PHYSICAL MEDICINE AND REHABILITATION: STATE OF THE ART REVIEWS (PM&R: STARS)

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WHAT IS THE TRUE SPECTRUM OF FUNCTIONAL DISORDERS IN REHABILITATION?

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INTRODUCTION: FUNCTIONAL DISORDERS IN REHABILITATION

"Why won't my patient get better?" In rehab settings, this oft-heard plaint is commonly applied to patients whose objective injuries fail to explain the range or severity of functional disability that prevents them from returning to their previous work and lifestyle. Many such patients are experiencing *traumatic disability syndromes*,[60] such as postconcussion syndrome, chronic pain, and posttraumatic stress disorder, and a substantial proportion of these patients eventually have contact with the legal system.[5,92] In addition to their direct injury-related disabilties, these patients may display other types of psychological disturbances that may be variously described in clinical reports as "psychosomatic," "hysterical," or "malingering," especially in the context of adversarial forensic evaluations.

Confusion reigns. Orthopedists make ill-conceived psychiatric diagnoses, branding patients as nut cases or cheats, while mental health clinicians lack clear guidelines on how to treat these patients in the practical, here-and-now, results-oriented setting of the rehab clinic. Unfortunately, many orthopedists, neurologists, physiatrists, and other rehab clinicians tend to see things in either-or terms: either the patient has a "real" disability, or he's making it up. At the same time, the legal and insurance system's frequent need for cut-and-dried determinations of disability, liability, responsibility, intent, and percentage of impairment can put case managers and attorneys at odds with the comparatively untidy diagnostic world of

psychological disorders, personality traits, neuropsychological impairment patterns, and disability syndromes.[61,62]

"The first step to making a diagnosis is to think of it."[93] Accordingly, this chapter describes the major classes of functional psychological syndromes seen in common rehabilitation practice, based on a selective review of the literature, and my own experience in the trenches of daily rehab practice. The intent is to help all those who deal with traumatically disabled patients—doctors, therapists, case managers, and attorneys—understand the main classes of diagnostic categories that may complicate their cases.

Psychological disturbances can constitute the primary focus of a rehab case (e.g., postinjury depression or substance abuse), or they can create confusion in a case based primarily on straightforward physical injury (e.g., persistent chronic pain or cognitive deficits after a "minor" injury). Indeed, many of these syndromes are characterized by a longstanding, exacerbating, and remitting pattern of psychological impairment; however, their expression may be elicited or intensified by the psychological stress that attends an accident or other injury. Finally, a psychological component to disability does not by any means imply untreatability; on the contrary, adequate case conceptualization and appropriate treatment of psychiatric disorders and psychodynamic issues in the rehab setting can often make the difference between a productive recovery and a "chronic case."[60]

SOMATOFORM DISORDERS

The common feature of the *somatoform disorders* is the presence of subjective physical symptoms that suggest a medical illness or syndrome, but are not fully explainable by, or attributable to, a general medical condition, substance abuse, or other type of mental disorder.[4] Needless to say, a thorough medical workup is necessary to rule out actual physical illness, and there is no clinical rule that prohibits somatizing patients from developing real illnesses or sustaining real injuries. Indeed, in rehabilitation settings, a documented injury frequently becomes the nidus for one or more somatoform disorders. In the current classification of DSM-IV,[4] somatoform disorders include several subtypes. Although these tend to be relatively consistent from patient to patient, it should be recognized that patients may show more than one subtype, a combination of subtypes, or alternation between several subtypes as a continuum of coping style.[96]

Somatization Disorder

Somatization disorder, formerly referred to as "Briquet's syndrome" or "hysteria," involves a history of multiple unexplained physical symptoms and complaints, beginning before age 30, and often traced to childhood and adolescence. Outbreaks of numerous and varied symptoms may occur in clusters that wax and wane over time, often in response to interpersonal, vocational, and other stressors. Associated features include anxiety, depression, impulsivity, relationship problems, psychosocial discord, and possibly substance abuse. Symptoms in somatization disorder may closely mimic standard traumatic disability syndromes, or they may be atypical or frankly bizarre in quality, location, or duration. The patients typically describe their symptoms in exaggerated, florid terms, and several physicians may be consulted concurrently, leading to secondary problems associated with medication abuse and unnecessary surgical treatment. Rehab clinicians who carefully review medical records will typically be impressed by the sheer number and variety of past injuries, illnesses, and unexplained symptoms, covering a wide range of organ systems and medical diagnoses.

From the point of view of rehabilitation, examiners should determine whether the present symptoms that are claimed to be related to the injury actually fall into a prior longstanding pattern and history of multiple symptoms and complaints. Intention in "pure" somatization disorder is typically unconscious: the patient genuinely believes that he or she is ill or impaired. The underlying motivation is frequently inferred to be a quest for support and reassurance, or manipulation of the affection of a significant other. The psychodynamic goal is the satisfaction of dependency needs by reliance on caretakers or on the protective role of medical authority.

Case Example. A 53-year-old female office manager had undergone numerous diagnostic tests and treatments for headaches, dizziness, anxiety attacks, and gastrointestinal symptoms since her early 20s. Over the years, she frequently voiced fears of having a brain tumor or a "silent stroke," despite the lack of medical evidence. Two years ago, she sustained a mild closed head injury in an auto accident. Since then, her headaches and dizziness have worsened, accompanied by hypersensitivity to light and noise, tinnitus, anxiety attacks, and severe "forgetfulness." Brain MRI and EEG are within normal limits, and neuropsychological testing shows mild, equivocal findings. She is now concerned about permanent brain damage and continues to seek consultations. In addition, she has developed cardiac palpitations and intestinal cramps.

Conversion Disorder

The essential feature of *conversion disorder* is the presence of sensory or motor deficits that appear to suggest a neurological or medical illness or injury. In conversion disorder, intention is almost always unconscious, the patient unshakably convinced of the impairment. The underlying motivation typically involves the attempted resolution of psychological conflicts, such as dependency wishes, by channeling them into physical impairment. Alternatively, there may be an actual symbolic "conversion" of a particular psychological conflict into a representative somatic expression, as in pseudoparesis of an arm in a patient who fears acting on a hostile impulse or severe, incapacitating lumbosacral pain in a worker who believes that "I was a *stand-up guy*, but my company didn't *back me up* when I needed them." Exacerbations are typically precipitated by psychosocial stresses related to job or family, including the stress of the accident itself, with resulting financial and legal hassles. A common psychodynamic force in traumatic disability cases seen in rehab settings is a combination of anger and derailment of overcompensated striving that had previously been utilized as a defense against unconscious dependency wishes.[17,32]

Typical conversion symptoms in the rehab setting include sensory impairment (visual, auditory, somesthetic), motor impairment (weakness, poor coordination, "falling spells"), genitourinary and sexual dysfunction putatively related to lumbosacral spinal cord injury, or impaired memory and pseudoseizures in postconcussion cases, as well as intractable chronic pain (see below). There is often a nonphysiologic pattern to the symptoms, which correspond to the patient's notion of impairment (especially in medically unsophisticated subjects), rather than neuromedical reality—although in this age of ubiquitous medical newsletters and websites, patients may be quite accurate in their replications of organic disability. Unlike the anxious, agitated, angry, or depressed emotional state of many injured patients, conversion patients may display "la belle indifference," a bland, almost nonchalant demeanor that seems to suggest that the conviction of physical impairment is of little concern—and indeed, actually strangely reassuring—to the patient, despite his or her protests of catastrophic ruin.

The question of the actual nervous system changes involved in conversion disorders is fascinating and controversial (see references 32, 33, 38, 55, 97 for reviews), raising the question of what is really meant by a "functional disorder." In many cases, there does appear to be some measureable functional disturbance in the nervous system's processing of sensorimotor information that underlies the symptom (see reference 97 for a recent example of research in this area). Secondary changes, such as disuse atrophy, may result from prolonged immobility of a limb or body part.

Case Example. A 36-year-old male construction foreman sustained a low back injury at work, resulting in a mildly herniated lumbar disk and some soft tissue sprain-strain injury. He had always prided himself on being a very hard-working, independent, and capable man, holding two jobs and supporting his wife and children, plus his wife's mother. He was now told that his injury would necessitate his going on light-duty work with a minimal decrease in pay but a substantial loss of job status. Over the course of several weeks, the low back pain worsened and was accompanied by progressive weakness and numbness in his legs. Eventually, his right leg "gave out," and he now walks with crutches. He is convinced of his total incapacity for work and is claiming total permanent disability, despite the lack of medical evidence of serious injury, and even in the face of his insurance company's threatened termination of benefits because of his noncompliance with treatment. When queried, he exudes an eerie calm and says, "This is what they've done to me, but I've learned to accept my fate."

Pain Disorder

As a new somatoform category in DSM-IV,[4] previously classified as a subspecies of somatization disorder,[2,3] the essential feature of *pain disorder* is chronic pain that causes significant distress or impairment in social, occupational, or other important areas of functioning, and in which psychological factors are judged to play a significant role in the onset, severity, exacerbation, or maintenance of the pain. The pain is not intentionally produced or feigned as in malingering or factitious disorder (see below), but rather expresses, represents, or disguises an unconscious need, fear, or conflict, closer to somatization disorder. In addition, pain caused by documented physical injury can be exacerbated by real-life external stressors, setting up a vicious cycle, which may partly explain the etiology of such syndromes as fibromyalgia.[9] In many cases, no "legitimate" medical explanation for the degree of severity and/or length of persistence of the pain can be discovered, and patients make the rounds from clinician to clinician until they are eventually shunted into the mental health system or rejected outright from further treatment.

In many cases, the chronic pain syndrome has a characteristic evolution and course.[22] The problem typically begins with some accident or injury that causes an expectable degree of acute pain requiring medical treatment. In a certain proportion of these patients, the pain and disability never seem to get better, and in fact, are reported by the patient to worsen with time. Various medical strategies are tried by the treatment team, but nothing seems to work. Excessive physical disability related to sleep and appetite disturbance complicates the picture and is often exacerbated by the side effects of excessive and varied medication.

The patient's ongoing struggle with continual pain results in depression, obsessive somatic preoccupation, and a tendency to increasingly conceptualize most life events, activities, and problems solely in terms of how much pain it will cause, leading to a vicious cycle of hopelessness, helplessness, and despair. Each new treatment or physician may briefly inspire hope, which is then dashed by disappointment when the procedure fails to cure or significantly relieve the pain. Resentment and bitterness grow toward the medical profession, and this antipathy is reciprocated, as doctors come to dread visits by the "crock."

Pain now becomes the central focus of the patient's life. The patient progressively withdraws from family and social activities, and interpersonal interactions are fraught with tension and anger. Sometimes the patient develops a symbiotic alliance with a close family member or sympathetic clinician who becomes the patient's advocate and champion, further fueling the sense of victimhood. Attorneys are almost inevitably involved, entrenching an adversarial relationship among patient, family, doctors, and insurance company. Problems with medication and with alcohol and drug abuse may compound the problem by producing toxicity and addiction. Pain behavior becomes a major coping mechanism, progressively allowing the patient to avoid any kind of stressful task or issue. The patient now claims total disability, but the insurance company keeps sending him for more "independent medical opinions" in their efforts to settle the case. After considerable passage of time, even the patient's own doctors and attorneys begin to lose patience and subtly or overtly urge the patient to "give it up." In the worst situations, this leads to further incapacitation and the inexorable decline toward total invalidism.

How patients think about and conceptualize their pain and its implications for their future may be an important factor influencing response to treatment and long-term outcome. Cognitive distortion is a factor that can have important emotional and behavioral effects.[23] Chronic low back pain patients are prone to "cognitive errors," such as catastrophizing, overgeneralization, personalization, and selective abstraction, which are associated with depression.[24-26] The majority of chronic pain patients overestimate their baseline pain when asked to recall it following treatment,[27] and subjects' recall of the reactive and emotional quality of their pain appears to be particularly subject to distortion.[29,84]

Hypochondriasis

The conviction that one has a serious illness or injury, in the face of numerous medical pronouncements to the contrary, is the defining characteristic of *hypochondriasis*. Patients are preoccupied with the fear of pathology, injury, disease, or deterioration, and tend to misinterpret normal bodily signals as signs of dire illness or injury.[4] Unlike the varied clinical presentations of somatization disorder, hypochondriacs tend to focus on one or a few chosen symptoms and remain preoccupied with them, although the focus may shift over time from one symptom or disorder to another—e.g., from memory impairment, to dizziness, to headaches, to back pain—and the associated anxiety may wax and wane over time. Unlike conversion disorder, there may be no actual observed or experienced impairment per se: it the *fear* of insidious damage that is the problem. Common rehab examples include fear of "stroking out" after a head injury, fear of further injury and becoming a "cripple" upon return to work following a knee or shoulder injury, and fear of contamination and genetic damage after toxic exposure.[44,49]

The unconscious motivation in hypochodriasis typically involves a deflection of anxiety away from issues of broader psychosocial concern, such as career or relationships, with a focus on a more delimited, and hence "controllable" source of concern in the form of somatic symptoms and fear of further injury. These tortured souls search endlessly for the one enlightened medical expert or miracle diagnostic technique that will either conclusively validate or rule out their worst fears. Yet with each reassurance, more fears arise.

Case Example. A 27-year-old male warehouse worker was exposed to fumes from an accidental chemical spill at work. He subsequently became preoccupied with subjective disturbances in breathing and underwent many tests and consulted numerous cardiologists and pulmonary specialists. Shortness of breath, painful inhalation, gasping and wheezing, hyperventilation, and other symptoms were described, all focused on the respiratory system, and all believed to have been caused by exposure to the putatively toxic gases from the spilled chemicals, despite the fact that no medical abnormalities could be determined. Clinical history revealed that at age 11, he witnessed his grandfather gravely ill with congestive heart failure, and he had subsequently become obsessed with "heart problems," spending a good portion of his adolescence and early adulthood fearing sudden cardiac death and undergoing many unrevealing cardiologic workups. The preoccupation with respiratory symptoms following his exposure to fumes appears to have been one form of extension and redirection of those fears.

Body Dysmorphic Disorder

In rehab settings, *body dysmorphic disorder* may occur where the injury has resulted in some degree of disfigurement or loss of function that impacts on the patient's self-image. Diagnostically, this disorder involves a preoccupation with an imagined defect in appearance, or overconcern with a minor defect. This may include facial scars or asymmetry, lost athletic prowess, reduced work capacity, or weight changes due to immobility after an orthopedic injury. Alternatively, it may present itself as a form of *cognitive dysmorphic disorder* associated with the postconcussion syndrome following a traumatic brain injury,[45,46,63,65,67,72,74] in which diminished intellectual skills, interpersonal functioning, or employment status is the main source of self-deprecation. Unconsciously, the motivation for such preoccupation with self-perceived ugliness or worthlessness may involve deep-seated and longstanding feelings of self-loathing that are now, postinjury, projected onto a more objectifiable physical or mental impairment that serves as the (new) focus of "badness."

Case Example. A 40-year-old female high school teacher in the midst of a contentious divorce was assaulted by a student who hit her in the face with a canvas backpack. She was momentarily dazed and sustained a mild cervical sprain-strain injury, which subsequently resolved. However, she also sustained several scratches and bruises on her face, which, although healed and virtually invisible to the close inspection of doctors and friends alike, continued to plague the patient each time she looked in the mirror. In addition, she became convinced that the neck injury had caused her head to tilt at an ugly angle and that she now "looked like a gimp." She was about to undergo cosmetic surgery, but the plastic surgeon hesitated when he reviewed the history and subsequently requested a psychiatric consultation.

FACTITIOUS DISORDER

Factitious disorder is defined as the deliberate production, manipulation, or feigning of physical or psychological signs and symptoms to satisfy psychological needs rather than for material gain.[4] Because the intentionality of symptom production is conscious and deliberate, it is diagnostically separated from the somatoform disorders. However, unlike malingering (see below), where a utilitarian motive for the deception can usually be discerned, the motive in factitious disorder is primarily to assume the sick role, with all the attendant care, solicitous concern, and relief from responsibilities of normal life that this entails, even at the price of substantial

cost in money, health, or freedom—i.e., the motive would be viewed by most people as "senseless" in terms of significant practical gain. In many cases, there also appears to be great satisfaction, perhaps only partly unconscious, derived from manipulating the medical system and "fooling the experts."

Historically referred to as *Munchausen's syndrome*, the manifestations of factitious disorder are limited only by the imagination and ingenuity of these patients. Medically sophisticated patients, such as nurses or mental health clinicians, may be quite clever in feigning credible medical and psychiatric illnesses and impairments by the surreptitious use of chemical substances or medical apparatus, or by displaying realistic postconcussive, depressive, or PTSD symptoms. Less knowledgeable patients may resort to cruder methods such as drinking toxic concoctions, bruising or cutting themselves to simulate injuries, or acting like their imagined version of a "brain-damaged" or "crazy" person.

MALINGERING

Malingering is not classified as a true psychiatric disorder per se, but rather is defined as the conscious and intentional simulation of illness or impairment for the purpose of obtaining financial compensation or other reward; evading duty, responsibility, obligation; or exculpation or mitigation for the consequences of criminal or other illicit behavior.[4,8,73] In other words, there is a practical and "sensible"—albeit ill-intended—motive for the simulation and therefore it does not represent a true "symptom" of psychopathology, although malingering patients may certainly have other psychiatric syndromes and personality disorders (see below). The incidence of malingering differs across clinical and forensic settings and populations, with estimates ranging from 1% to 50%.[83,85] Many experts view malingering in terms of a continuum, based on the degree to which the subject is consciously aware of his or others' motivation,[70,94] although such a fuzzy conceptualization of intentionality may blur the diagnostic distinction between true malingering and other syndromes discussed in this chapter.[51,52,56,57,65,67,74]

Based on Lipman's[28] typology, malingering can be categorized into four main categories, which I have summarized by the mnemonic acronym FEEM[67]:

1. Fabrication: The patient has no symptoms or impairments resulting from the injury, but fraudulently represents that he has. Symptoms may be atypical, inconsistent, or bizarre, or they may be perfect "textbook" replicas of real syndromes. In common rehabilitation practice, this wholesale invention of an impairment syndrome is the rarest form of malingering.

Example. A man trips in a store, gets up, has no symptoms other than momentary embarrassment, but a week later decides to file a false claim for damages against the store owner, claiming severe "brain damage." On neuropsychological examination, he reports multiple symptoms that he secretly looked up in the library, and purposely performs extremely poorly on neuropsychological tests.

2. Exaggeration: The patient has symptoms or impairments caused by the injury, but represents them to be worse than they really are. This is probably the most common form of malingering in clinical and forensic practice.

Example. A woman is jostled in the course of a low-speed auto collision, is momentarily dazed, and subsequently experiences a moderate headache, some neck soreness, and transient chest irritation and shoulder soreness from the seatbelt. On clinical examination, she show up wearing a cervical collar and falsely reports unrelenting, excruciating headaches, severe shoulder and arm weakness that precludes her from working, and virtual neck immobility from "whiplash."

3. ***Extension:*** The patient has experienced symptoms or impairments caused by the injury, and these have now recovered or improved, but he falsely represents them as continuing unabated, or even as having worsened over time.

Example. After 6 months, the low back pain symptoms caused by a work-related lumbar disc bulge and radiculopathy have virtually disappeared, but on his follow-up orthopedic examination, the patient falsely reports being as agonized as ever: "There's been no let-up in the pain, Doc, and the pills you gave me don't seem to work anymore."

4. ***Misattribution:*** The patient has symptoms or impairments that preceded, postdated, or were otherwise unrelated to the index injury, but he fraudulently attributes them to that injury.

Example. A man reports that he was "just fine" until 6 months ago when his leg was carelessly pushed into a restaurant wall by a waiter rushing by. He is suing the restaurant because he now has difficulty standing or walking; the affected leg frequently "gives out," causing him to fall; and he has had to borrow crutches from a neighbor just to make it to this appointment. Further historical exploration reveals a prior injury to the knee in a high school football mishap, as well as a second injury to the same joint in a motorcycle accident while intoxicated 4 weeks prior to this examination.

As noted above, malingered exaggeration of existing symptoms is more frequent than pure fabrication of totally nonexistent illnesses or injuries.[28,83] Also, more than one category of malingering may be observed in the same patient at the same or different times. To compound matters further, more than one syndrome may be the subject of different types or degrees of malingering, e.g., postconcussion syndrome, chronic pain, fibromyalgia, toxic exposure, anxiety, depression, PTSD. Finally, malingering can co-occur with other psychological syndromes, such as the somatoform disorders or personality disorders. In many cases, malingering is suspected when patients exaggerate impairment beyond the level of clinical believability, or when they are observed (e.g., on insurance company surveillance) performing activities that they are supposedly incapable of doing.[4,51,52,57] It is here that the evaluator's knowledge of typical and atypical syndromes is crucially important in making the correct diagnosis and appropriate clinical or forensic recommendations.[6,32,33,36,46,56,57,60–62,65,67,72,74,92]

PERSONALITY DISORDERS

Personality disorders are not traumatic disability syndromes per se, but may strongly influence the psychological reaction to injury and thereby complicate recovery. Although all human beings are characterized by different clusters of personality traits, a *personality disorder* is defined as an enduring pattern of inner experience and behavior that deviates markedly from the expectations of the individual's culture, is pervasive and inflexible, has an onset in adolescence or early adulthood, is stable over time, and leads to distress or impairment.[4,89] In the current DSM-IV psychiatric classification, these disorders appear on Axis II as longstanding, stable patterns of behavior, as opposed to other psychiatric disorders that appear on Axis I, and are presumably more transitory and are potentially amenable to treatment and symptom resolution.

Antisocial personality disorder is a pattern of disregard for, and violation of, the rights of others. It is typically associated with impulsivity, criminal behavior, sexual promiscuity, substance abuse, and an exploitive, parasitic, and predatory lifestyle. These "psychopaths" or "sociopaths" have no qualms about malingering impairment for material gain and may be quite slick, engaging, and convincing in their performances, often eliciting sympathy from well-meaning doctors, lawyers, and insurance adjusters. Many clinicians have observed otherwise intellectually limited patients who are nevertheless veritable psychopathic geniuses when it comes to intuitively sizing up social situations and manipulating those around them. Demographically, there is often a complex, cyclical relationship between an impulsive, thrill-seeking, risk-prone, accident-laden lifestyle and the presence of multiple injuries and healthcare-seeking behavior on the part of antisocial individuals. In rehab settings, there is a high association with malingering and medication-seeking behavior, and common Axis I comorbidities include alcohol and substance abuse.

Histrionic personality disorder is a pattern of excessive emotionality and attention-seeking. Symptoms will typically be reported with theatrical flamboyance, and all attempts at medical explanation or reassurance will be evaded or resisted by persistent complaints of total, catastrophic, and heart-wrenching disability. If engaged in treatment, rehabilitation progress may be impeded by excessive bids for attention, reassurance, and support—although this suggestibility might be utilized positively by the rehab staff to shape constructive behavior. Common Axis I comorbidities include depression and somatization disorder.

Borderline personality disorder is a pattern of instability in interpersonal relationships, self-image, and emotion, along with marked impulsivity, including self-injury and suicidality. The pervasive anger of many borderlines, along with their tendency to alternately idealize and devalue others, may impel them to furiously pursue lawsuits to "punish" treacherous employers, doctors, or insurance companies for hurting or betraying them. Borderlines may also alternately idealize and devalue their doctors and rehab therapists, thereby complicating clinical cooperation. Common Axis I comorbidities include panic disorder, posttraumatic stress disorder, bipolar disorder, and substance abuse.

Paranoid personality disorder is a pattern of distrust and suspiciousness, so that others' actions and motives are interpreted as persecutory or malevolent. Believing that "the system is out to get me," these patients may feel no compunction about "beating the bastards at their own game" by exaggerating impairment and making excessive disability claims. Common Axis I syndromes include mood disorders and psychotic disorders, and this personality disorder may evolve into psychotic paranoid delusional disorders over time and/or with the increased stress of injury and frustration in obtaining access to treatment.

Narcissistic personality disorder is a pattern of grandiosity, entitlement, need for admiration, and lack of empathy. "How dare the defendant or insurance company not compensate me for my loss and suffering?" These patients may feel they have the right to feign or exaggerate disability in order to win their case and get what they "deserve." Less maliciously, their wounded narcissistic pride at not being able to return to work or other important roles may spur exaggerated claims of injury simply to save face by presenting themselves as "totally disabled" and therefore "unable" to work. In rehab settings, they may try to take partial or full control of the treatment plan: "It's my body—I know what's best for me." Common associated Axis I diagnoses include bipolar disorder and substance abuse.

Dependent personality disorder is a pattern of submissive and clinging behavior that stems from an excessive need to be taken of. These patients may latch onto the sick role as a way of conscripting and prolonging the nurturing care and support of doctors, family members, and solicitous attorneys. Axis I anxiety and dysthymic disorder are common.

Avoidant personality disorder is a pattern of social inhibition, feelings of inadequacy, and hypersensitivity to negative evaluation or criticism, often accompanied

by Axis I anxiety disorders, panic disorder, phobic disorders, and alcohol abuse as self-medication.

Schizoid personality disorder is a pattern of aloof detachment from social interaction, with a restricted range of emotional expression, which may represent a clinically less severe phenotypic variant of schizophrenia.

Avoidants fear people and schizoids don't need people, so both of these types may welcome the enforced invalidism and isolation of a traumatic disability syndrome to maintain their distance from unwanted social interaction.

Obsessive-compulsive personality disorder is a pattern of preoccupation with orderliness, perfectionism, and control. These patients may drive doctors and lawyers crazy with their incessant and repetitive demands for medical information and details about the progress of their cases. Heightened anxiety and obsessive hypochodriacal preoccupation may lead to the overinterpretation of mild symptoms or deficits as catastrophic. Comorbid Axis I diagnoses include anxiety disorders, substance abuse as self-medication, dysthymia, and (although less commonly than would be expected) obsessive-compulsive disorder.

Passive-aggressive personality disorder is currently not formally included in DSM-IV–Axis II, but in prior classifications[2,3] referred to a pattern of negativistic attitudes and passive resistance to demands for appropriate behavior. Often noted for their cynical demeanor, these patients may derive great satisfaction from their power to deceive, control, and manipulate clinicians, attorneys, and caretakers. To perpetuate their martyred victimhood, they may sabotage their own treatment, all the while maintaining the appearance of innocent cooperation, a behavioral pattern known as *pseudocompliance*, or "yes-but" behavior. Mood disorders are common Axis I concomitants.

THE POSTCONCUSSION SYNDROME

The *postconcussion syndrome*[91] is included in this chapter, not because it represents a functional disorder per se—indeed, traumatic brain injury would seem to be the epitome of an "organic mental syndrome"—but because functional disorders are frequently admixed and/or confused with postconcussion phenomena. Focus on physical and orthopedic injuries in accident cases—from the emergency room to the rehab clinic—often overlooks mild postconcussive symptoms, especially when there are few or no "objective" signs, or more commonly, when these have been insufficiently explored.[46,64,65,72,74]

Postconcussion Symptoms

Many commonly reported postconcussive symptoms may be confused with psychological stress reactions, functional disorders, or other psychopathology, and in fact may be comorbid with these. Postconcussive symptoms that may become the focus of particular diagnostic confusion in the rehab setting include the following:

Difficulties in Attention and Concentration. Patients have trouble following directions, keeping on track with daily rehab activity schedules, maintaining continuity of activities in the face of distractions, or remembering why they got up to go into another part of the clinic. They are slow to focus on tasks or figure out what to do next. A typical complaint is that "I'm not as sharp as I used to be." Others may describe them as "space cadets."

Learning and Memory Problems. These include difficulty retaining material heard or read, forgetting names or faces of staff members or other patients, confusing one person with another, having trouble recalling information "that I know I used to know," and struggling to remember things that used to be learned easily. In general, it is harder for new information to get processed, and what does get in seems to be forgotten more quickly. Staff may grow frustrated at repeated instructions or demonstration of exercises.

Slowness and Inefficiency. Tasks take longer to do, and they may have to be done over and over again. In many cases, the basic skills and knowledge necessary to perform a task may be essentially preserved, but the quickness and efficiency with which those abilities are applied to the task or problem at hand have been impaired. This may be mistaken for willful noncooperation or passive-aggressive sabotage of treatment goals.

Concreteness. Postconcussion patients generally do better with tasks and in situations that are familiar rather than novel, structured rather than open-ended, and specific rather than ambiguous. Patients may not appreciate jokes that involve shifting or reversing one's point of view, and they may have difficulty perceiving more than one side (their own) of an argument or putting themselves in "another person's shoes," which is often interpreted by others as shallowness or selfishness.

Depression and Mood Swings. Patients may show a lability of emotional responsiveness over the course of minutes, hours, or days. Irritable outbursts or crying spells may occur with minimal provocation. Manic highs may alternate with depressive lows, and family members, caretakers, rehab clinic staff members, and fellow patients may feel yoked to the patient's emotional roller coaster, the observers never relaxed, always vigilant for the next abrupt mood surge, which tends to put everybody on edge. In clinical settings, this may be confused with mood disorders or personality disorders.

Agitation, Irritability, Paranoia, and Rage. A smoldering edginess may be seen, and persistent carping, complaining, and hostility may strain relationships with clinic staff, fellow patients, and family members. Many postconcussion patients seem to have developed a "short fuse" that at intervals flares into aggression and rage, in part fueled by the increased suspiciousness and paranoia that sometimes develop after traumatic brain injury.

Impulsivity and Inertia. Patients may alternate between mute passivity and frenetic overactivity, may take dangerous physical and social risks in the clinic, and generally show little foresight or judgment. This type of syndrome may be especially associated with damage to the brain's frontal lobes, the "executive control system" for modulating thought, feeling, and action. Lack of appreciation of postconcussive effects may lead clinicians to interpret these behaviors as symptoms of a psychiatric disorder, personality disorder, willful obstructionism, or "just not giving a damn about his own recovery."

POSTTRAUMATIC STRESS DISORDER

Although persisting and debilitating stress reactions to wartime and civilian traumas have been recorded for centuries,[60,95,99] *posttraumatic stress disorder*, or PTSD, first achieved status as a codified psychiatric syndrome in 1980.[2] While other kinds of psychological syndromes, such as phobias, anxiety, panic attacks, and depression, may follow exposure to traumatic events, the quintessential psychological syndrome following psychological traumatization is PTSD.[4,30,31,47,60,68,69,87] As with postconcussion syndrome, PTSD in rehab settings may be conspicuous by its diagnostic absence. Especially in the predominantly male, blue-collar, work-injury population, "mental stress" may be underreported owing to its perceived association with weakness and cowardice. In such settings, the emotional distress may be more heavily somatized into physical symptoms and impairment.

Diagnostically, PTSD is a syndrome of emotional and behavioral disturbance following exposure to a traumatic stressor that injures or threatens self or significant others, and that involves the experience of intense fear, helplessness, or horror. As a result, there develops a characteristic set of symptoms, which may include the following.

Anxiety. Patients describe a continual state of free-floating anxiety and maintain an intense hypervigilance, scanning the environment for impending threats of danger. Panic attacks may be occasional or frequent. This is likely to distract patients from rehab activities.

Physiological Arousal. The patient's nervous system is on continual alert, producing increased bodily tension in the form of muscle tightness, tremors, restlessness, heightened startle response, fatigue, heart palpitations, breathing difficulties, dizziness, headaches, or other physical symptoms. Naturally, this may play havoc with any kind of biofeedback or relaxation therapies, and may contribute to increased muscular tension and heightened pain, as well as reduced tolerance for exercise.

Irritability. There is a pervasive edginess, impatience, loss of humor, and quick anger over seemingly trivial matters. These "attitude problems" will quickly exhaust the good will of clinicians, rehab staff, and fellow patients alike.

Avoidance/Denial. Patients try to blot out the event from their minds. They avoid thinking about or talking about the traumatic event, as well as news items, conversations, or TV shows that remind them of the incident. Part of this is a deliberate, conscious effort to avoid reminders of the trauma, while part involves an involuntary psychic numbing that blunts incoming threatening stimuli. In some cases, this may mimic brain injury–related frontal lobe inertia and/or right hemisphere aprosodic emotional flattening, and be mistakenly attributed to a postconcussion syndrome. In other cases, patients may be rebuked for being unmotivated and "not paying attention."

Intrusion. Despite patients' best efforts to keep the traumatic event out of their minds, the disturbing incident pushes its way into their consciousness, typically in the form of intrusive images or flashbacks by day and/or frightening dreams at night. Patients may experience panic attacks triggered by a combination of increased cardiovascular activity during exercise and frightening recollections or flashbacks of the traumatic event.

Repetitive Nightmares. Sometimes the patient's nightmares replay the actual traumatic event; more commonly, the dreams echo the general theme of the trauma, but miss the mark in terms of specific content. The emotional intensity of the original traumatic experience is retained, but the dream may partially disguise the actual event. Sleep disorders may lead to clinic absences or daytime fatigue with associated disruption of activities.

Withdrawal and Isolation. The patient shuns friends, schoolmates, and family members, having no tolerance for the petty, trivial concerns of everyday life. The hurt feelings this engenders in those rebuffed may spur resentment and counteravoidance, leading to a vicious cycle of mutual rejection and eventual social ostracism of the patient. In rehab settings, patients may seem to adopt a "who-gives-a-damn" attitude, as pervasive hopelessness erodes motivation for treatment.

Acting-Out. More rarely, traumatized patients may abruptly walk off the clinic site, or take unaccustomed risks with clinic equipment, exercise overexertion, or medication abuse. This may be interpreted as antisocial personality disorder or organic frontal lobe impairment. The diagnostic situation is further complicated by the well-documented association of TBI with premorbid impulsivity, violence,

substance abuse, and antisocial behavior, which may in turn become exacerbated by the effects of postconcussion syndrome and PTSD following work-related or other accidental injury.[34,37,42,45,48,53,59,71,82,98]

Subsyndromal and Atypical Presentations of PTSD

Subsyndromal, partial, or atypical forms of PTSD can cause significant impairment in family, vocational, and psychosocial functioning,[90] and clinicians must be alert to the types of atypical expressions of PTSD that present primarily with an intensification of one or more particular posttraumatic symptoms, but may not fulfill all of the official diagnostic criteria for PTSD per se.[1] These include the following.

Depressive PTSD Subtype. This subtype presents with psychomotor retardation, social withdrawal, inability to deal with everyday matters, loss of interest and motivation, low self-esteem, self-criticism, guilt feelings, and possible suicidality.

Dissociative PTSD Subtype. This involves a predominance of flashbacks, pseudohallucinatory experiences, depersonalization and derealization, fugue-like automatistic behavior, and possibly symptoms of multiple personality disorder.

Somatomorphic PTSD Subtype. The primary manifestation is chronic pain or other physical symptomatology, typically without clear localization or identifiable cause. Chronic pain and conversion symptomatology may be especially associated with PTSD.[5,86]

Psychotic-like PTSD Subtype. The patient displays distortions of consciousness, fantasizing, staring, inattentiveness, impaired motivation and activity, paranoia, and behavioral regression.

Organic-like PTSD Subtype. These patients present with impaired attention, concentration, learning, memory, and reasoning, along with confusion, slowness in thought, speech, and behavior; in some cases, the presentation is so severe as to suggest frank dementia.

Neurotic-like PTSD Subtype. This is characterized by anxiety, phobic avoidance, restlessness, hyperactivity, obsessionalism, and panic attacks.

NEUROSENSITIZATION SYNDROMES

Many of the syndromes we see in mental health and rehabilitation settings reflect problems in adaptation and coping with injury of some kind, including postconcussion syndrome, chronic pain, posttraumatic stress disorder, multiple chemical sensitivity, and depression. Indeed, inasmuch as many injuries occur under frightening circumstances and may produce long-term disability that is demoralizing, many of these traumatic disability syndromes[60] are frequently comorbid, creating vicious cycles of impairment and reduced quality of life.

These syndromes may also share common pathophysiological mechanisms involving patterns of neuroplasticity that are important to understand from both theoretical and practical clinical perspectives. Accordingly, the present author has been attempting to tie together some of the recent empirical and theoretical work in the area of traumatic disability syndromes, with specific reference to the commonly comorbid syndromes of depression, chronic pain, postconcussion syndrome, and posttraumatic stress disorder (PTSD), in order to develop a broader model of what I have called *neurosensitization syndromes* (NSS).[54,66] A NSS may be defined as: (1) a syndrome of subjective discomfort and objective functional disability; (2) that often appears excessive in duration and severity with respect to the identified initiating injury or event; (3) that may be resistant to conventional medical and psychological treatment modalities; and (4) that is hypothesized to develop as the result of progressively

enhanced sensitivity or reactivity of central nervous system (CNS) mechanisms at the neurophysiological, biochemical, and intracellular levels.

Depression

For most patients, the entry point into the mental health system is some variation of persistent dysphoric affect, and disorders of mood pervade and modify the clinical expression of most forms of psychological disability. Mood disorders tend to occur in cycles, waxing and waning along with circumstances in the life of the traumatically disabled individual, but often a depressive episode seems to occur in the absence of any identifiable precipitant, in which case it is typically regarded as "endogenous."

To explain these cycles of affective disorder, Post and colleagues[77–80] have appealed to the paradigm of neurophysiological kindling, in which intermittently repeated application of a stimulus to sensitive limbic structures, especially the amygdala, results in a cumulative sensitization that in turn produces some form of episodic paroxysmal emotional, or behavioral expression. This can be in the form of a mood disorder, a violent or suicidal outburst, a psychotic episode, or a frank seizure.

Kindling appears to represent a relatively permanent change in neural excitability. After many repetitions of kindled behavioral phenomena, *spontaneity* may occur, in which behavioral changes can now develop in the absence of any signficant external stimulation. This model explains how stress-induced mood alterations become sufficiently sensitized to occur spontaneously, providing one possible model for recurrent cycling mood disorders. In this model, severe depressive states that appear to arise spontaneously may actually reflect the summation of depressogenic influences on mood-mediating brain mechanisms occurring over a critical period of time.

At the microneuronal and intracellular levels, kindled activation of neurotransmitter pathways produces not only acute events associated with rapid alternations in neural firing and short-term neural adaptation, but also much longer-lasting intracellular changes at the level of genetic transcription by way of DNA changes involving induction of messenger RNA. Such transcription factors may provide the basis for a biochemical cascade that results in more enduring neurotransmitter, receptor, and peptide changes. These, in turn, would provide the biochemical and anatomical basis for long-term synaptic adaptation and memory that could last indefinitely.

The sensitization pattern of kindling suggests that the "symbolic" aspects of previous events that have precipitated a depressive response might be conditioned so that they later come to elicit the depression even in the absence of the original stress or trauma. Stressors related to separation, loss, and devalued self-esteem that are associated with traumatic disability may play an important pathophysiological role in triggering repeated depressive episodes with each successive failure, frustration, or disappointment, and lead to progressively heightened long-term vulnerability to subsequent recurrences. Eventually, even anticipated stresses or imagined losses may be capable of producing the behavioral, physiological, and biochemical alterations of a full-blown depressive disorder: the patient "gives up trying" and "just won't get better."

Chronic Pain

A common diagnostic association occurs between depression and chronic pain.[24,25,39,40,41,43,76] Similar to Post's[78] conceptualization of depression, Coderre et al.[11] have proposed a *central neuroplasticity model* of chronic pain that appears to meet the present criteria for an neurosensitization syndrome. Research and clinical experience suggest that peripheral injury can produce CNS changes that far outlast the original injury, probably resulting from either a reduction in the threshold of pain receptors or a sensitization-induced increase in the excitability of CNS neurons involved in pain transmission.

Pain episodes, as well as other stressors, may trigger a number of autonomic and musculoskeletal reactions, most notably sympathetic activation and elevated muscle tension. If stress or pain-related muscular contractions occur repeatedly and/or are sustained, muscular and sympathetic reflexes lead to: (1) increases in muscle tension via dysregulation of the gamma motoneuron system; (2) sympathetically mediated vasoconstriction, leading to pathological sympathetic overflow; and (3) pathological inflammatory cascades involving the hypothalamic-pituitary axis. These phenomena may explain many cases of myofascial pain, reflex sympathetic dystrophy, and other intractable neuromuscular and neurovascular pain syndromes.[12,16]

Coderre et al.[11] theorize that persistent noxious stimulation may result in increased expression of intracellular messenger proteins in CNS structures involved in pain transmission, including the periaqueductal gray, thalamus, habenula, and somatosensory cortex. Thus, activation of intracellular, molecular messenger systems in response to sensitization is invoked as an explanatory mechanism in both Post's[78] model of affective disorder and Coderre et al.'s[11] model of pathological chronic pain. It is not difficult to conclude that the vicious cycles of agony and despair we see and treat in our patients reflect the operation of this kind of "out-of-control" positive feedback loop, one of the factors that makes chronic pain syndromes so difficult to treat.

Postconcussion Syndrome

Traumatic brain injury often accompanies orthopedic injuries that induce chronic pain,[35,58] and like chronic pain, postconcussion symptoms frequently persist. In the postconcussion syndrome, there is often an immediate postinjury "grace period," followed over the course of days and weeks by a worsening clinical state, with progressive functional disability sometimes seen years down the road, often interpreted as somatization or malingering. However, this kind of clinical presentation presents the possibility that neurosensitization mechanisms may underlie many postconcussion symptoms.

Recent experimental and human models of postconcussive effects[7,19,21,81] suggest that neuronal axons are typically stretched, but rarely severed, in the immediate impact injury. This causes a focal disruption of the axonal cytoskeleton that leads to focal axoplasmic swelling, stretching, infolding, and eventual detachment, typically taking up to 12 hours to develop. Although only a small percentage of the brain's axons are damaged in most mild-moderate concussions, diffuse axonal degeneration ultimately translates into diffuse deafferentation of the synaptic target sites of the destroyed axonal terminals.

Diffusely deafferented postsynaptic sites can be reoccupied over time with terminals derived from related intact fiber populations, but with anomolous "rewiring" patterns different from those originally lost. Thus, diffuse axonal injury and its subsequent diffuse deafferentation most likely contribute to the early clinical impairment syndrome seen in mild head injury, while the compensatory neuroplastic changes—collateral spouting, dendritic arborization, and neurotransmitter changes—would account for the symptomatology observed during the typical 3–12 month postconcussive recovery course. Cases of persisting or delayed-onset postconcussion

symptoms may involve the less-than-optimal reafferentation of traumatically damaged synaptic pathways—a "defective rewiring" of brain circuits mediating subtle but important aspects of cognition, emotion, personality, and behavior.[13,74]

At the inter- and intracellular level, cerebral concussive impact may cause widespread neuronal depolarization that produces a large, nonspecific release of excitotoxic neurotransmitters. Resulting abnormal activation of receptors could produce changes in intracellular signal transduction pathways resulting in transient, long-lasting, or irreversible alterations in neuronal function. Traumatically induced tissue deformation opens ion channels, resulting in a massively abnormal potassium efflux inducing indiscriminate neurotransmitter release and further destructive and destabilizing depolarization.[20,21] Thus, according to the neurosensitization model, diffuse axonal injury and excitatory neurotransmitter release may induce sensitization of pathological postconcussive symptomatology, much as occurs with the sensitization of affective disorder, chronic pain, and PTSD. Indeed, this may partially account for the comorbidity of these traumatic disability syndromes.[54,60,66]

Posttraumatic Stress Disorder

Painful injuries and postconcussive symptoms typically have their origin in some form of traumatic event such as a traffic, worksite, or assault injury. In fact, PTSD is so frequently comorbid with other disorders, such as chronic pain, postconcussion syndrome, depression, and somatoform disorders,[45,46,60,74,86] that "pure" chronic PTSD would seem to be the exception, supporting the concept of a *cascade* of symptoms or syndromes evolving over time, initiated by either the original trauma or by the PTSD itself.

Charney et al.[10] have elaborated a psychobiological model of PTSD that appears to characterize it as a neurosensitization syndrome. According to this model, sensitization by fear associated with traumatic stress results in a change in excitability of amygdaloid neurons. This in turn influences the functioning of a variety of limbic and brainstem structures involved in the somatic and autonomic expression of fear and anxiety. For example, a reduced activation threshold of the locus coeruleus results in increased norepinephrine output at locus coeruleus projection sites. In addition, functioning in mesocortical dopaminergic neurons is elevated.[9,15]

Stress-induced impairment of long-term potentiation, mediated in part by excitatory amino acid, noradrenergic, and opioid receptor systems, may be responsible for the development of the learning and memory deficits observed in PTSD. Because extinction appears to involve an active learning process—an idea that goes back to Pavlov[75]—deficits in learning may impair normal extinction in patients with PTSD, leading to the abnormal persistence of emotional memories. This may partially explain the "paradox" of learning and memory deficits coexisting with abnormally intense intrusive recollections in PTSD. In patients with PTSD, specific sensory phenomena, such as sights, sounds, and smells circumstantially related to the traumatic event, persistently produce a recrudescence of traumatic memories and flashbacks. The continued revivification of traumatic memories associated with a painful injurious event may thereby act as both trigger and reinforcer of depression, chronic pain, or somatoform disorders as coping responses to trauma, thereby perpetuating dysfunctional traumatic disability in yet another series of vicious cycles.

CONCLUSIONS

As clinical science progresses, the distinctions between "functional" and "organic" continue to blur. Therefore, the answer to the question posed in this chapter's title is a resounding "*unknown*." After all, the purpose of diagnostic classification and labeling should be increased understanding for the purposes of treatment. Just as most medical and psychiatric syndromes have both organic and psychosocial etiologies, so will most of the therapies applied. The remainder of the chapters in this volume will guide the reader toward more knowledgeable and effective care of those patients who come to us with diagnostic challenges and the need for our healing skills.

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APPLIED PSYCHOPHYSIOLOGY: STATE OF THE ART

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SCOPE AND DEFINITION

Applied psychophysiology and biofeedback have a broad scope ranging from the clinical settings of psychotherapy and health care to a number of areas including performance enhancement[21] and ergonomic applications to increase work efficiency and worker productivity and to decrease injury.[72] More esoteric applications include psychophysiological polygraphs in forensic and employment screening for deception detection.[59] There is some promise and application of biofeedback devices in transpersonal and psychospiritual realms.[49] In the consideration of functional aspects of physical symptoms, psychophyisiological assessment can have very important roles because: (1) aspects of autonomic nervous system functioning can be assessed, (2) surface electromyography (SEMG) can provide data about muscle recruitment and activation patterns inaccessible to palpation or needle electromyography, and (3) monitoring brain wave functioning in various activation states provides one of the least expensive means of visualizing brain functioning.

Nearly everyone has read something about biofeedback, as the term became a household word in the late 1960s and early 1970s. In the wake of such successful name recognition, the major professional organization of providers of biofeedback changed its name and its journal to a more expansive and less familiar title of "applied psychophysiology and biofeedback." The question of "what's in a name" has developed, and an entire journal issue[2] has been devoted to attempting to clarify what is meant by "applied psychophysiology and biofeedback." From a practical