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Brain injury and functional disorders part IV. *Robert L. Heilbronner; Michael F. Martelli; Keith Nicholson; Nathan D. Zasler.*

Neurorehabilitation professionals are often asked to provide an opinion about the presence or absence of brain dysfunction after reported or suspected minor head injury (MHI). This is often after other medical practitioners (e.g., neurosurgeons, neurologists) have already rendered an opinion about whether or not any reported cognitive, behavioral, and emotional sequelae are neurologically based. Especially when significant symptoms are reported and persist, professionals often demonstrate predispositions with regard to interpreting symptoms as neurologically based (i.e., due to brain injury), due to motivational factors (e.g., malingering or "compensation neurosis"), or representative of secondary psychological effects (e.g., anxiety, somatic hypervigilance, secondary gain, etc.). Indeed, there are controversies about whether the post-concussion syndrome (PCS) is an organic phenomenon, (1,2,3) and a professional's beliefs clearly influence how a patient's condition is conceptualized and treated.

In fact, neurorehabilitation efforts may depend more on the philosophy of the treating professional than on the patient's symptoms per se. Further, the opinions of health professionals can influence how a person responds to his or her injury. (4) Finally, as the course of time passes from the onset of injury, psychological (versus organic) factors exert a greater impact on the expression of postconcussive symptoms. (5,6,7) This can occur for many reasons, including that the patient might have returned to pre-injury activities (e.g., work) too soon and could be under situations of greater stress or demand or because other stresses, such as the effects of litigation or new onset medical conditions or unrelated life stresses, could exert an influence.

Symptoms occurring earlier post injury appear organic in nature (e.g., headache, dizziness, double vision), while those occurring later appear more psychoemotional (e.g., depression, anxiety, irritability). For some patients, however, symptoms tend to worsen over time and earlier "organic" type symptoms may be reported later. This deviates from the expected pattern of recovery after mild head trauma (8,9,10,11) and raises suspicion that functional or even pathological factors like malingering or "compensation neurosis" may be playing an increasing role.

Not all such patients, however, are feigning or exaggerating difficulties, even if they demonstrate impairments on neuropsychological tests or in psychoemotional functioning that appears disproportionate to head injury severity. In a small number of cases, increased vulnerability to neurologic injury or greater than realized injury severity may be responsible. In the vast majority of cases, however, other factors, such as emotional distress, certain personality vulnerabilities, or environmental demands, can lead to greater functional disability than would be expected based solely on injury severity variables (e.g., length of unconsciousness, post-traumatic amnesia).

Some of the risk factors that have been associated with persistence of PCS symptoms include preexisting psychiatric history, history of drug or substance abuse, previous head injury, female gender, advanced age, and other conditions causing loss of neural and psychosocial coping reserves. (12,13) Importantly, a diagnosis of a formal mental disorder using the DSM-IV (14) is not a necessary precondition for psychological vulnerabilities for poor post injury adjustment.

Individual coping vulnerabilities that leave persons "at risk" for psychoemotional disturbance that are clearly present in an individual's personality dynamics (15,16) may not be sufficient to disrupt pre-injury functioning in the absence of significant stress. The stresses associated with injury and adverse reactions to transient physical or cognitive changes and life disruption, however, may represent demands that decompensate ongoing coping and produce psychiatric sequelae. Symptoms may also arise from stress associated with return to work, self doubt or anxiety about ability to return to pre-injury level of functioning and meeting expected demands, associated fear of rejection, fear of loss of a supportive safety net, anger and resentment, the effects of litigation, or the psychological consequences of the injury itself, and an interaction of these and other factors. (17)

As time post injury increases, a multitude of factors can impact a person's day-to-day functioning. Losses relating to, for example, work, income, cognitive efficiency, family role changes, variable stresses unrelated to injury, etc., can represent additional stresses that further decompensate adjustment and further exacerbate poor psychoemotional adjustment.

One of the potential pitfalls in the neurobehavioral assessment of suspected brain injury is the potential to over diagnose brain injury based solely on a compatible set of patient complaints. Clearly, accurate clinical assessment requires familiarity with the expected recovery patterns following mild traumatic brain injury (MTBI) (18,19,20) as well as differential patterns associated with exaggeration and malingering. (21,22,23,24,25,26,27)

When there is a marked discrepancy between the person's claimed distress or disability and the objective findings, when there is a medicolegal context, and when there are suspicions about motivation, the possibility of exaggeration response bias and malingering should be closely scrutinized. Conversely, the possibility of under diagnosing brain injury and over diagnosing malingering also exists. Clearly, there is no uniformity of responses following MTBI, so a predisposed professional could easily over interpret natural variability as inconsistency or interpret psychological reactions to impairments as causal.

The types of deficits seen with post concussive cognitive sequelae are not specific to brain injury and can result from multiple other factors, singularly and in combination. These include medication effects, depression and other psychiatric states, chronic pain, sleep disturbance, developmental learning disabilities, medical illnesses such as hypertension, diabetes, COPD, and sleep apnea, and others. (e.g., 28) Clinicians must be familiar with psychiatric or other syndromes that may present as organic brain injury. Of course, the presence of a psychiatric syndrome or response bias does not necessarily exclude the diagnosis of another organic syndrome. This certainly complicates the process of disentangling multiple clinical entities that sometimes co-exist. Only methodical assessment can differentiate sequelae secondary to brain injury from the multiple other factors, which can produce similar symptomatology.

DIFFERENTIAL DIAGNOSIS

Traditionally, the most common differential diagnosis neurorehabilitation professionals are asked to address in cases of mild head trauma is the determination of whether or not symptoms are neurologically

or functionally based. With an increasing number of personal injury cases, the differential also concerns whether symptoms are "real" or manufactured. This distinction is really not dichotomous or mutually exclusive. [See part II and especially part III in the "Masquerades" series for a review of response bias.] (29,30)

Further, there are other psychiatric diagnoses that must be considered in assessing functional disability. Unfortunately, there are no simple guidelines for reliably distinguishing neurologic sequelae following brain injury from other possible diagnostic entities. Disentangling the effects of preexisting conditions on cognition functioning can be difficult, and examiners must rely on other sources of pre-injury information (e.g., medical records, education, and employment history, etc.) to assist in differential diagnosis.

Accurate differential diagnosis requires familiarity with the major functional medical disorders. In this paper, we address issues of differential diagnosis. Causality and apportionment are important additional considerations that are beyond the scope of this paper but will be elaborated in a future Masquerades of Brain Injury issue. The following syndromes represent the major functional medical disorders. (31) These are syndromes that present as physical disorders with symptomatology that produces functional disability that is primarily mediated by psychological disturbances.

POST-TRAUMATIC STRESS DISORDER

Post-traumatic stress disorder (PTSD) is a psychological reaction to an extremely distressing event, which is usually experienced with intense fear, terror, and helplessness. The most common symptoms of PTSD are recurrent and intrusive recollections of the event, distressing dreams during which the event is re-experienced, deliberate efforts to avoid thoughts or feelings associated with the event, as well as activities or situations that arouse recollections of it. (32) Like those with mild head trauma, patients with PTSD complain of concentration difficulties, forgetfulness, sleep difficulties, irritability, and poor frustration tolerance; they are likely to become depressed, anxious, and exhibit cognitive problems (secondary to emotional and psychological factors).

Until recently, the prevailing opinion has been that cerebral concussion and PTSD could only co-occur in the absence of a loss of recall for the trauma or events surrounding an accident. Recent evidence, however, suggests that patients who sustain MTBI with loss of consciousness and amnesia can also develop phobic-kinds of responses and generalized fears, which can produce disability. Traumatic pain experience, islands of consciousness with partial uncertain recollections or even reconstructed memories, and heightened physiological anxiety that generalizes to injury-related symptoms or stimuli, can all contribute to post-traumatic stress symptomatology. Importantly, because the cognitive and other symptoms associated with PTSD closely overlap with difficulties frequently endorsed by MTBI patients, distinguishing the two are accomplished only through careful and deliberate assessment.

FACTITIOUS DISORDERS

Factitious disorders are characterized by physical and psychological symptoms that are produced by the individual and, like malingering, are under voluntary control. The judgment that the behavior is under voluntary control is based, in part, on the patient's ability to simulate illness in such a way that he or she is not discovered. This is made by excluding all other possible causes of the behavior. Although similar to malingering in some respects, the person with a factitious disorder usually has more obvious or severe character pathology, such as a borderline personality disorder, and the only apparent or primary goal is

to assume the patient role. A history of repeated "accidents" with a compensatory tinge may exist, which may reflect the person's impulsivity and intense anger or passive-aggressive behaviors in certain situations. (33)

The most common type of factitious disorder is chronic factitious disorder with physical symptoms, otherwise known as Munchausen's syndrome. This involves multiple hospitalizations, and often multiple surgeries, for symptoms with no apparent true physical disorder. The complaints of patients are typically dramatic and colored by the patient's knowledge of the disorder and hospital procedures. Although there is a conscious attempt to fabricate symptoms, the underlying motivation and reasons may be outside of the patient's conscious awareness.

Another common factitious disorder is the factitious disorder with psychological symptoms or Ganser syndrome. This is often characterized by the symptom of giving approximate answers or talking past the point, (34) and patients often provide "near misses" of the correct response during mental status testing or neuropsychological evaluation. Patient's who respond to obvious questions with near misses frequently arouse suspicion in examiners and might also become extremely negativistic and uncooperative to further questioning. Although this style of responding and seemingly oppositional behavior might suggest the presence of malingering because less than optimal amount of effort is being put forth, the goals are clearly disparate.

SOMATOFORM DISORDERS

The somatoform disorders involve the presence of physical symptoms that suggest a physical disorder for which there are no demonstrable organic findings or known underlying pathophysiologic mechanism. There is also positive evidence, or at least a strong presumption, that the symptoms are not intentionally produced but are linked to psychological factors or conflicts. (35) Even settlement of a legal case would not likely ameliorate the symptoms if the underlying conflicts were not addressed. (36)

Unlike factitious disorder or malingering, the symptom production in somatoform disorders is not under voluntary control, and the person does not experience the sense of controlling the symptoms. Of the somatoform disorders, somatization disorder and conversion disorder are perhaps the two most often considered in cases of suspected or purported MTBI. The former involves multiple somatic complaints for which no physical cause can be found, and the patient often makes repeated visits to physicians and may have numerous hospitalizations over the course of several years. Typical complaints include pseudoneurologic or conversion-type symptoms, gastrointestinal complaints, psychosexual difficulties, cardiopulmonary problems, chronic pain, and symptoms in the female reproductive system. There cannot, by definition, be any identifiable organic etiology for the symptoms. (37)

The essential feature of a conversion disorder is a loss of, or alteration in, physical functioning that suggests a physical disorder but which instead is an expression of a psychological conflict or need. The most obvious and "classic" conversion symptoms are those that suggest neurological disease (e.g., paralysis, seizures, etc.). Like malingering, there are different kinds of "gains" that a patient with conversion disorder can achieve, but the symptoms are not under voluntary control. In one situation, the person achieves "primary gain" by keeping an internal conflict or need out of awareness, and usually there is a temporal relationship between an environmental event that relates to the psychological conflict or need and the initiation or exacerbation of the symptom. In another situation, the individual might achieve "secondary gain" by avoiding a particular activity that is aversive, or by getting support from the environment that might not otherwise be forthcoming. Some MTBI patients with bona fide symptoms

become incapacitated and require significant others to assume certain tasks for them. In these cases, somatoform disorder and malingering should be ruled out.

Most conversion symptoms develop in response to extreme psychological stress and appear rather suddenly. In contrast to mild head trauma patients who may be overly concerned or distressed by their symptoms, patients with conversion typically demonstrate a relative lack of concern (la belle indifference) over their reported symptoms that are out of keeping with the severity of the impairment. In these cases, it is usually a family member who is acutely aware of changes in the person's overall level of psychological and social functioning. From a diagnostic standpoint, the relative degree of awareness or unawareness of deficits versus the proportionality of related concern a person has might be another way, in combination with a comprehensive assessment that includes a thorough medical exam and neuropsychological evaluation, to distinguish between MTBI and a conversion disorder.

PSYCHOGENIC SEIZURES

Psychogenic seizures are a subcategory of nonepileptic seizures (NES) or "pseudoseizures" characterized by episodic or paroxysmal phenomena that resemble epileptic seizures but do not have the same characteristic changes in underlying brain activity. (38) As with epileptic seizures, patients with NES may demonstrate falling, self-injury, and may even be incontinent; however, NES do not generally include such epileptic sequelae as tongue biting, unprotected falling or incontinence. (39)

NES are frequently misdiagnosed as seizures. Up to 25 to 30 percent of people with nonepileptic seizures may also have epileptic seizures and nonepileptic events may generate from physiologic or psychological causes. The main differential diagnoses for physiologic events that present as NES include autonomic disorders, cardiac events, cerebrovascular disease, drug toxicity, metabolic disorders, migraines, and sleep disorders.

Psychogenic seizures (also known as hysterical epilepsy, conversion fits, pseudo-attacks, and somatoform spell disorder) refer to the subcategory of NES with a psychological etiology. Psychogenic seizures may occur as part and parcel of disorders associated with anxiety, depression, and psychosis, and a history of childhood trauma is common. They occur most commonly in adulthood. Malingering, factitious disorder, and dissociative disorder also must be considered under the differential diagnosis of NES from psychological or functional causes.

The diagnosis of psychogenic seizures is accomplished by first ruling out epilepsy (e.g., observation and clinical symptom correlation, EEG video monitoring, post seizure blood prolactin levels, placebo or suggestion induction; familial epilepsy risk, responsiveness to anti-seizure medication). (40) Secondarily, physiological syndromes (e.g., cataplexy, transient ischemic attacks, syncope) must be ruled out. Finally, psychogenic seizures are diagnosed by analyzing the patient's history. A number of signs suggesting psychogenic rather than epileptic episodes, including frequent episodes unaffected by anticonvulsants, coexistence of psychological symptoms or associated psychiatric disease or vulnerabilities (e.g., anxiety, depression, inappropriate affect or lack of concern, somatization or hysterical personality traits, childhood abuse or trauma, a history of poor adjustment or under achievement, abnormal interaction with significant others, and the presence of emotional triggers). Behavioral techniques, which have been found somewhat successful in ameliorating epileptic seizures are considered the primary treatment of choice for psychogenic seizures. (41,42)

The diagnosis of psychogenic seizures is ultimately probabilistic and fallible. Indeed, accurate diagnosis should allow consideration of the following cautions: (43)

* Epilepsy suggestive EEG can occur in asymptomatic patients.

* At least some patients labeled as having pseudoseizures are eventually diagnosed with epilepsy or as having structural brain lesions using more sensitive recording procedures (e.g., depth recordings, MRI) or other physiologic conditions.

* Real and pseudoseizures commonly co-exist in patients.

* Nonepileptic seizures and psychogenic seizures are too often used synonymously and are confounded in the literature.

* Differentiation of nonepileptic seizures is much more difficult for partial seizures versus tonic or clonic seizures.

FUNCTIONAL AMNESIA

There is often some disturbance of mnemonic function with mild or other traumatic brain injury and with other disorders involving structural brain lesions. In the case of MTBI, memory problems are usually discrete and limited, resolving fully within weeks to months. Although there may be some complications (e.g., poor memory associated with premorbid ability structure, interfering effects associated with headache or other pain, affective distress, sleep disturbance or other (44) assessment of memory problems following mild traumatic brain injury is generally straightforward.

There are sometimes, however, dramatic memory problems that may represent a functional amnesia. The most common and striking of these are perhaps the cases of profound retrograde amnesia in which there is complete or near complete loss for explicit recall of personal, autobiographical information from prior to the trauma usually with preservation of anterograde memory or capacity for new learning, and preservation of semantic memory, implicit memory, and well learned skills. (45,46,47)

Functional retrograde amnesia, sometimes termed psychogenic amnesia, may be seen subsequent to a variety of traumatic experiences. It is widely accepted that psychological trauma can produce such a presentation. Whereas these memory problems do not involve structural brain lesions, abnormal brain activity has been demonstrated with functional imaging. (48,49) This indicates, as would be expected with any psychological act, that there is an underlying neurobiological substrate manifesting in what has been termed an "mnestic block syndrome." (50,51)

These disorders, however, are reversible and resolve spontaneously with appropriate psychotherapeutic interventions, or under sodium amytal interviews, although they sometimes persist for lengthy periods. As with diagnosing other disorders, dissimulation must be ruled out. Further, the possibility of an iatrogenic effect of professional suggestion on the part of medical and legal professionals should be considered.

CONCLUSIONS

Neurobehavioral assessment in cases of mild head trauma represents a burgeoning area of growth for neurorehabilitation professionals, and our understanding of the many factors involved in symptom production and persistence has grown dramatically over the past decade. Whereas post-traumatic symptoms were once believed to be either exclusively "organic" or "neurotic," a more complex understanding of the multiple factors determining functional disability and outcome has emerged. (52) As a result, clinicians in hospital practice and in the private sector must become more familiar with the different medical and psychiatric diagnoses that are commonly involved in the differential diagnosis of MTBI. Legal professionals, too, must be aware of the different diagnoses that mimic or look very much like MTBI so that they can judge the merits and liabilities of their cases.

Whereas identification of neurobehavioral impairments in the acute stage after mild head trauma may be relatively simple, assessment of persisting symptoms after a few weeks or months is a more complex enterprise. The longer after the accident or injury that post-traumatic symptoms persist, the greater the likelihood that secondary, psychological factors play a major role. (53,54) In addition, the clinician must determine if the individual is intentionally producing the symptoms or not. If it is determined that the patient is consciously producing their symptoms, then the next decision is to assess whether or not there is an obvious goal that the person is trying to achieve. In clinical practice, these distinctions are not always that easy to make.

Patients presenting with significant functional disabilities after seemingly mild injuries represent complex assessment challenges for physiatrists, neuropsychologists, and other rehabilitation professionals. At a minimum, clinicians must have an understanding of the pathophysiology and neurobehavioral sequelae associated with MTBI. They also must have a familiarity with other more "traditional" psychiatric disorders to assist in differential diagnosis. Data from neuropsychological testing, in combination with other objective and subjective psychological data (e.g., a thorough history, clinical interview, review of school records, reports of collaterals, etc.) and information from other medical disciplines, promises the greatest method for differentiating between premorbid factors and post morbid residua secondary to an accident or injury.

Many cases of mild head trauma are not simple or clear-cut, but consideration of some of the other functional disorders mentioned in this article may lead to a greater understanding of some of the complexities involved in differential diagnosis and provide a better foundation for rendering opinions about the causes, needed treatment, and eventual prognosis of symptoms following an accident that purportedly involves MTBI.

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Masquerades of Brain Injury Part V: Pre-Injury Factors Affecting Disability Following Traumatic Brain Injury

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TABLE OF CONTENTS

Traumatic brain injury (TBI) constitutes a major health and societal problem in the United States. Traumatic brain injuries occur in a tri-modal distribution with the highest incidence in children (*i.e.*, younger than five years old), young adults (*i.e.*, 16 to 34 years), and adults (*i.e.*, 65 years and older). TBI rates are highest for males age 15 to 24 years and for both sexes after the age of 70.[1] In general, adult men represent two-thirds of the brain injuries sustained between ages 15 to 70.

It is well-known that recovery from brain injury is a long-term process and that there is considerable patient variability in long-term outcome. TBI outcomes range from subtle changes in personality to profound physical, cognitive, and psychosocial disability. It is also widely accepted that neurobehavioral, cognitive, and adjustment difficulties, rather than physical impairments, represent the most disabling long-term effects of TBI.[2]

OUTCOME FOLLOWING TRAUMATIC BRAIN INJURY

Given the large individual variability, outcome following moderate to severe brain injury is difficult to predict. Persons with TBI represent a diverse group, and, to some extent, the variability in outcome is a function of pre-injury differences in personality, social roles, and intellect. Pre-injury status may be particularly important when evaluating long-term outcome and adaptation following brain injury.[3]

Diversity in patient outcome also arises from post-injury differences in pathophysiology and associated sequelae.[4] Variables such as length of coma, duration of post-traumatic amnesia, the presence of seizures, and type of brain injury are well documented and known to influence outcome from traumatic brain injury.[5,6,7,8] Furthermore, perceived outcome is often subjective. What one patient and family may consider "positive" may be viewed as an insurmountable loss by another individual and his or her family.

In limited studies, it has been found that persons with a pre-injury history of substance abuse, psychiatric disorder, low intelligence, and poor occupational adjustment tend to have more complicated recoveries than similarly injured patients without such histories.[9] Researchers, however, have generally paid less attention to premorbid factors that might influence outcome in comparison with trauma related variables (*e.g.*, duration of coma) or other post-injury biomedical factors (*e.g.*, radiological indicators of brain injury). This is due, at least in part, to the difficulty in obtaining reliable information concerning the nature of premorbid factors and the vast array of characteristics and events from which to choose.[10]

It is intuitively appealing to consider that when a traumatically brain injured person is confronted with the long-term outcome of TBI, premorbid psychological coping characteristics become engaged. The impact of the interaction between premorbid and post-injury personality variables and long-term outcome has not yet been determined, but clinicians involved in the rehabilitation of persons with TBI are frequently impressed that personality variables, both pre- and post-injury, contribute greatly to long-term outcome.[11] Given the variability in patient recovery, some empirical support, and a widely accepted but only partially investigated view that premorbid intellectual, personality, and sociocultural influences interact with acquired brain injury to produce a complex symptom picture,[12] further investigation is clearly warranted.

STRESS AND COPING MODELS OF ADAPTATION

Increasingly sophisticated models of behavior are emerging in the fields of medicine and psychology that assist with conceptualizing and designing treatment interventions for challenging healthcare situations. Biopsychosocial models represent alternative theoretical approaches to dualistic and reductionistic biomedical models that explain disease and health primarily in terms of measurable biological variables. A stress, coping, and vulnerability formulation of brain injury postulates that the injury results in multiple cognitive, emotional, social, and neurophysical

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demands which constitute, singularly and in combination, severe stressors which not only challenge the coping capabilities of the individual but directly diminish available resources through loss of premorbid skills and a combination of reductions in social and financial supports.[13] This formulation includes a complex interaction of factors surrounding brain injury, the history the individual brings to the injury, and the environment that the individual confronts afterwards.

In 1992, Kay[14] proposed a concept of individual vulnerability that suggests that a large number of variables, ranging from biological to psychosocial, influence the impact and outcome of a brain injury for any given individual. Individual differences in brain structure, hormonal and neurotransmitter balances, and other biologic systems represent pre-injury differences that may render one brain more susceptible to, or magnify, neurologic impairment. Subsequently, a wide variety of personality and psychosocial variables interact with the particular impairments to produce a unique functional outcome.

Importantly, case study review revealed several pre-injury personality styles that resulted in vulnerability to poorer relative outcome following mild TBI (MTBI). For example, highly driven, perfectionistic, overachiever types seem specifically prone to elevated stress levels and complicated recoveries due to catastrophic reactions to reductions in cognitive performance. Grandiosity and narcissistic features, as well as borderline personality disorder features, also complicated recoveries, as did prior traumatic experiences, or preexisting somatoform or dependency features. Ruff, Mueller, and Jurica[15] report on additional case study data supporting these and a few other "vulnerable" personality styles. The major "vulnerable" personality styles are included in Exhibit 1.

Satz,[16] after reviewing the clinical literature, proposed a "threshold factor" to account for instances of protection from, or vulnerability to, clinical symptoms when the central nervous system is diseased. The concept of "brain reserve capacity," as espoused by Satz, offers a useful explanation of threshold differences in the onset of clinical symptoms or the expression of impairments and disabili-

Exhibit 1: Vulnerable Personality Styles

Style	Premorbid traits	Postmorbid reactions
Overachiever	Sense of self derived from driven accomplishments, which is frequently accompanied by obsessive compulsive traits	Catastrophic reaction if drop in performance is perceived
Dependent	Excessive need to be taken care of, frequently leading to submissive behaviors and a fear of separation	Paralyzed by symptoms if critical erosion of independence occurs
Borderline personality traits	Pattern of instability in interpersonal relationships and self-image with fear of rejection or abandonment	Exacerbation of personality disorganization, including despair, panic, impulsivity, instability, and self-destructive acts
General insecurity	Weak sense of self, which can include shame, guilt, and dependency needs	Magnification of symptoms
Grandiosity	Overestimation of abilities and inflating accomplishments, can include need for admiration and lack of empathy	Minimization or denial of symptoms; if failure results, crash of self-esteem can result in catastrophic reaction
Antisocial traits	Tendency to be manipulative or deceitful, temperamental, impulsive, and irresponsible; lacks sensitivity to others	Possible exaggeration or malingering, increased risk taking, irritability, takes little responsibility for recovery
Hyperactivity	Restless, unfocused, and sometimes disorganized	Attentional difficulties and impulsivity may be compounded; possible oppositional behavior
Depressed	Mood fluctuations dominated by negative affect	Increase of depressive symptoms, despondency
Histrionic style	Emotionality and attention seeking behavior	Dramatic flavor to symptom presentation; blaming behavior
Somatically focused	Preoccupation with physical well being, reluctance to accept psychological conflicts	Endorsement of multiple premorbid physical symptoms intermixed with new or changing post-morbid residua
Post traumatic stress disorder	Prior stressors produced an emotional reaction of fear and helplessness	Decreased coping ability, cumulative effect of traumas with exaggerated reaction to current crisis

Reference: Ruff, RM, Mueller, J and Jurica, P. (1996). Estimation of Premorbid Functioning after traumatic brain injury. NeuroRehabilitation, 7, 39-53.

ties after brain injury. Psychosocial factors, intelligence, and educational level represent indirect, albeit imprecise, measures of this construct.

When an individual sustains a brain injury, a dramatic imbalance in psychological, biological, and environmental function occurs. The observed diversity in individual outcome certainly reflects the influence of pre-injury differences in neurologic status, personality, and coping skills, intellect, and social resources and roles interacting with post-injury differences in pathophysiology and associated sequelae and changing environmental demands.[17,18,19,20]

Neurologic disease occurs within a multiaxial matrix of a person's physiologic, psychologic, and social history and environment to produce a complex presentation in which diversity in outcome is expected. In conceptualizing adaptation and outcome from a "demands versus resources" model, Satz argues that an individual's unique history is critical in determining what vulnerabilities and resources he or she brings to the injury, as these resources act as "deposits" in a resource "bank" that enhance future adaptation.[21]

When confronted with managing the long term impact of brain injury, an individual's premorbid personality and coping resources, including premorbid intellectual, personality, and sociocultural factors, interact with the sequelae of acquired brain injury to produce a complex symptom picture.[22] This conceptualization is consistent with the stress and coping literature,[23] which conceptualizes coping as an individual's cognitive and behavioral efforts to master demands and conflicts, including the sequelae of neurologic disease, and where an individual's traditional mastery of coping is expected to influence his or her responses to injury.

Despite criticisms about the lack of studies investigating the influence of pre-injury characteristics on brain injury outcome,[24,25] a few studies have incorporated such recommendations. Two empirical investigations to date have sought to elucidate a theoretical model derived from the cerebral reserve/individual vulnerability and stress and coping literature.[26,27] This model posits that individuals possess adaptational reserve for meeting neurologic and other demands and accordingly, greater degrees of reserve would be associated with higher levels of resilience and improved adaptation and recovery from neurologic trauma, while individuals with limited or previously depleted adaptational reserve would be expected to demonstrate increased vulnerability and poorer response.

Martelli and colleagues[28] specifically investigated a vulnerability, stress, and coping model of adjustment following TBI. They argued that the multiple cognitive, emotional, physical, and social sequelae of TBI constitute, singularly and in combination, severe stressors that both challenge coping capabilities and directly diminish available coping resources through loss of abilities, independence, self-esteem and identity, financial, and social supports. Recognizing that recognized prognostic variables sometimes exert relatively weak or variable influences, they developed a composite vulnerability to disability scale including premorbid psychiatric (presence of symptoms of mental illness), neuropsychological (estimated premorbid IQ), attitudinal (victimization perception), relationship (marital status), as well as injury related (*i.e.*, length of post traumatic amnesia, presence of post traumatic seizures) variables.

Using this scale, they combined vulnerability factors and differentiated patients based on a clinically derived cutoff score into high and low vulnerability groups. Group membership (*i.e.*, high versus low vulnerability) was highly accurate in discriminating post-traumatic vocational and disability status. In terms of vocational status, only one of the eight subjects in the high vulnerability group was employed or in school, while 14 of the 20 low vulnerability subjects were employed or in school. With regard to disability status, six of the eight high vulnerability subjects were receiving disability compensation versus five of the 20 low vulnerability subjects. These results are presented in Exhibit 2 and Exhibit 3.

Macmillan *et al*[29] conducted an investigation focusing on a select subgroup of vulnerability variables identified by Martelli *et al*.[30] Investigating premorbid psychiatric and substance abuse history, Macmillan *et al*[31] found that the presence of pre-injury psychiatric and substance abuse problems were both very highly associated with lowered employment and independent living status. Dividing subjects into high and low vulnerability groups (*i.e.*, simple addition of rating scores for premorbid psychiatric and substance abuse histories), they found that only two of the 20 persons (10 percent) in the high vulnerability group were working, and this was in a part time capacity.

In contrast, 19 of 23 of the low vulnerability subjects were employed. In addition, 90 percent of persons with no sig-

Exhibit 2: Employment Status for High and Low Vulnerability Groups

	Current Work Status By Vulnerability Group		
Current Work Status	**High Risk**	**Low Risk**	***Total***
Not Working/No School	*7*	6	*13*
Part-Time Work/School	1	6	*7*
Full-Time Work/School	0	8	*8*
Total	*8*	*20*	

Exhibit 3: Disability Status for High and Low Vulnerability Groups

	Current Disability Status By Vulnerability Group		
Current Disability Status	**High Risk**	**Low Risk**	***Total***
No Disability	2	14	*16*
Receiving Disability	6	6	*12*
Total	*8*	*20*	

nificant premorbid substance abuse were living independently, while only a small minority (*i.e.*, 23 percent, or 3 of 13) of those with premorbid substance abuse were living independently. Interestingly, none of the vulnerability variables were associated with patient's assessment of their own functional status. These results are included in Exhibit 4 and Exhibit 5.

In another study on the influence of a preexisting vulnerability on subsequent brain injury, Raskin[32] investigated whether repeated sexual abuse and subsequent mild traumatic brain injury (MTBI) led to poorer neuropsychological testing results. Based on hypothesized neuropathological changes secondary to the stress associated with sexual abuse, results indicated that individuals with a history of both sexual abuse and MTBI had a greater number of deficits than normal controls or those with either condition alone.

Further evidence of the influence of psychiatric factors on test performance comes from Klonoff and Lamb[33] who cited the neuropsychological test performance of nine individuals with MTBI in which the presence of psychiatric disturbance and/or malingering better explained the poor neuropsychological performance over the trauma. Similarly, Greiffenstein and Baker,[34] in a selected sample prospective study, reported that pre-existing somatoform problems may predict symptomatology in late postconcussion claimants. Finally, Youngjohn *et al*[35] highlighted the need to consider psychiatric disturbances when conducting forensic neuropsychological evaluations.

Taken together, these studies provide support for the assertion that preexisting stress/vulnerability factors can have an appreciable impact on both neuropsychological and daily living functioning subsequent to TBI.

CONCLUSION

There are an estimated two million traumatic brain injuries each year in the United States. Psychosocial and neurobehavioral disorders, rather than physical impairments, are the most disabling consequences. Variability in outcome following TBI more often is the rule rather than the exception, and this phenomenon has not been well understood. Overwhelmingly, outcome studies have focused on the effect of post-injury variables and generally employed gross measures of physical and cognitive status versus quality of life and adaptation to disability.

Many studies have excluded persons with psychiatric and substance abuse histories. There is increasing appreciation, however, that pre-injury characteristics such as coping history may influence outcome and contribute to individual variability in terms of vulnerability to persistent disability. This vulnerability undoubtedly reflects a complex combination of both premorbid and post-injury variables.

Exhibit 4: Employment Status for High and Low Combined (Premorbid Psychiatric, Substance Abuse) Vulnerability Groups

	Current Work Status by Vulnerability Group		
Current Work Status	**High Risk**	**Low Risk**	***Total***
Not Working/No School	0	11	*11*
Part-Time Work/School	2	8	*10*
Full-Time Work/School	20	4	*24*
Total	*22*	*23*	

Exhibit 5: Living Status for High and Low Combined (Premorbid Psychiatric, Substance Abuse) Vulnerability Groups

	Current Living Status by Vulnerability Group		
Current Living Status	**High Risk**	**Low Risk**	***Total***
Independent	0	11	*11*
Home-Supv/Attendant	2	8	*10*
Assisted Living/Nursing Home	20	4	*24*
Total	*22*	*23*	

From the studies presented, it is clear that premorbid vulnerability factors such as psychiatric status, substance abuse, and sexual abuse histories can exert a very strong influence on neuropsychological functioning and ability to cope with and adapt to demands and challenges associated with TBI. Perhaps more importantly, convergent findings fit nicely with the growing trend toward biopsychosocial conceptual models of adaptation following TBI. Incorporating specific concepts of individual vulnerability and cerebral reserve identified through a multifactorial, biopsychosocial interactive model has strong implications for assessing impact of injury and illness on adaptation and disability.

These conceptualizations offer the promise of more useful assessment procedures and treatment interventions. Increasingly, researchers and clinicians are recognizing the importance of evaluating the collective influence and interaction of psychological, social, and cultural factors with biological factors in explaining disease and its variable expression in terms of healthcare outcomes. Such an effort offers a strategy for better understanding the multiple factors mediating the functional expression following impairment that produces more or less disability, and, ultimately, for enhancing adaptation following brain injury.

An increased sensitivity to the role of vulnerability, stress, and coping factors is ultimately required for any efforts to optimize functional capabilities and improve design of interventive strategies in this patient population. Evaluating the influence of premorbid and post-injury variables should result in better predictions with increased understanding of the risk factors associated with TBI and the design of more appropriate intervention programs. The

importance of understanding the variables that mediate the relationship between impairments, disability, and handicap cannot be underestimated, and continued investigations of vulnerability and stress and coping formulations are clearly indicated.

Importantly, a biopsychosocial model, as described, represents a more integrated and through understanding of the effects of brain injury. Such a model reveals more complex pathways for explaining causality and apportionment than ones describing simpler trauma dose response relationships. For the medicolegal setting, these formulations represent challenging models that require innovate approaches by both expert witnesses and attorneys. The implications of a complex biopsychosocial model of injury, impairment, and disability will be reviewed in future articles that specifically address causality and apportionment issues.

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ABC of psychological medicine

Chronic multiple functional somatic symptoms

Christopher Bass, Stephanie May

The previous article in this series described the assessment and management of patients with functional somatic symptoms. Most such patients make no more than normal demands on doctors and can be helped with the approach outlined. However, a minority have more complex needs and require additional management strategies. These patients typically have a longstanding pattern of presenting with various functional symptoms, have had multiple referrals for investigation of these, and are regarded by their doctors as difficult to help.

Terminology

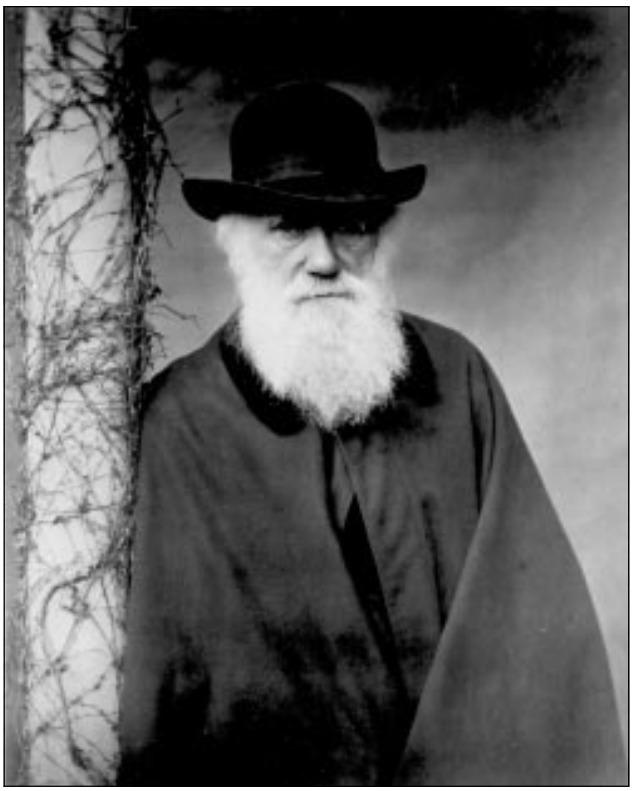
Because such patients may evoke despair, anger, and frustration in doctors, they may be referred to as "heartsink patients," "difficult patients," "fat folder patients," and "chronic complainers." The use of these terms is inadvisable. If patients read such descriptions in their medical notes they are likely to be offended and lose faith in their doctor and may make a complaint. In psychiatric diagnostic classifications these patients are often referred to as having somatisation disorder. We prefer the term "chronic multiple functional symptoms" (CMFS).

Epidemiology and detection

Over 4% of the general population and 9% of patients admitted to tertiary care have CMFS. Each primary care doctor will have on average 10-15 such patients.

Most patients with CMFS are women. They often have recurrent depressive disorder and a longstanding difficulty with personal relationships and may misuse substances. There is an association with an emotionally deprived childhood and childhood physical and sexual abuse. Some patients will clearly have general disturbances of personality.

The risk of iatrogenic harm from over-investigation and over-prescribing for somatic complaints makes it important that patients with CMFS are positively identified and their management planned, usually in primary care. Potential CMFS patients may be identified simply by the thickness of their paper notes, from records of attendance and hospital referral, and by observation of medical, nursing, or clerical staff.



Charles Darwin (1809-82) suffered from chronic anxiety and varied physical symptoms that began shortly after his voyage in the *Beagle* to South America (1831-6). Despite many suggested medical explanations, these symptoms, which disabled him for the rest of his life and largely confined him to his home, remain medically unexplained

Management in primary care

Assessment

It is helpful if one doctor is identified as a patient's principal carer. Once a patient is identified as possibly having CMFS a systematic assessment is desirable. The case notes should be reviewed and the patient seen for one or more extended consultations.

Case notes—Patients with CMFS often have extensive case notes. Unless these are reviewed, much potentially useful information may remain hidden. It is also helpful to compile a summary of these records and to evaluate critically the accuracy of any previously listed complaints and diagnoses. The summary should include key investigations performed to date and any information about patients' personal and family circumstances.

"Fat files" are a simple indicator of a high level of contact with medical services, which may indicate multiple chronic functional somatic complaints

Long appointment—During one or more long appointment a patient's current problems and history should be fully explored. Patients should be encouraged to talk not only about their symptoms but also about their concerns, emotional state, and social situation and the association of these with their symptoms. At the end of the assessment, patient and doctor should agree a current problem list, which can then be recorded in the notes.

Management

The initial long interviews serve not only to derive a problem list but also to foster a positive relationship between doctor and patient. Thereafter, the doctor should arrange to see the patient at regular, though not necessarily frequent, fixed intervals. These consultations should not be contingent on the patient developing new symptoms. Consultation outside these times should be discouraged.

Planned review

All symptoms reported by patients during these consultations must be acknowledged as valid. A detailed review of symptoms enhances the doctor-patient relationship and minimises the likelihood of missing new disease.

Reassurance that "nothing is wrong" may be unhelpful, possibly because a patient's aim may be to develop an understanding relationship with the doctor rather than relief of symptoms. Focused physical examination can be helpful, but there is a risk of patients receiving multiple diagnostic tests and referrals to specialists, and these should be minimised. Patients also often accumulate unnecessary prescribed drugs, and if so these should be reduced gradually over time.

If a satisfactory rapport can be established with a patient, new information about his or her emotional state, relationship difficulties, or childhood abuse may be revealed. In such cases the doctor may need to offer the patient a further long appointment to reassess the need for specialist psychological care.

Support for doctors

General practitioners managing patients with CMFS should arrange ongoing support for themselves, perhaps from a partner or another member of the primary care team with whom they can discuss their patients. A doctor and, for example, a practice nurse can jointly manage some of these patients if there is an agreed management plan and clear communication.

Referral to psychiatric services

Not all doctors will consider that they have the necessary skills or time to manage these patients effectively. Review by an appropriate specialist can then be helpful. Unfortunately, the decline in the number of "general physicians" and specialist mental health services' increasing focus on psychotic illness mean there are few appropriate specialists to refer to.

If referral is sought two questions must be considered: "Are there any local and appropriate psychiatric services?" and "How can I prepare the patient for this referral?" If available, liaison psychiatry services are often the most appropriate and experienced in this area of practice. To prepare the patient, a discussion emphasising the distressing nature of chronic illness and the expertise of the services in this area, together with a promise of continuing support from the primary care team, can help to make the referral seem less rejecting. If possible, the psychiatrist should visit the practice or medical department and conduct a joint consultation.

Assessment of chronic multiple functional somatic symptoms

- Elicit a history of the current complaints, paying special attention to recent life events
- Find out what the patient has been told by other doctors (as well as friends, relatives, and alternative practitioners). Does this accord with the medical findings?
- Elicit an illness history that addresses previous experience of physical symptoms and contact with medical services (such as illness as a child, illness of parents and its impact on childhood development, operations, time off school and sickness absence)
- Explore psychological and interpersonal factors in patient's development (such as quality of parental care, early abusive experiences, psychiatric history)
- Interview a partner or reliable informant (this may take place, consent permitting, in the patient's presence)
- After the interview attempt a provisional formulation

Useful interviewing skills for doctors managing patients with multiple physical complaints

- Adopt a flexible interviewing style—"I wonder if you've thought of it like this?"
- Try to remind the patient that physical and emotional symptoms often coexist—"I'm struck by the fact that, in addition to the fatigue, you've also been feeling very low and cannot sleep"
- Try "reframing" the physical complaints to indicate important temporal relationship between emergence of patient's somatic and emotional symptoms and relevant life events
- Respond appropriately to "emotional" cues such as anger
- Explore patient's illness beliefs and worst fears—"What is your worst fear about this pain?"

Management strategy for patients with chronic multiple functional somatic symptoms

- Try to be proactive rather than reactive—Arrange to see patients at regular, fixed intervals, rather than allowing them to dictate timing and frequency of visits
- During appointments, aim to broaden the agenda with patients—This involves establishing a problem list and allowing patients to discuss relevant psychosocial problems
- Stop or reduce unnecessary drugs
- Try to minimise patients' contacts with other specialists or practitioners—This will reduce iatrogenic harm and make containment easier if only one or two practitioners are involved
- Try to co-opt a relative as a therapeutic ally to implement your management goals
- Reduce your expectation of cure and instead aim for containment and damage limitation
- Encourage patients (and yourself) to think in terms of coping and not curing

Explanations to the patient

Present patient's problems as a summary with an invitation to comment:

"So let me see if I've understood you properly: you have had a lot of pain in your abdomen, with bloating and distension for the past four years. You have been attending the (GP) surgery most weeks because you've been very worried about cancer (and about your husband leaving you). You also told me that these pains often occur when you are anxious and panicky, and at these times other physical complaints such as trembling and nausea occur.

"I'm struck by the fact that all these complaints began soon after you had a very frightening experience in hospital, when your appendix was removed and you felt that 'No one was listening to my complaints or pain.'

"Have I got that right, or is there anything I've left out?"

Summary of a 15 year "segment" of the life of a patient with chronic multiple functional somatic symptoms

Date (age)	Symptoms (life events)	Referral	Investigations	Outcome
1970 (18)	Abdominal pain	GP to surgical outpatients	Appendicectomy	Normal
1973 (21)	Pregnant (boyfriend in prison)	GP to obstetrics and gynaecology outpatients	Termination of pregnancy	—
1975-7 (23-25)	Bloating, abdominal pain, blackouts (stressful divorce)	GP to gastroenterology and neurology outpatients	All tests normal	Diagnosis of irritable bowel syndrome and unexplained syncope. Treated with Fybogel
1979 (27)	Pelvic pain (wants to be sterilised)	GP to obstetrics and gynaecology outpatients	Sterilised, ovaries preserved	Pelvic pain persists for 2 years after surgery
1981 (29)	Fatigue (problems at work)	GP to infectious disease clinic	Nothing abnormal detected	Diagnosis of myalgic encephalomyelitis made by patient. Joins self help group
1983 (31)	Aching, painful muscles	GP to rheumatology clinic	Mild cervical spondylosis. No treatment	Treated with Tryptizol 50 mg on referral to pain clinic. Improves
1985 (34)	Chest pain and breathlessness (son truanting from school)	Accident and emergency to chest clinic	Nothing abnormal detected, probable hyperventilation	Refer to psychiatric services

Specialist assessment

Before interviewing a patient, it is useful to request both the general practice and hospital notes and summarise the medical history. A typed summary of the "illness history" can be kept as a permanent record in the notes. This summary can guide future management and is especially useful when a patient is admitted subsequently as an emergency or when the receiving doctor has no prior knowledge of the patient.

Several important interviewing skills should be used during the assessment. These skills can be learnt using structured role playing and video feedback. They form the basis of a technique called reattribution, which has been developed to help the management of patients with functional somatic symptoms.

Specialist management

If a patient can understand and agree an initial shared formulation of the problems, an important first stage is reached. From this a plan of management can be negotiated. It is best to adopt a collaborative approach rather than a didactic or paternalistic manner. If it is difficult to arrive at an understanding of why the patient developed these symptoms at this particular time, then an alternative approach may have to be adopted. In essence this involves the doctor attempting to address those factors that are maintaining the symptoms.

Assessment and management go hand in hand. One of the main aims of management is to modify patients' often unrealistic expectations of the medical profession and to remind them of the limits to medicine. In many cases hopes may have been falsely raised, and patients expect either a cure or at least a considerable improvement in symptoms. Although this is desirable, it may not be attainable. Instead, the doctor should attempt to broaden the agenda, with an emphasis on helping patients to address personal concerns and life problems as well as somatic complaints. It is also necessary to encourage them to concentrate on coping rather than seeking a cure.

This process requires patience, and a capacity to tolerate frustration and setbacks. It may require several discussions in which the same issues are reviewed. In the long term, however, it can be rewarding for both patient and doctor.

Common problems in management

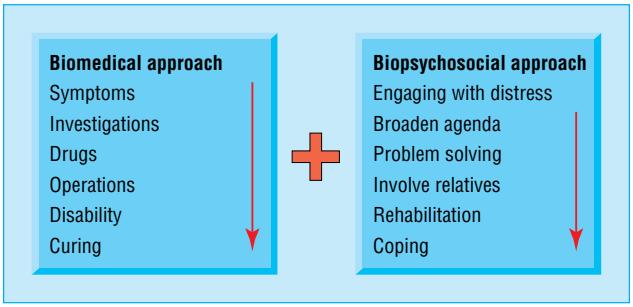
Management may be complicated by various factors. Firstly, preoccupation and anxious concern about symptoms may lead patients to make unhelpful demands of their doctor, which prove difficult to resist.

What is the cause of functional somatic symptoms?

- A variety of biological, psychological, and social factors have been shown to be associated with functional symptoms; the contribution of these factors will vary between patients
  Recent developments in neuroscience show altered functioning of the nervous system associated with functional symptoms, making the labelling of these as "entirely psychological" increasing inappropriate
- With our current knowledge, it is best to maintain "aetiological neutrality" about the cause of functional symptoms
- The main task of treatment is to identify those factors that may be maintaining a patient's symptoms and disability

Maintaining factors that should be focus of treatment in patients with multiple somatic symptoms

- Depression, anxiety, or panic disorder
- Chronic marital or family discord
- Dependent or avoidant personality traits
- Occupational stress
- Abnormal illness beliefs
- Iatrogenic factors
- Pending medicolegal claim



The aim of treatment for patients with chronic multiple functional symptoms is to add a biopsychosocial perspective to the existing biomedical approach

Secondly, there may be evidence of longstanding interpersonal difficulties, as indicated by remarks such as "Nobody cares" or "It's disgusting what doctors can do to you." Such comments may suggest that the patient's relationship with the doctor may reflect poor quality parental care or emotional deprivation in childhood. They are important for two reasons: firstly, the doctor may take these remarks personally, become demoralised or angry, and retaliate, which will destroy the doctor-patient relationship; and, secondly, the attitudes revealed may require more detailed psychological exploration.

Finally, iatrogenic factors may intervene that are beyond the treating doctor's control. Because these patients have often visited several specialists, conventional and alternative, they may have been given inappropriate information and advice, inappropriate treatment, or, in some cases, frank misdiagnosis.

Factitious disorders and malingering

Factitious disorders

Factitious disorders are characterised by feigned physical or psychological symptoms and signs presented with the aim of receiving medical care. They are therefore different from functional symptoms. The judgment that a symptom is produced intentionally requires direct evidence and exclusion of other causes. Most patients with factitious disorders are women with stable social networks, and more than half of these work in medically related occupations. Once factitious disorder is diagnosed, it is important to confront the patient but remain supportive. When factitious disorder is established in a person working in health care it is advisable to organise a multidisciplinary meeting involving the patient's general practitioner, a physician and surgeon, a psychiatrist, and a medicolegal representative.

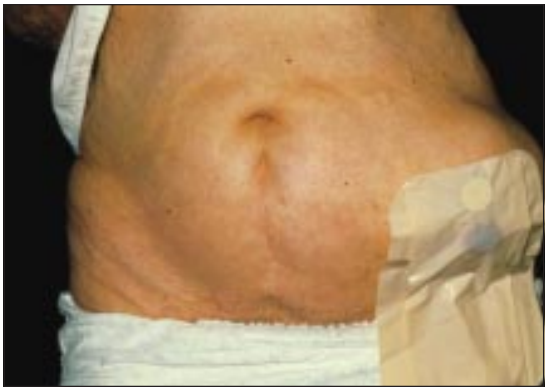
If, and only if, the deliberate feigning of symptoms and signs can be established (such as by observation of self mutilation) should patients be confronted. It is helpful if both a psychiatrist and the referring doctor (who should have met to discuss the aims, content, and possible outcomes of the meeting beforehand) can carry out the confrontation jointly. This "supportive confrontation" is done by gently but firmly telling the patient that you are aware of the role of their behaviour in the illness whilst at the same time offering psychological care to help with this. After confrontation, patients usually stop the behaviour or leave the clinic. Only sometimes do they engage in the psychiatric care offered.

Malingering

A distinction should be made between factitious disorders and malingering. Malingerers deliberately feign symptoms to achieve a goal (such as to avoid imprisonment or gain money). Malingering is behaviour and not a diagnosis. The extent to which a doctor feels it necessary to confront this issue will depend on the individual circumstances.

Conclusion

Patients with multiple longstanding functional symptoms are relatively uncommon, but their interaction with the health system is memorable in that it often leaves both them and their doctors frustrated. Their effective management requires that special attention be paid to their interpersonal difficulties (including those arising in their relationship with the doctor), the limiting of unhelpful demands, and the avoidance of iatrogenic harm. As with any chronic illness, confident management and getting to know a patient as a person can change what is often a frustrating task into a rewarding one.



Failing to recognise and institute appropriate management for patients with multiple functional somatic symptoms may lead to iatrogenic harm from excessive and inappropriate medical and surgical intervention

Münchausen's syndrome

- Münchausen's syndrome is an uncommon subtype of factitious illness in which the patient, who is often a man with sociopathic traits and an itinerant lifestyle, has a long career of attending multiple hospitals with factitious symptoms and signs
- Management is as for factitious disorder, but engagement with psychiatric treatment is rare

Evidence based summary

- Prevalence of chronic multiple functional somatic symptoms depends on how many functional symptoms are required—the fewer symptoms the higher the prevalence
- Patients with chronic multiple functional somatic symptoms (somatisation disorder) can be effectively managed in primary care, with resulting cost savings

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Case Reports

Comorbid Factitious and Conversion Disorders

LEWIS M. COHEN, M.D., F.A.P.M.
KENNETH CHANG

Oh what a tangled web we weave,
When first we practice to deceive!
—Sir Walter Scott (1771–1832), Marmion, canto VI, stanza 17

The presence of deception is the key to distinguishing between factitious and conversion disorders.[1,2] The former condition is marked by active dissimulation that permits the individual to assume the role of the patient; the latter is prompted by unconscious conflicts and symptoms that are not intentionally produced. Coexistence of the two disorders in the same patient highlights the fluid nature of prevarication. Furthermore, since patients with either diagnosis are frequent targets for intense countertransference and are consequently the objects of scorn, irritation, and humor, the following case underscores the role of therapeutic confrontation.[3,4]

Case Report

Ms. A was a 36-year-old woman who was hospitalized in the oncology service and was then rapidly recognized as having a factitious disorder with physical symptoms. Her chief complaint was acute leg weakness, which she reported had manifest during a course of chemotherapy for breast cancer. She explained to the admitting physician, Dr. Cox (all names are pseudonyms), that she was receiving treatment from a local oncologist, Dr. Seth Levine, who was on vacation. The results of a physical examination were inconsistent with this history, as Ms. A appeared to be in excellent health. No neuromuscular abnormality was detected that could explain the paresis, there was no scar from a biopsy or lumpectomy, and she had a full head of hair that had recently been shaved (thus differing in appearance from hair after chemotherapy). The following day, Dr. Cox was able to reach a Dr. Steven Levine, who had a hematology-oncology practice in the area, and he confirmed that he had never treated Ms. A. Dr. Cox directly confronted Ms. A, who continued to insist that she had breast cancer.

Two years previously, Ms. A was determined to have a conversion disorder of pseudoseizures. She had also been depressed for several years and was hospitalized in a psychiatric facility on one occasion for suicidal ideation. She had graduated from college and briefly worked in a medical clinic, but she was now unemployed and living with her parents.

When the psychiatric consultant arrived at the oncology nursing station, Ms. A's factitious disorder was the topic of boisterous conversation. Upon entering her room, he was immediately struck by her closely cropped head. After introducing himself and the reason for the consultation, he explained that there was some "confusion" about her outpatient physician and cancer treatment. Throughout the interview, Ms. A steadfastly maintained that she had breast cancer, was receiving chemotherapy, and was being followed up by Dr. Levine. After asking her permission, the psychiatric consultant picked up the bedside phone and called directory assistance for the office number of Dr. Seth Levine. While looking at Ms. A, he explained that there was no such listing. He then asked her which pharmacy was providing her medications and again sought the number from the operator. The pharmacist quickly obliged by listing her medicines, and the psychiatrist explained to Ms. A that none of these were chemotherapeutic agents. Both Ms. A and the psychiatrist agreed that this was a mystery.

The consultant suggested the following:

I understand from your physician that you are going to be discharged shortly from this unit and will be returning home with your father. Let me suggest that you look for the telephone number of the doctor that you say is treating the cancer, give him a call, tell him what has transpired, and have him contact me so that I can clarify matters. If,

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however, you can't find this physician, then you need to give some serious thought to your having an emotional or psychological condition, rather than a physical one, and I would like you to call me so we can discuss getting some help.

Three days later, Ms. A called and stated, "I don't think I have Munchausen's [this term was never mentioned by the consultant] because I knew what I was doing, but I can't get it under control and need some treatment." She went on to say, "I am lying outright to everybody." Ms. A could not explain her leg weakness, which had started and ended spontaneously.

Asked to recount the circumstances of her situation, she described how, 6 months previously, her mother had suddenly become symptomatic from congenital heart disease and her father had required surgery for carcinoma of the prostate. She panicked "because they needed my help, and up until then, it was always the other way around." During a conversation with a close friend, "It slipped out . . . and soon everyone knew I had breast cancer."

The consultant discussed referral to a local mental health clinic and offered to contact Ms. A again in a month to find out how she was doing. During the subsequent follow-up phone contact, Ms. A reported that she had attended an intake appointment and was beginning therapy. She stated, "I don't have the words to express my appreciation for what you did for me." Asked to explain, she said, "They simply did not believe me in the hospital. You gave me a chance to go home and figure it out." Pushed to clarify the extent to which she knew she was fabricating, Ms. A vacillated between acknowledging her lies and appearing to be surprised at the truth. She concluded, "I couldn't see past this huge spider web that I was creating. . . . You gave me the option to figure things out. . . . My parents and friends are now aware of the deception."

At her 5-month follow-up, Ms. A was seeing a therapist on a weekly basis and reported feeling much better. When asked about the time leading up to her hospitalization, she stated, "It was so confusing; it was easier to believe that I had breast cancer."

Discussion

Breast cancer is a rare form of factitious disorder.[5–9] Patients appear with self-induced dermatosis, aggravation, ulceration, or infection of the breast. At least one prophylactic mastectomy has been performed for a woman who falsely claimed to have a family history that was strongly positive for the disease.[10] The crescendo of media attention accorded breast cancer may be increasing the prevalence of this type of factitious disorder.[11] Management involves preventing unnecessary medical/surgical interventions and ensuring that appropriate psychiatric follow-up is arranged. Unfortunately, it is all too common for such patients to peregrinate from physician to physician and never receive psychiatric treatment.

The present case involves a woman with factitious breast cancer and a comorbid conversion disorder. Ms. A conceded that she had prevaricated regarding her breast cancer but maintained that her presenting symptom of leg weakness was genuine. That she had a propensity for conversion disorders is not surprising, given a past history of pseudoseizures. This case is interesting because of the combination of factitious behavior, conversion signs, and inconsistent insight into her deception. Ms. A stated that there were times when she was convinced that she really did have a malignancy. It is unclear, even to her, how much of the disease was contrived and how much of it she really believed.

We would postulate that parental illnesses drove Ms. A to assume a sick role and deceive friends, family, caretakers, and ultimately herself. The effort and conflict required to maintain the facade of breast cancer led to the eruption of the conversion disorder. Explaining the leg weakness as a side effect of chemotherapy required her to take other steps to complete the picture of chemotherapeutic treatment. To this end, she cycled back into factitious behavior, shaved her head with a razor, and was hospitalized.

It is understandable that sometimes the line between reality and fantasy can blur. Polage[12] has described a fascinating experiment with normal subjects who were instructed to tell a lie to an experimenter. She reported that after lying to the researcher for an extended period of time, up to 10% of the subjects subsequently became convinced of the truth of the tale and denied that they had been lying. This phenomenon is also evident in law enforcement and is manifest in the reopening of the Central Park jogger case, in which five defendants were convicted after falsely confessing to the crime.[13,14] The videotaped confessions are compelling in the detail and seeming truthfulness of the accounts, but they were refuted by DNA and other evidence. In patients prone to suggestibility (i.e., those with a history of conversion), it is reasonable to assume that memories will be altered by repeated false statements and further reinforced by family and friends who provide a social

influence that helps perpetuate the false belief. This is an especially problematic area in dissociative disorders.[15]

Spence and associates[16] have made use of positron emission tomography to study a small group of patients with conversion disorders involving motoric function of their upper extremities. They compared the patients with normal individuals who were requested to feign limb weakness, as well as a control group. The conclusions are preliminary because of the small group size and the absence of patients with factitious disorders. Nevertheless, it is interesting that all of the patients with conversion disorder who attempted to move their affected limbs were found to have left prefrontal hypofunction, while right prefrontal hypofunction characterized the group that feigned having a disorder ($p<0.001$). The authors hypothesized that hysterical pathophysiology specifically involves dysfunction of the left dorsolateral prefrontal cortex, and this region is activated in the feigners who chose to slow down and limit their movements.

In conversion disorders, direct confrontation is generally accepted as being an ineffective approach, as it directly challenges the subjective experience of the patient. More supportive, insight-oriented, cognitive, and behavioral techniques are recommended that focus on understanding the symptoms as part of a biopsychosocial system.[17,18] Behavioral reinforcement has been particularly useful in dealing with conversion in children, in whom insight-oriented therapy is less effective.[19] All patients should be offered encouragement that symptoms will remit.

The literature is divided as to the management of factitious disorders. Most authors recommend confrontation by the primary physician after discovery of irrefutable evidence of duplicitous behavior.[20] In one study, only 13 of 33 confronted patients acknowledged self-inducing symptoms, but almost all reported an improvement in the physician-patient relationship.[21] Other experts have suggested that a nonconfrontational approach improves patient compliance.[22,23] The goal is not to invalidate the patients' symptoms but rather to build rapport and make psychiatric follow-up acceptable. This would be particularly applicable to situations where it is not clear how much is believed and how much is contrived.

In our patient's case, the attending physician and the psychiatrist enacted a variation of "good cop, bad cop." The patient was confronted in the traditional manner by the internist, while the consultant used a gentler and more empowering approach. Since patients rarely accept referrals immediately, the consultant provided the patient with a task and asked her to consider seeking psychiatric help if that task could not be accomplished. This allowed the patient the time to come to terms with her denial and provided her with the responsibility for arranging care. This combination of interventions proved to be effective in helping Ms. A recognize the nature of her symptoms and to seek outpatient care.

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Traumatic brain injury when symptoms don't add up: conversion and malingering in the rehabilitation setting. (Traumatic Brain Injury).

Author/s: Patricia Rogers Babin

There are occasions in rehabilitation when a person presents with symptoms that are inconsistent, exaggerated, do not fit with any known medical diagnosis, or are frankly unbelievable, leading the rehabilitation specialist to label the person as a malingerer. Often, however, people demonstrate symptoms of a psychological disorder that is not malingering - the rehabilitation specialist may actually be observing an unconscious psychological need to be "ill" in some way, i.e., a disorder that is conversion or conversion-like. In the literature, there are few articles discussing the presence of conversion in the rehabilitation setting (Speed, 1996; Teasell & Shapiro, 1994), and there is a dearth of literature that explores the difficulty of differentiating between conversion (and conversion-like disorders) and malingering.

This paper will focus on the special case of traumatic brain injury (TBI). The authors have found those persons with mild, moderate, and even severe TBIs occasionally present with symptoms that are in excess of or inconsistent with what would be expected for their diagnosis. In the case of mild traumatic brain injury, most people experience symptom resolution within one to three months (Dikmen, McLean, & Temkin, 1986; Gentilini, Nichelli, & Schoenhuber, 1989; Levin, Eisenberg, & Benton, 1989). However, 10% to 15% of people with mild TBIs continue to report persistent deficits and impairment after this three month time period (Alexander, 1995). Understandably, total symptom resolution is not expected in persons with moderate to severe TBIs. However, cognitive and functional gains are generally made in a more or less linear fashion over the course of months and years, barring significant medical problems.

There have been several reasons conjectured to account for the ongoing and at times excessive complaints of persons with TBI. Psychiatric problems are often thought to contribute to symptom exaggeration (Lishman, 1988). Just as often, persons with TBI are misjudged and accused of malingering. It is necessary to explore the conversion versus malingering differential as patients reporting

"unbelievable" symptoms are often incorrectly perceived as malingering and not given the treatment they need. This paper serves to outline conversion, malingering, and related disorders, describe assessment tools used to make a differential diagnosis, delineate two pertinent case studies, and discuss treatment options for persons with TBI who are demonstrating and reporting symptom exaggeration.

Conversion

Conversion disorder falls under a class of disorders known as the somatoform disorders. The common feature of all somatoform disorders "is the presence of physical symptoms that suggest a general medical condition ... and are not fully explained by a general medical condition, by the direct effects of a substance, or by another mental disorder" (Diagnostic and Statistical Manual of Mental Disorders, 4th Edition (DSM-IV), 1994). Conversion disorder is the unconscious expression of what is thought to be psychological conflict through physical symptoms. The DSM-IV (1994) delineates diagnostic criteria for conversion disorder. Namely, the symptoms must cause significant distress or impairment in social, occupational, or other areas of functioning, the deficits presented must be motor or sensory, and the symptoms are not due to pain, sexual, or mental disorders (e.g., schizophrenia). In addition, medical diagnoses do not fully account for the symptoms, psychological conflicts and stressors precede the symptoms, and the symptoms are not intentionally produced.

The presence of a psychological conflict or stressor is a fine, but important, point. The patient may not fully acknowledge the significance of such a conflict or stressor or may deny that the conflict or stressor has affected him or her. Regardless, the role of the conflict/stressor is critical. The symptoms that arise from the conflict/stressor are symbolically related to unconscious drives. Symptoms are believed to allow partial expression of a forbidden wish or unacceptable need, disguising the wish so that the patient need not consciously confront it. For example, a single mother of three children under five years of age resented that her large extended family was not helping her more. She always felt that her family was not supportive, causing her to act independent and overly self-reliant throughout her childhood. She developed amnesia for her entire childhood and severe new learning deficits after a minor vehicle accident in which there was no loss of consciousness and no positive medical findings. The amnesia necessitated that her family intervene and subsequently she obtained the help she needed without having to acknowledge and express her anger and unmet dependency needs.

Epidemiology and Etiology

Prevalence rates of conversion disorder have ranged from as low as 11/100,000 to as high as 300/100,000 in the general population (DSM-IV, 1994; Ford & Folks, 1985). In medical centers, incidence rates have ranged from 5% to 20% (Zeigler, 1970). A number of predisposing factors may make people susceptible to developing a conversion disorder, including being female, adolescent psychopathology, pre-existing psychopathology, and history of serious medical illness (Barsky, 1989; DSM-IV, 1994).

A number of theories purport to explain the onset of conversion symptoms. One theory, based on a psychodynamic conceptualization of the symptoms, posits that the person derives primary gain by keeping the internal conflict out of conscious awareness. On the other hand, learning theory suggests that a person derives secondary gain by avoiding an activity or activities that are stressful or noxious and by gaining emotional or social support that may not otherwise be available.

Diagnosis

Conversion disorder is very difficult to diagnose. The rehabilitation specialist must invest energy, time, and financial resources to rule out a medical disorder prior to making a diagnosis of conversion. As discussed above, conversion disorders are diagnosed if a medical diagnosis does not fully account for the symptoms. Ergo, the patient may have an underlying, very real medical disorder and still demonstrate conversion symptoms. Diagnosing a psychiatric disorder, such as conversion, is complicated by the fallibility of medicine and diagnostic tests. One study found that of patients given the diagnosis of conversion, in 60% of those cases an "organic" cause for the symptoms was eventually found (Gould, Miller, Goldberg, & Benson, 1986).

Differentiating medical diagnoses from psychiatric diagnoses is further complicated because psychological factors are often associated with the onset of many medical disorders, e.g., depressive and anxiety symptoms. Also, secondary gain or external incentives are common with many medical disorders. For example, it is not uncommon for someone with a stroke or cerebral vascular accident to demonstrate depressive symptoms or histrionic features under the stress of the physical, functional, and psychosocial changes or losses that occur. Furthermore, symptoms associated with a TBI may include irritability, fatigue, and deficits in attention, memory, and executive functioning. Someone experiencing these problems after TBI may not be able to follow through on work or social obligations, and may need to rely on others for emotional, financial, and functional support--seemingly deriving secondary gain. While in this situation secondary gain appears obvious, in reality the reliance on others is a natural and perhaps appropriate result of losing one's independence.

Making the differential diagnosis between malingering and somatization type disorders, such as conversion, can be difficult. Conversion symptoms can be inconsistent and unbelievable, very similar to malingering. It is commonly believed that patients who are in pursuit of compensation frequently report symptoms with longer duration (Mittenberg, Diguilio, Perrin, & Bass, 1992). The authors of the current paper contend that duration of reported symptoms does not necessarily imply the secondary gain of compensation associated with malingering. It is possible that patients may employ an unconscious psychological process that also involves secondary gain, but of the nature that meets or modifies a psychological need, i.e., conversion or conversion-like disorder.

Finally, the critical difference between conversion disorder and malingering is intent. That is, is the person consciously and volitionally

producing symptoms? Unfortunately, even the most precise tests cannot measure or determine whether someone is consciously or unconsciously motivated to report symptoms. In the case of TBI, expectancies for TBI symptoms commonly occur in normal populations, i.e., people who have not experienced a TBI can accurately describe common sequelae (Alves, Macciocchi, & Barth, 1993). Because TBI symptoms are commonly known and predictable, patients can relatively easily report symptoms. Patients who are at risk demographically for conversion disorder are more susceptible to developing conversion symptoms if they have some knowledge of a medical disorder. Similarly, patients who are intentionally feigning symptoms, as in the case of malingering, can more easily do so if the disorder has predictable symptoms.

Related Disorders

There are a number of DSM-IV diagnoses that must be considered in the differential between conversion and malingering. As noted above, conversion disorder is a subtype of the somatoform disorders, several of which can be confused with conversion disorder, including somatization disorder, undifferentiated somatoform disorder, pain disorder, hypochondriasis, and somatoform disorder not otherwise specified. A differential diagnosis must also be made between a somatoform disorder such as conversion, and a DSM-IV category of conditions known as Psychological Factors Affecting Medical Condition. Lastly, one must also consider Factitious Disorder in the differential. A full explanation of all related disorders is beyond the scope of this paper. See the DSM-IV for complete diagnostic criteria of related disorders.

Malingering

Malingering is the intentional production of medical or psychiatric symptoms to obtain an external incentive (DSM-IV, 1994). In the psychiatric literature, the incentive is known as secondary gain, and may consist of monetary or other gain, or avoidance of negative consequences. Because malingering is not a psychiatric or medical diagnosis, it was assigned a nondiagnostic DSM-IV V-code. Intent to defraud is difficult to prove to a certainty, so that many clinicians avoid labeling a person as a malingerer (Binder, 1992).

Malingering is often associated with litigation or Worker's Compensation claims. There are numerous articles published on the incidence of malingering or likely malingering in persons with TBI. Many studies have found that persons in litigation or pursuing Worker's Compensation claims performed with less consistency on neuropsychological tests (Reitan & Wolfson, 1995; Reitan & Wolfson, 1996), performed with neuropsychological test score patterns that do not occur in nonlitigating persons with TBI (Reitan & Wolfson, 1992), or performed with questionable motivation (Fox, 1994; Schmand, Lindeboom, Schagen, Heijt, Koene, & Hamburger, 1998; Youngjohn, Burrows, & Erdal, 1995). In contrast, Ruff, Wylie, and Tennant (2000) found no differences between litigants and nonlitigants with TBI on neuropsychological test performance. Suhr, Tranel, Wefel, and Barrash (1997) argue that factors other than pending litigation contribute to poor or inconsistent performance. In addition, there is

no evidence that TBI patients, as a group, intentionally feign or exaggerate deficits on neuropsychological testing more often than other diagnostic groups (Leininger & Kreutzer, 1992).

Epidemiology and Etiology

Because there are disincentives for the malingerer to reveal feigning a disability, the incidence is difficult to determine. Estimates range from 1% to 50%, depending on the setting and population (Grant & Alves, 1987; Resnick, 1988; Schretlen, 1988). However, most patients try to appear psychologically normal and to minimize their cognitive deficits (Pankratz, 1988), including those seeking compensation (Lezak, 1995). Some experts say the incidence of malingering is much less common than expected given the amount of attention focused on it in the literature (White & Proctor, 1992). Nevertheless, a Rand Corporation study found 35% to 42% of the medical costs claimed in motor vehicle accidents in 1993 involved staged or nonexistent accidents, or inflated claims (Carroll, Abrahamse, & Vaiana, 1995).

Antisocial traits, antisocial personality disorder, and substance abuse are associated with deception and can increase the chances of malingering (Miller, 1989, 1990). As a result, angry and challenging affect may be seen. The reported circumstances surrounding the precipitating accident may be vague or odd. Falls may be unwitnessed, or the patient may demonstrate intact abilities after the injury, which he subsequently "loses." Malingerers typically do not cooperate well during examination and treatment. Excessively detailed complaints, bizarre or unusual complaints, or claimed stress out of proportion to the precipitating agent are common. Responses may be excessively slow, as the malingerer tries to determine what response best suits each question. For example, a patient with severe documented orthopedic trauma tried to claim a brain injury as well. However, a basic question such as, "What color is the sky?" elicited the improbable answer, "Greenish-blue?" after a lengthy pause.

Diagnosis

There are a number of assessment tools used to explore psychological factors that contribute to symptom presentation. When the patient with a TBI presents with a complicated and confusing symptom picture, the authors suggest the following assessment strategy: clinical interview, collateral interview with significant other, interviews with staff working with patient (e.g., physical, occupational, and speech therapists), review of medical records, neuropsychological testing, MMPI-2, and appropriate tests of malingering.

The first step to assessing malingering is for the examiner to establish the severity of the initial injury using standard medical procedures and measures. For example, the Glasgow Coma Scale (GCS, Teasdale & Jennett, 1974), a measure of coma severity, is expected to be 13 to 15 in cases of mild TBI, 8 to 12 in cases of moderate TBI, and below 7 in cases of severe TBI. Loss of consciousness (LOC) must be less than 20 minutes in mild TBI (Rimel, Giordiani, Barth, 1981). LOC in a concussion may be present or absent without affecting symptom outcome. Mild TBI subjects complaining of symptoms one to 24 months post-injury who had brief LOC did not differ

neuropsychologically from those without LOC (Leininger, Gramling, Farrell, Kreutzer, & Peck, 1990).

An accident history incompatible with the patient's report should be noted. However, some apparent discrepancies may be explained. For example, a person with a recorded GCS of 14 and ability to follow commands in the emergency room who claimed two days of coma may not be dissimulating if he or she is referring to post-traumatic amnesia (PTA). PTA estimates the amount of time after injury before day-to-day memory function can be documented (Rosenthal & Griffith, 1985). Retrograde amnesia (RA) refers to the period of memory loss preceding the TBI. Periods of PTA and RA spanning from weeks to years occur with severe TBI, but not with mild TBI. With complicated mild TBI in which there are CT scan findings, usually of a subdural hematoma or intracerebral contusion, results similar to moderate TBI can be expected (Williams, Levin, & Eisenberg, 1990).

Neuropsychologists cannot accurately identify malingering using neuropsychological tests alone (Faust, Hart, Guilmette, & Arkes, 1988), except perhaps in obvious cases (Trueblood & Binder, 1997). Having said that, some test patterns are suggestive of dissimulating. Obvious malingering may be seen when feigning subjects believe a test is more difficult (Slick, Hopp, Strauss, & Spellacy, 1996). Frequently, better performance on more difficult tests and poor performance on much easier tests may be interpreted as an attempt at malingering while also attempting to preserve ego. A case in point is that old over-learned information is preserved in most persons with TBI. One patient with an unwitnessed fall from a truck claimed he did not know what the numbers one or two were, presumably because his memory was impaired. He continued to deny knowledge of the numbers one and two even after adding them to obtain three. Of course, such widespread memory loss does not occur with traumatic brain injury patients who have intact attention, as this patient did.

In addition to exaggerated or inconsistent findings, possible malingerers tend to over-endorse symptoms. They may report having every symptom about which they are questioned. More intelligent or psychopathic individuals may have researched appropriate responses. However, they will lack the subtle findings associated with the disorder. For example, intrusion and repetition errors are common on list-learning tasks with mild TBI, a sign of diminished self-monitoring. Malingerers may have no more of these errors than non-brain injured individuals.

Many attempts were made to adapt neuropsychological measures to detect malingering during the course of a standard battery of tests. Malingering indices were developed for simple reaction time (Strauss, Spellacy, Hunter, & Berry, 1994), Digit Span (Iverson & Franzen, 1994), and for the Paced Auditory Serial Addition Test (Strauss et al., 1994). Common memory tests were adapted to assess malingering, such as the Wechsler Memory Scale-Revised (Bernard, Houston, & Natoli, 1993; Mittenberg, Azrin, Millsaps, & Heilbronner, 1993), the Recognition Memory Test (Iverson & Franzen, 1994; Millis, 1992, 1994), and the Rey Auditory Verbal Learning Test (Bernard, 1991; Bernard et al., 1993; Binder, Villanueva, Howieson, & Moore, 1993).

To date, none of these adaptations is either thoroughly cross validated or widely used.

Psychology Tests Used to Detect Malingering

The MMPI and its successor, the MMPI-2, are the most common tests administered to detect malingering. The Psychopathic Deviate Scale (Scale 4), a measure of antisocial traits, may be elevated in some malingering individuals. However, it frequently is not. The Infrequency scale (F), Back Infrequency scale (Fb), Infrequency-Psychopathology scale, and the Dissimulation scale-2 (D-sub(s2)), are scales that are typically analyzed in persons presenting with exaggerated complaints. Although these scales can differentiate between personal injury litigants and controls, the scales were not able to differentiate litigants and clinical, nonlitigants. The Fake Bad Scale (FBS) was able to differentiate between litigants and clinical, non-litigants (Tsushima & Tsushima, 2001). Regarding MMPI/MMPI-2 clinical scales, Boone and Lu (1999) found that the 1-3/3-1 (Hypochondriasis and Depression) code types showed evidence of non-credible cognitive performance on malingering and neuropsychological tests. Some sophisticated feigners of malingering produce valid MMPI-2 profiles, while the most unsophisticated feigners produce suspicious profiles (Slick, et al., 1996). One neuropsychological test expert, Lezak (1995) contends that no "Malingering Profile" exists on the MMPI or MMPI-2.

The Structured Interview of Reported Symptoms (SIRS; Rogers, 1986, 1992) was developed for psychiatric patients. Its primary scales include compendiums of rare, improbable and absurd symptoms, as well as blatant versus subtle symptoms. The supplementary scales include a direct appraisal of honesty as well as subscales for defensive symptoms, overly specified symptoms, and symptom inconsistency, which can be helpful in interviewing a suspected malingerer.

Case Study: Suspected Malingering with Ultimate Conversion Diagnosis

History of injury: The first case study involves a 39-year-old man (H.B.) who sustained a moderate TBI in a motor vehicle accident. H.B. was a passenger traveling to his job, when the car he was in was hit head-on. H.B. lost consciousness at the scene. CT scan on admission and on follow-up three days post injury revealed a large amount of intraventricular hemorrhage. Upon admission to acute rehabilitation almost two weeks after his accident, he was confused, restless, distractible, and logorrheic. He was also mildly anomic, but speech was generally fluent. At discharge from inpatient treatment two weeks later, he was oriented in all spheres and had mild to moderate deficits in memory. His thoughts were generally organized and he could independently complete all simple activities of daily living. He was discharged to his home with his girlfriend and began treatment in an outpatient brain injury program.

Social and Mental Health History: H.B.'s medical history was unremarkable. With regard to mental health history, he reported a depressive episode after the death of his mother. He self-medicated with alcohol, but never received treatment for depression or alcohol

abuse. Prior to the accident, he was a moderate social drinker. H.B.'s work and relationship history was somewhat complex. He had numerous careers and jobs throughout his adulthood. At one point he studied ballet, but most of his jobs were unskilled or semi-skilled. He attended college, but did not complete any course of study. He had a history of close, and sometimes chaotic social relationships. Most recently, he lived with his girlfriend of eight months. Apparently, they became seriously involved after two weeks of dating. During his outpatient treatment, the girlfriend revealed she was dissatisfied in the relationship prior to the accident. He had a relatively unremarkable legal history, but admitted to nonpayment of taxes for five years. This was a source of conflict in his relationship with the girlfriend. H.B. also noted that he was intent on suing the driver of the car that caused the accident.

Clinical Picture: Outpatient treatment in a comprehensive Brain Injury Program consisted of individual speech, occupational, and psychotherapy, as well as group cognitive therapy, adjustment group, and community re-entry group. The outpatient staff initially saw H.B. make good progress. After a few weeks in outpatient therapy, his girlfriend complained that H.B. was too dependent on her. She was encouraged to set limits with him as he was capable of completing many if not all complex activities of daily living. Soon though she left the home, and then finally the relationship. During the dissolution of the relationship, H.B.'s performance on cognitive tests and tasks declined. In fact, as time went on his deficits became more "severe". He also developed slurred speech, with prominent tongue protrusion. Medical workup did not reveal physiological or medical explanations for the change in his performance. He was prescribed Effexor by his physiatrist for self-reported depressive symptoms. Interpersonally, he cooperated in group but clearly demonstrated passive-aggressive, narcissistic, and histrionic personality traits. A few members on staff strongly suspected malingering, i.e., that H.B. was intentionally producing cognitive deficits in order to gain attention and support. Despite the staff's conviction, there were clear predisposing factors, which could have contributed to development of a conversion disorder; i.e., an unintentional or unconscious production of symptoms. These factors included Axis I and possible Axis II psychopathology, major life stressors, knowledge of TBI and related deficits. However, in the conversion versus malingering argument, H.B. also demonstrated what are thought to be typical malingering behaviors: inconsistent performance, engaging in a lawsuit, and avoiding work and other responsibilities. H.B. was administered the MMPI-2 (see Figure 1) to begin assessing the psychological and emotional factors which could have been affecting his rehabilitation progress. A neuropsychological test battery was not administered due to the patient's severely and profoundly impaired performance on all cognitive tests administered during speech therapy. It was clear that neuropsychological testing would have also shown severe deficits in all areas of cognitive functioning.

[FIGURE 1 OMITTED]

Analysis: Analysis of the MMPI-2 administered did not reveal any over or under-reporting of concerns. He was not necessarily trying to put himself in an overly positive light. Validity indicators suggested a valid

profile. His code-type, 1-3-8, was interpreted with his TBI deficits in mind. Typically, 1-3-8 profiles are viewed as schizophrenic. In this case, the elevation on scale 8 can be explained in part by deficits reported by many patients with TBI. The 1-3 elevation suggests classic conversion symptoms.

Treatment: The staff was encouraged to begin suggesting to H.B. that he should improve over time, regardless of his complaints that he was severely impaired. As difficult as it was, the staff was encouraged to remain supportive and nonreactive to his apparent symptom exaggeration. To address his progressive speech problem he was put on a behavioral program. To reduce the likelihood that H.B. would interpret the intervention as "psychological", the speech therapist introduced the plan to him. To "correct" the tongue protrusion he was instructed to speak with clenched teeth for five days. Reminders were given. He was given a pseudo-scientific explanation for the intervention and told that for his particular disorder, five days of teeth clenching was the therapy and cure. It was suggested to him that if his speech did not improve, there must be some "other" nonmedical explanation for his speech problem. In addition, if he slipped back into maladaptive speech production, he was instructed by staff and his peers to clench his teeth. Within two weeks, tongue protrusion decreased and H.B.'s speech production improved dramatically.

To address the psychological and emotional issues contributing to H.B.'s decline in functioning, he continued to participate in both individual and group psychotherapy. Psychotherapy addressed his unmet dependency needs and focused on his strengths and abilities. Group psychotherapy focused on helping develop a more flexible interpersonal style, i.e., less demanding of and more appropriately assertive with others. Group psychotherapy also served to provide healthy models of recovery and coping for H.B. After four more months of treatment, he was discharged able to function independently, with less focus on his deficits, and adequate speech production. Tongue thrusting was virtually eliminated. A follow-up MMPI-2 was administered three months after the initial administration (see Figure 2).

[FIGURE 2 OMITTED]

A neuropsychological test battery was also administered to assess the patient's improved functioning. Full Scale IQ was 100 with Verbal and Performance IQ scores at 107 and 91. He demonstrated mild inconsistencies with some evidence of limited effort on easier tests, and more effort on difficult ones. For example, Digit Span was average, but Arithmetic was superior. Despite some inconsistencies, H.B.'s overall performance was consistent with moderate TBI, his acute care hospital diagnosis. Specifically, he demonstrated deficits in speed of processing, memory retrieval, planning, and organization.

Analysis: Although H.B. demonstrates some of the hallmarks of malingering, i.e., exaggerated deficits, inconsistent performance, history of psychological difficulty, recent severe psychosocial stress, secondary gain, the change in H.B.'s MMPI-2 profile clearly suggests that he improved. Improvement alone without resolution of legal or

relationship issues strongly suggests that H.B.'s symptoms were not intentional or conscious, but instead were likely unconscious attempts to meet dependency needs, which surfaced under extreme distress. Validity indicators on the follow-up MMPI-2 were again within normal parameters: H.B. did not attempt to over-report or under-report symptoms. Analysis of his profile indicates an overall lowering of all clinical scales. H.B.'s new code-type was 3-8-5, suggesting mild to moderate histrionic traits with ongoing cognitive and sensory disturbances. Results of neuropsychological testing appeared to be a relatively accurate reflection of his cognitive strengths and weaknesses.

Case Study: Suspected Malingering

History of injury: J.R. was a right-handed 35-year-old man with a high school education. He had an unwitnessed fall on flat ground while working as a construction site plumber. He was found face down, got up, walked a few steps, and reportedly fainted. He regained consciousness with paramedics present. At the emergency room, a CT scan of the brain was negative, and he was diagnosed with a contusion and muscle strain. His wife picked him up three hours after the incident. Later that night, he became dazed and bumped into the walls. His wife took him back to the ER where another CT scan was negative. His wife noted childish behavior, bad temper, and cognitive "fuzziness" for a month following the accident.

Clinical Interview: J.R. was angry, irritable, and challenging throughout the interview and testing. He complained of balance problems, headache, blurred vision, and memory problems. He claimed to lack memory of the injury itself, for the week before the accident and for one month following. Inconsistent history reporting was evident. For example, he did not recall any details of his birthday the week before the fall. Yet, he remembered slipping on items another worker left on the ground just prior to the fall. He was noncompliant with medications, claiming Alprazolam (Xanax) caused memory problems and Amitryptilline (Elavil) 25 mg made him sleep from 11 A.M. to 2 P.M. the following day. When asked why he had a bandage on a forefinger, he claimed the finger was broken "with the bone coming through the skin," but that he had not consulted a physician. Other physical complaints were equally bizarre or over-elaborated. He held his head claiming severe headache, refusing to perform serial 7 subtractions. He denied problems with appetite, sleep, or energy, but complained of severe depression. He said that most of all, he just wanted to return to work.

Social and Mental Health History: J.R. had a 15-year history of alcohol abuse, but admitted only occasional alcohol use in the last two years. He was married one year to his third wife, with an 8-month-old child. He paid child support to his first two wives. The couple admitted financial stress predating the accident. J.R. had been on the job less than three months at the time of the incident. This was his fourth Worker's Compensation claim. He told the insurance case manager that he was suing an examining doctor who injured his neck. His case manager reported that J.R.'s father was disabled most of his life following a Worker's Compensation injury. He denied a history of psychiatric difficulties.

Learning Test (CVLT), delayed recall was particularly low, one of 16 list items. Recognition memory was worse than expected for mild TBI, 11 words with five false positive errors. On the other hand, J.R.'s performance on the test lacked the common mild TBI subtle findings of repetition or intrusion errors. The Geriatric Depression Scale was in the range for severe depression despite the lack of vegetative findings for sleep, appetite, or energy.

[FIGURES 3-4 OMITTED]

Analysis: The inconsistent history, over-elaborate complaints, and test findings strongly suggested malingering. The couple was told there was no cognitive reason that he could not return to work. They immediately said his balance was too poor to work on construction sites, as he would have to stick his head down in holes or go up ladders. He did not seem pleased when other alternatives to working construction were suggested, despite the claim that he only wanted to return to work.

Treatment: As noted previously, setting limits is the primary treatment for suspected malingerers.

Discussion

Treatment considerations will focus on conversion disorder, as the only treatment for obvious malingering is limit setting. Conversion disorder should be conceptualized as a real and treatable problem. There is potentially much overlap between somatoform disorders such as conversion disorder and malingering. Both diagnoses may be associated with inconsistent and seemingly exaggerated performances, secondary gain, various psychopathologies, and stressful life events. The single most important difference between the two diagnoses is whether or not the patient is unintentionally or unconsciously producing the symptoms (somatoform or conversion disorder) versus intentionally or consciously producing the symptoms (malingering).

The patient with a TBI, mild or moderate, may be especially vulnerable to being diagnosed with malingering or intentional feigning of symptoms. This vulnerability may stem from the variable and the sometimes inconsistent nature of TBI sequelae, the appearance of secondary gain such as law suits or family attention, and the difficulty coping that some patients have after TBI due to the subtle, but significant changes in how they think and function. In addition, patients with TBI are frequently misjudged by the public due to the misperceptions that the public has about brain injury and recovery. These misperceptions about recovery may be strongly influenced by the media, e.g., the seemingly complete recoveries that football players make after sustaining multiple concussions. The entertainment industry also fuels the misperceptions regarding TBI recovery as can be seen by the complete recoveries that actors make after coma. As a result of these misperceptions, many people expect the patient who has sustained a concussion or TBI to make a full recovery and when the patient continues to report symptoms, they are seen as poor copers, exaggerators, or at worst malingerers.

interviews and observation of rehabilitation therapies) will help determine where the conscious versus unconscious line in drawn.

It is critical that the healthcare professional working in rehabilitation not assume that the TBI patient is intentionally or consciously producing symptoms, i.e., malingering: doing so would impede the needed treatment that the patient with TBI deserves. The rehabilitation specialist, who suspects possible conversion or conversion-like disorder, should refer the patient to a qualified mental health professional without necessarily discharging the patient from medically based treatment. Keeping the patient in a medical setting, treated by an interdisciplinary team, can be an ideal situation. When treating the patient who is over-focused on his or her symptoms, the rehabilitation specialist should not convey that the patient's symptoms are psychologically based. Doing so will encourage resistance and symptom exacerbation (Speed, 1996).

After determining that a medical diagnosis cannot fully explain the TBI symptoms, a mental health professional should assess the patient with appropriate psychological and neuropsychological tests. Next, a behavioral approach to treatment should be taken with the adjunct of psychotherapy. The goal of such a program should be to help the patient unlearn a maladaptive response, and learn more appropriate ways of dealing with the environment (Trieschmann, Stolov, & Montgomery, 1970). After a program is established, the patient should be given a pseudo-scientific explanation for his/her disorder. It should be implied that if there is no improvement after the specified course of treatment, the disorder could not be medically based (Teasell & Shapiro, 1994).

For the patient with a TBI, treatment should focus on setting hierarchical goals and providing cognitive related interventions. It is important to give positive reinforcement for improved function and "punish" signs of dysfunction (Speed, 1996). Dysfunction or maladaptive behaviors may be punished by systematic ignoring, removal of a special activity such as an outing, or returning to work on a lower level goal. This strategy is especially useful in group treatment settings. The treatment team should remain positive and patient and keep in mind that most people with conversion disorder improve eventually. TBI, regardless of severity, is a complex diagnosis affecting all aspects of a person's life. The person with a TBI is usually under a severe level of distress considering cognitive changes, physical changes, or both; relationship changes; work changes; and financial changes. Given the stressors with which the person with a TBI may have to deal, he or she may indeed exhibit psychological or emotional symptoms that complicate the picture. At the very least, persons who present with complicated presentations should be given the benefit of the doubt and, therefore, the benefit of rehabilitation treatment.

Table 1
[Tests of Malingering]

Table 1: Tests of Malingering

Name of Test	Author(s)	Description
CVLT-II (Forced Choice Recognition Subtest)	Delis, Kramer, Kaplan, & Ober, 2000	Forced Choice Recognition of words on learned list versus novel unrelated words - > 1 error suggests poor motivation.
Dot Counting Test (DCT)	Rey, 1941 Lezak, 1983	Counting grouped dots versus counting ungrouped dots -- counting grouped dots should take less time.
Rey Malingering Test (RMT)	Rey, 1958	15 items presented in 5 groups of 3 -- subject should be able to draw at least 3 rows.
Symptom Validity Test (SVT)	Pankratz, Fausti & Peed, 1975 Pankratz, 1988	Forced choice -- subject should score at least 50%
b Test	Boone, 2000	15-page booklet of b's, q's, d's, etc. Cut-off scores for time, omissions and commissions.
Portland Digit Recognition Test (PDRT)	Binder & Willis, 1991 Binder, 1993	Forced choice recognition of digits -- subject should score at least 50%.
Test of Memory Malingering (TOMM)	Rees & Tombaugh, 1996	Forced Choice recognition of 50 line drawings -- subject should score at least 50%.
Victoria Symptom Validity Test	Slick, Hopp, & Strauss, 1992, 1995	Computerized version of PDRT -- reaction time also measured. Results classified as malingered, questionable, or valid.
Validity Indicator Profile (VIP)	Frederick, 1997	Assesses consistency of effort over time -- results indicate 1 of 4 response styles: compliant, careless, irrelevant, or malingered.

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Factitious Disorder

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Synonyms and related keywords: Munchausen syndrome, Munchausen's syndrome, Munchausen syndrome by proxy, Munchausen's syndrome by proxy, FD, factitious illness, pseudologia fantastica, Ganser syndrome, narcissism, sociopathy, somatoform illnesses, malingering, somatization disorder, conversion disorder, hypochondriasis, pseudocyesis, pain disorder, body dysmorphic disorder, major depression, delusional disorder, somatic delusions

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Disclosure

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INTRODUCTION

Background: Few patients are more challenging and troublesome to busy clinicians than those with factitious illnesses. The term factitious disorder (FD) refers to any illness deliberately produced or falsified for the sole purpose of assuming the sick role. Patients waste valuable time and resources with lengthy and unnecessary tests and procedures at a cost, according to one estimate, of $40 million per year. Moreover, patients with FD often generate feelings of anger, frustration, or bewilderment in the physician. These patients violate the following unwritten rules of being a patient: (1) patients should provide a reasonably honest history; (2) symptoms result from accident, injury, or chance; and (3) patients hold the desire to recover and cooperate with treatment toward that end.

FDs likely have always been present throughout history and have appeared in the literature since the time of the Roman physician Galen, who wrote about them in the second century. In the 1800s, the British physician Gavin described how some soldiers and seamen pretended illness to excite compassion or interest.

The modern history of FD begins in 1951, when Asher described case reports of patients who habitually migrate from hospital to hospital, seeking admission through feigned symptoms while embellishing their personal history. He assigned the name Munchausen syndrome to this condition after Baron von Munchausen, a well-respected, retired German cavalry officer who had tales of his life stolen and parodied in a booklet in 1785. Persons with Munchausen syndrome were said to typically (1) exhibit numerous surgical scars, especially abdominal surgical scars, (2) display a truculent or evasive manner, (3) provide a dramatic medical history of questionable veracity, and (4) attempt to conceal such documents as hospital discharge forms or insurance claims. Asher distinguished abdominal, hemorrhagic, and neurologic subtypes.

Since the publication of Asher's article, numerous reports of patients producing or falsifying almost every conceivable kind of illness have appeared in the literature. The type of patient described by Asher is now thought to represent a minority of cases of FD. The term Munchausen syndrome most appropriately refers to the subset of patients who have a chronic variant of FD with predominantly physical signs and symptoms. In practice, however, many still use the term Munchausen syndrome interchangeably with FD. In 1976, the term Munchausen syndrome by proxy entered the medical lexicon and came to describe cases in which an individual artificially produces illness in another person, typically a mother who produces illness in a young child.

The *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV)* requires that the following 3 criteria be met for the diagnosis of FD: (1) intentional production or feigning of physical or psychological signs or symptoms, (2) motivation for the behavior is to assume the sick role, and (3) absence of external incentives for the behavior (eg, economic gain, avoiding legal responsibility, improving physical well-being, as in malingering).

The *DSM-IV* recognizes the following 3 types of FD: (1) FD with predominantly psychological signs and symptoms, (2) FD with predominantly physical signs and symptoms, and (3) FD with combined psychological and physical signs and symptoms.

A fourth type, FD not otherwise specified, includes those disorders with factitious symptoms that do not meet the criteria for FD. The *DSM-IV* places FD by proxy (ie, Munchausen syndrome by proxy) into this category, defining it as "the intentional production or feigning of physical or psychological signs or symptoms in another person who is under the individual's care for the purpose of indirectly assuming the sick role." FD by proxy has yet to be recognized as an official separate category in the *DSM-IV*. Appendix B of the *DSM-IV* lists the following research criteria for FD by proxy.

- FD by proxy is the intentional production or feigning of physical or psychological signs or symptoms in another person who is under the individual's care.

- The motivation for the perpetrator's behavior is to assume the sick role by proxy.
- External incentives for the behavior (such as economic gain) are absent.
- The behavior is not better accounted for by another mental disorder.

Pathophysiology: As with many psychiatric illnesses, the pathophysiology of FD is unclear. Case reports of abnormalities on MRIs of the brains of patients with chronic FD suggest that brain biology may play a role in some cases. In addition, some patients with FD have displayed abnormalities on psychological testing. Results of EEG studies have thus far been nonspecific.

Frequency:

- **In the US:** The prevalence of FD is unclear. Many authorities believe the condition is underdiagnosed because it involves willful deception, which may be missed by medical staff. Conversely, the prevalence of chronic FD may be overdiagnosed in some cases because the same patients with FD may migrate from hospital to hospital. The frequency of presentation of various factitious illnesses (eg, which factitious illnesses are most common) is unclear. However, most researchers agree that the prevalence of factitious psychological symptoms is much lower than the prevalence of factitious physical symptoms. Studies investigating the prevalence of FD have found the following:
    - Of patients referred for evaluation of fever of unknown origin at the US National Institute for Allergy and Infectious Disease, 9.3% had FD.
    - Of material submitted by patients as kidney stones, 2.6% was found to be nonphysiologic and probably fraudulent.
- **Internationally:** Whether the epidemiology of FD differs in countries other than the US is unclear.
    - Of patients referred to the consultation-liaison service of a large teaching hospital in Toronto, 0.8% (10 of 1288) had FD.

- Of infants brought to a clinic in Australia because of serious illness, 1.5% were cases of FD by proxy.

Mortality/Morbidity: FD can result in morbidity and mortality from the patient's re-creation of actual medical conditions (eg, exogenous administration of insulin) or from the procedures undertaken by the physician to diagnose or treat the condition (eg, unnecessary cardiac catheterizations, surgeries). No studies have quantified the total estimated morbidity and mortality from FD.

Sex: Persons with FD are usually female and employed in medical fields such as nursing or medical technology. Working in the medical field provides knowledge of how disease might be produced artificially and provides access to equipment (eg, syringes, chemicals) with which to do so.

- Persons with chronic FD (ie, Munchausen syndrome) tend to be unmarried men who are estranged from their families.
- Perpetrators of FD by proxy are typically mothers who induce illness in their young children; however, sometimes fathers or others are responsible.

Age: Persons with FD tend to be women aged 20-40 years. Persons with chronic FD (ie, Munchausen syndrome) tend to be middle-aged men.

History: Patients may feign illness by means of a factitious history alone (eg, falsely claiming to have had a syncopal episode), by a factitious history plus the use of external agents that mimic disease (eg, adding exogenous blood to urine and claiming hematuria), or by a factitious history plus inducing an actual medical condition (eg, injecting bacteria to produce infection, ingesting CNS-active medications to induce psychiatric symptoms).

Detection of FD is typically slowed by the natural tendency among physicians to believe what patients say. Indeed, this tendency may be even greater because many patients with FD work in the health care field and are colleagues.

- The presence of the following factors may raise the possibility that the illness is factitious:
  - Dramatic or atypical presentation
  - Vague and inconsistent details, although possibly plausible on the surface
  - Long medical record with multiple admissions at various hospitals in different cities
  - Knowledge of textbook descriptions of illness
  - Admission circumstances that do not conform to an identifiable medical or mental disorder
  - An unusual grasp of medical terminology
  - Employment in a medically related field
  - Pseudologia fantastica (ie, patients' uncontrollable lying characterized by the fantastic description of false events in their lives)
  - Presentation in the emergency department during times when obtaining old medical records is hampered or when experienced staff are less likely to be present (eg, holidays, late Friday afternoons)
- Other clues that may arise during the course of treatment include the following:
  - A patient who has few visitors despite giving a history of holding an important or prestigious job or one that casts the patient in a heroic role
  - Acceptance, with equanimity, of the discomfort and risk of diagnostic procedures
  - Acceptance, with equanimity, of the discomfort and risk of surgery
  - Substance abuse, especially of prescribed analgesics and sedatives
  - Symptoms or behaviors only present when the patient is being observed

- Controlling, hostile, angry, disruptive, or attention-seeking behavior during hospitalization
- Fluctuating clinical course, including rapid development of complications or a new pathology if the initial workup findings prove negative
- Giving approximate answers to questions (eg, a horse has 3 legs; 7 X 6 = 41), usually occurring in FD with predominantly psychological signs and symptoms (see Ganser Syndrome)

Physical: Suspicion of FD is raised when the patient has multiple surgical scars or a gridiron abdomen, indicating the chronic form of FD, or with evidence of self-induced physical signs.

- Mental Status Examination: Patients with FD may vary in their presentation, and no findings have been shown to be pathognomonic. The following findings are possible:
  - Appearance may include physical findings described above.
  - Attitude may range from cooperative with assessment and treatment to evasive and vague regarding details.
  - Mood and affect may be brighter than what would be expected given the patient's medical condition.
  - Perceptual abnormalities, such as hallucinations and disturbances of thought process or thought content, and suicidality and/or homicidality, may be present with FD with predominantly psychological signs and symptoms. Patients having FD with predominantly physical signs and symptoms usually do not confess to thoughts of harming themselves or others, even when they have actually harmed themselves by deliberately inducing physical illness.
  - Cognitive functioning may be aberrant if the patient presents with Ganser syndrome.

Causes:

- The causes of FD are not well defined. One psychodynamic explanation asserts that patients with FD, who often have a background of neglect or abandonment, are attempting to reenact unresolved early issues with parents. The following explanations are also possible:
  - Underlying masochistic tendencies
  - A need to be the center of attention and to feel important
  - A need to assume a dependent status and receive nurturance
  - A need to ease feelings of worthlessness or vulnerability
  - A need to feel superior to authority figures (eg, the physician) that is gratified by being able to deceive the physician
- Explanations offered for FD by proxy parallel those for FD, except that the parent is using the children to meet these needs. Thus, the child is used as a tool with which to recreate unresolved issues with parents and authority figures.
  - Alternatively, the mother is presumed to gain vicarious satisfaction of attention and nurturance needs that may be missing from her marriage through projective identification.
  - Another explanation asserts that the behavior stems from narcissism, sociopathy, and the desire to manipulate authority figures.
- The risk factors for developing FD remain largely unclear. Based on the histories of patients with FD, the following can be projected as characteristics that may predispose an individual to develop a factitious illness:
  - Presence of other mental disorders or medical conditions in childhood or adolescence that resulted in extensive medical attention
  - Holding a grudge against the medical profession or having had an important relationship with a physician in the past
  - Presence of a personality disorder, especially borderline, narcissistic, or antisocial personality disorder

Delusional Disorder
Depression
Schizophrenia

Other Problems to be Considered:

FD appears in the differential diagnosis for many illnesses. Accordingly, FD must be distinguished from true or real general medical conditions or mental disorders, including those that are (1) due to accident or chance, (2) due to noncompliance with treatment, (3) iatrogenic, or (4) the result of attempted suicide, homicide, or self-mutilation.

FD must also be distinguished from the somatoform illnesses and malingering. FD has been believed to fall on a continuum between these illnesses.

Somatoform disorders include the following conditions:

- Somatization disorder (ie, multiple physical complaints over many years)
- Conversion disorder (ie, defects in sensory or motor functioning having a psychological origin)
- Hypochondriasis (ie, preoccupation with imagined disease or illness)
- Somatoform disorder not otherwise specified (eg, pseudocyesis)
- Pain disorder (ie, severe pain in which psychological factors have a strong component)
- Body dysmorphic disorder (ie, intense preoccupation with a real or imagined defect in appearance)

With somatoform disorders, the production of the symptoms of illness is not intentional, and the motivation for illness is unconscious. In FD, symptoms are produced intentionally but for unconscious reasons. In malingering, symptom production is intentional and conscious to achieve an external incentive beyond assuming the sick role (eg, evading the police, obtaining compensation, getting a bed for the night). In practice, determining whether an external incentive exists is sometimes difficult.

The differential diagnosis for FD by proxy includes the following possibilities:

- Real medical illnesses
- Overanxious parenting
- Normal variability between illnesses
- Illnesses resulting from discontinuation of medicines
- Malingering (by an older child)

Patients with other psychiatric diagnoses can also present with somatic preoccupation that is not supported by findings from physical examination, laboratory testing, or imaging. Patients with major depression with psychotic features and delusional disorder (somatoform type) can present with somatic delusions. Associated features of these conditions should facilitate the differential diagnosis.

Lab Studies:

- The diagnosis of FD is typically made late, after other diagnostic possibilities have been exhausted. Laboratory studies can be especially helpful in facilitating the diagnosis of many physical illnesses as factitious.
    - For example, patients with hypoglycemia can be assessed for exogenous insulin injection by a finding of increased serum insulin/C-peptide ratio (>1.0) during a hypoglycemic episode.
    - Similarly, patients who complain of kidney stones can be asked to filter their urine for stones, and the submitted material can be tested for composition.
    - A tissue biopsy can be helpful in revealing the factitious nature of lesions in which foreign material has been injected to simulate naturally occurring disease.
- Because the range of factitious illnesses is limited only by the imagination of the perpetrator, listing all possible laboratory tests that might prove useful is impossible. However, suspicion that an illness is factitious should be conveyed to the pathologist, who may be helpful in identifying ways to confirm the diagnosis.

Medical Care: Provide medical care as needed to treat comorbid conditions and complications arising from induced illness.

- Psychiatric care
    - Patients with FD must be evaluated fully and assessed for comorbid axis I and axis II diagnoses. By treating axis I disorders, improvement or resolution of factitious behavior may also occur.
    - Pharmacotherapy must be monitored carefully to prevent patients from perpetuating self-destructive behavior. Medications to treat the symptoms of personality disorders, such as selective serotonin reuptake inhibitors (SSRIs) to reduce impulsivity, may be of benefit.
    - Psychotherapy should focus on establishing and maintaining a relationship with the patient. Supportive psychotherapy may help contain the symptoms of FD.
    - Family therapy may help families to better understand patients and their need for attention.
    - Cognitive-behavioral therapy may prove difficult when patients are unable to form a collaborative team, such as with comorbid antisocial personality disorder.

Surgical Care: Provide surgical care as needed to treat comorbid conditions and complications arising from induced illness.

Consultations:

- Psychiatrists
    - Obtaining a psychiatric consultation is recommended when the practitioner believes an illness is possibly factitious.
    - Health care providers should work as a team, together with nursing, social work, and legal personnel.
    - The patient should be gently confronted with the team's suspicions in a supportive manner that focuses on the patient's psychological distress as the source of illness.
    - Psychiatric treatment should be offered to the patient.
    - The patient with FD will probably try to split the team, and this is a danger for the psychiatric consultant who attempts to establish a therapeutic relationship with the patient. Accordingly, some authorities feel that therapy should not be attempted with patients who have FD unless they can make a good-faith showing of desire for therapy.
    - Patients who are confronted typically deny that they have manufactured disease, although a few admit it.
    - Patients with the chronic form of FD typically become angry and discharge themselves from the hospital to try to perpetuate their illness elsewhere.
    - A few patients with FD consent to psychiatric treatment.

- Where FD by proxy is suspected, the law requires physicians to notify the authorities and to initiate steps for the immediate protection of the child.
    - Protection may involve removal of the child from the home, at least until the situation can be completely assessed.
    - Once protective measures are in place, the mother should be confronted with the evidence. She will almost certainly deny the charge and will attempt to remove the child from the hospital.
    - Criminal prosecution of the perpetrator may also be necessary.
    - Evaluation should not be limited to the child involved but should also include his or her siblings.
    - Psychotherapy should be offered to the mother, the affected children, and the family.
    - Pharmacotherapy may be appropriate when the mother has comorbid axis I or axis II conditions that are amenable to treatment.
    - The family requires careful long-term monitoring, especially because of the danger that the mother could move her family and seek to perpetrate such behavior in a new location.

MEDICATION

No medications are shown to be efficacious in treating FD per se. However, pharmacologic therapy for concurrent psychiatric diagnoses is indicated.

FOLLOW-UP

Further Inpatient Care:

- Further inpatient care may be required if patients relapse. This includes treatment of any medical or surgical conditions and psychiatric hospitalization when necessary.

Further Outpatient Care:

- Close psychiatric follow-up care and monitoring in the outpatient setting is indicated to prevent relapse. Close medical follow-up care may also be necessary, depending on the condition.

Transfer:

- Transfer from the medical floor to an inpatient psychiatric department is indicated if patients agree to treatment. In rare cases, involuntary hospitalization may be possible if the patient's health is jeopardized severely by continued production of factitious illness (eg, the patient has already lost a kidney because of FD and is in danger of losing another).

Deterrence/Prevention:

- Deterrence and prevention involve clear documentation of patients with a known history of FD, although it does not involve blacklisting.

Complications:

- Complications may arise from the induction of factitious illness or arise iatrogenically from the workup or treatment for the condition, in addition to producing high health care costs.

Prognosis:

- Chronic FD appears to follow an unremitting course. Treatment may transiently ameliorate symptoms but does not appear to last.
- Patients with simple FD follow a more variable course. Some of those who seek treatment may be able to overcome their illness. In any event, simple FD appears to remit in the fourth decade of life.

Patient Education:

- The patient confronted with staff suspicions that the illness is factitious may be unreceptive to attempts at patient education. Still, education should be attempted in the same gentle and supportive manner with which the patient is confronted. If the patient has given permission, educating family members about the patient's condition may also be helpful. Education as to risks of noncompliance with treatment recommendations is also important, ethically and legally, because the patient may wish to sign out against medical advice.
- Education
    - Convey empathy for the patient's distress that has led to the feigning or intentional production of illness.
    - Inform the patient that his or her distress may improve with treatment.
    - Point out that without treatment, the patient may again seek hospitalization.
    - Emphasize that each episode of producing or feigning illness can result in significant morbidity or even mortality for the patient through the production of illness or the undergoing of unnecessary tests or treatments.
- If the patient is receptive to psychiatric treatment, patient education may be an important component of psychotherapy. Information from this article or other sources may be used to help the patient understand more about his or her illness, including the presumed origins of factitious behavior and the importance of regular follow-up care with the psychiatrist.

Medical/Legal Pitfalls:

- Physicians should keep in mind that the patient with FD is entitled to the same rights to privacy and confidentiality of information as any other patient.
    - Although patients with FD waste valuable resources, notifying other hospitals of patients with FD or circulating a blacklist of such patients probably violates the physician's ethical and legal duties; therefore, this practice should be discouraged. Keep in mind that patients with FD can and do litigate.
    - In a similar vein, searching a patient's belongings without permission for items used in perpetrating factitious illness violates the patient's privacy unless the search is conducted with the patient's consent. Consent can sometimes be gained by revealing the suspicions of FD to the patient while asking permission to search because patients may insist they have nothing to hide.
    - The use of video cameras to monitor patient behavior, if already in routine use to monitor patients' rooms (eg, in some critical care wards) does not appear to violate privacy considerations. The covert operation of cameras for the specific purpose of catching the patient with FD or the perpetrator of FD by proxy is a controversial method that is sometimes used.

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Case Report

Factitious Physical Disorders, Litigation, and Mortality

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This case report describes four patients who died of factors directly related to factitious physical disorder and whose cases involved civil litigation. The causes of death varied among the patients and included massive aspiration pneumonia, cardiac arrest, opioid overdose, and septic shock. We discuss how individuals with factitious disorder may enter the legal system through the process of civil litigation. This entry into the legal system, in which monetary gain plays a significant role, illustrates that the boundary between malingering and factitious disorder can be permeable. Nonetheless, individuals with factitious physical disorder have a strong tendency toward self-injurious behavior that may eventually result in death. In the case series of 20 patients from which these four patients were drawn, four (20%) patients died as a direct result of factitious disease. This mortality rate should serve as a warning sign to physicians who provide care for these patients that they are dealing with a potentially fatal disease.

Factitious physical disorders are those in which an individual consciously creates signs or symptoms of disease. The individual may create the signs or symptoms using a fictitious history, a simulation of disease, or by the actual production of disease states.[1] These cases can be quite vexing for physicians and caregivers because they violate the basic patient-doctor contract. In this contract, patients are allowed to remain in the sick role with the expectation that they want to get out of that role as soon as possible. The patient with factitious disorder violates the contract by seeking to remain in the sick role as a primary goal. DSM-IV[2] established the following criteria for a diagnosis of factitious disorder:

- Intentional production or feigning of physical or psychological signs or symptoms
- The motivation for the behavior is to assume the sick role
- External incentives for the behavior are absent

Factitious disorder can be with predominantly physical or psychological signs and symptoms or both. In contrast to patients with factitious disorder, individuals with malingering have clear secondary gains motivating their behavior. However, in cases in which litigation is present, the boundary between factitious disorders and malingering can become blurred. Litigation creates an external or secondary gain in the form of a potential monetary award and validation by the authority of the court. We will discuss four cases that demonstrate this configuration.

The cases represent a subset of 20 cases that have been described previously and featured both factitious disorders and civil litigation.[3] One or both of the authors served as expert consultants to attorneys involved in the litigation but were not involved in the clinical care of any of the patients. This role as an expert gave the authors data and a longitudinal perspective on the patients that facilitated confirming the factitious diagnosis. The four cases were selected because they were known to have ended in death and were only described within aggregate summary statistical data in the previous publication. We will describe the cases and discuss the implications regarding mortality risk among individuals who litigate factitious physical disorders.

Case Report

Ms. A was a 35-year-old Asian woman who claimed to have sustained a head injury in an altercation with a geriatric patient in the health care setting where she worked as a nurse's aide. After going to a hospital emergency room, she passed out and appeared to be comatose for 3 days. When she awoke, she remembered the incident and where she was injured but failed to remember her family, includ-

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ing her husband and children. She slowly recovered her memory over a period of months but remained unable to work and claimed to have a variety of chronic pain problems, for which she received opioid management and consultation from a psychiatric pain specialist who never diagnosed factitious disorder. Despite a lack of objective evidence for any organic injury (including a normal EEG, computerized tomography scan, and magnetic resonance imaging), her workers' compensation carrier awarded her $500,000 in settlement because of her alleged disability. She and her husband sued the facility in which the alleged injury had taken place and settled out of court for approximately $1.5 million. The settlement was driven by the fact that the defendant facility had insurance up to this amount and did not wish to face any potential risk for liability beyond this level. Within 1 year of receiving her civil litigation award, Ms. A died of an opioid-related respiratory arrest at home. She had remained in a wheelchair until she died.

Mr. B was a 38-year-old married physical therapist who claimed to have a latex allergy producing a work-related disability. In addition to a workers' compensation claim, he initiated litigation against latex glove manufacturers, alleging that their faulty manufacturing process produced excess antigens and that this was the etiology of his illness. While pursuing a medical evaluation for presumed latex-related pulmonary symptoms, Mr. B was hospitalized repeatedly. A psychiatric consultant raised the possibility of factitious disorder as a diagnosis on several admissions. On several of these hospitalizations, Mr. B complained of chest pain and had ECG evidence of ischemia. He received two cardiac catheterizations, which both showed completely patent coronary arteries. On a subsequent hospitalization, he suffered another episode of chest pain, and an ECG demonstrated ventricular tachycardia. In this episode, he suffered massive cardiac ischemia and went on to have brain death as a result of cardiac failure. Before Mr. B had been transferred to the coronary care unit, where he died, a psychiatric consultant suggested a room search, which revealed several syringes filled with epinephrine taped to the underside of his bed. The injection of epinephrine appeared to cause coronary artery spasm in clean coronary arteries and was concluded as being the etiology of his arrhythmia and ischemia. His case was settled by his estate for an undisclosed amount.

Ms. C was a 52-year-old female factory worker. She was in a single car accident in which her pelvis was fractured. After orthopedic surgery to reduce the fractures, she had a long series of wounds that did not heal. She refused psychiatric consultations when they were attempted. Her wounds were complicated by infections that eventuated in polymicrobial (fecal flora) cellulitis that was only poorly responsive to antibiotic treatments. She usually required indwelling central venous access for delivery of antibiotic treatment. During one episode, she was admitted to the hospital for septicemia, and a Hickman catheter was placed for antibiotic treatment. While recovering from the anesthesia, she aspirated a large volume of gastric contents and died. She supposedly had not had anything to eat or drink for more than 12 hours at the time of the aspiration. Her husband continued litigation that had begun before her death against the original surgeons who had repaired her pelvic fractures. In addition, he added the hospital that was last involved with her treatment to the list of defendants. Eventually, the case was settled for an amount below the National Practitioner Data Bank threshold.

Ms. D was a 42-year-old single white woman who had worked as a pharmacy technician. She had a 17-year history of cellulitis in different extremities of her body. For 12 years she had a wound in her left anterior thigh that failed to heal, despite numerous debridements and antibiotic trials. After this, she developed a similar lesion in her right thigh. Although she was able to work intermittently, her time spent in the hospital progressively increased as the years went on. She refused psychiatric treatment, saying that she did not trust psychiatrists because of negative experiences in the past.

During her last hospitalization, she was admitted with new evidence of infection with polymicrobial organisms, including a gram-negative organism, in her right thigh wound. Shortly after admission, she went into septic shock and died despite rigorous treatment. Her family continued a lawsuit that they had encouraged her to initiate before her death, claiming that several of her doctors had treated her inadequately and caused her problems. After her death, a settlement was reached based in part on the argument that her septicemia had been inadequately treated.

Discussion

In factitious disorder, the patient seeks the sick role for many potential reasons. The role may gratify dependency needs, act out patterns derived from childhood, or fulfill other psychological motivations. One common feature of factitious physical disorder is that it allows the individual to obtain socially sanctioned approval. Indeed as Parsons

first described the sick role,[4] individuals are relieved of the normal social demands of society because of their illness as long as they try to recover as soon as they can. In factitious physical disorder, individuals seek the sick role. In fact, when litigation is present, they may be attempting not only to obtain socially sanctioned approval but, in addition, judicially certified approval. The judge or jury may be seen as bestowing a judicial shield from any potential accusation of fabrication. Litigation may also result in additional rewards of obtaining a financial benefit as well as venting various emotions aimed at the defendants.

When factitious physical disorder is mixed with litigation, the process may fit with the individual's psychodynamic motivations that drive the factitious behavior in the first place. For example, when Mr. B sued his employer for his latex allergy, it may have allowed him to vent angry feelings toward authority figures that he might similarly have acted out by deceiving his doctors. This acting out may well have been related to childhood abuse at the hands of his father that had been noted in his history. In other cases, litigation itself, with the promise of a large lifetime annuity award, may parallel the dependency strivings found in factitious physical disorder.

In addition to psychodynamic considerations, the psychological impact of litigation on factitious behavior may be conceptualized from a learning theory model. If individuals learn that the sick role produces benefits, such as increased care or attention that would not otherwise be forthcoming, they may replicate the behavior to obtain the potential rewards. From this perspective, litigation may again serve as a potentially strong reinforcer for sick-role behavior.

Regardless of the theoretical perspective from which one views factitious physical disorders mixed with litigation, it appears that just as winning litigation may procure a judicial seal of approval, it may also yield a lifetime sentence. For example, Ms. A remained in the sick role and in her wheelchair despite no organic basis for the limitation, even after obtaining substantial monetary awards. Winning such an award may also make it even more difficult for the individual to relinquish the sick role without a marked loss of face. Outright malingerers are more prone to relinquish the sick role once the external reward has been obtained. Indeed, this ready relinquishment of the sick role may differentiate patients with factitious disorder from malingerers. Unfortunately, this type of judgment can often be made only in retrospective analysis. Furthermore, even some malingerers may be locked into the sick role lest they suffer a loss of face with a rapid recovery.

The four cases described in this series of patient deaths provide the opportunity for a final retrospective analysis. The outcome of death suggests that the factitious disorder appeared to be aimed at achieving a primary psychological gain. Even though litigation raised the question of secondary gain playing some role, the fact that the patients died because of conditions related to their factitious behavior highlights the persistent pursuit of the sick role. Even the approximately $2 million that Ms. A received was not enough to shift her behavior from the sick role. Indeed, one way of conceptualizing the difference between factitious disorders and malingering is that in factitious disorder, the primary gain of the psychological benefit accruing with the sick role is more important than any secondary gain. In malingering, however, the secondary gain is greater than the primary gain, and once the secondary gain has been achieved in the form of an external reward, the fabricated illness behavior diminishes.

The family of the patient with factitious disorder can play an important role in the pursuit of litigation. For example, family members may encourage a patient to pursue litigation when they do not believe that their relative actually has a self-induced disorder. In some instances, the family member encourages litigation in order to obtain monetary redress for the suffering that their relative has undergone without realizing it was self-induced. In all of our cases, family members were co-plaintiffs alleging complaints such as loss of consortium and loss of earnings from the plaintiff. Family members may inadvertently reinforce factitious behavior when they encourage litigation or become a party to it. In addition, family members may find the defendants readily available targets upon which to focus their anger.

This ability to externalize may allow the family to avoid closer inspection of the possible factitious etiology of their relative's health problems. This psychological process may help relieve the family from any sense of responsibility for the disorder, which could be heightened if they were aware of its factitious etiology. Thus, litigation may diminish the guilt or shame that can arise with the diagnosis of a psychological disorder. This family reaction seemed evident with patient D. Despite the fact that Ms. D had suffered for many years with episodes of cellulitis that had been identified by multiple physicians as being factitious, the family ignored this possibility and instead focused on the doctors and the hospital as being at fault. As Appelbaum and Gutheil[5] have described, a bad outcome coupled with bad feelings may make a family more likely to pursue litigation.

These cases raise another issue regarding factitious disorders once litigation is present. Factitious physical disorder should be regarded as a serious condition that can be life threatening. When factors are present that lead to litigation, such as the increased need to obtain judicial endorsement of the presence of an illness, there may be an increased mortality rate associated with the condition. Moreover, it is possible that the litigation process, with its attendant stressors, may accelerate a worsening in patients with factitious physical disorder. The four cases described in this article involved litigation before the deaths occurred. This combination of factitious disorder and litigation had a mortality rate of four of 20 cases, or 20%. Our follow-up was limited to a 2-year period. Because of the difficulty in correctly ascribing medical illness to a factitious etiology and the loss of patients in the follow-up process, it is possible that the actual mortality rate is even higher. Although our group size was small and thus limits generalizability, the mortality rate in our case series appears higher than in one other study related to patients with factitious disorder who were not identified as being involved in litigation. In that study, Krahn et al.[6] reported a mortality rate of 2% in their case series that had follow-up over a mean period of 64 months.

Consulting psychiatrists and psychologists can serve an important role in educating physicians to recognize factitious disorders. Once recognized, clinicians treating patients with factitious disorder should be alert to the possible presence of litigation. Given the mortality rate in our series, once litigation is present, it may be a marker for an elevated risk of death and should prompt clinicians to take all possible preventive steps. The mortality rate observed in our case series should alert clinicians to consider that in patients who are identified as having factitious physical disorders and reveal that they are in litigation, there is significant risk of a life-threatening condition.

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Article

Patients Who Strive to Be Ill: Factitious Disorder With Physical Symptoms

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Objective: Factitious disorder with physical symptoms characterizes patients who strive to appear medically ill and assume the sick role. Clinical suspicion is highest for female health care workers in the fourth decade of life. This study was designed to analyze the diagnosis of factitious disorder, the demographics of affected patients, and intervention and treatment.

Method: Retrospective examination was of 93 patients diagnosed during 21 years. Two raters agreed on subject eligibility on the basis of DSM-IV criteria and absence of a somatoform disorder and a plausible medical explanation.

Results: The group included 67 women (72.0%); mean age was 30.7 years (SD=8.0) for women and 40.0 years (SD=13.3) for men. Mean age at onset was 25.0 years (SD=7.4). Health care training or jobs were more common for women (65.7%) than men (11.5%). Most often, inexplicable laboratory results established the diagnosis. Eighty had psychiatric consultations; 71 were confronted about their role in the illness. Only 16 acknowledged factitious behavior. Follow-up data were available for only 28 patients (30.1%); maximum duration of follow-up was 156 months. Two patients were known to have died. Few patients pursued psychiatric treatment. Eighteen left the hospital against medical advice.

Conclusions: Factitious disorder affects men and women with different demographic profiles. Diagnosis must be based on careful examination of behavior, motivation, and medical history and not on a stereotype. Laboratory data and outside medical records help identify suspicious circumstances and inconsistencies. Confrontation does not appear to lead to patient acknowledgment and should not be considered necessary for management.

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Factitious disorder with physical symptoms is challenging for health care providers. DSM-IV offers two inclusion criteria: physical symptoms are intentionally produced, and the patient's motivation is to assume the patient role. The only exclusion criterion is the lack of external incentives seen in malingering. The DSM criteria define an extremely heterogeneous population with coexisting medical and psychiatric disorders.

Case series are an invaluable data source for factitious disorders, which can cause irreversible medical consequences for the patient, tremendous cost to society, and strong emotions in health care providers (1–4). The incidence and prevalence of factitious disorder with predominantly physical symptoms are unknown because its inherently secretive nature thwarts traditional epidemiological research. Sutherland and Rodin (5) estimated the incidence at a tertiary medical center of 0.8% on the basis of 10 patients (70% female) referred to psychiatry out of 1,288 psychiatric consultations. Population-based studies that use either surveys or review of comprehensive medical records have not been conducted.

Most literature regarding factitious disorders has been based on hundreds of case reports and a few large series. In 1983, Reich and Gottfried (4) described a 10-year experience with 41 patients with factitious disorders in a hospital population. This group was 95% female, their average age was 33 years, and 68% had health-related jobs. Carney and Brown (1) described 42 patients, 76% of whom were female. The mean age was 34 years, and 50% were in "caring professions." The profile of the young female health care worker with factitious disorder is widely accepted (6).

Method

This study was approved by the Mayo Foundation's institutional review board. Effective Jan. 1, 1997, Minnesota law requires patient consent for all medical records review for research. Consent is not required for patients seen before that date unless they return for subsequent care. Data were collected from February until June 1997; three potential patients were excluded.

Two databases were used to identify patients. The computerized master list of dismissal diagnoses from 1976 to 1996 was searched for "factitial disorder," "factitial symptoms," "Munchausen's syndrome," and "polysurgical syndrome." However, this database was not sufficient because some patients with a compelling diagnosis of factitious disorder were not included. In some cases, the primary medical or surgical service elected not to state "factitious disorder" as the dismissal diagnosis but preferred a less provocative diagnosis, for example, "anemia of unknown origin." To identify cases of this type, the psychiatric consultation service list from 1980 to 1996 was manually reviewed for all re-

TABLE 1. Selected Examples of Inexplicable Laboratory Results for 93 Patients With Factitious Disorder

Presenting Complaint	Laboratory Evidence
Hematuria	Red candy found in urine sample
Recurrent hypoglycemia	Exogenous insulin identified
Nonhealing wound	Mouthwash found in wound
Pheochromocytoma after adrenalectomy elsewhere	Normal adrenal tissue
Diarrhea	Stool sample consisted purely of water
Recurrent renal colic	Glass fragments found in urine sample
Recurrent polymicrobial infections	Unusual pathogens found (aquarium water)
Hypokalemia, diarrhea	Thiazide diuretics detected in urine toxicology screening

quests concerning these disorders. Because psychiatric consultations were not conducted on all patients, this resource was also insufficient. The two databases were used in a concerted effort to identify all patients with possible factitious disorder in the 20-year interval. The same inclusion and exclusion criteria were used for both sources of data. Missing records from the psychiatric consultation database determined the 4-year staggered start.

Data Collection

U.S. residents who were inpatients 18 years or older at index treatment were eligible. This study was designed to test the hypothesis that patients with factitious disorder have a shorter life expectancy than a national age- and sex-matched normative population. The aim was to collect follow-up data and use the Social Security Administration Death Master File to determine the age at death of the patients. Because of tremendous difficulty obtaining follow-up data, this part of the study was not completed. Problems included no permanent address, undocumented Social Security number, false names, and records that disappeared at dismissal.

All medical records were examined by two reviewers. DSM-IV criteria were used. Patients were included if their physical signs or symptoms were intentionally produced and their motivation was to assume the sick role. If external incentives such as economic gain were present, patients were excluded. Several additional issues not referred to in DSM-IV were also addressed. Patients were excluded if their medical records revealed one or more of the following: plausible medical explanation, possible somatoform disorder (suspected unconscious symptom production and motivation), exclusively psychological factitious symptoms, or inadequate data. Patients with symptoms limited to the hair and skin (apart from nonhealing deep wounds) were excluded because this set of patients was large; in this distinct subgroup, motivation to assume the sick role was generally absent. Both raters evaluated patients independently and then reached consensus. Patients were excluded if either reviewer determined a possibility that the symptoms were not factitious, usually because of a plausible medical disorder.

The index treatment was defined as the hospitalization during which the treatment team concluded that the patient's illness was factitious. This index treatment became a reference point, and all subsequent data were considered follow-up or outcome data. Data were sought to support the classic diagnosis of Munchausen's disorder, which requires a self-inflicted medical condition, visits to multiple medical centers (peregrination), and pathologic lying (pseudologia fantastica) (7). This step was undertaken because many physicians persist in using Munchausen's terminology and criteria when referring to patients with factitious disorders. Peregrination was identified as having previously visited three or more medical centers for the same problem. The authors failed to develop a definition for pseudologia fantastica that could be operationalized.

TABLE 2. Demographic Characteristics of 93 Patients With Factitious Disorder

Variable	Patients With Variable			
	N	%		
Sex				
Male	26	28.0		
Female	67	72.0		
Race				
Non-Hispanic/white	84	90.3		
African American	2	2.2		
Other or mixed	7	7.5		
Education				
Less than high school	10	10.8		
High school graduate or some college or technical school	48	51.6		
College graduate or higher	18	19.4		
Unknown	17	18.3		
Health care training				
None	49	52.7		
Nursing	24	25.8		
Medicine	2	2.2		
Other (such as technical, emergency medical technician, medical illustrator)	18	19.4		
Employment				
Employed and/or student	64	68.8		
Disabled	15	16.1		
Worker in health care field	41	44.1		
State of residence				
Minnesota	17	18.3		
Illinois	10	10.8		
Wisconsin	8	8.6		
Other (23 states)	58	62.4		
	Mean	SD	Median	Range
Age at index evaluation (years)[a]	33.8	10.6	32	18–68
Men	40.0	13.3	39	21–68
Women	30.7	8.0	31	18–64
Age at onset (years)[b]	25.0	7.4	26	13–39
Men	26.0	10.2	25	17–38
Women	24.8	7.0	26	13–39

[a] Significant difference between groups ($p<0.003$, Wilcoxon rank-sum test).
[b] Nonsignificant difference between groups ($p=0.70$, Wilcoxon rank-sum test).

Patients were included only if there was conclusive evidence that the patient intentionally produced or feigned physical signs or symptoms (DSM-IV criterion A). The specific categories of evidence and examples are as follows:

1. Inexplicable laboratory results (foreign material in biopsy samples, positive results of toxicology screens, or a history of abnormal findings from biological fluids collected in private but normal findings from fluid collected while patient was under observation) (Table 1).
2. Inconsistency between the history and results of physical examinations.
3. Patient admission of self-induced illness.
4. Records from other institutions (patients denying recent diagnostic evaluations in the context of contradictory information or criminal conviction for Munchausen's syndrome by proxy).
5. Observed tampering and inappropriate behavior (removal of dressings, manipulation of catheters, or syringes containing medications or contamination).
6. Surreptitious use of medications (suspected medications were found in the patient's possession).
7. Family confrontation of patient.

TABLE 3. Evidence of Factitious Disorder in 93 Patients

Evidence	Patients With Evidence[a] N	%
Inexplicable laboratory results	42	45.2
Inconsistent or implausible history	33	35.5
Patient admission of self-induced illness	16	17.2
Outside records	15	16.1
Observed tampering, syringes, etc., found	11	11.8
Hidden medications found	4	4.3
Family confronted patient	3	3.2

[a] Total is more than 93 because some patients had more than one type of evidence.

Statistical Analysis

The data were entered into SAS 6.0 (SAS Institute, Cary, N.C.), and descriptive statistical analysis was conducted. Wilcoxon's rank-sum test (also known as the Mann-Whitney U test) was used to test whether the median age was significantly different between male and female patients in the study. A two-sided test was used (8). The reported p value was based on the normal approximation; p values of 0.05 or less were considered statistically significant. The median, range, mean, and standard deviation were reported for age for each sex.

Results

The study included 93 hospitalized patients with factitious disorder. Twenty cases were obtained from the institutional master list and 73 from the psychiatry consultation service list. The characteristics of the patients are described in Table 2. The study group was predominantly female, and the women were significantly younger than the men ($p<0.003$, Wilcoxon rank-sum test). Women were more likely to have health care training or jobs (N=44, 65.7%) than men (N=3, 11.5%). Peregrination was identified in 16 men (61.5%) and 28 women (41.8%). Table 3 lists the type of evidence that supported factitious disorder, and Table 4 provides several features associated with this study group. Complaints of pain were the most frequently associated feature, and problems with prescription medications were common. A subgroup had close relationships with local physicians. Several patients exhibited unusually immature behavior with hospital staff or family and friends, a finding suggesting poor coping skills.

The interventions pursued by primary medical or surgical services are listed in Table 5. The majority of the patients (76.3%) were confronted with their diagnosis; however, only a small number (17.2%) acknowledged that their illness was self-induced or simulated.

Follow-up data are included in Table 6. A small number of patients received continuing medical care at the same institution after the diagnosis of factitious disorder was established. The medical records revealed three patients who subsequently sought inappropriate medical care elsewhere. In some cases, this outcome was determined because other institutions requested medical records. Eighteen patients left against medical advice, and four patients refused dismissal. A small group of patients agreed to receive psychiatric treatment, but it was difficult to determine to what extent they engaged in treatment and modified their behavior.

TABLE 4. Associated Features in 93 Patients With Factitious Disorder

Feature	Patients With Feature N	%
Self-referred	44	47.3
Pain complaints	85	91.4
Visited three or more medical centers previously for the same problem	28	30.1
Alleged abuse of the patient	21	22.6
Personal involvement with local physician (such as close friend, employee)	13	14.0
Immature relationships		
With medical staff	24	25.8
With family or friends	19	20.4
Chemical dependency issues		
Opioids	14	15.1
Benzodiazepines	9	9.7
Alcohol	8	8.6
Other DSM-IV psychiatric disorders	30	32.3

TABLE 5. Interventions for 93 Patients With Factitious Disorder

Intervention	Patients Given Intervention N	%
Psychiatric consultation		
Obtained	80	86.0
Never ordered	7	7.5
Patient refused	8	8.6
Patient left before assessment	8	8.6
Patient confronted	71	76.3
Patient acknowledged	16	17.2

Discussion

Determining what evidence is sufficient for establishing the diagnosis of factitious disorder remains difficult. Five levels of factitious disorder behavior have been proposed: 1) fictitious history, 2) simulation, 3) exaggeration, 4) aggravation, and 5) self-induction of disease (6). These levels are awkward to apply because they overlap, and patient presentation varies over time. Most of the patients included in this study group would be placed in level 4 or higher because conclusive laboratory data and physical examination data are more readily available. At lower levels of enactment, physicians must rely on inconsistent medical histories. Medical records from elsewhere offering contradictory information are useful, but secretive patients are often reluctant to authorize their release. Insightful family members, if available, also may provide invaluable data regarding a fictitious history or simulation of symptoms.

Differential diagnosis in patients with self-destructive behaviors is extensive, and the disorders include somatoform, eating, chemical dependency, personality, psychotic, and malingering. Verifying that a patient's objective is to assume the sick role rather than, for example, to access drugs, is critically important. Evidence that was acceptable for this retrospective study was deliberately con-

TABLE 6. Follow-Up Data for 93 Patients With Factitious Disorder

Outcome	Patients With Outcome		
	N	%	
Confirmed dead	2	2.2	
Threatened to sue institution	4	4.3	
Ongoing care at institution			
Inpatient	22	23.7	
Outpatient	7	7.5	
Agreed to psychiatric treatment			
Inpatient	11	11.8	
Outpatient	8	8.6	
Subsequently sought inappropriate medical care elsewhere	3	3.2	
Left against medical advice	18	19.4	
Refused dismissal	4	4.3	
No follow-up data	65	69.9	
	Mean	SD	Range
Duration of follow-up (months)	64	51	1–156

servative. Additional patients may have presented with suspicious behaviors that were never documented as potentially factitious by the hospital teams. As a result, these patients are likely an underrepresentation of the number of patients with factitious disorder seen at our institution.

The two databases used for this study yielded similar cases existing in different contexts. The computerized master list primarily included patients with clear-cut situations. This source provided data regarding the 23 patients who were not seen by the psychiatry consultation team; reasons for this were patient refusal, patient dismissal, or lack of a request. Fewer data concerning past psychiatric history and social concerns were available for these patients. Primary services were hesitant to record factitious disorder as the dismissal diagnosis, sometimes with even robust evidence. The psychiatric consultation log revealed 121 patients with a questionable factitious disorder. In 48 instances, patients were excluded from the final study group because of insufficient evidence or exclusion criteria, but in 73 cases, the factors supporting the DSM-IV diagnosis were present in the opinion of the examining psychiatrist. However, sometimes the primary surgical or medical team still declined to explicitly state factitious disorder on the dismissal summary. The willingness for psychiatrists and nonpsychiatrists to document factitious disorder varies considerably within this institution and nationally. Even published case reports can generate controversy to consider factitious disorder in the presence of other psychiatric diagnoses (9).

In our experience, physicians are reluctant to consider a factitious process in the differential diagnosis unless definitive proof is available. If the threshold of evidence is too high, patients undergo unnecessary, risky, and expensive procedures. However, when the standard for evidence is too low, patients can be inappropriately confronted about their role in inducing an illness. In our opinion, factitious disorders must remain diagnoses with exclusion and inclusion criteria. Simply identifying core inclusion symptoms does not address the extensive psychiatric and medical differential diagnosis (10). New diagnostic results, such as low C-peptide levels in suspected exogenous insulin use, can simplify documenting a factitious process (11, 12). However, more in-depth understanding of the vast array of medical disorders and their variants can make it more difficult to confidently consider a factitious explanation.

The need for diagnostic rigor is clear. Once a treatment team becomes suspicious that a patient is deliberately fabricating or simulating an illness, countertransference issues potentially interfere with the provision of compassionate medical care. Furthermore, if the treatment team decides to confront a patient about a suspected factitious disorder, the patient-physician relationship is likely to be irrevocably damaged.

The high percentage (72.0%) of female patients in this study group challenges the DSM-IV assertion that factitious disorder is more common in men. Additional reports describing relatively large study groups would assist in determining the sex prevalence. Clearly, published single case studies may be misleading in this regard.

Our study confirms that a significant subgroup (47.3% of the total) consisted of female health care workers. The well-known stereotype of the patient with factitious disorder has likely biased this retrospective study group. Nonetheless, the study method also permitted identification of a small majority of patients (52.7%) who had a different demographic background. The vague inclusion criteria used previously may have unfairly weighed demographic factors in studies in which the patients were overwhelming female (5). The use of two reviewers to determine potential cases was expected to reduce the ascertainment bias inherent in a project of this type. The relative number of health care workers in case series of factitious disorder is striking (1, 4, 5). These patients have the knowledge and skills needed to induce a plausible illness. Whether this pattern diminishes over time with the evolution of television programs depicting realistic and graphic medical scenes remains to be seen. Close relationships with local physicians, which included family members, employees, and friends, were observed, and the illness conceivably facilitated more contact or the relationship interfered with detection of the factitious process.

Most of the patients (71.3%) were well educated (high school education or higher), and most (68.8%) were either employed or full-time students. This socioeconomic distribution is unlikely to be explained by persons having insurance coverage that permitted access to a tertiary medical center. During this study, unemployed and disabled patients would have had ready access because of government insurance programs. Patients with factitious disorders have been described as belonging to higher socioeconomic groups, but this characterization has not been a satisfactory explanation (13). The age at onset was in early adulthood for both men and women.

Evidence indicating the most effective intervention and treatment is still lacking. Immediate confrontation appears ineffective in most patients. The approach advocated by Eisendrath and associates (10) is likely preferable, in which patients suspected of having a factitious process are treated in a supportive manner without confrontation. Physicians often feel that confrontation is necessary to complete the evaluation; however, data show that few patients admit their behavior. The alternative method is gradually to develop an empathetic relationship that induces the patient to give up the maladaptive behaviors. The assistance of a psychiatrist is beneficial to assess the patient for coexisting psychiatric disorders and to advise the primary health service on management (14). However, when patients place themselves at risk of iatrogenic injury, a more direct approach is sometimes mandatory. Treatment of any coexisting chemical dependency or psychiatric disorder is optimal.

The generalizability of these observations made at a midwestern tertiary care center is unclear. Psychiatrists and nonpsychiatrists at medical centers renowned for specialized diagnostic evaluations likely encounter similar patients. This study group clearly represents a referral group because patients traveled from all over the United States for medical care. However, for many patients, the desire to obtain multiple medical opinions appears to be an element of the disorder. The impact of managed care restrictions on reducing peregrination is unclear. In our opinion, no psychiatric or medical disorder served meaningfully as a control group. In this respect, this study departs from conventional research standards. If future prospective studies are feasible for examining long-term outcome, quality of life, or health care costs, for example, some type of comparison group will be necessary.

The investigators are aware that some of the information in the medical records of this group of patients may be inaccurate. For example, the information regarding visits to previous health care centers could not be easily verified. Patients are likely to have underestimated the number of visits if they were concerned that doing otherwise would expose them to scrutiny for a factitious disorder. Several patients had social and developmental histories that were clearly implausible, but no means were available to verify their educational or occupational histories. The diagnosis of Munchausen's syndrome requires the presence of a self-induced illness, visits to multiple medical centers, and pseudologia fantastica (7). This terminology did not prove to be useful with this study group because of the inherent difficulty in operationalizing these criteria. Because of this hurdle, we recommend that physicians use the DSM terminology of factitious disorder rather than this outdated classification system.

Medical records from other institutions were essential for establishing the diagnosis of factitious disorder for 15 patients. Outside records permitted the treatment team to be aware of the suspicions and working diagnoses of previous health care providers. They also informed the team of which diagnostic tests had been performed previously, and this information helped the team understand the patients' relentless quest for further evaluations, which might include exploratory operations. Outside records will be increasingly difficult to access because of new federal privacy regulations. The effects of new federal privacy regulations that allow patients to request alterations to their medical records if they detect a mistake remain to be seen. Privacy legislation may encourage some patients to obtain much-needed health care. In the case of a patient with a factitious disorder, the regulations may make establishing a diagnosis addressing self-destructive behavior even more difficult.

This study was conducted in compliance with Minnesota state law regulating researchers' access to medical records (15). Legislation is likely to interfere substantially with research on secretive disorders. Patients are unlikely to provide consent for research authorization. Compounding a patient's inherent propensity to falsify information, this legislation may make it increasingly difficult for health care providers to understand the risk factors, appropriate interventions, and outcomes of patients with factitious disorders.

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Evidence-Based Medicine and Medicine-Based Evidence: The Expert Witness in Cases of Factitious Disorder by Proxy

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The UK media has recently devoted much attention to the role of expert witnesses in child protection cases. One or two particular pediatricians who have given expert testimony have been the subject of personal vilification and professional investigation. These cases raise questions about the use of medical expert testimony when there is real uncertainty in the scientific community and the emotional stakes are high. Do doctors use scientific evidence to make diagnoses in the same way that the courts use evidence to make judgments? The cases also raise questions about the personal credibility and trustworthiness of experts: should we allow ourselves to be seen as personally powerful witnesses? Are we responsible for how we are seen by the jury? In this article, these questions are addressed, with the conclusion that distress and anxiety about child maltreatment influences all the players in the justice process and may interfere with the process of justice.

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The British media has recently been much exercised about the role of expert testimony in child protection and maltreatment cases. There have been several highly publicized cases relating to multiple or repeated cot deaths in a family, which have then resulted in the mother's being charged with murder. In one case, the mother was acquitted. In two other cases, the mothers' convictions for murder were overturned, apparently because of concerns about the reliability of the expert evidence. In all three cases, the prosecution had used the same expert, an eminent professor of pediatrics, who is now being vilified by the press as a man who has injured families and tarnished the names of innocent mothers.

These cases raise several fascinating questions for all those who give expert testimony and especially for forensic psychiatrists. Specifically, I argue that there are two questions of primary interest: first, is medical reasoning compatible with legal reasoning? Or, to put it another way, how does evidence-based medicine relate to medicine-based evidence in the courts? Second, if a case based on expert testimony is overturned on appeal, what does that say about the expert? Is an expert liable for what lawyers do with her testimony?

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Munchausen Syndrome by Proxy and Cot Death

In 1977, Professor Roy Meadow first described abnormal parenting behavior in mothers, which he called Munchausen syndrome by proxy (MSBP).[1] The behavior involved mothers who consciously deceived health care professionals into believing that the mothers' children were ill by giving false accounts of symptoms or signs or inducing symptoms in children. Professor Meadow believed that the mothers did this to gain attention for themselves. All his cases involved the presentation of children to hospital doctors.

Since then, there has been considerable research interest in this phenomenon, and many different

types of false presentation have been described.[2,3] Once pediatricians started to look more carefully at how parents, especially mothers, presented their children for health care, it became clear that there is an enormous spectrum of what might be best thought of as abnormal illness behavior by proxy—that is, when a caregiver in charge of a dependent other elicits health care on the charge's behalf in an abnormal way.[4] Doctors play the essential role of validating illness[5] through diagnosis. They either validate the parental fears or reassure that nothing is wrong. Normal rules of illness behavior (in the West, at least) assume that the parent will accept the doctor's findings and comply with the advice given. Doctors, in turn, generally assume that what patients, and in this case, patients' parents, say is true.

However, if parents actively deceive doctors about their children's illnesses, then the normal doctor-patient relationship is fatally undermined in terms of expectable role. Pediatricians can find themselves in the role of possible crime investigators, who need a low threshold of suspicion.[6] This adversarial attitude extends into research as well as normal clinical practice. Researchers making a study of the causes of apnea attacks in infants found 14 cases in which the children's only breathing difficulties were the result of their parents' attempt to smother them.[7] This study led to another of a larger sample of children with breathing difficulties, including 39 cases in which the pediatricians were suspicious that the "apnea" was in fact child abuse. Police investigation showed that they were correct in 33 (84.16%) of the 39 cases.[8]

Professor Meadow himself[9] reviewed the deaths of 81 children who had been found by the criminal or family courts to have been killed by their parents. Most of these deaths had originally been categorized as sudden infant death syndrome (SIDS). There are several theories about why infants might suddenly die (genetically acquired thermoregulation problems, sleeping posture, toxic mattress content, or heart or respiratory problems) and some limited evidence for all of them.[10] But it is also clear that one cause of sudden infant death is murder and that, even if there are other possible causes, deliberate smothering by a parent is also a possibility.

We need not rely only on medical expert evidence for this assertion. Parents who have actually smothered their children may tell professionals that they have done so. Admittedly, this usually happens after the children have been removed from their care because of suspicions about poor care. Such admissions are often made to social workers or therapists and may not result in prosecutions. This means that these accounts are rarely published, either in court transcripts or in the academic press. Nevertheless, experts working in this field all have had experience of parents describing how they smothered their children. This is not to say that it happens often, but the fact that it happens at all is evidence that smothering is one cause of SIDS. There is also evidence from police investigations in hospitals. When there is a high index of suspicion, UK national guidelines on the management of child protection cases support the use of covert video surveillance by the police.[11] Research based on data extracted from this evidence has been published in peer-reviewed journals.[8,12]

Evidence-Based Medicine: "Lies, Damn Lies, and Statistics"

Because of his research experience, Professor Meadow has been frequently instructed by lawyers in relation to child protection cases, especially when there is uncertainty about the cause of the child's injuries or illness. In a recent case, it was alleged by the Crown that the mother had caused the death of her two children by some abusive means. The defense claimed that the children had died of SIDS. Numerous pediatric experts were called by both sides, of which Professor Meadow was one. He stated[13] that the experts agreed that the children had not died of SIDS (although they did disagree about what had caused the infants' deaths and the significance of the post-mortem findings).

At the trial, Professor Meadow cited a published statistic in his testimony, indicating that the chance of a second cot death happening in a middle class home was 1 in 73 million.[14] This figure has come back to haunt him. The mother was eventually convicted and went to prison. She appealed, and the first appeal failed. She appealed again (she was a lawyer, as was her husband) and was successful. This time on the grounds that a prosecution medical expert (not Professor Meadow) had failed to disclose evidence that might support a medical cause for her children's deaths. Throughout both appeals, the media repeatedly referred to Professor Meadow's statistic and ridiculed it as flawed and incredible. The appeals court also made reference to this statistic as "misleading evidence," even though it did not form the substance

of the successful appeal. Since this second appeal, Professor Meadow has been the subject of extensive personal criticism. He has been reported to the General Medical Council on the grounds of alleged misconduct, and all the criminal cases in which he has given expert testimony are to be reviewed.

The more hysterical criticism of some medical experts claims that they set out to exert an unnaturally strong influence over juries in a deliberate way, to pursue their own mad ideas and promote their own careers. The less hysterical criticism states simply that medical experts can cease to be impartial in their evidence. The accusation is that the Professor is identified too much with the prosecution and molds his interpretation of the data to fit the prosecution's case. This identification with the cause, and not the facts of his opinion,[15] could mean that he either consciously or unconsciously gave misleading evidence to the court, which would be unprofessional behavior.

These criticisms of one expert witness should give pause for thought about what we should be doing when we appear in court. There are existing standards of ethics for experts participating in the justice process, which make it clear that the experts' primary duty is to the court and not to those who instruct them.[16,17] To perform a negligent examination or give false or misleading evidence, even if not deliberately, would violate several principles of medical ethics. This is hardly contentious. The contentious implications raised by Professor Meadow's experience relate to two aspects of expert testimony: first, the duty of the expert when there is real uncertainty in the scientific community, and second, the personal credibility of the expert and its influence in the courts.

Medicine-Based Evidence, Not Evidence-Based Medicine

The first problem lies in the different ways that clinicians approach medical facts. In relation to SIDS, in which there is real uncertainty about the cause of death in an infant, pediatricians act like scientists. As good scientists, doctors are taught to be parsimonious in their explanations of scientific facts, not to generate needless hypotheses when there are perfectly good explanations at hand. We are taught, in fact, to take an almost Sherlock Holmesian view of medical investigations, so that, when all investigations for all possible causes of illness have been performed, whatever explanation is left after all the others have been excluded must be the cause, however improbable. For pediatric experts, if all other medical causes of breathing difficulties have been excluded, then smothering must be a real possibility, and they testify thus in court.

The problem, however, is that smothering is not a diagnosis, but a crime. Detectives (like Sherlock Holmes) investigate crime (which is morally and legally deplored). Doctors investigate symptoms of diseases (which are meant to be value neutral). A person cannot be accused of having a disease. In fact, to have a disease is usually to be the subject of sympathy and concern.

A mother who smothers her infant is not necessarily mentally ill. She has demonstrated harmful behavior; she may or may not have a medical diagnosis as well. It cannot be said that she is suffering from smothering behavior, any more than it can be said that she suffers from blowing-nose syndrome or stone throwing. Thus, it makes no sense to ask, as the courts so often do, whether this mother "suffers" from MSBP, because MSBP is not a diagnosis with explanatory power.

Any psychiatric diagnosis in an abusive mother may be used as an explanation for her criminal behavior. It is clear that there is a relationship between some types of mental disorder and risk of criminal harm to others, and in such circumstances, any diagnosis comes to be seen as both an explanation for an odd event and, simultaneously, an accusation of a crime. The problem then is that one is accusing a mother who has lost a child of being responsible for that loss.

Courts, therefore, confuse mental disorders with behavior in ways that psychiatry does not. Courts also do not apply parsimony of explanation in analysis, especially in criminal cases. The case of *R. v. Cannings*[18] makes this abundantly clear. This was one of the cases recently reviewed because Professor Meadow gave evidence at the trial of Mrs. Cannings for the murder of her two children. The conviction has now been overturned, and in judgment, Lord Justice Judge specifically addresses the issue of parsimony of explanation:

> Throughout the [criminal] process great care must be taken not to allow the rarity of these sad events, standing on their own, to be subsumed into an assumption, or virtual assumption, that the dead infants were deliberately killed, or consciously or unconsciously to regard the inability of the defendant to produce

> some convincing explanation for these deaths as providing a measure of support for the Prosecution's case. If on examination of all the evidence every possible known cause has been excluded, the cause remains unknown [Ref. 18, ¶ 177].

The problem with this argument is that Lord Justice Judge appears to be stating that doctors must not include deliberate harm to a child in making a differential diagnosis. Although this fits philosophically with the premise that crime is not a diagnosis, it means that pediatricians should focus only on medical causes of symptoms, and if they cannot identify a medical cause, they should cease to consider the issue. This, however, is to abandon any involvement in child protection, which requires such consideration. Furthermore, both the courts and social services rely on the medical use of parsimonious explanation to trigger investigation. If there is no obvious disease to cause death, then crime may be the only obvious explanation. Even if the cause is an unknown disease not yet understood, the possibility that a crime has been committed is necessary for both police and social service investigations even to begin.

Of course, the child protection point of view is usually too close to the prosecution's point of view for adversarial comfort when it comes to the court. The family courts and the criminal courts are different theaters from an ethics viewpoint. One pursues the best interests of the child, the other pursues a truth beyond reasonable doubt. Pediatric evidence that concludes that smothering is the likely cause of a child's breathing problems clearly assists only one side in an adversarial hearing (although this is not necessarily evidence of bias on the part of the expert). The other side must use any means it can to cast doubt on the pediatric evidence, including undermining the credibility of the expert and advancing alternative explanations for the findings. The judgment in *Cannings* has given further support to this process, stating that if the outcome of a trial depends "exclusively or almost exclusively" on disagreement between medical experts, then "it will be unwise and unsafe to proceed" (Ref. 18, ¶ 178).

What if the defense case goes well beyond the current medical evidence base? In a recent case reported in the British press,[19] a father was charged with murder after his 10-week-old daughter died, with evidence of 32 separate fractures on her body. The defense argued that the father had been using a new scientific technique for feeding his daughter, called "assertive alimentation," which involved his overcoming her resistance to being fed with a bottle. There was no scientific evidence advanced to support this new technique; rather, there was only medical expert testimony that the fractures were consistent with significant force being used on the child. However, because of the judgment in *Cannings*, the trial was abandoned. The relationship between 32 fractures and the cause of death of a little girl is "unknown."

The Discreet Charm of the Expert: Personal Credibility

The other matter raised by the criticism of Professor Meadow is the extent to which experts are responsible for the impression they give of themselves in court—that when it comes to giving evidence, it is they who are convincing and not their testimony. Personal credibility is part of the evidentiary process, down to details such as dress code and demeanor. In an adversarial setting, both sides seek to undermine each others' testimony, and this can and will include undermining the personal credibility of the expert on several matters.[20] The jury decides which evidence it prefers, presumably affected by several factors, of which expert credibility is only one.

If the judge and jury find one expert more persuasive than the counterevidence, then this is part of due process. However, many people might find it alarming to think that personal standing and credibility matter more than science and justice when it comes to expert testimony, especially when there is real uncertainty about the scientific evidence. However, this is a phenomenon associated with all kinds of trials involving experts, not just child protection cases. It could be argued that it is a fault of the adversarial system that the personal attributes of the expert are not neutralized by the examination process, but this need not undermine the justice of the proceedings as a whole.

The counterargument is that if credibility of scientific evidence can rest on attributes as flimsy as personal appearance or charisma, then trust in the justice process as impartial begins to fade, especially trust in the testimony of medical experts. If the public still places high levels of trust in doctors (as surveys repeatedly show), then presumably one basis for that trust is a perceived lack of personal investment or wish for personal glory as the basis for professional altruism. If doctors acting as experts are after personal acclaim and kudos, then this undermines the

perceived altruism that contributes to claims of impartiality and disinterestedness in the doctor-patient relationship.

Appelbaum[21] is undoubtedly right that justice is the trumping virtue in the ethics of the expert and that attention to truth and objectivity are essential for ethically justifiable practice by experts. But objectivity may be difficult in highly emotionally charged cases involving children. It may also be hard to be objective and truthful when there is really comparatively little evidence about which to be objective. One cause of sudden death in children is smothering, and expert pediatricians have every right to say so. It could be argued, however, that in that situation, an expert must be very sure that all other explanations have been explored and excluded, especially if they are contrary to his or her pet theory. There is a danger of experts becoming identified with their view in a way that reduces objectivity, especially if what is at stake appears to be the protection of the most vulnerable.

It is hard not to think that much of the hostility that the pediatric experts meet is caused by the fact that they are accusing those who are the most idealized among people, the mothers of small infants. It still seems to be very difficult for people to accept that mothers (or anyone in a mothering role) may have hostile feelings toward their infants, and reminders of this unpleasant message tend to result in attacks on the messenger. It is also interesting to note Lord Justice Judge's reference to the influence of "unconscious assumptions" in these cases. As a forensic psychotherapist, I am glad to see that the courts acknowledge that unconscious reasoning may be active in the criminal justice process, and I agree that this may affect how participants in the justice process see criminal defendants, especially in cases involving children. But I would add that unconscious process may also affect how experts are seen, and that much of the hostility toward experts like Professor Meadow arises from unconscious anxiety about criticizing parents.

The Limits of Testimony

There is another problem for the pediatricians in cases of unexplained infant death or illness—the pressure they come under to explain what has happened. This pressure may lead them to speak beyond their expertise. It could be said that Professor Meadow is not an expert in MSBP, despite the fact that he wrote the eponymous paper. Although an undoubted expert in the causes of ill health in children, he is not an expert in the field of child maltreatment, or all its causes, or its relationship with mental disorder. However, it is likely that, in the courtroom, he is put under pressure to be all these things.

My own work in this area has taken me frequently to the family courts in cases in which it is claimed that a child's unexplained illness is the result of abnormal illness behavior by the mother. I am repeatedly asked by lawyers to assess the mother in such a case, to see if she is "suffering" from MSBP. Leaving aside the fact that it is not possible to suffer from a behavior, it usually turns out that the cause of the child's illness is disputed, and the lawyers are then seeking to use psychiatric evidence to prove the facts, (i.e., if she "has" MSBP, then she must have done it).

Most forensic psychiatrists are wise to this type of ploy and quickly make it plain that psychiatric expertise cannot determine what happened when facts are disputed and that no diagnosis determines behavior, either past or future. In the family court, there will then be a need for a split hearing: the first part to make a finding of fact as to what happened to the child and the second to hear any relevant psychiatric testimony, once the facts are established. However, pediatric experts are necessary for the finding of fact because it is their evidence that will help the court to determine the cause of the child's injuries or illness. Without training or advice, they may be tempted (or pushed) to speak beyond their expertise, to comment on psychiatric issues in the parent or on treatment of psychiatric disorders, or even to identify a perpetrator where the perpetrator is unknown. I have witnessed pediatric experts giving just such evidence.

Not only is this going well beyond the remit of pediatric expertise, it is abandoning all scientific rigor. Although cohort studies of MSBP-perpetrating mothers suggest that personality disorder and factitious or somatizing disorders are overrepresented in this group,[22] these are data collected retrospectively. There is no evidence base that would allow one to state prospectively that the presence of any of these disorders makes the MSBP behavior more likely. Further, there is ample evidence that most individuals with such disorders do not exercise these behaviors with their children and that most MSBP behavior is displayed by psychiatrically normal people. Pediatric experts who make such statements in court would not accept this type of empirical analysis

if it were presented in the same way to an editorial board for publication as a paper, but some apparently feel able to do so when it forms part of expert evidence. This may reflect the different ways that evidence is assessed by courts and journals—lay review of the personal charisma of the expert by the court as against peer review by anonymous reviewers.

The British courts have not examined the status and admissibility of expert testimony as the U.S. courts have, for example, in *Daubert v. Merrell Dow Pharmaceuticals, Inc.*[23] The duties of the expert are set out in *Anglo Group Plc v. Winther Brown & Co Ltd.*[24] The expert should be able to provide evidence that is not clear to the ordinary person (*R. v. Turner*[25]). The test of the status of the medical evidence would probably mirror the test for negligence; it should reflect a reasonable body of medical opinion (*Bolam v. Friern Hospital Management Committee*[26]), which does not mean that there are not opposing opinions (*Maynard v. West Midlands Regional Health Authority*[27]) and it should be logical (*Bolitho (deceased) v. City & Hackney HA*[28]). On the *Bolitho* view, the type of retrospective inferences offered by some pediatric experts would fail on the grounds of logic.

There is no doubt, however, that both pediatric and psychiatric experts come under great pressure in the family courts to provide medical evidence that will determine who perpetrated the abuse on the child (once established). This expectation is a particular problem when more than one parent is suspected, and there are other vulnerable children in the family. The best interests of children require that they be protected from abusive parents, but it is not in their interests to be separated from a nonabusive parent. The anxiety in the family court to do the right thing is often almost palpable and may tempt experts to abandon both their objectivity and the empirical rigor that underpins it.

Protecting Children: Rocks and Hard Places

Currently, the British media, including the quality press and BBC radio, have gone on record as doubting the "existence" of Munchausen syndrome by proxy, stating that it is not a diagnosis but a theory without evidence promoted by one lone professional. As one radio presenter put it, "What is easier to believe: that a professor dreams up an unbelievable theory or that mothers actually harm their children?" Pediatricians have responded by pointing out that if they are to be vilified publicly for being suspicious and for participating in police or social service investigations of child abuse, then they will cease to do this work. Indeed, there is evidence that complaints against pediatricians have gone up (although they are rarely upheld)[29] and child protection posts are left unfilled.

But pediatric expert testimony is crucial in both the family and criminal courts. Professor Meadow and many other eminent pediatricians have been repeatedly instructed by the courts because of their research and clinical experience. There are no data available about how often which side instructed these experts. The recent publicized cases have dwelt on the fact that these experts often appear for the prosecution in criminal cases, which is perhaps unsurprising. In family cases, English courts now favor the appointment of a single joint expert, whose primary duty is to the court, to reduce the amount of expensive expert testimony and speed up procedures. The Royal College of Pediatricians and Child Health has set up a register of expert witnesses who will be trained and accredited by them.

These interventions will probably not address the problem of the unknown cause of death in a child, as raised by the judgment in *Cannings*.[18] At the time of writing, one mother whose murder conviction was going to appeal has just admitted that she did kill the child in question and a previous sibling.[30] Medical expert testimony is never likely to be flawless in its truth, any more than any other evidence. Perhaps much of the vilification of child protection experts comes because there is such a strong wish that they provide a "truth" for the court that will mean that children will always be safe and justice will always be done.

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Functional Somatic Syndromes

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The term *functional somatic syndrome* has been applied to several related syndromes characterized more by symptoms, suffering, and disability than by consistently demonstrable tissue abnormality. These syndromes include multiple chemical sensitivity, the sick building syndrome, repetition stress injury, the side effects of silicone breast implants, the Gulf War syndrome, chronic whiplash, the chronic fatigue syndrome, the irritable bowel syndrome, and fibromyalgia. Patients with functional somatic syndromes have explicit and highly elaborated self-diagnoses, and their symptoms are often refractory to reassurance, explanation, and standard treatment of symptoms. They share similar phenomenologies, high rates of co-occurrence, similar epidemiologic characteristics, and higher-than-expected prevalences of psychiatric comorbidity. Although discrete pathophysiologic causes may ultimately be found in some patients with functional somatic syndromes, the suffering of these patients is exacerbated by a self-perpetuating, self-validating cycle in which common, endemic, somatic symptoms are incorrectly attributed to serious abnormality, reinforcing the patient's belief that he or she has a serious disease. Four psychosocial factors propel this cycle of symptom amplification: the belief that one has a serious disease; the expectation that one's condition is likely to worsen; the "sick role," including the effects of litigation and compensation; and the alarming portrayal of the condition as catastrophic and disabling. The climate surrounding functional somatic syndromes includes sensationalized media coverage, profound suspicion of medical expertise and physicians, the mobilization of parties with a vested self-interest in the status of functional somatic syndromes, litigation, and a clinical approach that overemphasizes the biomedical and ignores psychosocial factors. All of these influences exacerbate and perpetuate the somatic distress of patients with functional somatic syndromes, heighten their fears and pessimistic expectations, prolong their disability, and reinforce their sick role. A six-step strategy for helping patients with functional somatic syndromes is presented here.

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The term *functional somatic syndrome* refers to several related syndromes that are characterized more by symptoms, suffering, and disability than by disease-specific, demonstrable abnormalities of structure or function. Physicians in many medical specialties are increasingly confronted by patients who have disabling, medically unexplained, somatic symptoms and who have already arrived at a diagnostic label for their illness. The functional somatic syndromes have acquired major sociocultural and political dimensions. Their definitive status in public consciousness and popular discourse contrasts markedly with their still uncertain scientific and biomedical status. Patients with these syndromes often have very explicit disease attributions for their symptoms, and they resist information that contradicts these attributions (1, 2). These patients often have a strong sense of assertiveness and embattled advocacy with respect to their etiologic suppositions, and they may devalue and dismiss medical authority and epidemiologic evidence that conflicts with their beliefs (3).

The functional somatic syndromes include multiple chemical sensitivity, the sick building syndrome, repetition stress injury, chronic whiplash, chronic Lyme disease, the side effects of silicone breast implants, candidiasis hypersensitivity, the Gulf War syndrome, food allergies, mitral valve prolapse, and hypoglycemia. The incidence of several other functional somatic syndromes has apparently declined: chronic carbon monoxide poisoning; chronic mononucleosis; and symptoms resulting from exposure to video display terminals, carbonless copy paper, and weak electromagnetic fields. In three other syndromes—the chronic fatigue syndrome, fibromyalgia, and the irritable bowel syndrome—more uncertainty exists about the presence of demonstrable pathophysiology, but these syndromes are included in this review because they have extensive phenomenologic overlap with other functional somatic syndromes and the psychosocial factors discussed here apply to them.

Methods

English-language articles were identified through a search of the MEDLINE database from 1966 to

the present. The bibliographies of the retrieved articles were then searched for additional publications. Standardized or structured analysis of the identified papers was not possible because of variation in quality, design, and methods and because of the breadth of the articles included. Emphasis was given to empirical studies that used more rigorous diagnostic methods, larger samples, systematic analyses, appropriate comparison groups, and longitudinal follow-up.

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Historical Context

In the past, various conditions associated with the symptoms of functional somatic syndromes (such as headache, musculoskeletal pain, fatigue, gastrointestinal distress, memory difficulties, and insomnia) have arisen, attracted intense medical attention, and then declined in incidence. Neurasthenia, spinal irritation, chronic brucellosis, pinched nerves, railway spine, and soldier's heart were each initially thought to have a medical cause, but when no pathologic basis for these conditions could be established, they subsequently declined in incidence and prevalence. More recently, functional somatic syndromes such as mercury poisoning caused by dental fillings, symptoms resulting from use of video display terminals, and chronic mononucleosis have declined in popularity.

Somatic distress and medically unexplained symptoms have always been endemic to daily life, but the social and cultural characteristics of each era shape the expression, interpretation, and attribution of these symptoms. Thus, similar constellations of benign symptoms acquire different diagnostic labels and are attributed to different causes in different time periods (1, 3). A line of descent can be traced from the DaCosta syndrome through soldier's heart, shell shock, and battle fatigue to the Gulf War syndrome (4). Musculoskeletal pain in the workplace, which previously manifested as writer's cramp and telegraphist's wrist, is now termed *repetition strain injury* (5). There are similarities between railway spine, common in the early 20th century, and the more recent chronic whiplash syndrome (6).

Although the functional somatic syndromes are not new, patients who have these syndromes today differ from their predecessors by being less relieved by negative findings on medical evaluation and less responsive to explanation, reassurance, and palliative treatment (1, 7, 8). Several factors may account for this shift.

First, the authority and prestige of the physician have declined: The reassurance of one's personal physician and the opinions of medical and public health authorities are no longer as calming, reassuring, and palliative as they once were. With this erosion of physician authority and the increasing prevalence of a generalized antiscientific attitude (7), the determination that a functional somatic syndrome has no pathologic basis does not result in a rapid decline in the incidence of that syndrome, as it did in the past (8). This divergence of medical and scientific evidence and public opinion is particularly evident in the recent controversy over silicone breast implants (7).

Second, the current situation is powerfully shaped by the mass media (7–9), which often use hyperbole and uncritical reporting to portray the functional somatic syndromes (3, 7, 10, 11). Preliminary data, tentative findings, and the personal accounts of individual sufferers are reported as conclusive medical evidence (3, 12). The functional somatic syndromes are described as rapidly spreading epidemics, progressive and incapacitating, and some reports insinuate that powerful societal institutions are denying the existence of these syndromes to conceal their own negligence or culpability (3, 11). Such sensationalism and alarmism promote symptoms and distress (5, 13–17).

Finally, the contemporary climate is marked by the prominent political, legal, economic, and regulatory ramifications of the functional somatic syndromes (18–20). Individuals and organizations have strong vested interests in the status of these syndromes, and the actions of these persons and groups may reinforce sufferers' beliefs that their symptoms have a medical basis (21–24). The functional somatic syndromes form the basis for lawsuits and class actions seeking to attribute liability and fault. Medical specialists and clinics develop professional and financial stakes in one syndrome or another. Advocacy groups emerge to mobilize public opinion, influence scientific debate, and shape public policy. The functional somatic syndromes are a source of disputes over health insurance coverage; may propel the creation of environmental, occupational, and workplace regulations; and may qualify sufferers for worker's compensation or disability benefits.

Overlap and Common Characteristics

Each functional somatic syndrome is seen in a heterogeneous group of patients. In some patients, symptoms are attributable to a known disease entity; in others, they result from an unrecognized disorder that may involve physiologic or immunologic hyperreactivity and perceptual hypersensitivity. Other patients have symptoms that are caused by a

psychiatric disorder, and still others have symptoms that are best understood as a response to stressful life circumstances. Our knowledge of the functional somatic syndromes is incomplete, and we do not fully understand the etiologic roles of biological, psychological, and sociocultural factors in these syndromes. Although complex, poorly understood, and heterogeneous, the functional somatic syndromes nonetheless have enough in common to justify our discussing them together as variants of a common biopsychosocial process.

The similarities seen in the functional somatic syndromes have led some to propose that they share a common pathophysiology. Thus, they have been conceptualized as variants of "affective spectrum disorder" because a significant fraction of patients who have these syndromes respond to antidepressant medications of different, unrelated chemical classes (25–27). It has also been suggested that the functional somatic syndromes all involve the same pathophysiologic dysregulation and blunting of the central nervous system's response to stress (28). Further research may shed light on these interesting hypotheses, but they are currently largely speculative.

Phenomenology

Although individual functional somatic syndromes may present with some organ-specific symptoms and may differ with respect to which symptoms are most prominent (for example, neck pain in chronic whiplash and gastrointestinal symptoms in the irritable bowel syndrome), they generally lack characteristic clinical presentations or distinct symptom complexes that are consistent across cases and that distinguish the syndromes from one other (29, 30). The various functional somatic syndromes have remarkably similar symptoms that share two important characteristics: They are diffuse, nonspecific, and ambiguous, and they are very prevalent in healthy, nonpatient populations (31). Symptoms common to the functional somatic syndromes include fatigue; weakness; sleep difficulties; headache; muscle aches and joint pain; problems with memory, attention, and concentration; nausea and other gastrointestinal symptoms; anxiety; depression; irritability; palpitations and "racing heart"; shortness of breath; dizziness or light-headedness; sore throat; and dry mouth.

All of these symptoms have a high incidence in the general population. Surveys of healthy persons who are not patients show that fatigue, headache, joint aches and stiffness, upper respiratory symptoms, and diarrhea are common and generally resolve spontaneously, usually within 1 month (32). Significant fatigue, for example, is reported by more than 20% of adults (33–36), and 30% of persons report current musculoskeletal symptoms (37). Eighty-six percent to 95% of the general population has at least one somatic symptom in a given 2- to 4-week period (32, 38–41). The typical adult has a symptom every 4 to 6 days (32, 39), and 81% of healthy college students report having at least one somatic symptom in a 3-day period (42).

Overlap and Co-occurrence

The functional somatic syndromes have a high degree of overlap and co-occurrence (28, 43–45). Because the conditions are phenomenologically similar (31), the same person often meets the diagnostic criteria for several functional somatic syndromes simultaneously. Considerable overlap has been reported between multiple chemical sensitivity and repetition stress injury (46); between fibromyalgia and the chronic fatigue syndrome; between fibromyalgia and the irritable bowel syndrome (47–51); among multiple chemical sensitivity, the irritable bowel syndrome, and the Gulf War syndrome (31, 45, 50, 52, 53); and among the chronic fatigue syndrome, multiple chemical sensitivity, and fibromyalgia (31, 52). Over time, the same person may believe that he or she has several different functional somatic syndromes (43), a process referred to as *pathoplasticity* (1), and the diagnostic label given to a particular patient may be as strongly influenced by the context and medical specialty of the diagnostician as by the patient's symptoms (54). Confronted with the same polysymptomatic patient, a rheumatologist may focus on upper-extremity symptoms and diagnose repetition stress injury, an internist may inquire into constitutional symptoms and suspect the chronic fatigue syndrome, an allergist may diagnose the sick building syndrome, and a gastroenterologist may focus on bowel symptoms and identify the irritable bowel syndrome (54).

Epidemiology

The functional somatic syndromes have several epidemiologic similarities. They often begin in limited, sporadic outbreaks among small groups of people who are in close contact with each other (such as residents of small, rural towns; coworkers in the same office; or members of particular military units) and then "spread" to other persons with similar risk profiles after widespread publicity and alarm (1–3, 46, 55–60). The pattern of these epidemic-like outbreaks at first suggests infectious contagion or a common toxic agent. Epidemiologic scrutiny, however, shows that the spread occurs along lines of interpersonal communication, acquaintance, and familiarity as well as with physical proximity or exposure to the suspected "pathogen" (1, 5, 18, 20, 43, 46, 61). Prevalence rates vary widely in similar populations exposed to the same putative etiologic agent (62–64), and similar groups in various geo-

graphic locations do not necessarily develop a given syndrome (for example, keyboard operators outside of the United States, England, and Australia develop repetition stress injury less frequently, and non–English-speaking troops deployed to the Persian Gulf did not develop the Gulf War syndrome as often). No dose–response relation can be firmly established (5, 16, 66–70), and no pathogenic toxin, infectious agent, or physical vector is discovered after extensive evaluation (5, 46, 68, 71).

Comorbid Psychiatric Disorders

Patients with functional somatic syndromes have elevated rates of psychiatric disorders, particularly anxiety, depressive, and somatoform disorders. The cause-and-effect relation between the functional somatic syndromes and psychiatric disorders is widely debated because it is often difficult to determine which condition is antecedent and which is consequent (72). Nonetheless, the prevalence of axis I psychiatric disorders, both current and lifetime, is clearly higher in patients with functional somatic syndromes than in the general population or in similar groups of medically ill patients (18–20, 28, 44, 49, 50, 71–75). For example, the prevalence of psychiatric symptoms and psychiatric diagnoses is significantly higher in patients with fibromyalgia than in patients with rheumatoid arthritis or in healthy persons (25, 26, 76–80). Patients with the irritable bowel syndrome have more psychiatric diagnoses, personality disorders, and psychiatric symptoms than patients with inflammatory bowel disease do (81–84). The prevalence of premorbid and current psychiatric disorders is higher in patients with multiple chemical sensitivity than in numerous comparison groups (18, 19, 29, 71, 85), and elevated rates of anxiety and depressive disorders have been seen in several populations with the chronic fatigue syndrome (44, 73, 86–93).

Patients with functional somatic syndromes, including those with the chronic fatigue syndrome, multiple chemical sensitivity, fibromyalgia, and the irritable bowel syndrome, also have a higher prevalence of somatization, somatoform disorders, and medically unexplained symptoms that are unrelated to the functional somatic syndromes (19, 20, 44, 71, 72, 84, 92, 94–101). In some studies, somatization (the experiencing of somatic symptoms that do not have a demonstrable medical basis, the belief that these symptoms are due to demonstratable disease, and the seeking of medical attention for them) predates the onset of the functional somatic syndromes (4, 74); this suggests a preexisting tendency to experience and report bodily distress. For example, a group of patients with multiple chemical sensitivity had significantly more medically unexplained somatic symptoms and a higher prevalence of somatization disorder before the onset of multiple chemical sensitivity than a comparison group did (71). However, somatization occurs in almost everyone at some time and to some degree and does not itself indicate a psychiatric disorder. Because the functional somatic syndromes are determined by multiple factors and are much shaped by psychological, sociocultural, and circumstantial forces, they resist localization anywhere within our medical or psychiatric taxonomy.

Refractoriness to Treatment of Symptoms

The functional somatic syndromes are often refractory to usual medical treatments and standard palliative measures (2). Epidemiologic comparisons of patients who have self-diagnosed functional somatic syndromes with community residents who report the same symptoms suggest that refractoriness, chronicity, and intractability of symptoms are more characteristic of the former group. In those functional somatic syndromes for which an environmental cause is postulated, improvement does not reliably result from control or elimination of the putative toxic agent (46, 102–104). When a physical activity is thought to be pathogenic, rest and physiotherapy are generally not effective (46, 104–106). When restriction of a patient's activities and functioning fails to relieve a given symptom, this is often regarded not as evidence against the putative cause-and-effect relation but rather as an indication that the restrictions were not stringent enough. Patients are thus caught in a vicious cycle in which the ineffectiveness of a treatment strategy leads to its intensification rather than its abandonment.

Amplification and Maintenance of Somatic Symptoms

An Explanatory Model

No single mechanism accounts for the functional somatic syndromes, but the knowledge we have is enough to suggest an explanatory model for the genesis and maintenance of these conditions (11, 107). Distressing symptoms are omnipresent in daily life. They result from benign dysfunctions and self-limited ailments; chronic medical conditions; psychosocial stress; psychiatric disorders; and, less frequently, previously unknown or unrecognized medical conditions. Under the influence of medical scrutiny, public health concern, and media attention, a process of symptom amplification that alters the perception of these endemic symptoms can be set in motion. Learning about a disease of which we were previously unaware (through personal contact with a sufferer, word of mouth, or the media) may lead us

to tentatively reattribute previously ill-defined or treatment-resistant chronic symptoms to the "new" disease (74, 108). (For example, nasal stuffiness and headaches may be ascribed to the sick building syndrome.) This reattribution then amplifies the symptoms themselves, making them seem more intense, noxious, and troublesome (107, 109, 110). The assumption that one is seriously ill also heightens self-scrutiny and prompts a confirmatory search for other symptoms to corroborate one's suspicions. Ambiguous sensations that were previously ignored, dismissed as innocuous, or never consciously noticed are now interpreted as further evidence of the presence of the suspected disease (107, 109, 110). A self-validating and self-perpetuating cycle of symptom amplification and disease conviction ensues: The suspicion of disease heightens bodily awareness, symptom perception, and distress, and these, in turn, reinforce the belief that the sufferer is sick.

This process of confirmatory bias and symptom amplification operates in each individual sufferer. It may also serve as a mechanism for "transmitting" the syndrome from one person to another. A new syndrome may first appear when a few persons with an unusual or previously unknown or ill-defined medical condition are recognized. Under the influence of growing medical and public attention, these persons serve as a nidus around which aggregate other persons who have similar symptoms but do not actually have the same underlying condition. Media publicity, sympathetic physicians, special clinics devoted to the condition, hotlines, litigation, disability compensation, and patient advocacy groups serve as vectors and propel this amplification of symptoms and reattribution of preexisting somatic distress. This process is mediated by four mechanisms: the belief that one has a disease, negative expectations about the future course of the disease, the sick role, and stressful events.

The few persons originally affected may serve as a template for others with similar, preexisting symptoms who reattribute their symptoms to the functional somatic syndrome about which they have recently learned. Sociocultural forces then reinforce the reattribution and, ultimately, the symptoms themselves. Some persons (for example, those with a history of trauma, those with psychiatric disorders, those undergoing major life stress, and those whose families or caregivers reinforce their symptoms and illness behavior [2, 111, 112]) are more vulnerable to this process of amplification. The following discussion focuses on the four specific mechanisms involved in symptom amplification. These amplifiers were selected because they are particularly salient in perpetuating and in maintaining patient distress and because we have empirical evidence about their roles.

Psychosocial Factors That Amplify Symptoms

The Belief That One Is Sick

Bodily perception is an active, not passive, process. Myriad somatic and visceral stimuli are constantly filtered in the brain, and only a small fraction reach conscious attention (109, 113–115). Our suspicions about the causes of our sensations guide this filtering and appraisal process: Sensations thought to have pathologic significance are selected for conscious attention and are amplified. The influence of cognitive beliefs on somatic perception is evident in studies showing that disease labeling results in decreased psychological health and increased absenteeism (116). For example, patients who did not know that they were hypertensive show a threefold increase in days of work missed after diagnosis; this effect is independent of the antihypertensive regimen (117). In a prospective study of herpes zoster (118), the persistence of pain at follow-up was predicted by the extent of the patient's conviction about the disease at inception. Among patients with chest pain but not serious coronary artery disease, the persistence of pain was predicted by the patient's earlier belief that he or she was prone to serious heart disease (119). Similarly, the persistence of fatigue after viral infection has been associated with the patient's belief in his or her vulnerability to viruses and with the tendency to ascribe ambiguous bodily symptoms to disease (120). In two prospective studies of the chronic fatigue syndrome (22, 23), the strength of the sufferers' belief that their fatigue had a medical basis predicted poor subsequent symptomatic outcome. Finally, patients' convictions that they had severe lactose intolerance led them to misattribute various benign abdominal symptoms to this disorder (67).

Beliefs about disease also bias recall of past symptoms (121). In a comparison with uninjured controls, patients with whiplash were found to underestimate their preinjury history of neck symptoms (62, 122). Compared with women who had less negative views of menstrual distress, women who believed that menstruation is a negative experience recalled past menstrual periods as more symptomatic than they had reported them to be when they were experiencing them. The two groups of women did not differ, however, in their recall of intermenstrual symptoms (123). Similarly, informing healthy volunteers in an experiment that they had just tested positive for a disease caused them to recall symptoms that were said to characterize that disease and to recall more behaviors that were described as risk factors for the disease (124).

Thus, the more convinced patients with functional somatic syndromes are that their symptoms are serious and pathologic, the more intense, pro-

longed, and disabling the symptoms become. Such symptom amplification is fostered by physicians who prematurely focus exclusively on medical explanations for the symptoms, by alarming anecdotes in the popular press and on the Internet, and by organized campaigns to designate a particular syndrome as a serious disease (24, 125, 126).

Future Expectations and the Role of Suggestion

Suggestion amplifies and maintains symptoms because humans tend to perceive what they expect to perceive. The cognitive processing of current bodily sensation is guided by our expectations of what we will experience next. This was shown in a multicenter study of aspirin treatment for unstable angina (127). Patients whose informed consent forms explicitly mentioned possible gastrointestinal side effects had a significantly higher incidence of gastrointestinal symptoms (but not confirmed gastrointestinal disease) than did patients whose forms did not specifically mention these effects. Six times as many patients in the former group withdrew from the study because of gastrointestinal distress (127). In patients with the chronic fatigue syndrome, extremely negative expectations about the future consequences of exercise are associated with higher levels of fatigue and disability (128). Similarly, patients who are more concerned about the seriousness of whiplash at the time of injury have longer lasting symptoms (129), and the expectations of patients with mild head injury with regard to future symptoms explain as much of the variance in symptoms as the injuries themselves (62). The power of suggestion has also been shown to influence healthy persons: Instructing persons to attend to evidence of "nasal obstruction" as they breathed induced more symptoms than instructing them to attend to the "free passage of air" (109). Similarly, headache was induced in volunteers who were told that a mild electric current that produces headache would be passed through their heads, when in fact no electricity was administered (130).

Studies of communities exposed to toxic waste pollution are also relevant. Compared with residents of unexposed communities, exposed residents report a broader range of somatic symptoms than can be attributed to the pollutant, and symptoms are most prominent in persons who believe that toxic waste and environmental pollution are more threatening and dangerous (131–133). Media coverage, community and legal action, and allegations of cover-ups alter the perception of normally occurring benign symptoms in those who expect to become sick, causing them to misattribute symptoms to the pollutant (131, 132). One explanation for the increased incidence of somatic symptoms in Gulf War veterans may be the suggestions made by the media, some medical professionals, and advocacy groups about the negative health consequences of suspected toxic exposures (126). Similarly, persons investigating repetition stress injury have concluded that exaggerated media reports of this condition's seriousness and suggestions that the condition is progressive and incapacitating perpetuate the symptoms and disability associated with it (5, 16).

The Sick Role

Symptoms are also amplified by the act of becoming a patient. The assumption of the sick role can initiate far-reaching and pervasive changes—such as unemployment, altered social relationships and family dynamics, and medical help seeking—that in themselves amplify symptoms. Thus, the responses of family members, employers, and physicians to a patient's illness behavior can exacerbate or alleviate chronic pain and the symptoms of somatoform disorders (134–137), and the chronicity of medically unexplained symptoms has been empirically associated with such "secondary gains" (138). In general terms, social labeling theory posits that the connotations and implications of the label we apply to a condition or state influence the outcome of that condition or state. Once a person is labeled as ill, for example, he or she is regarded and treated in ways that make recovery more difficult: Continued illness is expected of the person, and symptoms therefore persist (139).

Health-contingent litigation, monetary compensation, and disability payments all have negative effects on symptoms (140). This was shown by a recent study of whiplash in a country that has little physician or public awareness of the syndrome, no litigation or compensation for it, and no involvement of insurance companies. Victims of rear-end motor vehicle accidents in this country did not have a higher incidence of postaccident headache and neck pain than did randomly chosen, uninjured, age- and sex-matched persons (63). A large body of literature indicates that injury compensation and worker disability payments are associated with a poorer symptomatic outcome after medical treatment and with a prolonged rehabilitative course (141–143). For example, recovery from surgery for the carpal tunnel syndrome is more prolonged and more symptomatic in persons who receive workers' compensation than in those who do not (144). Forcing someone to repeatedly prove that he or she is sick confounds the illness experience, impedes recovery from symptoms, and fosters invalidism (142, 145). When the continuation of benefits is contingent on the continuation of symptoms, the patient is trapped in the sick role. Thus, the incidence of repetitive stress injury is closely correlated with the availability and generosity of disability and workers'

compensation payments, and it declines after administrative and judicial decrees that it is not compensable (16, 104, 146).

When persons with functional somatic syndromes become patients and are given a diagnosis, they are admitted to the sick role. They may curtail or stop work, limit recreational or social activities, pursue legal action or receive disability compensation; read about their condition in magazines and on the Internet, meet and talk with fellow sufferers, and join an advocacy group. Although these steps may be adaptive and appropriate for some, they may also have unintended, long-term, negative consequences by strengthening expectations of future distress, reinforcing symptoms, and making recovery more difficult. Recovery is more difficult and requires greater face saving when sick role behaviors have become more extensive and ingrained: Clinical improvement may seem to call into question the patient's veracity or the legitimacy of his or her symptoms.

Stress and Distress

Stress exacerbates and perpetuates physical symptoms, lowers the threshold for medical help seeking, and makes us quicker to conclude that an ambiguous bodily sensation is due to disease (111, 147–152). Two types of stress are relevant: 1) daily life problems and recurring minor irritants and 2) major life changes and events requiring adaptation. Repetition stress injury, for example, is closely associated with daily stresses and hassles in the workplace and tends to occur when workers must adapt to a new technology that is demanding, threatens job security, and raises expectations for productivity. Clerical workers who report upper-extremity pain also report greater work demands, less control over their work, more job insecurity, and less camaraderie with their coworkers than do workers without such pain (153, 154). A similar relation exists between job stress and back pain (155–158), and perceived work intensity, mental strain, and stressful home lives are more common among workers who acutely develop the sick building syndrome (159). Recurrent, daily stresses have been shown to amplify pain in patients with rheumatoid arthritis (160, 161). Similarly, chronic whiplash symptoms 6 months after a motor vehicle accident were prospectively predicted by daily life stresses in the months before the injury, whereas neurologic signs did not predict subsequent distress (66, 162).

Stressful major life events have also been shown to amplify bodily symptoms. Natural disasters, such as floods (163–165); warfare; criminal victimization; and exposure to environmental pollutants (17, 166–168) result in medically unexplained symptoms. Emotionally laden stressors have been found to exacerbate or precipitate many functional somatic syndromes (28, 169–171). Military combat has resulted in a consistent syndrome of medically unexplained symptoms in U.S. soldiers since the U.S. Civil War (4, 17, 172). Medically unexplained somatic symptoms increase substantially in populations stressed by exposure to environmental toxins and in populations that are only rumored to have had such exposure (13–15).

Stress amplifies symptoms in two ways. First, because stress is widely known to be pathogenic, persons under stress are quicker to ascribe ambiguous bodily symptoms to disease rather than to attribute them to normal physiology, as they might otherwise do. Second, external stressors induce anxiety and depression, which have their own somatic and autonomic concomitants. Anxiety decreases the pain threshold and pain tolerance (173). It also causes apprehensive self-scrutiny and a sense of physical threat and jeopardy, which make symptoms more noxious, ominous, and worrisome (174–176). Depression, in addition to producing its own autonomic symptoms, amplifies and perpetuates other somatic symptoms (174–176). For example, patients who had more persistent and prolonged symptoms after an influenza outbreak were shown to have had higher levels of depression before becoming sick (177).

The more the functional somatic syndromes are thought of as ominous, incapacitating, and severe, and the more alarm and peril are associated with them (in short, the more stressful the experience of illness), the more intense and disabling symptoms become (5, 13–17).

Helping the Patient

The hyperbole, litigation, compensation, and self-interested advocacy surrounding the functional somatic syndromes can exacerbate and perpetuate symptoms, heighten fears and concerns, prolong disability, and reinforce the sick role. Excessive medical testing and treatment expose patients to iatrogenic harm and amplify symptoms. Exclusive emphasis on a search for structural abnormalities can distract physicians from eliciting the patient's beliefs, expectations, and personal circumstances. Patients with functional somatic syndromes can become so engrossed in establishing the legitimacy of their condition, so invested in discovering the cause of their symptoms, and so preoccupied with assigning fault and culpability that palliative treatment is made more difficult or is forgone.

Given these caveats, how should clinicians proceed? Medical management rests on six steps: 1) ruling out the presence of diagnosable medical disease, 2) searching for psychiatric disorders, 3) build-

ing a collaborative alliance with the patient, 4) making restoration of function the goal of treatment, 5) providing limited reassurance, and 6) prescribing cognitive–behavioral therapy for patients who have not responded to the aforementioned five steps.

First, clinicians must uphold their medical mandate with an appropriate search for a previously unrecognized medical disorder. In deciding how extensive this medical work-up should be, physicians must remember the adverse effects of overly aggressive investigation, of fostering the sick role, and of leading patients to expect a definitive medical explanation for all somatic distress. Caution is advised in ordering tests and obtaining specialty consultations solely to reassure the patient—negative findings provide little reassurance to most patients with chronic, medically unexplained symptoms and often ultimately heighten rather than assuage worry and anxiety (178–180). Furthermore, extensive medical testing carries the risk for iatrogenesis and solidifies the patient's conviction that his or her distress has a biomedical cause (24, 181). It is therefore helpful to have evidence-based guidelines for the appropriate extent of medical evaluation and the frequency with which such evaluation should be repeated. Currently, expert consensus has been promulgated for only a few functional somatic syndromes.

Second, the physician should search for diagnosable psychiatric disorders, particularly major depression and panic disorder (which are highly prevalent and treatable). Self-report screening questionnaires and brief, structural diagnostic interviews can assist the physician in this search. It is important to remember that the likelihood of a psychiatric diagnosis increases linearly with the number of somatic symptoms that the patient reports (97, 182–184). For example, compared with patients who have no pain, those who have medically unexplained pain at two sites have a fivefold higher prevalence of major depression, and those with three or more pains have eight times the risk for major depression (185). The stigma associated with a psychiatric diagnosis often makes patients feel that the legitimacy of their illness is being discounted and may make them cling more assiduously to a biomedical explanation of their symptoms (134). Patients must be assured that the presence of a psychiatric disorder in no way means that their somatic symptoms are imaginary or feigned. They should be told that psychiatric disorders are regarded less as causes of somatic symptoms than as amplifiers that exacerbate and perpetuate symptoms and impede recovery.

Third, a collaborative therapeutic alliance between physician and patient is crucial. The physician must take special care to acknowledge and legitimize the patient's suffering because a definitive biomedical explanation for the patient's symptoms has proven elusive. At the same time, the physician should discourage the patient from assuming the sick role, should undercut alarming expectations about the clinical course, and should avoid making distressing symptom attributions. Closely related to the establishment of a collaborative alliance is the process of making symptom palliation, coping, and rehabilitation the focus of the clinical enterprise. The goal of treatment becomes the identification and alleviation of factors that amplify and perpetuate the patient's symptoms and cause functional impairment. The focus is on coping rather than on curing, on improving functional status rather than eradicating symptoms. If this is to be accomplished, patients with functional somatic syndromes must be actively involved in the treatment process and must be dissuaded from assuming a passive role and waiting to be cured by medical procedures or interventions. Realistic, incremental goals should be set and should be specified in terms of observable behaviors. (For example, a gently graduated exercise program should be prescribed.) Patients should be encouraged to resume their activities as much as possible and to remain at work if they are at all able.

Limited, cautious reassurance is appropriate. Patients can be reassured that grave medical diagnoses have been ruled out and can be told clearly that they do not have a lethal or progressive disease. However, because these patients feel ill and symptomatic, it is not enough to tell them what they do not have without telling them what they do have. It is often helpful to describe the process of amplification, whereby sociocultural and psychological processes exacerbate distress and hinder recovery. Although it does not provide a definitive etiologic explanation for a patient's distress, such a discussion gives patients an explanatory model that focuses on processes and functioning rather than on structural abnormalities.

Finally, if these strategies are insufficient, cognitive–behavioral therapies can be effective in treating the persistent distress and disability resulting from functional somatic syndromes. Such therapies have been developed for the somatoform disorders and for some medically unexplained symptoms, including those of the irritable bowel syndrome, fibromyalgia, the chronic fatigue syndrome, headache, and atypical chest pain (186–198). Controlled intervention trials with long-term follow-up have shown the effectiveness of cognitive–behavioral treatment in reducing somatic symptoms, generalized distress, and disability (186–197, 199–204). These interventions help patients cope with symptoms by helping them reexamine their health beliefs and expectations and explore the effects of the sick role and of stress and distress on their symptoms. They help patients find

alternative explanations for symptoms, restructure faulty disease beliefs, alter expectations, and learn techniques of focused attention and distraction. Behavioral strategies, such as response prevention, systematic desensitization, graduated exercise regimens, and progressive muscle relaxation, help those with functional somatic syndromes resume normal activities, minimize role impairment, and curtail sick role behaviors. The cognitive–behavioral approach stimulates patients to assume a more active role in coping and rehabilitation, and it counters the assumption that cure results only from the application of technological interventions to passive patients.

The role of traditional psychotherapy is generally restricted to cases in which the patient with a functional somatic syndrome identifies a psychological problem or a source of emotional distress for which he or she wants treatment. Psychotropic medications are indicated when a pharmacologically responsive psychiatric disorder (such as major depression or panic disorder) is present. In addition, antidepressants sometimes alleviate somatic symptoms (particularly pain and insomnia) and may improve the functional status of patients who have functional somatic syndromes and subthreshold psychiatric disorders. The empirical evidence for the efficacy of antidepressants is strongest for the chronic fatigue syndrome, fibromyalgia, and the irritable bowel syndrome (27). Little is known about the use of alternative therapies in functional somatic syndromes. They may help some patients by providing an enhanced sense of self-efficacy and control over symptoms, but empirical data on this topic are not available.

Conclusions

The functional somatic syndromes cause great suffering, distress, and disability and have substantial societal costs. The public, therefore, needs 1) to be cautioned about prematurely concluding that symptoms indicate serious disease, 2) to become more cognizant of the ubiquity of benign symptoms and self-limited conditions, and 3) to appreciate the influence of psychosocial factors on the experience of illness. Research into the functional somatic syndromes must continue, but the search for biological and physical causes of symptoms should be accompanied by study of psychological and sociocultural factors. Finally, the media must offer the public a less sensational, more accurate, and more sophisticated model of the functional somatic syndromes—one that encompasses both biomedical and psychosocial factors. Such a comprehensive, biopsychosocial approach to functional somatic syndromes by the medical profession, the public, and the media should permit us to better understand and more effectively treat these conditions.

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REVIEW

Functional symptoms in neurology: questions and answers

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Between 10 and 30% of patients seen by neurologists have symptoms for which there is no current pathophysiological explanation. The objective of this review is to answer questions many neurologists have about disorders characterised by unexplained symptoms (functional disorders) by conducting a multidisciplinary review based on published reports and clinical experience. Current concepts explain functional symptoms as resulting from auto-suggestion, innate coping styles, disorders of volition or attention. Predisposing, precipitating, and perpetuating aetiological factors can be identified and contribute to a therapeutic formulation. The sympathetic communication of the diagnosis by the neurologist is important and all patients should be screened for psychiatric or psychological symptoms because up to two thirds have symptomatic psychiatric comorbidity. Treatment programmes are likely to be most successful if there is close collaboration between neurologists, (liaison) psychiatrists, psychologists, and general practitioners. Long term, symptoms persist in over 50% of patients and many patients remain dependent on financial help from the government. Neurologists can acquire the skills needed to engage patients in psychological treatment but would benefit from closer working relationships with liaison psychiatry or psychology.

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In a typical neurology outpatient clinic, 10–30% of patients will have symptoms which are not explicable by demonstrable structural or pathophysiological abnormalities.[1] Unexplained symptoms are particularly common amongst frequent users of healthcare services.[2] Up to two thirds of these patients have symptomatic psychiatric comorbidity and many describe suicidal ideation if asked.[3 4] Neurologists need to be able not only to diagnose symptoms as functional but also to communicate with and manage patients in whom no clear organic explanation for symptoms can be found. This article addresses some of the common questions which neurologists face when they see patients with functional symptoms.

WHAT ARE FUNCTIONAL SYMPTOMS?

Functional symptoms are physical complaints postulated to be associated with psychological distress, which are not primarily explained by pathophysiological or structural abnormalities. This explanation sounds succinct but suffers from several conceptual limitations. Firstly, the diagnosis depends on the exclusion of a medical explanation by clinical judgement or investigation (which may be impossible in paroxysmal disorders like epilepsy, or in conditions like migraine, in which the diagnosis relies on the subjective assessment of the examiner). Secondly, psychological distress is difficult to measure or objectify. Thirdly, functional symptoms can complicate medically explained disease processes which can cause difficulties with delineation and diagnosis.

Functional symptoms can mimic those of most recognised neurological disorders (fig 1), manifest acutely or as a more indolent problem, and can be persistent or intermittent in nature. Functional symptoms can occur together with symptoms of neurologically explained disorders or may be the defining manifestation of a functional disorder.

WHAT NAME SHOULD WE USE FOR FUNCTIONAL SYMPTOMS?

Functional symptoms have attracted many different terminologies. Some diagnostic labels which were initially acceptable had to be abandoned because they entered common usage as derogatory idioms.[5] Appropriate terminology is important because the explanation of functional symptoms is an important part of treatment, and the acceptance of the explanation given is associated with a better prognosis.[6 7] This communication may be hindered if patients feel that their symptoms are interpreted by the doctor as malingering or exaggeration. A recent study has demonstrated that ''functional'' is more acceptable to patients than the terms ''psychosomatic'', ''medically unexplained'', or ''stress related''.[8] In case of seizures, ''functional'' proved less offensive than ''hysterical'', ''pseudo'', ''stress related'', and ''psychogenic'' or the expression ''non-epileptic attack disorder''.[9] The term functional also lends itself to offering patients a positive explanation of symptoms (for example, ''there is no damage of nerve cells but a disruption of function'').

In the current taxonomies (Diagnostic and Statistical Manual of Mental Disorder, 4th edition (DSM-IV),[10] International Statistical Classification of Diseases, 10th revision (ICD-10)[11]) functional symptoms can be classified as manifestations of somatoform disorders (physical symptoms which suggest a general medical condition) or dissociative disorders (disruption

Abbreviations: DSM-IV, Diagnostic and Statistical Manual of Mental Disorder, 4th edition; ICD-10, International Statistical Classification of Diseases, 10th revision

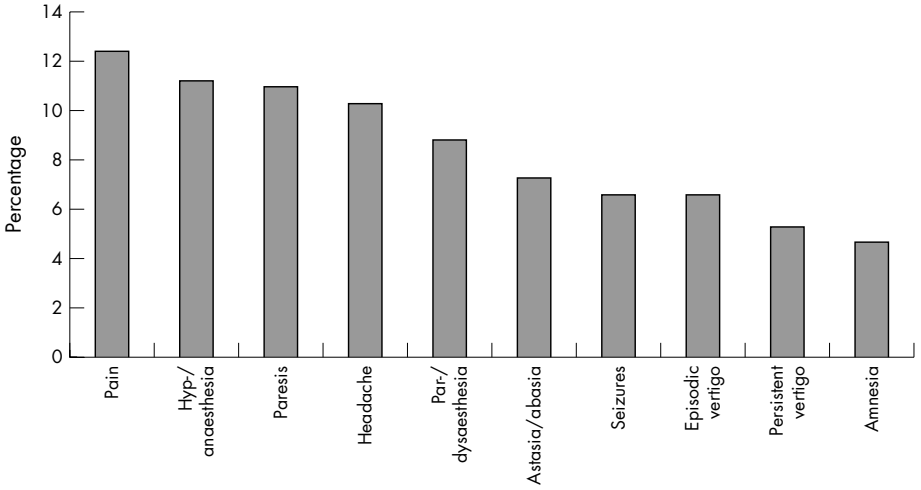

Figure 1 Distribution of 717 functional symptoms in 405 neurology patients.[19]

of the usually integrated functions of consciousness, memory, identity, or perception). However, many functional neurological symptoms (like amnesia or seizures) fulfil diagnostic criteria for both categories. Perhaps because of this, the present clinical criteria in the ICD and DSM systems do not perform well diagnostically if relied upon alone.[12] What is more, the term ''somatoform'' does not imply a positive explanation for the symptom, and there have been increasing criticisms of the somatoform classification with more emphasis being placed on symptomatology.[13]

Functional symptoms were previously called ''hysterical'', but the term's derogatory connotations and the ever widening meaning of the word (for example, mass hysteria, hysterical personality) make it inappropriate.[5] Functional problems are sometimes called ''psychogenic'' or attributed to ''conversion''. However, in the ICD-10 the term ''psychogenic'' is defined quite narrowly as signifying an association with recent trauma,[11] and the term ''conversion'' evokes an aetiological mechanism for which we have no evidence. These models are too simplistic in most cases.

The term ''medically unexplained'' is a better representation of scientific knowledge to date, but may make patients think that their symptom is not being taken seriously, is unlikely to inspire confidence, and may jeopardise engagement with future therapeutic endeavours.

ARE FUNCTIONAL SYMPTOMS WILFULLY PRODUCED?

In line with the current classificatory systems of mental disorders (DSM-IV, ICD-10), most clinicians attempt to distinguish between functional symptoms (which are *not* intentionally produced by the patient) from symptoms that are feigned. However, whereas variability of effort can be quantified, intentionality cannot.[14] Besides, there may not be a categorical distinction between patients who feign their symptoms and patients who do not.[15] Because of this, it is actually not possible to say whether or how often functional symptoms are wilfully produced. Fortunately, it is rarely necessary for a clinician to determine whether symptoms are intentional. Even if patients see no other way of reducing psychological distress than feigning illness or exaggerating pathophysiologically explained symptoms, it may be appropriate to offer medical or psychological attention.[16]

ARE THERE CLEAR DISTINCTIONS BETWEEN FUNCTIONAL, FEIGNED, AND HYPOCHONDRIACAL SYMPTOMS?

The DSM-IV and the ICD-10 encourage clinicians to divide intentional symptoms into malingered symptoms (wilfully produced for external gain) or factitious symptoms (wilfully produced for internal gain). However, it is very difficult for a clinical observer to judge the internal or external reward for a symptom. Similar problems arise with the concept of ''secondary gain'' (for example, benefits associated with taking on a sick role).[17]

The DSM-IV and ICD-10 further distinguish between somatoform disorders (which are characterised by functional symptoms) and hypochondriasis, in which there is predominant anxiety about illness, often in the presence of misinterpretation of physiological processes in the body. However, somatoform disorders are often also associated with anxiety about ''serious'' underlying pathology, and this categorical distinction may be difficult to make.

This does not mean that there is no difference between patients whose functional symptoms are unintentional, malingered, factitious, or hypochondriacal. The margins between these conditions are simply not as clear as the prototypical definitions in the DSM-IV and ICD-10 suggest.

ARE FUNCTIONAL SYMPTOMS DISPROPORTIONATELY PREVALENT IN NEUROLOGY?

Although many who have written about functional symptoms (Thomas Willis, Jean-Martin Charcot, Joseph Babinski, Sigmund Freud, to name but a few) had a neurological background, a comparative study found that functional symptoms were similarly prevalent in other medical specialties.[18] Within neurology, one study found that 9% of 4470 inpatient episodes were provoked by psychogenic disorders,[19] and a second reported that 11% of 300 consecutive neurological outpatient presentations were ''not at all explained'' by organic disease, 19% ''somewhat explained'', 27% ''largely explained'', and 43% ''completely explained''.[1] Several reports suggest that the risk of functional symptoms is increased in patients with physical disorders of the brain, or indeed the peripheral nervous system,[20–22] but (perhaps because of the indistinct conceptual boundaries) the prevalence of functional symptoms in the context of structural or pathophysiologically explained neurological disorders is unknown.

WHAT CAUSES FUNCTIONAL SYMPTOMS?

Recent attempts to explain functional symptoms have focussed on volition,[23] attention,[24] and auto-suggestion.[25] One attractive model suggests that somatisation represents an innate coping mechanism learned from early parent-child interactions.[26 27] The neurobiological substrate of functional symptoms remains elusive, but it is now clear that the brain can physically adapt to environmental challenges and stress,[28 29] so scientific understanding has moved beyond the dualistic concept of separation between mind and brain. Functional or voxel based neuroimaging techniques may bridge the gap between theoretical concepts and biological understanding in the future. However, whilst the studies completed to date are promising, they have been small and their results are difficult to interpret.[30–33]

In the absence of a clear pathophysiological understanding, patients with functional neurological symptoms are perhaps best understood using a multidimensional aetiological model, in line with that proposed for other unexplained somatic disorders.[34] This approach can accommodate biographical factors (like childhood trauma, abuse, life events), relevant biological features (like gender), psychological features (like poor coping styles, the tendency to dissociate, emotional expressiveness), neurological co-morbidity (for example learning disability or epilepsy), social aspects (such as a disturbed family environment, financial insecurity, absence of friends or confidants, limited coping resources), and broader cultural factors (such as attitudes to illness, gender roles). The biopsychosocial model of disease developed in the 1980s, which considers the interplay between all these factors, is a helpful conceptual approach in this context.[35]

The analysis of an individual patient can be informed by a pragmatic distinction between predisposing factors (conferring an increased vulnerability to functional symptoms), precipitating factors (triggering symptoms), and perpetuating factors (contributing to a chronically recurrent course).[36] These factors interact with other psychiatric disorders (like anxiety disorders, posttraumatic stress disorder, depression) and personality traits (especially those typical of borderline and dependent personality disorders).[37] The importance of the various factors differs substantially between patients and may even change in the same patient over time. In assessing the importance of possible aetiological factors it is important to note that the presence of one particular factor (for instance childhood abuse) does not automatically make it aetiologically relevant.

In many patients a history of trauma, a stressful life event, or an ''unspeakable dilemma'' can be identified.[38 39] Often functional disorders seem to be sparked off by a relatively small event which appears to serve as a symbolic reminder of more serious trauma or distress in the past.[40] Even if the identified problem does not seem serious enough to trigger a disabling functional symptom, it may be useful in engaging patients in psychological treatment.

HOW CAN WE DIAGNOSE FUNCTIONAL SYMPTOMS?

The first step in the diagnosis of functional symptoms is the exclusion of a neurologically explained problem. Clinicians have to strike a balance between the pursuit of diagnostic certainty and the fact that continuing investigations may not allow patients to address psychosocial issues and have the potential to cause anxiety and iatrogenic damage. The second, equally important step is the screen for aetiologically relevant psychological or psychiatric features. These are likely to be serious recent life events, but triggers could be more subtle in patients with pre-existing inadequate coping skills. This stress-diathesis model means that explicit psychological criteria for the diagnosis of functional disorder remain elusive although some recent progress has been made.[41]

The clinical signs of functional weakness and sensory disturbance,[42] disorders of gait or stance,[42–44] and non-epileptic seizures[45 46] have been described in detail elsewhere (table 1).

Of course, the clinical tests or investigations which are required to exclude neurological disorders differ between one symptom and another. Before asking for tests clinicians should attempt to review their patient's previous health records to avoid needless replication. What is more, thick notes with presentations to many different specialities can also act as a diagnostic pointer.[47] When requesting investigations clinicians should consider that tests with a low pretest probability of detecting pathology are associated with a high risk of false positive results. Many tests rely on the detection of inconsistency of the clinical problem (apparent in collapsing weakness or the inability to lift a leg off an examination couch whilst being able to walk). Inconsistency also becomes apparent when the quantitatively poor performance of patients with functional disorders in neuropsychological tests is analysed in a qualitative manner.[48 49] In patients with apparent weakness or sensory loss, inconsistency can also be found by demonstrating intact sensory pathways using neurophysiological methods like evoked potentials.[50 51]

Of course, the detection of inconsistency simply raises the possibility that a conventional syndrome does not fit—it does not follow that a functional disorder must be present. Patients with many neurologically explained disorders show marked diurnal variation or variable symptoms.

HOW CERTAIN CAN WE BE THAT A SYMPTOM IS FUNCTIONAL?

Several recent studies have shown that patients whose symptoms are considered neurologically unexplained after appropriate assessment rarely turn out to be diagnosed with a somatic neurological disorder later. However, it should be born in mind that these studies were carried out at specialist

Table 1 Clinical signs associated with functional neurological symptoms[42–46]

Symptom	Clinical sign or observation
Paralysis	Variable loss of function
	Hoover's sign
	Collapsing weakness
	Ipsilateral sternocleidomastoid weakness
	Lack of ipsilateral facial weakness
	Weakness with normal tone and reflexes
Gait and stance	Fluctuating nature
	Dragging of inverted or everted foot
	Excessively slow movement
	Romberg's test with patient falling towards examiner regardless of position of examiner
	Walking as if on ice
	Uneconomical gait
	Giving way of legs with prevention of fall
Sensory	Whole limb anaesthesia
	Hemisensory loss for all modalities to midline
Seizures	Precipitated by stressful situation
	Duration >2 min, tendency to present as seizure status
	Waxing and waning of motor activity or prolonged limp unresponsiveness
	Side to side head movements
	Pelvic thrusting
	Ictal or immediately postictal crying
	Partial responsiveness during seizures
	Closed eyes, resistance to eye opening
	Unexpectedly rapid or slow recovery

centres, and that the risk of a misdiagnosis of disorders characterised by functional symptoms may be higher in less well investigated patients. Stone *et al* found that a neurological explanation had been found in one of 42 patients with functional weakness or sensory loss after a mean of 12.5 years of follow up.[52] Crimlisk *et al* found that only three of 64 patients with functional weakness had developed an identifiable somatic disorder explaining the weakness 6 years after the initial assessment (two out of these three patients were misdiagnosed because they had disorders with variable symptoms).[5 53] Couprie *et al* found an unexpected somatic explanation in four of 56 patients with a functional neurological problem after 1.5–9.5 years.[54]

Although it therefore seems that the finding that a symptom is functional usually stands the test of time, physicians have a tendency to underdiagnose functional problems.[55] This is likely to be one of the reasons why patients with non-epileptic seizures for instance are often misdiagnosed as having epilepsy for many years[56] (fig 2). Several studies have shown that the delay in the diagnosis of functional symptoms is associated with poorer outcome.[53 57 58] Patients do therefore not benefit from a delayed diagnosis but may suffer as a result of it. The misdiagnosis of non-epileptic seizures as epilepsy is particularly dangerous—severe iatrogenic injury, pregnancy loss, and death have been reported.[59–61] Physicians who fail to identify medically unexplained disorders may be faced with litigation.[62]

This does not mean that neurologists should rush into calling a symptom functional. There are also risks of overdiagnosing functional problems.[63 64] In particular, patients with atypical or rare organic disease presentations may not receive appropriate treatment or be denied financial help for incapacity from the government.[65] Once a clinician has diagnosed a problem as functional, patients are less likely to be taken seriously by colleagues or to receive palliative or curative treatment.[66]

In the absence of a diagnostic gold standard, a degree of doubt about a neurologically explained disorder may remain. However, clinicians can usually be sure enough of the functional nature of a symptom to stimulate a search for possible (psychosocial) triggers and associated psychopathology, and to propose psychological forms of treatment.

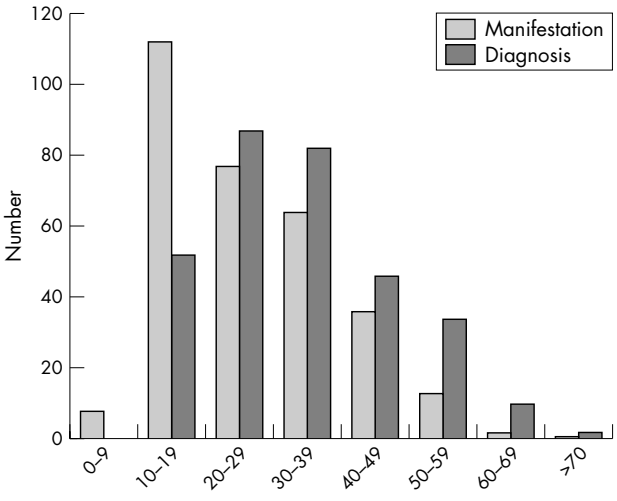

Figure 2 Age at seizure manifestation and correct diagnosis of 313 patients with non-epileptic seizures. Almost all patients were thought to have epilepsy and 75.5% had been treated with anticonvulsant drugs.[56]

WHAT IS THE BEST WAY OF COMMUNICATING THE DIAGNOSIS OF A FUNCTIONAL PROBLEM?

The basic communication skills required for patients with functional symptoms do not differ from those which are helpful with other patients.[67 68] If the initial communication goes well, symptoms settle in a substantial group of patients.[6 69 70] Communication is more likely to be successful if positive diagnostic factors (significant life events, psychiatric features) and negative factors (inconsistencies, negative investigations) have been elicited so that the relevance of psychological factors becomes more believable to the patient.[71] A good way to begin is to enquire what the patient's particular concerns about his symptom are, and what he may have been told by other doctors, so that specific reassurance can be given.

The initial explanation should cover the items listed in table 2. The aims of this conversation include reducing anxiety, instilling a degree of therapeutic optimism, and facilitating engagement in further psychological therapy. Outcome is likely to be better if a positive diagnosis is made (for example, "you have (a) functional weakness/numbness/seizures"), and this diagnosis is explained as much as this is possible (for example, "we are not entirely sure what causes functional weakness/numbness/seizures, but we often see them in patients who are under a lot of stress/who have suffered a loss/who have been badly upset by something…").[72] The physician may pause at this point of the explanation to give the patient a chance to volunteer a relevant traumatic event, conflict, or problem. At no point should the patient be made to feel coerced into making a disclosure, and it should be made clear that the patient should do this only at a time and place with which they feel comfortable. Patients may be probed gently ("I wonder if you recognise any of this in your own life?").

The possible co-occurrence of physical and psychological symptoms should be discussed if applicable (stressing that any separation is quite artificial). It may be helpful to include an educational element at this point to help the patient to understand how emotional factors could be causing physical symptoms. This is best given as a three stage explanation, linking mood, pathogenesis, and symptoms (for example, "When people are anxious the muscles in their neck tend to tense up and that can cause headaches").[73]

Finally, the neurologist should explain the plan for further management. If symptoms are acute, patients may improve with an explanation, encouragement, positive suggestion, and physiotherapy. In more chronic cases, and to anticipate and prevent relapse, a psychological approach should be considered and offered in most cases.

Neurologists will need to make themselves aware of local pathways of referral for psychological treatment. Ideally this should be closely integrated into the neurological diagnostic and therapy service. Finding a suitable therapist may not be easy because there is little evidence that the provision of psychotherapy services in the National Health Service has improved much since it was last investigated in 1996.[74]

The practice of copying medical correspondence to patients (which has been endorsed by recent government policy in the UK) offers a further opportunity to enforce the points which were made during the consultation. The effects of this practice have not been studied in the context of functional symptoms, but they have been studied in psychiatric practice.[75] Results show that there are many reasons why openness could be particularly helpful in this context. Of course, copied letters will cause offence and confusion if they do not reflect the discussion during the consultation.

Table 2 Management of patients with functional neurological symptoms

Management stage	Suggested approach	References and further reading
Initial assessment	Demonstrate that you believe and are interested in symptom and severity Elicit history of other symptoms, previous contacts with health service Find out what patient has been told about his symptom by other doctors Elicit patient's own beliefs about the symptom Screen for significant psychiatric disorder (especially depression and anxiety) Show interest in impact of symptoms on patients' life Ask about life events Obtain history from partner/relative/friend if possible Review previous clinical records if possible Arrange appropriate tests (if necessary)	Craig,[40] Williams and House,[47] Page and Wessely,[67] Creed and Guthrie,[71] Fink *et al*,[106] Morriss *et al*[108]
Communication of diagnosis	Admit uncertainty if investigations incomplete/inconclusive Clarify with the patient how structural disease has been excluded (taking account of patient's specific health concerns) Reframe symptoms (''I can see that since you lost your wife....'') Give a positive explanation of the symptom Convey the potential for substantial recovery Be honest and direct with patents (copying clinic letters is a good way of reiterating important issues)	Page and Wessely,[67] Jackson and Kroenke,[72] Morriss *et al*,[108] Coia and Morley[113]
Acute symptomatic therapy	Discuss potential acute/remote stressors Suggest that symptoms are likely to improve Encourage activity rather than rest/consider physiotherapy	Richardson and Engel[114]
Psychiatric medication	Ask the patient's view (will they take the tablets?) Consider antidepressants even in the absence of overt depressive/anxiety symptoms Explain length of treatment, possibly delayed effectiveness, lack of addictive potential	O'Malley *et al*,[98] Soloff,[99] Stone *et al*[102]
Referral for psychological/ psychiatric assessment	Point out that reducing stress and learning ways of coping with symptoms are useful to all patients regardless of the nature of their symptoms Consider joint appointment Be optimistic but avoid raising expectations to levels which are likely to disappoint	House[115]
Psychological management options	Consider patient held treatment plan, or patient held records Identify goals for treatment Work on identifying predisposing, precipitating, and perpetuating factors Look at potentially problematic patterns in interpersonal relationships Identify ongoing life stressors Identify and address patterns reinforcing abnormal behaviour Reframe and reattribute the links between psychological factors and symptoms Consider the use of specific psychotherapeutic techniques by those with appropriate training (for example, cognitive behavioural and analytical, interpersonal, behavioural psychotherapy) Use appropriate evidence based psychological interventions to treat anxiety and depression if present Discuss relapse prevention Consider goodbye letter to patients on completion of work reinforcing issues discussed and recording progress made.	Goldberg *et al*,[73] Guthrie,[85] Bleichhardt *et al*,[87] Sharpe *et al*,[96] Roth and Fonagy,[97] Fink *et al*,[106] Morriss *et al*[108]

SHOULD PATIENTS WITH FUNCTIONAL SYMPTOMS BE REFERRED TO A PSYCHIATRIST?

Many neurologists are uncomfortable continuing to see patients with functional symptoms. As a consequence, they may get little opportunity to improve their clinical practice in this area. Adult psychiatric services have had to focus on so called severe mental illness (that is, bipolar disorder and schizophrenia), and psychiatrists may therefore also have limited expertise in dealing with these problems. The fact physicians seem unable to address their problem is one of the reasons why many patients with unexplained symptoms keep seeking medical advice.[2]

Ideally, patients with a functional neurological symptoms should be screened for anxiety and depression and should be offered a psychological and psychiatric assessment. Whilst functional disorders do not sit easily within a categorical diagnostic system (see above), there is general agreement that they are a important marker of psychiatric morbidity.[76] In one study of 300 consecutive new patients seen in a neurological outpatient clinic, 67% of patients with unexplained symptoms but only 38% of patients with explained symptoms had depressive or anxiety disorders.[3] In another study, 33% of patients with functional motor symptoms but only 10% of patients in the control group had a major psychiatric disorder.[77] Whereas 13% of patients with unexplained symptoms had seriously considered suicide in the last 2 weeks, only 7% of patients with explained symptoms had done so.[4] These results fit with the observations in other areas of medicine that patients with unexplained symptoms are roughly twice as likely to suffer from psychiatric disorders.[78] Patients with more unexplained symptoms are at greater risk.[79 80]

Ideally the psychiatric assessment should be carried out by a psychiatrist with an interest in this area (or by a neurologist with relevant psychiatric skills). Unfortunately, liaison psychiatry services (and neurological services for patients with functional disorders) remain patchy and a substantial group of patients will decline the option of seeing a psychiatrist.[3] To some patients, a psychologist may be a more acceptable option (especially if they are part of the neurological team). Good communication between different disciplines is essential to avoid patients being given mixed or confusing messages.

WHAT SPECIALIST TREATMENT IS AVAILABLE?

There are insufficient randomised controlled trials in the area of functional neurological symptoms. Evidence from the treatment of other medically unexplained symptoms and similar disorders, however, supports the hypothesis that psychotherapeutic methods may be helpful in some

patients.[81–86] The evidence is perhaps strongest for variants of cognitive behavioural therapy.[87–89] Psychodynamic, interpersonal therapy, and hypnosis have been shown to be effective treatments for irritable bowel syndrome,[90–92] a disorder often comorbid with functional neurological symptoms.[93] Furthermore, biofeedback methods may be useful in the treatment of somatoform disorders.[94]

Little is known about which patients benefit most from which approach. Predisposing and precipitating factors are important in building up an understanding of the patient. However, the mere disclosure of traumatic factors alone is usually not sufficient.[95] Therapy needs to focus especially on factors perpetuating symptoms if it is to be of lasting benefit.[96] It is important to note that the kind of psychotherapy required is not the same as counselling (schooled listening) which is only likely to help in milder (or self limiting) psychiatric disorders.[97]

Antidepressants may be useful in the treatment of patients with functional symptoms. They may even be effective for those without a depressive syndrome.[98] Antidepressants have been shown to reduce emotional dysregulation,[99] a personality trait associated with functional symptoms.[100] One of the most common reasons for non-response to antidepressants is an inadequate dose given for an inadequate length of time.[101] Neurologists should anticipate that nearly three quarters of neurological outpatients think antidepressants are addictive, and nearly half think they can do physical harm.[102] Patients who can see the reasons for taking medication are more likely to complete the course.

Antipsychotic drugs may have a very limited role in the treatment of patients with quasi-psychotic dissociative symptoms resistant to other methods but should be used with caution because of long term side effects.[103]

DO NEUROLOGISTS NEED TO FOLLOW UP ALL THEIR PATIENTS WITH FUNCTIONAL SYMPTOMS?

Rapid discharge from a neurological clinic may be interpreted by the patient as a sign that the neurologist disbelieves him or her. They may then be forced to rely on financial help from the government, or are left dependent on their family or social support network (we know that patients with non-epileptic attacks are more likely to receive financial help from the government than comparable patients with epileptic seizures[104 105]). It is often wise to oversee any transfer of clinical care to psychological or psychiatric services as many patients do not engage easily.

Follow up should satisfy a number of aims: (1) to signal to the patient that his complaint is being taken seriously without the need for a further health crisis; (2) to facilitate engagement in a psychological treatment programme; (3) to reduce the risk of referral for a more palatable specialist opinion; (4) to offer the opportunity to review the diagnosis and ensure no alternative pathology has been missed (especially if new symptoms evolve); and (5) to avoid inappropriate diagnostic re-assignment to an organic disorder.

In practice, only a small number of patients with mixed physical and psychological disorders (for instance epilepsy and non-epileptic seizures) will require long term follow up by a neurologist. For patients with chronic medically unexplained symptoms who fail to improve with a psychological treatment programme the focus may shift to the maintenance of social functioning and minimisation of iatrogenic damage. Programmes for the effective management of such patients in primary care emphasise the regular assessment of patients by a single, identified health practitioner to pre-empt crisis generation, the withdrawal of unnecessary medication and specialist referrals, and the setting of achievable goals (damage limitation rather than cure).[106] As a minimum these programmes can reduce healthcare usage and cost.[107] Some patients may also be open to a process of reattribution. One influential approach to reattribution is based on the three stage process of making the patient feel understood, changing the agenda from a focus on symptoms to a wider view of abilities, and relating symptoms to psychosocial problems.[73 108]

WHAT IS THE PROGNOSIS OF FUNCTIONAL NEUROLOGICAL SYMPTOMS?

Studies of patients with functional paresis or sensory disturbance show that 37–83% of patients (mean age 36–39 years) continued to have symptoms 2–16 years after diagnosis.[52–54 58 109] The study with the longest follow up showed that 29% of patients had retired on medical grounds.[52] Outcome was similarly poor in unselected neurological outpatients with medically unexplained symptoms. Symptoms were worse or persisted in 54% of patients 8 months after the original assessment.[110] The prognosis appears even worse in patients with non-epileptic seizures. The largest study showed that 71% of patients continued to have seizures 11 years after manifestation, and 56% of patients were dependent on social security.[111] Patients who remain unimproved continue to consume healthcare resources. One follow up study of 64 patients with motor conversion symptoms diagnosed at a tertiary neurology referral centre found that 6 years after diagnosis 51% of patients had been re-referred to another neurologist (28% with the same symptom).[112]

There is a danger that new clinicians unfamiliar with a case will relaunch unnecessary investigations or diagnose organic disease which has previously been ruled out. The investigation of the outcome of patients with non-epileptic seizures found that despite advice to discontinue them, 41% of patients were still taking anticonvulsants 5 years later.[111]

Even with psychological treatment, outcome is not good in all patients. A long history of physical symptoms, particularly debilitating symptoms and entrenched support systems which reinforce illness behaviour, predict a poor prognosis. The prognosis is better if patients acknowledge emotional distress in relation to their symptoms and in patients with adequate support who live in a stable social environment. Poor compliance or an unwillingness to engage are likely to result in poor outcome.[85]

CONCLUSION

Functional neurological symptoms are common but poorly understood and frequently mismanaged. After appropriate investigations, clinicians should not be afraid of calling symptoms functional. However, there is a risk of both under and over diagnosis. When neurologists do not follow up such patients themselves diagnostic errors may be perpetuated. The outcome of disorders characterised by functional neurological symptoms is currently poor. Outcomes are likely to improve if the diagnosis of such disorders was sought more actively and communicated more successfully. Whilst many questions about nosology, aetiology, and long term effectiveness of treatment remain open, it is not appropriate to refuse treatment to patients with the biological potential to make a full recovery. Neurologists are well placed to work in collaboration with (liason) psychiatrists, psychologists, and general practitioners to develop treatment programmes for patients with functional neurological symptoms.

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FUNCTIONAL SYMPTOMS AND SIGNS IN NEUROLOGY: ASSESSMENT AND DIAGNOSIS

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It's a Tuesday morning at 11.30 am. You are already 45 minutes behind. A 35 year old woman is referred to your neurology clinic with a nine month history of fatigue, dizziness, back pain, left sided weakness, and reduced mobility. Her general practitioner documents a hysterectomy at the age of 25, subsequent division of adhesions for abdominal pain, irritable bowel syndrome, and asthma. She is no longer able to work as a care assistant and rarely leaves the house. Her GP has found some asymmetrical weakness in her legs and wonders if she may have developed multiple sclerosis. She looks unhappy but becomes angry when you ask her whether she is depressed. On examination you note intermittency of effort and clear inconsistency between her ability to walk and examination on the bed. She has already had extensive normal investigations. The patient and her husband want you to "do something". As you start explaining that there's no evidence of anything serious and that you think it's a psychological problem, the consultation goes from bad to worse….

In this article we summarise an approach to the assessment and diagnosis of functional symptoms in neurology, paying attention to those symptoms that are particularly "neurological", such as paralysis and epileptic-like attacks. In the second of the two articles we describe our approach to the management of functional symptoms bearing in mind the time constraints experienced by a typical neurologist. We also address difficult questions such as: "What causes functional symptoms?", "Are they real?", and "Is there anything that can be done?"

We emphasise the need for a transparent and collaborative approach. As we will explain this depends on giving up a purely "psychological" view of functional symptoms in favour of a biopsychosocial view of causation in which dysfunction of the nervous system is the final common pathway.

SYMPTOMS AND DISEASE

It is important to keep in mind the difference between symptoms and disease. Symptoms, like fatigue, are the patient's subjective experience. Doctors are trained to find a disease, such as multiple sclerosis, to explain the symptoms. When there is no disease it becomes tempting to suggest that the symptom must be "not real" or psychogenic. In fact, symptoms appear for multiple reasons of which disease is only one (fig 1). Symptoms arise from physiological factors (for example, physiological tremor), psychological factors (for example, paraesthesia during a panic attack), behaviours (for example, excessive rest), and cultural or external factors (for example, compensation and the welfare state). For some patients disease pathology is a major (but not the only) factor in causing symptoms and in others it is minor or absent entirely.

A crucial implication of this approach is that the patient does not have to have a "genuine" disease in order to have a "genuine" symptom.

WHAT SHOULD WE CALL THEM?

The large number of terms to describe symptoms unexplained by disease is a reflection of the diverse concepts that have been used to understand them. They include:

- Pure symptomatic labels (for example, chronic fatigue, low back pain)
- Symptom syndromes (for example, chronic fatigue syndrome)
- "Non-diagnoses" that describe what the diagnosis is not rather than what it is (for example, non-epileptic attacks, non-organic, medically unexplained)
- Diagnoses that imply an as yet unestablished disease cause (for example, reflex sympathetic dystrophy)
- Diagnoses that imply an as yet unestablished psychological cause (psychogenic, psychosomatic, "all in your mind")
- Historic diagnoses that do not fit in to any of these categories (for example, "hysteria", "functional")
- "Official" psychiatric diagnoses. These are found in psychiatric glossaries which are rarely used by neurologists and include:

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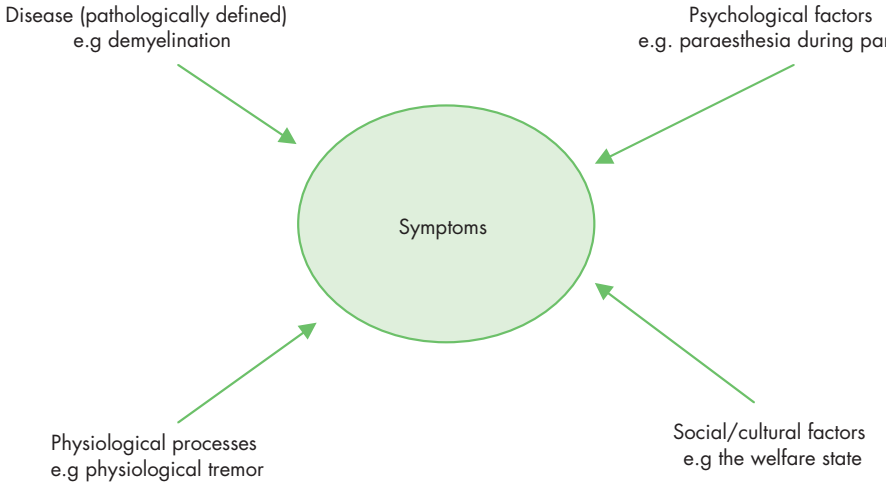

Figure 1 Disease is only one cause of symptoms.

- conversion disorder: a psychoanalytic concept that describes the occurrence of motor or sensory neurological symptoms other than pain and fatigue that cause distress, are not explained by disease, not malingered but are thought to relate to psychological factors
- somatisation disorder (Briquet's syndrome): refers to patients with lifelong functional symptoms including pain, neurological, gastrointestinal and sexual symptoms, again with the implication that psychological problems have been somatised or converted
- dissociative motor disorder: in which dissociation (or a failure of integration of psychological processes) is the putative mechanism
- hypochondriasis: a distressing state of anxiety about disease
- factitious disorder: symptoms consciously simulated in order to gain medical care
- malingering: a term (and not a medical diagnosis) for symptoms which are simulated for clear financial or material gain.

The terminology you use is important. It will not only reflect how you think about the problem, but will also determine the patients' reaction to your diagnosis. For reasons that we explain in the second article we prefer the term "functional" and will use it in these articles.

HOW COMMON ARE FUNCTIONAL SYMPTOMS?

Around one third of new neurological outpatients have symptoms regarded by neurologists as "not at all" or only "somewhat" explained by disease.[1] This finding is not unique to neurology and has also been reported in primary and secondary medical care worldwide. Table 1 illustrates some of the different functional somatic symptoms and syndromes that have been described by various medical specialties. Although superficially disparate, there is substantive overlap in the symptoms, epidemiology, and response to treatments of these functional somatic syndromes.[2]

Contrary to popular belief, even the more dramatic functional symptoms are surprisingly common. The incidence of functional paralysis is probably similar to that of multiple sclerosis (around 5/100 000). Non-epileptic attacks make up around 10–20% of the patients referred to specialist epilepsy clinics with intractable seizures and up to 50% of patients admitted to hospital in apparent status epilepticus.[3] In movement disorders clinics up to 5% of new referrals may have functional symptoms.[4]

FUNCTIONAL SYMPTOMS: WHY BOTHER?

When faced with a clinic full of patients with epilepsy and multiple sclerosis, many neurologists cannot help thinking that patients with functional symptoms should be at the bottom of their priorities. There are a variety of views. Many doctors believe that patients often exaggerate or make up their symptoms in order to gain sympathy or financial benefit. Alternatively, some doctors believe the patients' symptoms, but simply view the problem as "not neurological" and one that should be dealt with by a psychiatrist and not a neurologist.

One argument relates to the patients themselves. When patients with functional neurological symptoms are compared to those whose symptoms are associated with disease, they are found to have similar disability and even more distress.[1] Their symptoms tend to persist at follow up but only rarely become explained by disease. Distress and disability are by their nature subjective but we argue that ultimately it is the subjective that matters most.

The second argument relates to the work of a neurologist. Whether you like it or not, functional symptoms account for one third of your workload. If you allow yourself to become interested in the problem rather than irritated by it, you may find it has an effect on how much you enjoy your job generally.

Table 1 Examples of "functional" somatic symptoms and syndromes from different medical specialities

Neurology	Functional weakness, non-epileptic attacks, hemisensory symptoms
Gastroenterology	Irritable bowel syndrome, non-ulcer dyspepsia, chronic abdominal pain
Gynaecology	Chronic pelvic pain, premenstrual syndrome
ENT	Functional dysphonia, globus pharynges
Cardiology	Atypical chest pain, unexplained palpitations
Rheumatology	Fibromyalgia
Infectious diseases	(Post-viral) chronic fatigue syndrome
Immunology/allergy	Multiple chemical sensitivity syndrome

TAKING A HISTORY FROM SOMEONE WITH FUNCTIONAL SYMPTOMS: A PRACTICAL APPROACH

If you suspect that a patient's presenting symptoms are functional, there are ways of adapting the history to make it more efficient, more interesting for you, and more helpful for the patient. For the patient with functional symptoms, a good assessment is also the beginning of treatment.

''Drain the symptoms dry''

If your patient has a lot of symptoms, begin by making a list of all of them. Here is where you can save most time by resisting the urge to interrogate the features and onset of every symptom as you go. Instead, leave a few lines between each symptom on the list so that you can return to them as required. This allows the patient to unburden themselves quickly of all their symptoms, gives you a broad picture early on, and prevents new symptoms ''cropping up'' late in the consultation. Fatigue, sleep disturbance, memory and concentration problems, and pain can be routinely enquired about at this stage. However, for reasons we will explain, questions about mood are often better left to later.

The more physical symptoms a patient presents with the more likely it is that the primary presenting symptom will not be explained by disease.[2] A long list of symptoms should therefore be a ''red flag'' that the main symptom is functional.

Asking about disability

Ask the patient to describe ''What's a typical day like?''. Follow up questions such as ''How much of the day do you spend in bed?'' and ''How often do you leave the house?'' are more useful than the traditional disability questions about dressing and walking distance. Pay particular attention to *why* they are disabled—for example, someone may have a very mild hemiparesis which really does not impair gait but be very worried about falling which is why they do not go outside.

Finding out more about onset and course

Although you may want to take a detailed history of the course of some symptoms, if a patient has had the symptoms for many years it may be more useful to obtain the overall course of the illness by drawing a graph with time on the x axis and severity on the y axis (fig 2). This can be a quick way of condensing a large amount of information— the line of the graph demonstrates how the illness has gradually worsened, cycled, or perhaps just been static over the period in question. To find the starting point, a useful question is ''When did you last feel well?''. Other events can then be added using arrows—for example, to indicate when the patient stopped working, life events, or medical interventions.

Asking about dissociation

Dissociative symptoms include depersonalisation (feeling detached from oneself) and derealisation (feeling that the world is no longer real) and can be unfamiliar territory for neurologists. However, they commonly occur in patients with neurological disease (such as epilepsy and migraine), in patients with functional symptoms, particularly those with paralysis and non-epileptic attacks, and less commonly in healthy individuals. People find it difficult to describe dissociation and may just say they felt ''dizzy''. The following descriptions give an indication of what sort of thing to look for:

- ''I felt as if I was there, but not there, as if I was outside of myself''
- ''I was spaced out, in a place all of my own''
- ''Things around me didn't feel real, it was like I was watching everything on television''
- ''My body didn't feel like my own''
- ''I couldn't see but I could hear everyone, I just couldn't reply''.

Dissociative symptoms are not diagnostic of a functional problem, but are worth looking for, particularly in patients with functional paralysis or non-epileptic attacks, because:

- they are frightening to patients who are often relieved to discover that the symptom is common and does not indicate ''madness''
- where there is dissociation, there is a reasonable chance of finding that the patient has panic attacks (episodic severe anxiety)
- they can offer an extra way of explaining to patients the link between their experiences and the development of unusual symptoms such as a limb that no longer feels as if its part of them.

What happened with previous doctors?

Ask your patient to tell you about doctors who they saw previously. They may complain bitterly about Dr X or Y who ''didn't listen'' to them or who told them it was ''nothing serious''. You do not need to say whether you agree with Dr X or Y but hearing about this serves two important purposes. Firstly, it can warn you about explanations and treatments that are likely to be rejected. Secondly, by letting the patient talk openly about previous disappointing medical encounters you are showing them that you are interested in their suffering and understand their frustration.

Asking about illness beliefs

What does the patient think is causing their symptoms? What do they think should be done about them? Do they think they are irreversible or reversible? There is evidence that patients with functional neurological symptoms are *more* likely to be convinced that their symptoms are caused by disease than patients whose symptoms are actually caused mainly by disease—perhaps because they are trying to convince others that their symptoms are ''real''. These

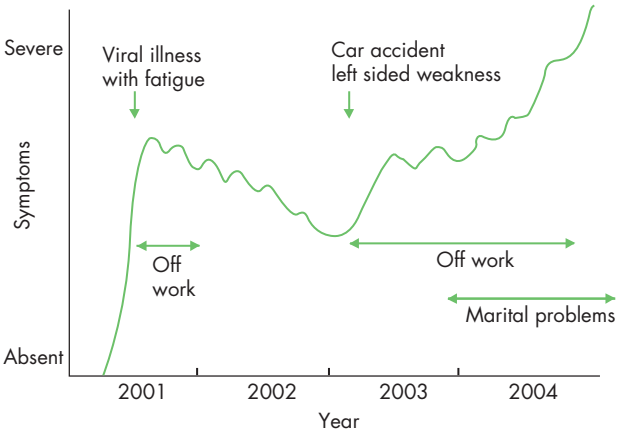

Figure 2 Using a graph to take a history from a patient with functional symptoms.

questions also guide the final explanation. For example, if a patient expresses fear that their symptoms are due to multiple sclerosis a specific explanation of why this is not the case will be needed.

Past medical history: ''get the notes''

Apart from the overall number of symptoms, the other general *diagnostic* red flag is whether there is a history of previous functional symptoms (table 1). The more functional symptoms they have had in the past, the more likely it is that the current symptom is also functional.[2] This reflects the fact that some people are more prone to developing symptoms than others, for reasons we will discuss later. There may also be a history of medical attempts to treat these symptoms with surgical operations (for example, hysterectomy at a young age, appendicectomy, laparoscopy to investigate abdominal pain). Patients may have forgotten previous problems or they may just sense that the more they tell you about previous medical encounters that ended in no diagnosis, the less likely you are to take their current symptoms seriously. That is why you need the notes. Previous psychiatric diagnoses may be particularly unforthcoming in the history. If the patient already has a neurological or other disease diagnosis, ask yourself if the evidence recorded in the notes justifies it—it may not. Alternatively, they may have a disease but one which is insufficient to explain the current symptoms.

Social history: work, money, the law, and marriage

An unpleasant job, being in a ''benefit trap'' (where money received on benefits is comparable to that earned at work), and involvement in a legal case should not be seized on as ''the cause'' of symptoms. This is just another form of unhelpful oversimplification. They could, however, be highly relevant obstacles to recovery. For example, patients with motor symptoms who got married or divorced have been found to do better than those patients whose marital status does not change.

Modelling

A history of similar illness in friends or family or contact with illness through work may lead to another simplistic explanation that the patient is copying or ''modelling'' their symptoms on others. Although plausible, there is little evidence to support or refute the idea that this occurs.

Asking about emotional symptoms: go carefully

Depression, anxiety, and panic are more common in patients with functional symptoms than those with disease. However, asking about psychological symptoms in the wrong way can make the patient defensive because they think that you are about to dismiss them as ''psychiatric''. We therefore suggest that you:

- make sure you have already asked about all the associated ''somatic'' symptoms first—for example, fatigue, poor concentration, poor sleep
- leave questions about emotions until the end of the history
- when you do ask, frame the question in terms of the symptom they are presenting with
- avoid, initially at least, psychiatric terms like depression, anxiety and panic.

For example, instead of ''Have you been feeling depressed?'' try ''Do your symptoms ever make you feel down or frustrated?''. Instead of ''Do you enjoy things any more?'' try ''How much of the time do your symptoms stop you enjoying things?''. When the patient replies that they can't enjoy things because they can't walk, etc, ask them how often they can enjoy the things they can do.

If you suspect your patient has been having panic attacks or is agoraphobic ask ''Do you ever have attacks where you have lots of symptoms all at once? When do these happen? Is it when you're outside or in certain situations?''.

Reading this you may ask yourself: why not just ask the patient directly about depression and anxiety? Many patients, and not just those with functional symptoms, regard anything ''psychological'' as mental weakness, madness, or an accusation that they are ''making up'' their symptoms. Being careful about how you ask questions about psychological symptoms and deferring them to later in the interview allows the patient to gain more confidence in you as a doctor. We find that once a patient trusts you are not going to use emotional symptoms ''against'' them they often will tell you important things they might otherwise not have done.

History of abuse: to ask or not to ask?

Childhood abuse and neglect is another factor that makes people more prone to functional symptoms. But unless you have a long time to spend with the patient or they volunteer the information, we would suggest leaving questions about early life experiences and abuse until subsequent consultations (or to someone else). The evidence from primary care currently does not support the idea that quickly ''getting to the bottom of things'' in this way improves outcome.

How long should all this take?

Like surgery, there is a limit to how quickly this can be done in a very complicated patient, even with the efficiencies we have suggested. Doing it in 10 minutes may be worse than not doing it all.

EXAMINATION

The diagnosis of motor and sensory symptoms discussed below depends on demonstrating positive functional signs as well as the absence of signs of disease.[5] Most of these signs relate to inconsistency, either internal (for example, Hoover's sign reveals discrepancies in leg power) or external (for example, tubular field defect is inconsistent with the laws of optics).

When considering functional motor or sensory signs remember that:

- inconsistency is evidence that signs are functional, but does not tell you whether they are consciously or unconsciously produced
- the presence of a positive functional sign does not exclude the possibility that the patient also has disease—they may have both
- all physical signs have limited sensitivity, specificity, and inter-rater reliability.

GENERAL SIGNS

La belle indifférence

''La belle indifférence'', an apparent lack of concern about the nature or implications of symptoms or disability, is a clinical feature that continues to receive prominence in standard descriptions of conversion disorder. However, it has no discriminatory value. Furthermore, in our experience most

patients who are said to have ''la belle indifférence'' are either: (1) making an effort to appear cheerful in a conscious attempt to not be labelled as depressed; or (2) factitious (because they are deliberately making up the symptom they are not concerned about it).

The laterality of the symptoms

Although often considered left sided, a recent systematic review found only a slight left sided preponderance (55–60%) for functional motor and sensory symptoms.

FUNCTIONAL WEAKNESS

Preliminary observation

Look for evidence of *inconsistency*. For example, compare their gait when they leave the consulting room to when they came in? What happens to their weakness when they have to take their clothes on or off or when they have to get something from their bag?

Hoover's sign and other tests of ''complemental opposition''

Hoover's sign, described in 1908, is the most useful test for functional weakness and the only one that has been found in controlled studies to have good sensitivity and specificity.[6] It is a simple, repeatable test, which does not require skilled or surreptitious observation. The test relies on the principle that we extend our hip when flexing our contralateral hip against resistance (you can test this out on yourself). It can be performed in two ways:

- *Hip extension*—Look for a discrepancy between voluntary hip extension (which is often weak) and involuntary hip extension (which should be normal) when the opposite hip is being flexed against resistance (fig 3). It is important when testing involuntary hip extension to ask the patient to concentrate hard on their good leg.
- *Hip flexion*—Test hip flexion in the weak leg while keeping your hand under the good heel. Look for the absence of downward pressure in the good leg.

A similar principle can be used to examine weakness of hip abduction which may initially be weak but then come back to normal if tested simultaneously with the ''good side''.

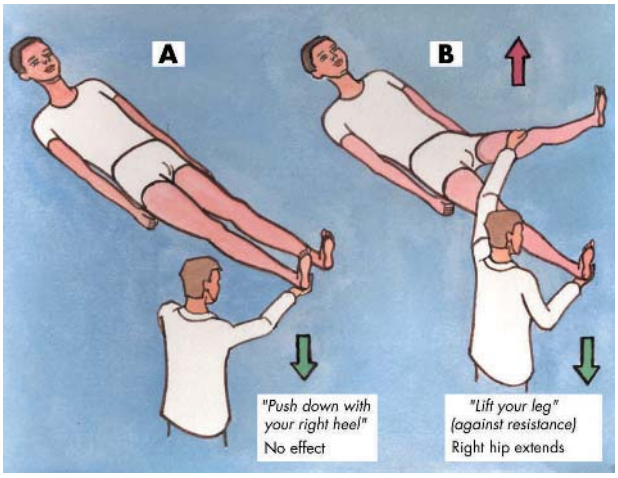

Figure 3 Hoover's sign. (A) Hip extension is weak when tested directly. (B) Hip extension is normal when the patient is asked to flex the opposite hip. Reproduced from Stone *et al*,[5] with permission of the BMJ Publishing Group.

These tests, although useful, should be interpreted cautiously for the following reasons:

- Pain in the affected hip may produce greater weakness on direct, compared with indirect, testing as a result of attentional phenomena (related to pain rather than weakness)
- Cortical neglect can cause a positive Hoover's sign
- The test may be mildly positive in normal individuals because of a splinting effect
- None of the studies testing its utility were blinded and none mention the problem of neglect.

Collapsing weakness

''Collapsing weakness'', the phenomenon in which a limb collapses from an instructed position with a light touch, is a common finding in patients with functional weakness. It is often associated with power that comes and goes or ''intermittency''. This should be not be described as ''intermittency of effort'' since you cannot directly assess someone's effort. Normal power can often be achieved transiently with encouragement, for example by saying to the patient, ''At the count of three, stop me from pushing down…''. Alternatively, gradually increase the force applied to the limb starting gently and building imperceptibly up to normal force.

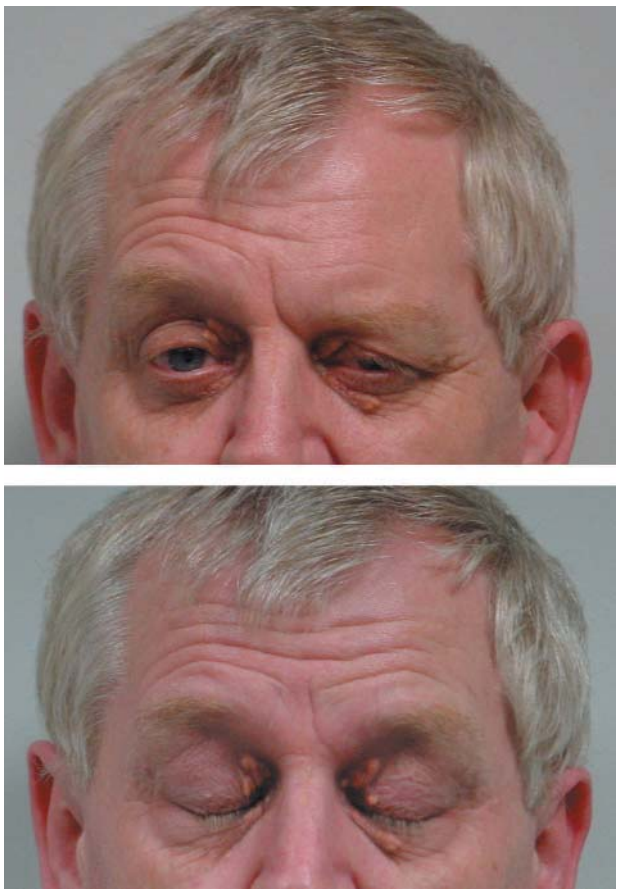

Figure 4 Pseudoptosis. This man presented with photophobia and difficulty elevating the right side of his forehead. The photograph shows his normal resting state (upper panel) and normal movement of his forehead with his eyes shut (lower panel). There is overactivity of his orbicularis oculis which had been incorrectly interpreted as ptosis. It improved with gradual exposure to light. Reproduced from Stone,[17] with permission of Blackwells Publishing.

An inability to understand the instruction, pain in the relevant joint, being generally unwell, and a misguided eagerness of some patients to "convince the doctor" may cause a false result. These concerns have been vindicated in the small number of validity studies of this sign which have found that it is a rather poor discriminator between functional and disease related symptoms.[7]

Functional weakness of the face, pseudoptosis, and "wrong way tongue deviation"

Organic unilateral ptosis is usually associated with frontalis overactivity, whereas in pseudo-ptosis a *persistently* depressed eyebrow with a variable inability to elevate frontalis, overactivity of orbicularis, and photophobia is characteristic (fig 4). Apparent functional weakness of the lower half of the face and tongue deviation towards the normal rather than paretic side may occur because of overactivity of the affected side rather than underactivity.

Other signs of functional weakness

- "Co-contraction" describes the contraction of an antagonist muscle—for example, triceps, when the agonist muscle, biceps, is being tested.
- When carrying out the "arm-drop", look for an unusually slow and jerky descent of the arm from an outstretched position on to the lap (better and less aggressive than dropping the arm on to the patient's face).
- Occasionally when the "arm-drop" test is performed the arms remain inexplicably elevated, so called "pseudo waxy flexibility", a phenomenon akin to that seen under hypnosis.
- It may be worth examining the strength of the sternocleidomastoid which is rarely weak in disease but may often be weak in unilateral functional weakness.

Using sedation/hypnosis

In the altered mental state induced by sedative drugs or hypnosis, patients with functional weakness may begin to move their limbs normally again. Showing a video recording of this to the patient can be helpful in demonstrating to them the potential for reversibility.

Important absent signs in functional weakness

Although the conventional examination of tone and reflexes should be normal, pain may increase tone, anxiety can increase reflexes, and in the patient with unilateral symptoms there may be mild reflex asymmetry, particularly if there is attentional interference from the patient. Pseudoclonus can occur, with irregular and variable amplitude. The plantar response should not be upgoing, but do not be surprised if the plantar response is mute on the affected side in functional weakness, particularly if there is pronounced sensory disturbance.

FUNCTIONAL SENSORY DISTURBANCE

Functional sensory disturbance may be reported as a symptom or may be detected first by the examiner. While a number of functional sensory signs have been described, none appear to be specific and they should not therefore be used to make a diagnosis.

Demarcation at the groin or shoulder

Patients may describe sensory loss that ends where the leg or arm ends, at the shoulder or groin.

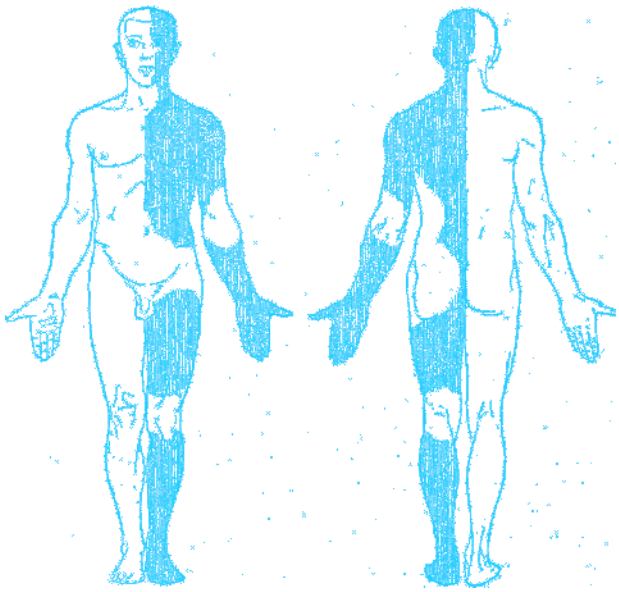

Figure 5 Hemisensory disturbance. From *Charcot's clinical lectures on diseases of the nervous system*, volume 3.[18]

The "hemisensory syndrome", midline splitting, and splitting of vibration sense

The hemisensory syndrome has been described for over a century and continues to be a well known but rarely studied clinical problem in neurology (fig 5). The intensity of the sensory disturbance often varies, and while it may be complete it is usually rather patchy, but with a distinct complaint from the patient that something is "not right" down one side or that they feel "cut in half".

Patients with hemisensory disturbance frequently complain of intermittent blurring of vision in the ipsilateral eye (asthenopia) and sometimes ipsilateral hearing problems as well. Hemisensory symptoms are increasingly recognised in patients with chronic generalised and regional pain.

"Midline splitting", the exact splitting of sensation in the midline, is said to be a functional sign because cutaneous branches of the intercostal nerves overlap from the contralateral side, so organic sensory loss should be 1 or 2 cm from the midline. However, midline splitting can also occur in thalamic stroke. Therefore the finding of reversible contralateral thalamic and basal ganglia hypoactivation using single photon emission computed tomography (SPECT) in patients with unilateral functional sensory symptoms is intriguing in relation to this sign.[8]

Similarly, patients with disease should not report a difference in the sensation of a tuning fork placed over the left compared to the right side of the sternum or frontal bone, as the bone is a single unit and must vibrate as one. Studies of both midline splitting and splitting of vibration sense have found they are common in patients with disease and so cannot be recommended.[7]

Tests involving doctor trickery

If you ask a patient to "Say 'Yes' when you feel me touch you and 'No' when you don't" they may indeed say "no" in the affected area. The problem in interpreting this test is firstly that the patient may be using "no" to mean "not as much", and secondly many patients will work out (at least in hindsight) that they were being tricked. This makes this test

Table 2 Attack features that can help to distinguish non-epileptic attacks from epileptic seizures. Reproduced from Reuber and Elger,[3] with permission

Observation	Non-epileptic seizures	Epileptic seizures
Situational onset	Occasional	Rare
Gradual onset	Common	Rare
Precipitated by stimuli (noise, light)	Occasional	Rare
Undulating motor activity	Common	Very rare
Asynchronous limb movements	Common	Rare
Purposeful movements	Occasional	Very rare
Rhythmic pelvic movements	Occasional	Rare
Opisthotonus, ''arc de cercle''	Occasional	Very rare
Side-to-side head shaking	Common	Rare
Tongue biting (tip)	Occasional	Rare
Tongue biting (side)	Rare	Common
Prolonged ictal atonia	Occasional	Very rare
Ictal crying	Occasional	Very rare
Closed mouth in ''tonic phase''	Occasional	Very rare
Vocalisation during ''tonic–clonic'' phase	Occasional	Very rare
Closed eyelids	Very common	Rare
Convulsion >2 minutes	Common	Very rare
Resistance to eyelid opening	Common	Very rare
Pupillary light reflex	Usually retained	Commonly absent
Reactivity during ''unconsciousness''	Occasional	Very rare
Lack of cyanosis	Common	Rare
Rapid postictal reorientation	Common	Rare

unhelpful if you want to adopt the transparent approach we favour.

NON-EPILEPTIC ATTACKS

There is a stronger evidence base for approaching the diagnosis of functional/non-epileptic attacks or pseudoseizures.[3] As for functional weakness, the history may be suggestive, but will usually not be in itself diagnostic.

Semiology

Non-epileptic attacks vary widely in their semiology but have been broadly divided into hyperkinetic/thrashing attacks and akinetic/motionless attacks. Table 2 lists some of the signs which have been tested in studies of both patients with non-epileptic attacks and epilepsy. In our experience symptoms of panic and dissociation are common in the prodromal phase, although patients may be reluctant to describe them.

As table 2 shows, there are no clinical signs of non-epileptic attacks which *never* occur in epilepsy, and apart from ictal electroencephalogram (EEG) abnormalities, there are no signs unique to epilepsy. For this reason, it is dangerous to use any of the listed signs in isolation to make a diagnosis.

There is a wide differential diagnosis for attacks that look ''odd''. ''Strangeness'' in itself should not lead you to a diagnosis of pseudoseizures. Frontal lobe seizures can look particularly bizarre. Paroxysmal movement disorders are another potential catch.

Prolactin measurement

Serum prolactin is often elevated 15–20 minutes after a generalised tonic–clonic seizure and should be normal after a non-epileptic attack. However, prolactin rise has been demonstrated after syncope and found to be normal after partial seizures. The test can be useful but in our experience it is often carried out badly in practice, with no baseline sample and a post-ictal specimen that is either measured too early or too late. For this reason, we do not advocate its use outside specialist units.

EEG and videotelemetry

EEG with videotelemetry remains the ''gold standard'' investigation for non-epileptic attacks. However, patients with partial epilepsy, particularly frontal lobe epilepsy, may not show any abnormalities on surface EEG recording when there is a deep ictal focus. In addition, some patients may not have attacks during monitoring.

Using placebo and suggestion to induce attacks

The use of intravenous placebo, such as giving a bolus of intravenous saline with the suggestion that it will bring on an attack, is controversial as it may involve deception by the doctor (depending how the procedure is explained to the patient). Verbal suggestion alone may be effective.[9]

FUNCTIONAL OR ''PSYCHOGENIC'' MOVEMENT DISORDERS

The diagnosis of a functional movement disorder is particularly challenging because of the unusual nature of some organic movement disorders. This is illustrated in the disproportionate number of movement disorders in cases where structural disease has been misdiagnosed as functional.

Further description of the features below can be found elsewhere[4] and useful video material can be found accompanying a recent textbook of movement disorders.[10]

There are some general features common to all functional movement disorders. These include:

- *Rapid onset*—This is more unusual in patients with organic movement disorder.
- *Variability*—Variability in frequency, amplitude, or distribution may be obvious during an examination or during observation at other times. It must be remembered that all movement disorders vary to some degree and will get worse during times of stress or worry, so minor variability is not helpful.
- *Improvement with distraction*—Distracting tasks include asking the patient to perform tests of mental concentration (for example, serial subtraction) or physical tasks

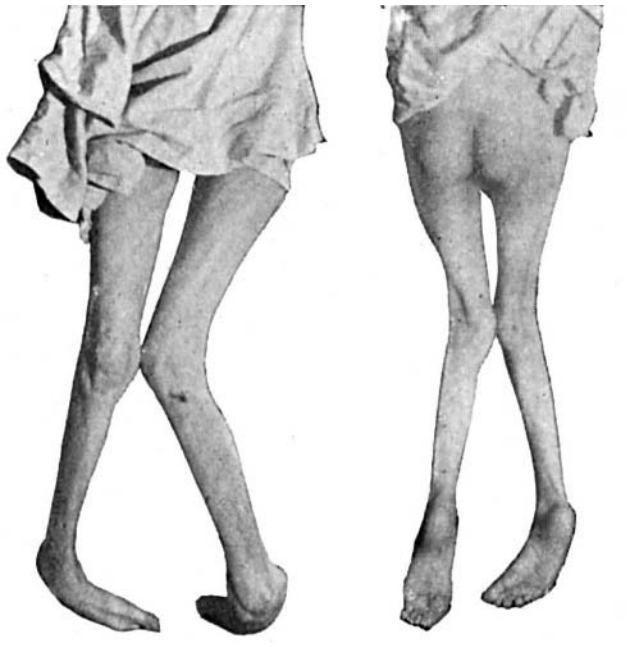

Figure 6 A patient with paraplegia and psychogenic/functional dystonia of 14 years duration before (left and middle panels) and after (right panel) treatment with psychotherapy. Reproduced from Purves-Stewart and Worster-Drought.[19]

with their normal limbs (such as rapid alternating hand movements). The inverse, worsening with attention, may also occur. Again, organic movement disorders may be susceptible to these factors to a degree.

Tremor

Entrainment

When testing for entrainment, a type of distraction task, the patient is asked to make a rhythmical movement with their normal hand or foot. Either the normal limb ''entrains'' to the same rhythm as the abnormal side or, more commonly, the requested rhythmical movement is irregular or incomplete. There is reasonable evidence for the reliability of this test from several controlled studies. A tapping frequency of 3 Hz may be more discriminant, and produce more variation, than a faster 5 Hz rate.

Tremor amplitude change with weights/co-activation sign

When weights are added to the affected limb, patients with functional tremor tend to have greater tremor amplitude whereas in those with organic tremor the tremor amplitude tends to diminish. This may be because of co-activation of agonists and antagonist, the so-called ''co-activation sign''. Related to this, patients with functional tremor may shake their limb more vigorously if it is held still.

Dystonia

Patients with hysterical contracture have been described since the late 19th century alongside organic dystonia (fig 6). Psychodynamic interpretations of dystonia (such as torticollis representing a ''turning away from responsibility'') encouraged misdiagnosis. When this error was realised there was a backlash and the diagnosis of psychogenic dystonia almost disappeared. More recently it is being recognised again and is included in the spectrum of ''fixed dystonia''.[11] The diagnosis is difficult but useful features include: an inverted foot or ''clenched fist'' onset in an adult, a fixed posture which is apparently present during sleep, and the presence of severe pain.

The ''gold standard'' for the diagnosis of functional dystonia is to demonstrate complete remission after administration of general anaesthesia, a suggestion, or placebo. Such a procedure, if handled carefully, may also be therapeutic. Be aware, however, that some types of organic dystonia may also remit spontaneously. A high proportion of patients with psychogenic dystonia have had an injury to the affected limb. There is an overlap between dystonia seen in relation to complex regional pain and psychogenic dystonia.

Other movement disorders

Psychogenic myoclonus is described as a myoclonus with variable amplitude and frequency. It may be strikingly stimulus sensitive—for example, to fluorescent lighting or with elicitation of deep tendon reflexes—in which case the latency between stimulus and jerk is often long and variable. Laborious research methods may demonstrate the presence of a ''Bereitschaftspotential'' one second before the jerk whereas in cortical myoclonus of organic origin there may be a cortical spike around 20 ms before the movement.

Psychogenic hemifacial spasm, parkinsonism, and paroxysmal movement disorders (some of which are like ''partial'' non-epileptic attacks) are also described.[4]

FUNCTIONAL GAIT DISTURBANCE

Several case series describe the features of functional gait disturbance[12] including one with video recordings.[13] Variability and improvement with distraction are noted but, as with movement disorders, just because a gait looks ''bizarre'' or ''ridiculous'' does not mean it is functional.

Unilateral functional weakness of a leg, if severe, tends to produce a characteristic gait in which the leg is dragged behind the body as a single unit, like a ''log'' (fig 7). The hip is either held in external or internal rotation so that the foot points inwards or outwards. This may be associated with a tendency to haul the leg on to an examination couch with both hands.

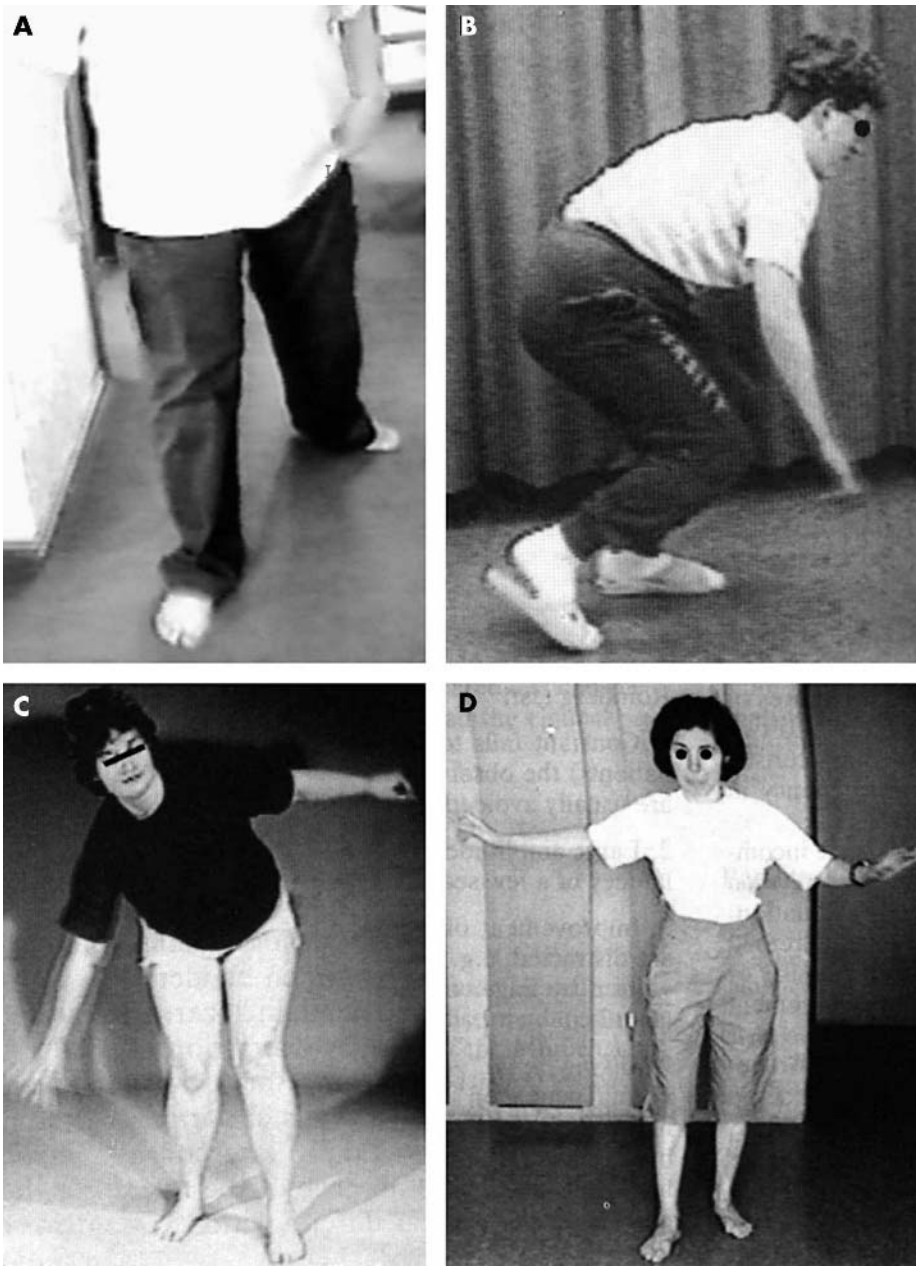

Figure 7 Functional gait disorders. (A) Dragging monoplegic gait. (B) Uneconomic posture. (C) Pseudoataxia. (D) ''Walking on ice'' gait. Fig 7A reproduced from Stone *et al*,[5] with permission of the BMJ Publishing Group. Fig 7B–D reproduced from Lempert *et al*,[12] with permission of Karger Publishing.

Other features suggestive of a functional gait (fig 7) include:

- *Excessive slowness*—Dramatic delay in gait initiation and subsequent ''foot-sticking'' without the subsequent improvement seen in extrapyramidal disorders.
- *Falling towards or away from doctor*
- *''Walking on ice'' pattern*—The gait pattern of a normal person walking on slippery ground. Cautious, broad based steps with decreased stride length and height, stiff knees and ankles. Arms sometimes abducted as if on a tightrope.
- *Uneconomic postures with waste of muscle energy*—A gait with an eccentric displacement of centre of gravity such as standing and walking with flexion of hips and knees. Often associated with fear of falling.
- *Sudden knee buckling*—Patients usually prevent themselves from falling before they touch the ground. Knee buckling can occur in Huntington's chorea and cataplexy.
- *Pseudoataxia*—A gait characterised by crossed legs with or a generally unsteady gait with sudden sidesteps.

OTHER SYMPTOMS

A brief summary of other symptoms (excluding cognition, pain, and fatigue) is given here mainly in order to direct the interested reader to the relevant literature

Dizziness

A full discussion of how to determine whether dizziness is predominantly functional, and indeed whether such a distinction can be made, can be found elsewhere.[14] A variety of terms have been used to describe the intersection of vestibular and psychogenic factors in dizziness including, phobic postural vertigo, ''excessive awareness of normal sensation'', and space and motion discomfort. Some key points are:

- Anxiety and phobic avoidance of situations or head positions that bring on dizziness does not necessarily indicate a ''psychogenic'' aetiology
- On the other hand, such phobic avoidance may continue after the initial pathology has resolved
- Panic attacks presenting somatically with dizziness should be considered in the differential diagnosis of dizziness—look for a fear of embarrassment and inability to escape from situations in which it is likely to occur, such as supermarkets, as well as for other autonomic symptoms
- Physiological vestibular sensitivity to particular visual stimuli such as patterned lines or bright lights (sometimes called visual vertigo) may lead to symptoms that also come on in crowded places
- Depersonalisation and derealisation may be described by the patient as ''dizziness''. If this sensation is there all the time, the patient may have depersonalisation disorder (a chronic form of dissociation)
- Asking the patient to hyperventilate to see if that reproduces the symptoms might appear straightforward, but it has a high false positive rate in patients with dizziness cause by disease.

A full assessment of vestibular abnormalities, provoking stimuli and emotional symptoms can lead to tailored treatment in the form of vestibular rehabilitation and/or a cognitive behavioural approach regardless of the aetiology.

Speech and swallowing symptoms

Typically, functional dysarthria resembles a stutter or is extremely slow with long hesitations that are hard to interrupt. The speech may be telegrammatic consisting only of the main verbs and nouns in a sentence. In its extreme form the patient may become mute. Be careful though, as these types of speech disturbance can also be seen in patients with disease.

Word finding difficulty is a common symptom in anyone with significant fatigue or concentration problems and may compound a functional dysarthria. True dysphasia as a more severe functional symptom, however, is rare.

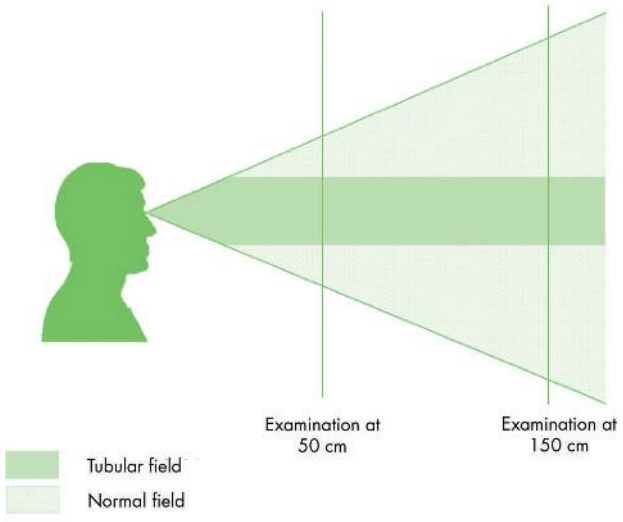

Figure 8 A ''tubular'' field deficit is inconsistent with the laws of optics and eye physiology. You can detect striking tubular field at the bedside.

Dysphonia is a much more common functional speech complaint and there is now quite a large literature outlining approaches to diagnosis and management.[15] Often the clinical presentation is of whispering or hoarse speech that is initially thought to be laryngitis by the patient but then persists for months or years. The possibility of spasmodic adductor or abductor dysphonia must always be considered.

Globus pharyngis or functional dysphagia is common and there is also a sizeable literature about it. The patient normally complains of a sensation of a ''ball in the throat'' and investigations do not reveal a cause. There is controversy regarding what constitutes a full set of investigations for this symptom.

Visual symptoms

Intermittent blurring of vision that returns to normal if the patient screws up their eyes tight then relaxes them again is commonly reported. Some of these patients have convergence or accommodative spasm, with a tendency for the convergence reflex to be transiently overactive, either unilaterally or bilaterally. In this situation lateral gaze restriction can sometimes appear to be present, but the presence of miosis may help to confirm the diagnosis. Voluntary nystagmus is described and appears to be a ''talent'' possessed by around 10% of the population.

Tests for functional visual acuity problems are described in detail elsewhere.[16] Simple bedside tests for a patient complaining of complete blindness are to ask them to sign their name or bring their fingers together in front of their eyes (which they should be able to do). They may have a normal response to menace and optokinetic nystagmus with a rotating drum (which equates to acuity of greater than 6/60). Decreased acuity in one eye can be assessed with a ''fogging test'' in which ''plus'' lenses of increasing power are placed in front of the ''good'' eye until the patient can only be using their ''bad'' eye to see.

Spiral or tubular fields are commonly seen clinically, are often asymptomatic, and can be elicited at the bedside. Remember to test the visual fields at two distances when looking for a tubular field (fig 8). Patients with functional hemianopia have been described who have homonymous hemianopia with both eyes open and then, inconsistent with this, have a monocular hemianopia in one eye with full fields in the other eye. Monocular diplopia or polyopia may be functional but can be caused by ocular pathology.

Auditory symptoms

Basic tests for deafness rely on a startle response such as making a loud unexpected ''clap'' out of sight of the patient. Auditory brainstem evoked responses or evoked otoacoustic emissions may be necessary to fully investigate a patient with this symptom.

INVESTIGATIONS

Even after finding clear positive evidence of functional symptoms, investigations are necessary in many (but not all) patients. Our criteria for performing tests are either (1) we are uncertain of the diagnosis, or (2) the patient remains uncertain of the diagnosis even though we are (and have done our best to explain it to them). Some patients really do not want tests; they just want a confident opinion. Others are only interested in the opinion of the scanner. As a general rule of thumb, if you are carrying out investigations to convince or reassure the patient, remember that this may

only be temporarily effective in patients with severe health anxiety who can become "addicted" to the reassurance of investigations. Similarly, patients who are convinced they have a certain disease like multiple sclerosis, but are not in the least anxious about this possibility, will not necessarily accept a negative investigation anyway. In a sizeable number of patients, normal investigations will be helpful and can speed recovery.

Preferably investigations should be performed as quickly as possible, as protracted testing maintains a focus on looking for disease rather than on rehabilitation. The need to look for disease also needs to be balanced against the risk of uncovering laboratory or radiological abnormalities that have nothing to do with the symptoms but which may delay or disrupt positive management. If tests are abnormal and relevant then positive functional signs should not necessarily be ignored. It may be necessary to make two diagnoses—one of an organic disease such as multiple sclerosis and another of additional functional weakness or disability.

CONCLUSIONS: ASSESSMENT AND DIAGNOSIS

Functional symptoms are one of the most common reasons for consulting a neurologist. The assessment of such patients can be made more efficient and interesting by adapting the history, obtaining all the symptoms early on, asking about illness beliefs, and being careful about how and when you ask about psychological symptoms. In making the diagnosis the presence of positive functional signs are of key importance but should be used cautiously. Finally, be prepared to make a diagnosis of additional functional disability in someone with a known organic disorder.

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Motor Conversion Symptoms and Pseudoseizures: A Comparison of Clinical Characteristics

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The authors prospectively studied consecutive neurological inpatients with either motor conversion symptoms or pseudoseizures of recent onset. Patients were administered a structured psychiatric diagnostic interview, a measure of perceived parental care, and a life events inventory. They found that patients with pseudoseizures (N = 20, mean age = 27 years): 1) were younger than patients with motor conversion symptoms (N = 30, mean age = 39 years), 2) were more likely to have a borderline personality disorder), 3) were more likely to have a lower perception of parental care and to report incest, and 4) reported more life events in the 12 months before symptom onset. These differences in their characteristics and associated factors raised the question of whether it is helpful to group patients with pseudoseizures and motor conversion symptoms in a single diagnostic category of conversion disorder. An alternative view, that gives primacy to the symptoms rather than a disorder, may enable more precise research questions to be posed.

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Conversion disorder is defined in DSM-IV as the occurrence of symptoms that mimic neurological disease (paralysis, seizures, and sensory disturbance but not pain, fatigue, or sexual dysfunction) that are not intentionally produced and do not occur exclusively in the course of somatization disorder. The definition also states that 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.[1] Conversion disorder remains a contentious diagnosis. It may be regarded as a refuge, both for patients with pseudoneurological symptoms who do not fulfil criteria for somatization disorder and for psychodynamic concepts in the DSM-IV. Unlike somatization disorder, there have been few studies of its diagnostic stability and reliability and none to our knowledge formally examining the rationale for the selection and grouping of paralysis, seizures, and sensory disturbance (excluding pain) in their own single category.

Studies of conversion disorder have either concentrated on individual symptoms such as dysphonia or pseudoseizures or have grouped various pseudoneurological symptoms as a single entity. As far as we are aware, no previous study has compared clinical characteristics and associations in patients with different types of conversion symptoms taken from the same patient population.

This study is of patients recruited from neurological centers who had only recently developed their symptoms for the first time. One group had weakness (paresis or paralysis of a limb) that was unexplained by neurological

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disease (that in this article we will refer to as "motor conversion symptoms") and the other had seizures that were unexplained by neurological disease (that we will refer to as "pseudoseizures"). The groups were compared on demographic variables, psychiatric diagnoses, and symptoms and personality disorder. We also compared the strength of association with the proposed etiological factors of adverse childhood experiences and recent life events.

The hypotheses tested were generated before data analysis from clinical experience and previous studies. These hypotheses were that patients with pseudoseizures, when compared to those with motor conversion symptoms, would 1) be more likely to be female,[2–10] 2) be younger in age,[4,7,10–16] 3) be more likely to have a borderline personality disorder,[17–19] 4) be more likely to report sexual abuse or problems in childhood,[20,21] and 5) report more recent life events.[22,23] We measured psychiatric disorder (DSM-IV, axis I) but did not expect to find a difference between groups in its prevalence.[24] We also examined the extent to which motor conversion symptoms and pseudoseizures occurred simultaneously in the same patient and the frequency of pain in both groups.

METHOD

Setting

The patients with motor conversion symptoms and those with pseudoseizures in this study have been previously described.[25,26] All patients were recruited over the same time period from two hospitals: the Department of Neurology at Umeå University Hospital in northern Sweden and the neurological section of the Department of Internal Medicine at the county hospital of Kalmar in the south of Sweden. Both hospitals have primary catchment areas of approximately 130,000 inhabitants, but Umeå University Hospital also offers neurological services to a secondary catchment area with a population of around 800,000 people.

Recruitment and Selection

In Umeå, the period of recruitment lasted 24 months for patients with motor symptoms and 48 months for patients with seizure disorders, whereas in Kalmar, the inclusion period lasted 22 months for both patient groups. The selection procedure at the two sites was identical. Non-Swedish speakers were excluded.

Consecutive patients with motor symptoms or seizures who were admitted to the in-patient wards of the two hospitals and fulfilled DSM-IV criteria for conversion disorder were considered for inclusion in the study. The diagnoses in all cases were confirmed by at least two different neurologists. Patients with concomitant somatic diseases other than neurological illnesses and patients with concomitant psychiatric syndromes other than somatization disorder were included. Patients with both conversion disorder and known neurological disease were excluded, as were patients with probable conversion disorder in which a neurological cause was judged unlikely but difficult to rule out completely.

Only patients with paresis or paralysis of less than 3 months' duration were included in the group with motor conversion symptoms. Patients with a psychogenic movement disorder or psychogenic gait disturbance were excluded. Patients with disease that could have caused their motor symptoms were excluded. Patients were assessed for the presence of disease by means of clinical assessment, including relevant radiological, neurophysiological, and biochemical investigations. The level of investigation depended on the certainty of the clinical presentation. However, all patients underwent neuroradiological investigations with computerized tomography or magnetic resonance imaging.

Only patients seen less than 12 months after the first pseudoseizure were eligible for inclusion in this group. Patients with pseudoseizures who also had epilepsy were excluded. Patients were assessed for the presence of epilepsy with long-term in-patient closed-circuit video and EEG monitoring, and those with a normal EEG during a clinical seizure were included.

Measures

Background information concerning previous medical and psychiatric disorders was collected by interview and by a review of hospital records. Information about previous medical and psychiatric disease among relatives was also obtained by a standardized interview. The research assessment was conducted during the patients' hospitalization by an interviewer who was blind to the specific hypotheses and consisted of the following.

Psychiatric diagnoses were determined with the Structured Clinical Interview (SCID) for Diagnostic and Statistical Manual for Mental Disorders (DSM-IV): the SCID-I for clinical syndromes and the SCID-II for personality disorders. These are structured clinical interviews linked to the DSM-IV diagnostic system.[27] They provide suggested wording for questions and criteria for judging the patient's

response but also allow for clinical judgment in interpreting whether the patient's responses meet the criteria. They have been shown to generate reliable diagnosis when used by trained clinicians.[28] The SCID-II interview covers the 11 personality disorder (axis II) diagnoses. The instrument has been validated against "longitudinal expert evaluation using all data"[29] and has high test-retest and interrater reliability.[30]

Patients also scored their level of psychological, social, and occupational functioning over the previous year according to axis V of DSM-IV by means of a validated self-report version of the Global Assessment of Functioning (GAF) scale.[31]

Perceived parental rearing practices were assessed by means of the Egna Minnen Beträffande Uppfostran (My Memories of Upbringing) self-rating inventory,[32] which is based on 12 qualities of child-rearing experiences: abusive, depriving, punitive, shaming, rejecting, overprotective, overinvolved, tolerant, affectionate, performance orientated, guilt engendering, and stimulating. A total of 81 questions can be answered in four ways for the father and for the mother separately: 1) it never occurred, 2) it could occur but it was exceptional, 3) it occurred quite frequently, 4) it was always so. A factor analysis has extracted three principal components: emotional warmth, rejection, and overprotection. The Egna Minnen Beträffande Uppfostran has been demonstrated to be highly convergent[33] in content to the widely used Parental Bonding Instrument,[34] which has only two dimensions (care and control/overprotection).

The occurrence of life events 12 to 4 months before and within 3 months of the onset of the symptom were assessed by using a 56-item life events inventory elicited by semistructured interview.[6,35] Life events were sorted into events concerning 1) work, 2) family life, 3) health problems among friends and relatives, and 4) events related to the patient's own health. It was also recorded whether the life event was expected or not, positive or negative, and easy or difficult to adjust to.

The study was approved by the research ethical committee of Umeå University in Sweden. All patients received oral and written information about the project, and written consent was obtained in all cases.

Analysis

The hypotheses were tested by comparing the groups. Unpaired t tests or Mann-Whitney tests were used, depending on whether the data were normally distributed. Fisher's exact test was used to compare proportions.

RESULTS

Twenty patients with pseudoseizures and 30 patients with motor conversion symptoms were recruited. Two patients in the pseudoseizure group and none in the motor conversion group refused to participate in the study. Neuroimaging was normal in all participating patients.

Basic demographic data are shown in Table 1. The patients with pseudoseizures were substantially and significantly younger (mean age = 27 years) than the patients with motor conversion symptoms (mean = 39 years) ($p<0.05$, unpaired t test) and were more likely to be unmarried and not to have children. There was no difference in the proportion of women. The patients with pseudoseizures were more likely to have completed a high school education ($p<0.01$, Fisher's exact test), although both had a similar social class profile.

The data from the diagnostic psychiatric interview (Table 2) indicate little difference between the patients with pseudoseizures and those with motor conversion symptoms. Both had high levels of emotional disorder and personality disorder. Although there was no major difference in the proportion with all personality disorder types, there was a substantially and significantly higher proportion of patients with borderline personality disorder in the group with pseudoseizures (35% c.f. 7%) ($p<0.05$, Fisher's exact test).

Table 3 summarizes factors relating to childhood. There was a higher incidence of parental divorce in the pseudoseizure group than in the motor conversion group. Six of the 20 pseudoseizure patients reported incest compared to only one patient in the motor conversion group. The perceived parental rearing inventory scores indicated significantly lower perceived emotional warmth from both parents and a higher perception of rejection from the father in the pseudoseizure group.

The results of the life events interviews are also shown in Table 3. The overall number of life events in the 12 months before assessment was significantly higher in the pseudoseizure group than in the motor conversion group ($p<0.0001$, unpaired t test) but not if only the 3 months before onset was considered. Patients in both groups reported life events that were perceived as negative, unexpected, or difficult to adjust to. Patients with motor conversion symptoms experienced their life events significantly more negatively than those with pseudoseizures ($p<0.01$, unpaired t test). A high proportion of life events reported by both groups related to personal health issues or health problems in family or friends. In the motor

symptom group, 71% of the life events experienced were of this nature compared to 53% in the pseudoseizure group (although the absolute number of life events compared to the pseudoseizure group was similar). The difference in the number of life events over 12 months was primarily made up of an excess of events relating to family life in the pseudoseizure group ($p<0.005$, unpaired t test).

One of the patients with pseudoseizures had a history of a transient motor conversion symptom in the past. Two of the patients with motor conversion symptoms had a history of pseudoseizures. Pain was a common symptom in both the pseudoseizure (30%) and motor conversion (50%) groups.

DISCUSSION

The data presented provide evidence of differences in the characteristics and etiological associations between the patients with pseudoseizures and the patients with motor conversion symptoms. Because these findings were from patients with similar symptom durations, the confounding effects of differential chronicity as a cause of these findings was reduced.

Patients with pseudoseizures have symptoms at a younger age and are more likely to report adverse childhood experiences. There was a 12-year difference between the mean ages of the two groups in this study, which confirmed our hypothesis. This difference probably explains the significantly higher number of previous hospital admissions with somatic symptoms in patients with motor conversion symptoms as well as the finding that patients with pseudoseizures were more likely to be unmarried and not have children.

Adverse childhood experience was particularly common in the pseudoseizure patients, in keeping with our hypotheses. Similarly, incest and borderline personality disorder, which themselves correlate strongly with measures of childhood abuse, were more common in this group. These data raise the possibility that the symptom of pseudoseizures is more strongly associated with adverse early

TABLE 1. Demographic Characteristics of Patients With Pseudoseizures and Patients With Motor Conversion Symptoms

Characteristic	Patients With Pseudoseizures (N = 20)	Patients With Motor Conversion (N = 30)	Analysis
Mean age (years) (range)	27 (18–54)	39 (18–74)	$p<0.005$[a]
Women/men	15/5	18/12	n.s.[c]
Mean duration (months) (95% CI)	5.4 (3.5–7.2)	1.6 (1.2–2.0)	$p<0.0001$[b]
Unmarried	70%	37%	$p<0.05$[c]
Children	30%	73%	$p<0.005$[c]
Number educated to high school level	50%	13%	$p<0.01$[c]
Not fully employed	65%	57%	n.s.[c]
Social status (1 = highest, 2 = middle, 3 = lowest) (95% CI)	2.6 (2.3–2.9)	2.7 (2.5–2.9)	n.s.[a]

[a]Unpaired t test.
[b]Mann-Whitney test.
[c]Fisher's exact test.

TABLE 2. Psychiatric and Medical Characteristics of Patients With Pseudoseizures and Patients With Motor Conversion Symptoms

Characteristic	Patients With Pseudoseizures (N = 20)	Patients With Motor Conversion (N = 30)	Analysis
Previous contact with a psychiatrist	45%	47%	n.s.[a]
Previous admission with somatic symptoms	35%	70%	$p<0.05$[a]
Pain	30%	50%	n.s.[a]
Any axis I disorder	55%	33%	n.s.[a]
Current major depression	30%	27%	n.s.[a]
Any personality disorder	65%	50%	n.s.[a]
Borderline personality disorder	35%	7%	$p<0.05$[a]
Axis V score (range 0–100) (95% CI)	66 (57–74)	67 (61–74)	n.s.[b]

[a]Fisher's exact test.
[b]Unpaired t test.

life events and the subsequent effect of those on personality than on motor conversion symptoms. In contrast, while these factors may be equally important for some patients with motor conversion symptoms, perhaps motor symptoms tend to arise on a broader range of psychosocial backgrounds in response to a greater variety of stimuli, such as fatigue, previous somatic disease, and pain.

Patients with pseudoseizures report more life events. We found a higher rate of life events, particularly related to family life, in the pseudoseizure group in the 12 months preceding the assessment. This supports our hypothesis and lends further support to the hypothesis that pseudoseizures may be more driven by life events than motor symptoms. It is interesting to note that there was not a particular excess of life events in the 3 months before onset (compared to the whole 12 months) in either conversion group. This suggests that if life events are important in the genesis of symptoms, they do not necessarily have to be closely temporally related to the onset of symptoms. This is contrary to definitions of conversion disorder that require such a temporal correlation. Even the most recent DSM-IV classification of conversion disorder still requires that the "initiation or exacerbation of the symptoms or deficit is preceded by conflicts or other stressors."

Limitations

The principal limitation of this study is the small numbers of patients involved. Given the number of tests performed, it is possible that some of the statistically significant differences between the groups occurred by chance. Although the interviewer was not blind to diagnosis—a problem with nearly all studies of this type—the data were collected blind to the specific hypotheses being tested.

The findings are only generalizable to patients who do not meet any of the exclusion criteria of this study, most notably DSM-IV somatization disorder. In practice, a number of patients presenting with conversion symptoms also meet criteria for this diagnosis. If they had been included, measures of distant predisposing factors, such as childhood and personality disorder may have been even higher, but the number of precipitating life events may have been lower since, in our experience, patients with this diagnosis often develop recurrent new symptoms without significant new life events producing them. It should also be emphasized that only patients with a DSM-IV diagnosis of somatization disorder (and not just a history of somatoform symptoms) were excluded. An additional limitation on the generalizability of these findings is that all the patients in

TABLE 3. Childhood Experiences and Recent Life Events of Patients With Pseudoseizures and Patients With Motor Conversion Symptoms

Characteristic	Patients With Pseudoseizures (N = 20)	Patients With Motor Conversion (N = 30)	Analysis
Parents divorced	55%	20%	$p<0.05$[a]
Incest	30%	3%	$p<0.01$[a]
Perceived parental rearing scores (0–100, 95% CI)[b]			
Emotional warmth (father)	42 (38–45)	47 (42–51)	$p<0.05$[c]
Emotional warmth (mother)	41 (38–44)	49 (46–53)	$p<0.005$[c]
Rejection (father)	49 (43–54)	39 (34–44)	$p<0.05$[c]
Rejection (mother)	39 (34–44)	40 (36–44)	n.s.[c]
Overprotection (father)	32 (31–34)	30 (28–32)	n.s.[c]
Overprotection (mother)	34 (32–35)	34 (31–36)	n.s.[c]
Life events preceding onset (95% CI)			
Number over 3 months	1.4 (1.1–1.7)	1.4 (1.0–1.8)	n.s.[c]
Number over 12 months	4.9 (4.0–5.7)	2.7 (2.3–3.1)	$p<0.0001$[c]
Type of life event (mean of total) (95% CI)			
Changes at work	16% (8%–22%)	15% (8%–22%)	n.s.[c]
Changes in family life	32% (24%–40%)	13% (6%–21%)	$p<0.005$[c]
Health problems among family or friends	26% (16%–36%)	33% (24%–42%)	n.s.[c]
Personal health issues	27% (18%–35%)	38% (28%–48%)	n.s.[c]
Quality of life event (mean of total) (95% CI)			
Unexpected	76% (66%–86%)	85% (72%–97%)	n.s.[c]
Negative	70% (59%–81%)	92% (81%–100%)	$p<0.01$[c]
With adjustment problems	73% (59%–87%)	85% (74%–96%)	n.s.[c]

[a]Fisher's exact test.
[b]Lower score equates to lower quality of perceived care.
[c]Unpaired t test.

this study were inpatients. Outpatients or patients with functional neurological symptoms in the community probably have less disabling symptoms and less psychiatric comorbidity.

However, there were also strengths to the sampling procedure used in this study. Consecutive patients seen as neurological referrals in a secondary referral center were recruited. This made them closer to a community sample than a sample from a psychiatric service or tertiary referral centers, such as specialist epilepsy services, which have formed the basis for most previous studies.

Finally, two of the principal areas being studied, life events and childhood experiences, are subject to problems of reporting bias, both in terms of accurate recollection and the influence of the subject's personality and emotional state.

Studies Comparing Pseudoseizures and Motor Conversion Symptoms

Only in the 1957 study by Ljungberg[9] can one find a degree of systematic comparison between a group of patients with motor conversion symptoms and a group of patients with pseudoseizures across a range of measures. Ljungberg found that in the group with paralysis, 53% were male compared to only 26% who were male in the pseudoseizure group. He also found no difference in the age of onset or prognosis between these two groups. He also found that the risk of hysteria in the mothers and sisters of patients with pseudoseizures (13%) was higher than that of the other groups (3.5%). Maxion et al.[36] demonstrated in their series of patients with conversion disorder that patients with pseudoseizures had a slightly lower age of presentation than patients with motor symptoms, although mean ages were not given. Two additional studies reported data showing similar symptomatic outcomes in pseudoseizures compared to motor symptoms but without other comparative data.[37,38]

A review of the published controlled studies on the childhood, life events, and psychiatric morbidity of patients with pseudoseizures and motor conversion symptoms is outside the scope of this article but can be found elsewhere.[26,39] Furthermore, these studies cannot be easily compared since they are drawn from heterogenous populations and have generally used different measures. Simple variables, however, are comparable. In a brief analysis, we examined all of the studies of motor conversion symptoms (paralysis) and pseudoseizures that we are aware of that were published since 1965 in which the sex or age of onset (or age of presentation combined with the duration of symptoms) are reported in consecutive patients. We excluded studies in which patients with paralysis were mixed with those with pseudoseizures or movement or gait disorder.[13–16,40–42]

There were six studies of 167 patients with paralysis reporting an average proportion of 48% women.[5–10,25] In one further study, 57% of 1,316 patients seen at a Chinese hysterical paralysis treatment center were women.[43] A review of 46 studies of pseudoseizures that we were aware of, with a total of 2,103 patients, indicated that 74% of these were women, which was significantly higher than the paralysis group ($p<0.0001$) (references available from the first author). There were insufficient studies to analyze age at onset in studies of paralysis only.[7,10] When we included studies reporting age at onset in gait disorder and mixed studies with high proportions of patients with paralysis, there were six studies reporting a combined average age at onset of 34 years.[7,10,13–16] In 22 studies of pseudoseizures, however, only two articles reported an average age of onset greater than 30 years (references available from the first author). This preliminary literature analysis supports two of the hypotheses of our study: that pseudoseizures tend to appear at a younger age and are more likely to occur in women than motor conversion symptoms.

In this study, there was a high frequency of pain in both groups but a much smaller overlap between pseudoseizures and motor conversion symptoms. This has been previously reported.[7,13–15,44,45] Studies that have found a large overlap in background factors between somatoform pain disorder and conversion disorder[46] echo the increasing realization that rather than forming separate and distinct categories, functional somatic symptoms consistently overlap.[47–49]

Implications for DSM Classification of Conversion Disorder

At present, the only reason pseudoseizures and motor symptoms seem to be grouped together in DSM-IV conversion disorder appears to be because they both imitate neurological disease. In ICD-10, the argument that they both are associated with a dissociative mechanism is arguably more logical. However, apart from the findings of Roelofs et al.,[50] most of the evidence that dissociation is in fact the mechanism comes from studies that have compared conversion disorder with organic disease controls. One must therefore ask whether dissociation, usually measured by dissociative scales, is specific to conversion symp-

toms or simply common to many different somatic and/or psychological symptoms.[2,51,52]

If it can be demonstrated 1) that patients with pseudoseizures have a consistently different psychosocial profile than patients with motor conversion symptoms and 2) that pseudoseizures cluster much more with other symptoms, such as pain, than they do with motor conversion symptoms, this raises questions about the homogeneity of the current diagnostic category of conversion disorder.

What about the natural history of patients with these symptoms as a differentiating feature? This cross-sectional study of recent-onset symptoms cannot tell us whether the younger patients with pseudoseizures may at a later date be especially vulnerable to motor conversion symptoms or vice versa. Despite a handful of studies demonstrating symptom persistence in a majority of patients and some crossover with somatization disorder,[53,54] longitudinal studies of large prospective cohorts are needed to establish the predictive validity of the diagnosis, particularly with respect to the rationale for grouping and separating pseudoneurological symptoms from other somatoform symptoms, such as pain.[48]

One may further question whether conversion disorder really is a disorder or merely a collection of patients with symptoms that imitate neurological disease. The two other criteria in DSM-IV that separate conversion symptoms from conversion syndrome are 1) that psychological factors are judged to be relevant and 2) that the symptom is not intentional or feigned. In practice, both of these criteria require judgments that are difficult or impossible to be certain of. We join other authors who have come to the conclusion that until evidence appears to the contrary, the phenomena described in this article are better described as a different type of conversion symptom rather than combined as conversion disorder.[55–57]

CONCLUSIONS

This study suggests that pseudoseizures appear at a younger age and are more likely to be associated with external factors such as life events and childhood adverse experiences, whereas motor conversion symptoms tend to occur later in life and are less dependent on life events. The symptoms of paralysis and seizure-like episodes unexplained by disease are probably best seen as symptoms rather than as manifestation of a disorder. They share factors, such as personality disorder and childhood experience, which are more common in cases than in patients with disease or the general population. However, these factors are similar to those associated with many other functional or medically unexplained physical symptoms, such as chronic pain and fatigue, and are also found commonly in patients with emotional symptoms, such as depression and anxiety.

Further studies of the pathways to the development of pseudoneurological symptoms and why some symptoms develop in preference to others are required.

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PTSD and Somatization in Women Treated at a VA Primary Care Clinic

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The authors examined the association between trauma, posttraumatic stress disorder (PTSD), and somatization in 264 women attending a Department of Veterans Affairs primary care clinic. Using a structured computerized interview (Composite International Diagnostic Interview), they found that traumatic events were reported by 81% of the women. The lifetime prevalence of PTSD was 27%; for somatization it was 19%. PTSD was the best predictor of somatization after control for demographic variables, veteran status, and other mood and anxiety disorders. Psychological numbing symptoms of PTSD emerged as a particularly strong predictor of somatization. The link between PTSD and somatization deserves further study.

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Somatization (bodily symptoms for which organic causes are not found) is a common occurrence in primary care settings. These multiple unexplained symptoms result in substantial disability, add to the cost of health care, and often lead to therapeutic disappointment.[1] The etiology of these unexplained symptoms remains unknown, although there is general consensus that psychological factors play an important role. Several psychiatric conditions have been associated with somatization, including mood and anxiety disorders.[2] Psychological trauma and abuse have been associated with somatization as well, but this relationship remains poorly understood. Patients suffering from posttraumatic stress disorder (PTSD), associated with both civilian and combat-related traumas, have shown disproportionately high rates of unexplained somatic symptoms.[3] To our knowledge, the association between PTSD and somatization has not been examined together with other possible predictors, such as other anxiety and mood disorders.

Since women are twice as likely as men to suffer from both PTSD and somatization[4] and since women in the military have reportedly experienced high rates of sexual and other violent traumas and their consequences, women who have served in the military may merit particular investigation.

The present study explores further the association between trauma, PTSD, and somatization in women attending a primary care clinic at a Department of Veterans Affairs (VA) medical center. In this group, containing both veteran and nonveteran women, we examined trauma history and mood and anxiety disorders as potential predictors of somatization. We also examined whether specific PTSD symptom clusters differed in their association to somatization.

METHOD

Subjects

Female patients scheduled for an appointment to visit a VA-based primary care women's health clinic were ap-

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proached at the clinic or by telephone and asked to participate in the study. Our goal was to recruit a group of approximately half veterans and half nonveterans (i.e., spouses of veterans) who were eligible for care in the VA system. Exclusion criteria were limited to the presence of psychosis and/or severe acute medical illness.

The human subjects institutional review board at the University of New Mexico approved the project. The nature and purpose of the study were described to patients who agreed to participate, and the subjects' informed consent was obtained. The protocol included a computerized structured diagnostic interview and paper-and-pencil questionnaires chosen to measure current overall health and severity of current PTSD symptoms. Participating subjects were compensated for their time with $20.

Assessment

The Composite International Diagnostic Interview,[5] version 2.1, was used to elicit demographic information, trauma history, and information regarding past and present psychiatric diagnoses according to ICD-10[6] and DSM-IV[7] criteria. This structured computerized instrument was developed by the World Health Organization and the U.S. Alcohol, Drug Abuse, and Mental Health Administration for use as a diagnostic tool in primary care psychiatric research.[5] The interview was developed after the NIMH Diagnostic Interview Schedule[8] (DIS) and has been fully validated and used in both national and international studies.[9–12]

Demographic information The Composite International Diagnostic Interview provided key demographic information; supplemental data were obtained by questionnaire.

Trauma Trauma is operationally defined as any stressful life event that is either outside the range of normal human everyday experience or poses a serious threat to the physical integrity of oneself and/or significant others (DSM-IV). During administration of the Composite International Diagnostic Interview, participants were asked whether they had ever experienced nine different types of trauma, including direct combat experience in a war; involvement in a life-threatening accident; involvement in a fire, flood, or other natural disaster; witnessing someone being badly injured or killed; experiencing rape, sexual molestation, or a serious attack or assault; being threatened with a weapon, held captive, or kidnapped; and being tortured or the victim of terrorists. Respondents were also asked to report any other extremely stressful or upsetting event and whether they had ever suffered a great shock because one of the events listed happened to someone close to them.

PTSD Respondents were asked during the Composite International Diagnostic Interview to indicate the "most upsetting" traumatic event they had experienced, and specific PTSD symptom responses were queried according to the event indicated. Consistent with DSM-IV, symptoms of PTSD were characterized by 1) intense fear, helplessness, or horror, 2) persistent re-experiencing of the event, 3) persistent avoidance of trauma-related stimuli and numbing of general responsiveness, and 4) persistent symptoms of increased arousal.

Somatization We assessed each of the 35 symptoms used in DSM-IV to diagnose somatization disorder. When a somatic symptom was reported during the Composite International Diagnostic Interview, a series of probes determined 1) the severity of the symptom, 2) whether it could be explained by physical illness, injury, or the use of medications, alcohol, or drugs, 3) whether the symptom had interfered with daily functioning, and 4) the onset and recency of each somatic symptom. A positive symptom score, based on the DSM-IV criteria, indicated that the symptom was not likely to be due to a physical disorder. For the purpose of this study, patients meeting the abridged somatization criteria of Escobar et al.[13] (four unexplained somatic symptoms for men and six unexplained somatic symptoms for women) were classified as "somatizers." The somatization items of the Composite International Diagnostic Interview were used to determine an abridged diagnosis, as well as the number of somatic symptoms (range = 0–35) or tendency to report somatic symptoms.

Other psychiatric disorders Lifetime diagnoses of depression, generalized anxiety disorder, panic disorder, and phobias were also determined by the Composite International Diagnostic Interview. For the purpose of this study, the designation of depression reflects recurrent cases (mild, moderate, and severe).

Statistical Analysis

SPSS 8.0 for Windows (Chicago, SPSS, 1997) was the statistical package used in the analysis of the data. Logistic regression generated odds ratios and confidence intervals (CIs). These values were used to determine associations between the predictor variables and abridged somatization.

RESULTS

Subjects

Of 601 eligible patients approached between June 1998 and September 2000, 334 agreed to participate and scheduled an appointment. The findings reflect completed data for 264 respondents, 134 veterans and 130 nonveterans. Subject ages ranged from 23 to 85 years. Ethnicity was predominately white of non-Hispanic origin (46%), and the remaining participants were identified as Hispanic white (30%), American Indian or Alaskan Native (3%), black of non-Hispanic origin (3%), Asian or Pacific Islander (2%), and Hispanic black (1%) (data for 15% not reported). Frequency of ethnicity by veteran status differed significantly for Hispanics, who were more prevalent among nonveterans than veterans ($\chi^2 = 22.05$, df = 1, N = 80, $p < 0.0001$). Ethnicity of participants and nonparticipants did not differ significantly, but the average age of nonparticipants was significantly higher than that of participants ($F = 25.9$, df = 1, 264, $p < 0.0001$).

Prevalence of Trauma and Psychiatric Disorders

The experience of some type of trauma was reported by 81.1% of the subjects, with 73.9% reporting self-experienced traumas (Table 1). Of those reporting self-experienced trauma, 24.6% reported one trauma only, 17.4% reported two traumas, and 58.1% reported three or more. An additional 7.2% reported suffering a great shock resulting from a trauma experienced by someone close to them.

The lifetime prevalences of psychiatric disorders, including somatization, PTSD, recurrent depression, generalized anxiety disorder, and panic disorder, are displayed in Table 2.

Associations Between Trauma, PTSD, and Somatization

Data collected on lifetime prevalence of psychiatric diagnoses indicated a significant relationship between PTSD and abridged somatization. In a stepwise logistic regression equation, abridged somatization was entered as the outcome variable with two levels: negative or positive diagnosis. The model controlled for demographic variables by including veteran status, age, and prevalent ethnic groups (Hispanics, 29%, and whites, 48%) in the first step. Next, trauma (self-experienced) was entered with two levels: no reported trauma or at least one reported trauma. The models and individual factors were not significant. Results are presented in Table 3.

When history of PTSD diagnosis (positive or negative) was added, the model attained significance ($\chi^2 = 20.12$, df = 6, N = 227, $p = 0.003$). Individuals with a history of PTSD were significantly more likely to meet the criteria for abridged somatization (odds ratio = 3.23, 95% CI = 1.53–6.85). The strength of the association did not vary by age, veteran status, or ethnicity.

PTSD, Other Common Psychiatric Disorders, and Somatization

Next, the relative strength of associations between abridged somatization and PTSD, versus other common psychiatric disorders, was examined. Again, demographic variables were controlled for in the first step of a logistic regression. Next, depression, generalized anxiety disorder, and panic disorder were added, and the model attained significance ($\chi^2 = 14.92$, df = 7, N = 226, $p = 0.04$). However, depression was the only single variable significantly associated with abridged somatization (odds ratio = 2.36, 95% CI = 1.13–4.91). When PTSD was added, the change in the model was significant (change in $\chi^2 = 8.62$, df = 1,

TABLE 1. Prevalence of Trauma Types Among Female Patients in a VA Primary Care Clinic

	Percent of Subjects		
Trauma Type	**Total Group (N = 264)**	**Veterans (N = 134)**	**Nonveterans (N = 130)**
Any trauma	73.9	82.8	64.6
Combat	1.9	3.7	0.0
Accident	26.1	34.3	17.7
Fire, flood, etc.	23.1	26.1	20.0
Witnessing someone else being badly injured or killed	36.0	39.6	32.3
Rape	33.3	44.0	22.3
Molestation	37.5	44.0	30.8
Attack	37.5	42.5	32.3
Threat	32.6	32.8	32.3
Torture	3.8	6.0	1.5

N = 234, p = 0.003). Depression did not retain significance in the model, and PTSD emerged as the only significant predictor of abridged somatization (odds ratio = 3.09, 95% CI = 1.46–6.52). Thus, subjects with a history of depression were more likely than subjects with generalized anxiety or panic disorder to meet the criteria for abridged somatization, but subjects with a history of PTSD showed an even greater likelihood of meeting the criteria for abridged somatization (see Table 4).

PTSD and PTSD Symptom Clusters as Predictors of Abridged Somatization

Finally, the relative strengths of trauma-related predictors (PTSD diagnosis, PTSD symptom clusters) of abridged somatization were examined. As before, demographic variables were controlled for in the first step of the model. When PTSD was included, the model attained significance ($\chi^2 = 12.78$, df = 5, N = 145, p = 0.03) and PTSD was a significant predictor in the model (odds ratio = 3.28, 95% CI = 1.44–7.48).

Next, the four PTSD symptom clusters (numbing, hyperarousal, intrusion, avoidance) were added to the model individually, in stepwise fashion. When the numbing symptom cluster was included, the change in the model was significant (change in $\chi^2 = 8.75$, df = 1, 151, p = 0.003). It was interesting that PTSD did not retain significance when numbing symptoms were included. When each symptom cluster was subsequently added to the model, none of the inclusions significantly added to the outcome variance. Therefore, when PTSD and all four symptom cluster categories were included as predictors, only the numbing symptom cluster was significantly associated with abridged somatization (odds ratio = 1.80, 95% CI = 1.21–2.69) (see Table 5).

Important to note is the high correlation between symptom clusters, suggesting that the numbing cluster did not offer unique predictive information. Rather, clusters other than numbing and the PTSD diagnosis overall did not offer predictive power above and beyond that which was accounted for by the numbing symptom cluster alone.

DISCUSSION

Our results confirm previous findings of very high rates of reported exposure to traumatic events in women attending a primary care clinic, particularly sexual assault in the veteran group. The lifetime prevalence rates of PTSD (27.3%)

TABLE 2. Lifetime Prevalence of Somatization and Other Common Psychiatric Disorders Among Female Patients in a VA Primary Care Clinic

	Percent of Subjects		
Disorder	**Total Group (N = 264)**	**Veterans (N = 134)**	**Nonveterans (N = 130)**
Somatization	19.3	25.4	13.1
PTSD	27.3	27.6	26.9
Depression	24.6	20.1	29.2
Generalized anxiety disorder	15.2	9.7	20.8
Panic disorder	4.2	5.2	3.1

TABLE 3. Logistic Regression (Stepwise) Showing Associations Between Trauma, PTSD, and Abridged Somatization Among 234 Female Patients in a VA Primary Care Clinic[a]

Variable	B	SE	Wald Statistic	Odds Ratio	95% CI
Added in step 1					
Age	−0.01	0.02	0.73	0.99	0.95–1.12
Veteran status	−0.45	0.39	1.35	0.64	0.30–1.36
Hispanic	−0.46	0.59	0.60	0.63	0.20–2.03
Anglo	0.02	0.52	0.00	1.02	0.37–2.81
Added in step 2					
Trauma	0.51	0.54	0.89	1.67	0.58–4.80
Added in step 3					
PTSD	1.18	0.38	9.40*	3.23	1.53–6.85
Constant	−0.93	1.04	0.80	0.37	

[a]Table reflects values as they appear in the full model (step 3): $\chi^2 = 20.12$, df = 6, N = 227, p = 0.003.
*p≤0.01.

and somatization (19.3%) were both high. The two conditions were significantly associated, and PTSD was the best predictor of abridged somatization after demographic characteristics, veteran status, and other mood and anxiety disorders were controlled for in our logistic regression model.

The emergence of PTSD as the best predictor of somatization is in agreement with the findings by Andreski et al.,[3] who prospectively studied a large group of patients in a health maintenance organization. In that study, subjects with PTSD developed significantly more somatization symptoms over a period of 5 years than comparison subjects. Previous large epidemiological studies of somatization using an older version of the Composite International Diagnostic Interview did not include a module for PTSD.[2] This may explain why mood and anxiety disorders were equally good predictors of somatization in those studies. Our data and those of others suggest that PTSD must be included in studies of somatization. Among the PTSD symptom cluster, numbing symptoms (excluding avoidance) were the best predictors of somatization. It has been suggested that numbing symptoms are independent of active avoidance and that they might have a different neurobiology.[14] To our knowledge, this is the first report to

TABLE 4. Logistic Regression (Stepwise) Showing Associations Between PTSD, Other Common Psychiatric Disorders, and Abridged Somatization Among 234 Female Patients in a VA Primary Care Clinic[a]

Variable	B	SE	Wald Statistic	Odds Ratio	95% CI
Added in step 1					
Age	−0.01	0.02	0.18	0.99	0.96–1.02
Veteran status	−0.72	0.41	3.08	0.49	0.22–1.09
Hispanic	−0.43	0.60	0.51	0.65	0.20–2.12
Anglo	−0.07	0.53	0.02	0.93	0.33–2.61
Added in step 2					
Depression	0.63	0.39	2.58	1.89	0.87–4.09
Generalized anxiety disorder	0.56	0.46	1.49	1.75	0.71–4.32
Panic disorder	0.40	0.76	0.27	1.48	0.33–6.64
Added in step 3					
PTSD	1.13	0.38	8.74*	3.09	1.46–6.52
Constant	−0.71	0.92	0.61		

[a]Table reflects values as they appear in the full model (step 3): $\chi^2 = 23.54$, df = 8, N = 225, p = 0.003.
*p≤0.01.

TABLE 5. Logistic Regression (Stepwise) Showing Associations Between PTSD, PTSD Symptom Clusters, and Abridged Somatization Among 151 Female Patients in a VA Primary Care Clinic[a]

Variable	B	SE	Wald Statistic	Odds Ratio	95% CI
Added in step 1					
Age	−0.02	0.02	0.62	0.98	0.94–1.03
Veteran status	−0.31	0.46	0.44	0.73	0.29–1.82
Hispanic	−0.54	0.71	0.58	0.58	0.14–2.35
Anglo	0.43	0.64	0.46	1.54	0.44–5.38
Added in step 2					
PTSD	−0.08	0.59	0.02	0.93	0.29–2.95
Added in step 3					
Numbing	0.49	0.23	4.70*	1.64	1.04–2.56
Added in step 4					
Hyperarousal	−0.02	0.23	0.01	0.98	0.63–1.53
Added in step 5					
Intrusion	0.23	0.22	1.08	1.25	0.82–1.92
Added in step 6					
Avoidance	0.09	0.28	0.11	1.10	0.63–1.90
Constant	−2.02	1.34	2.26		

[a]Table reflects values as they appear in the full model (step 6): $\chi^2 = 23.10$, df = 9, N = 141, p = 0.006.
*p≤0.05.

directly link numbing symptoms specifically to somatization. It is conceivable that numbing symptoms reflect social or emotional disengagement, causing increased awareness of and focus on internal sensory perception. It is also possible that some of the numbing symptoms of PTSD overlap with the alexithymia construct, which is known to be associated with somatization. If confirmed and replicated, the finding could lead to a new model for conceptualizing and studying somatization in the traumatized population.

Veteran status did not appear to be a significant predictor of somatization. The female veterans in our study reported more traumatic events, but the lifetime prevalence of PTSD was about the same in both groups (no significant difference).

Contrary to what we expected, rates of somatization did not vary with ethnicity. This finding differs from previous reports that suggested that Hispanic patients tend to somatize more.[15] This may reflect the level of acculturation of the Hispanics in the military and particularly in New Mexico.

Our study has some limitations, including the relatively low response rate and the lack of comprehensive data from the nonparticipant group. The use of lifetime prevalence rates for both somatization and PTSD makes it impossible to determine the temporal relationship of the association. However, the results deserve further investigation.

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Case report

Somatization in response to undiagnosed obsessive compulsive disorder in a family

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Abstract

Background: Somatization is a common problem in primary care and often presents puzzling problems for the family physician. A family or contextual approach is often useful in investigating and treating refractory symptoms.

Case presentation: A 63 year-old patient presented to his family physician with recurrent episodes of syncope, weakness and various other somatic symptoms. Lengthy clinical investigations found no organic pathological findings but a brief family assessment by the family physician revealed that the patient's wife was the "hidden" patient. Successful treatment of the patient's wife led to full recovery for both.

Conclusions: Exploration and treatment of the family context may often hold the key to the solution of difficult problems in somatizing patients.

Background

Family physicians are often faced with patients who present complex or puzzling symptoms that defy diagnosis or explanation despite intensive investigations [1]. Rigid adherence to the biomedical model is unsatisfactory in many cases. In the somatizing patient, one in whom multiple physical complaints suggest physical disorders without a disease or physical basis to account for them, the solution to problems may lie within the family context [2]. The objective of this case presentation is describe a successful family interevention in a patient with long-standing symptoms who was not helped by traditional investigations and treatments directed at the identified patient.

Case report

Mr. M. was a 63-year-old man of North African origin living in a deprived neighborhood in a city in Northern Israel. He was married and the father of five children. His youngest son, aged 17 years, was still living at home. He had worked in a large construction company, initially as a labourer then later as a manager until his early retirement. He presented to a new family physician in the neighbourhood clinic on a busy day, without an appointment, requesting to be seen urgently. He was known to this family physician from previous visits only for treatment of poorly-controlled Type 2 diabetes mellitus, from which he had suffered for 20 years. The patient was a tall obese, plethoric man. It was not immediately clear to the family physician why the patient, who was usually slow to speak and

bashful, was agitated and impatient. An immediate assessment was performed.

The patient reported that on the previous Friday evening when he rose to make the Sabbath blessings over wine, his legs shook, he was unable to speak and he fell to the floor. He recalls hearing his children call for an ambulance and remembers walking to the ambulance with minimal help from his sons, who accompanied him to the local hospital. He complained of frequent episodes during the past few years of dizziness, headache, tremor and sweating ending in loss of consciousness. He also complained of irritability and insomnia with early morning waking since his retirement. It was clear to the family physician that additional time would be required for further assessment, and a longer appointment was rescheduled for the end of the clinic day.

Review of the patient's thick medical chart before the second visit revealed frequent visits to many other physicians for similar symptoms in the past. The medical record showed a continuing pattern of emergency room visits, out-patient clinic visits to neurology and cardiology departments and hospital admissions for the same complaints. Unhelpful repeated investigations included several modalities of diagnotic imaging. Though his diabetes had been poorly controlled with oral medications, there was no evidence of diabetic complications. Numerous diagnostic labels had been applied and various medications had been tried without success. The family physician recalled a feeling of helplessness after such a long history of symptoms and extensive investigations but persisted in encouraging the patient to talk further about his symptoms.

Mr. M. had taken early retirement 6 months previously because of his symptoms. After retirement, his symptoms worsened. The physician noted that the patient's sons were the only ones involved in his care for each of the fainting episodes. The physician wondered about the wife's involvement in the care of her husband and asked about her role. As though a weight had been lifted from his chest, Mr. M. began to speak freely about how he suffered from his wife's behaviour. For the past several years his wife, Mrs. R., had ceased to function at home, and was pre-occupied with cleaning all day and every day. She was unable to tolerate even the smallest change in her home. This "craziness" as Mr. M. called it had driven their youngest son to leave home and move to a distant city. Mr. M. had also noted changes in her mood in recent years; episodes of anger and tears followed by laughter. She had outbursts of anger during which she left the house and went out shouting in the street. Mr. M. suffered acute embarrassment in front of the neighbors from these episodes and said he was "unable to stand this". In his words, the situation at home was what had led to his fainting spells and such was the case on the previous Friday night. With the approval of Mr. M., his wife was invited to a meeting with the psychiatric consultant in the clinic. Mrs. R. agreed to attend the clinic on condition that she would not be referred to the local community mental health clinic.

Mrs. R. was diagnosed as suffering from obsessive-compulsive disorder with depressive features. Treatment was begun with anafranil at a dose of 25 milligrams per day and was gradually increased to 75 milligrams per day over a period of 4 weeks. Marked improvement in her condition was noted over this period. Her mood improved, she became less anxious, her appetite increased and a marked decrease in her cleaning behaviors was noted. Two months after the start of drug treatment, the couple attended the clinic together to express their satisfaction with treatment. Mr. M. had no further episodes of syncope in 5 years of follow-up.

Comments

Obsessive-compulsive disorder (OCD) is a psychiatric disorder that afflicts approximately 1% to 3% of the population [3]. Impairment is evident in several areas, particularly in occupational and social maladjustment. It may go unrecognized, however, as many patients are embarrassed by their symptoms and are thus reluctant to report them. The disorder (OCD) often coexists with major depression (MD), with rates varying from 35 to 75% [4]. The risk for anxiety disorders is increased among the relatives of obsessional subjects compared with that for relatives of controls [5]. Several studies support extensive family involvement and accommodation of OCD symptoms, as well as the considerable burden placed on families who reduce their social activities and increase their isolation and distress [6,7]. Relatives of OCD sufferers, who are forced to participate in the patient's rituals, may report their distress in visits to the family physician and present an opportunity for diagnosis and treatment [8]. Patients treated with appropriate medication and behavioral modalities may show rapid improvement in adjustment levels with subsequent improvement in the function of all family members.

Conclusions

This case emphasizes the need for physicians to take a broader look at the family context when faced with patients with prolonged puzzling symptoms. Given the prevalence of OCD, family physicians have a role to play in the early identification and treatment of OCD. This case also highlights the need for support, advice, and education for family members of persons with OCD.

Competing interests

None declared.

Authors' contributions

RW conducted the initial treatment of the patient and his family and drafted the original manuscript. YF performed the literature review and wrote additional drafts. JY wrote the final draft and provided additional commentary in the Conclusion. All authors read and approved the final manuscript.

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Somatoform Disorders Among First-Time Referrals to a Neurology Service

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Consecutive new neurology inpatients and outpatients (N = 198) were assessed for somatoform disorders by using the Schedules for Clinical Assessment in Neuropsychiatry. Sixty-one percent of the patients (59% of the female patients and 63% of the male patients) had at least one medically unexplained symptom, and 34.9% fulfilled the diagnostic criteria for an ICD-10 somatoform disorder (27.7% of the male patients, 41.3% of the female patients, 20.5% of the inpatients, and 43.2% of the outpatients). The prevalence figures were about the same when DSM-IV criteria for somatoform disorders were used. Of the patients with a somatoform disorder, 60.5% also had another mental disorder. Somatization disorder, somatoform autonomic dysfunction, pain disorder, and neurasthenia were equally prevalent (6%–7%); dissociative (conversion) disorders and undifferentiated somatoform disorders were found in 2–3% of the patients. Fifty percent of the patients with somatoform disorders were identified by the neurologists.

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Mental disorders are highly prevalent among patients attending neurological services.[1–6] Patients presenting with physical symptoms not attributable to any known medical condition (i.e., functional or medically unexplained symptoms) are particularly common.[3,4,6–8] In ICD-10 and DSM-IV these conditions are classified mainly in the somatoform disorders group. In a study that used the same data set used in this study, somatoform disorders were the most prevalent psychiatric diagnoses among neurological inpatients and outpatients.[4] Little is known about the symptoms and types of somatoform disorders experienced by patients in neurological settings. In studies that have been conducted in this area, ICD-10 or DSM-IV criteria have not been used, or only one or a few of the diagnostic subcategories in the somatoform disorders group and not the whole diagnostic spectrum have been investigated.

The aims of this study were to determine the prevalence of medically unexplained symptoms and of somatoform and related disorders (classified according to ICD-10 and DSM-IV diagnostic subcategories) among new inpatients and outpatients seen in a neurological setting, determine the comorbidity of these disorders with other mental disorders, and assess whether somatoform and related disorders are diagnosed by neurologists.

METHOD

Inclusion

The study population included consecutive patients age 18 years or older referred for the first time to the neu-

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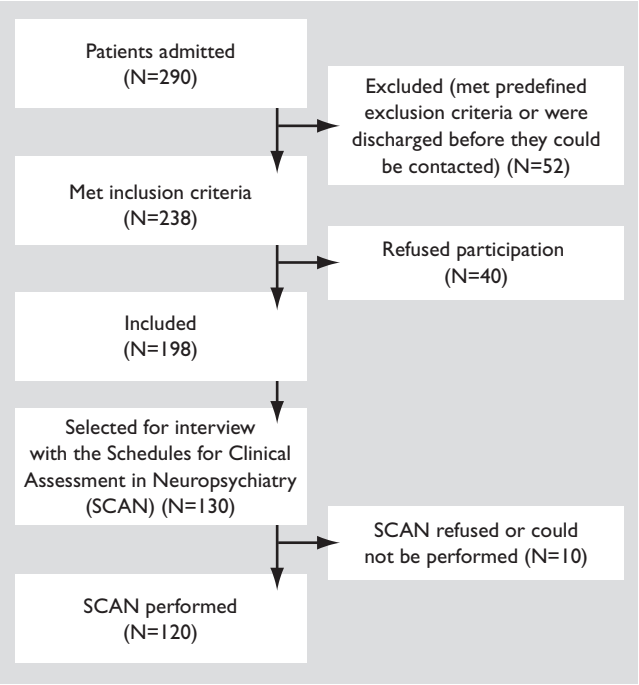
rological department of Vejle County Hospital, Vejle, Denmark, during a 3-month period in 1997. Only patients who had never previously been examined by a neurologist were eligible. The department provides all hospital-based neurological services for the general population of Vejle County, a total of approximately 356,000 inhabitants. The county is a mixed rural and urban area with four fairly large towns in which about one-half of the population is middle class. In total 290 patients were admitted either as inpatients or outpatients for the first time during the inclusion period (Figure 1). Excluded according to predefined criteria were patients who were not of Scandinavian origin (N = 5) and patients who could not be interviewed because they were too ill (N = 13) or because of deafness (N = 4), somnolence (N = 5), unconsciousness (N = 3), aphasia (N = 8), or disorientation (N = 3). In addition, 11 patients were discharged before a research worker could contact them. Forty patients refused to participate in the investigation. Thus, 198 patients were included.

The excluded patients were compared to the included patients on age, gender, and use of psychiatric and nonpsychiatric health care. These analyses, reported in a previous study,[4] showed only minor differences.

Table 1 summarizes the study subjects' sociodemographic and admission characteristics, and Table 2 lists their ICD-10 discharge diagnoses.

FIGURE 1. Sample Selection in a Study of Somatoform Disorders Among New Patients Seen in a Neurology Service



Procedure and Assessment

A two-phase design was used. At admission or first contact, all patients were interviewed by one of two research nurses. The interview included an eight-item version of the Symptom Check List (SCL-8d),[9] used to assess anxiety and depression, and the seven-item Whiteley index, which was slightly modified for use in the interview (available from the author on request). This scale measures illness worry and convictions about the presence of illness and has been shown to detect somatization.[10] The responses to each item were dichotomized.

Patients were selected in the following manner to undergo a diagnostic psychiatric interview. Patients with a score of 2 or more on the SCL-8d and/or 3 or more on the seven-item Whiteley index were considered high scorers. A random sample consisting of 50% of all patients was selected for psychiatric interview, and of the remaining 50% of patients, all high scorers were selected for the interview. Thus, a stratified subsample consisting of all patients with high scores and approximately one-half of the patients with low scores was selected. The psychiatric interview was conducted either during admission or at the first visit in the outpatient clinic. If the patient could not be interviewed in these settings, an appointment was made for the interview to take place as soon as possible after the first visit. Of the 130 patients selected for psychiatric interview, 10 refused to participate. Thus, 120 patients were interviewed.

The psychiatric interview was conducted with the Schedules for Clinical Assessment in Neuropsychiatry, version 2.1 (SCAN).[11] The SCAN interview is used to inquire about 76 physical symptoms in seven symptom groups. Each symptom is rated by the interviewer according to whether it can be attributed to a medical condition/dysfunction or not. Symptoms for which this distinction cannot be made reliably are rated with a separate code. In this study, we included in the analysis only those symptoms that were rated "definitely unexplained." The interviewers were free to explore aspects that were not fully clarified in the interview (e.g., by reviewing medical records or discharge letters). The interviewers met regularly during the course of the study to confer about specific cases and to discuss strategies for interpreting and rating ambiguous responses and symptoms that seemed to fall between the response categories. They were free to consult other physicians. The two SCAN interviewers, who had received psychiatric training during residency and who had been trained and certified at the World

Health Organization center in Aarhus, were blinded to the patients' answers to the interview at the index contact. The interrater agreement was high (agreement on 16 of 17 patients; $\kappa = 0.88$).

The SCAN interviews were used to develop computerized ICD-10 and DSM-IV diagnoses of somatoform disorders. At first contact and at discharge, the neurologist responsible for the examination/treatment of each patient was asked to rate whether the patient's symptoms were functional (i.e., without any adequate organic base); this rating was made on a 5-point scale ranging from "definitely not" to "definitely yes." The neurologist was also asked to rate whether the patient was more preoccupied with his or her symptoms than would be expected on the basis of the nature of those symptoms; this rating was made on a 4-point scale ranging from "definitely not" to "yes, very much." Furthermore, at discharge only, the neurologist used a 4-point scale ranging from "no" to "markedly" to rate whether the patient had exaggerated his or her symptoms.

Data Analysis

Data from the second phase of the two-phase design were analyzed by using weights that were inversely proportional to the sampling probabilities.[12,13] Prevalence estimates and approximate confidence intervals were calculated by weighted logistic regression analysis. The same method was used to estimate the associations between psychiatric disorders and other variables. In a few analyses we applied other statistical procedures using the same weights. In order to ensure valid standard errors and significance tests, the weights were scaled so that the sum of the weights was equal to the actual sample size (N = 120). SPSS for Windows, versions 6.1.3 and 10 (SPSS, Inc., Chicago), was used for statistical analysis.

TABLE 1. Sociodemographic and Admission Characteristics of 198 New Patients Seen in a Neurology Service Over a 3-Month Period and Assessed for Somatoform Disorders

Characteristic	Male patients (N = 92)	Female patients (N = 106)	Analysis
	Median	Median	p
Age (years)	49.3	50.7	0.64[a]
	%	%	p
Gender	46.5	53.5	0.18[b]
Occupation			0.33[c]
Employed[d]	56.5	50.0	
Unemployed	7.6	13.2	
Retired	21.7	27.4	
Disability pension	14.1	9.4	
Living conditions			0.21[c]
Alone[e]	25.0	35.8	
With partner	58.7	52.8	
With partner and child(ren)	16.3	11.3	
First contact[f]			0.77[c]
Inpatient admission[g]	41.3	43.4	
Outpatient examination	58.7	56.6	
Source of referral			0.63[c]
Family physician	57.6	49.1	
On-call general practitioner	8.7	12.3	
Emergency department	4.4	5.7	
Other department or specialist	18.5	24.5	
Other	10.9	8.5	

[a]Mann-Whitney U test.
[b]Binomial test, one-sided.
[c]Chi-square test.
[d]Including part-time workers and students.
[e]Including four (3.8%) female patients living alone with a child and one male patient living with his parents.
[f]Some patients first seen as outpatients may have been hospitalized later. Some patients first seen as inpatients may have been followed up as outpatients.
[g]One admission was planned; the remaining admissions were prompted by acute conditions.

RESULTS

Somatoform Disorders and Functional Symptoms

Sixty-one percent of the patients (59% of the female patients and 63% of the male patients) had at least one medically unexplained symptom. Figure 2 shows the symptom frequencies. Symptoms associated with other organ systems were as frequent as neurological symptoms. About 35% of the patients fulfilled the diagnostic criteria for an ICD-10 somatoform disorder, and the prevalence was about the same when DSM-IV criteria were applied (Table 3). As for the specified ICD-10 diagnoses, somatization disorder, somatoform autonomic dysfunction, pain disorder, and neurasthenia were equally prevalent (found in 6%–7% of patients); dissociative (conversion) disorders and undifferentiated somatoform disorders were found in 2–3% of the patients. The distribution of diagnoses was quite different when the DSM-IV criteria were used: somatization disorder was much less common (2%), and pain disorder was more common (11.6%). DSM-IV undifferentiated somatoform disorder was much more frequent than ICD-10 undifferentiated somatoform disorder (Table 3). Conversion disorder (2.9%) was equally prevalent according to both diagnostic systems. ICD-10 hypochondriasis was rare (0.6%), and DSM-IV hypochondriasis was more common (1.8%). Somatoform disorder not otherwise specified was diagnosed in 12.8% of the patients according to the ICD-10 criteria and in 7.1% of patients according to the DSM-IV criteria. As Figure 2 shows, only few patients complained of urogenital symptoms (apart from sexual indifference), and this low frequency may explain the low prevalence of DSM-IV somatization disorder, compared with ICD-10 somatization disorder.

TABLE 2. ICD-10 Discharge Diagnoses of 198 New Patients Seen in a Neurology Service Over a 3-Month Period and Assessed for Somatoform Disorders[a]

	Patients (N = 198)	
Disease group and code	**N**	**%**
Certain infectious and parasitic diseases (A00–B99)	3	1.5
Malignant neoplasms (C00–C97)	2	1.0
In situ and benign neoplasms and other neoplasms (D00–D48)	5	2.5
Endocrine, nutritional, and metabolic diseases (E00–E90)	1	0.5
Mental and behavioral disorders (F00–F99)	8	4.0
Diseases of the nervous system (G00–G99)	58	29.3
Diseases of the eye and ear, etc. (H00–H95)	8	4.0
Diseases of the circulatory system (I00–I99)	31	15.7
Diseases of the musculoskeletal system, etc. (M00–M99)	17	8.6
Congenital malformations, etc. (Q00–Q99)	1	0.5
Symptoms, etc., not elsewhere classified (R00–R99)	10	5.1
Injury, poisoning, etc. (S00–T98)	12	6.1
Factors influencing health status, etc. (Z00–Z99)	53	26.8

[a]Including main and subsidiary diagnoses. Each patient may have received more than one diagnosis, and the percentages sum to more than 100%.

Somatoform Disorders, Age, and Gender

The overall prevalence of ICD-10 (and DSM–IV) somatoform disorders was more than 10% higher among women than among men (Table 3). Adjusted for age, the gender difference was not statistically significant at a 5% probability level ($p = 0.10$; weighted logistic regression). Women had a higher prevalence of all the specified ICD-10 and DSM-IV diagnoses, except for neurasthenia (Table 3). Figure 3 shows the overall prevalence of somatoform disorders among female and male patients in three age groups. The prevalence among male patients fell markedly with increasing age ($p = 0.02$; test for trend in proportions). In the >60-year age group, the difference between genders in prevalence was 29.5%, but because of the small number of individuals, the difference did not reach statistical significance at a 5% probability level ($p = 0.06$; weighted logistic regression).

Somatoform Disorders Among Inpatients and Outpatients

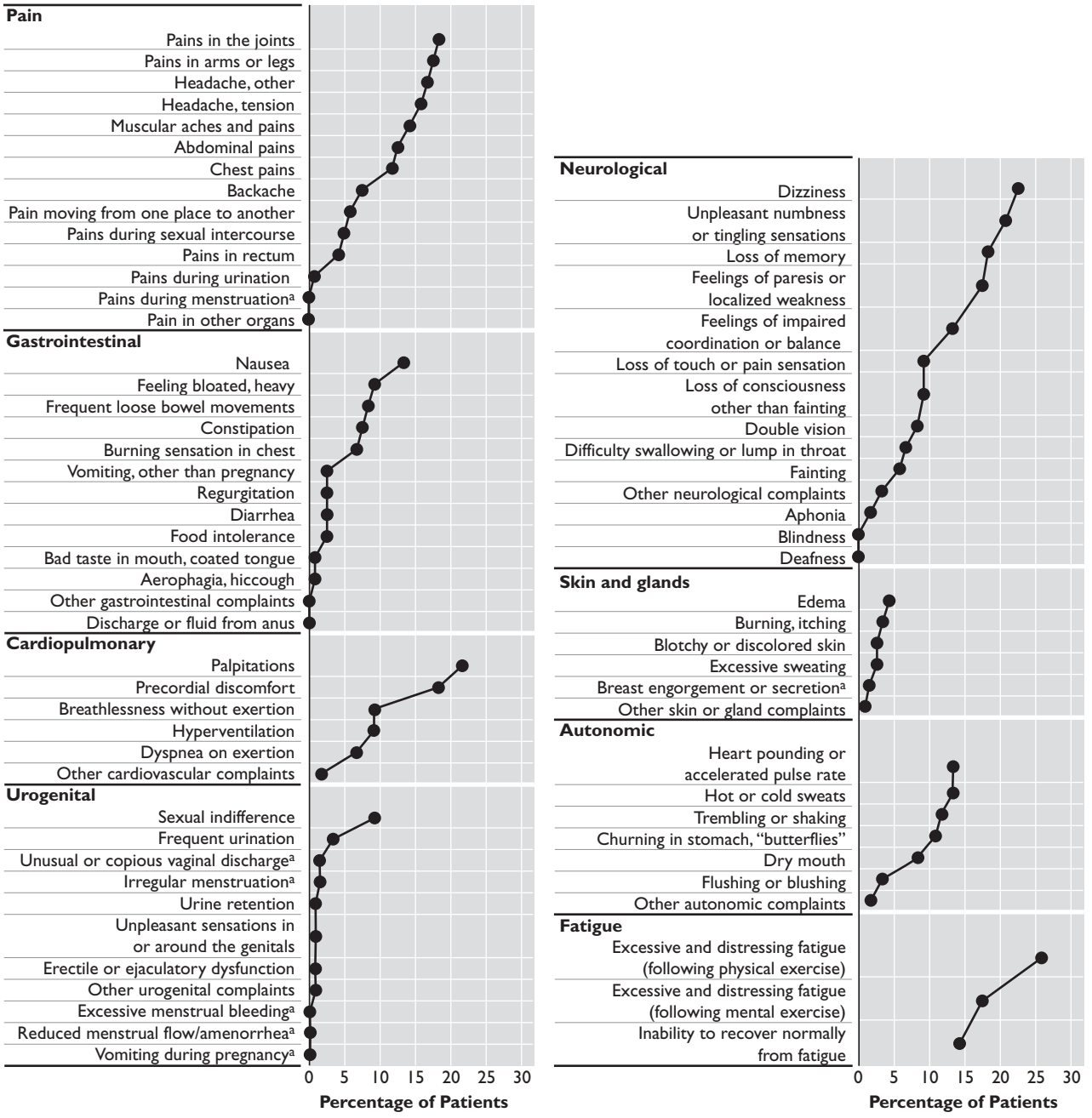
Most ICD-10 and DSM-IV diagnoses had a higher prevalence among outpatients than among inpatients, but the differences were statistically significant for only a few categories. ICD-10 and DSM-IV somatoform disorders were more than twice as prevalent among outpatients as among inpatients ($p < 0.02$; weighted logistic regression) (Table 3). This difference was partly accounted for by a higher prevalence of ICD-10 unspecified somatoform disorder among outpatients, compared to inpatients ($p = 0.03$; weighted logistic regression) and a higher prevalence of DSM-IV undifferentiated somatoform disorder among outpatients, compared to inpatients ($p = 0.01$; weighted logistic regression).

Comorbidity With Psychiatric Disorders

Table 3 reports rates of psychiatric comorbidity in the study subjects, i.e., the percentage of patients with a so-

matoform disorder who also had another ICD-10 mental disorder according to the SCAN interview. The overall rate of comorbidity was about 60%. The rate was higher in patients with some somatoform disorders and was lower in patients with other disorders, such as ICD-10 pain disorder (43.8%) and ICD-10 somatoform disorder, unspecified (39.6%). Several diagnoses, including four ICD-10 diagnoses and three DSM-IV diagnoses, had a comorbidity of 100%, i.e., all patients with these disorders had a comorbid psychiatric disorder. About one-quarter of the patients with

FIGURE 2. Frequency of Medically Unexplained Symptoms Among New Patients Seen in a Neurology Service Over a 3-Month Period and Interviewed With the Schedules for Clinical Assessment in Neuropsychiatry (N = 120)



[a]Percentage of female patients (N = 69).

TABLE 3. Prevalence of ICD-10 and DSM-IV Somatoform Disorders Among New Patients Seen in a Neurology Service Over a 3-Month Period[a]

		Estimated prevalence						Comorbidity of Somatoform Disorder With Other Mental Disorders (%)		
		All patients								
Diagnostic System and Disorder	Code	%	95% CI	Male Patients (%)	Female Patients (%)	Inpatients (%)	Outpatients (%)	Any other ICD-10 psychiatric disorder	ICD-10 depressive disorders, including dysthymia	Phobias, generalized anxiety, panic disorder
ICD-10										
Any somatoform disorder	—	34.9	26.9–43.8	27.7	41.3	20.5	43.2	60.5	23.8	26.8
Somatization disorder	F45.0	7.0	3.6–13.2	3.8	9.9	4.8	8.3	84.1	17.8	45.1
Undifferentiated somatoform disorder	F45.1	2.5	0.8–7.5	0.0	4.7	1.8	2.9	100.0	26.9	53.8
Hypochondriacal disorder	F45.2	0.6	2.9–12.0	0.0	1.1	0.0	0.9	100.0	0.0	100.0
Somatoform autonomic dysfunction	F45.3	6.0	3.5–12.9	4.1	7.8	4.8	6.8	100.0	0.0	63.6
Persistent somatoform pain disorder[b]	F45.4	6.8	0.1–5.8	6.2	7.4	6.1	7.2	43.8	35.4	8.5
Neurasthenia	F48.0	6.0	0.4–5.2	7.4	4.7	3.7	7.3	81.2	39.1	53.4
Dissociative (conversion) disorders (motoric etc.)[c]	F44.4-6	2.9	1.0–8.0	2.3	3.5	2.9	2.9	100.0	0.0	23.2
Somatoform disorder, unspecified	F45.9	12.8	7.9–20.0	9.2	15.9	2.9	18.4	39.6	22.8	13.7
DSM-IV										
Any somatoform disorder	—	35.4	27.4–44.3	28.7	41.3	20.5	44.0	61.1	23.5	24.8
Somatization disorder	300.81	1.1	0.2–5.8	0.0	2.0	2.9	0.0	100.0	0.0	0.0
Undifferentiated somatoform disorder	300.81	17.5	11.7–25.3	15.4	19.3	4.8	24.8	49.5	26.1	21.0
Conversion disorder	300.11	2.9	1.0–8.0	2.3	3.5	2.9	2.9	100.0	0.0	23.2
Pain disorder (chronic)[c]	307.80/89	11.6	7.0–18.7	8.7	14.2	9.8	12.7	57.6	32.2	27.3
Hypochondriasis	300.7	1.8	0.5–6.6	0.0	3.4	0.0	2.8	100.0	0.0	69.7
Somatoform disorder not otherwise specified	300.81	7.1	3.6–13.3	6.3	7.8	8.0	6.6	68.9	35.4	35.4

[a]Calculated by weighted logistic regression (N = 120; N = 51 male patients, N = 69 female patients).
[b]Symptom duration of 6 months or more.
[c]Dissociative motor or convulsion disorders or dissociative anesthesia and sensory loss.

a somatoform disorder had comorbid depression or anxiety disorder, but there was a large variation between the diagnostic subcategories. This variation may be artificial because of the relatively small patient sample.

Neurologists' Impressions of Patients' Symptoms and Symptom Preoccupation

None of the neurologists' ratings of the functionality of patients' symptoms, patients' preoccupation with symptoms, or aggravation of symptoms identified all patients with somatoform disorders (Table 4). The question that identified the most somatoform patients was whether the patient was preoccupied with his or her symptoms. At the first contact, 50% of the patients with somatoform disorder were identified by the neurologists as at least "a little preoccupied"; 42% of the patients were so identified at discharge.

Only three patients with somatoform disorder (1.5% of all patients) were referred to a psychiatrist or psychologist. All three patients were, prior to admission, in treatment by their family physician for a mental disturbance.

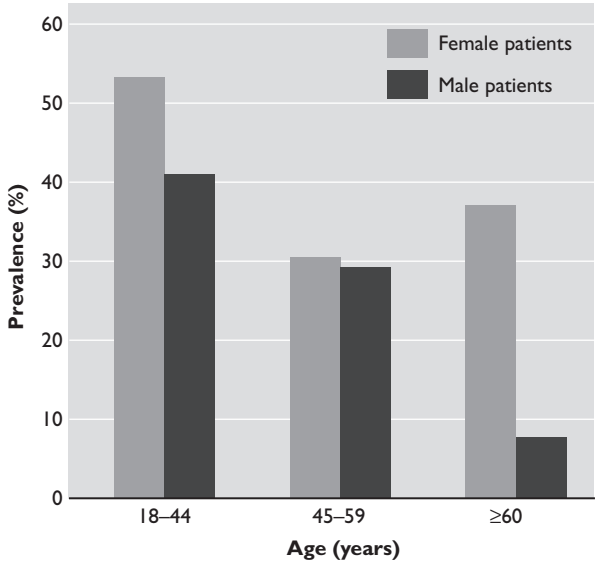
DISCUSSION

About one-third of new patients seen in a neurological service over a 3-month period fulfilled ICD-10 and DSM-IV criteria for a somatoform disorder. We are not aware of other reports on the prevalence of ICD-10 and DSM-IV somatoform disorders among neurology patients. The prevalence of somatoform disorders identified in this study was higher than that found by Fink et al.[14] among medical inpatients. However, in contrast to the previous study, the current study included only new patients, and the sample was younger and included both inpatients and outpatients. The divergence may therefore be partly explained by the different patient samples. The findings of a higher prevalence of somatoform disorders among women and among younger patients are in accordance with previous findings for medical inpatients.[14] The prevalence of somatoform disorders in the current study was higher than that found for primary care patients in an earlier study.[15]

We used a two-step design that included interviews with all patients who scored high on the screening instruments and with a random sample of the remaining patients. This procedure was cost-effective because it allowed us to avoid interviewing numerous patients without mental problems. One weakness of the procedure is that the statistical weighting procedures used for correction of the skewness imposed by the sampling procedure weakened the statistical power and resulted in wide confidence intervals.

Considering solely functional or medically unexplained symptoms, the prevalence was higher in the current study, compared to other studies in neurological settings.[3,7,16,17] The difference may be attributed to the fact that the other studies were based only on neurologists' judgments. Neurologists may be inclined to focus mainly on the symptoms that they believe are relevant for the particular patient. In this study we systematically asked about all kinds of symptoms, regardless of the relevance for the actual neurological referral. Some patients might thus have had a well-defined neurological disease besides their somatoform disorder. We do not know whether this difference may partly explain the neurologists' low rate of recognition of somatoform disorders, compared to the SCAN psychiatric interview, because we did not record whether the functional symptoms were the only reason for the neurological referral. Another explanation for the low level of agreement between neurologists' impressions and the psychiatric interview findings may be that the concepts of medically unexplained symptoms and somatoform disorders are largely unfamiliar to or even neglected by neurologists. Finally, the prevalence of somatoform disorders may be overestimated. Symptoms rated as medically unexplained in the SCAN interview may indeed not have been so. However, as the interviewers were free to explore case notes and other information and the interview was

FIGURE 3. Prevalence of Somatoform Disorders Among New Patients Seen in a Neurology Service Over a 3-Month Period (N = 198), by Gender and Age of Patients



carried out after the neurological examination, the findings of the neurological examination were taken into account by the interviewers. Both interviewers had had several years of training in medicine and surgery, and the one who did the main part of the interview had 1 year of training in neurology. To qualify for a somatoform disorder diagnosis, patients must have had symptoms for more than 6 months and must have had a substantial number of functional symptoms. (The second criterion applies to all somatoform disorder diagnoses except for unspecified somatoform disorder and pain disorder.) The diagnoses obtained by means of the SCAN interview thus seem substantial. The neurologists had high specificity in identifying patients with somatoform disorder, i.e., when they identified a functional disorder in a patient, that patient in most cases also received a somatoform disorder diagnosis according to the SCAN interview. This finding calls for more intensive study of the cases in which there were disagreement between the neurologists and the research interviewers.

No patient follow-up was conducted in our study, although follow-up data could have been used to investigate whether the symptoms were later explained by a differential diagnosis and to study the stability of the somatoform disorder diagnoses over time. However, medically unexplained symptoms only rarely are explained by a medical condition diagnosed at a later stage. For example, Crimlisk et al.[18] showed that at 6-year follow-up only 2%–3% of patients discharged from a neurological department with a diagnosis of functional paralysis had an organic neurological disorder that fully or partly explained their previous symptoms.

Only three (1.5%) of the 198 patients in this study were referred to a psychiatrist or to a psychologist, and all three had a somatoform disorder according to the SCAN interview. The low referral rate in the present study is in line with findings in other studies.[19] As the neurologists were able to identify many more than three of the patients with somatoform disorders, the low referral rate is not explained only by a lack of recognition of the disorders. Other factors such as lack of accessibility to psychiatric assistance, fear of stigmatizing the patient, or time pressure may be more important.

The findings of a high prevalence of functional complaints and somatoform disorders among neurology patients highlight the need for increased awareness of the problem among neurologists and for more research on the management of somatoform disorders.

The high rate of comorbidity between somatoform disorders and other mental disorders is in accordance with findings in other studies.[14,15,20] Despite agreement on the overall prevalence of somatoform disorders assessed according to the ICD-10 and DSM-IV diagnostic systems, there were great discrepancies in the prevalence of the specific somatoform disorder subcategories according to the two systems. This pattern raises doubts about the credibility of the diagnostic subcategories, which may be one reason why neurologists are reluctant to adopt these categories in their diagnostic practice. It is therefore very important that the diagnostic groups be validated and revised on an empirical basis to make them acceptable for use in clinical practice.

TABLE 4. Sensitivity and Specificity of Neurologists' Identification of Somatoform Disorders Among 198 New Patients Seen in a Neurology Service[a]

Criterion for Identifying Patients With Somatoform Disorder	Prevalence (%)	Sensitivity[b]	Specificity[c]
ICD-10 diagnosis of somatoform disorder (according to the Schedules for Clinical Assessment in Neuropsychiatry)	18	1.00	1.00
Neurologists' judgment at first contact			
Patient's symptoms possibly functional	17	0.40	0.94
Patient's symptoms most likely functional	3	0.09	1.00
Patient is a little preoccupied with symptoms	28	0.50	0.83
Patient is somewhat preoccupied with symptoms	11	0.20	0.93
Neurologists' judgment at discharge			
Patient's symptoms a little functional	14	0.27	0.93
Patient's symptoms somewhat functional	6	0.09	0.95
Patient is a little preoccupied with symptoms	26	0.42	0.82
Patient is somewhat preoccupied with symptoms	5	0.10	0.98
Patient's symptoms had been a little exaggerated	14	0.19	0.89
Patient's symptoms had been somewhat exaggerated	6	0.08	0.96

[a]Neurologists' judgments were compared with ICD-10 diagnoses of somatoform disorders.
[b]The percentage of patients with ICD-10 somatoform disorders who were rated by neurologists as having the criterion.
[c]The percentage of patients without ICD-10 somatoform disorders who were rated by neurologists as not having the criterion.

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Psychosomatic Diagnosis: a literature review.

Summary: Structural, epidemiological and medical biases tend to restrict the application of a proper diagnostic procedure in clinical practice. The aim of this review is to discuss factors affecting the occurrence, nature and presentation of psychosomatic diagnoses and their connotation with pejorative overtones. This paper investigated the multifactorial causes for these occurrences and revealed their implications for clinical management and medical research. It is suggested that psychological assessment should be provided in the early stages of the clinical investigation and some diagnoses should be reintroduced in current psychiatric classifications. Pejorative overtone has also been attributed to several diagnoses, particularly those related to somatisation. The use of moral remarks added to medical diagnosis should be banned from literature since it has distorted both practice and statistical results.

Introduction

Physical symptoms in the absence of an identifiable organic correlation are common in medical practice (Shepherd *et al.*, 1966; Von Korff *et al.*, 1988; Kroenke & Magelsdorff, 1989; Mayou, 1991). Many complaints of chronic pain, dyspepsia, headache, dyspnea, joint pain, chest pain, causalgia, dizziness, loss of function, palpitations and fatigue fall into this category. About 30% of these symptoms were attributed by the attending physician to "psychiatric disturbance" (Bain & Spaulding, 1967; Lipowski, 1988).

The medical profession finds these conditions difficult to describe with a satisfactory term. Those that have been used included non-specific, non-organic, functional, dysfunctional and idiopathic somatic symptoms. These "diagnostic puzzles" are difficult to treat. They are usually accompanied by over-investigation and consume considerable medical resources to little benefit. Independent of physical findings or excessive preoccupation with normal physiological signs, the progression of this expensive investigative process leads these initial unexplained medical symptoms to be later classified as functional or as psychological overlay. At other times these symptoms are categorized as psychogenic, psychosomatic, conversion, somatisation, somatoform disorder and hysteria. Usually, these labels are accompanied by the absence of physiological correlates, obvious pathological explanation or physical and laboratory findings. Applied to patients with a similar clinical picture, the term hypochondriasis emphasizes beliefs concerning illness rather than symptoms (Kellner, 1985). Other terms, like atypical or non-specific and functional somatic symptoms, are concerned with somatic symptoms that do not result from physical disease, but often originates from normal bodily sensations or some sort of autonomic dysfunction in its association with cognitive distortion. The majority of these labels have been seen by the medical profession as having a "pejorative overtone" (Sharpe *et al.*, 1992; Mayou 1975; 1976; 1991). The aim of this paper is to review the basis for these medical diagnoses and

to investigate why they became associated with such a depreciative adjective.

Historical perspective

For centuries, physicians have recognized somatising patients. They have given a variety of overlapping labels that were some times inter-changeable. This was particularly true to the terms "hysteria" and "hypochondriasis" (Veith, 1965; Fisher-Homberger, 1972; Boss, 1979) which originated from the Greek language.

Sydenham's Dissertation of 1682 represents a landmark in the evolution of medical thought about these diagnoses. He provided a description of the physical and mental symptoms of these disorders. Hysteria often exhibited a single symptom and was viewed as a malady of women. Hypochondriasis included a wide range of symptoms, which could imitate many diseases and prevailed in males. They were both considered a disturbance of the mind and an inconsistency of the body due to disorders of "animal spirits".

In 1733, Cheyne pointed out the difficulties in giving this diagnosis for patients in medical settings and how his colleagues attributed to them and to him "pejorative overtones"

Nervous distempers are under some kind of disgrace and imputations in the opinion of the Vulgar and Unlearned. They pass among the multitude for a lower degree lunacy. Often when I have been consulted in a case, and found it to be what is commonly called "nervous". I have been in the utmost difficulty when desired to define or name the distemper. If I called the case glandular with nervous symptoms (actual psychological overlay), they concluded I thought them pox'd or had the King's Evil. If I said it was vapour, hysteric or hypochondriacal disorders (actual somatisation), they thought I called them mad or fantastical (symptom in their minds) and was thought as rude, a fool, a weak and ignorant coxcomb, and perhaps dismissed in scorn form seeming to impeach their courage. Notwithstanding all this, the disease is as much a bodily distemper as the smallpox or a fever and I think never happens to any but those of the liveliest and quickest natural parts and particularly where there is the most delicate sensation and taste, both of pleasure and pain (p13).

Of course, it could be argued that words like hysteria and hypochondriasis do not in fact have the same meaning in today’s terminology. However, no matter what one calls these symptoms, the point to be stressed is that they were not explained physically and possibly had a psychological origin.

In 1799, Sims explicitly distinguished hysteria from hypochondriasis by pointing out the new feature of disease conviction. In hypochondriasis, patients had their mind "almost entirely taken up with the state of their health, which they imagine to be infinitely worse that it is and believed themselves afflicted with almost every disorder they have ever seen, read or heard of" (Lipowski, 1988). Hysteria was associated with changeable mood, while hypochondriasis resembled melancholia. Therefore, Sims described in these clinical pictures symptoms that we now associate with those of anxiety and depression.

Although described in obsolete words, the clinical cases reported above can easily be recognized in the actual medical practice. This suggests that culture, sociofamily factors and behavior can change over the centuries, but the influence of mental mechanisms on psychosomatic illness remains essential.

Hysteria and hypochondriasis may be viewed nowadays as the prototypes of somatoform disorders, which the DSM-III stated as "physical symptoms suggesting physical disorders". Moreover these entities represent prime examples of an abnormal body experience (somatisation) and of an abnormal cognitive disturbance (obsessional thoughts), respectively.

Epidemiology

The frequency of the somatisation may increase with the increase of medical care (White, 1961; Lipowski, 1988). Population surveys (Wadsworth *et al,* 1972) demonstrate that minor bodily symptoms are very common and that only a small proportion of them are reported to doctors. Studies in primary practice are particularly important. The 1980-81 National Ambulatory Medical Care survey in the US looked at about 90 000 visits to physicians and found that 72% of the patients who received psychiatric diagnosis had one or more physical symptoms for their complaints (Shurman *et al.*, 1985). In general practice, one fifth of all attendees present with physical symptoms of minor emotional disorder (Shepherd *et al,* 1966; Mayou, 1976, 1991). Psychiatrist who work in general hospitals consistently report hysterical symptoms as being more common than do their colleagues with more limited specialist experience (Mayou, 1975). At the secondary level of medical care the frequency of somatisation may be even higher. For example, in the UK during 1985 there were 330,000 hospital referrals for back pain, and 63,000 hospital in-patient treatments averaging 2 weeks. Of these patients, 60% had non-specific backache. The etiology of these cases were still not diagnosed by discharge.(Lipton & Jones, 1987). The problem

for diagnosing these cases is related in part to the delay in obtaining results from investigations and to the lack of training in psychological medicine. However, it may also be related to the intrinsic difficulty of providing a psychiatric diagnosis to patients who communicate their psychological distress through physical symptoms. This way of describing somatisation has been criticized for failing to reveal the crucial point that somatisers actually experience and communicate primarily somatic, not psychological, distress and this is the main feature that characterizes them. It is important to examine why this became so.

Somatisation in epidemiological research

Several authors have pointed out the influence of culture in the presentation of somatisation (Slater 1965, Carter 1972, Hare, 1974, Mayou, 1975). Others reported on social and cultural differences on the prevalence of mental illness and alcoholism at specific locations (Bebbington *et al*, 1981, Goodman, 1981, Cooper, 1987). Yet the influence of sociofamilial and cultural factors are directly related to the level of care in which the research is conducted. This may be particularly relevant since somatisation varies from culture to culture.

There is growing evidence that the prevalence of illnesses in the general population differs from primary and secondary care (Mayou, 1975, 1976, Lipowski, 1988). Thus in *general populations* there are individuals who seek care and those who do not, even with the same sort of diagnosis.

Those who reach *primary care* are more concerned with their symptoms, which they perceive as serious and they may insist in having further investigations. Usually, at this level an organic diagnosis is given, distinguishing cases from non-cases. Patients with organic pathology or functional somatic symptoms are likely to be identified as such, and they normally disclose functional illness behavior by accepting the doctor's guidance and reassurance. Investigations and proper counseling often remove the patient's psychological concern and any tendency towards unreasonable illness behavior. However, in some non-diagnosable cases, a mixture of organic and non-organic factors are usually present.

These patients are often referred to specialized services for further investigations, either in general hospitals or specialized clinics e.g. rheumatology or orthopedics. Thus, in *secondary care level*, psychological overlay remains frequent. Some patients, most of who lack a conclusive diagnosis and show dysfunctional illness behavior, become persistent attendees and are often

referred to *tertiary care*.

These cases are usually characterized by previous negative investigations and repeated failure to respond to treatment in previous levels of care. They are often seen in highly specialized services and the distinction made in this level is between somatisers and non-somatisers. The possible higher frequency of somatisation in these patients is often misinterpreted as bias in their referrals and study results are eventually dismissed because of "selected patient samples" (Mayou, 1976, 1994, 1996). Nevertheless, the presence of somatisation in tertiary level of care (e.g. Pain Clinics, Neurologic Clinics, Psychiatry) might be expected to be higher than in primary care, and certainly higher than in the general population. It is this process of psychological escalation that possibly reduces differences in rates of somatisation between samples from different cultures. Psychological enhancement is relevant to epidemiological research because psychosocial and family factors may be investigated on persistent chronic attendees. It is likely that the influence of psychological factors is indeed greater in tertiary than in previous levels of care. This would explain the different results from studies in those levels in which organic pathology and minor problems are likely to be identified and treated (Barker & Mayou, 1992, Mayou *et al*, 1993, Mayou, 1993, 1995).

Social and medical problems in assigning a psychosomatic diagnosis

The reluctance of patients to verbalize their psychological distress to doctors or to communicate them through physical complaints probably results from a multifactorial interaction that includes personal, medical, sociofamily and cultural aspects. This psychosomatic language of distress appears to be prevalent in cultures (e.g. Western societies) where expression of emotional distress in psychological terms is socially unacceptable or family inhibited (Kirmayer, 1984; Lipowski, 1988) or where communication of emotional distress in a somatic form is encouraged (Zborowsky, 1956). The latter examined the degree of mother over protection and concern in Italian and Jewish communities in New York.

Crying in complaint is responded to by parents with sympathy, concern and help. By their over-protective and worried attitude they foster complaining and tears. The child learns to pay attention to each painful experience and to look for help and sympathy which is readily given to him. In Jewish families, where not only a slight sensation of pain but also each deviation from

the child's normal behavior is looked upon as a sign of illness, the child is prone to acquire anxieties with regard to the meaning and significance of these manifestations (p. 28).
Zborowsky presents something of a caricature. However, his view of somatic complaints as inherent in the familial response to the child's health and illness is an important insight.

Other factors operate in doctor-patient relationships. These may explain why so many patients are discharged from hospital without proper diagnosis and treatment. The way care is provided associated with medical biases usually interferes with the mechanism of providing an adequate diagnosis. Few specialists are really concerned with careful diagnostic discrimination between organic and non-organic illness (Campero *et al.*, 1993; Verdugo & Ochoa, 1992,1994; Ochoa *et al.*, 1993, 1995, 1997).

In structural terms, the physician first sees the patient without a psychological and social assessment. At the beginning of the clinical investigation, there is a tendency to provide an organic label however tenuous. Although a psychologist or psychiatrist usually provides a diagnosis of somatisation, patients are only referred to them after receiving a provisional diagnosis. This way of referring not only crystallizes the idea of an organic illness but the psychiatrist is then unable to exclude it. The mental health professional only *adds* a psychological component, thus suggesting functional overlay instead of a more refined diagnosis indicating the type of somatisation. This is reinforced by the fact that most patients are obviously not mentally ill and do not fit into major psychiatric diagnostic categories. Finally, when patients ‘ crystallize’ the idea of an organic illness they tend to become much less tolerant of psychological assessment and reluctant to consider a psychological origin for their unexplained symptoms. As a result, we end up with a false impression that conversion and somatoform disorders are very rare.

The term *crystallization* can be employed to qualify a specific interaction between doctor and patient. In this relationship the doctor induces the patient to think he or she has a physical illness by the overuse of physical investigations or by suggesting the possibility of an organic illness by using medical terminology to explain findings which are not strictly related to the cause of the symptoms. The usual way to reinforce *crystallization* is to provide invasive treatment (e.g. nerve block, injection, and denervation) without a proper psychological assessment. This reinforcement also occurs with unnecessary drug prescription (Mayou, 1991, 1993) and unnecessary surgical intervention (Barker & Mayou, 1992). These authors found that patients who had normal

appendices removed had a worse prognosis than comparisons. In the year following admission, those patients presented recurrent and disabling pain associated with continued psychiatric symptoms.

The consequence of this medical bias, whereby the idea of an organic illness is made concrete to the patient, leads to several problems. Firstly, inducing misdiagnosis. This includes prevention of a proper organic diagnosis, bias in psychiatric assessment away from somatoform categories and invalid research results. The impediment of a correct psychiatric diagnosis and its subsequent treatment leads to poor clinical response and increases patient dissatisfaction with staff. Secondly, it increases long lasting and unrealistic treatments and an expansion in the take-up of social benefits. Chronic undiagnosed somatisers with an organic label are now more commonly seen than misdiagnosed hysteria with real organic pathology (Quill, 1985; Ron, 1994; De Lemos, 1997).

Diagnoses that are not objectively verifiable often condemn patients to chronicity and iatrogenesis (Ochoa, 1993, 1997). It seems that doctors' abnormal diagnostic behavior leads to patients' abnormal illness behavior that is compounded by abnormal treatment behavior (Awerbuch, 1985; Ochoa, 1997). To prevent these problems, psychological assessment should be provided much earlier in the clinical investigation, preferably before the allocation of a provisional organic diagnosis.

Professional bias

In clinical practice, the act of diagnosis is a professional process by which signs and symptoms inform a diagnostic dimension or category. If patients do not respond to treatment, the diagnosis is reconsidered and so is the treatment. In this way, after some time, the doctor can be close to the clinical reality of the patient, considering the physical, psychological, somatopsychic or psychosomatic pain.

The value of a diagnosis is that if the theory underlying it is scientifically sound, it implies the course the doctor is to follow. Thus it should provide a useful prediction for the course of the disease. This will imply its etiology, and perhaps most importantly of all, will suggest the most appropriate treatment. However, somatisation poses serious problems for diagnosis. The complexities of mechanisms involved in the production of a clinical symptom lead to a great

variety of characteristics that may not always be fully attributable. To understand somatisation and classify the symptom rationally, the physician must be aware of a range of clinical and psychological factors.

In terms of secondary and tertiary levels of care, a doctor's specialty may also introduce some bias on his or her conclusions about diagnostic adequacy, allowing room for idiosyncrasy. Twycross (1990) trying to understand pain in cancer patients, proposed the concept of *informed imagination*, whereby a symptom that is not readily classified will still be understood as part of a system of medical knowledge, and an attempt will be made to establish a diagnosis, however conjectural. To this concept can be added the complimentary notion of *concept extension* which describes an attitude whereby the diagnostic concept is extended to encompass symptoms that do not fit the classification.

In multidisciplinary pain clinics, for example, there is a tendency to extend the organic classification to include those patients with some sort of somatisation (e.g. atypical facial pain, pre-trigeminal neuralgia, sympathetic maintained pain) or to create new diagnoses in descriptive terms based almost exclusively on clinical examination (e.g. fybromyalgia, chronic fatigue syndrome, repetitive strain injury). The patient is therefore labeled with a diagnosis, which has no cause, no confirmed physiopathological mechanism and no laboratory findings to measure its progress (Croft *et al.*, 1994). Usually response to treatment does not include placebo comparison, making clinical conclusions rather unreliable (Campero *et al.*, 1993; Verdugo & Ochoa, 1994). The result is a hypothetical organic illness and an ill-formulated management that precludes a proper organic or psychological diagnosis and proper treatment.

The extended concept can be considered as a process of professional distortion, which can lead to specific views about the patient's symptomatology, either considering every symptom as purely physical or as mainly psychological. The psychologist and psychiatrist may be equally guilty when they extend the concept of some psychological illnesses solely because of negative physical findings. Whilst informed imagination is a more neutral, scientific and operational way of thinking, it is still based on medical theories which by and large do not include psychological theories. In any case, the doctor’s aim should be to discover the *clinical reality*, in other words the patient's pathology. However, the diagnostic label depends not only upon the apparent seriousness and nature of the clinical picture, but upon the attitude and behavior of patients and

the demands they makes upon doctors (Mayou, 1976).

Psychosomatic presentation

Difficulty in properly classifying cases with somatisation is an old problem. Several structural, social, medical and professional biases interfere with the appropriateness of medical diagnoses, particularly those related to the psychosomatic encroachment. Doctors may induce *crystallization*. Their choice for a specific theoretical approach may lead to different interpretation of unexplained physical symptoms and would promote changes in diagnostic classification. These factors have also contributed to exclude or to re-include some psychosomatic diagnoses in some of the most used psychiatric classifications (e.g. DSM-III, DSM-III-R and DSM-IV).

Independent of cognitive distortion and its association with psychophysiological mechanisms, as happens to Functional Somatic Symptoms, there may be other explanations for the emergence of unexplained somatic symptoms. It is possible that some of these symptoms arise from mental processes, no matter how these symptoms are labeled (e.g. somatoform, somatisation, psychosomatic, psychogenic or conversion). The inquest on why some individuals somatise more often than others and why somatisation frequently recurs imitating other psychiatric disorders supports the argument in favor of the involvement of mental processes in the genesis of some types of somatisation. This is even more so in cases with psychogenic pain, where these mental processes probably derived from the interaction between traumatic childhood experiences and a variety of other social factors (Roy, 1992; De Lemos, 1997). There are some additional reasons to support the relevance of mental mechanisms in these cases (De Lemos, 1997; Ochoa, 1997).

The former studied the impact of family atmosphere in chronic pain maintenance for 10 years and investigated whether psychogenic pain was a myth or reality. This study (not published in part because it's pejorative overtones) showed that the concept of psychogenic pain is a valid and useful concept. Patients with this type of pain were distinguished formally using discriminant function analysis including *any* or *all* of its phenomenological, behavioral and etiological characteristics. Using *all* the criteria reflects the clinician's ability to make the distinction between non-organic and organic pain. Being able to make it almost as well *on single* grounds also suggests that psychogenic pain is a *useful* category as well as consistent one. The latter was concerned to identify neurological inconsistencies that could be attributable to mental

mechanisms.

Several studies have addressed the issue of co-morbidity of mood disorder and chronic pain (Chaturvedi, 1987; Marshal *et al.*, 1992); Sullivan *et al.*, 1992). However, when a proper distinction between non-organic and organic pain is made (Peyrot *et al.*, 1993; De Lemos, 1997), depression and anxiety do not correlate with pain type. The latter study also showed that depression arises from pain rather than the reverse. Thus, there is weak support for considering psychogenic pain as a process influenced or generated by affective disturbance.

Excessive preoccupation with normal sensations (heart beat), functional bodily manifestations (palpitation) associated with the wrong interpretation of these autonomic symptoms have been considered as being important in the emergence of somatisation (Sharpe *et al.* 1992). This cognitive-behavioral model of etiology distinguishes between predisposing, precipitating and perpetuating factors and great emphasis is given to body surveillance and cognitive distortions as "barriers to recovery". Trying to explain somatisation, Mayou (1993) gives the following example.

A middle aged man with bad family history of heart disease may present with chest wall pain which he has misinterpreted as evidence of heart failure shortly after hearing the death of a close friend from heart attack (p.75).

In this example, in contrast with other descriptions, there is virtually no physiological link between the underlying heart function and chest wall pain, thus the model relies heavily on cognitive distortion. Moreover, it was derived from studies on recurrent acute pain rather than chronic fluctuating pain. In previous descriptions (Mayou, 1976), the model was anchored in an experiential aspect. Cognitive disturbance (chest wall pain) should be preceded by an over concern with a past episode of minor injury/illness (angina) and triggered by an autonomic dysfunction (tachycardia) associated with mood disorder (anxiety) and other complimentary factors (caffeine, nicotine).

Surely this model explains the emergence of some conscious (or pre-conscious) functional somatic symptoms. However, it does not explain why in some cases these somatic misinterpretations do not change after proper medical clarification. This reluctance probably indicates an unconscious component. The model also does not explain why some of these somatic complaints follow the pattern of recurrent psychiatric syndromes. Conversely to other non

relenting sensations, pain is not a physiological bodily function and does not have a correlate to be monitorised as happens to other bodily sensations (e.g. digestion, heartbeat or breathing). Thus, the behavioral model may not be applicable for the majority of chronic pain. Particularly in those cases where the onset of pain is followed by mild accidents incapable of accounting for autonomic nerve dysfunction or when it appears for no apparent reason. An observational study (De Lemos, 1997), conducted in a tertiary care level, showed no significant differences in social modeling in patients with psychogenic pain. Again there is little evidence to consider inexplicable pain as a functional somatic symptom or to assume that it is other than an expression of a mental process which may be in part unconscious and for this reason more resistant to clarification.

Psychosomatic diagnosis

Early this century, Freud who attributed the emergence of somatic symptoms to unconscious mental processes accepted the term hysteria. His views were mainly based on the psychiatric approach that prevailed at that time and derived from the hypnotic treatment that was routinely employed on hysterical cases (Freud & Breuer, 1895). Hysterical symptoms were conceived as resulting from the mental mechanism of dissociation and subsequent conversion. Conversion was understood as a displacement of mental energy towards the body to avoid unbearable mental stress. The clinical picture of these cases included personal suggestibility, loss of bodily functions and denial. The psychological sources of the pseudo neurologic hysterical symptoms were demonstrated by the use of hypnotic therapy.

One or two decades later, the term *somatisation* was introduced by one of his followers - Stekel - to described a more transient syndrome characterized by multiple symptomatology whereby a serious neurosis could promote bodily disorder. This new term was slightly different from the concept of conversion. These multiple symptoms were usually treated by dynamic psychotherapy (Hinsie & Campbell, 1960; Lipowski, 1988). Alexander *et al.* (1934) was concerned with the specificity of psychological factors and mental mechanisms in some illnesses regarded as psychosomatic. Following a similar theoretical approach, Menninger (1947) extended the concept of somatisation by defining "somatisation reactions" as the "visceral expressions of the anxiety which is thereby prevented from being conscious". Thus, the somatic symptom was not seen any more as a neurotic trait but as a result of a state of mind. In general terms,

psychoanalysis has used the concept of somatisation to indicate an unconscious defense mechanism and to address certain somatic complaints to a hypothesized psychogenic source. This view is not shared by some psychiatrists and is often regarded as unacceptable for those who are not psychoanalysts. They would favor a more neutral and descriptive approach that does not imply an etiologic mechanism. However, this approach is often conceived in relation to other theoretical approaches (e.g. cognitive-behavioral) and frequently includes some alternative explanation of the genesis of somatic symptoms (Sharpe *et al*, 1992).

Somatisation could be viewed as a tendency to experience and communicate somatic distress by patients that disclosed a specific clinical pattern. Their symptoms are unaccountable by pathological findings; they are attributed to physical illness, and they make patients seek medical help.

Bridges and Goldberg (1985), for example, stipulated that to be considered somatisers patients must meet the following criteria. They must seek medical help for somatic symptoms (and not for psychological manifestations of psychiatric disorders); must attribute their symptoms to physical illness, and must report, when properly interviewed, symptoms that justify psychiatric diagnosis. Although the patient seldom agrees, some physicians usually interpret somatisation as a response to psychosocial stress. Others would rather describe somatisation in terms of somatisation behavior, best seen as a behavioral syndrome (Barker & Mayou, 1992). For these authors, somatisation is understood as a wide concept that includes a wide variety of different physical complaints not normally associated with physical illness.

The general assumption of this approach (Sharpe *et al.*, 1992; Mayou, 1993; 1995) is that the presentation of somatic symptoms is originally related to mild psychophysiological mechanisms or minor physical illnesses, which are later misinterpreted through cognitive disturbance and influenced by concomitant mood disorder. The clinical picture is maintained or perpetuated by a reciprocal interaction between the patient's illness behavior and the reinforcing attitude of those who care for the patient (including doctors). Great emphasis is given to three aspects of the somatisation process: *experiential, cognitive and environmental.* The experiential aspect is what individuals perceive in regard to their bodies. It includes *transient changes in body functioning, non-specific psychological dysfunction* and *persistent physical complaints.* Examples of *transient changes in body functioning* are tachycardia, dyspepsia, dyspnea, headaches, asthenia, and

dizziness. Examples of *non-specific psychological dysfunction* are irritability, insomnia, lethargy, anxiety, depression, and fatigue. Examples of *persistent physical complaints* are chronic pains, such as atypical facial pain, chronic dysfunctions (e.g. irritable bowel syndrome), chronic loss of a function (e.g. vision, voice or hypoalgesia) and changes in appearance (e.g. dysmorphophobia.) These bodily complaints are *all* seen as variants presented in somatisation. However, these complaints are related to different concepts and completely different etiologic mechanisms for each symptom. While transient change and psychological dysfunction can be reasonably attributed to the emergence of functional somatic symptoms, chronic persistent complaints are more likely to be the expression of more serious types of somatisation (e.g. somatoform disorders, conversion) and serious cognitive distortion as sometimes seen in schizophrenia (e.g. dysmorphophobia).

This tendency to encompass a variety of experiential complaints under the rubric of somatisation is shared by most behavioral psychiatrists and it is in part responsible for weakening the frequency of "psychogenic" syndromes in clinical practice, which they regard as "pejorative" (best not being verbalized to patients).

At the end of the last century, however, the diagnosis of hysteria was fashionable and it was dropped from the DSM-III a couple of years ago. Conversely, hypochondriasis was largely displaced by the concept of neurasthenia at that time, but it was reincluded in the DSM-III (Fisher-Homberger, 1972) although considered by some psychiatrists as a *continuum* or as a reaction (Mayou, 1976). Recently, in the DSM-IV the previous diagnosis of Somatoform Pain Disorder was jettisoned and substituted by the diagnosis of Pain Disorder, in which both organic and non-organic aspects of pain should be present. This doubtful approach now makes chronic pain, as an isolated somatisation, impossible to be diagnosed on its own. Thus, "psychogenic" pain should now be included in more restricted categories (e.g. somatoform disorder or conversion), which are difficult to diagnose due to a great number of psychological and physical characteristics that patients must present. The actual diagnostic criteria reduce the prevalence and incidence of “psychogenic pain”.

Nevertheless, several authors still emphasize the importance of the concept of "psychogenic" pain, both in Pain Clinics and neurologic practice (Quill, 1985; Lipowski, 1988; Ron, 1994; Ochoa, 1997). Moreover, the theoretical or terminological reluctance to accept psychogenic diagnoses usually leads to a variety of invasive and surgical treatments with serious iatrogenic

results (Verdugo & Ochoa, 1992; Ochoa, 1995, 1997).

Independent of these "vagaries" of psychiatric nosology and terminology (Lipowski, 1988), changes in medical diagnosis are still very much with us. They raise questions as to what extent we are providing new diagnoses for old diseases (Mayou, 1994; Pearce, 1994). Also to what extent we are altering operational criteria to exclude or include some diagnoses according to underlying medical theories or fashion. It is questionable that these alterations really represent an evolution in the psychological comprehension of unexplained physical symptoms, because they do not take into consideration the meaning of somatisation and its connection with early family disruptive events.

Even accepting the view that favors the use of operational criteria based on symptoms instead of etiology to classify illnesses, new descriptive terminology does not essentially alter the clinical presentation of some ancient illnesses like hysteria or hypochondriasis. Irrespective of the DSM's nomenclature, fashionable diagnoses (and treatments) tend to be transient, while old consistent medical entities are likely to remain. Although some changes in psychiatric classification can be attributed to divergences about the importance given to mental mechanisms involved in somatisation, the great majority of these changes may simply be associated with the way health services and medical care are provided and utilized by patients.

Lets now look how the medical profession and society have enhanced, distorted and applied concepts that were originally developed inside a specific domain, and why this wider use led to the attachment of "pejorative overtones" to some diagnoses.

The pejorative connotation

In the past, pejorative tones attributed to doctors who used certain diagnostic labels could be interpreted in two different ways. It may be related to the insistence of patients to obtain help for disturbance in "animal spirits" or "vapours" in the medical setting, or it may be seen as a reluctance by doctors in accepting the psychological emergency of symptoms in a somatic form.

Today, this dichotomy between diseases regarded as originating from the body versus illnesses mainly attributed to the mind still persists. This controversy is additionally complicated by different psychiatric views towards mental illness and its association with abnormal somatic symptoms. As mentioned before, some psychiatrists tend to disagree with any terminology or

diagnostic label that implies an implicit mental mechanism or etiological assumption.

In the psychological treatment of patients with functional somatic symptoms, Sharpe *et al* 1992 stated.

Existing terms are unsatisfactory because they are used imprecisely, may convey etiological assumptions (such as somatisation), and have a pejorative overtone (p. 516).

However, the decision to assign negative adjectives to a noun is rather subjective. It depends very much on thoughts, beliefs and assumptions of those who employ them. On occasions, these adjectives reflect a moral perspective, other times they reveal the theoretical perspective of the physician or the psychiatrist who use them. There is, however, scarce scientific basis for their use.

The social appropriation of diagnostic concepts

It is well known that psychiatric terminology may spread to outside the medical field and would sometimes be employed with a completely different meaning or connotation. Thus, a label originally developed inside the psychiatric field is often slightly modified by the medical profession, misused at family level and finally applied to a wider sociocultural phenomenon. The concepts of hysteria and hypochondriasis will be regarded as paramount to exemplify the effects of this social dimension on diagnostic conceptualization.

Although both hysteria and hypochondriasis are persistent clinical entities, they have not been immune to the influence of fashionable medical theories. Psychoanalysis conceived hysteria as a result of a mental process characterized by the presence of specific mechanisms. First, conversion that meant the displacement of mental energy towards the body. Second, dissociation implying in a mental mechanism that prevents an association with a past memory. Third, the presence of psychological denial related to the origin of the symptom. Thus, a psychosomatic diagnosis was interpreted as a difficulty to communicate psychological distress (Balint, 1957).

Psychiatric appropriation followed and hysteria was then postulated as a psychiatric syndrome and later as a reaction, only to be recently dropped from psychiatric classifications and understood as a form of somatisation. The original diagnosis of hysteria suffered several changes. At medical level, the expression was applied to a variety of unexplained somatic symptoms, particularly those in which a dysfunction (e.g. hysterical cough) or a loss of function (e.g. hysterical paralysis) was involved. At family level, the term hysteria acquired a different

connotation either to describe one's tantrums accompanied by unreasonable complaints, or the angry behavior of a child. As shown, there was a substantial change in its conceptualization. At a sociocultural level, hysteria has also been identified with a variety of cultural phenomena, including "possession experience", collective panic, witchcraft and collective suggestibility (e.g. mesmerism). However, there is almost no connection between these social appropriations of the term and the original concept designed to explain non-neurological bodily symptoms. For example, one could have been accused of witchcraft simply because he/she had not agreed with the prevailing religious views even in the absence of any physical symptomatology. The same is valid to the possession experience and to collective panic. Once a psychiatric term has been displaced from its original context and socially relocated, the term loses its original meaning. It then does not retain its prime application and becomes easily tarnished with "pejorative" overtones.

The same course observed in hysteria can also be elicited in the meanders imposed to the diagnosis of hypochondriasis. Psychoanalytically, it was regarded as resulting from obsessional thoughts towards somatic symptoms that were understood as a result of a disease conviction resistant to medical clarification. Psychiatry utilized the concept of hypochondriasis as a syndrome, which disappeared from early classifications to return one century latter in the DSM-III (Fisher-Homberger, 1972). The medical appropriation of the expression was applied to multiple non-specific somatic complaints sometimes accompanied by low mood. At family level, the expression was used to denote one's seeking attention through illness behavior, associated with unreasonable recurrent malaise. Modifications of the original conceptualization can be easily recognized. At medical level, the susceptibility of medical students to imitate some organic diagnosis has been considered as an indication of hypochondriasis. At the social level, the term has also been applied to medication addiction or to the recurrent pursuit of surgical intervention. Again there is little connection between these new uses of the term and the original concept.

As happened to hypochondriasis, the term phobia followed a similar trajectory. In psychoanalysis it was used to indicate a defense mechanism against separation anxiety. At psychiatric level it was associated to obsessional thoughts in anxious patients. In its usual form, it was seen as a psychiatric syndrome, mainly related to avoidance of open spaces or crowds (e.g. agoraphobia). The management of neurotic problems in medical practice used the expression in

relation to cases characterized by the association of somatic symptoms with fear, either in terms of recurrent non-specific complaints (e.g. fear of disease), persistent specific complaints (e.g. dysmorphophobia) or in terms of treatment fear (e.g. needlephobia). The concept of disease fear can be seen as the reverse of disease conviction, while fear of treatment can be interpreted as a "barrier to recovery". At social levels, phobia acquired a variety of meanings including fear of work (e.g. workphobia), homosexuals (e.g. homophobia) and religious fears that led to mystical rituals. The majority of them have acquired pejorative connotations.

The social appropriation of these three diagnostic categories, and the conceptual distortion that followed, has made it hard for doctors to communicate them to patients, either because of the medical setting in which these psychiatric terms were provided or the superinposed "pejorative" overtones that they socially acquired.

In general terms, diagnoses should be given based on scientific accuracy and not on moral grounds. Is calling patients schizophrenic less pejorative than saying they have a somatoform disorder? Let’s examine what is essentially pejorative.

The problem of assigning an acceptable psychosomatic diagnosis in medical practice is complex. In structural terms, services provided in the medical setting do not include early psychological assessment. This makes the task of assigning the diagnosis and explaining the reasons for the patient's somatisation more difficult. Doctors tend to consider all patients as medical cases when in fact some of them are not, although they would like to be. Frequently, the diagnosis of hysteria and hypochondriasis may be misdirected because doctors instead of mental health professionals see the great majority of somatisers. Moreover, doctors tend to undermine the scientific validity and clinical reliability of diagnoses placed in the limit of the psychosomatic boundary (Ron, 1994; Ochoa, 1997). There is also a tendency to consider any non-organic diagnosis as "pejorative". Even though doctors have the intention to respect or to please patients, *any* diagnosis that does not describe an organic cause they would find unsatisfactory. Doctors assume that these diagnoses imply labeling reasonable patients mad. To prevent the maintenance of this problem, psychological examination should be considered essential in health care and multidisciplinary clinics should be extended to the majority of medical subspecialties. Such a policy would promote more rational distribution of existent resources and greater financial benefit for health care.

A further complication in assigning the right diagnosis is posed by family and social appropriation of psychiatric terminology, particularly when these terms have been consistently attributed to child behavior. This appropriation reinforces the impact of a "pejorative" tone in psychosomatic diagnoses, which make it extremely difficult to employ terms like hysterical or hypochondriac in the medical setting.

In addition, the indiscriminate acceptance of "pejorative tones" in relation to some psychosomatic diagnoses has created serious problems in conducting clinical investigation and interpreting statistical results. Psychosomatic categories tend to be regarded with suspicion. Research projects to investigate these diagnoses may be refused by ethical committees on the same basis. If the investigation is eventually approved, results showing prevalence of "psychogenic" illnesses in tertiary care (e.g. Pain Clinics) would not be easily accepted for publication.

Refusal for publication is also common where experimental studies obtain negative results. Prof. Everitt, professor of statistics of the Institute of Psychiatry in the Maudsley Hospital, is aware of this selective effect and its impact on research results, particularly those from metanalysis.

Clinical trials which fail to show any (treatment) differences are less likely to be published owing to investigators not writing up the results or to journals declining the paper. Conclusions of the therapeutic effectiveness based on reviews of only the published papers may consequently be seriously misleading. (p. 51).

Assessment of reviews on "medicine-based-evidence" about the prevalence of “psychogenic” entities would often be equally misleading. Therefore, the overall impression obtained from literature may become detached from actual medical practice. This gap could lead to distortions of health strategies. Although claiming to be on behalf of patients, a silent non-scientific censorship that in part relies on medical prejudice leads to lack of information and would continue to demean some consistent diagnostic categories because of their "pejorative" tones.

Conclusion

Taking into account data on prevalence, its relation to how diagnoses are conceived or allocated combined with the way health services are provided, we are now in a position to understand better the process of somatisation and to draw reasonable conclusions about it. As happens in depression

and anxiety, somatisation should be understood as a "continuum". In the general population there is a higher proportion of transient non-specific physical symptoms (indigestion, constipation, bloating stomach, cough, palpitation, headaches, asthenia, dizziness), while in primary care they tend to be more persistent and are usually associated with autonomic dysfunction (e.g. dyspepsia, dyspnea, tachycardia, insomnia). For those who seek medical attention in these levels, the cognitive-behavioral treatment seems appropriate for these syndromes because severe cognitive dysfunction is unlikely to be present unless patients become persistent attendees.

As patients progress to higher levels of care, somatic complaints overlap with non-specific psychological dysfunction (e.g. irritability, insomnia, lethargy, anxiety, depression, fatigue) and are accompanied by higher levels of abnormal illness behavior. The implication here is that a serious cognitive distortion is in operation often co-existing with mood disorder that commonly derives from a primary somatic complaint. A more refined categorical distinction in the dimension of disease conviction should be considered to diagnose cases with somatisation in secondary level of care. Hypochondriasis is probably the term that best describes the majority of these cases.

The tertiary level of care, however, is characterized by the existence of more persistent chronic complaints (e.g. low back pain, atypical facial pain, chronic dysfunction or chronic loss of a function e.g. vision, voice or hypoalgesia) associated with specific psychological dysfunction (e.g. psychogenic pain, somatoform disorder or conversion). Complaints of pain in this somatic domain may be due to primary psychopathology (Derbyshire *et al.*, 1994). This process implies a more serious type of somatisation, which usually presents a poor response to treatment. These somatic complaints probably result from a variety of mental mechanisms, some of which are probably unconscious, triggered by family factors and usually expressed by specific psychological symptomatology (previous psychosomatic illness) or recurrent and persistent psychiatric illnesses (somatoform disorders).

In summary, somatisation should be regarded as a "continuum" and its clinical presentation probably progress in a *crescendo*. In the *general population*, there are non-specific symptoms associated with physical complaints. In *primary care,* the emergence of functional somatic symptoms associated with autonomic dysfunction is frequent. In *secondary levels of care,* clinical overlap between psychological and organic factors prevails and is often associated with cognitive distortions (e.g. degree of disease conviction). Finally in *tertiary care*, there is the presence of

persistent somatisation often associated with somatoform disorders and conversion.

Studies on prevalence show that functional somatic symptoms originating from minor injuries and organic pathology are higher in general population and primary care (Sharpe *et al*, 1992; Mayou, 1993). On the other hand, the association of somatisation with a higher degree of dysfunctional illness behaviors leads to a higher proportion of hypochondriacs and hysterics in secondary and tertiary levels of care (De Lemos, 1997). Possibly, some terms, such as hysteria and somatoform *pain* disorder, should be reinstated in the DSM to indicate those unexplained neurological cases associated with recurrent and persistent somatisation in tertiary level of care. These diagnoses should be considered as a discrete psychiatric entity. They would help to reestablish a common language among doctors and common sense in medical practice.

The attachment of "pejorative overtones" to psychogenic diagnostic categories is multifactorial. It frequently results from the interaction between moral, theoretical and social factors. The widespread appropriation of specific psychiatric labels has influenced and distorted the way diagnoses are verbalized in clinical practice and sanctioned in psychiatric classifications. It has generated vagaries in diagnostic conceptualization, although some of these psychiatric categories, such as hysteria and hypochondriasis, have consistently resisted the test of time without major changes in their clinical presentations.

On the other hand, the use of depreciative appraisal in relation to many unexplained somatic diagnoses has serious implications for the medical practice and the interpretation of statistical results. To date, bias in accepting papers for publication has distorted what is generally considered "medicine based evidence". To justify the use of "pejorative overtones" based on conclusions obtained from the current available literature is, therefore, flawed. To promote a realistic balance, the author supports the view that negative results and negative overtones (or its synonyms) superimposed to some psychosomatic diagnoses should not be regarded as a deterrent for publication. Moreover, pejorative connotations should be viewed as misleading and should be avoided in medical and psychiatric practice. They do not assist diagnosis or management of patients with unexplained somatic symptoms and their use will greatly distort the interpretation of results described in published papers.

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Article

A New, Empirically Established Hypochondriasis Diagnosis

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Objective: The narrow ICD-10 and DSM-IV definition of hypochondriasis makes it rarely used yet does not prevent extensive diagnosis overlap. This study identified a distinct hypochondriasis symptom cluster and defined diagnostic criteria.

Method: Consecutive patients (N=1,785) consulting primary care physicians for new illness were screened for somatization, anxiety, depression, and alcohol abuse. A stratified subgroup of 701 patients were interviewed with the Schedules for Clinical Assessment in Neuropsychiatry and questions addressing common hypochondriasis symptoms. Symptom patterns were analyzed by latent class analysis.

Results: Patients fell into three classes based on six symptoms: preoccupation with the idea of harboring an illness or with bodily function, rumination about illness, suggestibility, unrealistic fear of infection, fascination with medical information, and fear of prescribed medication. All symptoms, particularly rumination, were frequent in one of the classes. Classification allowed definition of new diagnostic criteria for hypochondriasis and division of the cases into "mild" and "severe." The weighted prevalence of severe cases was 9.5% versus 5.8% for DSM-IV hypochondriasis. Compared with DSM-IV hypochondriasis, this approach produced less overlap with other somatoform disorders, similar overlap with nonsomatoform psychiatric disorders, and similar assessments by primary care physicians. Severe cases of the new hypochondriasis lasted 2 or more years in 54.3% of the subjects and 1 month or less in 27.2%.

Conclusions: These results suggest that rumination about illness plus at least one of five other symptoms form a distinct diagnostic entity performing better than the current DSM-IV hypochondriasis diagnosis. However, these criteria are preliminary, awaiting cross-validation in other subject groups.

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Somatoform disorders are among the most prevalent psychiatric disorders. Beside the suffering inflicted on the patients, somatoform disorders impose a considerable financial burden on health care (1–10).

The study of these disorders is, however, hampered by the lack of valid and reliable diagnostic categories (11–15). The individual diagnoses are poorly and arbitrarily defined and thus overlap, while other disorders are so narrowly defined that the diagnoses can hardly be applied clinically, notably in primary care (4, 7, 16–18).

Diagnostic overlap is partly rooted in research methodological practices, such as the propensity of most investigators to single out a particular diagnosis for special study rather than adopting a comprehensive view that facilitates detection of overlap. Furthermore, the empirical foundation of the somatoform diagnoses is poor, as it emanates mainly from a clinical tradition based on observation of patients in severely skewed psychiatric settings, despite the fact that these patients are mainly encountered in general medical settings.

The main focus of this study was the diagnosis of hypochondriasis, the principal diagnostic criteria for which are, according to DSM-IV, a nondelusional preoccupation with fears of harboring a severe physical disease (criterion A), persistence of the preoccupation despite appropriate medical evaluation and reassurance (criterion B), clinically significant distress or interference with functioning (criterion D), and a duration of symptoms of at least 6 months (criterion E) (19, ICD-10). Criteria B and D also apply to several other somatoform disorder diagnoses, and criterion A is frequently seen in patients with other somatoform disorders, implying an overlap problem. Furthermore, Gureje et al. (17) showed that nearly no patients in primary care fulfilled the diagnostic criteria for hypochondriasis and that the major cause for this was criterion B, i.e., it is unusual for patients not to respond to reassurance at all despite appropriate medical evaluation. Criterion E is also problematic because the time limit is arbitrary and it limits the value of the diagnosis in nonpsychiatric settings, particularly primary care, where most patients whose illness lasts more than 6 months are viewed as having chronic disorders (16). Moreover, it is hardly possible to study the effect of early intervention when by definition a diagnosis cannot be made at an early stage. Thus, the current DSM-IV hypochondriasis diagnosis satisfies neither clinical nor nosological diagnostic validity requirements.

TABLE 1. Sociodemographic Characteristics of Primary Care Patients Who Did or Did Not Complete a Psychiatric Diagnostic Interview

Characteristic	Overall Group (N=1,785)	Patients Selected for Interview: Completed Interview (N=701)	Patients Selected for Interview: Declined Interview (N=193)	Difference		
	%	%	%	χ^2	df	p
Female gender	62.4	66.5	54.9	8.8	1	0.003
Work status				19.3	5	0.002
Employed	53.6	53.5	39.4			
Student	15.2	15.1	13.5			
Unemployed	5.6	8.3	4.7			
Pensioner	8.3	12.1	7.8			
Housewife or child care provider	2.3	2.3	1.6			
Unknown	15.1	8.7	33.2			
Living situation				79.7	3	<0.001
Alone	25.3	26.5	25.9			
With partner	56.8	62.1	38.4			
With parents	3.6	3.4	4.2			
Unknown	14.3	8.0	31.6			
Education				81.5	2	<0.001
Basic school (grades 7–10)	39.6	42.9	40.4			
Further education	45.6	48.6	26.9			
Unknown	14.9	8.4	32.6			
Vocational training				61.0	5	<0.001
Unskilled	17.0	18.7	16.6			
Skilled	23.8	24.5	19.7			
Formal education <4 years	20.7	22.7	13.0			
Formal education ≥4 years	9.0	9.7	3.6			
Other	10.6	11.7	11.4			
Unknown	18.8	12.7	35.8			
	Mean (SD)	Mean (SD)	Mean (SD)	z[a]	p	
Age (years)	38.8 (12.9)	40.5 (12.7)	37.6 (12.7)	–2.74	0.006	
	Median (Q1–Q3[b])	Median (Q1–Q3[b])	Median (Q1–Q3[b])	z[a]	p	
Screening scores						
SCL somatization subscale (23)	2 (0–3)	4 (2–5)	3 (1–5)	–2.49	0.02	
Seven-item Whiteley index (22)	0 (0–1)	1 (0–3)	1 (0–3)	–1.80	0.08	
Eight-item SCL (20, 21)	0 (0–1)	2 (0–4)	1 (0–3)	–0.97	0.34	
CAGE (24)	0 (0–0)					

[a] Mann-Whitney U.
[b] Q1 and Q3 are the 25th and 75th quartiles, respectively. The quartiles were used because the difference scores were highly skewed.

The present situation therefore leaves us with an urgent need for empirical nosological studies on hypochondriasis. The studies must draw on appropriate patient populations, state-of-the-art assessment methods, and advanced statistical aids. Such studies should seek both to validate the current hypochondriasis diagnosis and to systematically examine whether inclusion of other symptoms known to be associated with hypochondriasis improves the validity of the diagnostic criteria.

In the present study we aimed to fill this gap by investigating whether a select range of candidate symptoms for a new hypochondriasis diagnosis cluster in certain individuals and by proposing a distinct and discrete nosological disorder entity separable from other somatoform and psychiatric disorders.

Method

The study group included 1,785 consecutive patients of Scandinavian origin (ages 18–65 years) who consulted their primary care physicians during a 3-week period for new medical problems. All participants were covered by the National Health Care Program, which includes 98% of the Danish population; each individual is registered with one primary care physician. A patient can consult only the primary care physician with whom he or she is listed. With a few exceptions, e.g., emergency cases, almost all specialized treatment, including hospital admission, requires referral from the primary care physician.

The Danish Health Care System is almost entirely tax financed, and with a few exceptions, all medical care is free of charge.

The study was a part of a large randomized, controlled trial on the effect of educating primary care physicians about the treatment of somatizing patients and the effect of diagnostic aids. All 430 primary care physicians from the 271 practices in the county of Aarhus were invited to participate in the study, and 38 agreed. There were no statistical differences between the included primary care physicians and those who declined the invitation to participate as to type of practice, length of postgraduate psychiatric training, age, or gender. However, the participating primary care physicians had less experience (mean=10.3 years, SD=7.2) than the nonparticipants (mean=14.1 years, SD=8.5) (likelihood ratio test: χ^2=9.0, df=1, p=0.005), and more of them had participated in extended (more than 3 days) courses in communication skills and psychological therapy: 52.8% (19 of 36) versus 39.6% (113 of 285) (likelihood ratio test: χ^2=4.0, df=1, p=0.05).

TABLE 2. Diagnostic Classes Determined by Latent Class Analysis of Six Hypochondriasis-Related Symptoms in Primary Care Patients

Variable	Total Group (N=701)	Class 1: Hypochondriasis (N=102)	Class 2: Other Somatoform Disorders (N=244)	Class 3: Nonsomatoform Disorders (N=355)	DSM-IV Hypochondriasis (N=59)[a]
	%	Probability of Class Membership[b]	Probability of Class Membership[b]	Probability of Class Membership[b]	%
Symptoms					
Rumination about harboring an illness	15.1	0.97	0.07	0.01	48.0
Preoccupation with body or illness	19.9	0.96	0.91	0.10	100.0
Suggestivity or autosuggestivity	14.4	0.57	0.21	0.00	48.0
Preoccupation with medical information	22.0	0.38	0.43	0.07	31.0
Fear of infection or contamination	7.7	0.37	0.09	0.00	24.0
Fear of medication	14.6	0.31	0.24	0.05	36.0
	%	%	%	%	%
Observed class prevalence (N=701)[c]		14.6	34.8	50.6	8.4
Expected prevalence from latent class model[c]		13.1	32.5	54.3	
Number of symptoms present					
None	44.1	0.0	0.0	87.0	0.0
1	20.0	0.0	38.5	13.0	22.0
2	17.0	16.7	41.8	0.0	20.3
3	11.8	38.2	18.0	0.0	25.4
4–6	7.2	45.1	1.6	0.0	32.2

[a] Without exclusion of other somatoform or psychiatric disorders but including a duration of 6 months or more (DSM-IV criterion E).
[b] Conditional probability that a patient having the symptom belongs to the class.
[c] Unweighted.

The predefined inclusion and exclusion criteria were met by 2,197 patients, of whom 274 declined the invitation to participate and 138 did not participate for other reasons (forgot their glasses, the secretary was too busy, etc.), leaving 1,785 patients for analysis. The mean age of those declining participation was 42.2 years (SD=13.1) compared with 38.8 (SD=12.9) among the included participants (t=–4.0, df=2046, p<0.001). There was no significant gender difference between the two groups. After complete description of the study to the subjects, written informed consent was obtained.

Study Design and Procedures

A two-phase design was used. First, a screening questionnaire was distributed to all patients in the waiting room. This questionnaire included, among other things, the eight-item version of the Symptom Check List (SCL) (20, 21) assessing anxiety and depression; the seven-item Whiteley index (22), which measures worrying and convictions about illness and somatoform disorders; the somatization subscale of the SCL, which checks for 12 common physical symptoms (23); and the CAGE, which consists of four questions screening for alcohol abuse (24).

After the consultation, the primary care physician filled in a questionnaire on the assessment results and his or her judgment of the patient.

Selection of Patients for Psychiatric Diagnostic Interview

The responses to each item were dichotomized. Patients with a total score of 2 or more on the eight-item SCL, the seven-item Whiteley index, or the CAGE or a score of 4 or more on the SCL somatization subscale were selected for the second phase—a psychiatric diagnostic interview. Furthermore, a random sample of one-ninth of the remaining patients was selected for interviews to produce a stratified subsample consisting of all patients with high scores and one-ninth of those with low scores. The cutoff values were chosen on the basis of a sample size calculation and the performance of the screening scales in a previous study in primary care (20, 25). The aim was to include 400 patients with somatoform disorders in the study while performing no more than 800 psychiatric interviews, which was the maximum capacity of the psychiatric interviewers. We selected 894 patients for interview, but 193 (21.6%) declined the invitation, leaving 701 patients for interview. A comparison of those declining the interview with those who completed the interview (Table 1) shows that the former group contained a higher proportion of men, was younger, and had more missing answers to the questions about sociodemographic variables. Fewer patients in the group declining interview were employed or living with a partner, and fewer had an education beyond basic schooling. They had lower scores on the SCL somatization subscale than did the patients who completed interviews (2003 unpublished study by T. Toft et al.).

Psychiatric Research Interview

The psychiatric interview was conducted as soon as possible after the index contact, in the primary care physician's office, in the research unit's office, or in the patient's own home. The patients' transportation expenses were reimbursed. For patients who could not be interviewed immediately, the interview did not include the period *after* the index contact.

The psychiatric interviews were made by means of the Schedules for Clinical Assessment in Neuropsychiatry (SCAN), version 2.1 (26, 27), which is a standardized comprehensive interview endorsed by the World Health Organization (WHO) that covers all types of mental disorders. It is a semistructured interview, and its use requires an interviewer who has received psychiatric training. The interview lasts between 30 minutes, in cases of no health problems, and several hours, in cases of more massive psychopathology (the maximum in this study was 7 hours). In this study the interviewers were free to explore aspects they did not find fully clarified by the interview, e.g., by reviewing medical records. However, they were not allowed to contact the primary care physicians.

In the literature a range of cognitive and emotional symptoms, beside those specified in DSM-IV (and ICD-10), are commonly reported in patients with hypochondriasis (19, 28–30):

1. Worrying about or preoccupation with fears of harboring a severe physical disease (DSM-IV and ICD-10 criterion A).
2. Bodily preoccupation, i.e., an absorption with the body and its functioning, e.g., body excretion, physiological function, sensations, appearance, and body performance.
3. Obsessive rumination with intrusive thoughts and ideas and fears of harboring an illness that cannot be stopped or can be stopped only with great difficulty.
4. Suggestibility or autosuggestibility: responding with alarm to the slightest hint of illness; alarm may arise from reading about a disease, knowing someone who becomes sick, etc.
5. Extensive fascination with medical information: reading medical books or journals, reading about medical subjects in an encyclopedia, watching television programs about health or medicine, being interested in news about health, etc.
6. Unrealistic fear of being infected or contaminated by something touched or eaten, by a person met, etc.
7. Pathological, excessive health-preserving behavior, i.e., eating special food, extensive exercise, or overdoing other things to keep fit.
8. Focusing primarily on causality: the patient wants an explanation for the symptoms and is more concerned about the meaning of the symptoms than the distress they cause.
9. Fear of taking prescribed medication.

These and other candidate symptoms included in the diagnostic research criteria for different somatoform diagnoses were added to the SCAN interview section on physical health. The new questions were integrated in such a way that they appeared to be a natural part of the interview, and the definition of each new symptom was added to the glossary, which is a part of the SCAN interview. Each of the symptoms is rated 0 ("absent"), 1 ("mild to modest preoccupation but no significant distress or impairment"), or 2 ("excessive preoccupation involving severe daily troubles or numerous consultations or self-medication"). In the analysis the symptom ratings 1 and 2 were collapsed. "The duration of hypochondriacal preoccupation" (months) caused by any of the rated symptoms and "interference with everyday activity because of these problems" are additionally rated as separate items in the SCAN. The modified version with definitions of all the symptoms included in this article is available from the authors on request.

The SCAN interviews were conducted by six psychiatric physicians (including T.T.) who were all highly skilled in psychopathological assessment, intensively trained in using the SCAN interview, and certified at the WHO center in Copenhagen. They had all received psychiatric residency training and had at least 2 years of medical and surgical residency experience. During the study they met at least once a week to discuss cases and how to interpret and rate dubious responses and symptoms that seemed to fall between the numeric ratings, and they were free to consult more senior doctors.

Patients scheduled for Thursdays at 1:00 p.m. were asked to give permission for the interview to be videotaped. The videotaped interviews of the eight patients who accepted were rated by all the interviewers, and these ratings showed a high interrater agreement on the presence or absence of any psychiatric diagnosis (kappa=0.86) as well as on the somatoform diagnostic category (kappa=0.82) (unpublished 2003 article by Toft et al.). The interviewers were blinded to the patients' responses on the screening questionnaires.

Data Analysis

The SCAN interviews were used for computerized DSM-IV psychiatric diagnoses with reference to the "present state" (current mental disorder) and "lifetime before" (the patient's psychiatric history). In this article we present only the data on current mental disorder. The section on physical health containing information on the somatoform disorders and related diagnoses was processed separately in order to make the diagnoses fit the DSM-IV criteria and to analyze the added symptoms.

TABLE 3. Suggested Diagnostic Criteria for Hypochondriasis

Criterion	Description
A	Obsessive rumination with intrusive thoughts, ideas, or fears of harboring an illness that cannot be stopped or can be stopped only with great difficulty
B	One (or more) of the following five symptoms
1	Presence of a, b, or both
a	Worrying about or preoccupation with fears of harboring a severe physical disease or the idea that disease will be contracted in the future or preoccupation with other health concerns
b	Attention to and intense awareness of bodily functions, physical sensations, physiological reactions, or minor bodily problems that are misinterpreted as serious disease
2	Suggestibility or autosuggestibility; if the patient hears or reads about an illness, he or she is inclined to fear that he or she has the same disease
3	Excessive fascination with medical information
4	Unrealistic fear of being infected or contaminated by something touched or eaten or by a person met
5	Fear of taking prescribed medication
C	If a medical condition is present, the patient's reaction clearly exceeds what would be expected from the medical condition alone
D	The symptoms are not better explained by another psychiatric disorder
E	The symptoms are present for most of the time for at least 2 weeks
F	Specify whether the disorder is severe or mild—severe: at least one of the symptoms in criteria A and B is severely disturbing or significantly interferes with everyday activities; mild: all others

We processed the data in STATA (31) and SPSS (32), and for the latent class analysis we used WINMIRA (33) and M*plus* (34).

Group comparisons were made by chi-square tests for categorical data, and the Mann-Whitney U test or Kruskal-Wallis test was used for nonnormal continuous data. We estimated prevalence by weighted logistic regression with the observed fraction of second-phase patients as sample weights (35). To assess the equality of prevalences in subgroups, likelihood ratio tests were performed.

The model selection in latent class analysis was based on different information criteria, with the sample-size-adjusted Baye's information criterion (SS BIC) as the primary criterion (34).

In order to assess the goodness of fit of the final selected model, the Pearson chi-square fit statistic and an empirical Pearson chi-square fit statistic were computed.

Two-sided p values are reported.

Ethics

All biomedical studies in Denmark must be approved by the local Science Ethics Committee, which is subject to the laws and regulations laid down by the Danish government. We obtained approval from the Science Ethics Committee of Aarhus County.

All the participating patients received written and oral information and gave written informed consent.

Results

Diagnostic Classes

The nine candidate symptoms for a new hypochondriasis diagnosis were tested in a latent class statistical model with the number of classes varying between two and six.

TABLE 4. Characteristics Associated With Diagnostic Classes Determined by Latent Class Analysis of Hypochondriasis-Related Symptoms in 701 Primary Care Patients

Variable	Class 1: Hypochondriasis				Class 2: Other Somatoform Disorders				Class 3: Nonsomatoform Disorders			
	Mild (N=21)		Severe (N=81)		Mild (N=102)		Severe (N=142)		Mild (N=292)		Severe (N=63)	
	N	%	N	%	N	%	N	%	N	%	N	%
Duration												
≤1 month	10	47.6[b]	22	27.2[b]	75	73.5	78	54.9[b]	282	96.6[b]	60	95.2[b]
2–5 months	2	9.5[b]	7	8.6[b]	5	4.9	3	2.1	0	0.0[b]	0	0.0
6–23 months	1	4.8	8	9.9[b]	6	5.9	9	6.3	3	1.0[b]	0	0.0
≥2 years	8	38.1[b]	44	54.3[b]	16	15.7	52	36.6[b]	7	2.4[b]	3	4.8[b]
Excessive health-preserving behavior	14	66.7[b]	51	63.0[b]	60	58.8[b]	85	59.9[b]	51	17.5[b]	17	27.0[b]
Focusing on causality	12	57.1	57	70.4[b]	62	60.8[b]	90	63.4[b]	68	23.3[b]	26	41.3
Persistence of preoccupation despite medical reassurance	8	38.1[b]	41	50.6[b]	23	22.5	50	35.2[b]	6	2.1[b]	3	4.8[b]
Psychosocial communication style	2	9.5	34	42.0[b]	8	7.8[b]	41	28.9[b]	7	2.4[b]	12	19.0
La belle indifférence	0	0.0	28	34.6[b]	4	3.9[b]	41	28.9[b]	7	2.4[b]	11	17.5[b]
Discrepancy between subjective complaints and observed behavior	0	0.0[b]	28	34.6[b]	9	8.8[b]	52	36.6[b]	5	1.7[b]	15	23.8
Conflicts with doctors												
Yes	0	0.0	15	18.5[b]	1	1.0[b]	21	14.8[b]	3	1.0[b]	5	7.9
Unsure	6	28.6[b]	12	14.8[b]	8	7.8	15	10.6	13	4.5[b]	5	7.9
Doctor shopping	0	0.0	8	9.9[b]	1	1.0	12	8.5[b]	3	1.0[b]	3	4.8[b]
	Median	Q1–Q3[c]	Median	Q1–Q3[c]	Median	Q1–Q3[c]	Median	Q1–Q3[c]	Median	Q1–Q3[c]	Median	Q1–Q3[c]
Number of medically unexplained symptoms[d]	5	2–9	9	5–18	4	2–7	11	6–17	0	0–2	5	2–9
Number of organ systems associated with complaints[e]	3	2–4	5	3–7	2	1–3	5	3–6	0	0–2	3	2–4
Age at onset (years)[f]	32	22–35	25	17–35	29	20–40	27	18–37	29	20–41	32	20–43

[a] Without the DSM-IV exclusion for other somatoform or psychiatric disorders. The DSM-IV duration criterion of 6 months was not used for the "Duration" portion of the table (N=88) but was included for the other variables (N=59).
[b] Significant at 5% level (adjusted standard residual >1.96 or <–1.96 by chi-square test).
[c] Q1 and Q3 and the 25 and 75 quartiles, respectively.
[d] Out of 86 symptoms explored in the Schedules for Clinical Assessment in Neuropsychiatry interview, including for each organ system a residual group of "other medically unexplained symptoms."
[e] Seven organ systems (general, CNS, cardiopulmonary, gastrointestinal, extremity [motor apparatus], skin and glands, other).
[f] Because some patients had no somatoform symptoms or did not supply data, values were not included for all patients. The numbers of missing values are as follows: mild class 1, N=3; severe class 1, N=2; mild class 2, N=15; severe class 2, N=2; mild class 3, N=179; severe class 3, N=10; DSM-IV hypochondriasis, N=0.
[g] The symptom is included in the diagnostic criteria for DSM-IV hypochondriasis (therefore, all patients had this symptom).

The model with the smallest SS BIC was chosen as optimal, but it did not have a meaningful interpretation. We assessed SS BIC as we excluded one symptom at a time. We excluded "pathological, excessive health-preserving behavior" and "focusing primarily on causality," and an interpretable three-class model emerged. However, the high intercorrelation (r_s=0.37, N=701, p<0.001) and marked prevalence in either somatoform disorders class of the symptoms "worrying/preoccupation" and "bodily preoccupation" made it expedient to collapse the two symptoms into one item. The final model, displayed in Table 2, thus includes six symptom items. This model's goodness of fit is acceptable (Pearson χ^2=50.9, df=50, p=0.19, empirical p=0.20). It is seen that 14.6% of the interviewed patients fell into latent class 1, in which positive responses to most items were prevalent. The symptom "rumination" was highly predictive for this class: only three of the patients reporting this symptom did not fall into this class. None of these three patients reported any of the other six symptoms. This group should therefore be classified as patients with hypochondriasis. Class 2 comprised 34.8% of the patients, and most of the investigated symptoms were prevalent in this class except for "rumination" and "fear of being infected." Class 3 included 50.6% of the patients, and few reported any of the explored symptoms. The prevalences of the symptoms in the hypochondriasis class (class 1) were similar to or higher than those among the patients fulfilling the diagnostic criteria for DSM-IV hypochondriasis; the most marked difference was for "rumination" (Table 2).

The key symptom (criterion A) of DSM-IV hypochondriasis, "preoccupation with fears of having…a serious disease," is thus frequent in both class 1 and class 2, and the poor specificity of the symptom is furthermore highlighted by the fact that 84.6% of the 26 patients with a somatization disorder diagnosis and 58.4% of the 77 patients with pain disorder also had the symptom.

All patients in the hypochondriasis class (class 1) reported two or more symptoms, and 45.1% reported four or

Analysis			DSM-IV Hypochondriasis (N=88/59)[a]	
χ^2	df	p	N	%
273.2	15	<0.001		
			21	23.9
			8	9.1
			11	12.5
			48	54.5
129.0	5	<0.001	34	57.6
108.0	5	<0.001	41	69.5
147.3	5	<0.001	—[g]	—[g]
110.4	5	<0.001	26	44.1
105.8	5	<0.001	26	44.1
123.5	5	<0.001	30	50.8
82.8	10	<0.001		
			16	27.1
			14	23.7
25.6	5	<0.001	11	18.6
Kruskal-Wallis χ^2	df	p	Median	Q1–Q3[c]
315.9	5	<0.001	14	7–21
309.4	5	<0.001	6	4–7
54.6	5	0.06	20	16–29

more symptoms. The patients in the two other classes and those with DSM-IV hypochondriasis reported fewer symptoms. Only 13.0% of the patients in class 3 (patients with nonsomatoform illness) reported any of the six symptoms (Table 2).

Clinical Diagnosis of Hypochondriasis

We manually reviewed the patterns of the latent class results to turn the statistical information derived from analysis of the hypochondriasis class into operational diagnostic criteria for clinical use and to establish a new set of diagnostic criteria for hypochondriasis (Table 3). The only difference between the hypochondriasis class (class 1) and the diagnostic criteria displayed in Table 3 is item E, i.e., the symptoms should be present for at least 2 weeks. This criterion was added from a clinical point of view to avoid diagnosing the most transient cases. We also introduced a severity item, item F, making it possible to distinguish between mild and severe cases. Patients with severe and mild cases did not display significantly different numbers of symptoms. In the remaining part of this article, we will report data on the latent classes subdivided into severe cases, i.e., in which the patient states that at least one of the symptoms listed in Table 2 is severe or distressing or states that any of the somatoform symptoms severely interferes with everyday activities, and into mild cases, i.e., not causing significant impairment or distress.

Associated Symptoms and Characteristics

For 54.3% of the patients with severe hypochondriasis (class 1), the illness lasted for 2 years or more. It lasted 5 years or more for 42.0% and 10 years or more for 24.7%. Its duration was a month or less for about 27.2%, which was about the same duration as for patients fulfilling the DSM-IV criteria (Table 4). The illness lasted a remarkable 2 years or more in 38.1% of the patients with mild class 1 hypochondriasis.

The patients with severe class 1 hypochondriasis had a median age at onset of 25 years; the earliest onset was at age 5, and the latest was at 60. For comparison, the patients with DSM-IV hypochondriasis had a median age at onset of 20 years, with a minimum age of 6 and a maximum of 52. The other groups had higher onset ages, but in some of the groups the number of patients was small, and in the class 3 group more than one-half of the patients were not counted because the patients had no somatoform symptoms, making it impossible to register onset age.

Table 4 also lists a range of symptoms commonly reported in different somatoform disorders.

"The preoccupation persists despite appropriate medical evaluation and reassurance" is criterion B of the DSM-IV criteria for hypochondriasis and was found in about one-half of the patients with severe class 1 hypochondriasis. However, the symptom was also frequent in those with mild class 1 hypochondriasis and in patients in the class 2 group (other somatoform disorders). In addition, the symptom was frequent among the 26 patients with somatization disorder (73.1%) and the 77 patients with pain disorder (24.7%) (data not shown in Table 4). Of the patients with severe class 1 hypochondriasis, 42.0% were found to use a "psychosocial communication style," i.e., the patients focused on the psychosocial consequences of their illness and the restrictions to their lives rather than on the symptoms' implications for their health. This symptom was, however, also frequent in the other groups with severe forms of illness. Except for doctor shopping, about one-third or more of the patients in the severe class 1 and class 2 groups and in the DSM-IV hypochondriasis group had each of the listed symptoms, showing that the symptoms are prevalent in somatoform disorders but none seems to be distinctively associated with one category in particular.

Except for the mild class 2 and class 3 categories, the average patient in each class presented multiple medically

TABLE 5. Comorbidity With DSM-IV Disorders of Diagnostic Classes Determined by Latent Class Analysis of Hypochondriasis-Related Symptoms in 701 Primary Care Patients

	Class 1: Hypochondriasis				Class 2: Other Somatoform Disorders				Class 3: Nonsomatoform Disorders				Analysis		DSM-IV Hypochondriasis (N=59)[a]	
	Mild (N=21)		Severe (N=81)		Mild (N=102)		Severe (N=142)		Mild (N=292)		Severe (N=63)		χ^2			
DSM-IV Diagnosis	N	%	N	%	N	%	N	%	N	%	N	%	(df=5)	p	N	%
Somatoform disorders[b]																
Somatization disorder (N=26)	1	4.8	10	12.3[c]	2	2.0	12	8.5[c]	0	0.0[c]	1	1.6	38.8	<0.001	12	20.3
Undifferentiated somatoform disorder (N=138)	6	28.6	21	25.9[c]	20	19.6	48	33.8[c]	14	4.8[c]	29	46.0[c]	89.6	<0.001	20	33.9
Pain disorder (N=106)[d]	3	14.3	28	34.6[c]	9	8.8[c]	51	35.4[c]	4	1.4[c]	11	17.5	118.2	<0.001	22	37.3
Hypochondriasis (N=59)[a]	3	14.3	25	30.9[c]	7	6.9[c]	24	16.9[c]	0	0.0[c]	0	0.0[a]	100.1	<0.001		
Nonsomatoform disorders																
Major depressive disorder (N=72)	1	4.8	14	17.3[c]	9	8.8	21	14.8[c]	19	6.5[c]	8	12.7	13.3	0.03	9	15.3
Anxiety disorder, any (N=107)	1	4.8	29	35.8[c]	8	7.8[c]	36	25.4[c]	23	7.9[c]	10	15.9	56.1	<0.001	25	42.4
Substance use disorder (N=30)	0	0.0	7	8.6[c]	1	1.0	9	6.3	8	2.7	5	7.9	12.6	0.03	4	6.8
All nonsomatoform DSM-IV disorders except simple phobia, nicotine-related disorders, and sleep disorders (N=178)	3	14.3	40	49.4[c]	18	17.6	50	35.2[c]	47	16.1[c]	20	31.7	51.1	<0.001	29	49.2

[a] Without the DSM-IV exclusion for other somatoform or psychiatric disorders.
[b] Without exclusion for other psychiatric disorders.
[c] Significant at 5% level (adjusted standard residual >1.96 or <–1.96 by chi-square test).
[d] Without exclusion of DSM-IV hypochondriasis.

unexplained symptoms from many organ systems, and the DSM-IV hypochondriasis group had the most (Table 4).

Comorbidity

A modest overlap or comorbidity between severe class 1 hypochondriasis and the other somatoform disorders was reflected in the fact that about one-tenth to one-third of the patients with severe class 1 hypochondriasis also fulfilled the criteria for one of the diagnoses listed in Table 5. For all somatoform disorder diagnoses, the overlap was higher among the patients with DSM-IV hypochondriasis, and for undifferentiated somatoform disorder it was considerably higher. Only one patient fulfilled the diagnostic criteria for conversion disorder, which were therefore excluded from Table 5. The category of somatoform disorder not otherwise specified is an exclusion diagnosis and was therefore also left out. Only 30.9% of the patients with severe class 1 hypochondriasis and 16.9% of those with severe class 2 disorders fulfilled the diagnostic criteria for DSM-IV hypochondriasis (Table 5). On the other hand, 83.1% of the DSM-IV hypochondriasis patients had severe class 1 or class 2 disorders.

Patients with severe class 1 hypochondriasis and those with DSM-IV hypochondriasis had similar rates of comorbidity with nonsomatoform psychiatric disorders: 49% in each group (Table 5). The interviewers judged depression to be predominant in three of the 14 patients meeting the diagnostic criteria for both major depression and severe class 1 hypochondriasis and in three of the nine patients with DSM-IV hypochondriasis and depression.

Only three patients fulfilled the criteria for obsessive-compulsive disorder (OCD), and none of these fell into class 1 or class 2, whereas one of them had DSM-IV hypochondriasis.

Primary Care Physician Assessments

The primary care physicians, not knowing the patients' diagnoses, judged that 40.0% of the patients with severe class 1 hypochondriasis had a low care-seeking threshold (Table 6). They also found that 62.0% were more preoccupied with bodily sensations or illness than would be expected and that 71.3% were overly fearing illness or worrying about illness, of whom 45.0% could not be reassured or could only with difficulty be reassured that their worrying was biomedically unfounded. In 75.9% of the patients with severe class 1 hypochondriasis, the primary care physicians reported that the patient frequently consulted them because of medically unexplained functional symptoms. The rates at which the primary care physicians attributed these characteristics to patients were highest for patients with severe class 1 hypochondriasis in all instances, but even patients with mild class 1 hypochondriasis had comparatively high rates. High rates were also recorded for some of the other groups but not for patients with mild cases of class 3 disorders. The rates for the patients with severe class 1 hypochondriasis were similar to those for the DSM-IV hypochondriasis patients. Since "the preoccupation persists despite appropriate medical evaluation and reassurance" is included in the DSM-IV diagnostic criteria but not among the class 1 symptoms, it is remarkable that the primary care physicians found it difficult to reassure the same fraction of class 1 and DSM-IV hypochondriasis patients.

TABLE 6. Primary Care Physicians' Assessments of Patients in Diagnostic Classes Determined by Latent Class Analysis of Hypochondriasis-Related Symptoms in 701 Primary Care Patients

Patient Characteristic Assessed by Primary Care Physician	Class 1: Hypochondriasis				Class 2: Other Somatoform Disorders				Class 3: Nonsomatoform Disorders				Analysis			DSM-IV Hypochondriasis[a]	
	Mild		Severe		Mild		Severe		Mild		Severe						
	N	%	N	%	N	%	N	%	N	%	N	%	χ^2	df	p	N	%
Low threshold for consultation[b]	6	28.6[c]	32	40.0[c]	22	21.6	44	31.0[c]	42	14.6[c]	15	24.2	29.9	5	<0.001	26	44.1
Excessive bodily preoccupation[b]	9	42.6	49	62.0[c]	41	40.6	74	52.1[c]	82	28.5[c]	27	43.5	40.6	5	<0.001	36	61.0
Excessive fear of or worrying about illness[b]													50.2	10	<0.001		
Yes, but easily reassured	7	33.3	21	26.3	28	27.7	37	26.1	70	24.2	15	24.2				11	18.6
Yes, difficult or impossible to reassure	8	38.1	36	45.0[c]	20	19.8	50	35.2[c]	46	15.9[c]	18	29.0				27	45.8
Frequent consultations with functional physical symptoms[d]	13	61.9	60	75.9[c]	52	52.5	102	73.9[c]	102	37.5[c]	40	69.0[c]	72.9	5	<0.001	45	77.6

[a] Without DSM-IV exclusion for other somatoform or psychiatric disorders.
[b] Missing assessment of primary care physician for 6–8 patients.
[c] Significant at 5% level (i.e., adjusted standard residual >1.96 or <–1.96 by chi-square test).
[d] Missing assessment of primary care physician for 34 patients.

Prevalence

Table 7 displays the weighted prevalences corrected for the biases introduced by the stratified patient sampling. The prevalence of severe class 1 hypochondriasis was 9.5%, compared with 5.8% for DSM-IV hypochondriasis without exclusion of comorbid diagnoses and 4.7% (95% CI=2.9%–7.6%) with the full DSM-IV criteria.

Severe other somatoform disorders (class 2) were significantly more prevalent in the patients who were 40–49 years old than in the other age groups, and the patients with severe nonsomatoform disorders (class 3) were significantly older than patients in the other classes. Inversely, patients with mild class 1 hypochondriasis were significantly younger than the other patients. There were no statistically significant differences as to age or gender among the other classes or in DSM-IV hypochondriasis.

Discussion

Robins and Guze (36) and later Kendell (37) listed a range of strategies for establishing the validity of clinical syndromes; the first is to identify and describe the syndrome by "clinical intuition" or by cluster analysis, and the second is to demonstrate boundaries or "point of rarity" between related syndromes by statistical methods. The current DSM-IV hypochondriasis diagnosis rests mainly on "clinical intuition" and tradition, and it is not supported by substantial empirical evidence. To our knowledge, no studies have established a "point of rarity" between the DSM-IV hypochondriasis diagnosis and other somatoform disorder diagnoses, and we did not find support for this in the current study either, as the key symptoms included in the DSM-IV diagnostic criteria (criteria A and B) were also common among patients with other somatoform disorder diagnoses. In this study we included a range of symptoms reported to be common or typical for hypochondriasis (19, 28, 29). We used latent class analysis, a robust statistical method that from a statistical point of view produces a rather clearcut result, establishing a "point of rarity" between classes. Six of the nine symptoms explored fitted satisfactorily into a latent class model with three classes: a hypochondriasis class (class 1), a class of "other somatoform disorders" (class 2), and a class of nonsomatizing patients (class 3). However, even class 3 included some patients with medically unexplained symptoms, and further studies are needed to establish whether this class is truly a nonsomatizing group or a somatoform disorder subgroup without cognitive or emotional symptoms. The suggested diagnostic criteria have to be viewed as preliminary, as the criteria, among other validation procedures, have to be cross-validated in another group of subjects.

From a methodological point of view, the major strength of this study lies in its empirical foundation, in particular its inclusion of a large number of patients gathered in a nonspecialized medical setting. Further strengths include almost complete absence of selection bias, owing to the fact that Danish health care is free of charge, and use of a standardized psychiatric interview, the SCAN, which is state of the art and probably the most advanced and comprehensive diagnostic tool for psychopathology and psychiatric diagnostics available today (26, 27). The interview section on physical issues comprises questions about all types of somatoform symptoms and hence allows simultaneous study of all known types of somatoform disorders. The interviewers were all highly skilled in psychopathology assessment and intensively trained in using the SCAN interview. The candidate symptoms we added to the SCAN interview were feasible and reliable according to the interviewers.

We expected "bodily preoccupation" to be a distinct hypochondriasis symptom as bodily symptom amplification

TABLE 7. Weighted Prevalences[a] of Diagnostic Classes Determined by Latent Class Analysis of Hypochondriasis-Related Symptoms in Primary Care Patients, by Gender and Age[b]

			Class 1: Hypochondriasis				Class 2: Other Somatoform Disorders			
	Number of Patients		Mild		Severe		Mild		Severe	
Group	Interviewed	Screened	%	95% CI	%	95% CI	%	95% CI	%	95% CI
Overall	701	1,785	2.6	1.3–5.3	9.5	6.6–13.3	16.1	11.9–21.4	12.2	9.5–15.5
Gender										
Male	235	636	3.3	1.0–10.5	9.3	4.7–17.4	17.8	10.7–28.0	9.1	5.4–14.8
Female	466	1,149	2.2	0.9–5.3	9.6	6.3–14.2	15.2	10.4–21.7	13.9	10.4–18.3
Age (years)										
18–29	180	505	5.9[c]	1.9–16.3	7.8	4.1–14.1	17.3	9.7–29.0	7.3[d]	3.8–13.7
30–39	161	470	1.6[c]	0.7–3.6	11.0	5.2–21.9	15.8	8.2–28.2	11.2[d]	7.6–16.1
40–49	173	406	0.6[c]	0.1–2.5	7.5	3.5–15.4	18.6	10.3–31.3	20.1[d]	12.3–31.2
50–65	187	404	1.9[c]	0.8–4.2	11.7	6.1–21.5	12.5	6.7–22.1	11.3[d]	7.7–16.3

[a] Corrected for the biases introduced by stratified patient sampling.
[b] Without DSM-IV exclusion of other somatoform or psychiatric disorders.
[c] Significant difference among groups (χ^2=11.0, df=3, p=0.02; the p value is the result of a likelihood ratio test of the equality of the prevalences).
[d] Significant difference among groups (χ^2=13.3, df=3, p=0.004; the p value is the result of a likelihood ratio test of the equality of the prevalences).
[e] Significant difference among groups (χ^2=8.1, df=3, p=0.05; the p value is the result of a likelihood ratio test of the equality of the prevalences).

has been suggested to be a basic mechanism in hypochondriasis (38, 39), but like "preoccupation with…disease," it was just as frequent among class 2 patients as among patients with hypochondriasis, and furthermore, the symptoms were frequent among other DSM-IV somatoform disorders. With the addition of "extensive fascination with medical information," the three symptoms may collectively be viewed as common symptoms of somatoform disorders in general. According to our findings, the missing discriminatory power of these symptoms may therefore be the explanation for the overlap between DSM-IV hypochondriasis and other somatoform disorder diagnoses in the current classification system, as the symptom "preoccupation with…disease" is the key symptom (criterion A) in the DSM-IV diagnostic criteria for hypochondriasis.

"Obsessive rumination" proved to have a strong power to discriminate between the patients with hypochondriasis and other patients, and "fear of being infected or contaminated" also appeared to be quite distinctive. These two symptoms outperformed the other symptoms in establishing class 1 hypochondriasis, which raises the question of whether hypochondriasis should be viewed as an OCD spectrum disorder, i.e., "OCD bodily type," or alternatively, a specific illness phobia. This question cannot be addressed on the basis of this study. None of the three patients with OCD found in this study had class 1 hypochondriasis, and other symptoms atypical for OCD were also common among the patients with class 1 hypochondriasis. It has been suggested that the hypochondriasis diagnosis be moved from the somatoform disorder category to the anxiety disorder category as a "health anxiety disorder," thus replacing the stigmatizing hypochondriasis label. Despite possible support for this view, it calls for caution regarding premature conclusions; we should keep in mind the discussion in the 1960s and 1970s about whether hypochondriasis should be viewed as a depressive disorder because hypochondriacal worrying is also common in depressive disorders (40). Instead, a more neutral replacement may be considered, for example the term "valetudin disorder," which originates from the Greek word *valetudo,* meaning "the state of health" (41).

The criteria for class 1 hypochondriasis enjoyed high statistical, clinical, and "face" validity, even compared with the assessments of primary care physicians, but confirmation from daily clinical practice is, of course, needed. It is remarkable that the primary care physicians' assessments of the patients with severe class 1 hypochondriasis were similar to those for the patients with DSM-IV hypochondriasis despite the fact that class 1 hypochondriasis was about twice as prevalent as DSM-IV hypochondriasis. This may indicate that the class 1 criteria do not pick up more clinically insignificant cases than the DSM-IV diagnosis. Furthermore, it indicates that the DSM-IV hypochondriasis cases not included in the class 1 category in fact may be other somatoform disorders and that they ought to be classified in other subcategories. The class 1 hypochondriasis diagnosis also enjoys the advantage of not being an exclusion diagnosis, i.e., not requiring exclusion of a medical explanation of physical symptoms. It relies solely on positive diagnostic criteria in the form of cognitive and emotional symptoms, hence matching the diagnostic principles of other psychiatric diagnoses. The present study does not allow us to establish whether the full syndrome may emerge as a reaction to a newly diagnosed severe physical disease, as it was conducted in a primary care setting, where the number of such cases was low. We do, however, expect such psychological reactions to be only transient. In patients with the full syndrome, the psychological reaction would probably be clinically significant and encourage intervention, even if the patient also had a severe physical disease.

The class 1 hypochondriasis diagnosis had some comorbidity with other somatoform disorders, but its overlap was smaller than that for DSM-IV hypochondriasis

Class 3: Nonsomatoform Disorders				DSM-IV Hypochondriasis[b]	
Mild		Severe			
%	95% CI	%	95% CI	%	95% CI
54.0	47.7–60.0	5.7	3.8–8.3	5.8	3.8–8.7
55.2	44.5–65.5	5.3	2.4–11.3	5.6	2.2–13.5
53.2	45.6–60.7	5.9	3.8–9.1	5.9	3.9–8.8
58.5	46.5–69.6	3.3[e]	1.8–5.8	5.1	2.1–11.9
54.9	42.1–67.0	5.6[e]	2.1–14.2	8.2	4.1–16.0
48.8	36.4–61.4	4.3[e]	2.4–7.5	3.7	2.0–6.7
52.4	40.5–64.1	10.2[e]	5.3–18.8	6.0	2.4–14.4

and smaller than that reported in other studies (17, 42, 43). The magnitude of the overlap between class 1 hypochondriasis and other somatoform disorders was comparable to the overlap between depressive disorder and anxiety disorder (data not reported). The overlap may not necessarily be due to inappropriate diagnostics or overlap of diagnostic criteria; it might also be due to the patients' suffering from two distinctly independent disorders.

The overlap of DSM-IV hypochondriasis and severe class 1 hypochondriasis was only modest; about one-half of the DSM-IV hypochondriasis cases fell into class 1. Another half fell into class 2, other somatoform disorders. This seems to highlight the poor discriminatory power of the current DSM-IV hypochondriasis diagnosis, as these patients could be split into two distinct groups by latent class analyses of six symptoms. The use of an exploratory approach requires imposition of a minimum of analytical restrictions, and the DSM-IV hierarchical exclusion rules were therefore not used for patients meeting the criteria for more than one diagnosis. Use of the full DSM-IV hypochondriasis criteria, however, made little change (data not reported).

The prevalence of severe class 1 hypochondriasis reached 9.5%, which testifies to its high prevalence among primary care patients. The prevalence of DSM-IV hypochondriasis based on the full criteria (i.e., including the exclusion criteria) was 4.7%, which is higher than in most other studies in primary care, in which prevalence rates have been 0.0%–6.3% (17, 44).

One of the strengths of the present study lies in its use of a more extensive diagnostic procedure (i.e., the SCAN interview) than was used in other studies and the use of experienced psychiatric interviewers. We included only "incident" cases, i.e., those of patients presenting with new health problems, thus excluding some patients with chronic physical diseases. In accordance with most other studies, we found no gender or age difference in the prevalence of hypochondriasis (17, 44, 45). About 10% of the primary care physicians in the county participated in the study. This may raise the possibility that a practice selection bias limits the generalizability of the prevalence figures. However, the participating primary care physicians seem to be remarkably representative of the overall population of primary care physicians in the county on most of the variables on which they were compared.

This study focused mainly on class 1 hypochondriasis, but class 2, nonhypochondriasis somatoform disorders, may be just as interesting. This calls for a more profound and focused exploration of the nonhypochondriasis diagnostic categories, which is outside the limits of this article, but we plan to conduct such separate analyses.

The hypochondriasis diagnosis as defined in ICD-10 and DSM-IV has shown to be so restrictive that few patients in primary care fulfill the diagnostic criteria (17). Gureje et al. (17) showed that one of the major problems is the symptom "the preoccupation persists despite appropriate medical evaluation and reassurance"(i.e., criterion B), and Barsky et al. (16) have pointed to the problem of the 6-month time limit (criterion E). The diagnostic criteria suggested in this study do not include these two symptoms and therefore overcome these shortcomings of the current DSM-IV hypochondriasis diagnosis. To replace the duration criterion of 6 months, however, from a purely clinical and not statistical point of view, we suggest the adoption of a severity criterion, i.e., that the symptoms must cause significant distress or impairment in order to be considered clinically significant. However, the patients with "mild" cases (i.e., those with no significant impairment or distress) may still be clinically relevant, especially in a primary care setting. The symptom durations were identical among patients with functional impairment or distress and patients without it, and the latter group also presented multiple medically unexplained symptoms. We may therefore speculate whether the "mild" cases are a type of latent or subclinical hypochondriasis with a high risk of evolving into the manifest clinical syndrome under stressful conditions. Further studies will elucidate the potential implications of the mild syndrome for health care utilization and health-related quality of life and may, if the results are affirmative, warrant inclusion of the mild diagnostic category in the diagnostic classification system as suggested here.

A third point in the validation process, according to Kendell (37), is to perform follow-up studies to establish a distinct course or outcome. This was not undertaken in the present study, but the included patients are being followed. However, as the symptoms had lasted for a long period in a high fraction of the patients with class 1 hypochondriasis, the present data indicate that the symptoms pursue a distinct course and that the diagnosis remains stable. Kendell (37) proposed three more strategies: 1) therapeutic trials to establish a distinct treatment response, 2) family studies establishing that the syndrome

"breeds true," and 3) demonstration of the association with some more fundamental abnormalities, i.e., anatomical, biochemical, or molecular. Such validation studies, as well as cross-validation studies in other subjects, have still to be planned.

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Measures of General Health Functioning and Behavior

- Millon Behavioral Health Inventory (MBHI) and its recent upgrade, the Millon Behavioral Medicine Diagnostic test (MBMD) (93) is one of the most frequently used health inventories for medical populations in the US. The MBHI provides information across four broad categories: basic coping styles, psychogenic attitudes, specific disease syndromes, and prognostic indices. It has good psychometric properties, a large normative database of representative medical patients, with specific disease scales developed for specific patient groups. The MBMD has updated and expanded the research base and clinical scales (165-items, 38 scales, 3 validity scales). The MBMD assists with: identification of significant psychiatric problems, making specific recommendations; pinpointing personal and social assets to facilitate adjustment; identifying medical regimen compliance problems; structuring post-treatment plans and self-care responsibilities in the patient's social network. Computerized scoring and an interpretive report facilitate use.
- The SF-36 Health Survey (SF-36)(94) surveys general health status in terms of physical and mental health and functional status. Widely used in research, and as an outcome measure (95), it assesses 8 areas, including limitations in physical functioning and social functioning and roles and activities, pain, mental health, vitality and health perceptions.
- The Neurobehavioral Functioning Inventory (NFI) (96). The NFI is a multipurpose inventory designed to measure current cognitive, physical, and emotional functioning in persons with traumatic brain injury or other neurobehavioral disorders. It is comprised of six independent scales reflecting problems frequently experienced by persons with neurobehavioral disorders: depression, somatic, memory/attention, communication, aggression and motor functioning. This well researched instrument includes separate forms for patient and family, demonstrates concurrent validity with neuropsychological test data and objective personality inventory profiles, can assist with treatment planning and allows measurement of change over time.
- The Sickness Impact Profile (SIP) (97) is a behaviorally based measure of health status designed to assess both psychosocial and physical dysfunction. It has sound psychometric properties, is used widely with chronic pain patients and can provide relevant information regarding degree of functional limitation in daily activity.
- The Illness Behavior Questionnaire (IBQ:(98)(99))provides useful information about attitudes, perceived reactions of others and psychosocial variables. It delineates 7 factors that include general hypochondriasis, disease conviction, psychological vs. somatic focusing, affective disturbance, affective inhibition, denial, and irritability. In addition, it has value in identifying patients who rely on illness behaviors as a coping style for need procurement.

General Psychological Measures: Mood, Anger and Anxiety

- The Beck Depression Inventory – 2 (BDI-2) (100) is a common self-report measure that assesses depressive symptomatology. It has been reported to differentiate chronic

pain patients with and without major depression utilizing an optimal cutoff score of 21 (101) and has well documented predictive validity.

- The Zung Self-rating Depression Scale (SDS)(102), seems especially well suited for medical settings, and has several advantages over other measures. It is shorter, simpler to administer and score, requires a lower reading level, fits well with medical and injury situations and can be easily administered in an interview format (62). Items are self ratings on a scale ranging from 1 to 4 ("Not at all" to "Most or all of the time") and are scored in the direction of increased depressive symptomatology, with a raw score cutoff for mild depression of approximately 40 points.
- The State-Trait Anger Expression Inventory-2 (STAXI-2)(103)and its recent update is a reliable, well-normed instrument for assessing the experience, expression and control of both current state and trait anger. Anger Expression and Anger Control scales assess four relatively independent anger-related traits: (a) expression of anger outward; (b) holding anger in; (c) controlling outward expression; and (d) controlling internal angry feelings. This instrument provides information regarding how experience, expression and control of anger may contribute to psychophysiologic arousal and symptoms and increase risk for developing somatic symptoms and medical problems. Indirectly, it offers suggestions for the direction of appropriate interventions. Importantly, anger is a frequent concomitant of chronic pain that has been unfortunately underappreciated (57).
- The Beck Anxiety Inventory (BAI)(104) is a screening measure of severity of patient anxiety. Specifically designed to reduce overlap with symptoms of depression, it assesses both physiological and cognitive components of anxiety in 21 items describing subjective, somatic, or panic-related symptoms. The BAI differentiates well between anxious and non-anxious groups in a variety of clinical settings.
- The Perceived Stress Scale (PSS)(105) is a widely used instrument for measuring the degree to which situations in one's life are appraised as stressful. Items measure how unpredictable, uncontrollable, and overloaded respondents find their lives and directly queries current levels of experienced stress. Higher PSS scores have been associated with greater vulnerability physical and psychological symptoms following stressful life events.

Comprehensive Personality Assessment Instruments

- The Minnesota Multiphasic Personality Inventory (MMPI:(106) MMPI-2 (107)) is the most widely used psychological assessment instrument in the US. The MMPI is a 567 item (true/false) objective measure of personality function and emotional status with 10 clinical and 3 (7 in revised version) validity scales that were derived through empirical discrimination). Its predictive abilities are based on more than fifty years of actuarial data collection and analysis. It is a very sensitive measure of psychological states, traits and styles (e.g., excessive anxiety, tension, depression, hostility and problematic anger, somatization tendencies, sociopathy, substance abuse, deviant thinking and experience, social withdrawal, etc.). Through configural interpretation of the relative scale elevations, tentative hypothesis regarding personality and coping style and relative degree of particular types of psychological disturbance can be gleaned. Importantly, although the MMPI can and is frequently misused and misinterpreted (e.g., application

of psychiatric norms to medical patients tends to beg psychiatric interpretations), it represents one of the most useful adjuncts to personality assessment and treatment planning. A cursory summary of potential utility of MMPI profile interpretation for assessing psychological reactions and contributions to physical conditions was offered by Fordyce (101), and roughly includes configural guidelines for interpreting: 1) Willingness to display physical symptom behaviors; 2) Distress/discomfort about illness ("How comfortably sick?"); 3) Poor general coping skills; 4) Depression complicating physical symptoms; 5) Tension (and sympathetic arousal) contributing to physical symptoms (High back, head, neck, shoulder, etc.); 7) Treatment outcome issues. A number of subtypes of chronic have been identified by other researchers although, as with other measures, it remains unclear to what extent results may inform the degree to which complaints are associated with organic (especially peripheral pathology) versus psychologic contributions to patient presentation (108)(109).

- The Personality Assessment Inventory (PAI) (110) a good measure of general psychopathology that can help with identification of a wide variety of risk factors that could adversely affect adjustment. It has good psychometric properties and contains 340 items, with 22 scales, including 4 validity scales. As with most other general psychological assessment measures, it has no norms for chronic pain and tends to overpathologize this group.
- Millon Clinical Multiaxial Inventory, 3rd edition (MCMI-III:) (111): includes scales assessing DSM-IV based psychiatric disorders, including affective, personality and psychotic disorders, somatization and others. It is useful for the differential diagnosis of personality disorders and psychological vulnerabilities for adaptation to pain. Like other psychiatric measures, it has limited pain group norms and may be prone to overpathologizing patients.

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