OVERLAPPING SOMATOFORM SYNDROMES IN PERSONAL INJURY LITIGATION

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A number of new syndromes have emerged in the latter part of the twentieth century which often have little if any objective medical pathology. They share remarkably similar symptoms, and are frequently the subject of personal injury litigation, as well as workers' compensation and disability disputes. Because psychological factors are implicated, they are referred to here as Overlapping Somatoform Syndromes. Cultural influence on these conditions is discussed, and their core symptoms are outlined. The role of psychiatric testimony and the points of focus in psychiatric assessment are reviewed. These syndromes more likely than not represent a complex biopsychosocial process rather than the simple attributions proposed in personal injury claims.

We have entered a new age of syndromes: Chronic Pain Syndrome, Failed Back Syndrome, Chronic Fatigue Syndrome, Total Allergy Syndrome, Sick Building Syndrome, and the list goes on. In the psychosocial area, we hear of Repressed Memory Syndrome, Empty Nest Syndrome, Battered Wife Syndrome, and more (1). Although these syndromes present as separate and distinct medical conditions, closer analysis reveals remarkably similar symptomatology, with overlaps to psychological disorders. To the extent medical pathology cannot adequately explain these syndromes, the mechanism of somatization may (2). Diagnostically, medically unexplained syndromes are properly classified as Somatoform Disorders. For heuristic purposes, I will refer to them here as Overlapping Somatoform Syndromes.

In the latter part of the twentieth century, these new syndromes have played themselves out on the stage of personal injury litigation, as well as workers' compensation, and disability disputes. In these forums, the leading testimony regarding these syndromes has typically not been psychiatric, but from another medical specialty, e.g. physiatry, rheumatology, pulmonology, immunology, neurology. Not uncommonly, these experts are the last line of referral when traditional diagnostic studies have been exhausted. Many of these experts take a particular clinical or research interest in the particular syndrome, providing opinions couched in medicalized jargon. Their opinions are often outside mainstream medical thought and difficulty to challenge by ordinary clinicians.

Frequently, psychiatric opinion is sought in these claims as well and has one of two purposes. The first is to assess the degree of emotional damages, since invariably litigants will claim that suffering from the syndrome and its consequences has led to psychological harm. The second is to determine whether there is a primary psychological cause for the symptoms in the event medical pathology cannot adequately explain them. But those qualified to give this psychiatric opinion should be wary about aiding and abetting in the legitimization of these syndromes. Before this new variety of syndromes can be investigated in particular, we must first understand the medically amorphous and culturally dependent nature of syndromes in general.

SYNDROME VERSUS DISEASE

Although the terms are often used interchangeably, disease and syndrome are different concepts. A disease is a destructive process in the body with a specific cause and characteristic symptoms, e.g., multiple sclerosis, coronary artery disease, pneumonia, hypertension, diabetes. A syndrome, on the other hand, is a concurrence of certain symptoms which together presume a destructive process in the body, e.g., Tourette's Syndrome, Premenstrual Syndrome, Irritable Bowel Syndrome (3, 4). As an extension, psychosocial syndromes presume a destructive psychosocial process.

It is true that medical conditions can generally be defined on different levels. Their definition may depend on structural pathology, etiology, deviation from some physiological norm, observable signs, or symptoms (5). Obviously, medical conditions can incorporate more than one level of definition. In general, however, when medical conditions are defined by structural pathology or known etiology, they gain certainty as a disease process. When they are defined instead by observable signs or symptom presentation, they lose certainty and only presume the presence of disease, i.e., syndrome. A condition's classification as disease or syndrome can change over time. Historically, some conditions known as syndromes have eventually become known as diseases once their specific causes were understood, e.g., Parkinson's Syndrome, now called Parkinson's Disease.

The emerging and overlapping syndromes of today properly fall outside the category of disease because they rely less on objective evidence and more on subjective observations and subjective symptoms. Their definition is uncertain. Yet, by attaching the label syndrome—a word with its roots in medicine—the condition seems to gain medical legitimacy.

Admittedly, grouping problems in terms of a syndrome has a practical value in establishing that something is amiss and needs correction, rather than waiting until medical science can trace the symptoms' origin or objectify them. In the psychosocial area, labeling behaviors as syndromes helps externalize them for easier modification. Behavioral techniques can then be used to address maladaptive psychosocial processes and extinguish them. Countless patients have regained control over their lives in this way, even when understanding the root cause of a syndrome has been elusive and the label itself artificial.

But, when syndromes are defined primarily by subjective characteristics, they are prone to vagueness, arbitrariness, overinclusiveness or underinclusiveness, and a variety of cultural influences. No doubt, this subjectivity has enabled the increased incidence of syndromes in personal injury litigation, workers' compensation, and disability disputes today.

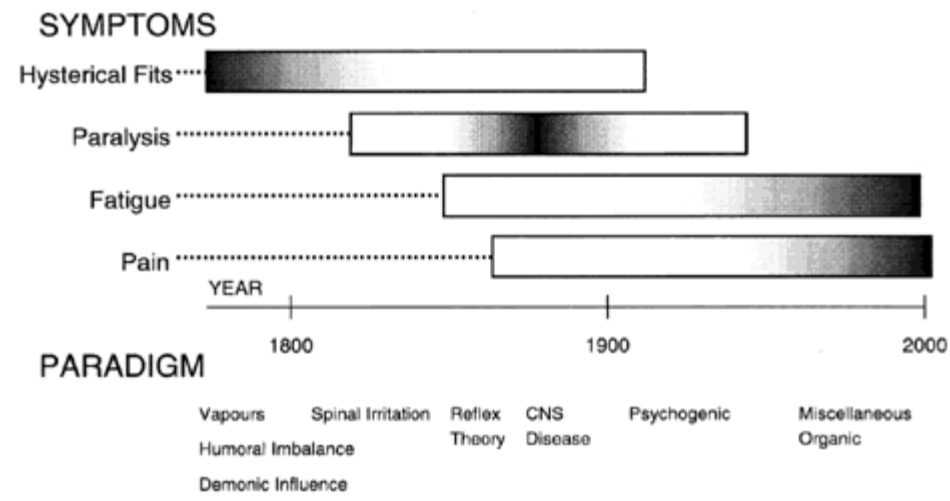
CULTURAL ISSUES

The history of medicine is replete with diseases and syndromes that have passed away with time. Many have passed because of an expanding body of medical knowledge, but some because of the powerful influence of culture. In psychiatry, there are examples of syndromes found only in certain societies. For example, Koro, a syndrome occurring primarily among Malaysians, involves a man's belief that his penis is shrinking and may gradually disappear into his abdomen, after which he will die (6). Piblokto occurs among Eskimos and is characterized by spells in which women scream and tear off their clothes while crying out like wild animals (7). Couvade is seen in some ancient and more primitive modern cultures when the husband of a woman who is giving birth experiences the pain of labor and delivery in excruciating intensity. These men have pain that is very real to them; culturally, the pain is a sign that they are the biological father (8).

Changing Syndromes in Western Society

Although these culturally influenced somatoform syndromes have occurred in more primitive societies, modern Western society is not without its own examples. Edward Shorter, in his book *From Paralysis to Fatigue*, traces psychosomatic illness in the modern era (eighteenth century and later). He concludes that the presentation of illness has varied in part according to what the culture deems legitimate (9). Over the past 200 years, he argues, the prevailing types of somatoform syndromes have changed in response to the prevailing medical paradigms of the time (see Figure 1).

Figure 1: Changing Somatoform Symptoms and Paradigms



The most common somatoform syndromes in the eighteenth and nineteenth centuries were labeled under the rubric of *motor hysteria*. They mostly consisted of hysterical fits characterized by uncontrollable shaking and/or different forms of paralysis. Paralysis could either be in the form of cataleptic fainting or regional paralysis of the body. Frequently, there was a mixed picture of both hysterical fit and paralysis. No doubt some of these problems were neurological conditions, i.e., choreiform movements, complex partial seizures, or narcolepsy. But, a substantial number were hysterical/conversion reactions.

Prior to the 1800s, the paradigms to explain such syndromes included vapours, humoral imbalance, or demonic influence. By the early 1800s, however, neuromuscular research identified the irritability of muscle fibers, leading to the theory of spinal irritation (9). Diagnosing spinal irritation involved finding a tender spot along the spinal column believed to be responsible for various peripheral symptoms, including distant pains, hysterical fits, blindness, dysphagia, menstrual difficulties, and many types of paralysis. Extensive treatment of the spinal irritation at the tender spot was universally followed, including cupping, blistering, and scalding the area.

As the 1800s progressed, further neurophysiological research identified the reflex arc in the spinal nervous system; this led to rampant attributions of medical maladies to *reflex theory* (9). Sensory irritation of nerves, most prominently in the pelvic area, was believed to result in motor abnormalities, such as the different forms of paralysis, rigidity, spasms, and convulsive movements. These reflexes were even believed to extend to the brain and cranial nerves, e.g., copiopia hysterica (meaning eye strain of uterine origin). Through reflex theory, every organ of the body was thought to be potentially influenced by every other organ.

With reflex theory, the incidents of paralysis grew to pandemic proportions through the late 1800s and into the early 1900s. Then, Charcot and others of his following changed the prevailing paradigm from reflex theory to nonspecific *central nervous system disease* (9). This new theory essentially held that exhausted cerebral centers were responsible for paralysis and other forms of motor hysteria. Only later, with the observations of Janet, Babinski, Freud, and others—as well as the recognition that suggestibility and hypnotic techniques could bring on or take away motor hysteria symptoms—*psychogenic theory* was introduced (9). With this theory's advent, the visible motor paralysis gained

an embarrassing psychological explanation. Consequently, symptoms that had once grown so dramatically began to decline.

Coincidentally, in the mid-1800s, George Beard adopted the concept of *exhausted cerebral centers* from central nervous system disease to explain sensory symptoms of fatigue, diffuse and persistent pain, neuralgia, insomnia, various sensory losses, and dyspepsia. He labeled the collection of these symptoms *neurasthenia* (9, 10). By 1900, the concept of neurasthenia was exported to Europe and described as an epidemic. Neurasthenia was the fashionable new disease.

Even after psychogenic theory became the paradigm to explain neurasthenia, the syndrome did not fall into disuse right away, but continued to be recognized in the psychiatric nomenclature into the 1960s (11). This was in part because sensory symptoms, unlike motor hysteria, were not as patently bizarre even when a psychological explanation for them was given. Furthermore, sensory symptoms of fatigue and pain were found in many other defined medical conditions so there was no automatic stigma to having them. By the end of the twentieth century, however, neurasthenia was dropped from psychiatric nomenclature (12) and miscellaneous organic syndromes were replaced as the modern paradigm for these same sensory complaints.

The shift from motor to sensory somatoform symptoms can be seen in the admission statistics for the past 100 years for psychiatric centers in Italy and Switzerland (see Table 1) (9, 13, 14). In the United States, the disappearance of motor hysteria was noted by Israel Wexler at Columbia University when he expressed that "hysterical paralysis has become a comparatively infrequent phenomenon...and astasia-abasia (hysterical inability to stand or walk) very rare." (9, 15). Meanwhile, by the end of the 1900s, fatigue and pain symptoms increased dramatically. It is estimated that there are presently 5 million Americans with undiagnosed Chronic Fatigue Syndrome, 400 local support groups for it, and 1,000 to 2,000 calls per month to The Center for Disease Control in Atlanta, Georgia, inquiring about the condition (9, 16).

Table 1: Psychiatric Admission Statistics in Europe for Hysterical Symptoms

YEAR	1900		1950	2000
	1898 - 1908	1910 - 1929	1938 - 1948	1970 - 1980
Psychiatric Hospital of Florence				
Hysteria	4 % (of all admissions)			0.1 %
Psychiatric Clinic of Lausanne				
Motor Hysteria	81 % (of all "hysterical" patients)			27 %
Fainting	47 %			31 %
Globus Hystericus	13 %			5 %
Trance-Like State	57 %			24 %
Amnesia	32 %			18 %
Fatigue	4 %			13 %
Psychogenic Visceral Symptoms	8 %			22 %

Similarly, the prevalence of pain—specifically, chronic pain—has increased dramatically. From 1971 to 1981, disability from back pain increased 168 percent (10). In headache complains alone, neurologists

have seen an eight-fold increase (17). Upper extremity pain is now the most prevalent disability in the industrial world (17). Chronic pain generally is thought to affect approximately 65 million Americans on an annual basis (19).

A Cultural Acceptance of Pain-Related Syndromes

In his recent work, *The Culture of Pain*, David Morris argues that the crisis of chronic pain in contemporary life is due in part to the failure of Western medicine to see pain as more than a sensation and recognize that bodily mechanisms are inextricably bound to the meaning that individuals and their culture give to pain (20). Pain is a mystery that is linked to more profound problems of life, suffering, and death. Modern science cannot answer the problem of pain without knowing the philosophical framework of the patient and the culture that influences that patient.

The culture's influence on disability claims for pain syndromes and other poorly defined complaints is dramatic. For example, repetitive motion injuries of the upper extremities once affected large numbers of Australian workers (up to 30 percent in some settings) until the diagnosis was no longer deemed legitimate and disability payments were curtailed (21). In Lithuania, it is theorized that the low incidence of persistent whiplash syndrome occurring after motor vehicle accidents is due in part to the fact that most drivers do not have personal injury insurance and, thus, the likelihood of disability compensation is remote (22). In Saskatchewan, Canada, a striking decline in whiplash injury and improved prognosis for the condition coincides with a change to the tort compensation system for traffic injuries in 1995, namely, no more payments for pain and suffering (23).

It is in the context of the United States' acceptance of pain-related syndromes through large jury awards in personal injury litigation, workers' compensation benefits, and disability time that the new variety of syndromes has developed in type and prevalence, creating the phenomena of Overlapping Somatoform Syndromes (OSS).

THE CORE SYMPTOMS OF OVERLAPPING SOMATOFORM SYNDROMES

Case Example

A 38-year-old man claims that as the result of accidental injury, he has been disabled for three-and-a-half years. He has been evaluated by numerous medical and surgical specialists who have not been able to find any objective medical pathology and have been unsuccessful in treating him. The man says that, if anything, he has been getting worse since the injury. His current treating physician reports that the patient is totally and permanently disabled, in part due to emotional distress because he is not getting better.

Among the man's most prominent symptoms are fatigue, chronic pain, headache, depression, anxiety, irritability, changeable mood, memory difficulties, poor concentration, confusion, feeling disorganized, word-finding problems, loss of task efficiency, dizziness, sleep disturbance, and nonspecific gastrointestinal distress.

From this hypothetical patient's symptoms, which is the most likely syndrome and its cause? Fibromyalgia or Myofascial Pain Syndrome from a motor vehicle accident involving whiplash? Complex Regional Pain Syndrome or Reflex Sympathetic Dystrophy (RSD) from a fall off of scaffolding, causing shoulder and arm injury? Mild Traumatic Brain Injury from a slip on the ice involving head trauma? Multiple Chemical Sensitivity Syndrome from exposure to carbon monoxide from a defective furnace? Gulf War Syndrome from military service in Operation Desert Storm? Chronic Fatigue Syndrome from overwork as an accountant?

In fact, the case example could fit all of these syndromes and more, including a number which have a notable minority following, e.g., Lyme Disease, Systemic Yeast Infection, Reactive Hypoglycemia. In the various syndromes that are claimed to be the result of personal injury, the symptoms presented in this case example are often the main complaints.

When medical pathology cannot adequately explain them, these syndromes may fall under the grouping of OSS. The core symptoms of OSS can be categorized as follows (see Table 2): fatigue, pain, emotional symptoms, cognitive impairment, variable sensory complaints, and other nonspecific symptoms. Of course, any one of these symptoms could represent a serious neuromuscular disorder, metabolic problem, or other medical condition that should be investigated. Together, they certainly seem alarming. But, by themselves, they do not *indicate* any particular illness. Whatever they could represent, they are too nonspecific to be of much diagnostic value. Once physical examination, laboratory testing, and other diagnostic studies fail to reveal objective medical pathology, few sound conclusions can be drawn from the symptoms alone.

Table 2. Core Symptoms in OSS

1. FATIGUE	5. VARIABLE SENSORY COMPLAINTS
Weakness	Visual Disturbance
Tiredness	Ringing in the Ears
Lack of Energy	Dizziness/Balance Problems
	Paresthesias
2. PAIN	
Muscular Aches	6. OTHER
or Pains	Sleep Disturbance
	Nonspecific GI/GU Distress
3. EMOTIONAL	Temperature Changes
Depression	Temperature Intolerance
Anxiety	Palpitations
Irritability / Anger	
Labile Mood	
4. COGNITIVE IMPAIRMENT	
Memory Difficulty	
Poor Attention and/or Concentration	
Confusion	
Feeling Disorganized	
Word Finding Problems	
Loss of Task Efficiency	

Because many of the core symptoms of OSS are claimed as evidence of traumatic brain injury, Lees-Haley and Brown studied their prevalence generally (24). They found a remarkably high percentage of the same symptoms in over 100 personal injury litigants who had not sustained a head injury, much less a traumatic brain injury (see Table 3). The Department of Defense found a similar symptom prevalence in Gulf War veterans (see Table 4) (25). A recent study from a large rheumatological center in Brazil found the same symptom prevalence in workers' seeking compensation for disabling

bilateral arm pain (see Table 5) (26). In these studies, subjective complaints of fatigue and pain were the most prevalent.

Table 3. Base Rate of Neuropsychological Symptoms In Personal Injury Litigants Without Head Injury

Anxiety	93 %
Sleep Disturbance	89 %
Depression	88 %
Headache	79 %
Fatigue	78 %
Decreased Concentration	77 %
Irritability	65 %
Disorganization	61 %
Confusion	59 %
Decreased Task Efficiency	56 %
Memory Problems	53 %
Dizziness	44 %
Word Finding Problems	34 %

Table 4. Complaint Prevalence In Gulf War Veterans

Joint Pain	49 %
Fatigue	47 %
Headache	39 %
Memory Loss	34 %
Sleep Disturbance	32 %
Rash	31 %
Difficulty Concentrating	27 %
Depression	23 %
Muscle Pain	21 %

Table 5. Pervasiveness of Symptoms Suffered By Workers Seeking Workers' Compensation For Disabling Bilateral Arm Pain

Fatigue	60 %
Sleep Disturbance	50 %
Dizziness	50 %
Nausea and GI Disturbance	40 %
Difficult Concentration	30 %
Memory Impairment	30 %
Anxiety	90 %
Depression	50 %
Headache	20 %

It can be argued that the core symptoms of OSS are merely a secondary psychological reaction to injury and, therefore, not surprisingly similar. But, in practice, the core symptoms are the primary signs of injury to the patient, who typically resists the implication that they are anything but. There is also usually little else but the core symptoms. Consequently, a circular logic sustains these syndromes: the symptoms indicate a reaction to an injury that consists only of the symptoms.

It can be argued that attributing psychogenic theory to these syndromes does not adequately take into account complex psychoneuroimmunological mechanisms or newly discovered physiological markers of stress and mental disorders. However, somatoform disorders do not need to be seen without physiological basis, only that the possible physiological substrates are subject to the vicissitudes of perception, motivation, secondary gain, reinforcement, and cultural influence. Moreover, the central modification and amplification of symptoms in these syndromes are more substantial reasons for their existence than the trauma on which the syndromes are allegedly based.

RECOGNIZING (AND EXCLUDING) OSS

Somatoform disorders in the psychiatric nomenclature are defined by the presence of physical symptoms that suggest a general medical condition, but are not fully explained by it (5). they include Somatization Disorder, Conversion Disorder, Pain Disorder, Hypochondriasis, Undifferentiated Somatoform Disorder, Body Dysmorphic Disorder, and Somatoform Disorder, NOS. OSS often consist of features of several of the subtypes, with Undifferentiated Somatoform Disorder being the most common classification. Because the hallmark of these disorders is the absence of a general medical condition to explain the symptoms, the clinician must exclude definable medical pathology. This needs to be accomplished by careful physical examination and diagnostic testing. Since the core symptoms for OSS are so ubiquitous, there are a significant number of patients who may seem to have OSS, but who actually have undiagnosed medical conditions. On the other hand, may of the modern emerging syndromes are actually OSS in disguise.

Fibromyalgia or Myofascial Pain Syndrome

Fibromyalgia, also known as fibrositis or fibromyositis, is a syndrome of generalized pain that is widespread throughout the body. If the pain is not widespread, then similar symptoms are often called Myofascial Pain Syndrome. The condition is chronic and typically has a poor prognosis in spite of aggressive treatment (27). Although recognized by the American Medical Association and the American College of Rheumatology, there is still considerable controversy about this condition and whether it actually exists as a distinct entity (28-31). Although the greater attention in Fibromyalgia and Myofascial Pain Syndrome is on muscular aches and pains, most of the core symptoms of OSS are also found, particularly fatigue.

Fibromyalgia has no known structural pathology, no known etiology, and no measurable deviation from a physiological norm. Observable signs on physical examination are said to include 11 of 18 tender (or trigger) points in specific body locations. At these points, evidence of a characteristic twitch or flinch and taut muscular bands may be found. If less than 11 tender points are located, the condition may fall under Myofascial Pain Syndrome. Unfortunately, the location of these tender points is not definitive and proponents say that the pain can be attributed to a location some distance away. Double-blind studies of experts looking for tender points have shown a large number of false positives, so there is low specificity for the diagnosis (29).

Fibromyalgia is more prevalent in countries where there are greater disability and insurance benefits or where there is higher cultural acceptance. For example, 11 percent of women in Norway, where Fibromyalgia is a readily accepted condition, meet the criteria (29). On the other hand, it is rarely seen in athletes, self-employed professionals, children, or the advanced elderly. Fibromyalgia is associated with higher rates of both mood and anxiety disorders, greater than that seen, for example, in rheumatoid arthritis patients (32, 33). Also, there is no consensus that Fibromyalgia originates from trauma (28). This should create some doubt whether Myofascial Pain Syndrome does either.

Chronic Regional Pain Syndrome or Reflex Sympathetic Dystrophy

Pain that spreads beyond the site of an original injury, usually to the extremities, and persists without evidence of structural damage is sometimes attributed to an abnormal sympathetic nervous system mediated response (34). Where there is direct damage to a peripheral nerve, this is known as *causalgia*. Where there is no direct nerve damage, it is known as *reflex sympathetic dystrophy* (RSD). Typical symptoms include exquisite pain even to light touch, edema of the skin and subcutaneous tissues, temperature and color changes, mottled or shiny skin, increased hair growth, and disturbed nail growth (34). Bone demineralization can also be seen at times on a bone scan of an affected extremity.

Sympathetic nerve blocks or sympathectomy can at times lead to dramatic relief, thus seeming to confirm the sympathetically mediated process. While there is ample evidence that RSD exists as a physiological phenomenon, many patients who claim to have it are suspected of a psychogenic disorder (35, 36). This suspicion arises particularly when pain spreads to other extremities (at times all four extremities) or when sympathetic nerve blocks have no effect. Many of these patients will also complain of the core symptoms seen in OSS.

Adding to the problem of what causes RSD is the fact that the disuse of an extremity over time can produce the same peripheral symptoms. Therefore, distinguishing which conditions are the result of a purely physiological response and which are the result of a poor motivation to recover can be difficult. In addition, even when sympathetic nerve blocks relieve symptoms, this can often be only temporary and due to a placebo effect, negating the inference that a sympathetically mediated process is occurring.

Adding to the complexity, there has recently been a reclassification of these disorders (34). The original RSD classification is now subsumed under *Chronic Regional Pain Syndrome I* (CRPS-I) and the original causalgia classification is now *Chronic Regional Pain Syndrome II* (CRPS-II). Of the new classifications, there are two subgroups, i.e., sympathetically *and* nonsympathetically mediated pain. While this reclassification was an attempt to create better definitions of these disorders, it has allowed conditions with no known physiological mechanisms to receive a legitimate medical label. Specifically, if CRPS-I or CRPS-II are nonsympathetically mediated, then there is no basis to link them to RSD or causalgia—or to draw inferences from those labels about the origin of the pain.

RSD and causalgia, as well as Fibromyalgia and Myofascial Pain Syndrome, are frequently referred to as *Chronic Pain Syndrome* (1). However, this is an even more nonspecific term, encompassing any number of other heterogeneous conditions, including Failed Back Syndrome, Repetitive Motion Syndrome, Occipital Neuralgia, Chronic Tendonitis, and others. Regardless of which diagnosis is being considered, Chronic Pain Syndrome frequently consists of the core symptoms seen in OSS.

Mild Traumatic Brain Injury

Claims of Mild Traumatic Brain Injury with persistent physical and mental symptoms are growing dramatically in this country (37). Often, the symptoms begin with a relatively mild direct head injury or an indirect jostling of the head in an accident. Although the effects of even Mild Traumatic Brain Injury can be devastating to an individual, the symptoms should eventually run their course. If the symptoms last longer than expected, however, concern should arise. This is especially the case when there has been no loss of consciousness or posttraumatic amnesia and the head injury itself has been minimal or nonexistent.

In many cases of Mild Traumatic Brain Injury, skull X-rays, EEG, CT and MRI scans are normal. The diagnosis is made on the basis of characteristic symptoms of Postconcussion Syndrome, with the continuation of some of the mental complaints said to be the residual effect of brain injury. But, most of the same symptoms that are attributed to Mild Traumatic Brain Injury are also seen with personal injury litigants who have not had any head injury (24). Therefore, those symptoms may be too nonspecific to be of value in making the diagnosis. Accompanying these symptoms are frequent pain symptoms of headache, neckache, or upper backache. Together, they also resemble the core symptoms of OSS.

In the absence of other diagnostic methods, neuropsychological testing is frequently used to identify cognitive impairment from Mild Traumatic Brain Injury. However, poor performance on these tests also occurs with depression, headache, chronic pain, fatigue, and preexisting attention problems. Severe stress alone has been shown to significantly—although reversibly—impair memory as a result of excess cortisol production (38). Thus, neuropsychological testing cannot make the diagnosis of Mild Traumatic Brain Injury, even though it is a valuable tool in assessing cognitive impairment and tracking the progress of the condition (37). Consistent with the limitations of neuropsychological testing is a statement by the American Academy of Neurology urging caution in attributing an etiology to any observed decrement in neural behavioral test performance, as these tests are extremely sensitive but not specific. No neuropsychological tests have been shown to have consistent diagnostic validity (39).

Multiple Chemical Sensitivity Syndrome

Multiple Chemical Sensitivity Syndrome (MCS) presents as an unusual and unexplained development of heightened sensitivity to environmental stimuli, manufactured products, chemicals, and so on, even when exposure is at a very low level (i.e., well within acceptable limits by most standardized measures) (40). In modern society, exposure to a variety of noxious agents is not unusual and can at

times have harmful consequences. With MCS, on the other hand, there has been a flurry of claims that cannot be objectively verified. This condition is also known as Environmental Illness, Environmental Somatization Syndrome, Total Allergy Syndrome, or Sick building Syndrome. Symptomatically, there are frequent claims of burning, watery eyes; irritated throat; itching and burning skin;' wheezing; and abnormal tastes and odors. However, the dominant complaints are in the form of the core symptoms of OSS.

Physical examination is often inconclusive. Challenge tests to the alleged toxic substance are usually not diagnostic, with placebo reactions quite common (41). Mild respiratory wheezing may be the only objective finding, although this has typically been used to support a diagnosis of occupational asthma in conjunction with MCS.

The results of inconclusive physical examination and challenge tests have led to the suspicion that MCS is a psychogenic or somatoform disorder (41-43). Interestingly, the syndrome is often "contagious" and/or culturally influenced. This can be seen when people in the same environmental vicinity or society as a whole suddenly experience the same symptoms to the same alleged toxic exposure (41). In several countries, for example, *electric hypersensitivity* increased dramatically when allegations of the visual toxic effect of video display units became publicized (44). However, there was no objective evidence of such an effect.

Gulf War Syndrome

A variant of MCS is a condition claimed by soldiers returning from the Desert Storm conflict in 1991. The reported symptoms had a strong similarity to Fibromyalgia and Chronic Fatigue Syndrome (45). Various possibilities for the syndrome, such as exposure to burning oil wells or an antidote to nerve gas (pyridostigmine bromide), have been considered, but not conclusively shown (25, 46). In fact, there has been a latency of onset from the time of the alleged exposure and a lack of association with the oil wells or the antidote from self-reported exposures (25).

The symptoms include eye and throat irritation, shortness of breath, wheezing, rashes, and joint pain. The core symptoms of OSS are typically present (47). Management guidelines for this spectrum of nonspecific symptoms resemble that for many "emerging overlap syndromes," i.e., working toward recovery in the absence of clear etiology (45).

Chronic Fatigue Syndrome

This nonspecific disorder, sometimes called Chronic Fatigue Immunodeficiency Syndrome or Myalgic Encephalomyelitis, is the prototype for conditions that define themselves by the core symptoms seen in OSS. It has increased so rampantly that a "hidden epidemic" has been claimed (48). The cause of Chronic Fatigue Syndrome is unknown and even proponents describe it as a heterogenous condition (49, 50). The role of viral infection (Epstein-Barr or Cytomegalovirus) has not been established and neither have the roles of allergy, dietary intolerance, poisoning, or hypoglycemia (51). Most scientific evidence points to a strong association with psychiatric disorders (52), In addition, diffuse pain frequently accompanies Chronic Fatigue Syndrome and many believe it is indistinguishable from Fibromyalgia (48, 49). As with most of the OSS, there is no known structural pathology, no known etiology, no deviation from a physiological norm, and no objective observable signs. The condition is entirely symptom-based.

Depression, Anxiety and Neurasthenia

Even without personal injury, depressive and anxiety disorders alone can include all of the core symptoms seen in OSS (5). Fatigue, muscular aches and pains, changeable mood, poor concentration,

difficulty thinking, memory problems, and vague physical disturbance are commonly seen in depressive disorders. Similarly, anxiety disorders can include a wide variety of sensory symptoms as well as the worry and preoccupation with health that is a feature of OSS.

Questioning patients about a psychological basis to their illness, however, frequently meets resistance. It is also not uncommon for patients to portray themselves psychologically in an overly favorable light in order to deflect the possibility of psychological factors (1). This can sometimes confound the diagnosis when nonpsychiatric clinicians are trying to rule out psychiatric disturbance. However, knowing the mechanism of *la belle indifference*, which occurs in Conversion Disorders (5), clinicians should not be surprised by the appearance of emotional well-being for OSS either.

The elimination of neurasthenia from the *Diagnostic and Statistical Manual of Mental Disorders* is unfortunate, since earlier psychological formulations of this condition have much to offer to our understanding of OSS. The symptoms of neurasthenia are almost identical with the core symptoms of OSS and, as discussed above, were once thought to be due to a general depletion of mental energy. Frequently, those with the condition were described as having difficulty expressing emotions or being psychologically unsophisticated. Not uncommonly, their personality style involved intense involvement with work, overexertion, and high degrees of responsibility. Their initial symptoms were often associated with anxiety and psychic depression (53). A review of the psychiatric history of patients with OSS shows similar characteristics in many cases.

THE NECESSITY OF A PSYCHIATRIC ASSESSMENT

Because the symptoms are so nonspecific in OSS, there is a high likelihood of false positives for any of the claimed conditions. The problem typically begins with the patient's need for attribution of his or her distress, especially in personal injury claims where disability is an issue. Once a patient attributes his or her symptoms to a particular traumatic event or injury, the history may be inadvertently shaped to fit that formulation, with other traumatic or troubling events dismissed. This history may then reinforce subsequent histories. Unfortunately, medical evaluations often fail to scrutinize the history beyond a search for superficial inconsistencies; thus, the evaluations can perpetuate the false data. This is not to imply that patients are necessarily fabricating the history or that the evaluators are not good clinicians. A mutually acceptable causation myth can easily be generated despite its inconsistency with the facts and become the sole basis for a diagnosis.

The problem in making the diagnosis correctly continues when a detailed psychiatric history is not taken, as is frequently the case in the absence of overt signs of depression, anxiety, or psychological disturbance. With the core symptoms of OSS, a psychiatric assessment involving the following points of focus needs to be done (see Table 6):

Table 6. Points of Focus in Assessment of OSS

1. Nature of Injury
2. Onset of Symptoms in Relation To Alleged Injury
3. Documented Objective Pathology
4. Progression of Symptoms in Natural Course
5. Psychiatric History
6. Objective Corroboration of 1. to 4.

1) Nature of injury: What is the nature of the claimed injury? What kind of accident or trauma occurred? Who saw it? What documentation exists? Are there police reports? Ambulance records? Emergency room notes? Employer statements? Is there any objective evidence of toxic exposure? If so, at what levels? Forensic psychiatrists should verify, whenever possible, the patient's description of the traumatic event. If this isn't possible, then psychiatric opinion about the effects of the traumatic event must be qualified.

2) Timing of symptoms to injury: Did the symptoms really arise following the alleged traumatic event or injury? Obviously without earlier medical records, it is impossible to judge whether some of the symptoms were preexisting. A patient's genuine assurance that he or she was feeling well until the injury may not be reliable. In countless cases, earlier medical records show core symptoms of OSS in one form or another stretching back many years. Did the symptoms really begin shortly after the injury (or at least a period of time that would be appropriate for that particular injury)? Complete medical and psychiatric records are important in order to ascertain this. Even when symptoms start much later, patients frequently forget this gap and mistakenly trace the symptoms to the time of the injury. Interestingly, family members can often join in this mistaken attribution, though gaining their perspective is still valuable.

3) Documented objective pathology: What is the documented objective medical pathology? Are physical findings transient and nonspecific? Are diagnostic tests equivocal? Do they show normal variants? What is the scientific literature about the proposed diagnostic findings and their relationship to the claimed disorder? Do the diagnostic studies for the condition have a significant percentage of false positives? Are the diagnostic methods supported within the scientific community? Because the jargon in OSS frequently has a pseudoscientific character, it can be confusing to anyone who is not specifically versed in the syndrome. As a consequence, clinicians often avoid closer analysis of the alleged syndrome or just give the patient and the other "expert" the benefit of the doubt.

4) Progression of symptoms: Did the symptoms progress in the natural course of the illness or injury? The natural course of most injuries is to heal. Certainly, some injuries leave lasting damage and will not heal. But, in personal injury litigation and workers' compensation disputes, it is surprising how often there is not only a lack of healing, but actually a worsening with time. Frequently, this is blamed on the psychological effects of the injury. However, the worsening is frequently not a secondary psychological reaction, but the core symptoms of OSS taking a dramatic and worsening course. This type of pattern should immediately raise a suspicion of OSS.

5) Psychiatric history: A thorough and detailed psychiatric history of the patient must be obtained. This is by far the most significant fault of most medical evaluators. They just do not know their patient. Without knowing a patient's life history, earlier psychiatric disturbances, dreams, failures,

defenses, reality testing, and conflicts during the time of the alleged injury, a clinician will always have an incomplete understanding of the cause and effect of that injury. It is not enough to simply record that the patient has had no previous psychiatric or psychological treatment. A patient's history may demonstrate that the *conditions* for OSS were there all along, with the injury serving merely as an opportunity for their expression.

KNOWING OUR LIMITATIONS

Karl Jaspers stated that "disease is always a biographical enterprise" (54). Nowhere is that more true than in the evaluation of OSS. If a psychiatric assessment with the above points of focus is not possible—as it may not be for the practicing clinician—there should at least be an appreciation of the limits of any subsequent medical opinion. It would be more scientifically precise to say, "Based on what the patient has told me and available records, it is my opinion that...", rather than blindly—and perhaps later embarrassingly—assume that an evaluation has been complete and conclusive. The diagnosis, treatment, and rewards of OSS in the context of personal injury litigation, workers' compensation, and disability claims are a major challenge facing medicine today. As medicine becomes more and more sophisticated, clinicians cannot forget the strong influence of culture on the prevalence of certain conditions or the possibility that false syndromes can be perpetuated through an erroneous collaboration between patient and physician, both looking to find a simple explanation to complex biopsychosocial problems. If we do forget these things, we will find that we have not progressed beyond the spurious and often comical diagnoses and treatments of centuries ago.

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