Somatization and Conversion Disorder

Trevor A Hurwitz, MD[1]

Somatization is the psychological mechanism whereby psychological distress is expressed in the form of physical symptoms. The psychological distress in somatization is most commonly caused by a mood disorder that threatens mental stability. Conversion disorder occurs when the somatic presentation involves any aspect of the central nervous system over which voluntary control is exercised. Conversion reactions represent fixed ideas about neurologic malfunction that are consciously enacted, resulting in psychogenic neurologic deficits. Treatment is complex and lengthy; it includes recovery of neurologic function aided by narcoanalysis and identification and treatment of the primary psychiatric disorder, usually a mood disorder.

(Can J Psychiatry 2003;49:172–178)

Information on author affiliations appears at the end of the article.

Highlights

- Somatic symptoms are a psychological defence against mental instability.
- The psychiatric disturbance that drives the somatoform disorders is in most cases a major affective illness.
- Conversion disorder is a specific form of somatization in which the patient presents with symptoms and signs that are confined to the voluntary central nervous system.

Key Words: *somatization, conversion disorder, narcoanalysis, depression, somatoform disorder*

Somatization is the tendency of individuals to experience and communicate psychological distress in the form of somatic symptoms and to seek medical help for them (1,2). This psychological process gives rise to somatoform disorders, which are typically first seen in nonpsychiatric settings. Their core features comprise somatic symptoms and signs that cannot be explained by known disease and that result in social and occupational impairment. Table 1 lists the clinical criteria.

Symptoms are typically multiple and vague and may refer to single or multiple body systems or functions. Presentations include cardiopulmonary, gastrointestinal, genitourinary, musculoskeletal, and neurologic complaints, as well as pain and fatigue (3,4). The DSM-IV-TR diagnostic categories that incorporate the concept of somatization include body dysmorphic disorder, conversion disorder, hypochondriasis, somatization disorder, pain disorder, undifferentiated somatoform disorder, and somatoform disorder not otherwise specified (5).

In clinical practice, somatoform disorders overlap and most commonly present with multifocal symptoms and signs (6). The spectrum of presentations varies from mild to severe and may be mostly symptom-based (hypochondriasis) or mostly sign-based (conversion disorder).

Sickness

Somatoform disorders are best understood within the context of sickness, because patients present as physically sick, which initially masks the underlying psychiatric disorder. Sickness comprises 3 components: disease, illness behaviour, and predicaments (7,8). Disease is the physical basis of sickness caused by tissue abnormality or malfunction. Clinically, disease manifests by observable signs (what the physician sees), such as edema, hemorrhage, or inflammation, and organ and system compromise, such as heart failure and paralysis. Illness behaviour refers to the subjective experiences and behavioural consequences of disease (what the patient complains of). Illness behaviour presents as symptoms. These may be nonspecific symptoms, such as fatigue or nausea. Symptoms may also be referable to specific systems or physical functions, such as shortness of breath or an inability to move; to specific mental dysfunctions, such as problems with concentration or sadness;

Table 1 Clinical criteria for psychogenic somatic symptoms and signs

Disease absent

- No identifiable organic cause
- Symptoms and signs do not correlate with known organic damage patterns

Disease present

- Symptoms and signs do not correlate with the damage patterns of the known disease
- Severity of symptoms and signs is disproportionate to the known disease
- Duration of symptoms and signs is disproportionate to the known disease
- Dysfunction induced by symptoms and signs is disproportionate to the known disease

Table 2 The differential diagnosis of psychogenic somatic symptoms and signs

	Somatoform disorder	Factitious disorder	Malingering
Conscious of motivation	No	No	Yes
Conscious of faking	No	Yes	Yes

or to impaired interpersonal behaviour, such as social withdrawal or compromised self-reliance. Predicaments are the psychosocial consequences of illness. These are the negative and positive interpersonal ramifications arising from being sick. Predicaments include interpersonal dependence; exemption from normal domestic and societal obligations such as work; financial compensation without labour; sanctioning of symptom-relieving interventions such as medications, including the use of narcotic analgesics; granting of special community privileges, such as reserved parking, special transport, and first access; and avoidance of noxious consequences by mitigation of responsibility, such as being relieved from doing military service or serving jail sentences.

Within the context of sickness, somatoform disorders have the appearance of disease, with the advantages and disadvantages of illness and predicaments. The conclusion—reached after appropriate investigation—that there is no disease engenders distrust and hostility on the part of the medical profession. Patients are poorly received and treated, and in this negatively charged physician–patient dynamic, the underlying disease of psychiatric disorder is often overlooked or ignored.

Pathogenesis of Somatic Symptoms

Somatic symptoms are a psychological defence against mental instability. Like all other intrapsychic defences, symptom formation reduces intrapsychic distress (9). This is known as the primary gain (10). Primary gain strives to restore psychological equilibrium, but at a price. Reality is distorted. Attention is redirected toward the presenting symptoms, and the real problem and source of mental instability is blocked out or only partly experienced and, hence, not addressed (9).

Once present, a symptom may be consciously used to achieve optimal interpersonal benefits. This is known as the secondary gain (10). Secondary gain is an ubiquitous interpersonal strategy sanctioned by society if the underlying problem is seemingly genuine or if the interpersonal advantages accrue via sophisticated or adaptive behaviour. By contrast, hostility and rejection are the usual responses when interpersonal advantages are extracted from seemingly fake problems, such as sickness without disease, or when interpersonal strategies are primitive and transparently manipulative and maladaptive. Such strategies include unsophisticated interpersonal coercion, avoidance of responsibility, avoidance of noxious consequences, undeserved compensation, and financial reward without labour. Negative physician response to secondary gain is unavoidably the result of a value judgement and applies regardless of whether behaviours have arisen from organic or from psychiatric conditions. In somatoform disorders, physician hostility is magnified when secondary gain is felt to derive from signs and symptoms without any underlying disease.

In somatization, the fundamental disturbance is a psychiatric illness that threatens mental stability. The threat to mental integrity produces anxiety that mobilizes the somatic defences (11). These defences are responsible for nonorganic physical symptoms or convert psychic pain into physical pain. This initial process constitutes the primary psychological gain. Such patients, like anyone else, use the emergent symptoms for interpersonal advantage to make the most of their predicament. This constitutes the secondary psychological gain.

Primary gain occurs unconsciously. As a result, the emergent somatic symptoms are experienced as an unwanted and uninvited disturbance. Patients believe and feel that they are sick. Somatizing patients are not aware of their underlying psychiatric disturbance—the motivation that drives the symptoms. These patients are also not aware that they are deliberating faking symptoms. However, the specific form of illness that surfaces reflects the patient's conscious beliefs about how disease should present. Symptoms and signs that derive from beliefs are known as ideogenic (12). Since they reflect a patient's concept of sickness, rather than organically disturbed anatomy or physiology, they appear atypical or bizarre to the examining physician. They either occur in the absence of identifiable disease or the pattern, severity, and duration of symptoms and signs, as well as the induced dysfunction, do not match any known disease. Symptom formation in somatization is best studied in conversion disorder, where the

symptoms are externalized as an observable neurologic deficit. This deficit can be reversed, revealing in the process the operative psychological mechanisms (12).

Malingering and Factitious Disorders

An identical cross-sectional presentation is found in 2 other confounding conditions and is partly responsible for the hostile reception given to somatizing patients. In malingering, the patient consciously decides to fake illness. The underlying motivation is also conscious and deliberate and is either to avoid noxious consequences or to gain undeserved benefits (13,14). Patients who malinger know that they are pretending to be ill. Malingered signs and symptoms are ideogenic in that they reflect the patient's conscious understanding of how illness should present. As such, the somatic presentation of malingering is indistinguishable from that of the somatizing patient. In factitious disorder, the patient makes a conscious decision to fake an illness. Unlike malingering, however, the underlying motivation has never been understood but is caused by an unidentified, unconscious psychological need. Nonetheless, factitious patients are fully aware that they are pretending to be ill, although they cannot clarify either to themselves or to others the purpose, other than to receive medical attention at any cost, that such a charade serves (14–16) Since the somatic symptoms are ideogenic, they too are indistinguishable from those of the somatizing patient or the patient who is malingering (Table 2).

Depression and Symptom Formation in Somatization and Conversion

The psychiatric disturbance that drives the somatoform disorders is in most cases a major affective illness. Major depression has been identified in 30% to 60% of patients with chronic pain and in 48% to 90% of patients who somatize. Depressive illness has also been found to be present in 54% to 88% of patients presenting with conversion symptoms (17–23).

There is no agreed-upon explanation about why patients use somatic defences. Factors that have been associated with somatization include increasing age, low social class, history of physical illness, family history of physical illness, membership in cultural groups that discourage emotional expression, alexithymia (nonpsychological mindedness), and fear of psychiatric stigmatization (24–27).

How or why a patient chooses a specific symptom or sign is also unclear. Explanations include a symbolic connection to an underlying conflict or, alternatively, symptom modelling in which patients mimic somatic symptoms that have previously occurred in themselves or in a family member as a result of organic disease (10). Where the symptom is modelled upon prior organic disease, the term "somatic compliance" has been used (28). The term "functional overlay" is used when preexisting organically determined symptoms and signs are elaborated and expanded upon by psychogenic mechanisms, resulting in nonorganic presentations (29).

Conversion Disorder

Conversion disorder is a specific form of somatization in which the patient presents with symptoms and signs that are confined to the voluntary central nervous system (5). When conversion symptoms occur in isolation, the primary diagnosis is conversion disorder. When conversion symptoms occur as part of a multisystem somatoform syndrome, the primary diagnosis is somatization disorder (10). "Conversion" or "conversion reaction" refers to the process whereby intrapsychic distress is converted into physical neurologic symptoms (30). Classically, patients present with psychogenic seizures or psychogenic motor-sensory deficits. Neurologic presentations may, however, involve any aspect of the central nervous system over which voluntary control is exercised. Thus, patients may present with a psychogenic dementia as well as loss of speech and language or a disturbance of any of the special senses (30,31).

The hallmark of a psychogenic neurologic presentation is that the disruption in voluntary neurologic function does not follow known neurologic damage patterns (30). This reflects the fact that the neurologic presentation is ideogenic and derives from patient beliefs about how neurologic symptoms should present. The emergent symptoms and signs are resilient and fail to resolve following negative neurodiagnostic investigations, reassurance, or care-provider rejection and persist across all settings. Typically, the observed signs—such as hemiparesis or blindness—are much worse when they are being formally evaluated or when circumstances bring the deficits to the patient's attention. However, whether observed or unobserved, the neurologic dysfunction remains present and interferes with the patient's functioning: the symptoms are fixed because the beliefs are fixed. Somatic symptoms and signs attributable to fixed beliefs provide the grounds for considering that such beliefs represent somatic delusions (12).

The pathophysiology of conversion symptoms thus begins with a psychiatric illness—most commonly depression—that threatens to destabilize mental functioning. The somatic defence, the conviction that "I am physically ill," forms unconsciously and surfaces as a fixed belief about the presence of a specific neurologic malfunction. The patient applies this belief consciously to govern behaviour, resulting in bizarre, atypical, and nonorganic findings on neurologic examination. The role of active inhibition has recently been demonstrated in some, but not all, functional-imaging studies (32–36). In a landmark positron emission tomographic blood flow study of chronic and total left-leg psychogenic paralysis,

attempts by the patient to move the paralyzed leg failed to activate the right primary motor cortex (32). Instead, the right orbitofrontal and right anterior cingulate cortex were activated. These activated areas were deemed to be responsible for the inhibition of the right premotor and primary sensorimotor cortex and the resultant left leg weakness. This active inhibition only became manifest when the patient was consciously trying to move the paralyzed leg. At rest, there was no significant asymmetry of activity in the motor cortices. A similar pattern of simultaneous activation of right frontal inhibitory areas and inhibition of right somatosensory cortex was found in case of left-sided paralysis and paresthesias studied by single-photon emission computed tomography (SPECT) during electrical stimulation of the left median nerve (33). Following neurologic recovery, the perfusion patterns normalized appropriately. In a functional magnetic resonance imaging study of psychogenic sensory loss, painful and tactile stimulation deactivated or failed to activate expected primary and secondary somatosensory cortices and associated cortical and subcortical networks. Simultaneously, and similar to the Marshall and others study (32), the anterior (rostral and perigenual) cingulate cortex was activated, but only during sensory stimulation that was not subjectively perceived. Among other observations, the study findings suggested abnormal cognitive or attentional processing during unperceived stimuli (34). In a SPECT study of acute unilateral psychogenic motor-sensory loss, bilaterally applied passive vibration led to hypoactivation of thalamus, caudate, and putamen contralateral to the deficit, which resolved with neurologic recovery. This study did not show any regions of significant asymmetric cortical activity (35).

These functional imaging studies provide a growing body of data supporting the role of conscious active inhibition in the genesis of psychogenic deficits. Symptom formation that depends upon conscious active inhibition explains why psychogenic neurologic deficits are less severe when patients are distracted and more severe when patients attend to their problem (12). The findings on neurologic examination are also consistent with cortical and subcortical inhibition controlled and shaped by higher-order brain centres. For example, neurologic deficits that are caused by a disturbance originating in the cortex itself should produce a typical damage pattern, such as the pyramidal distribution of weakness occurring in a patient who presents with paralysis (30). The physical findings, however, do not follow these known organic patterns. Instead, physiologically and anatomically naive beliefs applied consciously and mediated via higher-order brain centres inhibit downstream cortical and subcortical areas in such a way that the nonorganic neurologic pattern results. Last, the role of beliefs in consciously shaping symptoms and signs provides a coherent and consistent explanation for psychogenic neurologic presentations that involve positive neurologic phenomena such as psychogenic seizures and psychogenic movement disorders (37). On neurologic examination, these are also recognizable as psychogenic because they, too, fail to follow known organic damage patterns.

Like other somatoform disorders, conversion disorder can be a treacherous condition: in the past, up to 30% of patients diagnosed with conversion symptoms were subsequently discovered to have misdiagnosed organic illness (10). In contemporary medical practice, with the availability of sophisticated neuroimaging techniques such as magnetic resonance imaging, missed organic illness may account for 4% to 15% of individuals initially given a diagnosis of conversion disorder (38–43). To avoid this error, all patients must be thoroughly medically investigated.

Management of Conversion Disorder

Patients referred for the treatment of conversion disorder must first be medically cleared for any neurologic condition. All such individuals should have undergone appropriate neurodiagnostic investigations, including prolonged video-electroencephalographic monitoring in the event of a seizure presentation.

Conversion symptoms, especially when seen acutely, may resolve spontaneously with explanation and suggestion. In some patients, psychogenic sensorimotor deficits may respond to treatment in a rehabilitation unit using a behavioural approach with no other psychiatric intervention (44,45). In this group of patients, the primary psychiatric disturbance has seemingly settled. They are left with neurologic deficits from which they need a face-saving exit. Active rehabilitation provides such a vehicle.

Outpatient treatment of patients with conversion symptoms can be attempted using some of the strategies used in the inpatient setting. Patients with chronic and entrenched conversion symptoms usually require admission to an inpatient psychiatric unit that has experience with conversion disorders. They may undergo acute psychiatric decompensation as their neurologic symptoms resolve. This decompensation reflects the deconstruction of the somatic defence by treatment that unmasks the underlying psychopathology, usually depression. The unmasked psychopathology typically emerges over weeks, varies from mild to florid, and may include previously hidden psychosis (23,46).

Patients with sensorimotor disturbances are told that their neurologic deficits result from a loss of conscious control over the affected function caused by an underlying neurochemical disorder, usually, depression. This explanation provides the cognitive framework for treatment. It is accompanied by physiotherapy, which may be all that is needed for neurologic

recovery, provided that the patient's underlying psychiatric disorder has been identified and accepted and appropriate treatment started. In patients with entrenched conversion symptoms, narcoanalysis and narcosuggestion are required to initiate neurologic recovery. Methylphenidate 5 to 15 mg (a 1-time dose prior to starting each narcoanalysis) is administered orally. Thirty minutes later, amobarbital is administered intravenously at a rate of 50 mg/minute until the patient develops nystagmus and (or) dysarthria (46). The patient is encouraged to regain voluntary control through visual imagery and suggestion. Narcoanalysis is videotaped and reviewed the next day with the patient. Videorecorded neurologic recovery, no matter how small, cogently confirms that the deficits have a psychological basis. Narcoanalysis is repeated weekly over several weeks, provided that there are additional neurologic gains with each procedure. At least 2 narcoanalyses should be done, as the first narcoanalysis may not yield any neurologic gains. Recovery of neurologic function is usually slow and takes place over days, weeks, and months; it is rarely abrupt. Any recovered neurologic function is reinforced with physiotherapy and appropriate physical aids provided by a physical therapist familiar with psychogenic neurologic deficits (47).

Narcoanalysis is also helpful, but not essential, in uncovering the underlying primary psychiatric disorder. During the initial narcoanalysis, which may last up to 2 hours, a formal review of the patient's history and mental status is undertaken. Areas covered include a detailed review of the circumstances and psychological disturbances present at the onset of the conversion symptoms; a review of any relevant psychosocial stressors and conflicts; and the identification of psychopathology, including any source of symptom modelling and any secondary gain.

Exploring the patient's mental status, with or without narcoanalysis, is an ongoing process throughout treatment; it aims to identify and confirm the underlying primary psychiatric disorder. This may be immediately recognizable. More often, the underlying psychiatric disorder emerges over weeks, but it may surface precipitously. Relevant dynamics and psychosocial stressors are also sought and explored. A histrionic personality disorder is not a prerequisite for conversion disorder (10). La belle indifference is not diagnostically helpful, as it may be seen in organic conditions (48). Last, disturbed sexuality has no specific connection with conversion symptoms, although historically it has been identified as one of the primary conflicts associated with this condition (10).

In patients whose conversion symptoms have been present for fewer than 6 months, the underlying psychiatric disorder (most commonly, depression) is often readily discovered (21). With chronic conversion symptoms lasting more than 6 months, the underlying psychiatric disorder is often not easily identified. In these patients, the psychopathology may have been identifiable at the onset of their illness (21). However, months or years later, when some of these patients are finally recognized and accept treatment, their overt, acute, precipitating psychiatric disturbance has become submerged and entwined with their physical symptoms. The retained physical symptoms now provide a tenaciously held explanation for their pain, distress, despair, insomnia, lack of energy, and functional failure.

An essential part of treatment is the establishment of a therapeutic alliance that allows patients to recover with dignity and without loss of face. Staff need to be prevented from responding to these patients in nontherapeutic ways, such as rejecting them or becoming angry because of their "unnecessary" physical dependence and nursing needs or suggesting, subtly or overtly, that they are attention-seeking, manipulative, and exaggerating or faking their neurologic difficulties. Patients need to be helped to recognize their psychopathology and accept that their symptoms arise from a psychiatric rather than a neurologic condition. Patients must also be helped to accept orthodox psychiatric treatments. At every stage, vigilance needs to be maintained for new symptoms. Any emergent symptom needs to be adequately evaluated and not dismissed as another conversion reaction.

Psychogenic seizures require a different approach. Patients are told that they are having spells, not seizures, and that these spells are caused by a neurochemical, not an electrical, malfunction of the brain. Medical attention is then gradually shifted away from the spells. Anticonvulsants should never be abruptly abandoned but should be tapered slowly. Provided that the underlying psychiatric illness is addressed by treatment, the spells will resolve over time, which can be weeks or months.

Helpful psychotherapeutic strategies include cognitive-behavioural and expressive–supportive therapy. An overdetailed history, in which physical symptoms are reviewed on each and every occasion and in minute detail, often helps. This task can be demanding for physicians, but it reassures patients that their physical symptoms are not being dismissed or overlooked. A rational cognitive framework for understanding their symptoms helps patients. This is achieved by providing a tangible mechanism and nonblaming explanation to account for their neurologic symptoms (49). Patients are told that current evidence indicates that conversion disorder is caused by a neurochemical disorder of the brain, most commonly, depression. This neurochemical disturbance does not manifest through typical psychiatric symptoms but, rather, surfaces via unconscious mechanisms as physical symptoms. Typical psychological hurdles to be overcome include patients' reluctance to accept the idea that psychiatric disorder can cause the obvious and more acceptable somatic symptoms and their reluctance to accept orthodox psychiatric

therapies, even though they have usually tried many complementary therapies.

Family therapy is almost always necessary. Families have often invested heavily in patients' symptoms and devoted considerable time and resources to helping patients deal with their neurologic disabilities. The family will therefore also have to come to terms with the fact that the neurologic disability has been caused by a psychiatric illness. Helping families accept this condition as a genuine sickness, but one that is psychiatric rather than neurologic, ensures that they continue to support patients through the period of recovery and beyond.

Pharmacotherapy involves energetic psychotropic medication trials. Narcotic analgesics taken to control the nonorganic pain that may accompany psychogenic neurologic deficits need to be withdrawn. In this setting, they are presumably an attempt to self-treat the dysphoria of the underlying psychiatric disorder.

Patients with conversion disorder commonly demonstrate physiological and psychological medication hypersensitivity characterized by unusual side effects, in part linked to their reluctance to accept that they have a psychiatric disorder. They will prematurely abandon medication trials if not given adequate reassurance and support. Medications may therefore need to be started at low dosages and gradually titrated upwards. Here, the goal is to give antidepressants in appropriate dosages and for an adequate duration, similar to the dosing regimen of any mood disorder. Standard antidepressant medications are used and include the selective serotonin reuptake inhibitors, tricyclic antidepressants, and novel antidepressants such as venlafaxine and bupropion. Neurologic symptoms that fail to resolve with antidepressants alone should be treated with neuroleptics. Neuroleptics target somatic delusions (one hypothesis for the resistance of somatic symptoms in the face of incontrovertible evidence that no organic disease is present). Atypical neuroleptics are preferable because they carry a lower risk of tardive dyskinesia. If this strategy fails, patients should be offered a course of electroconvulsive therapy, which may succeed in recovering neurologic function and effectively treat the underlying psychiatric disorder (46).

Prognosis

Few studies describe the prognosis for psychogenic deficits and their associated primary psychiatric disorder. A shorter duration of psychogenic neurologic deficits is associated with a better neurologic prognosis (39–41). Even without specific treatment directed at recovering neurologic function, 28% to 63% of patients will have a complete remission of their presenting neurologic symptoms; 20% to 29% will show some improvement; and 10% to 52% will be unchanged or worse (38–42). In our unit, with treatment algorithm as described, 68% of patients demonstrated a full neurologic recovery, 12% had a variable recovery, and 20% failed to demonstrate any recovery (T Hurwitz and B Kosaka, unpublished observations). These 20% were felt to have a severe and treatment-resistant underlying psychiatric disorder. In a subgroup of these treatment-resistant patients, the acute precipitating psychiatric illness has seemingly fully resolved, but the neurologic symptoms persist, retained as a maladaptive behavioural pattern and perpetuated by the advantages of the sick role, wherein life as an invalid is preferable to all other options. Slater has described this group as "invalids of choice" (50).

Recovery of neurologic function does not, however, end the patients' difficulties. In a 6-year follow-up, 36% of patients had persistent, active, psychiatric problems; only one-third of patients were working full-time; and 47% were retired on the grounds of ill-health. At follow-up, the identified psychiatric disorder was mostly either a continuation or a relapse of the psychiatric disorder identified at the initial presentation (39). In another follow-up study of patients with conversion disorder, 34% were suffering an episode of major depression at follow-up. The mean number of years from the index clinical contact to the follow-up interview was 4.2 (42). These studies indicate the importance of identifying and treating the underlying psychiatric disorder.

Conclusion

Conversion disorder is a challenging psychiatric disorder that requires long-term commitment on the psychiatrist's part and uses the full spectrum of psychiatric skills. Conversion reactions represent a somatic defence against threats to mental stability that are most commonly attributable to an underlying mood disorder. Perseverance through the diagnostic and treatment phases is more often than not rewarded by positive results. Patients require long-term care to manage their mood disorders and associated psychosocial issues. One important role of the treating psychiatrist is to ensure that any new physical symptom receives appropriate medical investigation from the rest of the medical profession. Physicians are usually baffled and annoyed by patients with conversion disorders and remain too quick to dismiss any new somatic symptom as "more of the same," to the patients' great detriment.

References

1. Lipowski ZJ. Somatization: medicine's unsolved problem. Psychosomatics 1987;28(6):296–7.
2. Lipowski ZJ. Somatization: the concept and its clinical application. Am J Psychiatry 1988;145:1358–68.
3. Servan-Schreiber D, Kolb R, Tabas G. The somatizing patient. Primary Care 1999;26:225–42.
4. Servan-Schreiber D, Kolb NR, Tabas G. Somatizing patients: part I. Practical diagnosis. Am Fam Physician 2000;61:1073–8.
5. American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 4th ed. Text revision. Washington (DC): American Psychiatric Association; 2000.

6. Wessely S, Nimnuan C, Sharpe M. Functional somatic syndromes: one or many. Lancet 1999;354:936–9.
7. Pilowsky I. Abnormal illness behavior. Psychiatr Med 1987;5:85–91
8. Anonymous. Predicamants of hysteria. Lancet 1988;1(8583):452–3.
9. Vaillant GE. Theoretical hierarchy of adaptive ego mechanisms: A 30-year follow-up of 30 men selected for psychological health. Arch Gen Psychiatry 1971;24:107–18.
10. Lazare A. Current concepts in psychiatry. Conversion symptoms. New Engl J Med 1981;305:745–8.
11. Gabbard GO. Pschodynamic psychiatry in clinical practice. The DSM-IV edition. Washington (DC): American Psychiatric Press; 1994.
12. Hurwitz TA. Ideogenic neurological deficits: conscious mechanisms in conversion symptoms. Neuropsychiatry Neurospychol Behav Neurol 1989;1:301–8.
13. LoPiccolo C. Current issues in the diagnosis and management of malingering. Ann Med 1999;31:166–74.
14. Wise MG, Ford C. Factitious disorders. Primary Care 1999;26:315–26.
15. Cremona-Barbaro A. The Munchausen syndrome and its symbolic significance. An in-depth case analysis. Br J Psychiatry 1987;151:76–9.
16. Folks D. Munchausen's syndrome and other factitious disorders. Neurol Clin 1995;13:267–81.
17. Lipowski ZJ. Somatization and depression. Psychosomatics 1990;31:13–21.
18. Katon W, Ries RK, Kleinman A. Part II: a prospective DSM-III study of 100 consecutive somatization patients. Compr Psychiatry 1984;25:305–14.
19. Morrison J, Herbstein J. Secondary affective disorder in women with somatization disorder. Compr Psychiatry 1988;29:433–40.
20. Guze S, Woodruff R, Clayton P. A study of conversion symptoms in psychiatric outpatients. Am J Psychiatry 1971;128:643–6.
21. Roy A. Hysteria. J Psychosom Res 1980;24:53–6.
22. Wilson-Barnett J, Trimble M. An investigation of hysteria using the illness behaviour questionnaire. Br J Psychiatry 1985;146:601–6.
23. Hurwitz T, Kosaka B. Primary psychiatric disorders in patients with conversion reactions. Journal of Depression and Anxiety 2001;4:4–10.
24. Lloyd G. Medicine without signs. BMJ 1983;287:539–42.
25. Lloyd G. Somatization: a psychiatrist's perspective. J Psychosom Res 1989;33:665–9.
26. Taylor G. Alexithymia: concept, measurement, and implications for treatment. Am J Psychiatry 1984;141:725–32.
27. Kooiman CG. The status of alexithymia as a risk factor in medically unexplained physical symptoms. Compr Psychiatry 1998;39:152–9.
28. Nemiah J. Somatoform disorders. In: Kaplan H, Freedman A, Saddock B, editors. Comprehensive textbook of psychiatry. 3rd ed. Baltimore: Williams and Wilkins; 1980. p 1525–44.
29. Carter AB. The functional overlay. Lancet 1967:1196–200.
30. Hurwitz TA. Approach to the patient with psychogenic neurological disturbance. In: Kelley WN, editor. Textbook of internal medicine. Volume 2. Philadelphia: JB Lippincott Company; 1989. p 2518–21.
31. Mcevoy J, Wells C. Case studies in neuropsychiatry II: Conversion pseudodementia. J Clin Psychiatry 1979;40:447–9.
32. Marshall JC, Halligan PW, Gereon RF, Wade DT, Frackowiack RSJ. The functional anatomy of a hysterical paralysis. Cognition 1997;64:B1–B8.
33. Tiihonen J, Kuikka J, Viinamaki H, Lehtonen J, Partanen J. Altered cerebral blood flow during hysterical paresthesia. Biol Psychiatry 1995;37:134–5.
34. Mailis-Gagnon A, Giannoylis I, Downar J, Kwan C, Mikulis D, Crawley A, and others. Altered central somatosensory processing in chronic pain patients with "hysterical" anesthesia. Neurology, 2003;60:1501–7.
35. Vuilleumier P, Chicherio C, Assal F, Schwartz S, Slosman D, Landis T. Functional neuroanatomical correlates of hysterical sensorimotor loss. Brain 2001;124:1077–90.
36. Spence S, Crimlisk H, Cope H, Ron M, Grasby P. Discrete neurophysiological correlates in prefrontal cortex during hysterical and feigned disorder of movement. Lancet 2000;355:1243–4.
37. Miyasaki J, Sa D, Galvez-Jimenez N, Lang A. Psychogenic movement disorders. Can J Neurol Sci 2003;30 (Suppl 1):S94–S100.
38. Mace CJ, Trimble MR. Ten-year prognosis of conversion disorder. Br J Psychiatry 1996;169:282–8.
39. Crimlisk H, Bhatia K, Cope H, David A, Marsden D, Ron M. Slater revisited: 6 year follow up study of patients with medically unexplained motor symptoms. BMJ 1998;316:582–6.
40. Couprie W, Wijdicks E, Rooijmans H, van Gijn J. Outcome in conversion disorder: a follow-up study. J Neurol Neurosurg Psychiatry 1995;58:750–2.
41. Binzer M, Kullgren G. Motor conversion disorder: a prospective 2-to 5- year follow-up study. Psychsomatics 1998;39:519–27.
42. Kent D, Tomasson K, Coryell W. Course and outcome of conversion and somatization disorders. Psychosomatics 1995;36:138–44.
43. Moene F, Landberg E, Hoogduin K, Spinhoven P, Hertzberger L, Kleyweg R, and others. Organic syndromes diagnosed as conversion disorder: identification and frequency in a study of 85 patients. J Psychosom Res 2000;49:7–12.
44. Sullivan MJL, Buchanan DC. The treatment of conversion disorder in a rehabilitation setting. Canadian Journal of Rehabilitation 1989;2(3):175–80.
45. Teasell RW, Shapiro AP. Strategic-behavioral intervention in the treatment of chronic nonorganic motor disorders. Am J Phys Med Rehabil 1994;73:44–50.
46. Hurwitz TA. Narcosuggestion in chronic conversion symptoms using combined intravenous amobarbital and methylphenidate. Can J Pysychiatry 1988;33:147–51.
47. Behr J. The role of physiotherapy in the recovery of patients with conversion disorder. Physiotherapy Canada 1996;48:197–202.
48. Gould R, Miller B, Goldberg M, Benson D. The validity of hysterical signs and symptoms. J Nerv Ment Dis 1986;174(10):593–7.
49. Salmon P, Peters S, Stanley I. Patient's perceptions of medical explanations for somatization disorders: qualitative analysis. BMJ 1999;318(7180):372–6.
50. Slater ETO, Glithero E. A follow-up of patients diagnosed as suffering from "hysteria." J Psychosom Res 1965;9:9–13.

Manuscript received and accepted December 2003.
[1]Clinical Professor, Department of Psychiatry, University of British Columbia, Vancouver, British Columbia.
Address for correspondence: Dr T Hurwitz, Department of Psychiatry, University of British Columbia, 2255 Wesbrook Mall, Vancouver, BC V6T 2A1
e-mail: trevorh@interchange.ubc.ca

Résumé : Somatisation et trouble de conversion

La somatisation est le mécanisme psychologique par lequel la détresse psychologique s'exprime sous forme de symptômes physiques. La détresse psychologique de la somatisation est le plus souvent causée par un trouble de l'humeur qui menace la stabilité mentale. Le trouble de conversion survient lorsque la présentation somatique touche un aspect quelconque du système nerveux central sur lequel s'exerce un contrôle volontaire. Les réactions de conversion représentent des idées fixes sur la dysfonction neurologique qui sont consciemment adoptées, ce qui résulte en des déficiences neurologiques psychogènes. Le traitement est complexe et long; il comprend le rétablissement de la fonction neurologique à l'aide de la narcoanalyse, de l'identification et du traitement du principal trouble psychiatrique, habituellement un trouble de l'humeur.