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One Hundred Years of Hysteria

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A tough old word like hysteria dies very hard. It tends to outlive its obituarists.

—AUBREY LEWIS (1975, p. 12)

Actually, there have been at least 4,000 years of hysteria. Veith (1965, 1977) has traced the history of the syndrome from pharaonic Egypt, where the theory of the migrating uterus was first propounded, through Sydenham (1697), who was first to recognize the emotional cause of the syndrome, to Cullen (1796), who coined the terms *neurology* and *neurosis*, to Charcot (1877/1888), the neurologist who discovered that hysterics were highly suggestible, and, finally, to the beginnings of modern psychiatry in the late 19th century (for other historical treatments, see Merskey 1979, 1983; Micale, 1990). Hysteria has been centrally involved in major conceptual changes in psychiatry and clinical psychology. Ellenberger (1970) has shown how the analysis of hysteria by Freud (Breuer & Freud, 1893–1895/1955; Freud, 1905/1953) and Janet (1889, 1894/1901, 1907)—both of whom studied with Charcot—promoted psychogenic as opposed to somatogenic theories of the origins of mental illness.[1] More recently, analyses of hysteria by Szasz (1961) and of other forms of mental illness by others (Goffman, 1961; Scheff, 1966) laid the foundation for a social-psychological critique of the medical model of psychopathology, and a reinterpretation of mental illness in terms of strategic self-presentation, interpersonal communication, and social control (for a defense of the medical model of psychopathology, which emphasizes that it has nothing to do with somatogenesis, see Siegler & Osmond, 1974; Shagass, 1975).

Although the entire history of hysteria is fascinating, this chapter focuses only on the 100-year period since Charcot's death in 1893, which was also the year Janet's medical thesis on hysteria was accepted, and the

year that Breuer and Freud published the "Preliminary Communication" of their *Studies on Hysteria* (1955). (For psychologists interested in hysteria and dissociation, 1993 was a centennial year on three accounts.) It is over this period of time that clinicians and researchers began to develop a nosology for the systematic classification of psychopathology. As Grob (1991) has noted, up until the 19th century physicians were concerned with the diagnosis of individual cases, but not in organizing their diagnoses according to a systematic nosology (there were no psychologists yet, of course). A number of classifica-tory schemes had been proposed by Cullen (1796), Kant (1798/1978), and Pinel (1806/1962), among others. For example, Pinel distinguished between "physical" and "moral" neuroses, anticipating the later division between organic and functional mental illnesses (Spitzer et al., 1992). However, these nosologies did not play a major role in the treatment of individual patients.

Beginning in the mid-19th century, several factors combined to lead psychiatrists (then often known as alienists) to seek agreement on a classification scheme. One of these factors, at least in the United States, was the increasing scope and sophistication of the census and other social databases as instruments of social policy. Another was the need to imitate those branches of medicine that had gained social acceptance by virtue of their actual or perceived scientific rigor. As late as 1917, the Committee on Statistics of the American Medico-Psychological Association (AMPA; the forerunner of the American Psychiatric Association) noted that "The present condition with respect to the classification of mental diseases is chaotic. This condition of affairs discredits the science of psychiatry and reflects unfavorably on our Association" (Salmon, Copp, May, Abbot, & Cotton, 1917, p. 255).

This situation changed quickly. In 1918 the AMPA issued the first edition of the *Statistical Manual for the Use of Institutions for the Insane* (hereafter, referred to as *Statistical Manual*; Committee on Statistics, 1918), which was adopted by the Bureau of the Census in 1920 and went through nine revisions (Committee on Statistics, 1942). This laid the foundation for successive editions of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM; American Psychiatric Association, 1952, 1968, 1980, 1987, 1994). Interestingly, the place of hysteria in these manuals has shifted from one edition to another—it is as if institutional psychiatry does not know quite what to do with it. This is poetic justice, perhaps, for a syndrome that was once attributed to a wandering uterus, but it also reflects deep confusion and misunderstanding within psychiatry. The burden of this chapter is to argue that the mental disorders once grouped under the label "hysteria" are wrongly classified in the latest versions of DSM, and to argue for a new classificatory scheme.

THE HYSTERIA OF CHARCOT AND JANET

For Sydenham (1697), hysteria was characterized by physical symptoms produced by emotional causes (and women were hysterics, while men were hypochondriacs); for Briquet (1859), it was the label for patients with multiple and chronic physical complaints; for Breuer and Freud (1893–1895/1955), it was essentially synonymous with neurosis. However, modern psychopathology owes the classic description of hysteria to Charcot (1877/1888) and Janet (1889, 1894/1901, 1907), and it is to these authorities that we must turn for background on the present-day diagnostic dilemma of hysteria (see also Havens, 1973).

Veith (1965) related the story of how Charcot, a neurologist who was at the time chief of Medical Services at the Salpêtrière hospital in Paris, became interested in hysteria. A reorganization of the hospital led hysterics and nonpsychotic epileptics to be separated from the insane and housed together, after which the hysterical patients quickly began to display epileptiform seizures, which Charcot called *hystero-epilepsy* or *hysteria major*. Closer examination of these patients revealed a number of *stigmata*, or persistent symptoms, of what he called *hysteria minor*, such as disturbances of the tactile sense, including anesthesia and hyperesthesia; disturbances of the special senses, including deafness and tunnel vision; and disturbances of motor function, including paralysis. While the hysterical stigmata mimicked that of organic illnesses, Charcot recognized them as due to emotional disturbances and suggestion. Charcot's theory of hysteria is one of diathesis and stress: a hereditary constitutional weakness combines with a more or less traumatic stress to precipitate "dynamic or functional lesions" (Charcot, 1877/1888, p. 278). These lesions, he maintained, could not be localized with the tools available to him.

Charcot's insistance that hysterical stigmata result from lesions in the central nervous system did not prevent him from understanding the role of psychological factors in their genesis. He noted, for example, that the stressful events precipitating the illness did not always seem traumatic to an objective observer. What was important was that they were *subjectively* traumatic to the patient. This emphasis on what we would now call the mental representation of trauma, as opposed to trauma that is objectively defined, may be viewed as the official beginning of the psychogenic perspective in psychopathology; interestingly, it was put forth by a dedicated neurologist.

Moreover, hysterics were enormously suggestible, as indicated by the hystero-epilepsy observed after the reorganization of the Salpêtrière. The neurologist Babinski, along with Janet and Freud, one of Charcot's most distinguished pupils, even proposed that hysteria be renamed *pithiatism*,

after the Greek word *peitho*, meaning "to persuade" (Babinski & Froment, 1918). In addition, it was widely known that the symptoms of hysteria could be artificially produced by means of hypnotic suggestion. In fact, Charcot's demonstrations of hypnosis were the first laboratory models of psychopathology (Kihlstrom, 1979, 1984; Kihlstrom & McGlynn, 1991). Finally, there was the fact that hysterical stigmata conformed to the patients' ideas about the nervous system, rather than to neuroanatomical realities. It can be argued that Charcot's reputation as the most distinguished neurologist of his time lent credibility to the psychogenic theories of mental illness later formulated by Janet, Freud, and others (Havens, 1973; Veith, 1965, 1977).

Janet (1889, 1894/1901, 1907) followed Charcot in emphasizing the importance of a close clinical examination of his patients. His intention was to do for the neuroses what Kraepelin had done for the psychoses, that is, organize them into a coherent nosological scheme. While Kraepelin classified the psychoses into manic–depressive illness and dementia praecox, Janet classified the neuroses into hysteria and psychasthenia. Psychasthenia comprised obsession, anxiety, depression, and hypochondriasis. Janet thought that with each of these, the patient knows precisely what is wrong, and is distressed about it. With hysteria, however, there is a constriction of awareness: the hysterically amnesic does not know what he or she remembers, the hysterically blind and deaf do not know what they see or hear; and, as indicated by *la belle indifférence*, they do not care that they cannot remember, or see, or hear. Thus, Janet (1894/1901) defined anesthesia and amnesia as the primary stigmata of hysteria.

Janet (1889) analyzed mental life into elements known as psychological automatisms, each consisting of a complex act responsive to the details of the stimulus situation, and each including an idea and an emotion. In normal individuals, the entire set of psychological automatisms is bound together and accessible to phenomenal awareness and voluntary control. Under conditions of stress, however, one or more automatisms are split off, or dissociated, from the rest, inaccessible to phenomenal awareness and independent of voluntary control. According to Janet, somnambulistic states, to take one example, represent the involuntary repetition, in fantasy, of some forgotten traumatic experience, followed by amnesia for both the original event and its imagined repetition. In hysterical tunnel blindness, the patients are unaware of objects in the periphery of their visual field, even though their behavior is obviously influenced by these stimuli. Janet's ideas about dissociation were embraced by many of his contemporaries, including William James and Morton Prince in America (Fuller, 1982, 1986), but eventually were superseded by the psychoanalytic theory of Freud and his followers, which emphasized repression and an entirely different, impersonal construal of unconscious mental life.

More recently, Janet's point of view has been revived by Hilgard (1977/1986) in the form of the neodissociation theory of divided consciousness. Neodissociation theory conceptualizes the mind as a set of module-like components that monitor, organize, and control mental functioning in various domains. These mental structures are able to seek and avoid certain inputs, and facilitate or inhibit certain outputs, in accordance with global and local demands and intentions. In addition, these structures are subordinate to a central executive structure, which serves as the end point for all stimulus inputs, and as the origin of all motor outputs. This central structure is the cognitive basis for the phenomenal experience of awareness and intentionality. According to neodissociation theory, certain conditions can alter the relations among the various substructures, and between each substructure and the central executive. If, for example, the communication lines that link one substructure with the central executive structure are disrupted, then that substructure will process inputs, and generate outputs, independent of the central executive. This is the sort of thing that happens in hysteria. Indeed, Hilgard's neodissociation theory takes Janet's analysis of hysteria and places it within the conceptual framework of contemporary cognitive psychology.

In his foreword to Janet's *The Mental State of Hystericals* (1894/1901), Charcot the neurologist wrote that "hysteria is largely a mental malady" (p. v). Charcot and Janet differed on the role played by constitutional diathesis and organic lesions in hysteria. But they agreed on one fundamental point: Hysteria, as manifested in the stigmata that are pathognomonic of the disease, is fundamentally a disorder of consciousness. Hysterical patients are unaware of events of which they should, under ordinary circumstances, clearly be cognizant; and they are influenced by ideas, memories, affects, and needs that are excluded from or denied introspective access. This crucial point has been progressively lost over the successive revisions of DSM (for a similar argument, see Nemiah, 1991).

THE OFFICIAL DIAGNOSIS OF HYSTERIA: FROM DSM-I TO DSM-IV

The first edition of the AMPA *Statistical Manual* (Committee on Statistics, 1918) listed 22 categories of mental illness, which mostly consisted of various forms of psychosis (reflecting the somatogenic orientation of psychiatry at the time), but which also included a grouping of "psychoneuroses and neuroses." The 10th edition (Committee on Statistics, 1942) had a more differentiated list of psychoneuroses, including separate subcategories for hysteria, compulsive states, neurasthenia, hypochondriasis, reactive depression, anxiety state, anorexia nervosa, and mixed psycho-

neuroses. After World War II, a number of social and institutional pressures, including the increasing power of the psychodynamic viewpoint of Freud and the psychosocial perspectives advocated by Adolph Meyer and the brothers Menninger, a concern for reliable (if not necessarily valid) diagnostic procedures, and an expanded interest in less severe forms of mental illness necessitated a revised nosology, which became the American Psychiatric Association's *Diagnostic and Statistical Manual of Mental Disorders* (Grob, 1991).

DSM-I

The first edition of DSM (American Psychiatric Association, 1952), which was heavily influenced by psychoanalysis and other psychodynamic theories of mental illness (Grob, 1991), divided the psychiatric syndromes into three superordinate categories:

1. Disorders caused by impairment of brain tissue function
2. Mental deficiency
3. Disorders of psychogenic origin

The last category was a real catchall; it included any "general difficulty in adaptation of the individual . . . in which any associated brain function disturbance is secondary to the psychiatric disorder" (American Psychiatric Association, 1952, p. 9). It also included psychotic disorders, which were further subdivided into affective reactions, schizophrenic reactions (including the five traditional subtypes of simple, hebephrenic, catatonic, paranoid, and undifferentiated), and paranoid reactions; psychophysiological autonomic and visceral disorders (further classified according to the organ system affected); personality disorders (divided into personality pattern disturbances, personality trait disturbances, and sociopathic personality disturbance); transient situational personality disorders (e.g., adjustment reactions of adulthood, infancy, childhood, adolescence, and late life); and, last, but by no means least, the psychoneurotic disorders.

The psychoneurotic disorders, in turn, consisted of anxiety reaction, dissociative reaction, conversion reaction, phobic reaction, obsessive compulsive reaction, depressive reaction, and a wastebasket category of other forms of psychoneurotic reaction (see Figure 17.1). In line with psychoanalytic theory, the neuroses were characterized by either the conscious experience of anxiety (anxiety reaction, phobic reaction, and obsessive compulsive reaction) or the unconscious control of anxiety by means of certain defense mechanisms. Depressive reaction, in which anxiety is "allayed, and hence partially relieved, by depression and self-depreciation" (p. 33), stood somewhat outside of this dichotomy.

It should be noted that two forms of psychoneurosis were specifically related to the classic concept of hysteria: *dissociative reaction*, which included

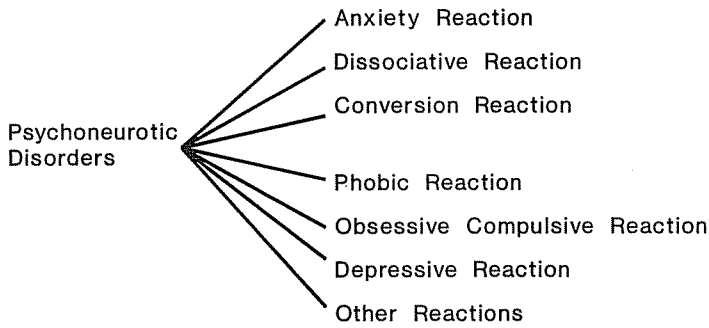

FIGURE 17.1. Classification of dissociative and conversion disorders in DSM-I.

"various symptomatic expressions, such as depersonalization, dissociated [multiple] personality, fugue, amnesia, dream state, somnambulism, etc." (p. 32); and *conversion reaction*, which included such "symptomatic manifestations . . . as anesthesia (anosmia, blindness, deafness), paralysis (paresis, aphonia, monoplegia, or hemiplegia), and diskinesis (tics, tremors, posturing, catalepsy)" (p. 33). Again in line with psychoanalytic thought, both dissociative and conversion disorders were attributed to the repression of anxiety-evoking impulses. This schema essentially followed the tradition initiated by Charcot and Janet, which has been abandoned in the latest editions of the DSM.

Moreover, it should be noted that both the dissociative and conversion reactions, being forms of psychoneurosis, were clearly differentiated from the psychophysiological disorders. (DSM-I had no listing for a hysterical personality disorder.) DSM-I explicitly rejected the label of *somatization reaction* for this category, on the grounds that the disorders in question were not simply psychoneurotic in nature. The psychophysiological disorders were further differentiated from the conversion disorders due, in part, to the idea that the symptoms of the latter reflected actual structural changes in the organs involved.

DSM-II

DSM-II (American Psychiatric Association, 1968) represented a rearrangement and expansion of the DSM-I nosology. The ten categories at the highest level of organization were:

1. Mental retardation
2. Organic brain syndromes
3. (Functional) psychoses

4. Neuroses
5. Personality disorders
6. Psychophysiologic disorders
7. Special symptoms (e.g., sleep or eating disorders)
8. Transient situational disturbances
9. Behavior disorders of childhood and adolescence
10. Various nonpsychiatric maladjustments

The changes introduced in DSM-II were subtle but important. For example, the term *reaction* was generally dropped from the nosology. The term *schizophrenia* replaced that of *schizophrenic reaction*, *major affective disorders* that of *affective reaction*, and *anxiety neurosis* that of *anxiety reaction*. The foreword to DSM-II averred that "The change of label has not changed the nature of the disorder" (American Psychiatric Association, 1968, p. ix), but this nosological move must have reflected an emerging construal of the psychoses and neuroses as legitimate medical syndromes, if not full-fledged disease entities.

Nevertheless, the neuroses were still characterized in psychodynamic terms as involving either the direct expression or the unconscious control of anxiety. The subcategories of neurosis were expanded to include neurasthenic neurosis (also known as neurasthenia), depersonalization neurosis (depersonalization syndrome), and hypochondriacal neurosis, as well as the traditional categories of anxiety neurosis, obsessive compulsive neurosis, and depressive neurosis (Figure 17.2). Interestingly, the broad category of neuroses now included a single subcategory of *hysterical neurosis* "characterized by involuntary psychogenic loss or disorder of function" (p. 39). Hysterical neurosis itself was further subdivided into two types: *hysterical neurosis, conversion type*, in which "the special senses or voluntary nervous system are affected, causing such symptoms as blindness, deafness, anosmia, anaesthesias, paraesthesias, paralyses, ataxias, akinesias, and dyskinesias" (pp. 39–40); and *hysterical neurosis, dissociative type*, in which "alterations may occur in the patient's state of consciousness or . . . identity, to produce such symptoms as amnesia, somnambulism, fugue, and multiple personality" (p. 40).

Like DSM-I, DSM-II insisted that *hysterical neurosis, conversion type* be distinguished from the psychophysiological disorders. In part, the distinction was made in terms of the organs involved, that is, those of the central or somatic nervous system versus those of the autonomic nervous system. Another distinction, retained from DSM-I, was that the psychophysiological disorders involved actual changes in organ function.

Similarly, the personality disorders included a subcategory of hysterical personality (also known as histrionic personality disorder), which was "characterized by excitability, emotional instability, over-reactivity, and

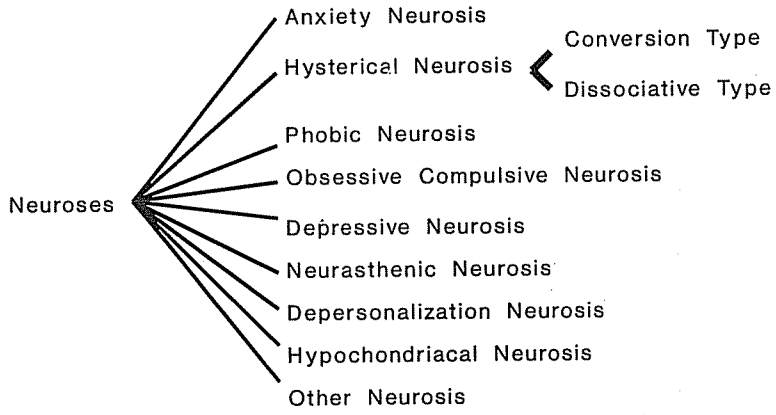

FIGURE 17.2. Classification of dissociative and conversion disorders in DSM-II.

self-dramatization" (p. 43; see also Shapiro, 1965; Horowitz, 1977). DSM-II clearly indicated that hysterical personality should be distinguished from hysterical neurosis, a point repeatedly insisted upon by Chodoff (1954, 1974; Chodoff & Lyons, 1958; see also Ziegler, Imboden, & Meyer, 1960). Nevertheless, the confusion among conversion hysteria, hysterical personality, and other "hysterical" syndromes had begun. For example, a literature review by Temoshock and Atkisson (1977) listed at least 14 different nosological labels related to hysteria (and proposed a psychosocial theory to explain them): dissociated personality, hysterical character disorder, idiosyncratic hysterical psychosis, sociopathy, Briquet's syndrome, anorexia nervosa, conversion symptoms, hysterical personality traits, socially patterned hysterical psychosis, dissociated states, hysterical contagion, millenarian movements, and reactive movements (for a similar list, see Roy, 1982).

DSM-III and DSM-III-R

DSM-III (American Psychiatric Association, 1980) changed everything. As Wilson (1993) has noted, this edition marked a transformation in American (and, for that matter, world) psychiatry, specifically, a return to Kraepelin-style descriptive diagnosis (as opposed to defining syndromes according to speculative theories such as psychoanalysis), and a shift from the psychodynamic, psychosocial, and biopsychosocial models of mental illness to a more strictly biological view. According to the psychosocial model, mental health, neurosis, and psychosis are on a single continuum; mental illness is a "reaction" to environmental events and circumstances. Hence,

Kraepelinian categorical diagnosis are viewed as both arbitrary and irrelevant—useful for statistical reporting and collecting third-party payments, perhaps, but not informative in the treatment of individual cases. Partly in reaction to the antipsychiatry movement, and partly to counter the hegemony of the psychodynamic, psychosocial, and biopsychosocial models of mental illness, DSM-III revived a concern for a theory-neutral descriptive psychopathology, of the sort preferred by Kraepelin, and for the first time canonized a set of diagnostic procedures as well as diagnostic labels.[2] The 17 major classifications of mental illness (18 categories in DSM-III-R) were:

1. Disorders of infancy, childhood, or adolescence
2. Organic mental disorders
3. Substance abuse disorders (in DSM-III-R, "psychoactive")
4. Schizophrenic disorders
5. Paranoid disorders (in DSM-III-R, "delusional" disorders)
6. Psychotic disorders not elsewhere classified
7. Affective disorders (in DSM-III-R, "mood" disorders)
8. Anxiety disorders
9. Somatoform disorders
10. Dissociative disorders
11. Psychosexual disorders (in DSM-III-R, "sexual" disorders)
12. Factitious disorders
13. Disorders of impulse control not elsewhere classified
14. Adjustment disorders
15. Psychological factors affecting physical condition
16. Personality disorders (listed on Axis II)
17. Conditions not attributable to a mental disorder
18. Sleep disorders (added in DSM-III-R).

While acknowledging the superiority of etiological over symptom-based classification, the framers of DSM-III argued that the origins of most mental disorders were unknown, and so merely speculative etiological labels should be abandoned. Accordingly, an attempt was made to eliminate *psychosis* and *neurosis* as superordinate categories (although the terms themselves were marginally preserved, in a political compromise with the psychoanalysts; Grob, 1991). The term *hysteria* was completely eliminated, along with theoretical statements about the unconscious expression of anxiety in dissociative and conversion symptoms. Classification was put on theory-neutral, descriptive grounds. Interestingly, however, DSM-III (and DSM-III-R) retained the far-from-theory-neutral terms *dissociative disorder* and *conversion disorder*, and added another theory-laden term for good measure: *somatization disorder* (for a review, see Kellner, 1990). These terms are the only diagnostic labels remaining in the psychiatric nosology that invoke, or at least imply, a specific etiological mechanism. Obviously, all three are derived

from the traditional concept of hysteria. Lewis (1975) was right: Even when the word *hysteria* dies, its underlying concept lives on.

At the same time, however, DSM-III marked a radical departure from the traditional classification of hysteria (Hyler & Spitzer, 1978). Not only was conversion disorder separated from dissociative disorder, conversion disorder was joined to somatization disorder, psychogenic pain disorder, and hypochondriasis under the new rubric of *somatoform* disorder. The somatoform disorders remained clearly separated from the psychophysiological disorders (renamed *psychological disorders affecting physical condition*, a sorry label; hereafter, in this chapter, they will be called *psychosomatic disorders*), hysterical personality disorder (renamed *histrionic personality disorder*), and the new category of *factitious disorders* (including Munchausen's syndrome). The separation between dissociative and conversion disorders was further reinforced by listing them at different levels in the taxonomy: Dissociative disorder was listed in a superordinate category, on the same level as schizophrenia and affective disorder, while conversion disorder was listed as a subset of the somatoform category. This structure was preserved in DSM-III-R (see Figure 17.3).

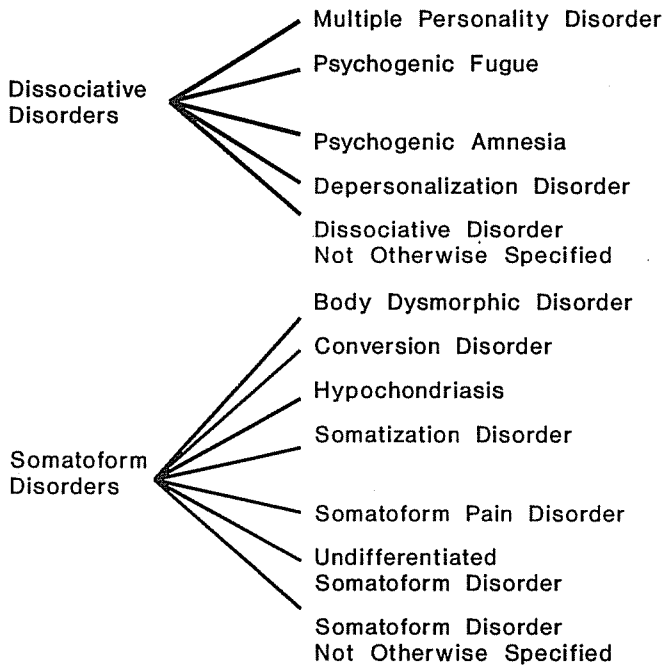

FIGURE 17.3. Classification of dissociative and conversion disorders in DSM-III and DSM-III-R.

So far as the dissociative and somatoform disorders are concerned, the essential structure of the diagnostic system is not changed in DSM-IV (American Psychiatric Association, 1994). In particular, as indicated in Figure 17.4, conversion disorder will remain separated from dissociative disorder, and listed under the rubric of somatoform disorder (Martin, 1992, 1993).

This choice perpetuates what Ryle (1949), in another context, called a category mistake:

> It is not merely an assemblage of particular mistakes. It is one big mistake and a mistake of a special kind . . . It represents the facts of mental life as if they belonged to one logical type or category (or range of types or categories) when they actually belong to another. (p. 17)

ALTERATIONS IN CONSCIOUSNESS IN CONVERSION AND DISSOCIATION

DSM-III created the new superordinate category of somatoform disorder in order to encompass all syndromes characterized by physical symptoms suggesting physical disturbances, but in the absence of organic (anatomical or physiological) findings that would explain the symptoms (for reviews, see Cloninger, 1986; Maxmen, 1986). In somatization disorder, the patient has an extended history of recurrent, multiple bodily complaints. *Somatization disorder* is also known as Briquet's (1859) syndrome, and is frequently identified with classical hysteria (Goodwin & Guze, 1989; Kellner, 1990). Also identified with classical hysteria, of course, is *conversion disorder* (or *hysterical neurosis, conversion type*), which is defined as an alteration or loss of physical function (e.g., paralysis, aphonia, blindness, and anesthesia) expressive of a psychological conflict or need (Ford & Folks, 1985; Lazare, 1981; Martin, 1992, 1993). *Body dysmorphic disorder*, previously known as dysmorphophobia, is diagnosed when the patient is preoccupied with an imagined defect in his or her physical appearance. When the belief is of delusional intensity, the patient may be diagnosed with *delusional* (or paranoid) *disorder, somatic type* (Phillips, 1991; Phillips & Hollander, 1994; Phillips, McElroy, Keck, Pope, & Hudson, 1993). In *somatoform pain disorder*, there is a preoccupation with pain, while in *hypochondriasis* (or hypochondriacal neurosis) there is a preoccupation with serious illness—either the patient believes that he or she has a certain illness, or fears contracting it.

Cloninger (1986) has characterized somatization disorder as *chronic hysteria* (on the basis of its slow onset and poor prognosis) and conversion disorders—along with dissociative disorders—as *acute hysteria* (on the basis

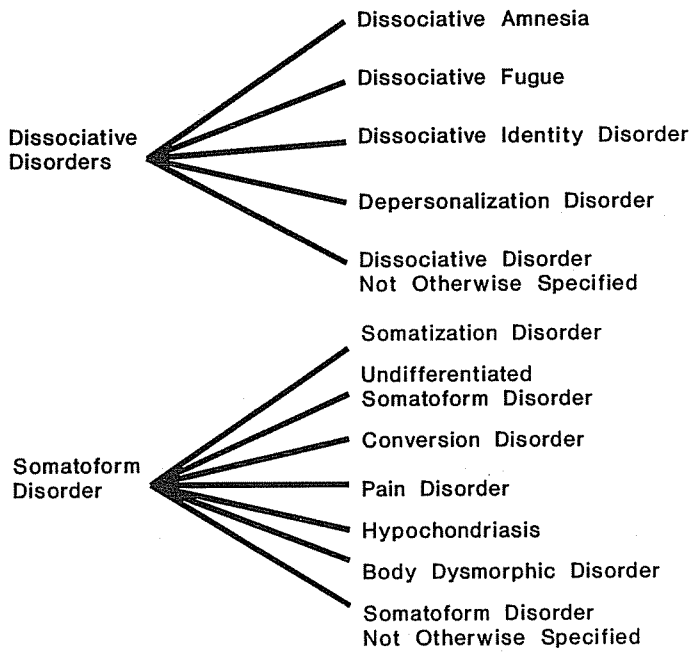

FIGURE 17.4. Classification of dissociative and conversion disorders in DSM-IV.

of rapid onset and remission). However, there is no evidence that individuals who present with conversion symptoms graduate to Briquet's syndrome; hence, the acute–chronic continuum seems inappropriate in this case. As a group, the somatoform disorders are distinguished from other mental disorders involving physical complaints by four criteria (Hyler & Spitzer, 1978): No physical mechanism explains the symptoms; the symptoms are plausibly linked to psychological factors (e.g., traumatic stress or interpersonal conflict) in the patient's life; the initiation of the symptom is not under voluntary control; and the symptom does not necessarily meet an obvious social goal (e.g., financial compensation or insanity defense). Thus, with *factitious disorders* and *malingering* the symptoms are under voluntary control, while with *psychological factors affecting physical condition* and *undiagnosed physical illness* they are not.[3] In factitious disorder and undiagnosed physical illness, there is no environmental goal met by the symptom, while in malingering the goal is obvious. In the case of the psychosomatic disorders, there are obvious physical mechanisms (e.g., chronic autonomic arousal) that explain the symptom (for a discussion of the classification of these disorders, see Looney, Lipp, & Spitzer, 1978).

The rationale for separating dissociative from conversion disorders, and

for linking the latter with somatization disorder, hypochondriasis, and the like, was that conversion disorders involve physical symptoms, while the dissociative disorders involve alterations in consciousness affecting memory and identity. This argument misses the essential feature that joins the conversion disorders to the dissociative disorders, and distinguishes them from the somatoform, factitious, and psychosomatic disorders. In the somatoform, factitious, and psychosomatic disorders the symptoms are physical, of the sort usually treated by internal medicine. The somatizer complains of abdominal pain, then pain in the extremities, later shortness of breath and heart palpitations. The patient with dysmorphophobic disorder complains that her nose is bumpy or her lips crooked, that he is losing his hair or his neck is shrinking. Despite all medical assurances, the hypochondriac fears that he has cancer, or AIDS, or has suffered a heart attack, or will soon do so. In the dissociative and conversion disorders, however, the symptoms in question are mental and pseudoneurological. Conversion disorder patients complain of anesthesia or paralysis, deafness or tunnel vision; dissociative disorder patients cannot remember what they have been doing, or even who they are.

Put another way, both the conversion and dissociative disorders are fundamentally disorders involving the monitoring and controlling functions of consciousness. These patients do not consciously perceive, or remember, events that they should be clearly aware of, and they cannot consciously initiate voluntary motor activities. Nothing like these alterations in conscious awareness and control occurs in the case of the somatization, factitious, or psychosomatic disorders.

Dissociative Disorders

The alteration in consciousness is so clearly expressed in what are now called the dissociative disorders that it is incorporated into the very definition of the category: "a disruption in the usually integrated functions of consciousness, memory, identity, or perception of the environment" (American Psychiatric Association, 1994, p. 477; for reviews of the dissociative disorders, see Kihlstrom, 1992a; Kihlstrom, Tataryn, & Hoyt, 1993; Kihlstrom & Schacter, in-press; Schacter & Kihlstrom, 1989; Cardeña, Lewis-Fernandez, Bear, Pakianathan, & Spiegel, 1994). In *dissociative amnesia*, the core symptom is an inability to recollect autobiographical memories, that is, events, including traumatic experiences, from one's personal past. In *dissociative fugue*, knowledge of one's identity is lost, as well as knowledge of one's past. In *dissociative identity disorder* (more familiarly known as *multiple personality disorder*), a single individual alternates between two or more identities, each associated with its own set of autobiographical memories; these identities are separated by a symmetrical or asymmetrical amnesia, so that while the

person displays one personality, he or she is unaware of the others, and their associated autobiographical memories. In *depersonalization disorder*, the person perceives him- or herself (depersonalization) or the surrounding environment (derealization) as changed or unreal. As Reed (1974/1988, 1979) has pointed out, this amounts to a failure to recognize objectively familiar objects as such.

That these memory failures are disorders of consciousness is clearly indicated by the fact that they are reversible: When the amnesia or fugue terminates, the person can remember those events that he or she could not remember before. In the case of fugue, the person is now unable to remember events that transpired during the fugue state itself. With multiple personality disorder (MPD), when the multiple personality shifts from one alter ego to another, the memories associated with the new identity are recallable, while those associated with the former identity are not (until the personality shifts again). Reversible memory disorders are disorders of retrieval; they occur because the individual cannot, at the moment, gain access to memories that have been adequately encoded, and remain available in storage. Retrieval, and accessibility, are phenomena of consciousness as they entail bringing available memories into phenomenal awareness.

The role of consciousness in the dissociative disorders is further indicated by the fact that the temporarily inaccessible memories nevertheless exert an impact on the patient's experience, thought, and action outside of awareness. In other words, psychogenic amnesia, fugue, and multiple personality entail a dissociation between two forms of memory, explicit and implicit (Schacter, 1987; Schacter, Chiu, & Ochsner, 1993).[4] Explicit memory refers to the person's conscious, intentional recollection of some event. Implicit memory refers to any change in behavior that is attributable to a past event, independent of conscious recollection of that event. Explicit memory is evident in acts of recall and recognition, which require the person to bring some event into conscious awareness; implicit memory is exemplified by savings in relearning and priming effects, which do not have this constraint. That is to say, subjects can show priming or savings even though they do not remember the events responsible for these effects. Studies of brain-damaged patients with the organic amnesic syndrome, and of intact subjects who studied material under degraded encoding conditions, show that implicit memory can be spared even when explicit memory is grossly impaired.

The same contrast can be observed in patients suffering from the functional amnesia that accompanies dissociative disorders (for comprehensive reviews, see Kihlstrom & Schacter, in press; Kihlstrom et al., 1993; Schacter & Kihlstrom, 1989). For example, Madame D, a patient studied by Janet (1904), lapsed into an amnesic state after being victimized by a practical

joke. During her waking hours she had no recollection of the joke, or of anything that had happened during the 6 weeks prior to the event. Nevertheless, she froze in terror whenever she passed the location where the joke had been played, and her nocturnal dreams contained a clear but unrecognized representation of the event itself. Similarly, a rape victim studied by Christianson and Nilsson (1989) became upset whenever she returned to the scene of her assault, a footpath constructed from crumbled bricks; she also reported the frequent intrusion of the words "bricks" and "bricks and the path" into her thoughts. Another patient, Jane Doe, was unable to identify herself or provide any information about her family or place of residence; but when asked on a number of occasions to randomly dial a telephone, she consistently produced a number that proved to belong to her mother (Lyon, 1985).

In a classic experimental case study of multiple personality, Ludwig and his colleagues administered a number of memory tasks to each of several alter egos separated by an interpersonality amnesia (Ludwig, Brandsma, Wilbur, Bendfeldt, & Jameson, 1972). One personality could not recall paired associates taught to the other personalities, of whom he was unaware. Nevertheless, when this personality was asked to memorize, rather than recall, pairs taught to one of the others, it showed a considerable learning advantage. This clearly indicates that the unrecalled items were available in memory, and interacted with ongoing learning processes—albeit outside of awareness. Similarly, a more recent study of MPD showed poor explicit memory when one alter ego was asked to recall paired associates studied by another; however, tests of repetition priming and proactive interference, among others, gave evidence of the transfer of implicit memory across personalities (Nissen, Ross, Willingham, Mackenzie, & Schacter, 1988).

Conversion Disorders

Similar alterations of consciousness, albeit affecting perception in this case, are commonly observed in the conversion disorders, a group of syndromes whose central feature is symptoms or deficits affecting voluntary motor or sensory function" (American Psychiatric Association, 1994, p. 452; for reviews of the conversion disorders, see Kihlstrom, 1992a; Kihlstrom, Barnhardt, & Tataryn, 1992; Martin, 1992, 1993). DSM-IV divides the conversion disorders into three subcategories. *Conversion disorder with sensory symptom or deficit* includes blindness, double and blurred vision, tunnel vision, deafness, tactile anesthesia or analgesia, and hallucinations (including hypersensitivity to both touch and pain). *Conversion disorder with motor symptom or deficit* includes aphonia, impaired balance, paralysis or localized weakness, tremors, urinary retention, difficulty swallowing, and difficulty

breathing. *Conversion disorder with seizures or convulsions* includes fainting and other epileptiform behaviors. Although the official definition of conversion disorder does not include the word *consciousness*, it is apparent that all of the disorders listed involve impairments in normal conscious functioning, which affect either the sensory–perceptual system or the voluntary motor system.

Like the dissociative disorders, the conversion disorders are reversible. Patients with functional blindness and deafness eventually recover their sight and hearing; those with functional paralysis eventually regain voluntary movement. Such recuperation is rarely possible in cases of actual lesions in the central nervous system: Rehabilitation of actual nervous system damage involves learning to cope with lost functions rather than the restoration of those functions (Baker & Silver, 1987; Delargy, Peatfield, & Burt, 1986).

The role of consciousness in the conversion disorders is further supported by evidence that unseen, unheard, or unfelt stimuli nevertheless influence the patient's experience, thought, and action. In other words, these patients give evidence of experiencing spared *implicit perception*. Implicit perception is an extension of the concept of implicit memory (Kihlstrom, Barnhardt, & Tataryn, 1992). Explicit perception occurs with everyday acts of seeing and hearing, wherein the person consciously detects the presence of some object in the environment, or consciously perceives its form, motion, or distance from him- or herself. Implicit perception is exemplified by priming effects that are attributable to such stimuli, but which do not require conscious detection, description, or identification. Studies of brain-damaged patients with "blindsight," and of intact subjects who are presented with material under conditions of masking, show that implicit perception can be spared even when explicit perception is grossly impaired.

The distinction between explicit and implicit perception is easy to demonstrate, and in clinical practice constitutes the means by which the differential diagnosis of functional and organic symptoms can be made (Pincus & Tucker, 1985). In functional blindness, optokinetic nystagmus can be induced by slowly rotating a vertically striped drum in the patient's visual field; in cases of unilateral blindness, patients continue to perform visual tasks normally even though a distorting prism has been placed over the ostensibly good eye. And in the case of patients with functional deafness, they will speak more loudly when their speech is masked with white noise. An early series of case studies (which are now recognized as pioneering examples of behavioral assessment and therapy; Yates, 1970) applied the procedures of classical and instrumental conditioning to patients with functional deafness and tactile anesthesias (Cohen, Hilgard, & Wendt, 1933; Hilgard & Marquis, 1940; Malmo, Davis, & Barza, 1952–1953; Sears &

Cohen, 1933). The ability of anesthetic and deaf patients to acquire conditioned responses to tactile and auditory stimuli clearly indicates that the unfelt or unheard stimuli have been processed by the sensory–perceptual system. Hilgard and Marquis (1940) suggested that conditioning techniques permit the measurement of sensory thresholds without reliance on verbal report; apparently, they also permit such measurements without reliance on conscious sensation.

In a classic study, Brady and Lind (1961) employed another learning paradigm, the differential reinforcement of low rates, to investigate and treat a case of functional blindness. The patient was required to press a button every 18–21 seconds; correct responses were signaled by feedback, social approval, or hospital privileges. At the end of the baseline phase, the patient was responding within the target interval on the majority of trials—evidence of temporal conditioning. However, when the experimenters introduced a visual cue, which marked the onset of the target interval, response rate declined precipitously. Although the patient denied that he could see the light, visual perception was implicit in that his behavior was responsive to the stimulus. A follow-up study of this patient by Grosz and Zimmerman (1965), using another visual discrimination task, yielded similar results. In the words of Grosz and Zimmerman (1965, p. 260), the patient "was denying visual functioning while functioning visually" (for further discussion of this case, see Brady, 1966; Zimmerman & Grosz, 1966).

Such paradoxical behavior is evidence of implicit perception, and it has been observed in a number of studies of functional blindness (e.g., Bryant & McConkey, 1989; Grosz & Zimmerman, 1970; Keehn, Keuchler, & Wilkenson, 1973; Miller, 1968, 1986; Theodor & Mandelcorn, 1973) and functional deafness (Barraclough, 1966). Similarly, psychophysiological studies reveal essentially normal somatosensory event-related potentials in cases of functional anesthesia and hemianesthesia (Levy & Behrman, 1970; Levy & Mushin, 1973).

Paradoxes also can be observed in functional paralysis, and again provide the clinical basis for a differential diagnosis (Pincus & Tucker, 1985). Patients may display astasia and abasia while walking, for example, but rarely fall and even more rarely hurt themselves. In the case of functional hemiplegia, patients who are trying to lift their "good" leg while reclining will press down with their "bad" leg, just as intact individuals do. Rehabilitation specialists report that patients with functional paralysis usually show normal muscle tone and dampened reflexes, which clearly indicates that the connections between the effectors and cortical projection areas are intact (Baker & Silver, 1987; Withrington & Wynn-Parry, 1985).

Theorists of a social-psychological persuasion, such as Grosz and Zimmerman (1965; Zimmerman & Grosz, 1966), believe that such patients are malingering—that they see and hear perfectly well while denying vision or audition. Janet (1907) himself rejected such a "crude explanation"

(p. 171). Rather, he argued that the sensory–perceptual deficits observed in hysteria reflected the disconnection of sensations from the person's consciousness: While the sensations were denied representation in phenomenal awareness, they continued to affect reflex behaviors, and voluntary movements, outside of awareness. In other words, the functional anesthesias (defined broadly to include all the sensory modalities) are dissociative in nature.

TOWARD DSM-V: A NEW CLASSIFICATION OF DISSOCIATIVE DISORDERS

The dissociative disorders and conversion disorders share two fundamental features. Both are pseudoneurological in nature, and both involve disruptions in consciousness. Specifically, current and past experiences are temporarily inaccessible to phenomenal awareness, yet they continue to influence the person's experience, thought, and action in the form of implicit percepts and memories; goal-directed actions consciously planned by the individual cannot be executed; and the performance of other actions, planned outside of awareness, is accompanied by the experience of involuntariness. These considerations strongly suggest that the conversion disorders should be separated from the somatoform disorders, and rejoined to the dissociative disorders (their partners in DSM-II) as a major category of psychopathology (for a similar argument, see Nemiah, 1991).

In passing, it must be admitted that the collective label for these disorders, *dissociative*, is somewhat vexatious, because the term has a number of meanings in psychology. Thus, in cognitive psychology, dissociation refers to an experimental outcome in which some condition or manipulation affects one variable and not another. For example, neuropsychological studies of language processing reveal that global dyslexics perform poorly when reading both nonsense and irregular words while surface dyslexics can read nonsense words but not irregular words, and phonological dyslexics can read irregular but not nonsense words. The dissociation between reading irregular and nonsense words indicates that these two functions are served by two somewhat different brain systems. In this sense, dissociation is analogous to the interaction term in the analysis of variance, and does not necessarily implicate consciousness in any way.

Perhaps Janet's own French word *désagrégation* would eliminate confusion, but it is too late for such a move now. Moreover, both *désagrégation* and *dissociation* imply a particular etiology in the syndromes with which the terms are associated. As such, resort to either label would seem to violate the goal, in DSM-III and its successors, of adopting a theory-neutral, purely descriptive nosology. However, it is not necessary to embrace a dissociative explanation for functional amnesia, anesthesia, or paralysis. In what

follows, the label *dissociative* will not be construed as referring to some hypothetical underlying mechanism, but rather to the fact that, descriptively, the symptoms in question represent the exclusion of some mental contents or processes from consciousness.

The essential feature of the dissociative disorders, as defined here, is a disruption of the monitoring and controlling functions of consciousness—that is, failures of conscious perception, memory, or motor control—that are not attributable to insult, injury, or disease affecting brain tissue, or to the effects of psychoactive drugs (Kihlstrom & Schacter, in press; Schacter & Kihlstrom, 1989). Further, the disorders are reversible, either temporarily, by means of hypnosis or barbiturates, or permanently, when the crisis resolves and the dissociative process remits. Moreover, careful examination of cases of dissociative disorder usually yields evidence of intact functioning outside of awareness. In this respect, the clinical and experimental meanings of *dissociation* come together: With the dissociative disorders, there is evidence of a dissociation between explicit and implicit expressions of perception, memory, and action. In fact, evidence of such a dissociation might be considered the defining behavioral (as opposed to subjective) feature of the dissociative disorders.

Following this line of reasoning, the dissociative disorders may be further classified according to the specific mental functions affected by the alteration in consciousness: memory and identity, sensation and perception, and voluntary action (see Figure 17.5).

In the *dissociative disorders of memory and identity*, the division of consciousness affects memory, broadly construed to include both episodic knowledge of one's personal history and semantic knowledge of one's identity and self-concept. In *dissociative amnesia*, there is a loss of conscious access to all or part of the individual's autobiographical memory. During the amnestic period, the patient remains cognizant of who he or she is, but cannot bring certain memories into phenomenal awareness. In *dissociative fugue*, there is a temporary loss of identity as well as of autobiographical memory. Again, awareness shifts: During the fugue state, the patient is unaware of his or her premorbid identity and history; after the fugue has resolved, access to premorbid personal knowledge is restored, but awareness of events occurring during the fugue, and any identity adopted in that time, is lost. In *multiple personality disorder*, awareness alternates between two or more identities, each with its own associated fund of autobiographical memories. While the dissociative dysmnesias involve the loss of explicit memory, implicit expressions of memory may often bridge the amnesic barrier.

It should be noted that the memory symptoms of the dissociative disorders fall into two broad classes, negative and positive, paralleling the familiar division between positive and negative symptoms in schizophrenia (Andreason & Olsen, 1982). Thus, dissociative amnesia, and those cases

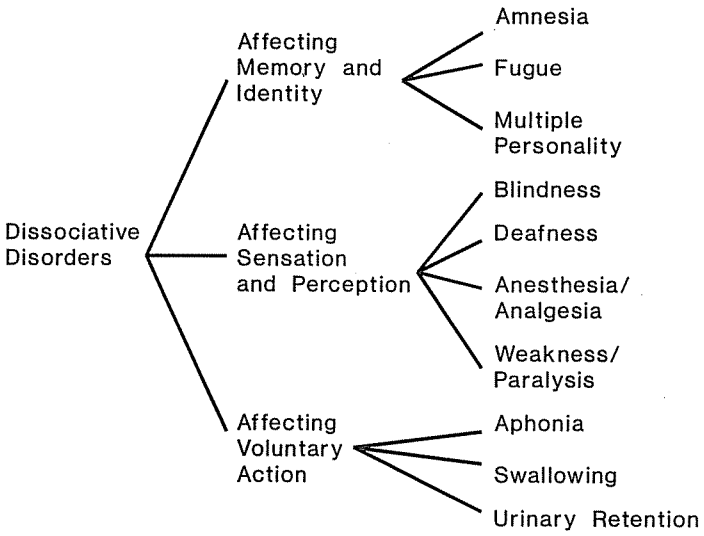

FIGURE 17.5. Classification of dissociative disorders proposed in this chapter: Specific examples listed on the right-hand side of the figure are only meant to serve as illustrations.

of dissociative fugue in which there is a loss but not a shift of identity, involve negative symptoms, that is, the absence of something (memory, identity) that is normally present. Instances of dissociative fugue in which there is an assumption of a new identity, and of MPD (in which there is the assumption of one or more new identities by definition), involve positive symptoms, that is, the presence of something (a second or third identity) that is normally absent. This distinction between positive and negative symptoms is also helpful in classifying the other syndromes of a dissociative nature.

In the *dissociative disorders of sensation and perception*, the division in consciousness affects sensory and perceptual function. The most familiar cases involve negative symptoms: blindness, deafness, tactile anesthesia, analgesia, etc. In each case, the patient is unaware of stimulation in a particular modality, although it should be noted that this unawareness may be partial, as in the case of tunnel vision (which affects the peripheral but not the central visual field), or even content-specific, as in the case of selective deafness to one voice but not another. In any event, it should be understood that the dissociative esthesias affect explicit perception; careful examination will show that implicit perception has been spared, in that the objects and events of which the patient is unaware will continue to influence ongoing experience, thought, and action.

Cases of positive sensory–perceptual symptoms, of which the most familiar type is *psychalgesia*, or functional pain affecting the back, joints, extremities, sexual organs, and bodily orifices should also be placed in the above category. *Psychalgesia* is frequently encountered in the neurological clinic. At first glance, the presence of such positive symptoms would seem to violate the core definition of the dissociative disorders—that they involve a disruption of awareness. If anything, the psychalgic patient has *too much* awareness. From the point of view of neodissociation theory, however, the patient's subjective experience of pain results from the construction of vivid mental images of pain, presumably based on memories of past experience. Hence, dissociation is a relevant term since the patients are unaware that they are generating these experiences for themselves. Because of this failure of reality monitoring, the pains are experienced as sensations rather than as images (for a further discussion, see Kihlstrom, in press).

Finally, the *dissociative disorders of motor function* affect the conscious control of voluntary efferent functions, rather than the conscious monitoring of present or past events. The most familiar instances involve localized weakness, paralysis of the extremities, aphonia, difficulty swallowing (what used to be called *globus hystericus*), and urinary retention. In neodissociative terms, some of these negative symptoms, such as weakness in the extremities, may reflect a failure of communication between the central executive, in which conscious intentions are formed, and the subordinate motor control centers that execute these ideas. Other cases, such as paralysis in the extremities, may reflect the active inhibition of motor activity, although, as in the case of psychalgesia, a dissociative barrier may prevent the person from being aware that this inhibition is the product of his or her own mental activities (Kihlstrom, in press).

The dissociative motor symptoms also include tics, fainting, vomiting, pseudoepileptic convulsions (which, like psychalgia, are rather commonly encountered in the neurological clinic), and the like. Again, it should be clear that despite appearances, these symptoms represent the generation of voluntary motor behaviors on the part of the patient. However, as in the cases of the mental images underlying psychalgia, and the active inhibitions involved in paralysis, the initiation of these behaviors is isolated from the patient's awareness, so that he or she experiences them as involuntary (for a more detailed analysis, see Kihlstrom, in press).

MECHANISM AND PHENOMENOLOGY

The framers of DSM-III and its successors were certainly right to return to Kraepelinian descriptive nosology as the starting place for scientific clinical practice, to emphasize the patient's phenomenology and behav-

ior over clinical inferences and speculations about etiology and underlying mechanisms, and to develop an explicit set of rules that would permit diagnoses to be reliably made. However, in their treatment of the syndromes historically associated with the concept of hysteria, two mistakes have been made and perpetuated.

The first, and less serious, mistake involved retaining the labels *dissociation*, *conversion*, and *somatization*. *Dissociation* can be a purely descriptive term, either referring to a lack of association between or integration of ideas, or to a state in which certain mental contents and processes are excluded from consciousness. Unfortunately, "dissociation" can also refer to the hypothetical process by which this exclusion is achieved—a process that is different from competing alternatives such as repression, suppression, or denial. In contrast *conversion* and *somatization* are exclusively theoretical in nature; they refer to particular processes postulated by psychoanalytic theory, by which unconscious conflicts are symbolically represented as manifest symptoms. *Dissociation* may still have a useful life as a purely descriptive label (Hilgard, 1977/1986), and it may be possible to reconstrue *somatization* in purely descriptive terms as well. But *conversion* appears to be too closely bound to psychoanalysis ever to serve as a theory-neutral descriptive label.

Fortunately, the label *conversion* is unnecessary: The conversion disorders are inherently dissociative in the descriptive sense in that they involve the exclusion of mental contents and processes from conscious awareness and control. At present, the conversion disorders are grouped with the somatoform disorders because they share "physical symptoms suggesting physical disorder" (Martin, 1994). But this is the second mistake. The symptoms of the conversion disorders are not physical, but mental in nature. And they do not suggest physical disorder, but rather a disorder in consciousness. Just as functional amnesia, fugue, and MPD are disorders of consciousness that affect memory and identity, so are functional blindness, deafness, anesthesia, and paralysis disorders of consciousness that affect sensation, perception, and voluntary action. Hence, we can only hope that when DSM-V appears, the term "conversion disorder" will have been abandoned once and for all, and that the syndromes now listed under this label will be returned to the dissociative disorders where they belong.

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NOTES

1. The earlier role of James Braid in setting the stage for psychogenic theories of psychopathology has been unfairly ignored. See appreciations by Kravis (1988), Kihlstrom (1992b), and Gravitz (1993).
2. Perhaps the most remarkable feature of DSM-III, however, was one that went largely unremarked: A clear change had occurred in the rules by which diagnostic decisions are made. Previous edition of DSM had provided succinct descriptions of the various mental disorders, leaving it up to the individual practitioner to arrive at a diagnosis for the individual case in the absence of specific rules. Before DSM-III, the diagnostic categories were considered, at least implicitly, to be classical proper sets, in which specific symptoms served as singly necessary and jointly sufficient defining features, yielding sharp boundaries between adjacent categories, the perfect nesting of superordinate and subordinate categories, and homogeneity within categories. However, analyses of actual diagnostic judgments showed that practitioners actually treated the syndromes as "fuzzy sets" represented by summary prototypes or sets of exemplars (Cantor & Genero, 1986; Cantor, Smith, French, & Mezzich, 1980; Horowitz, Post, French, Wallis, & Siegelman, 1981; Horowitz, Wright, Lowenstein, & Parad, 1981; Medin, Altom, Edelson, & Freko, 1982), a tendency that is broadly characteristic of natural-object categorization (Medin, 1989; Mervis & Rosch, 1981; Rosch, 1975; Smith & Medin, 1981). By emphasizing correlated rather than defining features, categorization by family resemblance, and within-category heterogeneity, DSM-III implicitly acknowledged that the fuzzy-set view of categorization applied to psychiatric diagnosis as well (Millon, 1991).
3. In DSM-IV, the label for these disorders is *psychological disorders affecting nonpsychiatric medical condition*—a diagnostic term that does not roll easily off the tongue. The Task Force on DSM-IV has solicited proposals for a simpler designation. Dare one propose a simple return to the former *psychophysiological disorders*, or even *psychosomatic disorders*?
4. Other labels for this distinction are direct versus indirect memory (Johnson & Hasher, 1987; Richardson-Klavehn & Bjork, 1988), memory with versus without awareness (Eich, 1984; Jacoby & Dallas, 1981), or declarative versus nondeclarative memory (Squire, Knowlton, & Musen, 1993).

REFERENCES

American Psychiatric Association. (1952). *Diagnostic and statistical manual: 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 of mental disorders* (3rd ed., rev). Washington, DC: Author.

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

Andreasen, N. C., & Olsen, S. (1982). Negative versus positive schizophrenia: Definition and validation. *Archives of General Psychiatry, 39*, 789–794.

Babinski, J., & Froment, J. (1918). *Hysteria or pithiatism and reflex nervous disorders in the neurology of war.* London: University of London Press.

Baker, J. H., & Silver, J. R. (1987). Hysterical paraplegia. *Journal of Neurology, Neurosurgery, and Psychiatry, 50*, 375–382.

Barraclough, M. (1966). A method of testing hearing based on operant conditioning. *Behaviour Research and Therapy, 4*, 237–238.

Brady, J. P. (1966). Hysteria versus malingering: A response to Grosz and Zimmerman. *Behaviour Research and Therapy, 4*, 321–322.

Brady, J. P., & Lind, D. L. (1961). Experimental analysis of hysterical blindness. *Archives of General Psychiatry, 4*, 331–339.

Breuer, J., & Freud, S. (1955). *Studies on hysteria.* In J. Strachey (Ed. and Trans.), *The standard edition of the complete psychological works of Sigmund Freud* (Vol. 2). London: Hogarth Press. (Original work published 1893–1895)

Bryant, R. A., & McConkey, K. M. (1989). Visual conversion disorder: A case analysis of the influence of visual information. *Journal of Abnormal Psychology, 98*, 326–329.

Briquet, P. (1859). *Traité clinique et thérapeutique à l'hystérie.* Paris: Ballière et Fils.

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. deS., & Mezzich, J. (1980). Psychiatric diagnosis as prototype categorization. *Journal of Abnormal Psychology, 89*, 181–193.

Cardeña, E., Lewis-Fernandez, R., Bear, D., Pakianathan, I., & Spiegel, D. (1994). Dissociative disorders. In A. Frances & T. Widiger (Eds.), *Source book for DSM-IV.* Washington, DC: American Psychiatric Press.

Charcot, J.-M. (1888). *Clinical lectures on certain diseases of the nervous system.* Detroit: Davis. (Original work published 1877)

Chodoff, P. (1954). A re-examination of some aspects of conversion hysteria. *Psychiatry, 17*, 75–81.

Chodoff, P. (1974). The diagnosis of hysteria: An overview. *American Journal of Psychiatry, 131*, 1073–1078.

Chodoff, P., & Lyons, H. (1958). Hysteria, the hysterical personality, and "hysterical" conversion. *American Journal of Psychiatry, 114*, 734–740.

Christianson, S.-A., & Nilsson, L.-G. (1989). Hysterical amnesia: A case of aversively motivated isolation of memory. In T. Archer & L.-G. Nilsson (Eds.), *Aversion, avoidance, and anxiety* (pp. 289–310). Hillsdale, NJ: Lawrence Erlbaum.

Cloninger, C. R. (1986). Somatoform and dissociative disorders. In G. Winokur & P. Clayton (Eds.), *The medical basis of psychiatry* (pp. 123–151). Philadelphia: W. B. Saunders.

Cohen, L. H., Hilgard, E. R., & Wendt, G. R. (1933). Sensitivity to light in a case of hysterical blindness studied by reinforcement inhibition and conditioning methods. *Yale Journal of Biology & Medicine, 6*, 61–67.

Committee on Statistics. (1918). *Statistical manual for the use of institutions for the insane.* New York: National Committee for Mental Hygiene.

Committee on Statistics. (1942). *Statistical manual for the use of institutions for the insane* (10th ed). New York: National Committee for Mental Hygiene.

Cullen, W. (1796). *First lines of the practice of physic, with practical and explanatory notes by John Rotheram.* Edinburgh: Bell, Bradfute.

Delargy, M. A., Peatfield, R. C., & Burt, A. A. (1986). Successful rehabilitation in conversion paralysis. *British Medical Journal, 292*, 1730–1731.

Eich, E. (1984). Memory for unattended events: Memory with and without awareness. *Memory and Cognition, 12*, 105–111.

Ellenberger, H. F. (1970). *The discovery of the unconscious: The history and evolution of dynamic psychiatry.* New York: Basic Books.

Ford, C. V., & Folks, D. G. (1985). Conversion disorders: An overview. *Psychosomatics, 26*, 371–383.

Freud, S. (1953). *Fragment of an analysis of a case of hysteria.* In J. Strachey (Ed. and Trans.), *The standard edition of the complete psychological works of Sigmund Freud* (Vol. 7, pp. 1–122.). London: Hogarth Press. (Original work published 1905)

Fuller, R. C. (1982). *Mesmerism and the American cure of souls.* Philadelphia: University of Pennsylvania Press.

Fuller, R. C. (1986). *Americans and the unconscious.* New York: Oxford University Press.

Goffman, E. (1961). *Asylums.* New York: Anchor Books.

Goodwin, D. W., & Guze, S. B. (1989). *Psychiatric diagnosis* (4th ed). New York: Oxford University Press.

Gravitz, M. A. (1993). Etienne Felix d'Henin de Cuvillers: A founder of hypnosis. *American Journal of Clinical Hypnosis, 36*, 7–11.

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

Grosz, H. J., & Zimmerman, J. A. (1965). Experimental analysis of hysterical blindness: A follow-up report and new experimental data. *Archives of General Psychiatry, 13*, 255–260.

Grosz, H. J., & Zimmerman, J. A. (1970). A second detailed case study of functional blindness: Further demonstration of the contribution of objective psychological laboratory data. *Behavior Therapy, 1*, 115–123.

Havens, L. L. (1973). *Approaches to the mind: Movement of the psychiatric schools from sects toward science.* Boston: Little, Brown.

Hilgard, E. R. (1986). *Divided consciousness: Multiple controls in human thought and action* (rev. ed.). New York: Wiley-Interscience. (Original work published 1977)

Hilgard, E. R., & Marquis, D. G. (1940). *Conditioning and learning.* New York: Appleton-Century-Crofts.

Horowitz, L. M., Post, D. L., French, R. d. S., Wallis, K. D., & Siegelman, E. Y. (1981). The prototype as a construct in abnormal psychology: 2. Clarifying disagreement in psychiatric judgments. *Journal of Abnormal Psychology, 90*, 575–585.

Horowitz, L. M., Wright, J. C., Lowenstein, E., & Parad, H. W. (1981). The prototype as a construct in abnormal psychology: I. A method for deriving prototypes. *Journal of Abnormal Psychology, 90*, 568–574.

Horowitz, M. J. (Ed.). (1977). *Hysterical personality.* New York: Jason Aronson.

Hyler, S. E., & Spitzer, R. L. (1978). Hysteria split asunder. *American Journal of Psychiatry, 135*, 1500–1504.

Jacoby, L. L., & Dallas, M. (1981). On the relationship between autobiographical memory and perceptual learning. *Journal of Experimental Psychology: General, 110*, 306–340.

Janet, P. (1889). *L'automatisme psychologique.* Paris: Felix Alcan.

Janet, P. (1901). *The mental state of hystericals.* New York: Putnam's Sons. (Original work published 1894)

Janet, P. (1904). Amnesia and the dissociation of memories by emotion. *Journal de Psychologie, 1*, 417–453.

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

Johnson, M. K., & Hasher, L. (1987). Human learning and memory. *Annual Review of Psychology, 38*, 631–668.

Kant, I. (1978). *Anthropology from a pragmatic point of view.* Carbondale, IL: Southern Illinois University Press. (Original work published 1798)

Keehn, J. D., Keuchler, H. A., & Wilkenson, D. A. (1973). Behavior therapy in a transactional context: The case of a blind drunk. *Behavior Therapy, 4*, 147–149.

Kellner, R. (1990). Somatization: Theories and research. *Journal of Nervous and Mental Disease, 178*, 150–160.

Kihlstrom, J. F. (1979). Hypnosis and psychopathology: Retrospect and prospect. *Journal of Abnormal Psychology, 88*, 459–473.

Kihlstrom, J. F. (1984). Conscious, subconscious, unconscious: A cognitive perspective. In K. S. Bowers & D. Meichenbaum (Eds.), *The unconscious reconsidered* (pp. 149–211). New York: John Wiley.

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

Kihlstrom, J. F. (1992b). Hypnosis: A sesquicentennial essay. *International Journal of Clinical and Experimental Hypnosis, 40*, 301–314.

Kihlstrom, J. F. (in press). Conscious awareness and the awareness of control. In H. J. Crawford (Ed.), *Consciousness and dissociation: Esssays in honor of Ernest R. Hilgard* Washington, DC: American Psychological Association.

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

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., & Schacter, D. L. (in press). Functional disorders of autobiographical memory. In A. Baddeley, F. Watts, & B. Wilson (Eds.), *Handbook of memory disorders.* London: John Wiley.

Kihlstrom, J. F., Tataryn, D. J., & Hoyt, I. P. (1993). Dissociative disorders. In P. J. Sutker & H. E. Adams (Eds.), *Comprehensive handbook of psychopathology* (2nd ed., pp. 203–234). New York: Plenum Press.

Kirmayer, L. J. (1986). Somatization and the social construction of illness experience.

In S. McHugh & T. M. Vallis (Eds.), *Illness behavior: A multidisciplinary model* (pp. 111–133). New York: Plenum Press.

Kluft, R. P., Steinberg, M., & Spitzer, R. L. (1988). DSM-II-R revisions in the dissociative disorders: An exploration of their derivation and rationale. *Dissociation, 1*, 39–46.

Kravis, N. M. (1988). James Braid's psychophysiology: A turning point in the history of dynamic psychiatry. *American Journal of Psychiatry, 145*, 1191–1206.

Lazare, A. (1981). Conversion symptoms. *New England Journal of Medicine, 305*, 745–748.

Levy, R., & Behrman, J. (1970). Cortical evoked responses in hysterical hemianaesthesia. *Electroencephalography and Clinical Neurophysiology, 29*, 400–402.

Levy, R., & Mushin, J. (1973). The somatosensory evoked response in patients with hysterical anaesthesia. *Journal of Psychosomatic Research, 17*, 81–84.

Lewis, A. (1975). The survival of hysteria. *Psychological Medicine, 5*, 9–12.

Looney, J. G., Lipp, M. R., & Spitzer, R. L. (1978). A new method of classification for psychophysiologic disorders. *American Journal of Psychiatry, 135*, 304–308.

Ludwig, A. M., Brandsma, J. M., Wilbur, C. B., Bendfeldt, F., & Jameson, D. H. (1972). The objective study of a multiple personality: Or, Are four heads better than one? *Archives of General Psychiatry, 26*, 298–310.

Lyon, L. S. (1985). Facilitating telephone number recall in a case of psychogenic amnesia. *Journal of Behavior Therapy and Experimental Psychiatry, 16*, 147–149.

Malmo, R. B., Davis, J. F., & Barza, S. (1952–1953). Total hysterical deafness: An experimental study. *Journal of Personality, 21*, 188–204.

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

Martin, R.L. (1994). DSM-IV diagnostic options for conversion disorder, proposed autonomic arousal disorder and pseudocyesis. In A. Frances & T. Widiger (Eds.), *Source book for DSM-IV*. Washington, DC: American Psychiatric Press.

Maxmen, J. S. (1986). *Essential psychopathology*. New York: W. W. Norton.

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

Medin, D. L., Altom, M. W., Edelson, S. M., & Freko, D. (1982). Correlated symptoms and simulated medical classification. *Journal of Experimental Psychology: Learning, Memory, and Cognition, 8*, 37–50.

Merskey, H. (1979). *The analysis of hysteria*. London: Bailliere Tindall.

Merskey, H. (1983). Hysteria: The history of an idea. *Canadian Journal of Psychiatry, 28*, 428–433.

Mervis, C. B., & Rosch, E. (1981). Categorization of natural objects. *Annual Review of Psychology, 32*, 89–115.

Micale, M. S. (1990). Hysteria and its historiography: The future perspective. *History of Psychiatry, 1*, 33–124.

Miller, E. (1968). A note on the visual performance of a subject with unilateral functional blindness. *Behaviour Research and Therapy, 6*, 115–116.

Miller, E. (1986). Detecting hysterical sensory symptoms: An elaboration of the forced choice technique. *British Journal of Clinical Psychology, 25*, 231–232.

Millon, T. (1991). Classification in psychopathology: Rationale, alternatives, and standards. *Journal of Abnormal Psychology, 100*, 245–261.

Nemiah, J. C. (1991). Dissociation, conversion, and somatization. *Annual Review of Psychiatry, 10*, 248–260.

Nissen, M. J., Ross, J. L., Willingham, D. B., Mackenzie, T. B., & Schacter, D. L. (1988). Memory and awareness in a patient with multiple personality disorder. *Brain and Cognition, 8*, 117–134.

Phillips, K. A. (1991). Body dysmorphic disorder: The distress of imagined ugliness. *American Journal of Psychiatry, 148*, 1138–1149.

Phillips, K. A., & Hollander, E. (1994). Body dysmorphic disorder: DSM-IV source book review. In A. Frances & T. Widiger (Eds.), *Source book for DSM-IV*. Washington, DC: American Psychiatric Press.

Phillips, K. A., McElroy, S. L., Keck, P. E., Pope, H. G., & Hudson, J. I. (1993). Body dysmorphic disorder: 30 cases of imagined ugliness. *American Journal of Psychiatry, 150*, 302–308.

Pincus, J. H., & Tucker, G. J. (1985). *Behavioral neurology* (3rd ed.). New York: Oxford University Press.

Pinel, P. (1962). *A treatise on insanity* (2nd. ed.). New York: Hafner. (Original work published 1806)

Reed, G. (1979). Anomalies of recall and recognition. In J. F. Kihlstrom & F. J. Evans (Eds.), *Functional disorders of memory* (pp. 1–28). Hillsdale, NJ: Lawrence Erlbaum.

Reed, G. (1988). *The psychology of anomalous experience* (rev. ed.). Buffalo, NY: Prometheus. (Original work published 1974)

Richardson-Klavehn, A., & Bjork, R. A. (1988). Measures of memory. *Annual Review of Psychiatry, 39*, 475–543.

Rosch, E. (1975). Cognitive representations of semantic categories. *Journal of Experimental Psychology: General, 104*, 192–223.

Roy, A. (Ed.). (1982). *Hysteria.* Chichester: John Wiley.

Ryle, G. (1949). *The concept of mind.* London: Hutchinson.

Salmon, T. W., Copp, O., May, J. V., Abbott, E. S., & Cotton, H. A. (1917). Report of the Committee on Statistics of the American Medico-Psychological Association. *American Journal of Insanity, 74*, 255–260.

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

Schacter, D. L., Chiu, C.-Y. P., & Ochsner, K. N. (1993). Implicit memory: A selective review. *Annual Review of Neuroscience, 16*, 159–182.

Schacter, D. L., & Kihlstrom, J. F. (1989). Functional amnesia. In F. Boller & J. Graffman (Eds.), *Handbook of neuropsychology* (Vol. 3, pp. 209–231). Amsterdam: Elsevier.

Scheff, T. J. (1966). *Being mentally ill: A sociological theory.* Chicago: Aldine.

Sears, R. R., & Cohen, L. H. (1933). Hysterical anesthesia, analgesia, and astereognosis. *Archives of Neurology and Psychiatry, 29*, 260–271.

Shapiro, D. (1965). *Neurotic styles.* New York: Basic Books.

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

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

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

Spitzer, R. L., First, M. B., Williams, J. B. W., Kendler, K., Pincus, H. A., & Tucker, G. (1992). Now is the time to retire the term "organic mental disorders." *American Journal of Psychiatry, 149*, 240–244.

Squire, L. R., Knowlton, B., & Musen, G. (1993). The structure and organization of memory. *Annual Review of Psychology, 44*, 453–495.

Sydenham, T. (1697). *Dr. Sydenham's method of curing almost all diseases, and description of their symptoms, to which are now added five discourses of the same author concerning the pleurisy, gout, hysterical passion, dropsy, and rheumatism* (3rd. ed.). London: Newman & Parker.

Szasz, T. S. (1961). *The myth of mental illness.* New York: Harper & Row.

Temoshock, L., & Atkisson, C. C. (1977). Epidemiology of hysterical phenomena Evidence for a psychosocial theory. In M. J. Horowitz (Ed.), *Hysterical personality* (pp. 143–222). New York: Jason Aronson.

Theodor, L. H., & Mandelcorn, M. S. (1973). Hysterical blindness: A case report and study using a modern psychophysical technique. *Journal of Abnormal Psychology, 82*, 552–553.

Veith, I. (1965). *Hysteria: The history of a disease.* Chicago: University of Chicago Press.

Veith, I. (1977). Four thousand years of hysteria. In M. J. Horowitz (Ed.), *Hysterical personality* (pp. 7–93). New York: Jason Aronson.

Wilson, M. (1993). DSM-III and the transformation of American psychiatry: A history. *American Journal of Psychiatry, 150*, 399–410.

Withrington, R. H., & Wynn-Parry, C. B. (1985). Rehabilitation of conversion paralysis. *Journal of Bone and Joint Surgery, 67*, 635–637.

Yates, A. J. (1970). *Behavior therapy.* New York: John Wiley.

Ziegler, F. F., Imboden, J. B., & Meyer, E. (1960). Contemporary conversion reactions: A clinical study. *American Journal of Psychiatry, 116*, 901–910.

Zimmerman, J., & Grosz, H. J. (1966). "Visual" performance of a functionally blind person. *Behaviour Research and Therapy, 4*, 119–134.