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THE DETECTION OF EXAGGERATED SENSORY SYMPTOMS

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A well-known study of the early 1980s posited that over $20 billion were spent in the investigation and treatment of symptoms that appeared to be without a specific identified disease, a dollar amount that has surely risen over time.[5] It is known that individuals with extensive medical utilization also often have high lifetime prevalence of co-morbid psychiatric disorders, e.g., depression, anxiety, somatization, and other somatoform disorders including conversion.[9] Thus, while rates vary according to setting and criteria, it has been estimated that between .2 and 20 percent of medical patients evidence symptoms that are either exaggerated or have a psychiatric etiology.[26] While some argue that the rate of misdiagnosis has decreased over time through the use of improved technology (e.g., MRI to diagnose multiple sclerosis),[18] there still remains a significant chance of encountering these patients in everyday practice.

The *Diagnostic and Statistical Manual of Psychiatric Disorders* (DSM-IV[1]) defines a group of somatoform disorders that include somatization, conversion, and pain disorders, along with hypochondriasis. The common features of these disorders are the unconscious physical expression of underlying psychologic distress. Factitious disorders, on the other hand, represent a conscious effort to create symptoms to obtain the "sick role," while malingering implies the above symptoms to attain an external benefit—compensation, disability, etc. The presence of concurrent psychiatric conditions such as depression or a personality disorder (along with low education and recent stressful life events) has been

implicated as a risk factor for developing a conversion syndrome.[4] Rather than an "all or none" phenomenon, researchers have also suggested that there can also be varying degrees of symptom exaggeration in the above-noted spectrum of conversion and factitious disorders.[16]

Psychiatric conditions have also been observed and documented in the practice of rehabilitation. While motor symptoms are frequently the focus of restorative treatment, cases have been reported of "feigned" sensory disorders either combined with or in the absence of weakness. Some common sensory complaints include blindness, anosmia, paresthesia, anesthesia, akinesia, dyskinesia, and tunnel vision.[18]

Determining the validity of a sensory complaint begins with the physical examination. Psychogenic conditions can have features in their presentation that are not usually encountered in daily practice, which triggers one to focus the physical exam more closely on specific symptoms. For instance, patterns of complaints may be inconsistent, nonanatomic, or at times dramatic.[18] A thorough sensory examination may reveal split temperature and pain findings, despite their pathways traveling together in the periphery and spinothalamic tracts. Pinprick sensation may abruptly change in the midline of the forehead, which is inconsistent with dermatomal patterns and sensory nerve distribution. Vibration loss may be seen over half of the face, jaw, sternum, or spine, but in reality vibration spreads across bony structures. Additionally, loss of facial sensation without scalp deficits is inconsistent with the anatomic distribution of the trigeminal nerve. True sensory deficits usually preclude functioning with closed eyes due to lack of tactile and proprioceptive feedback. Thus, a Romberg sign is present when there is loss of balance while standing erect with feet together and eyes closed, which is usually absent with psychogenic pathology.

Tone and reflexes are objective findings that are helpful in delineating nonorganic pathology from true sensory deficits. Upper motor neuron pathology may present with hyperreflexia, increased tone, and abnormal reflexes such as Babinski, Hoffmann, and clonus. Peripheral neuropathy may present with hyporeflexia and decreased tone in conjunction with lower motor neuron pathology. Conversely, tone and reflexes will be normal with psychogenic pathology.

Some provocative tests that can be used include Mankopf's maneuver, which may reveal an increased heart rate with painful stimulation (although our experience with this procedure has been mixed). Magnuson's test consists of having the patient point to an area of pain or sensory loss repeatedly over a period of time. Inconsistencies in reporting are suspicious for nonorganic lesions. A temporal delay to a nociceptive stimulus may also raise suspicions. Additional revealing tests include assessment of sensation with a limb in supination/pronation or internal/external rotation to again check for inconsistencies. Sensory testing can be done with the patient watching in a mirror, which can confuse a patient who is being disingenuous. Classic subjective symptoms, such as "la belle indifference" whereby the patient seems unconcerned despite alleged significant neurological and functional deficits, have often been considered pathognomonic signs of deception.[24] The use of this as a marker of symptom exaggeration must be tempered, however, by the recognition that a lack of prosody and concern (anosdiaphoria) can be symptoms of legitimate neurological dysfunction. In addition, the research on the utility of such classic psychiatric signs of conversion has been mixed at best.[6,14]

DETECTION IN MORE DIFFICULT CASES

Despite the knowledge of neuroanatomy and neurophysiology presented above, diagnosis in some cases is difficult, as both "real" and "exaggerated" symptoms can co-exist. For instance, it is well established that a sizable proportion of individuals diagnosed with epilepsy will also display pseudoseizures.[12] Similarly, cases will occasionally present with actual motor or sensory loss due to a neurological disease, yet the symptoms may seem greater than expected given the known pathology. In addition, violation of the above-noted laws of neuroanatomy can periodically be seen due to CNS dysfunction.

Thus, while the physical examination is the first step in determining the validity of symptoms, the physician may, at times, need to resort to additional diagnostic techniques. For example, electrodiagnostic studies can effectively assess the peripheral nervous system. Normal sensory nerve conduction studies usually preclude a peripheral (neuropathy) etiology to sensory deficits, which allow one to focus on the central nervous system as a possible site of pathology. Factors such as advanced age, limb edema, and obesity can reduce the validity of nerve conduction studies.[25] CNS assessment is accomplished through neurologic imaging consisting of CT or MRI of the brain, and will reveal most intracranial lesions. Normal imaging may support inconsistencies found on physical examination. Recent advances in functional neuroimaging (PET, SPECT, fMRI) also suggest a potential for use in diagnosing and understanding these conditions.

In addition, needle electromyography may differentiate between authentic weakness and poor patient effort. Reduced activation of motor unit potentials (MUP) may be due to true weakness, pain, or poor effort. By examining the frequency of MUP firing, one can identify a poor effort by a patient who is feigning illness. True weakness is often accompanied by a rapid MUP firing frequency; thus with normal firing rates unaccompanied by pain, psychogenic pathology may be considered. Somatosensory evoked potentials (SSEPs) may detect sensory lesions in the periphery and assess sensory transmission through the spinal cord and brain.[13]

FORCED-CHOICE TESTING

Forced-choice testing, described in detail elsewhere in this volume, is a long recognized technique for the detection of feigned or exaggerated memory and sensory deficits.[10,11] For sensory disorders, the clinician identifies a stimulus that is reproducible and relates to the patient's perceived deficit. While patients may protest that they cannot perceive the stimulus, we have often framed their sensory perception as similar to "blindsight," where an individual is cortically blind yet can still perceive some visual stimuli due to intact subcortical structures. Thus, we instruct the patient that while sensation is not consciously processed early in recovery, it is being perceived at some level of brain functioning. Performance at statistically significant below-chance levels on this two-alternative, forced-choice procedure, as calculated by the z formula, raises suspicions of symptom exaggeration/decreased motivation. Favorable detection rates have been reported in numerous studies.[2,3]

For sensory examinations, forced-choice tests of finger agnosia and fingertip graphathesia are included in a standard sensory-perceptual examination. Without visual feedback, a subject is asked to perform two repeated-trial tasks: (1) to identify which finger of one hand has been touched; and (2) to identify four numbers that are written on alternating fingers of each hand. Standard testing protocol involves 40 (20 per hand) presentations for finger agnosia and fingertip number writing testing.[15] Extended forms of the finger graphathesia procedure (using only two number choices) have been utilized to detect possible somatosensory exaggeration.[2,9,11] Even when an extended form of the procedure is not utilized, z formulas have been calculated based on the standard number (40) of administrations of the sensory perceptual

examination in order to aid with the detection of lessened effort.[17] By utilizing these methods, the clinician can ascertain the odds by which the results are due to symptom exaggeration or decreased motivation, a number that is often valuable in litigious situations. Still, forced-choice methods are inherently limited in that a lack of a clear finding on these tests (e.g., at chance-level performance) significantly reduces the conclusions about patient motivation that can be rendered. In addition, the technique is well described in the scientific literature and thus could be discovered and manipulated by savvy patients.

TREATMENT

Once identified, the literature regarding the treatment of sensory or any exaggerated physical symptom varies. Standard clinical practice advises that the symptoms be addressed in some active manner, either through psychological or physical treatment programs. Oftentimes both of these modalities are utilized in an integrated rehabilitation milieu. The idea of not confronting the patient's symptoms as of psychiatric origin is not always followed, and successful cases have been reported whereby the patients are "confronted," or at least told that the results of their tests suggest a psychiatric origin to their problem. The patients thereupon "flee into health," or not wanting to face further embarrassment, give up their symptoms and leave the clinic or hospital.[13,19] Certainly, numerous case studies exist regarding the enrollment of these individuals (with primarily left-sided deficits) in active rehabilitation programs.[18–20,22] The nature of the treatment ranges from typical rehabilitative care to one based on numerous behavioral interventions that encourages the patient to give up the symptoms and the sick role. Education regarding the influence of "stress," anxiety, and depression on physical functioning can be utilized in order to allow the patient to consider that the symptoms may be caused by something other than physical malfunctioning. For more refractory cases, the "double bind" has been utilized whereby the patient is told that unless the symptoms resolve, their origin must be considered psychiatric.[20] Thus, not wanting to be labeled as a "psychiatric case," the patient improves.

PROBLEMS WITH THE PSYCHIATRIC MODEL

The difficulties with typical psychiatric explanations of conversion spectrum disorders are that in practice it is often difficult to: (a) ascribe an underlying motive for the appearance of a symptom, (b) find a recent life event or stressor that appears to precede the conversion episode, or (c) readily identify the secondary gain derived from the symptom. Certainly, we have seen numerous cases in practice that, despite in-depth investigation, did not appear to fit the traditional psychiatric pattern or explanation of conversion. Thus, as discussed in several chapters in this volume, there has been a recent trend toward a more cognitive neuroscience model of symptom exaggeration and maintenance. This change in philosophy is partly driven by the recognition that a percentage of those diagnosed with a conversion spectrum disorder eventually go on to develop a diagnosable physical disease.[23] The advent of functional neuroimaging is also furthering our understanding of conversion symptomatology. Two recent reports have investigated activation patterns in brain areas with known relations to hypnotic states, inhibitory motor mechanisms, and sensation.[7,21] Thus, neurobiological explanations for the co-occurrence of motor and sensory symptoms (involvement of the basal ganglia and thalamus) and the frequent involvement of left-sided deficits (right hemisphere locus of activation) are being offered.[12] Still, functional neuroimaging studies do not solve the question of whether

the symptom (and brain activation area observed) is in reaction to the psychologic conflict or is the cause of the observed deficit.

CONCLUSIONS

The majority of individuals presenting with unexplained sensory disorders can usually be diagnosed on the bases of a thorough history and physical examination. Functional deficits almost always defy known laws of neuroanatomy. In addition, there is a wide variety of physical testing that can be performed to detect inconsistencies in presentation that strengthens the certainty of a non-organic pathology. More difficult cases are occasionally seen, whereby the testing suggested above is inconclusive or, more likely, the symptoms are seen in conjunction with a concurrent diagnosed disease state that could explain some or all of the inconsistencies. When such cases arise, more sophisticated measures of physical status (e.g., neuroimaging, SSEP, EMG) or statistical methods of consistency/effort (symptom validity testing) must be utilized to refine the diagnosis. Still, owing to inherent limitations of the diagnostic measures employed, there may be a few cases that cannot be definitively diagnosed. Usually, the weight of the evidence in such cases suggests a psychiatric origin to the symptoms and these patients are subsequently enrolled in a rehabilitation program.

The success of various physical and psychological treatments of conversion symptoms in a rehabilitation setting is well documented. Utilizing a psychiatric model of causation suggests that such treatment does not address the underlying cause of the symptom production and thus would likely reoccur with new stressors. Little research to support this notion is present in the literature, although the psychiatric explanation of conversion symptoms is currently being challenged by a more neurologically and biologically based conceptualization. Thus, oftentimes the methods described in this article will elucidate the degree of symptom exaggeration but not the cause. Truly, this is a nascent area of research whose results could change the diagnoses and treatment of conversion-based sensory disorders in rehabilitative practice.

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SYMPTOM VALIDITY ASSESSMENT IN THE REHABILITATION SETTING

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It should come as no surprise to health care providers that patients are occasionally dishonest, or perhaps simply inaccurate, when describing their emotional, cognitive, and somatic ailments. Indeed, readers may recall a time in their own past when they "played sick" or exaggerated their ills to avoid unpleasant or undesirable responsibilities, or to garner sympathy from an employer or family member. Health professionals are by necessity overly reliant on patient self-report, which unfortunately can lead to deleterious consequences such as misdiagnosis, misapplied treatment, and diminished outcome. We tend to make clinical judgments about the validity of patient self-report and behaviors, which to the casual observer probably seem to be a rather crude litmus test of patient honesty. Even when there has been a strong suspicion of invalidity, untoward effects are possible when the clinician has relied solely on patient self-report to the exclusion of corroborative information or objective tests.

Inaccuracies may range from minor distortions of occupational or academic performance[39] to gross misrepresentation of somatic ills.[14] Inaccurate self-report has been consistently shown to be more frequent among patients with certain psychiatric disorders compared with community-dwelling controls. For example, schizophrenia has been strongly associated with fabrication of information.[61,76] Patients with post-traumatic stress disorder[37] and alcohol abuse[60,74] have been shown to be less accurate in describing the history of their symptoms. Different forms of neurological illness, which admittedly overlap with many psychiatric conditions, may predispose patients to inaccurately report pertinent information.[38,64] Even