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Brain Injury and Functional Disorders Part IV

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Neurorehabilitation professionals are often asked to provide an opinion about the presence or absence of brain dysfunction after reported or suspected minor head injury (MHI). This is often after other medical practitioners (*e.g.,* neurosurgeons, neurologists) have already rendered an opinion about whether or not any reported cognitive, behavioral, and emotional sequelae are neurologically based. Especially when significant symptoms are reported and persist, professionals often demonstrate predispositions with regard to interpreting symptoms as neurologically based (*i.e.,* due to brain injury), due to motivational factors (*e.g.,* malingering or "compensation neurosis"), or representative of secondary psychological effects (*e.g.,* anxiety, somatic hypervigilance, secondary gain, etc.). Indeed, there are controversies about whether the postconcussion syndrome (PCS) is an organic phenomenon,[1,2,3] and a professional's beliefs clearly influence how a patient's condition is conceptualized and treated.

In fact, neurorehabilitation efforts may depend more on the philosophy of the treating professional than on the patient's

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symptoms *per se*. Further, the opinions of health professionals can influence how a person responds to his or her injury.[4] Finally, as the course of time passes from the onset of injury, psychological (versus organic) factors exert a greater impact on the expression of postconcussive symptoms.[5,6,7] This can occur for many reasons, including that the patient might have returned to pre-injury activities (*e.g.,* work) too soon and could be under situations of greater stress or demand or because other stresses, such as the effects of litigation or new onset medical conditions or unrelated life stresses, could exert an influence.

Symptoms occurring earlier post injury appear organic in nature (*e.g.,* headache, dizziness, double vision), while those occurring later appear more psychoemotional (*e.g.,* depression, anxiety, irritability). For some patients, however, symptoms tend to worsen over time and earlier "organic" type symptoms may be reported later. This deviates from the expected pattern of recovery after mild head trauma[8,9,10,11] and raises suspicion that functional or even pathological factors like malingering or "compensation neurosis" may be playing an increasing role.

Not all such patients, however, are feigning or exaggerating difficulties, even if they demonstrate impairments on neuropsychological tests or in psychoemotional functioning that appears disproportionate to head injury severity. In a small number of cases, increased vulnerability to neurologic injury or greater than realized injury severity may be responsible. In the vast majority of cases, however, other factors, such as emotional distress, certain personality vulnerabilities, or environmental demands, can lead to greater functional disability than would be expected based solely on injury severity variables (*e.g.,* length of unconsciousness, post-traumatic amnesia).

Some of the risk factors that have been associated with persistence of PCS symptoms include preexisting psychiatric history, history of drug or substance abuse, previous head injury, female gender, advanced age, and other conditions causing loss of neural and psychosocial coping reserves.[12,13] Importantly, a diagnosis of a formal mental disorder using the DSM-IV[14] is not a necessary precondition for psychological vulnerabilities for poor post injury adjustment.

Individual coping vulnerabilities that leave persons "at risk" for psychoemotional disturbance that are clearly present in an individual's personality dynamics[15,16] may not be sufficient to disrupt pre-injury functioning in the absence of significant stress. The stresses associated with injury and adverse reactions to transient physical or cognitive changes and life disruption, however, may represent demands that decompensate ongoing coping and produce psychiatric sequelae. Symptoms may also arise from stress associated with return to work, self doubt or anxiety about ability to return to pre-injury level of functioning and meeting expected demands, associated fear of rejection, fear of loss of a supportive safety net, anger and resentment, the effects of litigation, or the psychological consequences of the injury itself, and an interaction of these and other factors.[17]

As time post injury increases, a multitude of factors can impact a person's day-to-day functioning. Losses relating to, for example, work, income, cognitive efficiency, family role changes, variable stresses unrelated to injury, etc., can represent additional stresses that further decompensate adjustment and further exacerbate poor psychoemotional adjustment.

One of the potential pitfalls in the neurobehavioral assessment of suspected brain injury is the potential to over diagnose brain injury based solely on a compatible set of patient complaints. Clearly, accurate clinical assessment requires familiarity with the expected recovery patterns following mild traumatic brain injury (MTBI)[18,19,20] as well as differential patterns associated with exaggeration and malingering.[21,22,23,24,25,26,27]

When there is a marked discrepancy between the person's claimed distress or disability and the objective findings, when there is a medicolegal context, and when there are suspicions about motivation, the possibility of exaggeration response bias and malingering should be closely scrutinized. Conversely, the possibility of under diagnosing brain injury and over diagnosing malingering also exists.

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Clearly, there is no uniformity of responses following MTBI, so a predisposed professional could easily over interpret natural variability as inconsistency or interpret psychological reactions to impairments as causal.

The types of deficits seen with post concussive cognitive sequelae are not specific to brain injury and can result from multiple other factors, singularly and in combination. These include medication effects, depression and other psychiatric states, chronic pain, sleep disturbance, developmental learning disabilities, medical illnesses such as hypertension, diabetes, COPD, and sleep apnea, and others.[e.g. 28] Clinicians must be familiar with psychiatric or other syndromes that may present as organic brain injury. Of course, the presence of a psychiatric syndrome or response bias does not necessarily exclude the diagnosis of another organic syndrome. This certainly complicates the process of disentangling multiple clinical entities that sometimes co-exist. Only methodical assessment can differentiate sequelae secondary to brain injury from the multiple other factors, which can produce similar symptomatology.

DIFFERENTIAL DIAGNOSIS

Traditionally, the most common differential diagnosis neurorehabilitation professionals are asked to address in cases of mild head trauma is the determination of whether or not symptoms are neurologically or functionally based. With an increasing number of personal injury cases, the differential also concerns whether symptoms are "real" or manufactured. This distinction is really not dichotomous or mutually exclusive. [See part II and especially part III in the "Masquerades" series for a review of response bias.][29,30]

Further, there are other psychiatric diagnoses that must be considered in assessing functional disability. Unfortunately, there are no simple guidelines for reliably distinguishing neurologic sequelae following brain injury from other possible diagnostic entities. Disentangling the effects of preexisting conditions on cognition functioning can be difficult, and examiners must rely on other sources of pre-injury information (*e.g.,* medical records, education, and employment history, etc.) to assist in differential diagnosis.

Accurate differential diagnosis requires familiarity with the major functional medical disorders. In this paper, we address issues of differential diagnosis. Causality and apportionment are important additional considerations that are beyond the scope of this paper but will be elaborated in a future *Masquerades of Brain Injury* issue. The following syndromes represent the major functional medical disorders.[31] These are syndromes that present as physical disorders with symptomatology that produces functional disability that is primarily mediated by psychological disturbances.

POST-TRAUMATIC STRESS DISORDER

Post-traumatic stress disorder (PTSD) is a psychological reaction to an extremely distressing event, which is usually experienced with intense fear, terror, and helplessness. The most common symptoms of PTSD are recurrent and intrusive recollections of the event, distressing dreams during which the event is re-experienced, deliberate efforts to avoid thoughts or feelings associated with the event, as well as activities or situations that arouse recollections of it.[32] Like those with mild head trauma, patients with PTSD complain of concentration difficulties, forgetfulness, sleep difficulties, irritability, and poor frustration tolerance; they are likely to become depressed, anxious, and exhibit cognitive problems (secondary to emotional and psychological factors).

Until recently, the prevailing opinion has been that cerebral concussion and PTSD could only co-occur in the absence of a loss of recall for the trauma or events surrounding an accident. Recent evidence, however, suggests that patients who sustain MTBI with loss of consciousness and amnesia can also develop phobic-kinds of responses and generalized fears, which can produce disability. Traumatic pain experience, islands of consciousness with partial uncertain recollections or even reconstructed memories, and heightened physiological anxiety that generalizes to injury-related symptoms or stimuli, can all contribute to post-traumatic stress symptomatology. Importantly, because the cognitive and other symptoms associated with PTSD closely overlap with difficulties frequently endorsed by MTBI patients, distinguishing the two are accomplished only through careful and deliberate assessment.

FACTITIOUS DISORDERS

Factitious disorders are characterized by physical and psychological symptoms that are produced by the individual and, like malingering, are under voluntary control. The judgment that the behavior is under voluntary control is based, in part, on the patient's ability to simulate illness in such a way that he or she is not discovered. This is made by excluding all other possible causes of the behavior. Although similar to malingering in some respects, the person with a factitious disorder usually has more obvious or severe character pathology, such as a borderline personality disorder, and the only apparent or primary goal is to assume the patient role. A history of repeated "accidents" with a compensatory tinge may exist, which may reflect the person's impulsivity and intense anger or passive-aggressive behaviors in certain situations.[33]

The most common type of factitious disorder is chronic factitious disorder with physical symptoms, otherwise known as Munchausen's syndrome. This involves multiple hospitalizations, and often multiple surgeries, for symptoms with no apparent true physical disorder. The complaints of patients are typically dramatic and colored by the

patient's knowledge of the disorder and hospital procedures. Although there is a conscious attempt to fabricate symptoms, the underlying motivation and reasons may be outside of the patient's conscious awareness.

Another common factitious disorder is the factitious disorder with psychological symptoms or Ganser syndrome. This is often characterized by the symptom of giving approximate answers or talking past the point,[34] and patients often provide "near misses" of the correct response during mental status testing or neuropsychological evaluation. Patient's who respond to obvious questions with near misses frequently arouse suspicion in examiners and might also become extremely negativistic and uncooperative to further questioning. Although this style of responding and seemingly oppositional behavior might suggest the presence of malingering because less than optimal amount of effort is being put forth, the goals are clearly disparate.

SOMATOFORM DISORDERS

The somatoform disorders involve the presence of physical symptoms that suggest a physical disorder for which there are no demonstrable organic findings or known underlying pathophysiologic mechanism. There is also positive evidence, or at least a strong presumption, that the symptoms are not intentionally produced but are linked to psychological factors or conflicts.[35] Even settlement of a legal case would not likely ameliorate the symptoms if the underlying conflicts were not addressed.[36]

Unlike factitious disorder or malingering, the symptom production in somatoform disorders is not under voluntary control, and the person does not experience the sense of controlling the symptoms. Of the somatoform disorders, somatization disorder and conversion disorder are perhaps the two most often considered in cases of suspected or purported MTBI. The former involves multiple somatic complaints for which no physical cause can be found, and the patient often makes repeated visits to physicians and may have numerous hospitalizations over the course of several years. Typical complaints include pseudoneurologic or conversion-type symptoms, gastrointestinal complaints, psychosexual difficulties, cardiopulmonary problems, chronic pain, and symptoms in the female reproductive system. There cannot, by definition, be any identifiable organic etiology for the symptoms.[37]

The essential feature of a conversion disorder is a loss of, or alteration in, physical functioning that suggests a physical disorder but which instead is an expression of a psychological conflict or need. The most obvious and "classic" conversion symptoms are those that suggest neurological disease (*e.g.,* paralysis, seizures, etc.). Like malingering, there are different kinds of "gains" that a patient with conversion disorder can achieve, but the symptoms are not under voluntary control. In one situation, the person achieves "primary gain" by keeping an internal conflict or need out of awareness, and usually there is a temporal relationship between an environmental event that relates to the psychological conflict or need and the initiation or exacerbation of the symptom. In another situation, the individual might achieve "secondary gain" by avoiding a particular activity that is aversive, or by getting support from the environment that might not otherwise be forthcoming. Some MTBI patients with bona fide symptoms become incapacitated and require significant others to assume certain tasks for them. In these cases, somatoform disorder and malingering should be ruled out.

Most conversion symptoms develop in response to extreme psychological stress and appear rather suddenly. In contrast to mild head trauma patients who may be overly concerned or distressed by their symptoms, patients with conversion typically demonstrate a relative lack of concern (la belle indifference) over their reported symptoms that are out of keeping with the severity of the impairment. In these cases, it is usually a family member who is acutely aware of changes in the person's overall level of psychological and social functioning. From a diagnostic standpoint, the relative degree of awareness or unawareness of deficits versus the proportionality of related concern a person has might be another way, in combination with a comprehensive assessment that includes a thorough medical exam and neuropsychological evaluation, to distinguish between MTBI and a conversion disorder.

PSYCHOGENIC SEIZURES

Psychogenic seizures are a subcategory of nonepileptic seizures (NES) or "pseudoseizures" characterized by episodic or paroxysmal phenomena that resemble epileptic seizures but do not have the same characteristic changes in underlying brain activity.[38] As with epileptic seizures, patients with NES may demonstrate falling, self-injury, and may even be incontinent; however, NES do not generally include such epileptic sequelae as tongue biting, unprotected falling or incontinence.[39]

NES are frequently misdiagnosed as seizures. Up to 25 to 30 percent of people with nonepileptic seizures may also have epileptic seizures and nonepileptic events may generate from physiologic or psychological causes. The main differential diagnoses for physiologic events that present as NES include autonomic disorders, cardiac events, cerebrovascular disease, drug toxicity, metabolic disorders, migraines, and sleep disorders.

Psychogenic seizures (also known as hysterical epilepsy, conversion fits, pseudo-attacks, and somatoform spell disorder) refer to the subcategory of NES with a psychological etiology. Psychogenic seizures may occur as part and parcel of disorders associated with anxiety, depression, and

psychosis, and a history of childhood trauma is common. They occur most commonly in adulthood. Malingering, factitious disorder, and dissociative disorder also must be considered under the differential diagnosis of NES from psychological or functional causes.

The diagnosis of psychogenic seizures is accomplished by first ruling out epilepsy (*e.g.,* observation and clinical symptom correlation, EEG video monitoring, post seizure blood prolactin levels, placebo or suggestion induction; familial epilepsy risk, responsiveness to anti-seizure medication).[40] Secondarily, physiological syndromes (*e.g.,* cataplexy, transient ischemic attacks, syncope) must be ruled out. Finally, psychogenic seizures are diagnosed by analyzing the patient's history. A number of signs suggesting psychogenic rather than epileptic episodes, including frequent episodes unaffected by anticonvulsants, coexistence of psychological symptoms or associated psychiatric disease or vulnerabilities (*e.g.,* anxiety, depression, inappropriate affect or lack of concern, somatization or hysterical personality traits, childhood abuse or trauma, a history of poor adjustment or under achievement, abnormal interaction with significant others, and the presence of emotional triggers). Behavioral techniques, which have been found somewhat successful in ameliorating epileptic seizures are considered the primary treatment of choice for psychogenic seizures.[41,42]

The diagnosis of psychogenic seizures is ultimately probabilistic and fallible. Indeed, accurate diagnosis should allow consideration of the following cautions:[43]

- Epilepsy suggestive EEG can occur in asymptomatic patients.
- At least some patients labeled as having pseudoseizures are eventually diagnosed with epilepsy or as having structural brain lesions using more sensitive recording procedures (*e.g.,* depth recordings, MRI) or other physiologic conditions.
- Real and pseudoseizures commonly co-exist in patients.
- Nonepileptic seizures and psychogenic seizures are too often used synonymously and are confounded in the literature.
- Differentiation of nonepileptic seizures is much more difficult for partial seizures versus tonic or clonic seizures.

FUNCTIONAL AMNESIA

There is often some disturbance of mnemonic function with mild or other traumatic brain injury and with other disorders involving structural brain lesions. In the case of MTBI, memory problems are usually discrete and limited, resolving fully within weeks to months. Although there may be some complications (*e.g.,* poor memory associated with premorbid ability structure, interfering effects associated with headache or other pain, affective distress, sleep disturbance or other[44] assessment of memory problems following mild traumatic brain injury is generally straightforward.

There are sometimes, however, dramatic memory problems that may represent a functional amnesia. The most common and striking of these are perhaps the cases of profound retrograde amnesia in which there is complete or near complete loss for explicit recall of personal, autobiographical information from prior to the trauma usually with preservation of anterograde memory or capacity for new learning, and preservation of semantic memory, implicit memory, and well learned skills.[45,46,47]

Functional retrograde amnesia, sometimes termed psychogenic amnesia, may be seen subsequent to a variety of traumatic experiences. It is widely accepted that psychological trauma can produce such a presentation. Whereas these memory problems do not involve structural brain lesions, abnormal brain activity has been demonstrated with functional imaging.[48,49] This indicates, as would be expected with any psychological act, that there is an underlying neurobiological substrate manifesting in what has been termed an "mnestic block syndrome."[50,51]

These disorders, however, are reversible and resolve spontaneously with appropriate psychotherapeutic interventions, or under sodium amytal interviews, although they sometimes persist for lengthy periods. As with diagnosing other disorders, dissimulation must be ruled out. Further, the possibility of an iatrogenic effect of professional suggestion on the part of medical and legal professionals should be considered.

CONCLUSIONS

Neurobehavioral assessment in cases of mild head trauma represents a burgeoning area of growth for neurorehabilitation professionals, and our understanding of the many factors involved in symptom production and persistence has grown dramatically over the past decade. Whereas post-traumatic symptoms were once believed to be either exclusively "organic" or "neurotic," a more complex understanding of the multiple factors determining functional disability and outcome has emerged.[52] As a result, clinicians in hospital practice and in the private sector must become more familiar with the different medical and psychiatric diagnoses that are commonly involved in the differential diagnosis of MTBI. Legal professionals, too, must be aware of the different diagnoses that mimic or look very much like MTBI so that they can judge the merits and liabilities of their cases.

Whereas identification of neurobehavioral impairments in the acute stage after mild head trauma may be relatively simple, assessment of persisting symptoms after a few weeks or months is a more complex enterprise. The longer after the accident or injury that post-traumatic symptoms

persist, the greater the likelihood that secondary, psychological factors play a major role.[53,54] In addition, the clinician must determine if the individual is intentionally producing the symptoms or not. If it is determined that the patient is consciously producing their symptoms, then the next decision is to assess whether or not there is an obvious goal that the person is trying to achieve. In clinical practice, these distinctions are not always that easy to make.

Patients presenting with significant functional disabilities after seemingly mild injuries represent complex assessment challenges for physiatrists, neuropsychologists, and other rehabilitation professionals. At a minimum, clinicians must have an understanding of the pathophysiology and neurobehavioral sequelae associated with MTBI. They also must have a familiarity with other more "traditional" psychiatric disorders to assist in differential diagnosis. Data from neuropsychological testing, in combination with other objective and subjective psychological data (*e.g.,* a thorough history, clinical interview, review of school records, reports of collaterals, etc.) and information from other medical disciplines, promises the greatest method for differentiating between premorbid factors and post morbid residua secondary to an accident or injury.

Many cases of mild head trauma are not simple or clear-cut, but consideration of some of the other functional disorders mentioned in this article may lead to a greater understanding of some of the complexities involved in differential diagnosis and provide a better foundation for rendering opinions about the causes, needed treatment, and eventual prognosis of symptoms following an accident that purportedly involves MTBI.

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