Psychiatric Diagnosis and Treatment of Somatizing Neuropsychiatric Disorders

November 28, 2014
By Daniel T. Williams, MD [1] and Alla Landa, PhD [2]

Although the somatizing disorders cover a vast array of symptomatic domains across many medical specialties, this article addresses the broad topic conceptually.

Somatic symptoms associated with psychosocial stressors have presented diagnostic and treatment problems for centuries. In more recent times, DSM-IV attempted to delineate this terrain by segregating it into several categories: somatoform disorders, factitious disorders, and psychological factors affecting a general medical condition. It is reasonable, for the purposes of this article, to consider all of these categories to be “somatizing neuropsychiatric disorders.” The distinguishing characteristics of these diagnostic categories have traditionally been the presence of at least some partially medically unexplained symptoms, as well as the level of awareness (intentionality) and emotional distress on the part of the patient in generating these symptoms.¹

More recent clinical studies have led to a significant reorientation of perspective in this area, leading to a revised series of diagnoses in DSM-5. The role of the psychiatrist often begins as a consultant, on referral from the medical physician who is first called on to evaluate the physical symptoms. For primary care physicians and other medical specialists, the diagnostic challenge is discerning which portion (if any) of the presenting physical symptoms are due to identifiable medical diagnoses and which are due to abnormal reactivity to psychological stressors.

When sufficient clinical evidence points to the significant role of psychological factors, the psychiatrist is often called on to assist with a differential diagnosis and treatment. Although the somatizing disorders (SDs) cover a vast array of symptomatic domains across many medical specialties, this article addresses the broad topic conceptually. The clinical applications of these concepts to the protean symptomatic variants encountered in general medical practices and various medical specialties will, we hope, become clear.

Phenomenology
Somatization connotes the tendency to experience somatic symptoms to convey emotions that are difficult to express directly, often because they are socially unacceptable, damaging to one’s self-esteem, or due to a poorly developed ability to express emotions in non-somatic ways. Somatization is widely used as an avoidance mechanism, sometimes harmlessly, as when someone tries to diminish the discomfort of declining a social invitation or unwanted work opportunity with a consciously or unconsciously generated report of “feeling unwell.” However, excessive use of this “escape route” from social, work, or other obligations can become pathologically overused, leading consciously or unconsciously to chronic symptom formation.

Epidemiology of somatizing disorders
SDs are highly prevalent; up to 30% of the general population experiences clinically significant symptoms at some point in their lives. In the general population, the prevalence of SDs is similar in boys and girls (2% to 4%). Predictors of persistence of SDs over time include female sex, depressive symptoms, poor self-rated health, history of trauma or abuse, and reported SDs in the parents.² The persistence of female preponderance, manifested as severe and chronic somatic symptom disorder (SSD, formerly somatization disorder) is reflected in a 1% incidence of SDs in women. There have been various reasons postulated for the female preponderance of SDs in population studies, including cultural-environmental and biological influences.³ It is noteworthy that higher rates of SDs are encountered in primary medical care settings (up to 16%) and in general neurology clinics (up to 30%).⁴

Developmental course of somatization
The frequently documented coexistence of anxiety, depression, and dissociation in patients with SD as well as the frequent finding of “alexithymia” among them has led to all of these features being considered risk factors for SDs.⁵,⁶ A plausible hypothesis is that internalizing vulnerabilities produces inadequate routes for expressing emotional distress, and poor ability to express emotions in more
mature ways leads to somatic symptoms as an indirect expression of distress. A related developmental question arises: Do undiagnosed or untreated SDs in childhood predispose to persistent somatization in adolescence and adulthood? Follow-up studies suggest that the answer is yes. Preventive public health considerations point to the merits of early clinical intervention in these disorders to avoid excessive medical consultations, unneeded medical testing, inappropriate treatments, and the potential chronic disability that can ensue in the development of “illness as a way of life.”

**Postulated pathogenic influences**
A number of theories have been advanced to explain the development of somatizing propensities in vulnerable persons. Psychoanalytic theory in its classic form invokes the presumptive role of repressed conflicts regarding socially “unacceptable” sexual or aggressive impulses that are consciously suppressed or unconsciously repressed and then intuitively converted into physical symptoms, generating escape from the conflict. Modern psychodynamic theories focus on difficulties in expressing emotions verbally, which is frequently observed among somatizing patients, and on the tendency to revert to developmentally less mature somatic expression of emotions.

Learning theory postulates the gratification of dependency needs in somatizing individuals. Physical symptoms elicit help and comfort from others when an individual is frightened or overwhelmed. The ensuing relief from stress reinforces the symptoms’ perpetuation. Moreover, failure to learn higher-order ways of emotional expression (eg, verbal) as a result of a suboptimal interpersonal learning environment can lead to a tendency for a predominantly somatic expression of emotion. Behavior analysis focuses on the patient’s behavioral reaction to environmental factors. Physical symptoms allow withdrawal from aversive social stressors (eg, school, work). The emphasis here, which overlaps with learning theory, is on those environmental contingencies that can be modified.

Recent advances in social-affective neuroscience suggest that early interpersonal environment may interact with genetic predisposition and epigenetic changes to affect the neural circuits involved in interpersonal emotions and physical pain. This type of predisposition makes a person particularly sensitive to emotional stressors and presents difficulties in regulating emotional and somatic distress. This could explain the variable vulnerability to somatization under similar stressors among different individuals. It also points to the need to carefully evaluate these relevant vulnerabilities in psychotherapeutic exploration of each patient’s unique biographical narrative.

Attention to autoimmune sensitization, particularly regarding postulated somatic consequences of depression, may have particular relevance to psychological factors that influence medical conditions. Examples include rheumatoid, postinfectious, fibromyalgic, irritable bowel, neuropathic pain, and chronic fatigue syndromes. These somatic syndromes could be influenced by metabolic, immuno-inflammatory, autonomic, and hypothalamic-pituitary-adrenal axis dysregulations that can be exacerbated by stress.

Dissociation appears to play a role in many cases of somatization. It involves the relative suspension of awareness of oneself or of the environment, as an accompaniment of focal concentration. This often occurs unconsciously in cases of somatization that emerge outside of the individual’s awareness. In this situation, a learned withdrawal response to stress develops, wherein anxiety, depression, or obsessive-compulsive features generate a dissociative response. This response, in turn, precipitates somatic symptoms as a route of escape from stress.

**Treatment implications**
Implicit in the theoretical formulations presented above is the presumption that a maladaptive pattern of avoidance of emotions and withdrawal has been pathologically “learned” by the patient. Psychotherapeutic (and sometimes adjunctively pharmacological) treatment can help restructure these learned patterns. Supportive explanation and learning adaptive ways to express and regulate painful emotions can attenuate symptoms and improve adaptive functioning. Functional MRI studies have shown that clinical states of anxiety, depression, obsessive-compulsive, and SDs involve altered states of brain physiological activation that can, in many cases, be normalized with appropriate treatment. This is consistent with the observation that “different forms of psychotherapy lead to different structural changes in the brain, just as other forms of learning do.”

It is worth considering the power of relevant clinical imagery in establishing a therapeutic rapport so as to capture the patient’s and his or her family’s attention and to facilitate change in their beliefs, which are often initially wedded to the notion of a primary or exclusively organic basis of the presenting physical symptoms. Hence the example of distinguishing chest pain and shortness of breath from a heart attack and a panic attack: both are real and treatable, yet they have a different pathophysiology and require different treatments. The importance of a thorough differential
diagnosis is emphasized, but the need for open-mindedness and effective collaboration between primary care physician and psychiatrist is highlighted.

Another example of the power of metaphor in facilitating patient and family conceptual reorientation is using a computer model to delineate the distinction between neurological and psychiatric impairment in generating physical symptoms. In this model, neurological impairment is depicted as “hardware” damage, compared with psychogenic (somatoform) impairment, depicted as “software” malfunction. The latter represents better news for the patient, since “reprogramming” of brain function with psychotherapy is often successful, whereas brain parts are not replaceable. The goal of patient engagement (as well as of potentially supportive family members) is to bridge the commonly encountered gap of brain-mind dualism, thereby reducing the stigma frequently activated by the presentation of possible “psychogenic symptoms.” This process can instill hope and optimism regarding the patient’s capacity for clinical improvement, especially if failed treatments have been premised on a purely physical etiology. Indeed, it may be reasonable to consider psychotherapy in this situation as a type of “reconversion,” away from a misguided belief in a purely physical and treatment-unresponsive illness. The patient is then free to effectively participate in treatment geared to restoring health, with a more accurate diagnosis and a new treatment approach.

**Diagnostic considerations: DSM-IV and DSM-5**

The most significant shift diagnostically between DSM-IV and DSM-5 regarding these disorders is the elimination of the presumption of an unconscious basis of the category of symptoms that had been characterized in DSM-IV as somatoform disorders. Since it is often not feasible for an evaluating psychiatrist to objectively judge intentionality on the part of a patient, this component of the definition was dropped. The full gamut of somatizing symptoms in DSM-5 has been subsumed under the heading of SSDs. An explanation of all the changes in categorization between DSM-IV and DSM-5 and discussion of their effects on clinical practice are beyond the scope of this article, but major changes are summarized in the Table.

A few comments on the changes in DSM-5 as they pertain to the SDs are warranted. First, the definition of SSDs, aside from removing the requirement for the clinician to judge the patient’s intentionality, also deemphasizes the central role of medically unexplained symptoms. One reason for this is that unexplained symptoms may represent undiagnosed medical illness, which may therefore become a potentially dangerous pitfall for premature psychiatric diagnosis. Instead, DSM-5 defines SSDs on the basis of positive symptoms (ie, the presence of distressing somatic symptoms, together with excessive thoughts, feelings, and behaviors in response to these symptoms, beyond what appears medically explainable by available physical findings). Designated “specifiers” in certain diagnostic categories refer to symptom severity and to the possible presence of pain. The DSM-5 substitution of illness anxiety disorder for hypochondriasis in DSM-IV was based primarily on a wish to supplant the stigma that had become associated with the pejorative use of hypochondriasis. More controversy attended the proposal to supplant conversion disorder with functional neurological disorder, so both are options in DSM-5. The inclusion of psychological factors that affect a medical condition and factitious disorder under the rubric of SSDs in DSM-5 was based on the advocacy that clinicians consider all of these somatizing variants in the complex challenge of differential diagnosis.

Factitious disorder and its subtypes connote the purposeful falsification of physical or psychological signs or symptoms, or the induction of injury or disease associated with identified deception. In factitious disorders, the deceptive behavior is evident even in the absence of obvious, pragmatic external rewards; the presumptive psychopathological goal is to obtain medical attention or treatment. This is in contrast to malingering, which is not a psychiatric diagnosis. Malingering connotes a purposeful deceptive presentation of a physical symptom or other medical condition with the conscious intent of obtaining a pragmatic benefit, such as financial gain, narcotic medication, or exculpation from work responsibility or criminal activity. Our key clinical advisory in the initial approach to the presumably somatizing patient is to attribute an unconscious basis to symptom formation, as a reaction to cumulative, overwhelming life stressors. This allows an empathic therapeutic bond to form. Only in the face of substantial and persuasive evidence of dissimulation or conscious manipulation by the patient should the possibility of purposeful misrepresentation be addressed more directly. Doing so prematurely and in a disparaging tone immediately and possibly irrevocably transforms an initial therapeutic alliance into an adversarial relationship. It should be remembered that even in the case of more classic SSDs, there may be some “secondary benefits” to the symptoms, while the symptoms may have initially emerged as a purely unconsciously based stress reaction.
Approach to treatment
When transitioning from initial diagnostic impression to treatment plan formulation, it is wise to consider SSD as a working diagnostic hypothesis. If one has elicited, beyond the medical history, an adequate delineation of personal, family, vocational, and social history, one can consider whether the symptoms are a plausible psychological “protective function.” That is, do the symptoms serve to avoid a constellation of stressors with ensuing functional impairment, by allowing the patient to retreat into “the sick role”? Moreover, might the symptoms be the body’s reaction to overwhelming stress?

The psychotherapist explores possible predisposing, precipitating, and perpetuating factors contributing to symptom genesis. Many patients may not be able to articulate the complex environmental stressors that produce feelings of shame or inadequacy. They may cling to the identity of the medically ill patient as a “safer” refuge from life’s adversities. Therefore, the psychiatrist should present the diagnostic hypothesis of SSD tentatively and supportively, noting that it is not mutually exclusive of coexisting physical illness.

As the psychotherapeutic relationship evolves toward a treatment plan, it is important to achieve diagnostic consensus between the referring primary care physician, the psychiatrist, the patient, and any involved family members. It is less important to pin down initially the subtype of SSD than it is to have the patient sufficiently comfortable with the concept that psychological factors (many patients prefer the term “stressors”) play a significant role in their presenting medical condition. If the initial psychiatric evaluation occurs on an inpatient medical unit, it is helpful to have a conjoint diagnostic debriefing and treatment plan formulation meeting involving the patient, psychiatrist, and primary care physician, as well as relevant family members and other relevant hospital staff, with the patient’s authorization. This tends to cognitively and emotionally reinforce the reorientation of the patient’s understanding of the symptoms, so that treatment can proceed effectively. If the psychiatric evaluation occurs on an outpatient basis, then a thoughtfully formulated clinical report from the psychiatrist to the referring primary care physician, with a copy to the patient, may be comparably helpful in advancing this goal.

Treatment options
As with any clinical intervention, treatment planning needs to be individually formulated. There are multiple options for therapeutic intervention that may be helpful for particular patients. Those for which some supportive clinical evidence is available follow.

Reassurance, placebo, suggestion, and psychoeducation all have a long history of anecdotal efficacy. These interventions can be provided by a primary care physician or other clinician in acute and short-duration SSDs. Cases that arise in response to self-limited situational stress from which the patient can rebound with minimal but adequate short-term support need not necessarily be formally psychotherapeutic.

Individual and/or family psychotherapy is often needed, however, when SSDs are longer in duration and more impairing of function. In this case, a more thorough and sustained clinical intervention is required. Various approaches, which are not mutually exclusive, can be used. Psychodynamic strategies that focus on uncovering the painful affects and working through the patient’s interpersonal history and schemas that often make it difficult for a patient to express emotions are at the core of psychodynamic treatments. The treatment involves learning adaptive ways to express emotions. The therapist explores “unconscious conflicts” that are elicited in the narrative history and attempts to generate destigmatization regarding painful conflicts as well as a more enlightened strategy to negotiate the conflicted areas in a more appropriately self-actualizing manner. This allows disengagement from the cumbersome and eventually unneeded somatic symptoms.

Behavior modification restructures relevant environmental influences to improve adaptive functioning. This approach has the greatest applicability with children, adolescents, and severely impaired adults. The caregiver of these individuals may be positively reinforcing the patient’s symptoms and associated dependency by well-intentioned overprotection that derives from a misunderstanding of the illness. Alternatively, this pathological reinforcement of the patient’s SSD may derive from a pathological conscious or unconscious need to perpetuate the pathologically dependent relationship. In the more benign variant, the caregiver may be a powerful ally in the therapeutic reorientation. In the more malignant form (factitious disorder imposed on another), outside intervention, such as by child protective services, may be necessary.

Cognitive-behavioral therapy has a body of empirical support with a substantial number of randomized clinical trials. This approach focuses on conceptual reformulations, so that the emphasis is on restructuring the patient’s cognitive approach to understanding the conditions that...
generated disability and by clarifying how the patient can deal with those conditions more effectively. The typical strategies include hypothesis testing, exposure and response-prevention to feared situations, and symptom and improvement monitoring. These strategies are pursued in a more directive manner than is usual with a psychodynamic approach.

**Other interventions**

Group psychotherapy can be very helpful either as an adjunct to individual treatment or as a stand-alone therapeutic approach. A unique benefit of this approach is destigmatization and normalization of the patient’s symptoms, as well as a decrease in alienation that many patients with SSD often experience.

Mindfulness meditation, progressive relaxation, and deep breathing techniques can be very helpful as an adjunct treatment for somatization. These approaches promote healthy mind-body regulation, decrease physiological and psychological stress, and increase a sense of control over somatic symptoms among patients.

Hypnosis can be useful as a ceremonious facilitator, using guided dissociation as a restructuring aid. This can be integrated with any of the psychotherapy strategies noted above and may be particularly helpful in receptive, hypnotizable patients.

Physical and occupational therapies can be invaluable when actual muscular weakness has developed as the result of a functional neurological (conversion) symptom. They also may have powerful psychological value in a face-saving sense for patients who have difficulty in grasping the concept of somatization, who are uncomfortable with psychotherapy, and who want hands-on help to overcome their physical symptoms.

Psychopharmacological agents may have specific therapeutic benefit for comorbid psychiatric disorders, including anxiety, depression, obsessive-compulsive disorder, and psychosis, all of which may coexist with and complicate SSDs. In addition, these agents may have nonspecific (placebo) benefits. For patients who have difficulty in grasping the concept of somatization, who have discomfort with psychotherapy, or who want a “medicine” to legitimize the validity of their physical illness and recovery, a supportive discussion of the role of these medications in normalizing brain neurotransmitter function can be helpful. The medicine can be the needed aid that helps the psychotherapy go down.

For severely or chronically impaired patients with SSDs in whom sustained outpatient efforts at treatment have failed or who do not have access to appropriate treatment locally or are living in a psychologically toxic or abusive environment, it is sometimes very helpful to arrange for inpatient treatment on a medical unit where intensive psychiatric and physical rehabilitation resources are available.

While it may be challenging to secure insurance pre-clearance, the documented capacity to intensively integrate the above range of therapeutic modalities on an inpatient service with good results not otherwise feasible for complex, treatment-resistant cases can be persuasive. A thoroughly documented combination of medical and psychiatric consultative reports, forwarded to the insurance company, may effectively make the case for investing in an intensive short-term treatment to generate sustained clinical improvement and thus diminish the longer-term greater costs of disability.

**Conclusion**

Further research is needed for the deeper understanding of the etiology and underlying mechanisms of somatization. Recent advances in translational research and the integration of basic and clinical research findings provide new insights into this challenging-to-treat condition.

Table: DSM diagnostic changes in classifying the somatizing disorders

**Disclosures:**

*Dr Williams is Attending Psychiatrist at the Columbia University Medical Center and Special Lecturer in Psychiatry at the Columbia College of Physicians and Surgeons, Neurological Institute, New York.*
Dr Landa is Assistant Professor of Clinical Psychology in Psychiatry at the Columbia University Medical Center. The authors report no conflicts of interest concerning the subject matter of this article.

References:


**Source URL:**
http://www.physicianspractice.com/printpdf/psychiatric-diagnosis-and-treatment-somatizing-neuropsychiatric-disorders/page/0/1

**Links:**