

Somatoform Disorders

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Disorders of the mind are often linked to physical symptoms and signs of psychiatric disorders: for example, the trembling, palpitations, and hyperpnea of panic disorder and the constipation, dry mouth, furrowed brow (omega sign), and weight loss of depressive disorders. The connection between somatic symptoms and depression or panic disorder, for example, is almost always readily accepted by patient and primary care physician; the task is to chart a course of action to treat symptoms with medication and psychological intervention. However, the primary care physician often misses the diagnosis of somatoform disorders for weeks to years. When finally informed of the diagnosis by the primary care physician, the patient is typically not prone to accept it. The patient is convinced of the physical origin of the undiagnosed distress and is equally convinced that the psyche plays no part in the disease process. Thus, when a patient has a somatoform disorder, a disconnect is initially established between patient and physician in the diagnostic process.

As the term somatoform implies, this heterogeneous group of disorders are at the mind-body interface: bodily symptoms as psychiatric disorder. Somatoform disorders thus exemplify mind-brain interactions: the brain, in ways still not well understood, sends various signals that impinge on the patient's awareness, indicating a serious problem in the body.

From a nosological perspective somatoform disorders were only grouped together for the first time in 1980 in the third edition of Diagnostic and Statistical Manual of Mental Disorders (DSM-III) as those disorders in which bodily sensations or functions, as the patient's predominant focus, are influenced by a disorder of the mind. This clustering was not based on theoretical construct or laboratory findings.

There are now five specific somatoform disorders: (1) somatization disorder, (2) conversion disorder, (3) pain disorder, (4) hypochondriasis, and (5) body dysmorphic disorder. There are also two residual categories that do not meet the fullblown criteria of the other five: (1) undifferentiated somatoform disorder and (2) somatoform disorder not otherwise specified.

In routine medical practice, classic cases of somatoform disorders are less common than less well differentiated syndromes.

Chronic fatigue syndrome is a recently prevalent disease without identified pathogen. The Centers for Disease Control (CDC) has recently set up criteria for this disorder. Several features of this syndrome seem to overlap with some of the somatoform disorders and might be considered in the differential diagnosis of somatization disorder. From a symptom perspective, chronic fatigue syndrome has much in common with certain other ill-defined maladies such as multiple chemical sensitivity syndrome, the syndrome of clinical hypoglycemia, the effort syndrome, and neurasthenia. There is no doubt that those afflicted with these disorders of unknown cause suffer. Those disorders do have some syndromic overlap with clearly defined psychiatric disorders such as the somatoform disorders.

CORE FEATURES

Characteristic of somatoform disorders are three enduring clinical features: (1) somatic complaints that suggest major medical maladies yet have no associated serious, demonstrable, peripheral organ disorder; (2) psychological factors and conflicts that seem important in initiating, exacerbating, and maintaining the disturbance; and (3) symptoms or magnified health concerns that are not under the patient's conscious control.

Because of their intense bodily perceptions, restricted level of physical functioning, and morbid beliefs, these patients have become convinced they harbor serious physical problems. Moreover, their symptoms are not willfully controlled. Whatever their faults and problems, these patients are not malingerers. Yet their physicians' physical and laboratory examinations persistently fail to evince significant substantiating data about physical infirmity other than the patients' vigorous and sincere complaints. Patients with somatoform disorders are convinced that their suffering comes from some type of presumably undetected and untreated bodily derangement.

DIAGNOSTIC ISSUES

When a somatoform disorder occurs without another comorbid psychiatric condition, the primary care physician and the patient usually do not initially consider a psychiatric condition. The patient's morbid preoccupation with bodily concerns, not emotional feelings or disordered interpersonal relationships, is paramount. Often this preoccupation is so severe that it interferes with the patient's capacity for living, loving, or working; it usually has sent the patient on a ceaseless odyssey from physician to physician looking for effective symptomatic relief.

Because of the patient's focus on bodily issues, the psychiatrist's questions (should such a referral ever occur) about stress and family matters might seem off the mark to the patient. However, research literature as well as the clinical experience of seasoned psychiatric consultants has demonstrated the utility of psychiatric input into managing somatoform patients from a psychosocial perspective with attention to physical health status, mental health status, global outcome, and cost.

The 10th edition of International Statistical Classification of Diseases and Related Health Problems (ICD-10), which includes most of the somatoform disorders in the fourth edition of DSM (DSM-IV) (except for conversion disorders, which are included with the dissociative disorders), states that "the main feature of [the somatoform] disorders is the repeated presentation of physical symptoms together with persistent requests for medical investigations, in spite of repeated negative findings and reassurances by doctors that the symptoms have no physical basis. If any physical disorders are present, they do not explain the nature and the extent of the symptoms or the distress and preoccupation of the patient." The ICD-10 criteria for somatoform disorders are presented in Table 16-1.

Table 16-1. ICD-10 Diagnostic Criteria for Somatoform Disorders

Somatization disorder

A. There must be a history of at least 2 years' complaints of multiple and variable physical symptoms that cannot be explained by any detectable physical disorders. (Any physical disorders that are known to be present do not explain the severity, extent, variety, and persistence of the physical complaints, or the associated social disability.) If some symptoms clearly due to autonomic arousal are present, they are not a major feature of the disorder in that they are not particularly persistent or distressing.

B. Preoccupation with the symptoms causes persistent distress and leads the patient to seek repeated (three or more) consultations or sets of investigations with either primary care or specialist doctors. In the absence of medical services within either the financial or physical reach of the patient, there must be persistent self-medication or multiple consultations with local healers.

C. There is persistent refusal to accept medical reassurance that there is no adequate physical cause for the physical symptoms. (Short-term acceptance of such

reassurance, i.e., for a few weeks during or immediately after investigations, does not exclude this diagnosis.)

D. There must be a total of six or more symptoms from the following list, with symptoms occurring in at least two separate groups:

Gastrointestinal symptoms

- (1) abdominal pain;
- (2) nausea;
- (3) feeling bloated or full of gas;
- (4) bad taste in mouth, or excessively coated tongue;
- (5) complaints of vomiting or regurgitation of food;
- (6) complaints of frequent and loose bowel motions or discharge of fluids from anus;

Cardiovascular symptoms

- (7) breathlessness without exertion;
- (8) chest pains;

Genitourinary symptoms

- (9) dysuria or complaints of frequency of micturition;
- (10) unpleasant sensations in or around the genitals;
- (11) complaints of unusual or copious vaginal discharge;

Skin and pain symptoms

- (12) blotchiness or discoloration of the skin;
- (13) pain in the limbs, extremities, or joints;
- (14) unpleasant numbness or tingling sensations.

E. Most commonly used exclusion clause. Symptoms do not occur only during any of the schizophrenic or related disorders, any of the mood [affective] disorders, or panic disorder.

Undifferentiated somatoform disorder

A. Criteria A, C, and E for somatization disorder are met, except that the duration of the disorder is at least 6 months.

B. One or both of criteria B and D for somatization disorder are incompletely fulfilled.

Hypochondriacal disorder

A. Either of the following must be present:

- (1) a persistent belief, of at least 5 months' duration, of the presence of a maximum of two serious physical diseases (of which at least one must be specifically named by the patient);
- (2) a persistent preoccupation with a presumed deformity or disfigurement (body dysmorphic disorder).

B. Preoccupation with the belief and the symptoms causes persistent distress or interference with personal functioning in daily living and leads the patient to seek medical treatment or investigations (or equivalent help from local healers).

C. There is persistent refusal to accept medical reassurance that there is no physical cause for the symptoms or physical abnormality. (Short-term acceptance of such reassurance, i.e., for a few weeks during or immediately after investigations, does not exclude this diagnosis.)

D. Most commonly used exclusion clause. The symptoms do not occur only during any of the schizophrenic and related disorders or any of the mood (affective) disorders.

Somatoform autonomic dysfunction

A. There must be symptoms of autonomic arousal that are attributed by the patient to a physical disorder of one or more of the following systems or organs:

- (1) heart and cardiovascular system;
- (2) upper gastrointestinal tract (esophagus and stomach);
- (3) lower gastrointestinal tract;
- (4) respiratory system;
- (5) genitourinary system.

B. Two or more of the following autonomic symptoms must be present:

- (1) palpitations;
- (2) sweating (hot or cold);
- (3) dry mouth;
- (4) flushing or blushing;
- (5) epigastric discomfort, "butterflies," or churning in the stomach.

C. One or more of the following symptoms must be present:

- (1) chest pains or discomfort in and around the precordium;
- (2) dyspnea or hyperventilation;
- (3) excessive tiredness on mild exertion;
- (4) aerophagy, hiccough, or burning sensations in chest or epigastrium;
- (5) reported frequent bowel movements;
- (6) increased frequency of micturition or dysuria;
- (7) feeling of being bloated, distended, or heavy

D. There is no evidence of a disturbance of structure or function in the organs or systems about which the patient is concerned.

E. Most commonly used exclusion clause. These symptoms do not occur only in the presence of phobic disorders or panic disorder.

A fifth character is to be used to classify the individual disorders in this group, indicating the organ or system regarded by the patient as the origin of the symptoms:

Heart and cardiovascular system

Includes: cardiac neurosis, neurocirculatory asthenia, da Costa's syndrome.

Upper gastrointestinal tract

Includes: psychogenic aerophagy, hiccough, gastric neurosis.

Lower gastrointestinal tract

Includes: psychogenic irritable bowel syndrome, psychogenic diarrhea, gas syndrome.

Respiratory system

Includes: hyperventilation.

Genitourinary system

Includes: psychogenic increase of frequency of micturition and dysuria.

Other organ or system

Persistent somatoform pain disorder

A. There is persistent severe and distressing pain (for at least 6 months, and continuously on most days), in any part of the body, which cannot be explained adequately by evidence of a physiological process or a physical disorder and which is consistently the main focus of the patient's attention.

B. Most commonly used exclusion clause. This disorder does not occur in the presence of schizophrenia or related disorders, or only during any of the mood (affective) disorders, somatization disorder, undifferentiated somatoform disorder, or hypochondriacal disorder.

Other somatoform disorders

In these disorders the presenting complaints are not mediated through the autonomic nervous system, and are limited to specific systems or parts of the body, such as the skin. This is in contrast to the multiple and often changing complaints of the origin of

symptoms and distress found in somatization disorder and undifferentiated somatoform disorder. Tissue damage is not involved.

Any other disorder of sensation not due to physical disorder, which is closely associated in time with stressful events or problems, or which results in significantly increased attention for the patient, either personal or medical, should also be classified here.

Somatoform disorder, unspecified

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Primary Physician's Diagnostic Process

Physicians have been trained to record faithfully the patient's medical history, to perform a physical examination, and to make use of laboratory tests. Often during the primary care physician's evaluation of the patient for tiredness, poor appetite, weakness, or tense nerves, an underlying psychiatric condition is spotted by the astute clinician who senses considerable emotional turmoil. The clinician then elicits specific findings on the mental status examination. In the routine practice of medicine, as long as the patient can render a concise chronicle of events and present a straightforward account of bodily perceptions underlying pathophysiological events, the primary care physician has a remarkably good chance of making an appropriate diagnosis and instituting corrective treatment. However, it becomes understandably difficult for the physician to develop an accurate diagnosis when the somatoform patient forgets (represses) or refuses (suppresses) to share with the physician certain medically relevant, critical events or when the somatoform patient continually attributes potentially fatal diseases to benign internal stimuli.

Moreover, when the patient's expectations of a clear-cut diagnosis of physical illness are not substantiated by the results of the physical examination and laboratory findings, the clinician may have questions about the somatoform patient's competency, motivation, or even integrity. Is this account of suffering actually deliberate distortion? Is it simple forgetfulness when the patient fails to tell the physician that there was a similar episode of such symptoms 10 years ago (with benign outcome)? To the probing clinician it might appear at first that somatoform patients are guilty of blatant, intentional manipulation, or at the least of considerable distortion. Most physicians are not primed to consider that the lack of concordance between subjective suffering and objective physical findings can stem from a set of distorted perceptions and attributions or unusual beliefs.

In an emergency room or office practice setting, the application of specific diagnostic criteria for the somatoform disorders can be helpful. Even patients with somatoform disorders do get ill and eventually die of something. In the past physicians have been far too ready to attribute atypical presentations of certain protean medical diseases (e.g., multiple sclerosis, lupus, or myasthenia gravis) to psychiatric causes—coupling such reductionistic thinking with the conclusion that a putative psychiatric disorder does not merit further investigation or even referral.

Since the 1970s clinical research has allowed the inclusion criteria for some of the somatoform disorders to be considerably simplified, which has helped to make some of these diagnoses easier to use. More concise criteria have further supported the maxim that it is inappropriate to make a psychiatric diagnosis just because another medical diagnosis cannot be determined (i.e., the process of psychiatric diagnosis by

exclusion). Table 16–2 lists a few of the disorders commonly confused with somatoform disorders, especially early in their course.

Table 16-2. Conditions Commonly Confused With Somatoform Disorder

Multiple sclerosis	Acute intermittent porphyria
Central nervous system syphilis	Lupus erythematosus
Brain tumor	Hyperthyroidism
Hyperparathyroidism	Myasthenia gravis

BIOMEDICAL THEORIES

Of the few research findings on somatoform disorders that are relevant to underlying physiological or structural brain abnormalities, few have been widely disseminated. This makes the internist's task of telling patients that they have a psychiatric disorder even more complicated.

To comprehend the underlying basis of the somatoform disorders, the brain should be viewed both as a transducer of experience and as a practiced, highly trained organizer of perceptions from the milieu interieur and the milieu exterieur. The brain filters, amplifies, or dampens afferent and efferent stimuli from all parts of the body and from the brain itself. It then produces messages that explain the matrix of the patient's experiential world; that is, the brain edits and interprets its material (signals) and then sends out "sound bites" and an executive summary about bodily function in light of past incidents (including material seen on television or in the press). Somatoform disorders may involve a considerable variety of neuronal pathways—from brain-brain signals to pain pathways and perceptual pathways, in addition to efferent signals to motor apparatus and blood vessels.

CONVERSION DISORDER

Definition

A conversion disorder is a disturbance of bodily functioning that does not conform to current concepts of the anatomy and physiology of the central or the peripheral nervous system. It typically occurs in a setting of stress and produces considerable dysfunction.

Many conversion disorders simulate acute neurological pathology (e.g., strokes and disturbances of speech, hearing, or vision). However, conversion disorders are not associated with the usual pathological neurodiagnostic signs or the underlying somatic pathology. Conversion symptoms (e.g., anesthetics and paresthesias produced by a conversion disorder) do not conform to usual dermatomal distribution of the underlying peripheral nerves; rather, the signs and symptoms of a conversion disorder typically conform to the patient's concept of the medical condition.

Conversion disorders seem to change or convert the psychic energy of the turmoil of acute conflict into a personally meaningful metaphor of bodily dysfunction.

Turbulence of the mind is transformed into a somatic statement, condensing and focusing concepts, role models, and communicative meanings into one or several physical signs or symptoms of dysfunction. These somatic representations often simulate an acute medical calamity; initiate urgent, sometimes expensive medical investigation; and produce disability. In primitive settings, however, certain conversion symptoms have been taken as tokens of religious faith and even as expressions of witchcraft.

Although most conversion reactions are transient (hours to days), some can persist. Chronic conversion disorders can actually produce permanent conversion complications, such as disuse contractures of a "paralyzed" limb that remains long after the psychic strife that prompted the conversion has been resolved. In many cases

a chronic conversion disorder serves to help stabilize an otherwise dysfunctional family. In addition to sensorimotor symptoms, marked autonomic disturbances such as protracted (psychogenic) vomiting, hyperemesis gravidarum, urinary retention, and pseudocyesis are also seen, although less commonly. Conversion disorders challenge the diagnostic competence of internists, neurologists, otolaryngologists, ophthalmologists, and psychiatrists.

Like the other somatoform disorders, conversion disorders are not volitional. Rather, ego defense mechanisms of repression and dissociation act outside of the patient's awareness. Many patients with conversion disorders experience *la belle indifférence*, an emotional unconcern or even flatness in a setting of catastrophic illness; but some patients do experience considerable anguish over their new symptoms.

A conversion disorder can be considered when a patient manifests a loss or alteration in physical functioning suggesting a medical or neurological disorder and the condition cannot be explained by any other known medical disorder or pathophysiological process. A conversion disorder cannot be diagnosed just because a medical disorder cannot be ruled in. Failure to prove a physical illness is a necessary but not sufficient condition for making the diagnosis of conversion disorder.

HISTORY

Until the middle of the nineteenth century, somatization disorder and conversion disorder (which often travel together) were considered to be one condition called hysteria. The term hysteria was derived from the Greek word *hystera*, meaning uterus. Descriptions of conversion disorders appeared as far back as 1900 BC when multiple symptoms were attributed by Egyptian physicians to a wandering of the uterus within the body.

In the middle of the century, Pierre Briquet originated the modern concept of conversion disorder. He considered the disorder to result from a dysfunction of the central nervous system (CNS). He proposed that conversion symptoms occurred in those with a constitutional predisposition when a receptive part of the brain was impacted by extreme stress. Later, Russel Reynolds described clinical cases in which the loss of function or the persistence of severe pain could be attributed to an idea that the patient had about the body.

Jean-Martin Charcot then expanded on the biological concepts of Briquet and the psychological constructs of Reynolds, adding heredity to factors that influence predisposition. Moreover, Charcot suggested that a traumatic event gave rise to the idea, which then led to the brain's dynamic dysfunction; Charcot also suggested that the idea could be produced in the brain by hypnosis.

The term conversion was first used by Sigmund Freud and his associate Josef Breuer. It was used to describe the clinical case of Anna O., whose undischarged psychic energy was bound in a somatic symptom. This symptom represented the unconscious conflict. That is, a repressed thought was converted to a somatic symptom. Freud then worked out his concept of talking therapy as a catharsis through which unconsciously repressed material might become conscious. With catharsis in psychotherapy and with hypnotic suggestion, somatic conversion symptoms were shown to diminish and even disappear.

In 1929, following from Charcot, Pierre Janet observed that conversion disorders were preceded by a lowering of conscious threshold and were associated with dissociation. He recognized that a constitutional weakness in an individual might be accentuated by shock or fatigue, resulting in aspects of consciousness being split off. His concept of what is now considered to be conversion disorder did not, however,

include the concept of repression; thus, he was not concerned with the significance of the dynamic unconscious as Freud was.

COMPARATIVE NOSOLOGY

In 1952 the American Psychiatric Association's first edition of DSM (DSM-I) used the diagnostic term "conversion reaction," stressing (1) the reactive part of the disorder, (2) issues of symbolism—symbols or ciphers from the individual's unconscious that relate to significant life experiences, and (3) secondary gain—tangible benefits that accrue to the individual upon assumption of the sick role. In 1967 the second edition of DSM (DSM-II) changed the diagnostic term to "hysterical psychoneurosis, conversion type." Important in this formulation was *la belle indifférence*. Follow-up studies of specific phenomena associated with conversion disorder were constructed by assigning relative weights to certain features associated with conversion studies. These studies indicated, however, that there is no pathognomonic validity (using outcome as a gold standard) to symbolism of the symptom, secondary gain, hysterical personality, and *la belle indifférence*.

In 1980 DSM-III revised the diagnostic term again to conversion disorder. Removed from the cluster of symptoms was pain disorder, a symptom-based pain disorder with criteria otherwise comparable to that of conversion disorder. DSM-III and the revised third edition of DSM (DSM-III-R) required conversion disorder to be judged by the clinician to be etiologically related to the conversion symptom because of a temporal relationship between it and a significant psychosocial stressor or else a demonstrated coupling of conflict and psychological need and the initiation or the exacerbation of a preexisting symptom. That is, it was one of the few diagnoses where the judgment of the clinician was explicitly sought in the making of a diagnosis based on psychodynamic mechanisms. A subjective rather than an objective component was written into the diagnostic criterion; this raised the issue of inter-rater reliability in the diagnostic process.

One of the major changes from DSM-III and DSM-III-R to DSM-IV has been the further removal of the etiological inference of unconscious mechanisms and psychodynamics involved in the productions of symptoms, with the statement that "psychological factors are judged to be associated with the symptom or deficit because it was preceded by conflicts or other stressors." That is, since diagnosticians are not mind readers, the role of unconscious motivation must be inferred.

A second difference in DSM-IV from DSM-III-R is in the exclusion category associated with the concept of "not fully explained by a known physical disorder." This was broadened to include culturally sanctioned behavior or experience, general medical condition, and the direct use of a substance.

A third change came in the distress-disability category, in which the concept has now been broadened to include important areas of functioning to the individual other than just social and occupational and the phrase "warrants further medical attention" has been added (e.g., distress to family or physician because of potential medical implications associated with the symptom or deficit).

A fourth change focused on the wording used to eliminate factitious disorder and malingering. The words "not conscious of intentionally producing the symptom" have been removed in favor of the simple concept "not intentionally produced or feigned."

In 1978 the ninth revision of International Statistical Classification of Diseases, Injuries, and Causes of Death (ICD-9) used the term "hysteria" to include conversion disorder and dissociative phenomena. Both conditions were defined as mental disorders in which a mechanism beyond the patient's awareness produced either a

restriction of the field of awareness or a disturbance of motor or sensory function. Hysteria was associated with psychological advantage or symbolic value. Conversion symptoms involved the body's function, whereas dissociative symptoms involved the mind's function.

When ICD-10 considered the somatoform disorders, it listed somatization disorder and its subthreshold companion diagnosis, undifferentiated somatoform disorder, plus hypochondriacal disorder, persistent somatoform pain disorder, autonomic (psychogenic aerophagia) and nonautonomic (psychogenic pruritus) somatoform disorders as part of the group of the somatoform disorders. However, ICD-10 assigned conversion disorder to the dissociative disorders. Presumably this ICD-10 change was made because conversion disorders, like dissociative disorders, typically have a sudden onset and a short duration whereas the other somatoform disorders have a gradual onset and a much more chronic course. Thus, the term conversion hysteria, coupled loosely and interchangeably over centuries now disappears whereas dissociative (conversion) disorders appear in ICD-10 with separate codings for dissociative disorders of movement and sensation, dissociative motor disorders, dissociative convulsions, and dissociative anesthesia and sensory loss.

EPIDEMIOLOGY

Conversion disorders are the most frequently occurring of the somatoform disorders. Affected persons can range in age from early childhood into old age. The annual incidence of conversion disorders seen by psychiatrists in a New York county has been estimated to be 22 cases per 100,000 population. In a general hospital setting 5 to 16 percent of all psychiatric consultation patients manifest some conversion symptoms. In a study of a rural Veterans Administration general hospital, 25 to 30 percent of all male patients had a conversion symptom at some time during their admission. By contrast, in a psychiatric emergency room or psychiatric clinic, the incidence of conversion disorder is far lower (1 percent of all psychiatric admissions), as different selection factors supervene. Lifetime figures for ever having any conversion symptom, even if only on a transient basis, are far higher, with some studies reporting a 33 percent prevalence rate. Conversion disorder occurs mainly in women, with a ratio of 2 to 1 up to 5 to 1 in some studies. However, there does not seem to be an overrepresentation of conversion disorders in female children.

The prevalence of the disorder is highest in rural areas and among the undereducated and the lower socioeconomic classes. It is more prevalent in military populations, especially in those exposed to combat. It is also more common in underprivileged persons, in those of subnormal intelligence, and in industrial settings where compensation neurosis may become an issue. There may be a tendency for familial aggregation and for the patient to be the youngest sibling in the family. The incidence of the disorder may be on the decline.

Clinicians frequently involved with these patients in general hospital settings contend that conversion disorders are more likely to be seen in individuals who are psychologically naive, who are not particularly introspective, and who are disinclined to believe that psychic factors can affect physical processes than in individuals who are psychologically sophisticated.

Comorbidity

Medical and especially neurological disorders occur frequently among patients with conversion disorders. Indeed in some series the majority of conversion patients have a well-documented neurological condition. What is typically seen in these comorbid neurological or medical conditions is an elaboration of symptoms stemming from the original organic lesion. Whether this tells the clinician something about the nature of

the sick role or about the nature of the brain with compromised functioning and an altered state of consciousness remains an open question.

Preexisting or emerging psychopathology also seems to predispose an individual to the development of a conversion. Among the Axis I psychiatric conditions, depressive disorders, anxiety disorders, and somatization disorders are especially noted for their association with conversion disorder. Conversion in schizophrenia is reported but is very uncommon. Studies of patients admitted to a psychiatric hospital for conversion disorder reveal that, on further study, one quarter to one half have a clinically significant mood disorder or schizophrenia.

Axis II personality disorders also frequently accompany a conversion disorder, especially the histrionic type (in 5 to 21 percent of cases); the passive-dependent type (9 to 40 percent of cases), and the passive-aggressive type of personality disorder. However, conversion disorders can occur in persons with no predisposing medical, neurological, or psychiatric disorder.

ETIOLOGY

Biological Factors

Conversion disorders represent pathology in the mind as well as dysfunction within the brain. Recent etiological research on conversion disorder has involved event-related potentials, structural and functional brain imaging, and neuropsychological testing to investigate aspects of corticofugal inhibition of afferent stimuli.

Imaging

Pierre Flor-Henry's important work on the etiopathology of conversion disorder has used bipolar derivations of the electroencephalograph (EEG) with measures of coherence to reflect synchrony, not between individual electrode sites but between regions of the brain. His findings emphasize (1) hypofunction of the dominant hemisphere systems, (2) a consequent dysfunctional overactivity of the nondominant hemisphere, and (3) abnormal interhemispheric relations. Defects in processing endogenous somatic signals and integrating sensorimotor signals appear to be the consequence of altered dominant hemispheric systems. Particularly in women there seems to be a secondary disorganization of the contralateral hemisphere that in turn is capable of producing the characteristic somatic symptoms seen in a conversion reaction. An EEG study of a woman with a left-sided paralysis as a conversion disorder has proved most illuminating. The attempt to move the paralyzed leg failed to activate the right primary motor cortex. Instead, the right orbito-frontal and right anterior cingulate cortex were significantly activated, suggesting these two areas inhibit the prefrontal (willed) effects on the right primary motor cortex when the patient tries to move her left leg.

NEUROPSYCHOLOGICAL TESTS

Impaired vigilance-attention and short-term memory have been demonstrated. Localizing studies using the Halstead-Reitan Battery and other neuropsychological tests have manifested dysfunction of both nondominant right and especially dominant left hemispheres. Increased field dependency and heightened suggestibility is also present.

Unilateral Symptoms and Localization

Taken together, clinical and research findings could suggest that patients with conversion disorder under extraordinary circumstances experience impaired intercortical communication and blockade of ordinary channels of verbal associations. The preponderance of left-sided, unilateral symptoms seen in conversion disorder plus the strong association of conversion disorders with depressive disorders could be used as evidence of a nondominant right-hemispheric vulnerability. Additional

complementary evidence for localization comes from the fact that the left hemisphere is phylogenetically associated with inhibitory influences. Thus, the motor and sensory symptoms of conversion suggest defects in processing and in analysis of sensorimotor signals, which leads to a failure in the integration of endogenous somatic signals. The proposed defect in understanding the signals in a conversion disorder is in some ways analogous to the failure of comprehension in a stroke (i.e., with receptive and expressive aphasia when acoustic-motor coordination of auditory signals involving language fails to occur).

Sex and Brain Localization

The fact that conversion disorders occur mainly in women is another piece of evidence used to support a theory of brain localization. Studies from a variety of sources indicate that women have greater instability of right-hemispheric organization. Thus, it has been proposed that a primary defect in the left hemisphere interferes with the normal transcallosal inhibitory stabilizing functions of the unstable contralateral right hemisphere. These circumstances could account for the symptoms of conversion disorder and for its almost exclusive restriction to the female pattern of cerebral organization.

Much more clinical and experimental work remains to be done if these heuristic hypotheses are to have widespread clinical relevance. The phenomena of conversion disorder and hypnosis have both been considered to result from blockade of corticofugal impulses induced by emotional rapport or intense emotional experience. Both conditions can lead to selective diminution of awareness of a bodily function. Interestingly, hypnosis can bring about temporary remission of conversion symptoms and can also produce a mimicry of conversion symptoms in those not afflicted with the condition.

These biomedical theories account for the how, but not the what or the why of conversion disorder. Obviously a multifactorial explanation is needed to render an understanding of the patient's plight and to serve as a framework for testing the most effective and efficient methods of treatment. Conversion symptoms represent a common pathway for the expression of a complex biopsychosocial event. A patient with conversion disorder, having a specific diathesis, experiences and creates (outside of his or her level of awareness) an illness in a setting of stress that is shaped to some extent on his or her model of disease.

PSYCHOSOCIAL FACTORS

Long before neuropsychological, neurophysiological, and imaging evidence was available to contribute to an understanding of symptoms of conversion, astute nineteenth-century clinicians focused their attention on the psychological aspects of their patients' internal and external worlds. Sigmund Freud and Josef Breuer carefully studied the ongoing emotional struggles and the relevant life circumstances immediately antecedent to conversion symptoms in patients with neuroses. In such a fashion the context of the acute psychic trauma and other aspects of the patient's life story often could be coupled meaningfully to the development and maintenance of the patient's conversion symptoms. Moreover, psychoanalytic and hypnotic techniques sometimes produced dramatic and sometimes permanent remission of the patient's symptoms.

Psychodynamic Factors

According to psychoanalytic theory a conversion reaction results when the anxiety of unconscious intrapsychic conflict is converted into somatic symptoms. When aggressive or sexual impulses emerge in a field of strong inhibition of their expression, the resultant intrapsychic conflict overwhelms the person's ordinary ego

defense mechanisms. In such a setting unconscious mechanisms facilitate a compromise as conversion symptoms emerge. The settlement allows a partial expression of the primitive impulse but disguises it so that the individual is unaware of the unconscious wish and the unacceptable desire. However, the symptom formation may impose a considerable price. Suffering and disability then serve as atonement for having had the unacceptable wish or impulse.

The decrease in anxiety and psychological distress after formation of the conversion symptom is the primary gain. Benefits that also accrue to the individual after the sick role is assumed are the secondary gain. Both primary and secondary gain are typically part of the syndrome associated with conversion disorder. Its permanence and severity can be reinforced by patients being enmeshed in irresolvable conflict for which they feel no responsibility.

In psychodynamic terms conversion symptoms represent a solution to an unconscious conflict between instinctual drives and superego prohibitions to their expression. The conversion symptom emerges when a latent conflict is activated by an event unconsciously perceived as related to the conflict. The specific meaning of the precipitating event resonates with the personal vulnerability of the patient and the patient's life experiences. Thus the formation of the conversion symptom embodies a symbolic aspect of the intrapsychic conflict. The conversion symptoms may derive from identification with a significant individual, often someone whom the patient associates with loss and who has also experienced such a symptom (a conversion model). Sometimes the conversion model is the patient's own somatic ailment, with symptoms now called forth in an effort to resolve some intrapersonal or interpersonal crisis.

Sociocultural Theory

Viewed alone or as complementary to psychoanalytic theory, conversion symptoms can be understood in sociocultural terms as a form of communication concerning an emotionally charged feeling or idea blocked from expression by personal or cultural restraints. Conversion symptoms can express the forbidden, using mimicry or pantomime instead of words. Moreover, the symptoms of a conversion disorder allow the individual to enter into the sick role, avoiding certain responsibilities or noxious situations. As such, the patient can control or otherwise manipulate the behavior of others. However, it is most likely that clinicians who view conversion disorder only as a nonspecific organic response to stress and merely as a manifestation of illness behavior have: (1) had difficulty obtaining clinical data from or about the patient; (2) have failed to engage the patient from a psychodynamic perspective; or (3) have not used an amobarbital (Amytal) or other drug-assisted interview.

Learning Theory

In terms of conditioned learning theory a conversion symptom can be seen as a piece of classically conditioned learned behavior; symptoms of illness, learned in childhood, are called forth as a means of coping with an otherwise impossible situation.

DIAGNOSIS AND CLINICAL FEATURES

A frequent but not invariable common denominator of conversion disorder is the pseudoneurological nature of the symptom. Common types of conversion symptoms are listed in Table 16-3.

Table 16-3. Common Symptoms of Conversion Disorder

Motor Symptoms

Involuntary movements

Tics

Blepharospasm
Torticollis
Opisthotonos
Seizures
Abnormal gait
Falling
Astasia-abasia
Paralysis
Weakness
Aphonia
Sensory Deficits
Anesthesia, especially of extremities
Midline anesthesia
Blindness
Tunnel vision
Deafness
Visceral Symptoms
Psychogenic vomiting
Pseudocyesis
Globus hystericus
Swooning or syncope
Urinary retention
Diarrhea

Motor symptoms—Abnormal gait, weakness, and paralysis may occur. There can be involuntary movements, rhythmical tremors, episodic jerks, tics, seizures, and falling. Yet when patients with conversion symptoms fall, they rarely are severely hurt. Blepharospasm, torticollis, and opisthotonos may also occur. All such symptoms tend to grow more intense when observed. Many neurological symptoms of patients with a known pathophysiological basis, such as Parkinson's disease, also tend to intensify when the individual experiences increased anxiety. The DSM-IV criteria for conversion disorder are listed in Table 16–4.

Table 16-4. DSM-IV Diagnostic Criteria for Conversion Disorder

- A. One or more symptoms or deficits affecting voluntary motor or sensory function that suggest a neurological or other general medical condition.
- B. Psychological factors are judged to be associated with the symptom or deficit because the initiation or exacerbation of the symptom or deficit is preceded by conflicts or other stressors.
- C. The symptom or deficit is not intentionally produced or feigned (as in factitious disorder or malingering).
- D. The symptoms or deficit cannot, after appropriate investigation, be fully explained by a general medical condition, or by the direct effects of a substance, or as a culturally sanctioned behavior or experience.
- E. The symptom or deficit causes clinically significant distress or impairment in social, occupational, or other important areas of functioning or warrants medical evaluation.
- F. The symptom or deficit is not limited to pain or sexual dysfunction, does not occur exclusively during the course of somatization disorder, and is not better accounted for by another mental disorder.

Specify type of symptom or deficit:

With motor symptom or deficit
With sensory symptom or deficit
With seizures or convulsions
With mixed presentation

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Astasia-Abasia

A staggering, ataxic gait with gross jerks and thrashing or wild waving of the upper extremities, often with an inability to stand without support, is called astasia-abasia. Surprisingly, such patients sometimes can dance to music after the clinician suggests that the ability to successfully perform a dance such as the fox trot or the Texas two-step is not impacted by the patient's inability to stand or walk.

Pseudoseizures

Pseudoseizures are paroxysmal episodes of altered behavior resembling epileptic attacks but devoid of the characteristic clinical epileptic and electrographic features. Convulsive behaviors identified as a conversion disorder often take place when the clinician walks into the patient's room or when the family visits. Various terms such as psychogenic or hysterical seizures in the past, these clinical episodes terminate without the patient having a period of sluggishness, sleepiness, or confusion (as might be seen following a true convulsion). Pseudoseizures, but not complex partial seizures, tend not to manifest extreme stereotypy in the overt motor sequence and lack a neurological indicator. These patients do not evidence an elevated serum prolactin level immediately following the clinical episode, and the patient's interictal cortical EEG is normal or ambiguous. The EEG of the pseudoseizure patient during the clinical episode does not show any of the correlates of an epileptic seizure, such as increasing frequency of spike discharges, sudden onset of focal or diffuse rhythmic activity, or postictal slow waves.

Patients with pseudoseizures tend to have a history of psychiatric treatment, suicide attempts, and borderline personality disorder. However, the Minnesota Multiphasic Personality Inventory-2 (MMPI-2) in one study demonstrated an overall accuracy rate of 67 percent (correctly identifying 71 percent of the group with pseudoseizures and 65 percent of the patients with epileptic seizures) using the video-monitored EEG as the gold standard. Families of pseudoseizure patients had considerably more health problems, distress, and criticism than families of patients with epilepsy. There have now been a number of reports linking an increased incidence of childhood sexual abuse with the initiation, precipitation, or exacerbation of symptoms in patients with pseudoseizures. A diagnosis of pseudoseizures is also more likely if there is a financial incentive. Pseudoseizures can also be seen as a form of chronic maladaptive behavior utilizing the benefits of the sick role.

The induction of a seizure by suggestion, formerly considered a hallmark of hysterical seizures, is also seen in complex partial seizures when there is elevated electrical lability. Semi-purposeful movements, thrashing and pelvic thrusting, often considered to be the hallmarks of pseudoseizures, can be seen with direct stimulation of the cingulate region and are common correlates of complete partial seizures with frontotemporal foci. Moreover, more than 70 percent of patients with pseudoseizures without documented bona fide seizures show interictal EEG abnormalities, neuropsychological impairment, or abnormalities on magnetic resonance imaging (MRI) and computed tomography (CT) scans.

Although tongue-biting, urinary incontinence, injury during falls, and seeming loss of consciousness do not usually occur with a pseudoseizure, all of these can occur. However, the preservation of corneal, pupillary, and gag reflexes, plus the absence of extensor plantar responses and the preservation of normal color during the attack all suggest a pseudoseizure. The eyes of a patient in a pseudoseizure reportedly deviate toward the ground when the patient is placed on his or her side.

The proportion of pseudoseizures in a given study typically reflects the nature of the referral source and their relationship with the evaluation. From a clinician's perspective about one third of patients evaluated for a pseudoseizure do not have a convulsive disorder or pseudoseizures but rather have some other neurological condition, another third of patients have true convulsions as well as pseudoseizures, and another third have just pseudoseizures. From a treatment perspective it is imperative to know the correct diagnosis so as to avoid polypharmacy and toxicity, hazardous diagnostic interventions, and neglect of underlying psychological distress. Although patients with pseudoseizures typically are psychologically naive, their very lack of sophistication may make them more respectful of the power of the physician's authority. They may be more suggestible and easier to manipulate, which may be effectively utilized in the service of their treatment.

Other Common Motor Symptoms

Other symptoms include paralysis or paresis, more frequently on the patient's nondominant side (i.e., on the left side if the patient is right-handed). Paralysis can occur in one, several, or all limbs. In contrast to a patient with neuropathy, the reflexes in a patient with conversion disorder with anesthesia or paralysis remain normal. There is no fasciculation, no other electromyographic abnormality, and no atrophy. However, in cases of longstanding pseudoparalysis, disuse atrophy and even contractures can occur as a complication of conversion.

Sensory Symptoms

Anesthesias, hypesthesias, and paresthesias are common conversion symptoms, especially in the extremities (Table 16–3). The pattern of distribution of the anesthesia does not conform to the underlying central or peripheral nerve distribution. Typical are glove or stocking and strict midline anesthesias. Despite a claimed loss of total sensation in the legs and feet, for example, conversion patients can walk in the dark without stumbling (unlike those with *tabes dorsalis* who lack position sense).

Conversion patients told to answer "yes" if they can feel anything when pricked with a pin in the anesthetic zone often will respond "no" when pricked, even when they are not looking at the area being tested.

Hysterical Blindness

Other sensory modalities can also be affected. Patients with hysterical blindness typically do not hurt themselves seriously when they bump into stationary objects. Despite their "lack" of vision, their pupils react to light and their visual evoked potentials on the EEG are consistent with those of normal vision. Some of the other findings in monocular and binocular blindness associated with conversion are shown in Table 16–3.

Hallucinations

Patients with conversion disorder can have positive or negative hallucinations. Patients who have positive hallucinations perceive an image or hear a sound that is not there. Hallucinations in conversion disorder are usually associated with intact insight, and the hallucinations are often visual, auditory, and tactile. They tend to be described by the person as part of an interesting story. Conversely, with a negative hallucination the patient with an intact nervous system does not see an object that

others can see or does not hear a sound that others can hear. When generalized to more than a specific object that is not seen, the individual seems to be blind even though the visual apparatus is still working.

Visceral Symptoms

Psychogenic vomiting can occur as a conversion disorder (Table 16–3). Typically such a patient will not suffer significant weight loss when observed for a week on an inpatient medical service; gastrointestinal workup will show no significant disease or disorder. Another visceral conversion disorder is urinary retention. On urological workup, conversion patients show normal intracystometric dynamics. Conversion as pseudocyesis manifests as a cessation of menses, a protuberant abdomen, and an elevation of serum hormones seen in early pregnancy. Other visceral conversions include globus hystericus, syncope, and diarrhea.

Important Caveats

As with all disorders with a low specificity and no confirmatory laboratory test, substantiation of the diagnosis is facilitated by the passage of time with no other countervailing diagnosis evolving. Also helping to confirm the diagnosis of conversion disorder is the development of another bout of conversion symptoms. About 25 percent of patients with conversion disorder will develop another episode during the following 1 to 6 years.

The diagnosis of a conversion disorder is made more secure if details of a prior set of conversion symptoms can be elicited. However, complicating the collecting of the past medical history in many patients with somatoform disorders is their frequent use of repression. Such patients thus may not recall important pieces of historical data. In adding collateral information a review of the patient's old clinical chart can be very helpful.

Another important caveat is that the diagnosis cannot be based solely on inexplicable neurological findings accompanied by relevant psychological factors. Patients with lesion-based neurological disorders also wrestle with the sick role. Moreover, although suggestibility is often seen in patients with conversion, some patients with organic disorders can also respond to suggestion, briefly altering their symptoms. It is not uncommon to find neurological disease coexisting with a conversion disorder. Critical review of the literature finds little or no empirical support for the necessity of a number of previously widely accepted classic accompaniments of conversion disorder including: *la belle indifférence*, hysterical personality, the presence of secondary gain, the symptom as symbolism, sibling position, disturbed sexuality, and conversion V pattern on the MMPI-2 (elevated hysteria and hypochondriasis scales, even higher than the depression scale).

Suggestibility, with a patient seizing on command (or when induced in a particular situation), used to be considered a sign that a convulsion was a pseudoseizure. However, recent clinical and EEG evidence indicates that the precipitation of overt, nonstereotyped seizures by instruction may not be a reliable diagnostic method when an insidious process is slowly elevating temporal and limbic lobe lability. When taken by itself, no single associated finding is pathognomonic for the diagnosis of conversion disorder. However, a number of features taken together help the clinician to determine the likelihood (i.e., possible, probable, definite) of a conversion disorder.

Setting

Marked psychological stress is almost always present. Precipitants typically may have been acute rage, truncated grief, sexual abuse, or physical abuse. Somatic symptoms may develop abruptly following a dramatic psychological blow, mechanical trauma, or a life-threatening experience. However, studies on patients with conversion

disorder demonstrate that their life experiences are not more extreme than those with other types of psychiatric disorders.

The initial mental status examination of the patient with conversion disorder may prove to be quite unremarkable: a calm person who may or may not be troubled by a new somatic symptom may have no insight into the symptom's underlying dynamics. At first glance the patient's family may appear to be happy and integrated, with the family predicament (if present) being covert. Family difficulties are common in patients with conversion disorders, but not more so than for families of patients who attend a psychiatric clinic for other psychiatric disorders.

Mrs. A. was a 22-year-old right-handed fundamentalist farmer's wife, homemaker, and mother of three from a sparsely settled Western state. Her past medical history was benign except for a motor vehicle accident 2 years previously that produced a sharp blow to the right temporal area, resulting in several hours' loss of consciousness. She had an unremarkable behavioral history without substance abuse, prolonged depressions, or unexplained somatic symptoms. Her demeanor had always been placid and unassuming. There was no family history of antisocial behavior or substance abuse.

On Thanksgiving Day, while taking her usual solitary afternoon walk along the creek behind the kitchen, she came upon the floating, lifeless bodies of two of her children. She shrieked, swooned, and fell to the ground. Relatives in the house rushed out to assist but were unable to revive the children. When she was helped up, she asked that her husband guide her back to her room. Later that afternoon she seemed calm, even detached, as others scurried about making arrangements. She admitted to a visitor that she seemed to have lost the gift of sight.

That evening the family physician was called to examine the newly sightless woman. He noted that her pupils were round, equal, and constricted briskly with a bright light; she was unable to touch the tips of her index fingers together in front of her; she failed to look at her own hands when instructed to do so; and she had no other neurological abnormalities, asymmetries, or complaints. The physician explained to the gathered family and patient that the woman was suffering from nervous shock, needed kind and quiet support, and should refrain from routine household chores for the moment. The physician also suggested that her eyesight would gradually return over the next week or so, perhaps following the funerals of her children. The patient's vision did slowly return over the next days and she gradually resumed her usual level of care for the home, her surviving child, and other members of the family.

PSYCHOPATHOLOGY AND LABORATORY EXAMINATION

Diagnostic workup is a multistep process that begins with a very thorough history and physical examination. There are a number of simple but specialized examinations that can be worked into a seemingly routine physical examination. Although some diagnoses can be made from the foot of the bed, chair, or stretcher with substantiating data collected in just a few minutes, it is not infrequent for an initial workup of a patient with a conversion disorder to take many hours even when conducted by a skilled clinician. Collateral sources need to be included to build a case based on circumstantial evidence of what was going on in the patient's life at the time of the acute shock. Table 16-5 lists examples of important tests that are relevant to conversion disorder symptoms.

Scroll right to see more columns.

Table 16-5. Distinctive Physical Examination Findings in Conversion Disorder

Condition	Test	Conversion Findings
Anesthesia	Map dermatomes	Sensory loss does not conform to

		recognized pattern of distribution	
Hemianesthesia	Check midline	Strict half-body split	
Astasia-abasia	Walking, dancing	With suggestion, those who cannot walk may still be able to dance; alteration of sensory and motor findings with suggestion	
Paralysis, paresis not on it	Drop paralyzed hand onto face	Hand falls next to face,	
	Hoover test	Pressure noted in examiner's hand under paralyzed leg when attempting straight leg raising	
	Check motor strength	Give-away weakness	
Coma	Examiner attempts to open eyes	Resists opening; gaze preference is away from doctor	
	Ocular cephalic maneuver	Eyes stare straight ahead, do not move from side to side	
Aphonia	Request a cough	Essentially normal coughing sound indicates cords are closing	
Intractable sneezing	Observe	Short nasal grunts with little or no sneezing on inspiratory phase; little or no aerosolization of secretions; minimal facial expression; eyes open; stops when asleep; abates when alone	
Syncope	Head-up tilt test	Magnitude of changes in vital signs and venous pooling do not explain continuing symptoms	
Tunnel vision	Visual fields	Changing pattern on multiple examinations	
Profound monocular blindness		Swinging flash light sign (Marcus Gunn)	
	Absence of relative afferent pupillary defect		
	Binocular visual fields	Sufficient vision in "bad eye" precludes plotting normal physiological blind spot in good eye	
Severe bilateral blindness	"Wiggle your fingers, I'm just testing coordination"	movements before realizing the slip	Patient
	Sudden flash of bright light	Patient flinches	
	"Look at your hand"	Patient does not look there	
	"Touch your index fingers"	Even blind patients can do this by proprioception	

There are also a few specialized laboratory-based procedures that can assist in the diagnostic workup of some patients with conversion disorder. Helpful in specific instances are simultaneous EEG and videotaping of behavior along with lack of elevation of serum prolactin levels in pseudoseizures; optokinetic drum test in conversion blindness; cortical evoked potentials for auditory and visual deficits; and electromyogram for fasciculation in lower motor neuron paralysis.

DIFFERENTIAL DIAGNOSIS

Almost any neurological symptom can have a conversion basis. Many serious neurological diseases can be mistaken for the conversion disorders (Table 16–6), and a number of psychiatric conditions need to be considered in the differential diagnosis

of conversion disorder (Table 16–7). Until the diagnosis is clarified, ongoing evaluation by a psychiatrist and a neurologist may be useful; premature closure obviously aborts further diagnostic consideration.

Table 16-6. Neurological Conditions in the Differential Diagnosis of Conversion Disorder

Myasthenia gravis
Periodic paralysis
Brain tumor
Multiple sclerosis
Optic neuritis
Partial vocal cord paralysis
Guillain-Barre Guillain-Barré syndrome
On-off syndrome of Parkinson's disease
Degenerative diseases of basal ganglia and peripheral nerves
Acquired myopathies including polymyositis
Subdural hematoma
Acquired and hereditary dystonias
Drug-induced dystonia
Creutzfeldt-Jacob disease
Early manifestations of acquired immune deficiency syndrome (AIDS)

Table 16-7. Psychiatric Conditions in the Differential Diagnosis of Conversion Disorder

Major depressive episodes
Catatonic schizophrenia
Pain disorder
Somatization disorder
Histrionic personality disorder
Adjustment disorder
Posttraumatic stress disorder
Malingering

COURSE AND PROGNOSIS

Course

Most patients diagnosed with conversion disorder experience a quick symptomatic recovery. Rapid improvement is especially seen in cases where symptoms are of recent onset, suggestive of voluntary control, inconsistent or variable, and of obvious immediate benefit.

Follow-up studies in the literature from the 1960s demonstrated multiple diagnostic errors in series of conversion patients followed for 2 to 20 years because clinicians, using subjective criteria, proved to be too liberal in their use of the diagnosis. Some long-term follow-up studies have shown that 25 percent of conversion disorder patients on psychiatric wards who subsequently died had inaccurate initial psychiatric diagnoses of conversion disorder. Long-term follow-up studies using selection criteria heavily weighted to psychodynamic issues (symbolism, secondary gain, la belle indifférence) from DSM-I, DSM-II, ICD-9, and even DSM-III have shown an appalling lack of diagnostic consistency. A frequently quoted British inpatient study reported that over half of the patients had an organic disease 7 to 11 years later that accounted for the symptoms of the supposed conversion disorder. Of note, however, two thirds of the patients in that series were not diagnosed as having

conversion disorder by a psychiatrist before they were accepted into the series. Many cases from that inpatient study who did not develop neurological disease instead developed or were discovered to have some other type of disabling psychiatric disorder.

An American study found that neurological disease explained the original conversion symptoms in only one fifth of the cases. The emergence of neurological disease was more frequent in conversion patients who did not have somatization disorder as a comorbid condition. Another study with a 20-year follow-up noted that fully one third of patients later experienced a psychotic illness, often paranoid schizophrenia. In that sample central nervous system disease, especially epilepsy, was often mistaken for hysteria. A third major study with a 10-year follow-up noted that a quarter of patients developed organic disorders over time that accounted for the presenting conversion symptoms: mainly degenerative diseases of the spinal cord, peripheral nerves, bones, muscle, and connective tissue.

Thus, follow-up studies from tertiary medical centers show that there is a distinct possibility of the emergence of other medical, neurological, or disabling psychiatric disorders to account for the original conversion symptoms. Thus, one can conclude from the older literature that either the inclusion criteria used for the initial diagnosis of conversion disorder were far too inclusive, or else that the diagnosis of conversion disorder must be made more cautiously.

Conversion disorder has been imprecisely and improperly diagnosed when clinicians used it as a diagnosis of exclusion in the face of the neurological examination not being consistent with full-blown neurological disease. Clinicians need to be wary that they do not erroneously think the patient has a psychiatric illness such as conversion disorder because of inexplicable neurological signs and the coincidence of emotional conflict.

Prognosis

A favorable prognosis of conversion disorder is associated with sudden onset; readily identifiable stressful events; good premorbid health with no comorbid psychiatric, medical, or neurological disease; and no ongoing compensation litigation.

In a retrospective American study of patients with conversion disorder without comorbid neurological or medical disorders, 90 of 100 patients recovered by the time of psychiatric hospital discharge; on follow-up 5 years later, 75 percent remained well. Another general hospital study demonstrated that half of patients on a medical surgical unit found to have a conversion disorder during a psychiatric consult experienced remission of their conversion symptoms by the time of medical discharge. Another study of conversion patients successfully treated with a 1-year follow-up, only one fifth had relapsed; symptom substitution was minimal.

Patients with chronic conversion disorders, however, do not have a good prognosis. In a recent 10-year British follow-up study of 56 patients hospitalized and well studied for their pseudoneurological symptoms, 30 had no relief from their original symptoms. Additionally, 11 patients had neurological findings, tentatively considered at symptom onset, which in retrospect accounted for their conversion symptoms.

TREATMENT

Most conversion symptoms remit spontaneously or after behavioral treatment, suggestion, and a supportive environment. Thus, for symptoms of very recent onset a variety of other therapies have also been utilized successfully. In practice clinicians tend to choose therapies that reflect their training. Irrespective of the technique used, most approaches seem to work when symptoms are not reinforced, when an

authoritarian approach is used, and when the patient's psychosocial plight is the focus of attention.

Common Denominators of Success

What seems least likely to be effective is trying to get the newly afflicted patient to accept the therapist's opinion that the somatic symptom is a direct manifestation of a psychosocial problem (e.g., that the physical disability is the representation of a psychiatric problem). Rather, the common denominator of successful treatment (irrespective of the clinician's theoretical framework) is the building of a caring, authoritative relationship. It is important to provide the patient with a safe environment to facilitate the gradual decrease in symptoms. The clinician can then deal indirectly with interpretations of the conversion symptom while trying to minimize or eliminate the symptom. It is not helpful to argue with the patient about the cause of the conversion disorder.

A multiplicity of types of therapies have their adherents (Table 16–8). When successful, some common elements in many of these treatments are the following: (1) a nonconfrontational approach, (2) discouraging retention of symptoms, and (3) manipulating the environment.

Table 16-8. Therapies Used in Conversion Disorder

Faradic stimulation	Hypnosis and other suggestive techniques
Physical therapy	Behavioral approach to symptom elimination
Electrosleep	Family therapy
Inexact interpretations	Long-term insight-oriented psychotherapy

Psychodynamic techniques and insight-oriented intensive psychotherapy may need considerable modification with psychologically naive patients. At times placing patients in a double bind by telling them that full recovery constitutes proof of an organic cause and failure to recover constitutes proof of nonorganic or psychiatric etiology, facilitates rapid recovery. For example, when the patient is failing to progress, three possible explanations can be made for the lack of progress: (1) the patient has psychological problems that will necessitate long-term self-examination in treatment; (2) the patient is not trying, which constitutes grounds for dismissal; or (3) the symptoms are caused by excessive overstimulation and fatigue, which would necessitate periods of deep rest without stimulation of any kind. Patients usually choose this latter option as the most likely explanation. Therapy would then begin with behavioral goals augmented daily and deep rest if the patient fails to meet those goals. Deep rest precludes reading, watching television, and socializing with staff members or other patients until the next therapy session. This precaution should be presented as an expression of concern about the patient's level of fatigue and as a means of preventing overstimulation. This behavioral paradigm must be seen not as punitive but as very supportive. In this way, patients rapidly lose symptoms and gain discharge; however, each behavioral intervention needs to be tailored to the specifics of the case.

If combined with a supportive and problem-solving approach, hypnosis may be helpful. On some occasions parenteral injections of amobarbital or lorazepam (Ativan) have also been seen as helpful. For example, 50-mg doses of amobarbital from an intravenous infusion in 5 percent dextrose could be administered until the patient feels sleepy. An interview might require a total of 200 to 700 mg of amobarbital. A nurse should always be present as witness and a crash cart must be available lest there be respiratory failure or cardiac arrest. Videotaping the interview can be a powerful tool to allow later feedback to the patient under the supervision of

the therapist. This gives patients the opportunity to see how their unconscious works; however, it is the physician's responsibility to help the patient integrate this information.

Oral anxiolytic medications also may reduce anxiety and allow the patient to engage in a psychotherapeutic process that might otherwise be too overwhelming to handle. Decreasing the need for secondary gain by opening up other channels of communication seems to help. Eventually the more emotionally healthy patients seem to be able to gain insight into the meaning of their symptoms. Using a variety of techniques to work at resolving the patient's problems as well as eliminating the conversion symptom seems to be the best approach. Symptom substitution may occur if the patient still needs the conversion symptom, but most authors believe that substitution is usually not seen if one works patiently and with tact, allowing patients to save face and to accomplish some of their covert goals.

Poor Outcomes

Not all symptoms remit in hours or days; some linger tenaciously despite skilled inpatient treatment. In such instances other psychopathology and immutably malignant social pathology are often present. The presence of a conversion disorder in one member of a family, the putative patient, may well serve as a beneficial cohesive factor in an otherwise chaotic dysfunctional family. Symptom removal and clinical remission seems to be least successful if compensation is at issue.

SOMATIZATION DISORDER

Definition

The essential feature of somatization disorder is recurrent, multiple somatic complaints requiring medical attention but not associated with any physical disorder. Somatization disorder is the expression of personal and social distress in a deeply ingrained idiom of bodily complaints linked with medical help-seeking behavior. The diagnosis requires a history of many physical complaints of several years' duration and a lifetime history, beginning before age 30, which result in medical treatment or alteration in life-style. The symptoms must not be fully explained by a known nonpsychiatric medical condition, or the resulting complaints or impairment must be excessive. Typically these symptoms, coupled to persistent complaints plus demands placed on caregivers and medical services, result in gain to the patient through relief from responsibilities, through caring responses and attention from others, or both.

The diagnostic syndrome of somatization disorder has been evolving since the 1950s with various refinements and simplifications in criteria of a disorder that has the same stable, chronic course. Like a personality disorder, somatization disorder begins at an early age, is chronic and unremitting and appears to be a deeply ingrained adaptation to life. Currently, eight symptoms are required, which must meet a specific pattern: four different sites of pain; two different gastrointestinal symptoms; one sexual or reproductive system symptom other than pain; and one neurological symptom. The subjective severity of the symptoms must be sufficient to lead the patient to consult a physician, take medicine, or make life-style changes.

History

The history of somatization disorder is complex. Essentially, over the centuries two complementary syndromes have been described: one monosymptomatic and the other polysymptomatic. The monosymptomatic syndrome is currently recognized as conversion disorder whereas the polysymptomatic syndrome has become known as somatization disorder. Historically, the two disorders have often been interrelated and commingled.

Hysteria Somatization disorder has had many names and many antecedents; one such predecessor has been referred to in older texts as hysteria, first recognized by the ancient Egyptians who believed that hysteria was caused by upward dislocation of the uterus and displacement of other organs. Migration of the uterus throughout the body thus provided the basis for the multiple symptoms. Doubts about the uterine origin of hysteria began in the seventeenth century. Thomas Sydenham dissociated hysteria from the uterus and linked it with a psychological disturbance known at that time as "antecedent sorrows," therein recognizing the emotional origin of the disorder. Further, Sydenham was also the first to recognize the disorder in men.

In 1859 Briquet emphasized the multisymptomatic aspects of the disease and its protracted course. His report of the 430 cases observed at the Hospital de la Charite Charité in Paris focused on polysymptomatic aspects of the disorder. Briquet also recognized hysteria in men and attributed the disorder to emotional causes.

Modern Era An important series of papers published between 1951 and 1953 presented the first modern conceptualization of the multisymptomatic concept of hysteria. The Washington University group in St. Louis concluded that hysteria is a definable syndrome with a characteristic clinical picture that begins before the age of 35. Using objectifiable criteria, they defined a prevalence of the multisymptomatic disorder in the general hospital of 2.2 percent of all admissions. While noting the similarities of their work to Briquet's, they initially deviated by suggesting that men did not have the disorder.

In the early 1960s two studies confirmed the original findings of a definable clinical syndrome, demonstrating diagnostic stability of the multisymptomatic concept of hysteria. In 1970 the eponym Briquet's syndrome was proposed to denote multisymptomatic hysteria. The disorder, characterized by at least 25 symptoms from 10 symptom groups, was known as Briquet's syndrome until the publication of the DSM-III. Ironically, after the decision was made to incorporate Briquet's syndrome as part of the new diagnostic nomenclature, an unrelated decision was made to drop all eponyms. Hence a new name—somatization disorder—had to be created.

DSM-III streamlined the criteria to 14 lifetime symptoms in women (12 in men) from a list of 37 symptoms; moreover, a requirement for symptom grouping by organ systems was dropped. With the advent of DSM-III-R and further time for detailed follow-up studies, the number of symptoms required for men and women were both changed to 13.

Comparative Nosology

There have been multiple predecessors to the current diagnosis of somatization disorder, the best validated of which has been Briquet's syndrome. Later studies have demonstrated only a moderate degree of diagnostic concordance between Briquet's syndrome and somatization disorder. In spite of these limitations, in most situations it appears reasonable to apply the findings from the literature on Briquet's syndrome and somatization disorder. Somatization disorder did not appear in DSM-I or DSM-II; it first appears in DSM-III and later in DSM-III-R. For DSM-IV the diagnostic criteria were simplified to require one or more symptoms from each of four symptom groups. The ICD-10 criteria for somatization disorder require (1) at least 2 years of multiple and variable physical symptoms with no adequate physical explanation; (2) persistent refusal to accept advice; and (3) some degree of impairment of functioning. There must be a total of 6 or more from a grouping of 14 symptoms in 2 of the 4 different areas (gastrointestinal, cardiovascular, genitourinary, plus skin and pain). There are no pseudoneurological symptoms in ICD-10 and DSM-IV has different gynecological and sexual symptoms from those found in ICD-10. Moreover, ICD-10 requires at least

6 symptoms from 4 groupings whereas DSM-IV requires 8 symptoms from its 4 groupings. Finally, ICD-10 is specific about duration of the disorder—2 years—whereas DSM-IV requires that some of the physical complaints have their beginnings before age 30.

Epidemiology

Several studies based on large populations have estimated the lifetime prevalence of somatization disorder as 0.13 percent of the general population. Other community-based surveys estimate the lifetime prevalence to be 0.2 to 2 percent in women and less in men. Because patients with somatization disorder believe themselves to be medically ill, they can be assumed to frequent physicians' offices. Recent work indicates that as many as 5 percent of patients seen in family practice settings meet criteria for the disorder whereas 9 percent in a general hospital population and 12 percent in a group of patients with chronic pain met criteria for somatization disorder. In a group of patients with irritable bowel syndrome in a private outpatient clinic, 17 percent of patients met the criteria. In one random selection of 49 women who underwent hysterectomy for noncancerous reasons, 27 percent were diagnosed with somatization disorder.

Persons that meet the full criteria for somatization disorder are typically unmarried, nonwhite, poorly educated, and from rural areas. In families with somatization disorder, children have 11.7 times as many emergency room visits as families less afflicted with somatizing. Moreover, children's somatization is predicted by parental somatization, substance abuse, and antisocial symptoms.

Although there may not be a large number of these patients in the general population that meet full-blown criteria, individually they are expensive to care for. Recent studies estimate that somatization disorder patients incur 6 times the national average of per capital expenditures for hospitals and 14 times the national average of per capital expenditures for physician services. Moreover, there may be thirty times as many patients with subthreshold somatization, that is, those that meet partial criteria for somatization disorder (i.e., undifferentiated somatoform disorder).

Sex Early reports found somatization disorder exclusively in women. However, it is now recognized that somatization disorder afflicts men, but less commonly than women. Men comprise 5 to 20 percent of those with somatization disorder.

Comorbidity As the severity, chronicity, and invalidism of somatization increase, so does the likelihood of a major psychiatric disorder. Frequent concomitants of somatization disorder are major depressive disorder, anxiety disorders, and personality disorders. Over half of somatization disorder patients from a primary care sample were found to have a lifetime history of a major depressive episode in addition to their somatization disorder. Several recent studies using structured diagnostic interviews in patients from primary care settings found that 61 to 72 percent of patients with somatization disorder also have co-occurring personality disorders. This rate would seem to be 2.5 to 11.6 times more common in somatization disorder patients than in general medical patients.

Somatization disorder does not seem to be associated with any one or any combination of currently defined personality disorders. In one study, the most frequent types in order were avoidant, paranoid, self-defeating, obsessive-compulsive, schizotypal, and histrionic personality disorders. Earlier studies from patients attending psychiatric clinics had only reported an association with histrionic personality disorder and antisocial personality disorder. Many patients with somatization disorder also have conversion symptoms.

Anxiety disorders, especially phobias, panic disorder, and generalized anxiety disorder, are also present in patients with somatization disorder. Suicide threats are common in patients with somatization disorder, as are suicide gestures; however, suicide attempts rarely are lethal or near-lethal. Doctor shopping is frequent, with patients moving from one doctor to another. Typically, somatization patients have very chaotic social lives with frequent divorces, separations, and remarriages; similarly, they have trouble maintaining jobs and often become too disabled to hold gainful employment.

Etiology

Numerous theories have been advanced to explain the psychosocial mechanisms involved in the process of somatization. By contrast few theories have been yet proposed to account for the biological basis of somatization disorder. The act of somatization can be understood as social and emotional communication. It can also be explained as the result of an intrapsychic dynamic, with somatization carried to an extreme.

Social Communication Somatization as social communication includes the use of bodily symptoms to manipulate or control relationships (e.g., an adolescent girl's developing unexplained abdominal pain to prevent her parents from going away for the weekend). Somatizing also can serve as emotional communication. Patients may be unable to verbally express their emotions; therefore, they may use somatic symptoms and somatic complaints to express their emotional state. Symptoms may be used to symbolically communicate emotions, as they are in conversion disorder. Some patients also use medical complaints as a coping device to deal with stress. Finally, physical symptoms may be used as a solution to an intrapsychic conflict, again as in conversion symptoms. Studies of psychological tests in somatization disorder have reported that compared to sex- and age-matched controls these patients have significantly more scale elevations on the MMPI-2.

Psychodynamic Factors Classical psychoanalytic theory has held that hysteria represents a substitution of somatic symptoms for repressed instinctual impulses. Freud postulated that the conflict was a phallic Oedipal one. However, more recent articles in the analytic literature emphasize a pregenital conflict as well.

Biological Factors Interesting preliminary data are now finally available concerning the biomedical underpinnings of somatization disorder. Neuropsychological testing demonstrates equal bifrontal impairment of the cerebral hemispheres and nondominant hemispheric dysfunction in patients with somatization disorder. However, some reports indicate that symptoms referable to the left side of the body may indicate that the right hemisphere of the brain is more involved than the left side. Preliminary evidence indicates that patients with somatization disorder may have an abnormality in cortical functioning, as evidenced by abnormal auditory-evoked potentials. In contrast to controls, patients with somatization disorder responded similarly to relevant and irrelevant stimuli, suggesting an impairment in selective attention. The data require much more extensive follow-through, without the confound of comorbid personality disorders influencing findings.

Christopher Bass at Oxford University is leading an effort to define the pathophysiology of symptoms experienced by patients with somatization disorder because not all patients with unexplained medical complaints have emotional disorders. The awareness, reporting, and seeking of medical help is complex and involves biopsychosocial processes. From a perceptual perspective, awareness of symptoms varies with the attention paid to them. Symptom reporting is elevated among individuals who live alone or work in unstimulating settings. The awareness of

symptoms and of disease is increased by reading about or observing medical disorders. Depressed and anxious moods are associated with greater reporting of bodily symptoms. Personality traits, neuroticism, and negative affectivity with introspective characteristics all tend to lower the threshold for noticing and reporting bodily sensations. Muscular tension, hyperventilation, sleep, and inactivity are all conditions that could contribute to the symptoms experienced by patients who have somatization disorder.

Considerable evidence now also points to familial and genetic associations in somatization disorder. Some data support the findings that groups of patients with somatization disorder have a higher-than-expected prevalence of antisocial personality disorder or manifest several traits of antisocial personality disorder. Other data, however, do not support such a strong association. One theory holds that antisocial personality disorder and somatization disorder may have a common genetic background. Some scholars consider somatization disorder to be the female expression of a genetic tendency, with antisocial personality disorder being its male counterpart.

Recent data is now emerging from a number of studies on families of patients with somatization disorder. In comparison to control families, those of patients with somatization disorder have had more physical and sexual abuse, and more disabling medical illnesses in one or both parents—all severe socio-environmental challenges. Thus somatizing behavior in many patients with somatization disorder seems often to emerge against a backdrop of emotionally disturbed or impoverished family life lacking in ordinary parental care in which physical symptoms had established meanings. Indeed Christopher Bass has argued that somatization disorder should be classified as a developmental personality disorder because of its patients' characteristic lifestyles and modes of relating to self and others. Relevant to this argument is evidence of persistent impairment associated with an illness-shaping background in an individual's formative years.

Diagnosis and Clinical Features

Table 16–9 presents the DSM-IV diagnostic criteria of somatization disorder. It is important to note that specific symptoms do not need to be considered legitimate by the clinician. Rather, patient's reports that they have the symptom is sufficient as long as the symptom meets the severity criteria. A patient can have somatization disorder even if a current or presenting symptom did not begin before the age of 30 years. A careful review for an early onset of any of the unexplained symptoms for which the patient has had problems is necessary to make the diagnosis and at least one of these symptoms must begin before age 30.

Table 16-9. DSM-IV Diagnostic Criteria for Somatization Disorder

- A. A history of many physical complaints beginning before age 30 years that occur over a period of several years and result in treatment being sought or significant impairment in social, occupational, or other important areas of functioning.
- B. Each of the following criteria must have been met, with individual symptoms occurring at any time during the course of the disturbance:
 - (1) four pain symptoms: a history of pain related to at least four different sites or functions (e.g., head, abdomen, back, joints, extremities, chest, rectum, during menstruation, during sexual intercourse, or during urination)
 - (2) two gastrointestinal symptoms: a history of at least two gastrointestinal symptoms other than pain (e.g., nausea, bloating, vomiting other than during pregnancy, diarrhea, or intolerance of several different foods)

(3) one sexual symptom: a history of at least one sexual or reproductive symptom other than pain (e.g., sexual indifference, erectile or ejaculatory dysfunction, irregular menses, excessive menstrual bleeding, vomiting throughout pregnancy)

(4) one pseudoneurological symptom: a history of at least one symptom or deficit suggesting a neurological condition not limited to pain (conversion symptoms such as impaired coordination or balance, paralysis or localized weakness, difficulty swallowing or lump in throat, aphonia, urinary retention, hallucinations, loss of touch or pain sensation, double vision, blindness, deafness, seizures; dissociative symptoms such as amnesia; or loss of consciousness other than fainting)

C. Either (1) or (2):

(1) after appropriate investigation, each of the symptoms in criterion B cannot be fully explained by a known general medical condition or the direct effects of a substance (e.g., the effects of injury, medication, drugs, or alcohol)

(2) when there is a related general medical condition, the physical complaints or resulting social or occupational impairment are in excess of what would be expected from the history, physical examination, or laboratory findings

D. The symptoms are not intentionally feigned or produced (as in factitious disorder or malingering).

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Patients with somatization disorder consider themselves to be severely ill. They report their health is worse than those with chronic, lesion-based medical conditions. In contrast, the mortality rate of somatization patients is similar to that of the general population and is substantially less than patients with major depressive disorder.

Ms. D. is a 52-year-old white woman who was referred to a general internist in the city for evaluation of persistent back pain and multiple other complaints. At hospitalization it was noted that the patient was disabled from her job as a machine operator at a shoe factory. Ms. D. gave a history of 10 operations: removal of a tumor from her right wrist, a dilation and curettage, a hysterectomy, three abdominal gastric operations, three breast biopsies, and leg surgery. She had received care from five different hospitals and seven different physicians in the past 2 years.

On physical examination, Ms. D. was an obese, chronically ill-appearing woman who came to the hospital wearing her transcutaneous electrical nerve stimulation unit. She was cooperative and showed her various scars with a certain amount of enthusiasm.

The remainder of her physical examination was within normal limits except for a decreased range of motion in the area of her lumbar spine and local muscle guarding, with some tenderness. Spinal radiographs revealed some degeneration of vertebral bodies L2 to L5. On mental status examination she was cooperative and pleasant, and her behavior was somewhat seductive. There was no pressure or eccentricities in her speech. She showed little hesitation in discussing intimate details of her life. Her mood was euthymic; her affect was appropriate to mood but possibly a little shallow. The remainder of her mental status examination was within normal limits.

Disallowing all back-related symptoms, Ms. D. was positive for eight pain symptoms: four gastrointestinal symptoms, two sexual symptoms, and two pseudoneurological symptoms with an age of onset of 26 years. During the previous 12 months, Ms. D. reported that she had been in bed 21 days, had made seven office visits to four physicians, and had been hospitalized for a total of 52 days.

Ms. D.'s case illustrates that the diagnosis of somatization disorder can and should be made in the presence of comorbid medical conditions. Patients with somatization disorder do become ill, and their problems need to be appropriately diagnosed and treated. However, the management of somatization disorder should continue unchanged.

Pathology and Laboratory Examination

There are no known neuropathological or routine laboratory findings specific for somatization disorder.

Differential Diagnosis

An important aspect of the differential diagnosis is distinguishing a somatic symptom secondary to another psychiatric disorder from a symptom of somatization disorder.

Table 16–10 explains how somatic complaints in other disorders differ from those found in somatization disorder.

Table 16-10. Nonsomatization Somatic Symptoms

Anxiety or depressive disorders	Usually one or two somatic symptoms of acute onset and short duration
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Panic disorder	Somatic symptoms experienced only during panic episode
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Hypochondriasis	Patient's focus is on fear of disease, not focus on a symptom
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Conversion disorder	Only one or two complaints
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Pain disorder	One or two unexplained pain complaints, not a lifetime history of multiple complaints
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Hypochondriasis can sometimes be difficult to distinguish from somatization disorder. Hypochondriasis is more likely to begin at a later age than somatization disorder whereas the latter is more likely to occur in women than in men. Hypochondriacal patients often misinterpret normal bodily sensations as an indication of a pathological process. They believe that they harbor a specific lethal disease whereas somatization patients focus on complaints about multisystem symptoms that lead them to have an inordinately high number of surgical procedures and consume an excessive amount of health care.

Course and Prognosis

By definition, somatization disorder is a chronic relapsing condition with no known cure. It usually begins in middle to late adolescence, but may start as late as the third decade of life.

Typically, patients develop a new symptom, or symptoms, during times of emotional distress. No research data are yet available as to how long a modal episode of illness lasts. Clinical wisdom indicates that a typical episode lasts 6 to 9 months, with quiescent periods lasting from 9 months to a year. It is unlikely that patients with somatization disorder can go for more than a year without developing a new symptom and seeking some type of health care. Periods of psychosocial distress seem to coincide either with the onset of new symptoms or with increased health—care—seeking behavior associated with some preexisting symptom. Although no research data exist on whether stress precipitates the relapse, anecdotally there does seem to be an association. This association is especially problematic for patients because somatization disorder considerably disrupts social aspects of living.

Poor Long-Term Health Somatization disorder patients typically consider their health to be poor. When standard measures for health status are applied, the patients report that all aspects of their health—physical, social, and mental—as well as their general health perceptions are severely impaired. Patients with somatization disorder report

worse health than do those with chronic medical conditions. Further, because patients who have somatization disorder perceive themselves to be sicker than the sick, it is not at all incongruous that they usually deem themselves disabled from work.

Treatment

Because the cause of somatization disorder is unknown and no curative or ameliorative treatment has been found, the clinician needs to focus on management rather than treatment, on coping rather than curing (care rather than cure).

General Management Strategies While taking the medical history, physicians should take note of and store away for future use psychosocial material that it brought up spontaneously by the patient: references to personal difficulties and life events that cause distress. These matters can be explored at a later time after the relationship with the patient is more developed. It is important initially to avoid confrontations or to alienate the patient in other ways. The physical examination is an opportunity for discussing symptoms and providing clear information about clinical findings.

Information to the patient must be clear and unambiguous. During regularly scheduled repeat visits, as the patient endlessly drones on about physical symptoms, the clinician should gradually change the agenda to inquire about psychosocial issues in an empathic manner. In actual practice, this type of inquiry does not consume inappropriate amounts of time, contrary to clinicians' fears. Limits also need to be placed on the numbers and types of investigations that the patient requests. Crises in the patient's life, provoked by psychosocial stresses, are often accompanied by an increase in somatic symptoms. At such times the physician may be more able to draw the patient's attention to the relationship between emotional and physical suffering.

Best practice would indicate that the patient's primary care physician follows the patient closely and treats, or refers for treatment, any complicating comorbid psychiatric condition, such as a mood disorder. When ordering laboratory studies, the physician should order for the patient the same set of tests as if the patient were not a somatizer. If called into the case after all medical evaluations have been completed, the psychiatrist is advised to inquire about symptoms and the patient's medical odyssey, avoiding the temptation to express any opinions prematurely and maintaining a position of empathic curiosity during the initial intake in an effort to understand the patient's experiences.

Several types of treatment techniques have been advocated, including behavioral (avoid prolonged bed rest, which only increases weakness and encourages the patient to focus on symptoms); cognitive (avoid catastrophizing); and interpersonal (elucidate and deal with family conflicts). Progress should be aimed at improving functional capacity and the patient's progress will typically be very gradual.

Management of somatization disorder has only been tested empirically in several studies, findings from which revealed that when certain specific management strategies were undertaken by the primary care physician, patients with somatization disorder improved their physical functioning; their health care utilization decreased at the same time too.

Cost estimates of the savings of a simple consultation letter to these patients' primary care physicians about specific principles was associated with a 12 percent decrease in per-patient annual health care cost. Net annual cost savings were \$295. Improvement to the patient is best reflected in the ability to perform the activities of daily living.

Patients whose physicians received a simple consultation letter from a psychiatrist were significantly clinically improved in this domain compared to patients whose primary care physician did not receive such a consultation letter. Table 16-11 presents helpful management strategies for somatization disorder.

Table 16-11. Helpful Management Strategies for Somatization Disorder

- Establish primary care physician as patient's main and (if possible) only physician
- Set up regularly scheduled visits every four to six weeks
- Keep outpatient visits brief
- Perform at least a partial physical examination during each visit directed at the organ system of complaint
- Understand symptoms as emotional communication rather than the harbinger of new disease
- Look for signs of disease rather than being symptom focused
- Avoid diagnostic tests, laboratory evaluations, and operative procedures unless clearly indicated
- Set a goal of getting at least selected somatization disorder patients referral-ready for mental health care

There is little data on the treatment of depression in patients with somatization disorder, but what evidence there is suggests that depression can be successfully treated; when it is treated successfully, the somatization itself may also improve. Doctor-Patient Relationship The cornerstone for successful management of the patient with somatization disorder is establishing a trusting relationship between the patient and one community physician. The doctor-hopping that frequently occurs in these patients is both frustrating and countertherapeutic.

Recently a form of group treatment was shown to be effective in somatization patients. The intervention tested was a time-limited, behaviorally oriented group with a structured protocol. The overall goals of the group were to be a source of peer support, to share methods of coping, to increase the ability to perceive an expressed emotion, and to allow the patient to enjoy the group experience. The study demonstrated that in the year following treatment the experimental group of patients with somatization disorder demonstrated better physical and emotional health and evidenced decreased health care charges than untreated controls with the same disorder. Moreover, the more group sessions the subjects attended, the greater their sense of well-being and the lower their net health care costs. Other studies using short-term cognitive-behavioral therapy, individually or in groups, for somatizing or somatization disorder patients have demonstrated decreased somatic preoccupation, anxiety, depression, and medication usage.

UNDIFFERENTIATED SOMATOFORM DISORDER

Definition

Undifferentiated somatoform disorder is characterized by one or more unexplained physical complaints of at least 6 months' duration. These symptoms impair the patient in some domain and are temporally associated with a stressor. Psychological factors are assumed to be associated with the symptoms or complaints because of a contemporaneous relationship between the initiation or exacerbation of the symptoms and stressors, conflicts, or needs. The complaint must be unattributable to any other known psychiatric condition or pathophysiological mechanism or, when it is related to a nonpsychiatric condition, the physical complaints or resulting social and occupational impairments must be grossly in excess of what would ordinarily be expected from the findings.

History and Comparative Nosology

Many who work in the general medical setting find the diagnosis of undifferentiated somatoform disorder helpful. Research indicates the validity or distinguishing undifferentiated somatoform disorder from somatization disorder. There appears to be

a dimensional or quantitative difference between undifferentiated somatoform disorder and somatization disorder rather than a qualitative difference between the two. However, the natural history of both disorders seems to be similar. The disorder was not included in DSM-I, DSM-II, and DSM-III; it was first introduced in DSM-III-R and remains unchanged in DSM-IV. Undifferentiated somatoform disorder was introduced to the psychiatric lexicon in DSM-III-R because somatization disorder was considered to be too restrictive by primary care providers to provide adequate coverage for many patients with significant somatoform complaints. Thus the subsyndromal grouping was formalized to facilitate learning about the natural course of a large cluster of patients. The criteria for undifferentiated somatoform disorder in ICD-10 are similar to the diagnostic criteria in DSM-IV.

Somatizing Syndrome This disorder, characterized by a lifetime history of four unexplained somatic complaints for men and six for women, has been called various names. It forms a subset of patients with undifferentiated somatoform disorder. All research data so far have used the term somatization syndrome rather than the broader term undifferentiated somatoform disorder. The remainder of this section refers to data from research on somatizing syndrome.

Epidemiology

The importance of undifferentiated somatoform disorder comes from the fact that it may be 30 to 100 times more prevalent than full-blown somatization disorder. Undifferentiated somatoform disorder has an estimated lifetime prevalence in the general population of between 4 and 11 percent with an estimated 1 percent 6-month prevalence. Javier Escobar has described a group of patients with this disorder who have six unexplained symptoms for women and four for men; these patients have many of the same socioeconomic and clinical manifestations of somatization disorder. Undifferentiated somatoform disorder typically affects women. It has also been shown in some reports to be associated with lower socioeconomic status, older age, and Hispanic or African-American origin. One study found a slight association with antisocial personality disorder; however, other studies have not confirmed this. One recent study reported these patients had higher waking salivary cortisol concentrations and higher heart rates under the stress of a mental task and less habituation than controls. Presumably, those with undifferentiated somatoform disorder are comorbid with at least one other lifetime psychiatric disorder, but this has not yet been firmly established.

Approximately 50 percent of patients with the disorder have comorbid psychiatric conditions (e.g., depression, anxiety, personality disorders) compared with 7 percent in the general population.

Etiology

There are multiple theories about the process of somatization; none of which is specific to undifferentiated somatoform disorder.

Diagnosis and Clinical Features

The DSM-IV diagnostic criteria are listed in Table 16–12. Patients with undifferentiated somatoform disorder have more comorbid psychiatric diseases than do general medical patients without the disorder. These comorbid conditions are primarily depressive disorders, anxiety disorders, and personality disorders.

Table 16-12. DSM-IV Diagnostic Criteria for Undifferentiated Somatoform Disorder

- A. One or more physical complaints (e.g., fatigue, loss of appetite, gastrointestinal or urinary complaints)
- B. Either (1) or (2);

- (1) after appropriate investigation, the symptoms cannot be fully explained by a known general medical condition or by the direct effects of a substance (e.g., the effects of injury, medication, drugs, or alcohol)
 - (2) when there is a related general medical condition, the physical complaints or resulting social or occupational impairment is in excess of what would be expected from the history, physical examination, or laboratory findings
- C. The symptoms cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.
- D. The duration of the disturbance is at least 6 months
- E. The disturbance is not better accounted for by another mental disorder (e.g., another somatoform disorder, sexual dysfunction, mood disorder, anxiety disorder, sleep disorder, or psychotic disorder).
- F. The symptom is not intentionally produced or feigned (as in factitious disorder or malingering).

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Many patients with this disorder have illnesses that wax and wane but continue to play a persistent role in their lives. In a study of 46 patients with chest pain, those without coronary artery disease had a higher rate of anxiety disorders than those with coronary artery disease. Eleven years later, those with chest pain but without coronary artery disease were found to have led lives of disability and chronic distress. They had had repeated interruptions in their lives associated with functional symptoms. Those in the study with chest pain without coronary disease also had higher research scale ratings for depression and anxiety and reported more physical symptoms.

Pathology and Laboratory Examination

There are no specific pathological or routine laboratory features for undifferentiated somatoform disorder.

Differential Diagnosis

In the differential diagnosis of undifferentiated somatoform disorder care must be taken to differentiate somatic symptoms that could be a part of many other psychiatric disorders. For example, somatic symptoms frequently occur in major depressive disorder. Similarly, the somatic delusion of schizophrenia and major depressive disorder also needs to be differentiated from undifferentiated somatoform disorder. Several other psychiatric disorders need to be differentiated from undifferentiated somatoform disorder. Somatization disorder is a more severe disorder characterized by a chronic history of multiple unexplained somatic complaints that fit a specific pattern and begin before the age of 30. Adjustment disorder with physical symptoms may also evidence unexplained somatic complaints; however, the duration of this disorder is by definition less than 6 months. Finally, the diagnosis of psychological factors affecting medical condition may at first appear similar; however, the disorder affects a known Axis III disorder rather than mimicking an Axis I disorder.

Course and Prognosis

The course of undifferentiated somatoform disorder is generally chronic and relapsing; however, little systematic research on the disorder has been accomplished to date. It is likely that some cases of the disorder can resolve after a single episode. There is substantial disability, work impairment, and excessive health care utilization in undifferentiated somatoform disorder. The dysfunction is not seen to the same extent as in somatization disorder. Extensive comorbidity has been shown for

somatization disorder with depressive, anxiety, and personality disorders. With the less restrictive criteria for undifferentiated somatoform disorder, there is also considerable comorbidity, although the rates are not quite as high.

Treatment

Recent studies have indicated that patients with undifferentiated somatoform disorder respond to the same treatment or management approach as patients with somatization disorder.

One recent study indicates that somatizing syndrome patients of physicians who received a simple psychiatric consultation letter explaining management principles of intervention were able to lower the annual medical care charges of these patients by \$289 (in constant 1990 dollars), which equals a 32.9 percent reduction in the annual median cost of their medical care. In addition, these patients experienced clinically significantly improved physical functioning after the intervention.

HYPOCHONDRIASIS

Definition

Hypochondriasis is a disorder characterized by preoccupation with the fear of developing a serious disease or the belief that one has a serious disease. The fear is based on the patient's interpretation of physical signs or sensations as evidence of disease even though the physician's physical examination does not support the diagnosis of any physical disorder. The unwarranted fear of or belief in a diseased state persists in spite of medical reassurance. However, the belief does not have the certainty of delusional intensity.

History

The concept of hypochondriasis has been a part of medical lore since ancient times. Prior to the early nineteenth century the area of the body below the rib cage, the abdomen, was called the hypochondrium. Thus, hypochondriasis referred to somatic complaints occurring in the abdomen. In the late 1920s, R.D. Gillespie provided the first modern description of the disorder.

Comparative Nosology

Hypochondriacal symptoms can be a part of another disorder such as major depressive disorder, dysthymic disorders, generalized anxiety disorder, or adjustment disorder. However, primary hypochondriasis or hypochondriacal disorder is a chronic and somewhat disabling disorder with hypochondriacal symptoms, not merely a part of another psychiatric condition.

Hypochondriasis was included as a diagnostic entity in DSM-I. The diagnostic criteria continued to be revised in DSM-II, DSM-III, and DSM-III-R; however, the changes have been primarily linguistic, not substantive. The only change between DSM-III-R and DSM-IV is the addition of a specifier to note that the patient has poor insight during the current episode. The ICD-10 criteria for hypochondriasis are essentially the same as those of DSM-IV.

Epidemiology

Hypochondriasis is rather common in primary care settings. In various settings, the prevalence has varied from 3 to 14 percent. Recent work indicates that in a 6-month period of observation, 4 to 6 percent of the general medical population meet the specific criteria for this disorder. The prevalence in either sex is comparable to that within the general medical population. There are no specific tendencies for overrepresentation based on social position, education, marital status, or other sociodemographic descriptors. There is a wide range of ages at onset. Although the disorder can begin at any age, onset is thought to be most common between 20 and 30 years of age. A preliminary family study of 19 cases and their 72 first-degree relatives

demonstrated no increase in the rate of hypochondriasis among their relatives compared with a control group.

Comorbidity with other psychiatric disorders is common with hypochondriasis, and must be treated accordingly. As an example, when case-matched controls and 42 hypochondriasis patients from a medical clinic were evaluated psychiatrically, the hypochondriasis patients had twice as many lifetime Axis I disorders and three times the number of personality disorders. Of the hypochondriasis patients in the study, 88 percent had one or more additional Axis I disorders, the overlap being greatest with depressive and anxiety disorders. Depression only accounts for a minor part of the total picture in hypochondriasis, so it is a mistake to think that all hypochondriasis is the result of some other Axis I disorder.

Etiology

There are four major etiological theories concerning hypochondriasis: (1) amplification of normal bodily sensations; (2) psychodynamic formulations; (3) social learning concepts; and (4) syndromic variant of some other psychiatric condition, such as a mood or an anxiety disorder.

Amplification The amplification hypothesis posits that hypochondriasis results from the augmentation of normal bodily sensations. Of the four hypotheses it has the most research interest: hypochondriacal patients might amplify their normal somatic sensations and then attribute them to some sort of pathological condition. For instance, a change in a patient's perception of peristalsis might be interpreted as abnormal, hence representing disease. However, studies of hypochondriacal patients' awareness of their own normal heartbeat now shows that they are no more accurate than are a comparison group of nonhypochondriacal patients. Moreover, in a test of tactile sensitivity, patients with hypochondriasis did not have a greater ability to discriminate on two-point touch. Yet hypochondriacal patients consider themselves to be more sensitive to benign bodily sensations and certainly report more functional somatic symptoms. When hypochondriacal patients develop clinical arrhythmias, they are not more sensitive to or more accurately aware of subtle changes in cardiac activity than nonhypochondriacal patients. A hypochondriacal patient's behavior would seem to indicate a response bias toward reporting somatic and physiological distress with an attributional tendency to invoke somatic pathology.

Psychodynamic Factors A variety of hypotheses purport that intrapsychological factors are responsible for hypochondriasis. These factors run the gamut from Freud's early theories about disturbed object relations and intense preoccupation with the self to the concept of hypochondriasis as ego defense mechanisms against guilt.

Other theorists advocate that in hypochondriasis aggressive and hostile wishes toward others are transferred into physical complaints via repression or displacement. The frustration or anger that these patients often express can be theorized to be caused by past losses, rejections, or disappointments. Sometimes these patients express anger by first soliciting and then rejecting the help and concern of others. Alternatively, hypochondriasis may be viewed as a defense against guilt, a result of low self-esteem, or a sign of excessive self-concern. Pain and somatic suffering then symbolically become a means of atonement or can be experienced as deserved punishment for past real or imagined wrongdoing.

Learning Theory Learning theory postulates that psychosocial learning has a strong etiological component in hypochondriacal disorder. The concept contends that a patient learns the sick role and that role is sufficiently reinforced through either social contact or some need gratification. Interestingly, when compared to case-matched controls, patients with hypochondriasis believe that to be in good health means to be

relatively symptom free, and they consider the presence of symptoms as indicative of sickness. Hence, personal theories of attribution, when coupled to a catastrophizing cognitive style, may be relevant to the focus on illness. The sick role then becomes a means of receiving caretaking from others.

Variants The variant theory holds that hypochondriasis is a modification of some other psychiatric disorder, such as depressive disorders, anxiety disorders, and certain personality disorders, such as obsessive-compulsive personality disorder. Although some scholars have maintained that hypochondriasis is a variant of a depressive condition, current research has not supported this hypothesis. Some theorists believe that hypochondriasis is a variant of an anxiety disorder, such as obsessive-compulsive disorder or a panic disorder, but the research data on this is not yet compelling.

Diagnosis and Clinical Features

Table 16-13 lists DSM-IV criteria for the diagnosis of hypochondriasis. The developmental background of hypochondriacal patients is of interest in that significantly more of these patients than matched controls report traumatic sexual contacts, physical violence, and major parental upheaval before the age of 17. Significantly more hypochondriacal patients also report being sick as children and missing school time for health reasons; thus, they recall more childhood trauma and illnesses even though they are not currently more medically sick.

Table 16-13. DSM-IV Diagnostic Criteria for Hypochondriasis

- A. Preoccupation with fears of having, or the idea that one has, a serious disease based on the person's misinterpretation of bodily symptoms.
- B. The preoccupation persists despite appropriate medical evaluation and reassurance.
- C. The belief in criterion A is not of delusional intensity (as in delusional disorder, somatic type) and is not restricted to a circumscribed concern about appearance (as in body dysmorphic disorder).
- D. The preoccupation causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.
- E. The duration of the disturbance is at least 6 months.
- F. The preoccupation is not better accounted for by generalized anxiety disorder, obsessive-compulsive disorder, panic disorder, a major depressive episode, separation anxiety, or another somatoform disorder.

Specify if:

With poor insight: if, for most of the time during the current episode, the person does not recognize that the concern about having a serious illness is excessive or unreasonable

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Patients typically present for the first time with the complaint of a symptom, pain, or sensation. With some gentle questioning they quickly move from concern about the symptom to fear of a disease. Almost uniformly these patients are not concerned transiently about a minor disease, but rather are persistently worried about a serious disease. The specific disease may change from over time. The duration of an episode of a feared disease may run from months to years; alternatively, the feared disease may not change at all throughout the course of the disorder.

Pathology and Laboratory Examination

At present there are no known pathological somatic features of the disorder nor are there known routine laboratory or electrophysiological tests that help to elucidate this disorder.

Differential Diagnoses

Essential to the process of differential diagnoses is ruling out underlying organic disease. In the vast majority of cases this can be completed by the primary care physician without referral to a specialist. The workup is usually a straightforward process and typically focuses around the disease that the patient is concerned about. Somatization disorder is the main somatoform disorder that needs to be differentiated from hypochondriasis. Although there are numerous differences between somatization disorder and hypochondriasis, the major distinction is that patients with somatization disorder are concerned about their symptoms, even dramatizing them, and are relatively indifferent to concerns about underlying disease. Patients with hypochondriasis have an exactly opposite preoccupation; they fear an underlying disease and concern about symptoms quickly fades. Typically, hypochondriacal patients do not have the plethora of symptoms present in somatization disorder patients. Hypochondriasis needs to be distinguished from factitious disorder with predominantly physical signs and symptoms and from malingering. In hypochondriasis, patients actually experience the symptoms they report rather than simulating them.

Patients with psychotic disorders, particularly major depressive disorder and schizophrenia, may have somatic delusions or concerns about the presence of a disease. Hypochondriacal concerns secondary to major psychiatric disorders are categorized with the more serious disorders. Candidate conditions include major depressive disorder, dysthymic disorder, generalized anxiety disorder, obsessive-compulsive disorder, and panic disorder.

Course and Prognosis

Hypochondriasis is a chronic, relapsing condition with waxing and waning symptoms. The disorder is usually longstanding, frequently over several years. Episodes typically last months and even a few years. There are often quiescent periods between episodes; however, frequently there are recurrences during times of psychosocial distress. Symptom severity of the disease is such that some degree of psychosocial impairment occurs. Typically, familial and spousal relationships become strained by living with a patient who persistently fears disease. The individuals ability to work may or may not be affected.

Historically hypochondriasis has been given a pessimistic prognosis; however, that reputation may be exaggerated. Some authors now state that approximately 50 percent of patients show improvement; the remainder show a chronic, fluctuating course. A recent case-matched study to examine diagnostic stability and outcome of 50 cases meeting DSM-III-R criteria indicated that after 1 year two thirds of the subjects continued to meet criteria for hypochondriasis and the remaining one third had persisting hypochondriacal symptoms. The hypochondriacal subjects were improved on most measures. However, severe symptoms, longer duration of illness, and coexisting psychiatric illness were predictive of a worse outcome. In a 2-year follow-up study of 24 patients with transient hypochondriacal symptoms that did not meet DSM-III-R criteria for hypochondriasis, these patients continued to have their hypochondriacal symptoms with only one patient going on to meet full DSM-III-R criteria of the full-blown disorder. These patients continued to magnify the importance of their physical symptoms, had more functional disability, and utilized more medical care than case-matched controls. Thus, recent research has validated the

clinical view that the diagnosis of hypochondriasis appears to be stable over time, with symptoms waxing and waning. Hypochondriasis carries a very substantial, long-term burden of morbidity, functional impairment, and personal distress.

The following characteristics, which bode well for a patient's general health status, bode well for patients with hypochondriasis as well: a high socioeconomic status; the presence of other treatable conditions, such as an anxiety or depressive disorder; a sudden onset; an absence of a personality disorder; and the absence of comorbid medical disease.

Treatment

To date there are no controlled clinical trials on which to base rational treatment, therefore inferences have to be made from the treatment of other disorders and from clinical lore used in treating hypochondriasis. The simplest treatment approach is to look for and treat any comorbid psychiatric conditions, such as obsessive-compulsive disorder, panic disorder, and depressive disorder. When these conditions are treated pharmacologically and with appropriate therapy, the hypochondriasis often will improve.

A recent open trial of high dosages of fluoxetine (Prozac) on hypochondriasis patients not meeting criteria for comorbid depressive disorders showed much promise, with 10 of 16 patients much improved at the end of a 12-week trial. Several other trials with selective serotonin reuptake inhibitors (SSRIs) with primary hypochondriasis have also shown some positive results. Previously this group of patients had been regarded as refractory to all psychopharmacological treatment.

The management of hypochondriasis has typically been in the domain of the primary care physician. At least initially, these patients strongly resist psychiatric referral. Their mental model of the problem is that they have a covert physical illness and so they do not believe they need to see a psychiatrist. Secondly, psychiatry's track record using traditional psychotherapies for these patients has been rather dismal.

Psychoanalytic treatment and sometimes even psychodynamically oriented therapy of patients with full-blown primary hypochondriasis have been unsuccessful in removing symptoms and improving the function of these patients. One pragmatic approach that has heuristic merit is the use of paradoxical intention, telling the patient that you recognize the suffering but that you are not certain that you can help very much. When little is expected, sometimes small gains are appreciated and there is some improvement.

Another approach that may possess merit is group treatment. A cognitive-educational group treatment has recently been proposed that at face value appears to have validity, especially given the success demonstrated by a similar approach in patients with somatization disorder. Currently several trials of cognitive-behavioral treatment, both in groups and individually, are ongoing.

Hypochondriasis patients usually accept referral for treatment of a comorbid psychiatric condition, and such conditions are not rare for them. Finally, management suggestions that have been shown to be effective in somatization disorder should be tried in patients with hypochondriasis.

PAIN DISORDER

Definition

With their expertise in the use of psychoactive medication plus their interest in the personal and family dynamics of patients, psychiatrists have the capacity to be involved in the treatment of patients with chronic pain. Astute clinicians have long known that some patients use their pain as a way of seeking human relationships; the

secondary gain they receive from their infirmity then can be reinforced, maintaining the pain.

Co-occurring psychiatric disorders are not rare when pain is severe. It is always gratifying when a pain syndrome dissipates as an accompanying psychiatric disorder is treated, both receding together. However, the clinical question remains: was pain the cause of a coexisting psychiatric disorder or was pain the consequence of another psychiatric syndrome? One study found that in 89 percent of one clinic's patients who had both depression and pain, there was no antecedent history of depressions. This data would be consistent with the hypothesis that pain often causes depression rather than vice versa.

Pain is a highly subjective matter and is extremely difficult to quantify. For example, it has always been difficult to specify just how much chronic pain ought to be associated with a given lesion. Assessing the degree to which emotional factors are intensifying the patients' complaints is also complicated. Moreover, patients with chronic pain are not always immediately motivated to give an intricate personal and family history because they fail to see its relevance to their pain.

There is no accepted objective way to measure pain, and a variety of different measures of pain intensity and severity are currently used in the field, which makes it difficult to compare different clinical studies. Finally, many studies in the pain literature have demonstrated considerable differences in study populations, with different personalities and different cultures experiencing and expressing themselves in divergent fashions.

Wilder Penfield's clinical wisdom about patients with chronic pain has now become rather widely accepted in the field: if the patient is not a malingerer, the complaints about the extent of the pain are to be believed. The issues that need to be explored clinically are: (1) how disabled by the pain is the patient and (2) to what extent are there complicating emotional factors and comorbid psychiatric conditions. The physician attendant to the suffering of a patient with chronic pain needs to render a diagnosis that communicates important underlying aspects of the case and leads to a treatment plan that encompasses the patient's emotional needs.

Comparative Nosology

There have been a variety of informal and formal diagnostic terms associated with pain. For example, the euphemistic term atypical has been used, as in atypical facial pain, or atypical pelvic pain, to indicate that psychiatric issues needed to be considered in the cause or the maintenance of the severe pain syndrome. ICD-9 used the term psychalgia and DSM-III incorporated the term psychogenic pain.

Previous terms such as chronic pain syndrome, psychogenic pain, and DSM-III-R's somatoform pain disorder were used as psychiatric diagnoses for patients in pain wherein psychological factors have played a pronounced role. DSM-III-R required preoccupation with pain for at least 6 months and either no organic pathology or no pathophysiological mechanism found after appropriate evaluation; if organic pathology is present, the extent of the complaints about the pain or the degree of social-occupational disability should be greater than could be expected by the examiner based on the physical findings.

Applying the diagnostic terminology of DSM-III and DSM-III-R, however, has proved difficult. Even when diagnostic criteria required only no organic pathology or impairment in excess of what would be expected, precise application of criteria was problematic. Judging whether the amount of a patient's subjective pain is excessive became a daunting task and lacked validity and reliability studies. Furthermore, these

diagnoses failed to adequately describe persons who have a physical cause for pain but react to their pain in a dysfunctional manner.

DSM-IV provides a more focused approach, not relying on the extent of the pain as a criterion but asking the clinician to make a judgment as to whether psychological factors have a major role in the onset, severity, exacerbation, or maintenance of the pain, and as to whether or not there are factors from the general medical condition that also make a major contribution to the symptoms.

DSM-IV divides pain disorder into acute and chronic subsets. Another new feature of DSM-IV is that it splits pain disorders into three subtypes: pain disorder associated with psychological factors, pain disorder associated with both psychological factors and a general medical condition, and pain disorder associated with a general medical condition (which is not considered to be a mental disorder but is included to help in the differential diagnosis). Patients with chronic pain do not readily fall into dichotomous groups, those either with physical or with mental illness. According to one student of chronic pain, "Many patients have both; a few have neither."

ICD-10 labels pain disorder as "persistent somatoform pain disorder." ICD-10 does not subdivide pain into subtypes in which psychological factors play a major role along with a general medical condition and in which psychological factors are the determinant of pain. ICD-10 excludes backache not otherwise specified, tension headache, muscle tension, and migraine. Additionally, ICD-10 retains more of a DSM-III-R approach regarding emotional factors involved in the cause of the pain, indicating that "persistent somatoform pain occurs in association with emotional conflict or psychosocial problems that are sufficient to allow the conclusion that they are the main causative influences." ICD-10 is inconsistent in that it does not specify, as DSM-IV does, that emotional factors can impinge on certain aspects of the pain condition (onset, severity, exacerbation, or maintenance). Finally, ICD-10 goes beyond DSM-IV's statement that "pain $\frac{1}{4}$ is the predominant focus of clinical attention and is of sufficient severity to warrant clinical attention" when it describes the outcome of persistent somatoform pain disorder in this way: "Usually [there is] a marked increase in support and attention, either personal or medical."

Epidemiology

Pain of one sort or another is among the most frequent reasons that patients consult their physician. One study found that 13 percent of patients in a private internal medicine practice suffered from chronic pain. In one sample of health maintenance organizations, 8 percent of patients had severe persistent pain and 2.7 percent had severe persistent pain with at least 7 or more days of pain-related activity restriction in the past 6 months. Most pain is remediable, but chronic pain carries with it a high price: more than \$10 billion was spent in 1980 in disability payments for chronic pain problems. Recent data from numerous local and regional surveys leads to a yearly estimate of \$60 billion as the annual cost to the nation from health care costs and loss of work from patients of chronic pain. Low back pain alone currently affects more than 7 million Americans. Another cost of pain disorders is suicide, with one report stating that the suicide rate among pain sufferers is 9 times that of the general population.

Because diagnostic criteria for the disorder have been changing (from DSM-III to DSM-III-R to DSM-IV), detailed investigations that pull together the databases are still lacking. Studies seem to indicate that there are twice as many women as men with pain disorder, with peak incidence in the fourth and fifth decades, especially in those in blue-collar jobs. There is also some tendency for familial aggregation, along with more depression and more chemical dependency use disorders in those families.

Comorbidity There is a high frequency of comorbid psychiatric diagnoses in most groups of chronic pain patients. One study reported that 56 percent of their sample of 283 chronic pain patients suffered from one of the depressive disorders and that alcohol or drug dependence occurred in 15 percent. Finally, 58 percent met criteria for a personality disorder (most frequently dependent, passive-aggressive, or histrionic). Since these studies of personality disorder are retrospective, it remains unclear whether the pain itself has caused the patient's personality traits to become a personality disorder, or whether having a personality disorder predisposes one to developing a pain disorder.

Etiology

Pain comes from many sources, but the peripheral afferent nerves, central nervous system processing, and central nervous system interpretation of the localization and the severity of the pain are almost always involved. In some cases of pain disorder of the psychological or the combined type, a purely neurosurgical approach (eliminating a painful lesion and its connectivity) can successively chop away at parts of a person's peripheral and central nervous system until the cortex is no longer in contact with the relevant nerves or with the involved spinal cord from an afflicted area. In some cases the pain can still continue.

No adult registers an experience on a tabula rasa; rather, persons all encounter life happenings in a context of (1) personal training based on critical developmental incidents; (2) family-centered culture transmitted early in life; and (3) societal values about appropriate behaviors, including the expression of feelings. These past experiences all interact with a person's constitutional hardwiring, including preprogrammed response modes.

Chronic pain syndromes are among the most frustrating conditions in all of medicine for patients and physicians. To even have a chance at being successful in dealing with patients in pain clinicians need to be able to appreciate the patient in a biopsychosocial context. Successful academic pain clinics have discovered the necessity of a multidisciplinary approach. Indeed, physicians caring for such a patient need to do more than just take a detailed history; they also need to understand the patient's life from the patient's as well as the family's perspective.

For reasons they may or may not be aware of, some individuals magnify a given pain; others pay little attention to it, diminishing their response to a lesion that might otherwise cause great discomfort. Pain thresholds, which can be clinically measured, vary from person to person. Pain tolerance, likewise, has considerable latitude.

Moreover, there is a broad range of ways in which individuals express their discomfort or even their agony. Some with a broken ankle will be stoic; others with the same fracture will scream out in agony, facies pinched, arms flailing.

Although there are conditions in which persons do not have pain fibers (familial dysautonomia), this is so rare that they are not a factor in everyday practice. It is easier to divide the discussion of issues in pain into four categories: intrapersonal factors, interpersonal factors, ethnic factors, and biological factors.

Intrapersonal Factors Intrapersonal factors can impact how pain is experienced.

Psychodynamically formative events in the early years can carry over later into how one processes illness and other types of distress. One psychodynamically oriented theory is that having pain and expressing distress attracts others to deliver needed attention. In later years the experience of pain can be associated with obtaining love from an otherwise absent significant caregiver.

Another psychodynamically oriented theory holds that painful experiences, such as repeated severe corporal punishment from an abusive parent, can set the stage many

years later for the person's response to a wrongful piece of behavior with self-imposed retribution. In this way a pain disorder may be used to pay for some self-perceived sin or transgression. However, simple interpretation (illuminating the repressed, suppressed, or even grudgingly embellished facts) rarely serves to remove the pain dramatically.

Recent research data suggests that pain patients have a background of past sexual abuse or trauma and the resultant posttraumatic stress disorder. Some patients can be vaguely or poignantly aware of the associations between past life events and being pain prone or even being overresponsive to a painful experience. For other pain patients, the associated early-life experiences of pain are deeply buried and emerge only through a lengthy process of free association, dream work, or psychological testing.

Not every one with severe pain and unusual pain behavior has had dysfunctional early-life experiences that caused a psychopathological organization of the character structure. However, astute clinicians are always alert to the possibility that there may be important masochistic, dependent, or narcissistic features to the personality, or traumatic life experiences that impact how a given patient handles pain.

No one psychological test can accurately delineate the extent of the psychological component in a pain syndrome. Any painful chronic disease highlights and even changes certain components of personality. Psychological testing and patient interviews can yield clear evidence that some patients with considerable emotional overlay to their pain are at least initially resistant to suggestions that their pain symptoms are caused by anything other than physical factors.

Finally, it is likely that intense pain affects attitudes and emotions, and that attitudes and emotions influence the reported perceptions of pain intensity.

Interpersonal Factors Interpersonal factors may also impact on the pain experience and behaviors can be reinforced or inhibited. For example, reinforcements given by others important to the pain-afflicted person can play a major role in day-to-day and even second-to-second experiences. Lavishing attention on an individual when chronic pain intensifies creates a different inner and outer world for the patient than when the pain is either ignored or treated with additional painful experiences.

Some persons use their pain to manipulate others. Clinicians often observe this when there is a chaotic and otherwise inattentive family or in the case of a dysfunctional marriage that becomes reorganized around the continued crisis of chronic pain.

Surprisingly, in some persons the persistence of pain is far less important than what it is traded for: a relationship that is far more to the patient's liking.

The consequences of pain are often gain. Secondary gains, such as monetary rewards or avoidance of distasteful activities, are not an unusual consequence of having prolonged pain. It is only when interpersonal and other rewards are stripped away from the pain experience and its attendant disability that the individual with pain can develop a more normal life.

Like a somatization disorder, a pain disorder can become a way of life that alters the behavior of other people. The caring physician must always be aware of interpersonal factors. Family, financial, and legal dynamics of a situation can have a dramatic influence on the patient's pain experience and pain behavior.

In one study, male patients with chronic pain who received financial compensation following injury at work showed a particularly high rate of personality disorder (83 percent) which supports a contention in the literature that patients with "compensation neurosis" were frequently passive-aggressive, hostile, and inadequate.

Ethnic or Cultural Factors Quantitative and qualitative research projects of chronic pain sufferers in New England and Puerto Rico by the same investigators reinforce what astute clinicians have known all along—that similar lesions in patients from different backgrounds can produce profound variations in pain experience and pain intensity. The style of the typical taciturn "old Mainer" is quite different from the emotionally labile and highly expressive native Puerto Rican. Despite higher pain intensity and more emotional responses among Puerto Ricans, there was no significant difference between the two groups regarding interference in daily activities. Attitudes, beliefs, and emotional and psychological states can differ according to ethnic group affiliation. Moreover, the individual's psychological coping style and internal or external locus-of-control style, which are important determinants of how an individual handles the pain, are to a considerable extent culturally determined.

Biological Factors Biological factors also play a large role in the onset and perpetuation of chronic pain. Cortical and subcortical centers process and filter afferent impulses. Sensory and limbic structural abnormalities can determine the severity of a pain experience. Neural pathways serving pain are not "hard wired," but demonstrate considerable plasticity in response to injury. After an injury, changes occur in the sensitivity of the pain receptors themselves and also in the excitability of neurons in the dorsal horn of the spinal cord. Thus symptoms of chronic pain following injury may have a physiological basis in changes in the brain, spinal cord, and peripheral nerves.

Neurological changes resulting from recalibration of pain thresholds may be the result not of gross external injuries but rather of nonobvious injury resulting from mechanical stresses such as increased muscle tension (associated with anxiety or depression), and mechanical stresses of the lumbar spine. Additionally, there could be an associated muscular ischemia with the release of pain-producing substances such as substance P and histamine that alter the pain threshold.

The gate control theory purports that a gating mechanism in the dorsal horn of the spinal cord handles afferent pain signals; competing signals as well as neurotransmitters then can open or close the gate on painful perceptions. Endorphin deficiency seems to correlate with the augmentation of afferent stimuli. Serotonin, presumably relatively diminished in some forms of depression, has now been implicated as the main neurotransmitter in descending inhibitory pathways. Such mechanistic studies still remain preliminary but offer interesting explanations into a patient's plight when pain is burdensome but organic lesions do not appear major. Thus, the underlying psychodynamic and neurobiological framework can impact on the complex ways a given person experiences and re-experiences pain.

Pathophysiology and psychopathology may well be reciprocally linked in self-perpetuating cycles.

Diagnosis and Clinical Features

Pain disorder as a psychiatric condition is diagnosed when a patient's preoccupation with pain is consuming and to some extent disabling. That is, pain becomes the predominant focus of the clinical presentation and the pain itself causes clinically significant distress or impairment and the patient's life becomes organized around the pain; also, psychological factors are judged to play a role in this disorder. Table 16–14 lists the diagnostic criteria for pain disorder.

Table 16-14. DSM-IV Diagnostic Criteria for Pain Disorder

A. Pain in one or more anatomical sites is the predominant focus of the clinical presentation and is of sufficient severity to warrant clinical attention.

- B. The pain causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.
- C. Psychological factors are judged to have an important role in the onset, severity, exacerbation, or maintenance of the pain.
- D. The symptom or deficit is not intentionally produced or feigned (as in factitious disorder or malingering).
- E. The pain is not better accounted for by a mood, anxiety, or psychotic disorder and does not meet criteria for dyspareunia.

Code as follows:

Pain disorder associated with psychological factors: psychological factors are judged to have the major role in the onset, severity, exacerbation, or maintenance of the pain. (If a general medical condition is present, it does not have a major role in the onset, severity, exacerbation, or maintenance of the pain.) This type of pain disorder is not diagnosed if criteria are also met for somatization disorder.

Specify if:

Acute: duration of less than 6 months

Chronic: duration of 6 months or longer

Pain disorder associated with both psychological factors and a general medical condition: both psychological factors and a general medical condition are judged to have important roles in the onset, severity, exacerbation, or maintenance of the pain. The associated general medical condition or anatomical site of the pain (see below) is coded on Axis III.

Specify if:

Acute: duration of less than 6 months

Chronic: duration of 6 months or more

Note: the following is not considered to be a mental disorder and is included here to facilitate differential diagnosis.

Pain disorder associated with a general medical condition: a general medical condition has a major role in the onset, severity, exacerbation, or maintenance of the pain. (If psychological factors are present, they are not judged to have a major role in the onset, severity, exacerbation, or maintenance of the pain.) The diagnostic code for the pain is selected based on the associated general medical condition if one has been established or on the anatomical location of the pain if the underlying general medical condition is not yet clearly established—for example, low back, sciatic, pelvic, headache, facial, chest, joint, bone, abdominal, breast, renal, ear, eye, throat, tooth, and urinary.

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Pain disorder patients make repeated visits to physicians, often successively or even concomitantly and doctor "shopping" is very common. There is a risk of excessive use of narcotic or sedative hypnotic agents because the patient is enveloped by pain and often has associated complaints of anxiety, depression, and insomnia. Thus, any related comorbid psychiatric conditions must also be treated to alleviate the patient's pain.

The major feature of pain disorder is an all-encompassing focus on the pain such that any other concern takes on less than its ordinary significance. The pain is believed by the patient to be the source for all the patient's misery.

If the patient is depressed, anxious, or has insomnia, those symptoms are almost always considered by the patient to be secondary to the pain. Other frequent ancillary symptoms include changes in appetite, loss of energy, decreased interest in social activities, decline in sexual interest, diminished physical exercise, and even diminished exertion. Also often noted are a change in normal recreational pursuits, a breakdown in family relationships, an increased amount of time spent in bed or lying down, an increased amount of self-absorption (including hypochondriasis), and multiple drug use or abuse. Frequent visits to physicians and requests for medical or even surgical approaches to obtain pain relief are commonplace.

One of the possible predisposing factors for handling pain poorly is childhood sexual abuse, for example, in patients with chronic pelvic pain. Another predisposing factor is pending litigation, as in the case of whiplash injury. Here the personal meaning of a symptom can mitigate or increase the pain's intensity and the severity of the incapacitation.

By contrast, sometimes refractory pain with a presumed strong psychogenic component turns out to be a treatable neurological or orthopedic condition that has been missed by the referring clinician. In one report from a multidisciplinary pain clinic keeping tally on these matters for 120 patients with chronic pain, the overall rate of inaccurate or incomplete diagnosis at referral was 40 percent. Commonly missed diagnoses were myofascial disease, facet disease, peripheral nerve entrapment, temporomandibular joint disease, thoracic outlet syndrome, and herniated discs. After comprehensive workup, an organic origin for the pain was found in 98 percent of these patients.

Sites of Pain Typical sites and types of pain involved in pain disorder are headache, atypical facial pain, low back pain, and chronic pelvic pain; however, any site is a potential target. Pain can emanate from almost any point source in the body or it can be a conversion symptom involving just pain, using a previous pain pattern from organic disease as a conversion pain model. If the pain is used as a metaphor for psychic turmoil converted into a somatic form, it may bring to the afflicted person primary and secondary gains that reinforce the initiation, maintenance, and even exacerbation of pain symptoms for psychological purposes. When pain is a conversion, its distribution may not conform to known anatomical patterns. From a diagnostic perspective, pain cannot be diagnosed as a conversion disorder according to DSM-IV.

Mr. L., a 72-year-old married, Ukrainian-born, pious, wealthy retailer and father of a large family from an east coast city was admitted to the orthopedic service of a general hospital for evaluation of unbearable pain in the arches of his feet. He had fled his native country following a pogrom when he was 9. During the year of flight he had endured enormous physical hardships, starvation, and beatings until the surviving family members finally were able to emigrate to the United States. With incessant hard work, he had prospered economically; married a patient, supportive wife; and witnessed his six children develop promising careers. He became the major contributor to his temple and gave unstintingly to local charities for the needy and unfortunate. He had little time for personal enjoyment. Over the years each time he and his wife had time alone together and she had been affectionate with him, he would develop some excruciating bodily pain shortly thereafter: blinding headache, severe back spasm, abdominal pain, facial pain, or pelvic pain. These pains usually receded several days after the weekend was over or the trip was completed. Some pains occurred more frequently than others. He sought medical attention rarely except for these pains, which occurred every few months. His mood varied from glum to

gloomy but he denied that he was depressed. He often claimed to have been blessed with good fortune. He led a temperate life, drank little, and had relatively good health between these episodes of pain.

Over the four decades that his physicians cared for him, they had become frustrated with this unassuming and humble man; his ardent complaints of pain were always so nonspecific and fluctuating in nature that they could not describe adequately for themselves any pathophysiological mechanisms to account for his pain. Their diagnostic tests were not revealing and Mr. L. usually refused their offers of narcotic or other analgesic relief. Laboratory workup for pains in the arches was noncontributory, and he was discharged when his symptoms cleared in 3 days. Four months later he was readmitted to the surgical service of the general hospital with severe, unrelenting, left-side upper abdominal pain. This time Mr. L. described this new pain in meticulous detail. A brief workup revealed very advanced carcinoma of the tail of the pancreas. He took the news from his physician stoically and asked to be discharged home that day.

Differential Diagnosis

All pain is subjective and only obvious if the patient tells the physician about it. Acute pain tends to distort the facies in a grimace, causing muscle tightening and elevation of autonomic measures, such as pulse and blood pressure. Chronic pain, however, produces none of these signs. Clinicians trying to explore the extent of psychiatric involvement have used a number of clues, the validity of which still remains to be proved (Table 16–15). Those are the underlying dynamics and the presence or absence of conflicts and stressors are open to multiple interpretations; only careful long-term follow-up studies of large series of patients will help increase validity.

Table 16-15. Clues to Underlying Psychopathology in Pain Disorder

Antecedent history of poor premorbid adjustment: alcohol or drug abuse, sexual difficulties, multiple marriage, inability to hold a job

Temporal relationship between an environmental stimulus, relevant intrapsychic conflict, and pain

Utility of pain in either gaining compensation or avoiding situation deemed noxious by the patient

Lack of variation in the amount of pain from distraction, suggestion, or fear

Patients deal with their chronic illnesses in ways characteristic of their dynamics and their own psychopathology. Those with a histrionic nature exaggerate; those who use a history of ill luck to manipulate their environment make the most of their pain for their own ends; those with a need for punishment for real or imagined sins may fulfill their needs for atonement.

Both acute and chronic pain tend to be very field dependent. For example, soldiers who have survived fierce fighting on the battlefield typically need far less morphine for relief than their civilian counterparts with the same degree of tissue injury.

Absolute pronouncements about no somatic pathology or no emotional components to the clinical picture must be viewed with skepticism. A positive response to a trial of placebo medication does not indicate anything about the organic nature of the patient's pain or the extent of the underlying psychopathology. The placebo response is positive in about one third of whatever population is tested. Pain associated with both melancholia and metastases can respond to placebo. Patients and their clinical syndromes need to be treated without too much loss of time deliberating about whether the pain caused the depression or the depression caused the pain. Individuals with personality disorders and chronic pain tend to have a more difficult time coping

with any type of difficulty, both in the hospital and at home, and they are a challenge to their physicians. Fortunately, intensive inpatient treatment of refractory chronic pain has had some success.

Depressive Disorder Of the psychiatric diseases, depression presents most often as a pain disorder. At times the individual will have had previous depressive episodes with an altogether different clinical presentation and with no associated pain. Sometimes the best clue to the presence of a mood disorder is just the family history of severe depression, with or without pain being part of the clinical picture. There is no specific type of pain picture to alert the clinician to the presence of depression. Moreover, the sadness accompanying the pain disorder can almost always be ascribed to the pain. Supporting the diagnosis of a mood disorder are the vegetative signs of depressive disease, especially sleep and libidinal disturbances. Cognitive signs of an accompanying depression include apathy, decreased interest in work, suicidal ideation, and a preoccupation with death. Collaborative sources of information are often needed to fully understand the patient's level of functioning.

Sometimes the best way to make the diagnosis of depressive disorder in a patient presenting with pain is simply to initiate a treatment trial of an antidepressant medication in addition to nonspecific modalities. Dramatic clinical relief of both the pain and the vegetative signs of depression provides the answer to the clinical question. Suicide in chronic pain patients must always be kept in mind, especially during the first 10 days of treatment with an antidepressant when the patient begins to look better and has more energy; such a state often occurs before the feeling of despair has lifted.

Conversion Disorder When a conversion mechanism is involved in the pain disorder, the clinician determines that (1) details of the pain picture have a symbolic or other specific meaning for the patient; (2) there is a conversion model, often a parent or other close family member; and (3) the pain follows no known anatomical pathway. By convention, pain involving a conversion mechanism is diagnosed as a pain disorder, not as a conversion disorder.

Treatment for this is similar to the psychotherapy of a patient with a conversion disorder. In addition, the clinician will use the same nonspecific treatment meted out to many or all pain disorder patients: physiotherapy, water pool massage, infrared heat, nonsteroidal anti-inflammatory agents, and other such modalities.

Psychosis When a psychosis associated with schizophrenia, medical disorder, or a dementing illness presents with pain, the pain itself is delusional and the pain pattern is atypical in quality and in distribution. Treatment must relieve the underlying psychiatric disorder. Delusional beliefs, however, may prevent the patient from cooperating with the treatment plan.

Malingering When someone is purposely lying about pain, the pain is hard to detect. Such individuals are not interested in the same outcome as the physician, which is relief of pain. Sometimes old records betray that the patient has tried to mangle in other settings and in other ways. Unlike the patient with Munchausen syndrome (factitious disorder with predominantly physical signs and symptoms), who is interested in staying in the sick role, the malingerer is consciously interested in some other benefit. Some clinicians have found that Munchausen syndrome patients will willingly agree to painful procedures whereas the malingerer will not.

Emotional Overlay Determining the extent of the emotional component of chronic pain is helpful, and care plans could then be effected, along with appropriate expectations and appropriate treatment. The well-known MADISON scale (Table 16-16) has not been rigorously validated but has been of considerable help to those

working in the field. Another instrument that has proven to be clinically valuable in assessing whether a patient has an organic lesion that potentially amenable to a corrective surgical approach is the Mensana Pain Test, a 10-minute structured medical interview.

Table 16-16. MADISON Scale for Markers of Considerable Emotional Overlay

M = Multiplicity: Pain is either in more than one place or of more than one variety; when treated, may recur elsewhere.

A = Authenticity: More interested in clinician's acceptance of pain as genuine than in a cure.

D = Denial: Especially exaggerated marital or family harmony; when admitting depression or anxiety, no impact on pain is admitted.

I = Interpersonal relationship: Although the connection to the presence of any particular person's company as worsening the pain may be denied, observation of the patient's nonverbal and interactive behavior indicates otherwise.

S = Singularity: When the pain is described as unlike that of anyone else, ever.

O = "Only you": When the patient immediately idealizes the physician as savior, despite numerous failures by other competent experts.

N = Nothing helps, or no change: When there is no relief whatsoever from any type of intervention, although all are tried (including narcotics) and there is no hour-to-hour or day-to-day fluctuation under a variety of circumstances.

Table adapted from Hackett TP, Cassem NH: Massachusetts General Handbook of General Hospital Psychiatry., St. Louis, 1978.

Course and Prognosis

Once diagnosis is complete, treatment on an outpatient basis can be carried out by concerned physicians who will see the patient on a regular basis, be interested in the patient's complaints, and assure the patient that treatment will continue if there is some improvement. Pain disorder patients with pending litigation often attain no, or only minimal, relief until after the legal proceedings are finished.

Pain-Prone Individuals Persons who are painprone often experience pain in response to losses or other stresses, presumably as a way of dealing with unresolved guilt.

Outcomes from treating the pain-prone are typically described as transient or poor.

Techniques that are most promising are supportive therapy, a modicum of insight, and, when indicated, antidepressant medication.

Treatment

Many patients, especially those with depression, can be considerably helped. The key to dealing with the pain disorder patient is accurate diagnosis and focus on the patient's level of functioning. The role of the family in the pain patient's life must be determined and efforts must be made to intervene in the dysfunctional components of family life. The patient's pain and predicament are always acknowledged at each visit, but the major focus is in moving on and regaining function. Pain clinics and inpatient pain treatment units both have had their share of success.

Treatment of the patient with any of the pain disorder subtypes needs to be multidisciplinary and multidimensional. Controlled trials seem to indicate that group therapy with antidepressant medication seems to have an advantage over individual therapy with antidepressant agents.

Treatment programs need to be culturally sensitive in terms of intake procedures and treatment planning. Treatment teams need to be aware of the potential effect of ethnic background on the communication, concerns, and coping styles related to the chronic pain experience of these patients.

Treatment team planning is important from the onset, coordinated as needed on a periodic basis. Multiple regimens often help in reducing pain intensity. Tailored to the patient, options include nonsteroidal anti-inflammatory agents, tricyclic medications, selective serotonin reuptake inhibitors, nerve blocks, localized electrical stimulation, visual imaging, relaxation, physical therapy, hypnosis, counseling, cognitive behavioral therapy, and supportive psychotherapy. Currently the choice of which form of psychotherapy to use is based on empiric grounds and personal preference because no case-matched studies have yet been performed. Research data is clear, however, that success in treatment result in a decrease in the patient's catastrophizing and an increase in the patient's perceived control over the pain.

If opioids have been used for chronic refractory pain, they should be tapered and discontinued because obviously they were not helping and may have been interfering with the patient's progress. Abuse of anti-anxiety medications may also be present in patients with chronic pain and must be dealt with. Antidepressant medications often play an important role in decreasing pain, even in the absence of a frank depressive syndrome. The possibility of a chronic sleep disorder must be investigated and appropriately treated, if present.

Price of Chronic Pain The clinician needs to be aware that any type of chronic pain can alter the patient's personality and family dynamics. Pain can also alter the way the patient relates to the environment and perhaps also the patient's internal locus of control style. At the onset of treatment the clinician needs to set guidelines for continuing and discontinuing treatment. No improvement in any way after 6 months is grounds for termination of treatment. If improvement is shown, the patient will need continued support. Often the patient's objective improvement far exceeds the subjective improvement.

BODY DYSMORPHIC DISORDER

Definition

Body dysmorphic disorder focuses on the patient's feelings of dislike or even loathing for some aspect of the body's appearance. Despite the starkness of the complaint, few empirical data about the condition have been gathered although clinical investigations into this disorder are greatly increasing.

Patients with body dysmorphic disorder have a pervasive subjective feeling of ugliness of some aspect of their appearance despite a normal or nearly normal appearance. The core of the disorder is the person's strong belief or fear that he or she is unattractive or even repulsive. This fear is rarely assuaged by reassurance or compliments even though the typical patient with the disorder is quite normal in appearance. Although a minority of patients with the disorder do have a minor defect, the patient's concern is disproportionate to the degree of defect. So great is the individual's preoccupation that there is significant impairment in social or occupational functioning, or there is marked personal distress. All other problems are illogically attributed to the perceived cosmetic defect, accompanied by the unrealistic expectation that surgery will correct the defect and the patient's deficient life. Finally, the preoccupation is not better accounted for by any other mental disorder.

History

Body dysmorphic disorder has been described in the European, Japanese, and Russian psychiatric literature for almost a century with various names. Emil Kraepelin considered it to be a compulsive neurosis. Pierre Janet called it an obsession of shame of the body, and Freud's famous Wolf-Man case was obsessively concerned about the size of his nose. In the United States this disorder was initially classified as an

atypical somatoform disorder, labeled first as dysmorphophobia; however, until the late 1980s it has been little used and little studied in the United States.

Comparative Nosology

In the United States body dysmorphic disorder first appeared in DSM-III with the term "dysmorphophobia" in the residual category of atypical somatoform disorder. Because dysmorphophobia inaccurately implied the presence of a behavioral pattern of prominent avoidance of the body, the term "body dysmorphic disorder" was introduced in DSM-III-R as a nondelusional somatoform disorder of undue preoccupation with imagined defects of appearance. Clinicians have wrestled over whether a patient's strong conviction about a feature of appearance was or was not of delusional intensity (i.e., whether the patient had a somatoform disorder or a delusional disorder with somatoform features). DSM-IV resolves that issue by not requiring that the conviction be defined in intensity as a criteria for inclusion. Delusional body dysmorphic disorder can be double coded as both a delusional disorder and as body dysmorphic disorder—a compromise that underscores the uncertainty about whether they are the same or different disorders. The delusional form of the illness may be a more severe type of the disorder. It was first incorporated into ICD-9 with the somatoform disorders and is grouped with them in ICD-10.

Epidemiology

Accurate prevalence data for this disorder in the general population are not available. From the clinician's perspective, the full-blown syndrome is uncommon. Concern over appearance is culturally determined and dissatisfaction of a transient nature is particularly high in adolescents. However, to justify the diagnosis the individual must have clinically significant distress or impairment, which is not often found in teenagers.

The largest series to date, with 188 cases, had an equal balance of men and women. The average age of patients first diagnosed with body dysmorphic disorder is 30 years, and a high percentage have never married and are unemployed. Patients with the disorder first develop symptoms in adolescence or young adulthood. Typically they come from middle-class families and constitute a small, underrecognized and distinct group of patients in the general population. Only about 2 percent of those attending a university hospital plastic surgery clinic meet criteria for the disorder. Body dysmorphic disorder is not uncommon as a comorbid condition in patients with major depressive disorder (current rate of 60 percent; lifetime rate of 80 percent), obsessive-compulsive disorder, and social phobia. Indeed in one study of 30 patients, all met DSM-III-R criteria for at least one other psychiatric diagnosis at some point in their lives, and usually concurrently.

Etiology

The cause of body dysmorphic disorder is unknown. The high comorbidity with depressive disorders, a higher-than-expected family history of mood disorders and obsessive-compulsive disorder, and the reported responsiveness to SSRIs indicates that, in at least some patients, the pathophysiology of the disorder may involve serotonin and may be related to other disorders. There may be significant cultural or social effects on patients with body dysmorphic disorder because of the emphasis on stereotyped concepts of beauty that may be emphasized in certain families and within the culture at large. In psychodynamic models body dysmorphic disorder is seen as reflecting the displacement of a sexual or an emotional conflict onto a nonrelated body part; such a putative association occurs through the defense mechanisms of repression, dissociation, distortion, symbolization, and projection.

Diagnosis and Clinical Features

There are several likely places to find patients with this uncommon disorder: in a mood disorders clinic, in a plastic surgery clinic, and in a dermatology clinic. A patient with a body dysmorphic disorder might request rhinoplasty; removal of facial sags, jowls, wrinkles, or puffiness; or breast reduction or augmentation. In a clinic for refractory and recurrent depression a patient with the disorder might request relief from a depression caused by an imagined but burdensome bodily defect, but because of embarrassment or shame might not reveal the self-loathing of a body part. In a dermatology clinic, the concern could be acne, scarring, or blemishes. It is a secret disorder, often not admitted to, yet it is often a time-consuming one that engages the patient for many hours during the day with activities such as excessive checking of the defect in mirrors or other reflective surfaces, skin picking, hair combing, or frequently asking others for reassurance. Frequent accompanying symptoms, presumably related to suffering from discomfort about appearance in social situations, are insomnia, depression, and anxiety. Social isolation, dissatisfaction with relationships, shame, and low self-esteem are also common.

Facial flaws are the most common defect in body dysmorphic disorder. Other body parts that sometimes become a focus include hair, breasts, and genitalia. Commonly associated with the distorted belief about appearance is an unrealistic concept of how much one's image can be improved by surgical intervention. Some clinicians report that these patients, for all their preoccupation with their defect, still are vague or inconsistent in the description of it.

Family histories of substance abuse and mood disorder are common in reported cases. Also predisposing to the disorder may be certain types of personality characteristics, especially a mixture of obsessional and avoidant traits, but no single personality pattern predominates. Reportedly the patients are shy, self-absorbed, and overly sensitive to their imagined defect as a focus of notice or criticism. As adolescents, some reports hold that they had few friends and seldom dated. There may be some ideas of reference related to the body dysmorphic disorder, but otherwise there is no formal thought disorder. The major abnormality on mental status examination is the lack of insight into the nature of the problem. Table 16–17 lists the DSM-IV criteria for body dysmorphic disorder.

Table 16-17. DSM-IV Diagnostic Criteria for Body Dysmorphic Disorder

- A. Preoccupation with an imagined defect in appearance. If a slight physical anomaly is present, the person's concern is markedly excessive.
- B. The preoccupation causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.
- C. The preoccupation is not better accounted for by another mental disorder (e.g., dissatisfaction with body shape and size in anorexia nervosa).

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Differential Diagnosis

Body dysmorphic disorder patients have an overvalued idea about their defective appearance. A number of other psychiatric disorders are also accompanied by odd or unusual ideas about the body. In general such patients are best accounted for diagnostically by considering other major psychiatric disorders as primary, and adding that body dysmorphic symptoms are also present. For example, especially in the early phases of schizophrenia some patients may have somatic delusions for which

they seek correction by cosmetic surgery. In such cases there is an absolute conviction of ugliness as well as other bizarre delusions and perhaps even hallucinations.

Other disorders with body dysmorphic symptoms include mood disorder, narcissistic personality disorder, and anorexia nervosa; in such instances, both diagnoses should be made. Melancholically depressed patients often have additional delusions about some aspect of their body's inadequacy or defect. The presence of a mood disorder, if suspected, can be further elucidated through the taking of the appropriate medical history. Patients with narcissistic personality disorder have a continual interest in their appearance and a long history of interpersonal difficulties that overshadows the body image problem. Constant preoccupation with feared obesity and inadequacy in an obviously malnourished person may be best classified as anorexia nervosa.

A certain proportion of patients with obsessive-compulsive disorder also have body dysmorphic disorder, in which case both disorders should be diagnosed. If the patient has obsessions about appearance and associated compulsive behaviors (such as mirror-checking), the diagnosis of obsessive-compulsive disorder is not made if the obsessions only concern aspects of appearance.

Isolated somatic delusions unrelated to appearance and accompanied by absolute certainty without other evidence of thought disorder are best classified with the delusional disorders. The belief that one's gender is not rightly assigned (according to the appearance of the external genitalia at birth) is better accounted for by a diagnosis of gender identity disorder. Opinions still vary as to whether monosymptomatic hypochondriacal psychosis (a delusion about a disease or disfigurement in a body part) and body dysmorphic disorder are one or two disorders. DSM-IV allows for double coding if it seems relevant to the clinician.

Course and Prognosis

Onset of the disorder may be gradual, during childhood, adolescence, or in the 20s. Discontentment may build for several years before the person considers some type of definitive surgical correction; however, surgical treatment produces no relief. Several long-term follow-up series of patients who have undergone cosmetic surgery have indicated the frequent later emergence of even more severe psychopathology.

Treatment

Neither surgical nor psychotherapeutic intervention has had any significant long-term impact on decreasing the preoccupation with defective bodily appearance in most patients with body dysmorphic disorder. There are case reports that psychotherapy alone has helped some patients with this disorder: presumably those helped most by this treatment modality are more emotionally intact, but current thinking in the field is that psychotherapy and medication are both indicated.

Case reports note positive effects from tricyclic drugs, monoamine oxidase inhibitors (MAOIs), and pimozide (Orap), a dopamine-receptor antagonist. Recent preliminary data now support the efficacy of SSRIs and cognitive-behavioral therapy. Moreover, the efficacy of SSRIs appears to be greater than that of tricyclic drugs, which in turn are more effective than the MAOIs. Augmentation of SSRIs with buspirone (BuSpar) and antipsychotic agents, as well as the combination of SSRIs also appears to be promising. However, to date there have been no controlled pharmacotherapy trials. Some patients refractory to the tricyclics and MAOIs have recently been shown in open trials to respond to antidepressant and antiobsessional agents with potent serotonin receptor blockade, such as fluoxetine (Prozac) and clomipramine (Anafranil).

After maximal treatment response, some patients with body dysmorphic disorder may still retain remnants of their bodily preoccupation, but symptom intensity is often

diminished enough to allow patients to resume full social and personal lives.

However, relapse when patients are taken off medication is common.

SOMATOFORM DISORDER NOT OTHERWISE SPECIFIED

This is the second of the residual categories for the somatoform disorders. Like the first residual category, undifferentiated somatoform disorder, this category was created in order to classify certain somatoform disorder patients whose symptoms and associated disability do not fit the full criteria for other somatoform disorders. The diagnostic criteria for somatoform disorder not otherwise specified are presented in Table 16–18. Patients with this disorder have clinically significant distress or disabilities in social, occupational, or other important areas of functioning.

Table 16-18. DSM-IV Diagnostic Criteria for Somatoform Disorder Not Otherwise Specified

This category includes disorders with somatoform symptoms that do not meet the criteria for any specific somatoform disorder. Examples include:

1. pseudocyesis: a false belief of being pregnant that is associated with objective signs of pregnancy, which may include abdominal enlargement (although the umbilicus does not become everted), reduced menstrual flow, amenorrhea, subjective sensation of fetal movement, nausea, breast engorgement and secretions, and labor pains at the expected date of delivery. Endocrine changes may be present but the syndrome cannot be explained by a general medical condition that causes endocrine changes (e.g., hormone-secreting tumor).
2. a disorder involving nonpsychotic hypochondriacal symptoms of less than 6 months' duration.
3. a disorder involving unexplained physical complaints (e.g., fatigue or body weakness) of less than 6 months' duration that are not due to another mental disorder.

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NEURASTHENIA

Neurasthenia, literally a lack of nerve energy, was described by George M. Beard at the end of the nineteenth century to account for the physical and mental exhaustion arising from the depletion of nervous resources. This finding has given rise to numerous theories but little controlled research. The diagnosis is not included in DSM-IV, nor was it included in DSM-III or DSM-III-R. In DSM-I the condition was called psychologic nervous system reaction and in DSM-II it was called neurasthenic neurosis.

Neurasthenia is still diagnosed with some frequency in Asia and Russia but not the United States. Particularly in Asia, researchers have found neurasthenia to be a culturally sanctioned idiom of distress. In ICD-10, two main types of neurasthenia occur, with considerable overlap. One type has a predominance of increased fatigue after mental effort, thus associated with a decrease in job performance or coping with activities of daily living. Difficulty in concentration, unpleasant distracting associations or recollections, and generally inefficient thinking have all been reported. The other type has physical weakness and exhaustion after only a minimal physical effort, accompanied by muscular aches and pains and an inability to relax. Both types also have a variety of unpleasant physical feelings including dizziness, tension headaches, and feelings of general instability. Patients also worry about decreased mental or physical well-being and have irritability and anhedonia. Varying degrees of

minor anxiety and depression are also common. Sleep may be disturbed with initial or middle-phase insomnia or hypersomnia.

According to ICD-10, the condition must be present for more than 3 months and have either (1) persistent and distressing complaints of feelings of exhaustion after minor mental effort or (2) persistent and distressing complaints of feelings of fatigue and bodily weakness after minor physical effort (Table 16–19). Moreover, the patient is unable to recover from the symptoms of exhaustion or fatigue by rest, relaxation, or entertainment.

Table 16-19. ICD-10 Diagnostic Criteria for Neurasthenia

- A. Either of the following must be present:
 - (1) persistent and distressing complaints of feelings of exhaustion after a minor mental effort (such as performing or attempting to perform everyday tasks that do not require unusual mental effort);
 - (2) persistent and distressing complaints of feelings of fatigue and bodily weakness after minor physical effort;
- B. At least one of the following symptoms must be present:
 - (1) feelings of muscular aches and pains
 - (2) dizziness;
 - (3) tension headaches;
 - (4) sleep disturbances;
 - (5) inability to relax;
 - (6) irritability.
- C. The patient is unable to recover from the symptoms in criterion A (1) or (2) by means of rest, relaxation, or entertainment.
- D. The duration of the disorder is at least 3 months.
- E. Most commonly used exclusion clause. The disorder does not occur in the presence of organic emotionally labile disorder, postencephalitic syndrome, postconcussional syndrome, mood disorders, panic disorder, or generalized anxiety disorder.

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Prevalence studies indicate that, using ICD-10 criteria, about 1 percent of the population have had this condition for more than 3 months and 10 percent have it for more than a month during the past 10 years. An epidemiological study of Chinese-Americans reported that 6.4 percent of the subjects had neurasthenia, and only 3.6 percent of those had no other current or lifetime psychiatric diagnoses.

The cause of neurasthenia seems to be pleomorphic, with proponents implicating mood disorders, anxiety disorders, or conversion disorders. Others propose viral sequelae while still other investigators search for trace mineral deficiencies. Obscure neuromuscular conditions, cryptic immune dysfunction, and chronic fatigue syndrome have also been implicated in the cause of neurasthenia. Despite the broad range of unproven theories, individuals can be considerably incapacitated with symptoms of fatigue and fatigability, with or without accompanying anxiety. Diagnostic workup of the fatigue syndrome by definition is unrewarding because a medical condition causing fatigue would by definition exclude the diagnosis of neurasthenia.

There is no best treatment for neurasthenia. Randomized controlled trials have not been completed. In clinical practice, trials on antidepressant agents such as the activating selective serotonin reuptake inhibitors and psychostimulants such as

methylphenidate (Ritalin) or amphetamines have been tried. It may be that sleep, tincture of time, and empathic support are the best treatment modalities, but this has not been proven.

Mrs. B. is a 45-year-old married accountant who had an especially promising career with a large national firm for 5 years until, 10 years ago she developed the gradual onset of neurasthenic symptoms of fatigue, diffuse muscle aches, and fitful sleeping following a series of major psychological stressors including a secret extramarital affair. Her primary care physician prescribed nonspecific treatments including nonsteroidal anti-inflammatory agents and rest, all to little avail.

Two years after the onset of her neurasthenia, she developed an acute depressive episode with multiple somatic complaints. She was referred to a psychiatrist who also noted that her mother had multiple sclerosis, requiring considerable physical care from all her children as they grew up.

During treatment for this major depressive episode, Mrs. B.'s depression and neurasthenia improved gradually, but she experienced multiple side effects from fluoxetine, cyclobenzaprine (Flexeril), and even the low-impact swimming program. In addition to medication, she was treated with behavioral therapy that focused on diminishing her catastrophizing about her bodily sensations. In supportive psychotherapy she benefitted from working through both some of her insoluble family conflicts and her grief about letting go of her high career aspirations.

Her energy gradually increased toward normal. Her sleep pattern of multiple interruptions and early-morning awakening improved considerably. Her overall outlook on a life of diminished expectations ameliorated markedly.

Now 10 years since the onset of her neurasthenia, she still has mini-relapses of persistent and vexing exhaustion; when others see her faring better, they make demands on her for emotional or physical support that she can not directly refuse. She has not returned to gainful employment.

CHRONIC FATIGUE SYNDROME

Fatigue is one of the most common symptoms in all of medical practice. The nature of chronic fatigue syndrome, however, remains very controversial. This syndrome has been defined by the Centers for Disease Control (CDC) in 1988 as a disabling disorder with a combination of a certain number of nonspecific symptoms such as fluctuating levels of fatigue, various combinations of neuromuscular and neuropsychological symptoms, chronic pain, malaise, mild fevers, and anxiety. According to the latest CDC criteria, it is a condition that has been clinically evaluated and still remains as an unexplained, persistent, or relapsing chronic fatigue that is of new or definite onset in a previously healthy person; is not the result of exertion; is not substantially alleviated by rest; and results in substantial reduction in previous levels of occupational, educational, social, or personal activities (Table 16–20).

Table 16-20. 1994 CDC Criteria for Chronic Fatigue Syndrome

A. Severe unexplained fatigue for over 6 months that is:

- (1) of a new or definite onset
- (2) not due to continuing exertion
- (3) not resolved by rest
- (4) functionally impairing

B. The presence of four or more of the following new symptoms:

- (1) impaired memory or concentration
- (2) sore throat
- (3) tender lymph nodes

- (4) muscle pain
- (5) pain in several joints
- (6) new pattern of headaches
- (7) unrefreshing sleep
- (8) postexertional malaise lasting more than 24 hours

Since the time of the original CDC definition there have been a few minor revisions in criteria. The debate continues on whether chronic fatigue syndrome is a psychiatric illness or a somatic one with emotional components.

The prevalence of chronic fatigue syndrome is yet to be determined. In one recent primary care clinic study, of 686 patients specifically examined for fatigue, 77 patients were identified as having chronic fatigue and 17 cases met criteria for chronic fatigue syndrome. To date, there has been no community-based epidemiological investigation of this condition, only studies from primary-care practices. Hence there is no data of this condition that is not biased by help-seeking and access to health care.

In the absence of a clear cause, chronic fatigue syndrome has been disparagingly called "the yuppie flu," neurasthenia, and masked depression. Viral or postviral etiologies have also been considered, with diagnoses such as myalgic encephalomyelitis and chronic Epstein-Barr virus disorder being in vogue for a while. Studies of these viral etiologies have not seemed to be relevant to a large segment of those with chronic fatigue syndrome, but recent studies note a persistent enterovirus or herpes virus 6 in some cases. An overlap with fibrositis or fibromyalgia has also been considered, but this is not particularly illuminating because it only links a poorly understood rheumatic condition with another even less clearly defined, fatigue-defined condition of unclear etiology.

Medical anthropologists have seen chronic fatigue syndrome as a vehicle for negotiation of change in interpersonal worlds. A physiologist recently suggested that there might be a nasal fatigue reflex, akin to the atavistic but well-documented diving reflex. According to this hypothesis, the nasal fatigue reflex could produce the debilitating fatigue that would in turn give the afflicted individual the time to heal before having to face a hostile environment. Brain magnetic resonance imaging (MRI) studies of 43 patients with chronic fatigue syndrome, compared to controls, demonstrated that no MRI pattern of white matter abnormalities is specific.

Psychiatric factors have been strongly associated in the etiology of chronic fatigue syndrome. For example, certain clinical samples have reported that almost half of their cases have had antecedent psychiatric disorders such as depression, phobias, or other anxiety disorders. In a recent matched study of 214 subjects with chronic fatigue syndrome from a nonspecialist, nonreferral setting, most of the index subjects were at considerably greater risk of current psychiatric disorder than were control subjects. The likelihood of psychiatric disorder was six times greater in these chronic fatigue syndrome patients than in the matched controls when evaluated either by interview or by questionnaire. Other studies of subjects who have undergone neuropsychological testing indicate that at least a subset of patients with chronic fatigue syndrome experience significant impairments in learning and memory. However, a primary psychiatric etiology for chronic fatigue syndrome is typically stoutly denied by patients with this syndrome, especially by those who are members of the chronic fatigue syndrome national peer support groups.

Chronic fatigue syndrome is most likely to be a heterogenous condition, with fatigue being only a final common pathway. At this phase of research on the condition, it is not possible to speak with certainty about the etiology of this complicated condition. Whether chronic fatigue syndrome should be considered as a special class of mood disorder with somatic symptoms (specifically fatigue), or a somatoform disorder not otherwise specified, or a combination of a psychiatric disorder with an unidentified infectious agent, or even some combination of these conditions, remains to be clarified. Using strict DSM-III-R criteria, a recent study demonstrates that very few chronic fatigue syndrome patients have somatization disorder.

Treatments for a presumably heterogenous condition with an unknown cause typically should involve a multidisciplinary approach involving psychological, physiological, and social factors. Possible concomitant psychiatric disorders could most likely benefit from a psychopharmacological trial. Many other types of treatments are being used for this debilitating illness, with even electric plum blossom needle therapy having its adherents in the literature. Two randomized controlled trials of cognitive-behavioral therapy (compared with relaxation therapy or routine practitioner care) indicate that cognitive behavioral therapy has substantial impact on the disability and symptoms of patients with this disorder.

Miss J. was a 35-year-old single white librarian with a benign medical past and no psychiatric symptoms prior to developing a flu-like illness. After 10 days the acute episode passed, but she continued to feel lethargic and readily fatigued. Two weeks after the onset of this illness, she returned to work but was unable to complete her usual 8-hour days because of increasing exhaustion and a newly developed set of symptoms: gradually evolving, diffuse muscle and joint pain.

Her primary care physician suggested naproxen (Naprosyn) and gave her encouragement while counseling patience. The physician noted nothing unusual about her mood, and prescribed hypnotic agents to improve her sleep. There was no improvement, however, on 10 mg of zolpidem (Ambien). She then started having squeezing bitemporal headaches. After 3 months, she was referred to a rheumatologist who tried to start her on amitriptyline (Elavil) 50 mg at night. The patient protested vehemently, denying that she was depressed, just in pain.

Previously she had been a conscientious employee and had rarely taken leave or missed work because of illness. After 3 months of this illness, however, she was forced to take a leave of absence, returning to live with her mother since she no longer had any income. She continued to "hurt all over," was lethargic and irritable, and slept poorly because of pain. When she slept, she reported that she no longer awoke refreshed.

Six months after the onset of her original symptoms, she self-referred to an academic health center's rheumatology clinic where she presented as an afebrile and otherwise healthy woman who was angry about her protracted illness and her living situation. She admitted to difficulty with concentration. Joint examination revealed full range of motion with no red, hot, or swollen joints; tender points were present at all 18 sites. Her rheumatologist prescribed amitriptyline 25 mg at night for 4 days and then told to increase the dose by one tablet until she achieved better sleep or reached a dosage of 150 mg. Still protesting that she was not depressed, she took the antidepressant medication because she was desperate for relief. A month later she returned to the rheumatology clinic, still hostile and impatient, with little change, and she was then prescribed 20 mg of fluoxetine in the morning in addition to the amitriptyline at night.

Within a month of this regimen, she was somewhat improved in her mood, sleep, and joint symptoms. However, she still continues to have episodes of fatigue, usually related to stressful life events. She had not yet returned to the work force.