Functional neurologic disorders and related disorders

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Introduction

This article includes discussion of psychogenic neurologic disorders, functional neurologic disorder, functional movement disorder, conversion disorder, and hysteria. The foregoing terms may include synonyms, similar disorders, variations in usage, and abbreviations.

Overview

Several behavioral disorders are related by (1) their resemblance to other, more familiar neurologic disorders; (2) lack of well-established biomarkers (eg, structural lesions on brain imaging studies, seizure waveforms on EEGs); and (3) aggravation of symptoms with the patient s attention to the disorder. However, the features and causes for these disorders are very different among themselves. This topic reviews functional neurologic disorder, Munchausen syndrome, Munchausen syndrome by proxy, and Ganser syndrome.

Key points

- Functional neurologic disorders are commonly encountered in general neurologic practices and, hence, knowing their manifestations and treatment is crucial for clinical care.
- The disturbance is involuntary, yet at the same time it can be controlled by the patient intermittently.
- Despite being self-controllable, the disturbance is generally disabling unless expert professional care is provided.
- There is no consistent association between functional neurologic disorder and either posttraumatic emotional stress or sexual abuse.
- Functional neurologic disturbances disorder responds best to empathetic concern by the clinician; demonstration that the disorder lacks a structural or permanent etiology; explanation that it can be improved with distraction; and guided attempts to reduce triggers of onset. Cognitive behavioral therapy, combined with physical therapy when warranted, is emerging as a successful intervention.
- Although most forms of functional neurologic disorder are relatively benign, Munchausen syndrome by proxy (the false reporting of illness by caregivers of dependent individuals) demands rapid intervention.

Historical note and terminology

The term "hysteria" was originally applied to diverse female fluctuating behavioral disorders that were attributed from classical times to a "wandering uterus" (Zimmer 2004). Offray de La Mettrie, for example, published in 1738 an account of episodic catalepsy (waxy immobility of the limbs) in a woman that he attributed to hysteria arising from amenorrhea (Walusinski 2012). Eighteenth century treatments for hysteria were radical and untested, including bloodletting, beatings, diet, fresh air, and writing (Meek 2013). In the 19th century the term “hysteria” came to specify functional disorders following the impression that primarily women simulate medical disorders for secondary gain. In contrast, men were thought to be more susceptible to hypochondriasis, the preoccupation with diverse bodily complaints (Crimlisk and Ron 1999). In 1859, Briquet published a landmark study that used "hysteria" to describe symptoms affecting diverse bodily systems. Functional disorders were subsequently emphasized by the works of Charcot, Freud, and Janet (Crommelinck 2014).

Reynolds published in 1869 a prescient overview of disorders of motor control and sensation that appeared to stem from an ideological fixation and were amenable to compassionate behavioral retraining (Reynolds 1869). Reynolds rejected terming such patients “hysteric” as the term was used at the time. In 1888 Blocq comprehensively described a case series of the acute inability to stand and walk despite full motor control of the legs while supine (which he termed “astasia-abasia,” a term that continues today) (Okun and Koehler 2007). Although he doubted that the disturbance had a purely psychologic etiology, his pathophysiologic hypothesis—that marked emotional distress can aggravate cerebral inhibition over spinal walking mechanisms—is surprisingly similar to some current pathophysiologic

hypotheses for functional disturbances (see below). Elsewhere, a good overview of the history of the recognition of functional seizures has been provided (LaFrance and Devinsky 2004). A surge of interest in functional neurologic disorders came with World War I, when European soldiers returned from combat with a variety of neurologic deficits without traumatic brain injury (Jones et al 2007; Linden and Jones 2013). At this time, these disorders were considered to be neurologic, even though their precise etiologies were unclear. However, the neurologic interest in the causes, physiological basis, and treatment of functional disorders became overshadowed, for the most part, by the advent of psychoanalysis (Crommelinck 2014). A return to interest in the neurophysiological basis of functional disorders began in the 1960s and continues to the present.

A wide variety of synonyms for these disorders have been used up to the present, which hampers understanding. These terms include hysteria, conversion disorder, medically unexplained disorder, psychogenic neurologic disorder, and pseudoseizure. Edwards and Bhatia emphatically recommend the term “functional neurologic disorder” on the grounds that patients find this less objectionable than rival terms (Stone et al 2002b; Edwards and Bhatia 2012), which helps to emphasize the reversibility of the disorder (Reuber et al 2005; Stone et al 2005). PubMed has shown a continually growing use of this term over the past decade.

"Somatization disorder" or "Briquet syndrome" is a variant of functional disorder in which nonspecific bodily complaints appear (eg, fatigue, insomnia, irritable bowel) without resembling specific neurologic disorders and without objective physiologic disturbance (Khouzam and Field 1999; Stone et al 2005). "Malingering" is the fully aware simulation of a medical disorder (frequently neurologic) for personal gain, particularly for money, material goods, or improved access to specific privileges (eg, transfer from jail). "Factitious disorder" is the willful simulation of a medical disorder without clear financial or opportunistic gain (Bauer and Boegner 1996). Instead, sufferers have a need for an enhanced feeling of control or attention. "Munchausen syndrome" is a variant of factitious disorder (often with diverse complaints), in which the patient undergoes frequent clinic or hospital evaluation, sometimes resulting in invasive, even injurious, testing or treatment. The term was coined by Asher (Asher 1951), who thought that the wide meanderings of afflicted patients from clinic to clinic and their elaborate health histories resembled the fantastic travels regaled by a fictitious character depicted by Raspe in 1785, Baron Munchausen (Pankratz 1986). (This individual was likely inspired by the real Baron Münchhausen; consequently, German spellings of this disorder also appear.) "Munchausen syndrome by proxy" refers to a caregiver's bearing false evidence of medical illness in another individual who is incompetent to represent himself (a child, in most cases described thus far) (Meadow 1977). Finally, Ganser syndrome is a controversial disorder that involves, among other features, the suggestion of simulated confabulation (Ganser 1898).

Clinical manifestations

Presentation and course

Functional neurologic disorder is a generic term for inconsistent neurologic deficits that are aggravated with the patient s attention to them. Within some individuals, there is 1 symptom; in others, a wide variety may occur. Functional symptoms may also evolve. The functional neurologic disorders can affect nearly any widely recognized neurologic function. Unlike traditional neurologic disorders, functional neurologic disorders are not associated with objective biomarkers on standard instrumented assessments (eg, structural damage evident on brain MRI; epileptic waveforms on EEG). In addition, traditional neurologic disorders appear either to be unaffected or improved by attention.

Common features of functional neurologic disorders include the patient s limited awareness that their attention to their symptoms can affect their severity, lack a sense of self-agency, limited awareness of intact neurologic control for the specific symptom, and perceived great effort when attempting to control their symptoms (Espay et al 2018; Teodoro et al 2018). In most instances, patients view their disorder as stressful and disabling. In contrast, la belle indifference, or the lack of concern for the neurologic symptom, does not occur in the majority of functional disorder patients and is not a specific finding (Stone et al 2006).

Commonly, functional neurologic disorder is preceded by an abrupt physical event in the preceding 3 months, most often physical injury, infection, or traditional neurologic illnesses (eg, facial neuritis, migraine, brachial neuritis) (Pareés et al 2014a). Therefore, physical triggers may be key to the onset of many forms of functional neurologic disorder. However, functional symptoms can also develop in progressively degenerative neurologic disorders, particularly in Parkinson disease (Wissel et al 2018). This observation also emphasizes that functional neurologic disorder can co-occur with traditional neurologic disorder, eg, functional epilepsy occurring with apparently self-uncontrollable epilepsy

associated with epileptiform waves on EEG (Sadan et al 2016).

The frequency of functional neurologic disorders has been estimated to be 1% to 15% of all presentations at neurologic clinics (Williams et al 2005; Ahmad and Ahmad 2016; Carson and Lehn 2016). Pediatric manifestations are relatively uncommon, especially before the age of 8 years (Ouss and Tordjman 2014); however, they may account for up to 15% of patients who are seen in pediatric neurologic clinics (Ferrara and Jankovic 2008). In 1 epidemiologic study of 405 patients with functional neurologic disorder, the leading manifestations were pain, negative motor symptoms (eg, astasia, paresis), vertigo, seizures (which can include positive motor phenomena such as clonic movements), and somatosensory disturbances (Lempert et al 1990).

According to Espay and colleagues, it is helpful to categorize the diverse manifestations of functional neurologic disorders according to either the part of the body involved or behavioral characteristics (Espay et al 2018).

Functional movement disorder affects predominantly 1 or more limbs while not walking. Functional paresis has an incidence of 4 in 100,000 (Stone et al 2010). In contrast, psychogenic functional parkinsonism (tremor, generalized weakness, rigidity) is much more rare (Lang et al 1995). Among psychogenic functional dyskinesias, action tremor is most common, followed by resting tremor, dystonia, bradykinesia, and myoclonus (Hinson et al 2003). Psychogenic palatal tremor has occasionally been observed. In contrast to movement disorders that characterize traditional neurologic disorders (eg, Parkinson disease), functional movement disorders typically originate acutely. However, as noted above, functional movement disorders may co-occur with traditional neurologic disorders. One finding is that Parkinson disease as diagnosed by Hoehn and Yahr diagnostic criteria, or abnormal 123I-ioflupane SPECT scanning (DATscan evaluation) consistent with Parkinson disease, was preceded by functional movement disorder in 26% of patients (Wissel et al 2018). At least for this movement disorder, functional movement disorder may be an inherent part of Parkinson disease, in the same way that rapid eye movement sleep behavior disorder may be an inherent behavioral part of Parkinson disease. Moreover, functional movement disorder may follow deep brain stimulator implantation for refractory parkinsonism (Breen et al 2018). These authors suggested that functional movement disorder resulted because of the patients unmet expectations for improved movement control. However, the report did not consider the alternative that direct brain injury that followed deep brain stimulator placement may have directly caused the functional movement disorder.

The conventional involuntary motor symptoms of Parkinson disease can improve with environmental modifications and therapeutic suggestions, and it can be aggravated by attention to symptoms, which may be regarded also as forms of functional movement disorder. Thus, freezing of gait can be aggravated by attention to walking and improved by distraction to high-contrast visual stimuli (Nutt et al 2011); gait speed can be increased when marching to an auditory metronome (Thaut et al 1996); rest tremor, rigidity, and bradykinesia can be reduced by placebo (Goetz et al 2000; Barbagallo et al 2018); and various adverse effects can follow placebo in drug studies (the nocebo effect), including aggravated dyskinesias (Stathis et al 2013). The latter may result of patients having been informed of potential adverse effects, as required by the ethics of human drug research, despite their having received placebo medication.

The functional movement disorders include nonhyperkinetic disorders, including functional limb paresis that resembles stroke effects and functional dystonia, which involve sustained muscular contraction. Among orthopedic surgery practices, the “clenched fist” or the “psycho-flexed fist” occur. In contrast to hyperkinetic functional movement disorders, the functionally maintained flexion can develop gradually. In many instances, functional flexed limbs can lead to soft tissue changes over lengthy periods, including reversible maceration, atrophy, contracture, swelling, and joint dislocation (Simmons and Vasile 1980; Frykman et al 1983; Feldman and Duval 1997; Srivastava et al 2008; Ziegler et al 2008; Tonogai and Sairyo 2018). These disorders can reveal their functional character by symptom improvement with testing the passive range of motion during anesthesia and following counseling.

Axial functional neurologic disorder affects the body s core. This most often appears as gait disorder. A common subtype is astasia-abasia (from the Greek for inability to stand and walk). Blocq recognized that although the disorder resembled other forms of functional neurologic disorders (termed hysteria at that time), he recognized this nonetheless as a neurologic disorder (Blocq 1888). Astasia-abasia involves very unstable and marked lower extremity incoordination during walking, but with normal leg control in other contexts, for example, when asking the patient to walk backwards and move in bed. This category also includes abnormal posture, such as functional camptocormia, (ie, variable marked forward flexion of the trunk that in the consistent manifestation is a feature of Parkinson disease) (Skidmore et al 2007).

Oculomotor functional disorder is most often observed as convergence spasm (Kaski et al 2015). In this disorder, the patient looks medially with 1 eye (ie, cross-eyed), even when attempting to look at a distant object. This finding may give the false impression of abducens nerve palsy. The functional aspect of this disorder can be revealed by normal eye movements during optokinetic stimulation. Other oculomotor disorders include functional nystagmus, oscillopsia, and blepharospasm.

Functional sensory disorder can affect nearly any of the special senses. Some of the more common presentations include functional visual field deficits, including tunnel vision (the mapped visual field deficit that changes in relation to the distance of the object that is being looked at) (Espay et al 2018). A classic presentation is splitting the dermatome when applying a vibrating tuning fork to the middle of the forehead. Seldom discussed in this regard is Morgellons syndrome, also termed delusional parasitosis, in which patients complain of the sensation of crawling arthropods on or under their skin, and even the assertion that alien fibers are protruding through their skin, despite normal tissue examination (Krooks et al 2018). In contrast to these preceding disorders, establishing functional auditory disorders, contrasted with other auditory disorders, has not been well developed (Baguley et al 2016). However, functional perception of tones without clear source has been recognized in incidents of in mass psychogenic illness (Bartholomew and Pérez 2018).

To a greater extent than functional auditory disorder, functional disequilibrium disorders have been recognized. The disorder termed persistent postural perceptual dizziness, or PPPD, has been recognized (Espay et al 2018). This involves fluctuating dizziness that is aggravated by looking at complex or moving visual patterns, and often following inner ear disease (Dieterich and Staab 2017).

Functional cognitive disorder is a recognized variety of functional neurologic disorder. This involves the patient s complaint of intermittent cognitive impairment (most often memory loss), yet with generally normal performance on standard cognitive assessments. However, some cognitive tests are consistently found, including slowed cognitive processing speed and working memory (Teodoro et al 2018). In addition, patients with functional amnesia have a “reversed temporal gradient,” ie, with better recall of recent rather than remote events, opposite to progressive memory deficit that typifies neurodegenerative diseases such as Alzheimer disease (Harrison et al 2017). A striking variety of functional cognitive disorder is fugue, which involves inconsistent disruption of autobiographical knowledge, along with wandering from home for extended periods, unaccountably. In some instances, patients temporarily fail to recognize family members, nor recall their own identities.

Functional seizure disorder is more often termed psychogenic nonepileptic seizures or PNES. Functional seizure is the most common of paroxysmal functional disorders; functional syncope or drop attack also occur (Hoeritzauer et al 2018). Functional seizure involves abrupt, episodic stereotyped movements and interruption of awareness, without epileptic waveforms on EEG (Asadi-Pooya 2017). Eye closure is common and characteristic (Chung et al 2006).

Mass psychogenic illness (MPI) is the currently preferred term for what was formerly called mass hysteria or mass sociogenic illness. Mass psychogenic illness is distinguished from the other functional disorders by their being contagious. Mass psychogenic illness occurs among individuals who are grouped in close settings, including school and the workplace (Bartholomew and Wessley 2002). The symptoms that have been reported include tics, low-frequency sound perceptions, vertigo, syncope, headache, tachypnea, tremor, abasia, disinhibited emotion, and inattention (Bartholomew et al 2012). Although many reports of mass psychogenic illness have involved populations that may be considered not sophisticated (children, people from developing countries), a notorious and controversial incident involved some of the American embassy employees in Havana, Cuba between 2016 to 2017 (Bartholomew and Pérez 2018). The mass psychogenic illness hypothesis was excluded in the initial report, but a convincing alternate explanation was not offered, and the authors did not appear to be familiar with presentations of functional neurologic disorders (Swanson et al 2018).

Dissociative identity disorder deserves special mention. This is another controversial illness, formerly termed multiple personality disorder. In this disorder, for the most part the symptoms do not involve neurologic deficits, but rather the presentation of entire “personalities,” ie, an array of habits and emotional characteristics that are associated with a distinct “identity” different from the patient s original personality (Brand et al 2016). The core personality is often unaware, or incompletely aware, of the other personalities, extending to amnesia of the experiences of the other personalities, thus overlapping functional cognitive disorder (see above). Dissociative identity disorder shares etiology with functional neurologic disorder in that it appears to be a response to severe stress. Moreover, dissociative identity disorder can include objective neurologic findings, including personality-specific suppressed visual evoked potentials

(Strasburger and Waldvogel 2015), nystagmus (Bhuvaneswar and Spiegel 2013), EEG (Birnbaum and Thomann 1996), and cerebral perfusion patterns (Schlumpf et al 2014).

Whereas functional neurologic disorder involves primarily on 1 symptom, the complaints in somatization disorder (or Briquet syndrome) involve multiple, typically autonomic dysfunctions and do not suggest well-defined neurologic or other medical disorders. Examples of such complaints include nonfocal abdominal pain, nonspecific chest pain, dysuria, backache, erectile dysfunction, food intolerance, and fatigue (Khouzam and Field 1999).

Factitious disorder is the fabrication of illness within oneself for personal attention rather than monetary or material gain. An unmet need to feel in control may be responsible in some instances. Neurologic illnesses that may be misrepresented by factitious disorder include meningoencephalitis, epilepsy, loss of consciousness, visual loss, speech disorder, limb paresis (Bauer and Boegner 1996), unilateral neglect during clock-drawing (Khan et al 2000), quadriplegia (Feldman and Duval 1997), or irregular breathing (Walker et al 1989). Personality disturbances attend factitious disorder, including aggressivity, impaired impulse control, suicidal threats, and self-destructive behavior. A history of aliases and changing of hospitals or physicians may occur. Factitious disorder may be triggered in children by a sudden change in lifestyle or other psychosocial trauma (Eden and McNinch 1992).

Factitious disorder associated with repeated hospitalization, particularly when the patient presents to a multitude of medical centers, qualifies as Munchausen syndrome (Asher 1951). Pseudologia fantastica is a characteristic finding (Pankratz and Lezak 1987). Not uncommonly, such patients endure repeated invasive procedures for diagnosis or treatment, to no avail. Munchausen syndrome has been reported to present as complex regional pain syndrome (formerly termed reflex sympathetic dystrophy) (Chevalier et al 1996), hemiplegia (Biver et al 1992), status epilepticus (Savard et al 1988; Christensen and Szlabowicz 1991), neurogenic bladder (Heimbach and Bruhl 1995), paraparesis (Lazar 1986; Kwan et al 1997), meningitis (Marchant and Brown 1990), metabolic encephalopathy (Strobel et al 1994), or syncope (Tellioglu et al 2000).

Munchausen syndrome by proxy is the fabrication of medical complaints in a dependent individual by another, usually by a mother for her underage child (Meadow 1977). The behavior allows the mother to have the social attention or improved feeling of importance that she feels she would otherwise not obtain, while also placing the child's health in peril. This is child abuse. A 9% mortality rate has been identified from literature review (Rosenberg 1987). The child undergoes needless evaluation that can be invasive, loses time from school or other activities necessary for maturation, suffers psychologic trauma from chronically being in a sick role, and may be harmed by unnecessary medication. To achieve the deception, the caregiver must have detailed medical knowledge. Nonneurologic manifestations of Munchausen syndrome by proxy may include applying iodine to the child's skin to simulate jaundice and adding menstrual blood to the child's urine to suggest hematuria (Baldwin 1994). Unfortunately, the diagnosis has been carelessly applied in the medical literature, extending to instances where the mother has sincere but mistaken beliefs concerning disease in the child (Stirling 2007).

The most common neurologic manifestation of Munchausen syndrome by proxy is alleged epileptic seizures because serious medical action may be prompted simply by false eyewitness testimony rather than from physical evidence or abnormal clinical exam. In a variation of Munchausen syndrome by proxy, a lawsuit in the United States has alleged that a pediatric neurologist fabricated the diagnosis of epilepsy in several hundred of his underage patients, with concomitant needless prescription of antiepileptic medications and implantation of seizure-control devices, to reap profits for himself and his hospital (Egan 2010). Other neurologic manifestations can include apnea, loss of consciousness, and in 1 instance, myalgic encephalomyelitis (MacDonald 1989). In contrast to the usual victim being a child, however, several case reports have indicated abuse of adults (Sigal et al 1986; Granot et al 2004), often involving covert poisoning by the caregiver. In 1 of these cases, a 71-year-old man had recurrent stupor for 2 decades that was reversed by intravenous flumazenil. The clinicians had become convinced the patient had a strange disorder that released an endogenous benzodiazepine, until they discovered that the patient s wife had confessed to surreptitiously intoxicating him (as well as her mother) with lorazepam. The investigators themselves confessed to having easily been misled by a beguiling, but ultimately false, hypothesis.

As with Munchausen syndrome by proxy, patients with Ganser syndrome do not originate complaints about their abnormal findings. The disorder involves the tetrad of recurrent obviously incorrect answers to questions despite intact speech comprehension (eg, 1 + 1 = “4;” how many legs does a duck have? = “8”), impaired arousal, functional somatic findings (eg, nonphysiologic distribution of somatosensory loss), and hallucinations (Ganser 1898). The first sign – incorrect answers (*vorbeigehen* [“passing over”] in Ganser s terminology) – suggests the simulation of dementia

or psychosis, and has been frequently used to diagnose Ganser syndrome even in the absence of the other original features. The 4 features may variably coexist from patient to patient (Sigal et al 1992), thus, questioning whether Ganser syndrome describes a genuine symptom complex. When inappropriate answers to questions appear prominently in illness without all of the other diagnostic features, clinicians either loosely diagnose “Ganser syndrome” (Parker 1989) or more properly report “Ganser symptom” (Miller et al 1997). Consistent with other functional neurologic disorders, Ganser syndrome may abruptly resolve with psychotherapeutic intervention (Dabholkar 1987). However, in other instances the symptoms may persist for months despite therapeutic interventions (Miller et al 1997) or the absence of obvious gain (Dalfen and Feinstein 2000), which challenges the impression that the confabulatory answers strictly result from an intention to simulate neurologic illness. Indeed, such responses considerably resemble behaviors encountered in a wide variety of cerebral disorders. Furthermore, Ganser syndrome is frequently associated with brain injury and in 1 instance followed an extensive cerebral infarction (Wirtz et al 2008). Consequently, confabulated responses to questions cannot by themselves indicate whether the disorder is functional.

Prognosis and complications

Because diverse factors may be responsible for functional illnesses, a simple statement concerning prognosis is not possible. Furthermore, research studies have used (and probably continue to use) inconsistent methods for diagnosing functional disorders (Miller 1999), thus, confounding metaanalysis at this point.

Functional movement disorders may endure for years and disrupt daily living activities and employment (Fine et al 2000; Steriopoulos et al 2000; Gelauff et al 2014). Only about one half of patients with functional tremor or paresis improve, generally in response to eliminating stressors and cultivating the patient s trust in the physician (Jankovic et al 2006; Gelauff et al 2014). Prognosis for improvement from functional dystonia is poor, but combined psychotherapy and physical therapy may be efficacious in some instances and, therefore, should be tried (Schrag et al 2004). Functional movement disorders are also costly, with an estimated annual cost of care exceeding $100 billion in the United States (Anderson et al 2007).

Functional seizures are likely to recur in patients with depression, personality disorders, a history of chronic abuse, or abnormal MRI findings (Kanner et al 1999). Approximately 30% of patients with functional seizures have no recurrence. Functional seizures may occur in pregnant women. One study has reported that some continue taking antiepileptic medication, despite having been admonished otherwise, or present to emergency rooms with nonepileptic seizures and receive intravenous anticonvulsants. Thus, they risk avoidable harm to the fetus (DeToledo et al 2000). It may be difficult to dissuade such patients from taking medications, due to heightened anxiety among family members who note increased seizure frequency when off the medication.

With the advent of fibrinolytic therapy for acute stroke and its hemorrhagic complications, the presentation of psychogenic hemiparesis or other stroke deficits becomes potentially dangerous if emergency medical personnel are unable to recognize the fabrication of symptoms. In 1 study, 4 out of 26 patients presenting to an emergency room with acute hemiparesis were found to have factitious disorder that was recognized by medical personnel, and fibrinolytic therapy was appropriately avoided, although not before 1 of the patients was enrolled in a neuroprotective stroke study (Hemphill and Chung 1999). However, current research has shown no harm from fibrinolytics given to persons presenting with acute stroke symptoms with unremarkable neuroimaging, including persons later diagnosed with functional stroke (Chernyshev et al 2010).

Munchausen syndrome by proxy is associated with a fatality rate of from 9% to 30% for involved children (Baldwin 1994).

Clinical vignette

A 43-year-old man presented to the hospital complaining of increasingly frequent seizures over the past few days. He reported seizure disorder for the preceding 25 years following thoracic surgery for a chest wound. He was new to the hospital and did not supply medical records but indicated he had been evaluated at multiple other hospitals throughout the United States for his seizures. He reported that phenytoin caused him to have urticaria, and valproic acid had been only temporarily helpful. Concurrent carbamazepine and gabapentin that was started 1 month earlier when the patient had been away on vacation seemed to have reduced the seizure frequency to 1 per day until the present admission.

The seizure disorder had begun while he was in the military service. He felt frustrated trying to obtain a pension through the Department of Veterans Affairs because 1 doctor had alleged that the seizures may have originated prior to his military service, due to an undocumented history of unexplained loss of consciousness during adolescence. He was upset with most other medical institutions' evaluations, and he felt that clinicians either had considered the seizures to be psychologic or had despaired with treating him due to lack of improvement.

Neurologic examination was normal. During the present hospitalization, he suffered multiple daily seizures, up to 4 per hour. Seizures consisted of tonic posturing of 1 of the upper extremities, followed by total body stiffening, lordosis, unresponsiveness to verbal stimulation, diaphoresis, and low amplitude axial contractions, for as long as 15 minutes. The patient was tachypneic during these episodes, with slight facial reddening. The contractions occurred with an irregular rhythm and sometimes were interrupted by a few seconds' suspension of movement before restarting. During these episodes, pupillary responses were preserved to light, and plantar responses could not be elicited. Vital signs recorded during the seizures indicated only mildly elevated blood pressure. The patient claimed to be amnestic for these episodes. Even prolonged seizures were followed a few minutes later by purposeful activity such as postural adjustments in bed, reaching for water, and coherent conversation. Intravenous lorazepam and diazepam did not reliably terminate these episodes. The patient was electively intubated and given continuous intravenous midazolam for 2 days, which produced profound sedation. EEG during this time was unremarkable apart from intermittent theta and delta rhythm during light sleep that was thought to be possibly due to benzodiazepine treatment. Extubation and withdrawal of the midazolam were followed by resumption of the seizures.

His gabapentin was continued at 1500 mg/day, the carbamazepine was raised to 1000 mg/day, and valproic acid was restarted at 1500 mg/day (following rectal loading). Nonetheless, seizure character, frequency, and duration did not change, despite technically "therapeutic" serum levels of the latter 2 drugs. Brain MRI scan, arterial blood gases, complete blood count, and serum electrolyte and hepatic enzyme levels were all normal.

He underwent 3 days of continuous video-EEG monitoring. Review of the video tape recordings and simultaneous EEG by 2 neurologists identified multiple seizures but no abnormal EEG activity. The consultant neurologist conferred at length with the patient and his fiancée and said that he had "nonepileptic seizures" whose basis could not be explained. The patient worried that he would be considered psychotic, but the neurologist reassured him that this was not so. The couple was pleased to review several video tape recordings of the seizures, in which the neurologist indicated the normal EEG. The patient was advised to continue present oral medication, as the fiancée felt that the episodes had become less violent. The neurologist indicated that he would have office visits with the patient several times a year and might reduce the medications if there were no overall worsening. The neurologist indicated that because vital signs and EEG were unremarkable during these episodes, emergency treatment was not needed for these seizures. The couple left the hospital satisfied with the attention of a qualified clinician who had taken a serious interest in the patient's problems.

Comment. The patient's history of evaluation at multiple medical centers and 25 years of uncontrolled generalized seizures with normal neurologic findings suggested functional seizures. This was supported by normal brain imaging, extended EEG recording, and observations of prolonged but irregular convulsive movements that were interspersed by rest periods and followed by rapid resumption of environmental interaction. Although the origin for these seizures was unclear, they may have been reinforced by the patient's failure to obtain a military pension. Because doctors had told him he had a psychologic disorder or had failed to maintain clinical follow-up, there was no chance for therapeutic success. Confidence in the neurology consultant was established through careful attention to the patient's complaints, frank discussion and review of the EEG findings, and avoiding diagnosing psychopathology or referral to a psychiatrist. Although there was no orthodox indication for antiepileptic medication, there appeared to be no short-term harm from its continuation. More important, the neurology consultant established clinical rapport, which so far had been lacking, through assuring regular follow-up and sincere interest in the patient's problems. This foundation is essential before exploring medication tapering and psychosocial stresses and to avoid the patient's possibly turning to unqualified clinicians for help.

Biological basis

Anatomic localization

The few studies to date on structural MRI in functional neurologic disorders have been inconsistent, which were based on statistical analyses at the group level, rather than at the level of the individual patient. Thus, increased cortical grey

matter was observed in patients with functional seizures (left insula, bilateral medial frontal areas, left supplementary motor area, right superior temporal gyrus, motor cortex) and functional hemiparesis (bilateral premotor cortices) (Aybek et al 2014; Ristic et al 2015; Kozlowska et al 2017; Mcsweeney et al 2018). In contrast, decreased cortical grey matter was reported in functional seizure (right motor area, bilateral cerebellum, bilateral precentral), functional motor disorder (left anterior cingulate cortex), and dissociative identity disorder (insula, anterior cingulate, parietal, temporal, and orbitofrontal cortices) (Aybek et al 2014; Labate et al 2012; Perez et al 2018; Reinders et al 2018). Therefore, at present no signature brain MRI finding characterizes functional neurologic disorders.

According to a comprehensive review of the literature, there is no preponderant involvement of 1 side of the body or the other (Stone et al 2002a). Thus, these reports do not point to a characteristic focal structural brain finding that is associated with functional neurologic disorders, even though statistically significant alterations of brain morphology have been observed, which are different from healthy individuals.

Pathophysiology

The etiology, if there can be only one, for functional neurologic disorders is highly controversial and unresolved. The past century of thought about functional neurologic disorder centered on the hypothetical mechanism of “conversion” that was purported by Sigmund Freud. According to this hypothesis, repressed psychological distress was ameliorated by subconsciously conversion to an involuntary bodily symptom. There is, however, no scientific evidence for this process, and indeed, the hypothesis is untestable (Carson et al 2016).

A major difficulty with positing a single pathophysiological etiology is that among all patients with functional neurologic disorders, there are many exceptions to “rules” or patterns that have been recognized. Thus, for example, psychiatric disorder has been invoked to cause functional neurologic disorders because this is common among the patients, and yet many patients do not have psychiatric complaints. Similarly, psychiatric disorder is common in stroke, Parkinson disease, multiple system atrophy, dystonia, and epilepsy (Fiest et al 2013; Lehn et al 2014; Mele et al 2018; Zhang et al 2018), but it has not been apparent that psychiatric disorder causes these neurologic disorders. The absence of distinct biomarkers in functional neurologic disorders that are available on clinically ordered brain MRI scan or EEG is also true for many other neurologic disorders, such as Parkinson disease, essential tremor, dystonia, and migraine.

Functional disorder is frequently preceded either immediately or within a few weeks by either psychological or physical trauma. Seldom recognized, but pertinent, is that functional disorders appear in other animals following emotional or physical trauma (Anonymous 1898; Higier 1898; Sanger and Handy 1962; Keehn 1982). These reports involved reversible neurologic changes that were not associated with neuroanatomical changes. A related response to threat to survival is “learned helplessness”—sudden immobility—in laboratory animals (Seligman 1972). Clinical studies in recent years have reported tonic immobility in victims of sexual assault that resembled learned helplessness (Marx et al 2008; Möller et al 2017). An insight by Kretschmer was that the recourse by various vertebrates to hyperactivity or feigning death as an instinctive antipredator mechanism could be the basis for diverse functional neurologic disorders in humans in response to perceived threat (Kretschmer 1937).

A comprehensive literature review by Edwards and colleagues suggests that for certain individuals, a traumatic event, either psychological or physical, markedly disrupts the patient s predictions for the quality of the external or internal world following the patient s actions (Edwards et al 2012). Such a distorted prediction of sensory experience may thus apply to functional blindness, anesthesia, or pain. Furthermore, the altered expectation for sensory input may pertain to kinesthesis as well, and thus, cause patients to anticipate that part of their body will have either degraded kinesthesis (the situation following paralysis) or fluctuating kinesthesis (eg, following tremor, dystonia, or tonic seizures). The patient may then unconsciously adjust limb movement to match prediction, and thus avoid a perceptual mismatch. Support for this comes from the observation that patients with idiopathic unilateral facial paresis (Bell palsy) may also show ipsilateral limb hemiparesis (Keane 1993), even though the locus of injury is physiologically incompatible with hemiparesis. Keane suspected that hemiparesis with Bell palsy arises because patients assume they have a stroke, and so ipsilateral weakness is to be expected. Keane reported that such patients hemiparesis can be resolved simply with counseling. In everyday life, involuntary adjustment of perceptual gain may occur when, for example, we “ignore” pain from an injury when we have to focus on escaping danger. As another example, we may become so preoccupied in our thoughts as to fail to recognize familiar passersby who technically happen within visual field. On the other hand, Edwards and colleagues cite experimental evidence that ordinary perception of a mildly noxious stimulus may be aggravated by a preceding aversive stimulus (Edwards et al 2012). Hence, the revision of perceptual gain may not be all that unusual in special circumstances. In the case of functional illness, however, such

resetting is greater, enduring, and difficult to voluntarily reset.

Marshall and colleagues evaluated a patient with psychogenic hemiparesis following unspecified functional trauma (Marshall et al 1997). Attempted movement of the hemiparetic limb was associated with increased metabolic activity of the contralateral orbitofrontal and anterior cingulate areas, which are thought to be involved with motor inhibition. The affective response to painful stimuli can be modulated by hypnotic suggestion. This response has been associated with altered activity of the anterior cingulate area in human volunteers (Rainville et al 1997). Patients with functional hemiparesis, regardless of symptom laterality, may show decreased activity in the left dorsolateral prefrontal cortex on PET scanning during voluntary movements (Spence et al 2000). The failure by such patients to activate motor-related frontal cortical areas while observing a moving hand (unlike healthy individuals) suggests a deficit of motor imagery (Burgmer et al 2006). Another study has reported decreased thalamic and caudate nucleus metabolism contralateral to the side of functional hemiparesis, which resolved when the hemiparesis resolved (Vuilleumier et al 2001). This observation may be related to the structural imaging finding of bilateral thalamic atrophy in chronic functional hemiparesis (Atmaca et al 2006; Nicholson et al 2014). In contrast, functional limb paresis has been found to be associated with hypertrophy of the premotor cortices, which may reflect the effects of increased motor inhibitory activity (Aybek et al 2014).

Similarly, reduced contralateral somatosensory cortical activity to peripheral tactile stimulation was demonstrated on fMRI in patients with unilateral functional somatosensory loss (Ghaffar et al 2006). In contrast, cortical activity in the deficient areas occurred when the patients underwent bilateral tactile stimulation; the authors speculate that this was because the patients were distracted from maintaining inhibited awareness of stimulation on the affected side. It was suggested from this evidence that the frontal cortex may activate the nucleus reticularis of the thalamus, which secondarily inhibits primary somatosensory cortex processing in individuals with functional somatosensory loss (Heilman and Watson 2009).

Increased metabolic activity in frontal and subcortical areas may appear in patients with suspected functional hemianopia, which suggests a focal cerebral inhibitory process in functional visual loss similar to that postulated for functional somatosensory loss (Werring et al 2001). An interesting case report indicated psychic blindness associated with specific personalities in dissociative personality disorder. Visual evoked responses were absent only in the personalities that were blind (Waldvogel et al 2007). The authors suggested that strong, “top-down” cerebral inhibition (perhaps mediated by the thalamus) could have this remarkable neurophysiological effect. Similarly, healthy subjects can become experimentally trained to forget emotionally upsetting scenes (Depue et al 2007). Such forgetfulness is associated with right inferior frontal increased metabolism, coupled with decreased metabolism in memory-associated and vision-associated brain areas. A related case report of a woman with selective loss of autobiographical memories from childhood demonstrated different areas of brain activity on fMRI when well-recalled episodes were probed compared to poorly recalled episodes (Botzung et al 2007). Combined, these results suggest that circumstances that lead to psychologic repression (either of memories, voluntary movements, or sensations) involve direct participation of inhibitory brain areas.

A case report of functional aphonia demonstrated sustained resolution following repetitive transcranial magnetic stimulation to the right motor cortex, whereas such stimulation to the left hemisphere had been previously unsuccessful (Chastan et al 2009). This finding, which deserves confirmation, suggests that interhemispheric inhibition may be a feature of functional disorders.

A case report identified astasia-abasia along with diffuse, continuous limb tremor 2 months after temporal lobectomy for refractory epilepsy (Arabi et al 2012). Although the patient s motor disorder dissipated following intensive psychotherapy, which suggested a psychological basis for the disturbance, of note was that the investigators did not identify any psychologic stressors, but did find that the patient also appeared depressed post-surgery. The depression also reduced with psychotherapy. A wide variety of somatic disorders as well as depression have been reported to follow temporal lobectomy (Naga et al 2004; Foong and Flugel 2007; Maixner et al 2010). Therefore, temporal lobectomy may elicit neurophysiologic changes that can give rise both to affective disorders and to behavioral changes suggesting functional disorder, but without the stressors that have usually been associated with the latter. The favorable responsiveness of an apparently neurophysiologic disturbance to psychotherapy is unsurprising, in view of the similar neurophysiologic changes that can be demonstrated to follow either successful psychotherapy or pharmacotherapy for obsessive-compulsive disease (Schwartz et al 1996). This provides evidence that successful psychotherapy can directly change brain physiology.

Abnormal gating of sensory experiences was described above. Pareés and colleagues found that individuals with functional dystonia did not have the typical reduction in judgement of the force when they applied the stimulus to themselves compared to when a robot applied the stimulus (Pareés et al 2014b). This altered judgement in the force of self-touching was considered to reflect a lack of appreciation of self-agency in individuals with seemingly automatic limb movements. It is not clear whether disturbed cerebral inhibition or excitation is involved; further research will undoubtedly evaluate these possibilities.

Increased functional connectivity on fMRI evaluations has been observed between emotionally related brain areas and motor-associated areas in persons with either functional dyskinesias, gait disturbances, or seizures (Voon et al 2010; van der Kruijs et al 2012). These results suggest, in contrast to the inhibitory mechanisms above, a vulnerability to excessive activation of motor areas by emotional stimuli. Consequently, it is presently unclear whether a unitary hypothesis regarding regional cerebral activation patterns and functional neurologic disorders can be advanced.

Epidemiology"

The frequency of functional neurologic disorders has been estimated to be 1% to 15% of all presentations of neurologic diagnoses (Williams et al 2005; Ahmad and Ahmad 2016; Carson and Lehn 2016). There is a substantial predilection of functional neurologic disorders for women (Lempert et al 1990; Bahtia and Schneider 2007; Chastan and Parain 2010; Sadan et al 2016). Peak incidence tends to be in young adulthood, and there is a high association with anxiety disorders and depression (Lempert et al 1990; Pakalnis et al 1991; Aldenkamp and Mulder 1997; Binzer et al 1997). Nonetheless, psychosocial stressors are not invariably identified in persons with functional neurologic disorders, whereas preceding physical trauma is frequently identified (Pareés et al 2014).

Differential diagnosis

Although the functional neurologic disorders resemble highly deleterious medical illnesses, examination for which should routinely be conducted in the work-up, functional neurologic disorder should *not* be considered a diagnosis of exclusion (Drapier and Vérin 2012; Stone 2014). Instead, the well-trained and insightful clinician should be highly suspicious of such disease in the presentation of attentionally-modifiable disorder or of a disorder that superficially resembles conventional neurologic disease but that shows markedly atypical characteristics (eg, astasia-abasia, concentric visual field loss that changes visual angle when testing with stimuli at different distances from the patient). A history of psychiatric disturbance or concurrent personality disorder raises the suspicion for functional disorder, but these do not by themselves rule out structural, physiologic, or infectious pathologic processes, nor do they conversely rule in functional neurologic illness.

The following can be considered only an incomplete overview of conditions that may be confused with functional disorders because new examples are published frequently.

Functional disorder is suggested in part by the variability of the symptoms, particularly for impairments that are chronic, such as paralysis or visual loss. Nonetheless, the clinician must recall that "traditional" neurologic disorders, even if chronic and "stable," may also have variably presentations. Thus, for example, the comprehension deficit in aphasia may vary in relation to how often the patient is tested: it is usually at its best on initial evaluation and then worsens (Beyn 1958). Tremor (as in Parkinson disease or essential tremor) typically abates during sleep and, thus, is clearly influenced by the extent of conscious arousal. Moreover, tremor can be dampened through voluntary reaching. A remarkable instance of voluntary, nonfunctional palatal tremor has been described (Biller and Espay 2013). Postanoxic action myoclonus and Parkinsonian tremor may be aggravated by anxiety and may be abate when the individual is emotionally calm (Rollinson and Gilligan 1979; Apartis 2014). Tics may be voluntarily suppressed, but only for a limited period (Koller and Biary 1989). Fatigue, intoxication, medication effects, or systemic infection may aggravate nearly any central nervous system disorder. Such disturbances are distinguished from functional disorders by their adherence to a replicable association with toxic agents, fatigue, or sleep deprivation. In contrast, functional disorders fluctuate over several seconds to minutes and sometimes in relation to who is observing (including the patient himself) or if the patient is aware of being observed.

Primary progressive aphasia is a dementing illness that involves gradual, irreversible impairment in language, with comparative retention of other cognitive functions. This may be mistaken for functional neurologic disorder but can be recognized by abnormal brain SPECT imaging (Philbrick et al 1994). Acute aphasia due to neurosyphilis has been mistaken for functional illness, due to the absence of corroborative CT scan abnormalities (Boyle and Zafar 1995).

Complex regional pain syndrome is a controversial disorder that was formerly termed either “reflex sympathetic dystrophy,” “shoulder-hand syndrome,” “Sudeck dystrophy,” or “algodystrophy.” It is frequently mistaken for malingering. In this disorder, a usually catastrophic physiologic event (traumatic limb injury, spinal cord injury, stroke, myocardial infarction, or extensive burns) precipitates severe pain in a limb, sometimes associated with focal autonomic changes (altered perspiration, hair growth, skin color, or edema) and in late stages with focal osteomalacia. In a strange alternation of cause and effect, 1 case of complex regional pain syndrome included unilateral leg edema and was apparently triggered by acute severe emotional distress (Grande et al 2004), rather than the behavioral change occurring in response to somatic illness. Afflicted individuals, particularly children, may manifest "incongruent pain," ie, cheerful affect and lack of protective measures, despite maintaining that the pain is severe. It is unfortunate that for such a frequently incapacitating illness, consistent objective laboratory test findings have not been identified. The disorder responds to the combination of physical therapy and psychotherapy, which best should be provided by hospitalization with the care of a multidisciplinary team (Sherry et al 1999).

A wide variety of perceptual disorders associated with brain injury may show a "covert" recognition of stimuli under special circumstances, despite the overt denial of awareness. The existence of intact but unconscious processing is indicated by autonomic changes (altered heart rate, respiration, skin conductance, pupillary size, etc.) for certain stimuli (eg, familiar faces or voices), despite overt denial. This has been termed "guilty knowledge" due to the similarity to the autonomic signs of criminality during polygraphy in legal investigations (Bauer 1984). Nonautonomic behaviors may also signify retained sensory processing despite an overt failure of recognition. Disorders with dissociations between conscious and unconscious sensory processing include prosopagnosia (impaired face recognition) (Bauer 1984), cortical deafness (Engelien et al 2000), visual agnosia (Milner et al 1991), acquired alexia (inability to read meaningfully), in which patients may still have intact lexical decision (eg, intact ability to categorize uncomprehended words) (Coslett and Saffran 1989), and unilateral neglect, where stimulus size may affect bisection judgments despite unawareness of peripheral stimulus extent (Marshall 1998). Such findings demonstrate that overt disability may coexist with covert ability, due to the failure of intact cognitive processes to attain conscious awareness following certain forms of brain injury. Although these disturbances superficially seem to be feigned, they are distinguished from functional disorders by their consistent manifestations and the usual absence of any gain from their appearance. In addition, they are almost invariably associated with focal structural brain lesions.

Contrary to what one might expect, patients with functional seizures only rarely have coexisting epilepsy (Benbadis et al 2001; Mendez et al 2003). Because functional seizures occur only sporadically, they can be difficult to distinguish from epileptic seizures, particularly if functional seizures are associated with interictal spike discharges on EEG (a normal finding in some healthy individuals). However, it is noteworthy that the occurrence of objective brain abnormalities (epileptiform potentials on EEG, structural brain MRI abnormalities) has been found in as many as 22% of persons with functional seizures (Reuber et al 2002a). Persons with functional seizures also have higher resting serum cortisol levels than do healthy control subjects (Bakvis et al 2010). The average time to diagnosis of functional seizures may be 7 years due to this difficulty (Reuber et al 2002b). Functional seizures may be suggested by absent response to antiepileptic drugs, but nonetheless certain functional seizures also occur (see below). Epileptic seizures reportedly may be aborted by passive painful dorsiflexion of the palm (Carasso et al 1992). Consequently, the cessation of seizures immediately following specific environmental stimuli cannot by itself imply a functional disorder.

Complex partial seizures originating from the frontal lobe may result in convulsive episodes not characteristic of generalized seizures, but suggesting functional seizures, including kicking, complex arm movements, tongue protrusion, and pelvic thrusting (Williamson et al 1985; Kanner et al 1990; Geyer et al 2000). Conscious interaction with the environment is possible with frontal lobe seizures (Hsieh and Pearl 2009). The diagnosis of epileptic seizures is benefited by noting the stereotypical features of the phenomena, supplemented by EEG. However, scalp EEG may be insensitive to these seizures, which, therefore, may require depth electrodes instead. The retention of consciousness in generalized seizures has been occasionally reported (Bell et al 1997). Therefore, the retention of consciousness during seizures does not by itself imply functional disorder.

A variety of functional seizures occur, which likely reflects their diverse etiologies (LaFrance and Devinsky 2004). These paroxysmal disturbances may include panic attacks, intense anxiety, dissociation, episodic dyscontrol, and other psychiatric disorders that can be confused with epileptic seizures due to their associated autonomic alterations (Alper et al 1995). Comprehensive psychiatric evaluation is best to diagnose these disorders.

However, a few etiologies for functional seizures have also been recognized. Strokes particularly involving the

brainstem, may provoke convulsive movements without ictal EEG changes (Saposnik and Caplan 2001). The disorder is rarely seen and its mechanism is not understood. Neuroglycopenia secondary to pancreatic insulinomas may present with nonepileptic attacks that include confusion, lethargy, bizarre behavior (eg, fidgeting, shouting, falling), limb or whole body shaking, amnesia, incontinence, autonomic hyperactivity, and prolonged postepisode confusion. EEG abnormalities typical of seizures are absent, although slowing may appear. The onset is insidious in midlife, with a female preponderance. The condition is easily mistaken for other neuropsychiatric disorders (Bazil and Pack 2001; Graves et al 2004). It can be diagnosed by the pattern of absent response to antiepileptic medication, attacks during fasting, concurrent hypoglycemia, and resolution with glucagon. Excision of the tumor resolves the illness.

Increasing attention has been drawn to the diagnostic confusion that can attend antibody-mediated epilepsies, in particular, NMDA-receptor (or NMDAR) encephalitis. In the latter disorder, there is a frequent occurrence of bizarre, rapidly fluctuating behavioral disturbances that may include paranoia, delusions, hallucinations, suicidality, and apparent seizures, despite generally unremarkable standard structural brain imaging and video EEG recording. The predominant occurrence of such findings in young women, which in many cases is associated with an ovarian teratoma, may lead to the mistaken impression of functional seizures (Labate et al 2009; Caplan et al 2011). When a teratoma is present, the disorder often markedly improves with surgical removal of the tumor and immunosuppressant medication.

Confabulatory responses to questions raise the possibility of Ganser syndrome and, thus, possible functional dementia. Functional illness should be suspected when the illness onset is abrupt and without identifiable physiologic causes and if sources for secondary gain can be identified. On the other hand, confabulatory responses in the setting of brain injury or without accompanying features of psychosis suggest either serious structural brain injury or schizophrenia.

Psychiatric delusion may cause abnormal somatic function without demonstrating a contributing structural or physiologic etiology. In 1 instance, a young woman chronically kept her left hand in a tight fist, causing edema, contracture, and cellulitis (Srivastava et al 2008). The patient believed that her hand held treasure and did not want anyone else to have it.

Opposite to functional neurologic disorder is anosognosia, the denial of neurologic deficit (eg, hemiparesis, amnesia, aphasia) (Babinski 1914). A classic example of anosognosia is Anton syndrome, the denial of blindness (Forstl et al 1993). Although a psychologic defense mechanism is plausible in certain cases following cerebral injury, the associated cognitive deficits suggest that such denial more often results from defective cognitive functions essential for self-monitoring (McGlynn and Schacter 1989). Acute brain injury may rarely be associated with denial of ability (eg, vision) (Hartmann et al 1991). Associated cognitive deficits and its appearance following focal structural brain injury suggest that this deficit also is an involuntary impairment of self-awareness rather than a willful defense mechanism.

Diagnostic workup

As with any complaints of bodily disorder or functional disability, or the observation of behavioral changes that suggest specific neurologic disorders (eg, seizures), patients should undergo the usually indicated workups, unless the patient has been recognized by staff to have presented frequently with the same complaints without objectively confirmed abnormalities. In such instances, the reliability of previous workups should be reviewed before deciding that the illness is functional. Otherwise, the suspicion of functional disorder should not preclude a conventional medical workup because functional disorder does not in itself exclude medically treatable disorders. The failure to identify structural brain lesion in a neurologic disorder should not lead to suspecting functional disturbance if the presenting complaint is consistent and the patient does not have evidence for psychiatric disturbance (Di Renzi et al 1997). It should be noted that a growing body of literature has identified “MRI-negative” stroke on standard MRI in as many as one third of patients with suspected stroke (Makin et al 2015), which may indicate crucial limits to routine neuroimaging for acute brain illness. In contrast, abnormal brain imaging findings or other objective findings are not inconsistent with functional disorder (Fenelon et al 1991). In addition, severe medical problems may directly follow functional disorders, such as chronic muscle atrophy in functional quadriplegia (Feldman and Duval 1997) and recurrent severe head injury in functional falls (Voermans et al 2005). Functional fixed dystonia of the hand, resulting in chronically maintained clenching of the fist (“clenched-fist syndrome” or “psycho-flexed hand”), may lead to local swelling and other chronic soft tissue changes (Srivastava et al 2008; Ziegler et al 2008). Essentially, the clinician must decide whether objective study results are consistent with the presenting complaint for the purpose of directing management. Similarly, the pattern of complaint reporting and presentation (numerous hospitalizations, various presentations) should be considered when deciding whether to incorporate behavioral management in the treatment.

Functional disorder may be suggested by a history of frequent changes in medical practitioners, particularly when workups failed to identify the disorder, or if historical details are vague. Multiple surgical scars may suggest Munchausen syndrome (Asher 1951). Fluctuation of a deficient function, such as limb use or vision, suggests functional disorder (Reuber et al 2005; Stone et al 2005). Paroxysmal disorders (eg, epilepsy) that appear to be triggered by suggestion or particular social settings also increase suspicion. Similar concerns apply to malingering or factitious disorder (Bauer and Boegner 1996). The suspicion for factitious disorder should be raised in patients who present themselves for diagnostic workup despite a longstanding history of repeated invasive diagnostic measures (as evidenced by multiple surgical scars). A history of using aliases also should alert suspicion for factitious disorder.

Although studies have indicated a high incidence of history of sexual abuse in persons with functional neurologic disorders (van Merode et al 2004; Sar et al 2009), Stone and colleagues advise deferring inquiry for sexual abuse to an expert in this area because of the considerable amount of skillful evaluation and discussion that would be needed (Stone et al 2005).

Based on the presenting symptom, a large number of clinical neurologic tests may improve diagnosing functional neurologic disorders. Stone and colleagues offer a brief overview of these tests and their degree of reliability (Stone et al 2011). The following provides some considerations with diagnosing specific functional neurologic phenomena.

Nonparoxysmal functional disorders are suggested by fluctuating manifestations. Nonetheless, tremors that are commonly thought not have a functional etiology may also diminish with distraction (McAuley et al 1998). This observation suggests that conventional tremor may in fact have a functional basis. Variable muscle recruitment during contractions or irregular intervals from stimulus to jerk onset are consistent with functional myoclonus or functional startle (Thompson et al 1992). Dystonia accompanied by pain and variable presentation suggests functional disorder (Lang 1995). In a large study, 37% of patients with fixed-hand dystonia (most of whom were women) fulfilled diagnostic criteria for functional etiology (Schrag et al 2004). This was considered by the authors to be underestimated, owing to the presumed difficulty with distracting the patient from maintaining the fixed posture of the limb (as compared to, for example, functional tremor).

Patients who present with functional motor disorders (including limb paresis or general motor disability) are more likely to have depression, personality disorders, susceptibility to hypnotic suggestion (Maldonado and Jasiukaitis 2003), and a history of psychiatric treatment relative to patients with conventional motor disorder (Binzer et al 1997). The occurrence of asynchronous rhythmic oscillations in different parts of the body (eyes, neck) can be self-induced through practice (Lee and Gresty 1993) and, therefore, does not exclude functional tremor or nystagmus.

In functional seizures that resemble complex partial seizures, recall of events during the ictus is usually better than in complex partial seizures (Bell et al 1998). However, it is unclear whether this observation can be reliably used on a case-by-case basis, unless patients who manifest complex partial seizure signs demonstrate considerable recall of ictal events. To evaluate functional seizures, routine EEG may be misleading because some seizures (particularly those that emanate from the medial temporal lobe) may be difficult to detect through scalp electrodes. Furthermore, as is well known among clinicians, the interictal EEG may be normal in true seizure disorder. On the other hand, the provocation of seizures through suggestion, particularly during EEG-video monitoring, suggests functional seizure disorder in the absence of characteristic spike-wave discharges during the ictus. Although provocation has classically been performed through intravenous saline injection (informing the subject that he may develop a limited seizure following injection) (Luther et al 1982), this is ethically objectionable because it requires the clinician to mislead the subject (ie, a saline injection is unlikely to provoke so great an electrochemical alteration in salt balance as to risk seizures). Accordingly, Benbadis and colleagues recommend using noninvasive measures that are already known to induce seizures in truly epileptic patients with reduced seizure threshold, such as photic stimulation or hyperventilation. The patient is told that such maneuvers can trigger an attack, and the EEG examination is intended to evaluate this possibility. This approach is reported to be as sensitive as intravenous saline (Benbadis et al 2000). However, the response to provocation may not accurately reflect the characteristics of the seizures that actually appear in the patient (Gates 2001), and so one must be careful with this technique. The foregoing concerns notwithstanding, intravenous saline was found by 1 research group to be the only way to establish a diagnosis in 32% of their patients considered to have functional seizures (Ribaï et al 2006).

Weeping at the end of episodes suggests nonepileptic functional seizures (Bergen and Ristanovic 1993). This can be fairly reliably found on EEG-video monitoring. Bergen and Ristanovic encountered 10 patients in 4 years who wept following functional seizures. In contrast, crying induced by epileptic seizures is rare. Eye closure during seizures is

remarkably reliable at distinguishing functional seizures (where eye closure is continuously present) from epileptic seizures (where eye opening usually occurs) (Chung et al 2006).

Assessing the relative change in heart rate from resting baseline can reliably distinguish epileptic seizures from functional seizures, whether convulsive or not. Functional seizures cause minimal change in heart rate (at most 7%), whereas epileptic seizures increase the heart rate by at least 17% (on average) (Opherk and Hirsch 2002).

During tilt-table testing, individuals who are considered to have functional syncope are highly likely to close their eyes on the onset of apparent syncope, in marked contrast to persons who have cardiovascular syncope (Tannemaat et al 2013).

Commonly seen in functional coma are normal vital signs, toxicology, complete blood count, and serum electrolytes, with normal tendon, pupillary, and vestibulo-ocular reflexes, normal muscular tone, and flexor plantar responses. In true coma the patient does not prevent his hand from striking his face when it is released from grasp above the face. In contrast, in functional coma (or mildly impaired arousal), the hand avoids striking the face. In the presence of such findings, cerebral neuroimaging is unlikely to be necessary. Ice-water caloric vestibular testing (after ascertaining integrity of the tympanic membrane) may induce nausea so severe as to provoke well-integrated limb or postural movements or arousal. Similar defensive maneuvers may follow presentation of concentrated ammonia ("smelling salts") to the nose. Failure to demonstrate integrated responses to noxious environmental stimuli should prompt evaluation with EEG to rule out nonconvulsive status epilepticus.

Assessing functional visual disorders must be tailored to the presenting symptom. Helpful tests for evaluating functional visual loss include observing the patient's pupillary and facial responses to silently presenting a mirror. Functional blindness patients often attend the clinic wearing sunglasses, which may be a useful sign (Bengtzen et al 2008). Blink to visual threat may signify integrity of visual awareness (as long as one prevents corneal stimulation through gusts of air). However, the absent blink to threat does not strictly imply visual loss but may be due to inattention (Denny-Brown 1950). It is conceivable that patients who anticipate testing methods for functional visual loss may willfully prevent reflexive eye movements such as blink to threat. Further recommendations for assessment are available (Bose and Kupersmith 1995). Automated assessment of visual fields unfortunately does not distinguish between functional and structural visual disorders (Smith and Baker 1987). Similarly, visual evoked responses can be episodically inhibited in dissociative personality disorder in individuals with personality-dependent visual loss, which suggests that this technique is not reliable when evaluating suspected mechanisms of visual loss (Waldvogel et al 2007).

Functional hemianesthesia may be suspected when the application of a vibrating tuning fork to the forehead is not felt past the midline (Khan et al 2000; Gilman 2002).

Functional hemiparesis of the lower extremities has been traditionally diagnosed through examining for the Hoover sign (Hoover 1908). This is tested by having the patient supine in bed or the examination table while the examiner places both hands beneath the patient's heels. When asked to force the feet down (essentially, hip extension), only 1 leg moves. Then the examiner tests leg elevation in the nonparetic side while keeping the hand beneath the paretic heel. In true hemiparesis, upward mobility of the nonparetic leg causes no forceful movement of the paretic limb. In functional hemiparesis, in contrast, the "paretic" leg will move down forcefully, to stabilize the upward movement of the other leg (Stone et al 2002c). Although useful, this sign is subjective and, thus, may be difficult to assess reliably. This difficulty may be overcome through using strain gauges attached to the lower extremities and even the upper extremities and calculating the ratio of voluntary activation versus involuntary activation (through mobilizing the opposite limb) (Ziv et al 1998). Although more objective, this measure is elaborate and, thus, unlikely to become popular. It should also be noted that concurrent pain with leg movement may confound interpreting the test, the test s reliability has not been assessed, and test performance among individuals with defined neurologic disease (eg, multiple sclerosis) has not been evaluated (Stone et al 2002c).

In contrast to the Hoover sign, a reportedly more reliable test is to assess whether there is voluntary stabilization of the “paretic” leg while the unimpaired leg is actively abducted against the examiner s resistance (Sonoo 2004). In nonfunctional hemiparesis, the paretic limb hyperabducts when the opposite leg abducts against resistance. In functional hemiparesis, the unimpaired leg hyperadducts when the “paretic” leg is required to abduct against resistance. Blinded study is needed to confirm the validity of this method.

The suspicion of functional paraparesis may be confirmed with the Spinal Injuries Center (SIC) test (Yugué et al 2004). In this test, patients who demonstrate inability to elevate their legs have their knees passively flexed while they are recumbent. The examiner gently removes contact. The maintenance of flexed posture is a positive test and has a reported 100% sensitivity and 98% specificity.

Unilateral functional sensorimotor deficit (resembling stroke) may be associated with increased N140 component of the event-related potential following somatic stimulation on the affected side (Maldonado and Jasiukaitis 2003). This asymmetry reverses following therapeutic hypnotic suggestion.

Functional paraplegia is frequently accompanied by dense sensory loss that does not respect dermatomal boundaries but rather the "underwear line" (Stone et al 2005). Lower extremity tendon reflexes, cremasteric reflexes, plantar responses, anal sphincter tone, and limb muscle tone are usually normal. Case studies demonstrate that normal motor evoked potentials support the diagnosis of functional paraplegia (Pillai et al 1992). Integrity of muscle tone (and, hence, some degree of voluntary muscle control) may be demonstrated by flexing the hip and knee while the patient is supine and noting whether the leg slowly falls to 1 side, if at all. The clinician may surreptitiously monitor for subtle postural adjustments of the lower extremities while pretending to attend to the patient's upper body. Monitoring during sleep (eg, by direct observation, video monitoring, or accelerometry) can disclose lower extremity movements that are asynchronous with upper extremity movements and, hence, not reflexive (Lauerma 1993). If the patient is not medically astute, then the clinician may purposefully mislead the patient by claiming that the flexor response to plantar stimulation was evidence for a slight reversal of the paralyzing illness, and further improvement is expected to follow. The clinician may also mislead the patient into believing that the pin sensory level has slowly been retreating. Further improvement may follow over several minutes to an hour, particularly if the patient is given serious attention and is encouraged to believe that slow but steady improvement often occurs in such illnesses.

The science of statistics can help uncover factitious amnesia. Subjects with true amnesia should have about a 50/50 chance of recalling whether individual test stimuli were present on a 20-item list they have been shown. In contrast, subjects with malingered amnesia, who are naive to the principles of probability, may fail to recall items correctly at greater than 50% probability (Brandt et al 1985), thereby indicating covert recognition of list items.

Munchausen syndrome by proxy may be suspected for unexplained pediatric illnesses in which a parent (usually a mother) shows obsessive concern with the illness and is medically astute while developing close social relationships with clinical staff. Frequently, the father is unaware of the child's disorder and may not accompany the child to the clinic (Baldwin 1994). Confirmation of the fabrication of the illness in hospitalized children may be obtained through surreptitious video recording. However, the legality of this measure may vary with the jurisdiction serving the particular hospital, and so legal counsel is needed when entertaining this approach. If a clinical program adopts this approach, a well-defined plan for taking action to protect the child must be in place (Stirling 2007). Careful monitoring of the child's visitors, objects taken to the child or left in the room with the child may aid in the diagnosis. Monitoring of physiologic functions such as respiration may also assist.

Management

Patients commonly refuse psychiatric referral because they do not believe that they have a psychological disturbance (Kanner et al 1999). However, some studies have found that counseling can benefit functional seizures (Shen et al 1990; Aboukasm et al 1998). This can include providing video feedback to the patient of his functional seizures or video demonstration of intact leg movements during sleep to patients with functional paraplegia (Jahn et al 2006). Hypnosis, which was first applied in the late 19th century (Bogousslavsky and Walusinski 2010), may be therapeutically successful in the hands of a skillful practitioner, at least when motor disturbances occur (Moene et al 2003). Praise given by clinicians or therapists for functional gains, however small, can help to accelerate recovery while allowing the patient to maintain dignity (Delargy et al 1986). Frank discussion of the diagnosis by itself can reduce attacks in a minority of persons with functional seizures (McKenzie et al 2010).

Distinct psychiatric disturbances in functional patients, such as depression or anxiety, should be treated according to the standard of care, which may include psychiatric or psychologic referral. The psychotropic medication venlafaxine has been associated with reduced nonepileptic seizures in patients who have coexisting affective disorders (Pintor et al 2010). Similarly, sertraline has benefited psychogenic seizures (LaFrance et al 2010). At present, however, prospective therapeutic trials to evaluate specific interventions are few and strongly deserve increased undertaking (Gaynor et al 2009).

Work preliminarily suggests that cognitive behavioral therapy is efficacious for functional disorders (LaFrance et al 2009; Goldstein et al 2010; Stone et al 2010). The approach includes relaxation training and teaching patients to control their illness, rather than being passive. Combining sertraline with cognitive behavioral therapy may considerably more reduce functional seizures than cognitive therapy by itself (LaFrance et al 2014). Inpatient treatment with cognitive behavioral therapy, for functional paresis at least, may be more efficacious than outpatient treatment (McCormack et al 2014). When diagnosing functional seizure distresses patients, extended empathic therapy and support are advisable (Thompson et al 2009).

In general, respectful encouragement for a positive prognosis can benefit, as well as indicate, that the thoroughly conducted workup has suggested that there is no life-threatening disorder. Reassurance while conveying sincere interest in the patient's welfare will prove valuable (Reuber et al 2005; Peckham and Hallett 2009; Stone et al 2010). Rather than convey lack of understanding, the clinician should provide a positive diagnosis, that the person has a “functional neurologic disorder.” The terms "hysteria" and "malingering" should not be used (Solyom and Solyom 1990). Patients must never be told that they are "faking" the disorder, or they will almost surely seek medical treatment elsewhere, including from unqualified and potentially harmful practitioners, or they may bring legal action. Although hospitalized patients with functional disorder may provoke resentment among staff, the clinician must always maintain a calm, professional, respectful attitude and be in charge. Avoid describing patients as "manipulative" because this amounts to admitting the clinician's failure to maintain control of a vexing clinical presentation and can induce hospital staff not to act in the best interests of the patient's long-term health. The clinician above all needs to be a team leader to maintain a coherent staff approach to the patient. Private meetings with hospital or clinic staff, conducted with complete respect for the patient and acknowledgment that functional presentations are common experiences, may help to unify the staff approach and maintain an empathetic attitude.

Physical therapy for functional restricted bodily movement has been reported to have enduring benefit (Ness 2007). The therapeutic process included establishing rapport with the patient. It is conceivable that such intervention undoubtedly is a form of psychotherapy itself, in providing to the patient reassurance, empathy, feedback, and encouragement. In similar manner, compassionate visual training exercises applied over weeks to months can improve functional blindness (Fischer et al 2013). Nonetheless, the efficacy of such approaches has not been established in controlled clinical trials. However, it is conceivable that physical rehabilitation can help physical deconditioning that may follow prolonged activity curtailment in various kinds of functional neurologic disorders.

One case report observed resolution of functional itch with topiramate, which suggests a potential role for pharmacologic treatments for functional diseases (Calabro et al 2013).

An exciting development is the application of repetitive transcranial magnetic stimulation (TMS) to motor-associated cortex in individuals with functional disorders. A chance observation led to finding an 89% success rate with this form of treatment for various forms of functional paresis (Chastan and Parain 2010). Successful outcomes have also been described following TMS for functional aphonia and functional movement disorders (Chastan et al 2009; Garcin et al 2013). Nonetheless, a review of studies to date found considerable inconsistency in results and the absence of placebo control interventions (Pollak et al 2014). In response, Chastan and colleagues are currently pursuing a clinical trial involving “large-field” TMS for functional movement disorders, which applies a circular coil to stimulate a cranial area 30 times as large as the standard figure-8 coil (Parain and Chastan 2014). Preliminary findings have been encouraging.

The basis for such improvement is unknown. Chastan and colleagues suggested that patients with functional paresis may be helped by observing their involuntary limb contractions that are induced by stimulation. However, this would not explain improvement for active, dyskinetic movement disorders. Instead, the improvement of interhemispheric inhibition than can follow TMS may possibly be responsible (Lanza et al 2013). Such treatment already has been shown to have ample success for depression that is refractory to medication (Wani et al 2013). Thus, it may also be possible that functional disturbances may improve following TMS, secondary to improving an underlying emotional disorder. One may expect to observe further exciting developments in this line of investigation.

Another emerging line of treatment is the ability to progressively train persons with functional tremor to slow the tremor frequency by following a changing metronome (Espay et al 2014). Preliminary findings, however, indicated that improvements were followed by relapses in 40% of the patients. Hence, it may be necessary to combine tremor retraining with other measures, such as cognitive behavioral therapy, in order to obtain more consistent improvements.

Clinical records must be worded to avoid blaming the patient for the disturbance, because such records may eventually be read by the patient and his legal counsel, with possible harm to clinician and patient alike.

More challenging is when the clinician is asked by the patient to document the patient's work limitations or inability to be employed. The clinician may feel tempted to decide whether the patient is malingering (a judgment of consistent, conscious management of the illness presentation) or is poorly able to control the functional disorder (a judgment of partial irresponsibility for the disorder). This is a subjective judgment that may never be satisfactorily or objectively resolved. Because such patients generally refuse psychiatric evaluation (Carson et al 2000a), the clinician may be without professional backing by mental health personnel. Referral for vocational assessment should be considered in such instances. Praising the patient s healthy progress may help reinforce the continuation of recovery (Delargy et al 1986). Essentially, the clinician's empathy may become the crucial "cure" for the disorder, rather than medications. This may considerably vex the clinician who desires a speedy resolution for the disorder. However, the adoption of a patient, encouraging outlook and private expectation for a long-term outpatient relationship will best serve both the patient and clinician alike.

The diagnosis of Munchausen syndrome by proxy requires assembling involved clinical staff and legal counsel to review evidence supporting the diagnosis and then contacting a child protection agency for the child's safety and removal. Such steps must be undertaken without informing the responsible caregiver first (Baldwin 1994). Advice or leadership from clinicians with expertise in diagnosing and intervening in the condition is recommended (Stirling 2007). Psychiatric evaluation of the caregiver may be required and may involve a court order.

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**References especially recommended by the author or editor for general reading.

Profile

Age range of presentation

06-12 years
13-18 years
19-44 years
45-64 years

Differential diagnosis list

astasia-abasia
concentric visual field loss that changes visual angle when testing with stimuli at different distances from the patient
psychiatric disturbance
personality disorder
paralysis
visual loss
tremor
tics
intoxication
systemic infection
fatigue
sleep deprivation
primary progressive aphasia
acute aphasia due to neurosyphilis
complex regional pain syndrome
reflex sympathetic dystrophy
shoulder-hand syndrome
Sudeck dystrophy
algodystrophy
prosopagnosia
cortical deafness
visual agnosia
acquired alexia
epileptic seizures
complex partial seizures
nonepileptic seizures
strokes
mild urinary retention
brain injury
schizophrenia
nonobstructive urinary retention

Other topics to consider

Neurologic disorders presenting with behavioral signs and symptoms
Pathologic yawning: neurologic aspects
Psychogenic seizures
Psychogenic movement disorders