vituity®
At the heart of better care.

WHY I'M AN OWNER

"**Ownership means I have a say** in creating sustainable change in my practice, my organization, and in emergency medicine. From clinical innovations to healthcare justice, my voice is heard. **I am empowered to make differences every single day.**"

Maureen Bell, MD | Vituity Partner
EM Medical Director, Howard University Hospital

Vituity has always been **100% owned** by its physicians, and you **join as a Partner from Day 1**.

Learn more at **vituity.com/EMcareers**

Now hiring in: *Arizona, California, Illinois, Indiana, Kansas, Missouri, Minnesota, Nevada, New Jersey, Oregon, South Carolina, Texas, Washington, and Wisconsin.*

Received: 26 February 2021 | Revised: 8 April 2021 | Accepted: 12 April 2021

DOI: 10.1111/acem.14263

SPECIAL CONTRIBUTION

Functional neurological disorder in the emergency department

Sara A. Finkelstein MD, MSc[1] | Miguel A. Cortel-LeBlanc MD, MHA[2,3,4] | Achelle Cortel-LeBlanc MD[2,4] | Jon Stone MB, ChB, PhD[1]

[1]Centre for Clinical Brain Sciences, University of Edinburgh, Edinburgh, UK

[2]Faculty of Medicine, University of Ottawa, Ottawa, Ontario, Canada

[3]The Ottawa Hospital, Ottawa, Ontario, Canada

[4]Queensway Carleton Hospital, Ottawa, Ontario, Canada

Correspondence
Sara A. Finkelstein, Centre for Clinical Brain Sciences, University of Edinburgh, Edinburgh, UK.
Email: Research@fndmd.com

Funding information
JS has received funding from a NHS Scotland NRS Career Fellowship for research support. JS reports honoraria from UpToDate for writing articles related to functional neurological disorders. JS receives payment for consulting as an expert witness in personal injury and negligence claims. JS runs a free self-help website, neurosymptoms.org, for people with functional neurological disorder (FND). JS is on the medical board of FND Hope and FND Action as well as being secretary of the FND society (www.fndsociety.org).

Supervising Editor: J. Stephen Huff, MD.

Abstract

We provide a narrative review of functional neurological disorder (FND, or conversion disorder) for the emergency department (ED). Diagnosis of FND has shifted from a "rule-out" disorder to one now based on the recognition of positive clinical signs, allowing the ED physician to make a suspected or likely diagnosis of FND. PubMed, Google Scholar, academic books, and a hand search through review article references were used to conduct a literature review. We review clinical features and diagnostic pitfalls for the most common functional neurologic presentations to the ED, including functional limb weakness, functional (nonepileptic) seizures, and functional movement disorders. We provide practical advice for discussing FND as a possible diagnosis and suggestions for initial steps in workup and management plans.

KEYWORDS
conversion disorder, functional movement disorder, functional neurological disorder, nonepileptic seizures, stroke mimic

INTRODUCTION

Functional neurological disorder (FND), also called conversion disorder, is an involuntary change in motor or sensory function, where clinical findings provide evidence of incompatibility or incongruency with other recognized neurological or medical disorders.[1] Patients with FND may present acutely to the emergency department (ED) with symptoms similar to epileptic seizure, stroke, or other neurological conditions.[2] These patients often have a high return rate to the ED[3] and their symptoms have traditionally been seen as difficult to manage in the ED setting. Shorter time from symptom onset to diagnosis is an important positive prognostic factor,[4] demonstrating the importance of identifying these patients in an acute care setting.

In recent years, understanding of and clinical practice around FND have changed substantially. There has been increasing research in evidence-based diagnosis in this patient group, focusing on the use of positive clinical signs to make a "rule-in" diagnosis.[1] Emerging evidence regarding the neural basis of FND and its treatment places it at the interface between neurology and psychiatry. In this new paradigm, ED physicians are well positioned to raise FND as a possible diagnosis with the patient, helping to improve outcomes and decrease unnecessary health care utilization.

This review aims to make the recognition of FND more accessible to emergency physicians, such that they can consider it as a likely or suspected diagnosis. We discuss in detail positive clinical signs observed in the most common FNDs presenting to the ED. Common diagnostic pitfalls are addressed as well as an approach to diagnostic testing. We then discuss how to have a conversation with patients about a possible FND diagnosis and first steps in management.

Methodology

A panel of four physicians coauthored this paper: two neurologists with subspecialty expertise in FND (JS and SAF), a general neurologist (ACL), and a board-eligible emergency physician (MACL). All authors agreed on an outline of important sections to include in the article at the beginning of the project. Various search strategies (e.g., PubMed, Google Scholar, academic books, and hand search through review article references) were then used to identify evidence-based and up-to-date references for each section. References were reviewed and evaluated for relevancy and included based on review by all authors.

A brief word on terminology

Terminology regarding functional disorders has evolved over time. Some terms, including "psychogenic," "psychosomatic," and "conversion" disorder, along with "somatization," presume an exclusively psychological cause, which is often not evident. "Nonorganic" suggests a dualism of brain and mind and "medically unexplained" suggests a problem where we have no idea about etiology, diagnosis, or treatment. Terms like "hysteria" or "pseudoseizures" are pejorative or suggest a problem that is faked. The research community have supported the use of the term "functional neurological disorder" as one that is etiologically agnostic. FND seizures will be referred to in this paper as functional seizures but are alternatively referred to in the literature as dissociative, psychogenic, or nonepileptic seizures or attacks.

Factitious disorder is the deliberate feigning of symptoms without external motivators, while malingering is deliberate feigning for the purposes of secondary gain such as financial benefit. These are distinguished from FND by their intentionality—FND symptoms are unintentional and involuntary (see "Dealing with doubt" section).

EPIDEMIOLOGY

The overall prevalence of FND in the ED has been reported as 0.4% to 4%, although studies likely underestimate rates due to inconsistency in diagnostic coding and underrecognition.[5,6] Patients with FND account for 9% of all acute neurological admissions.[7] Functional seizures represent around 10% of all seizures in the ED,[8] and of patients presenting with refractory status epilepticus resulting in intensive care unit care, 25% have FND seizures and not epilepsy.[9] Up to one-third of patients with functional seizures will develop functional status epilepticus,[10] often with accompanying ED visits. Of patients presenting with acute onset motor or sensory symptoms, up to 25% of cases have been found to be stroke mimics, with about one in 10 of those representing patients with functional neurological symptoms.[11–13] Patients with functional disorders, including FND, have a higher utilization of ED care correlating with higher health care costs, even after they have received a diagnosis.[3,14] Moeller et al.,[15] when examining diagnostic accuracy of neurological disorders in the ED, found that functional disorders were the leading cause of misdiagnosis of neurological presentations. Costs of ED treatment for FND in 2017 among around 40,000 adults and children from a population of around 130 million U.S. citizens was $163 million, compared to $135 million for refractory epilepsy.[5]

PATHOPHYSIOLOGY

Previous etiological ideas for FND were exclusively psychological. New ideas about the pathophysiology of FND retain the importance of psychological models, but introduce a neurobiological perspective that places FND at the interface of the brain and mind.[16–19] Research using functional imaging suggests that these disorders are associated with dysfunction of brain networks involved in attention and perception, sense of agency, and prior sensorimotor expectations (Figure 1). A number of functional neuroimaging and neurophysiologic studies have demonstrated differences in activations between patients with FND, healthy controls, and participants asked to feign symptoms. Symptom generation and maintenance is likely due to a combination of predisposing, precipitating, and perpetuating factors. These arise from the patient's biology, cognition, environmental factors, previous experiences, and in some cases acute triggers, which are more often a pathophysiological experience such as injury or migraine than a psychological one.[20–23]

Dysregulation of attention is a major component of FND. Most people are likely familiar with the effect of focused attention on the self altering the outcome of an intended action—for example, being more likely to mix up one's words during a public speaking engagement. Our nervous system is designed to balance "bottom-up" sensory information traveling from the body to the brain with "top-down" predictions about what that sensory information will be. Dysregulation of this system in patients with FND is supported by electrophysiologic studies.[24,25] There appears to be an abnormally high amount of involuntary attention directed toward symptom-related prior beliefs and expectations, serving to reinforce and perpetuate symptoms.[26] This may explain why FND symptoms tend to improve with distraction, which physiotherapists capitalize on to treat FND motor symptoms.[27,28]

MAKING THE DIAGNOSIS OF POSSIBLE OR LIKELY FND

The basis for FND diagnosis is the demonstration of clinical features of internal inconsistency (reversibility) and/or to a lesser extent

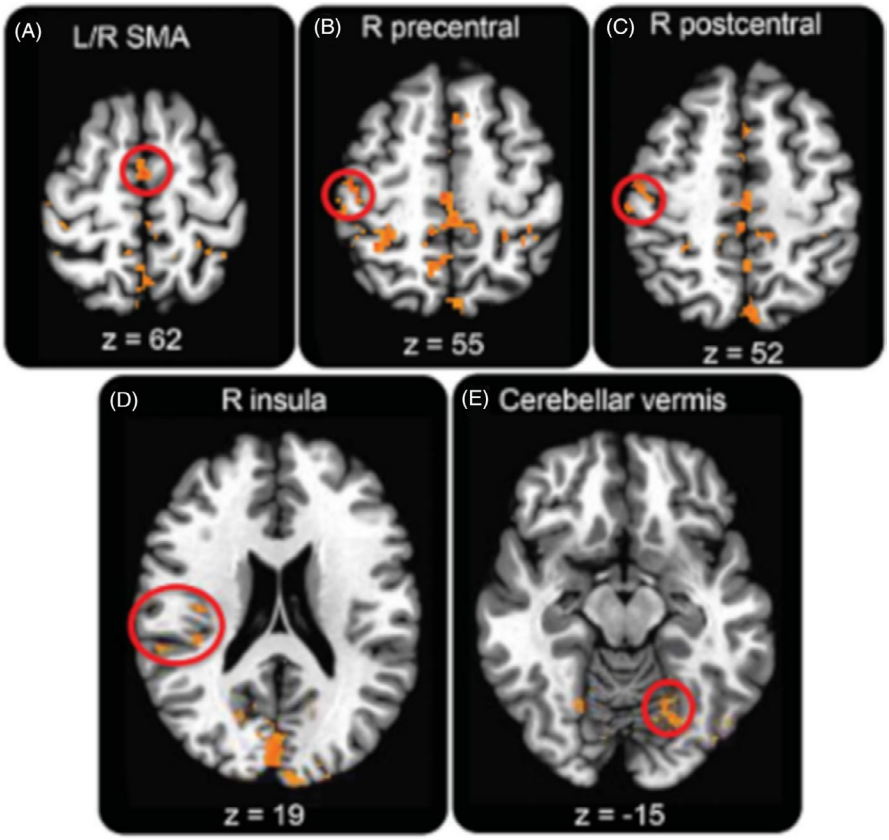

FIGURE 1 Decreased functional connectivity between the right temporoparietal junction and bilateral sensorimotor regions in patients with functional movement disorder*. *Reproduced from Maurer CW, LaFaver K, Ameli R, Epstein SA, Hallett M, Horovitz SG. Impaired self-agency in functional movement disorders: A resting-state fMRI study. Neurology. 2016;87(6):564-570. https://n.neurology.org/

TABLE 1 Selected clinical signs in functional weakness

Clinical sign	Description	Reliability[a]
Hoover's sign[20,35-37,39]	Weakness of voluntary hip extension that resolves with voluntary contralateral resisted hip flexion. Difficult to detect in bilateral leg weakness.	+++
Platysma overactivation[40]	Contraction of one side of the platysma, creating the effect of a facial droop.	++
Hip abductor sign[37]	Return of strength to hip abduction in the weak leg with contralateral hip abduction against resistance	++
Give-way/collapsing weakness[35,41,42]	Strength is initially normal and then collapses with resistance.	++
Dragging monoplegic leg[20,35]	Plegic leg is dragged behind body often with hip internal or external rotation and without hip circumduction.	++
Drift without pronation[35,43]	Isolated downward arm-drift without associated pronation.	+
Global pattern of weakness[35,44]	Equal weakness of both flexor and extensor muscles, both proximally and distally.	+
Motor inconsistencies[45]	Inability to produce one movement, while using the same muscles to produce a different movement. For example, a patient may have difficulty dorsiflexing while supine, but be able to stand on heels without difficulty.	+

+++ = highly reliable; ++ = reliable; + = suggestive.

[a]Reliability determined based on available clinical data[34] and author consensus.

incongruency with known patterns of structural neurological disease.[29] This is done primarily by looking for positive clinical signs of these disorders.[29] No clinical sign in isolation should be taken as confirmation of a functional disorder. Importantly, the need for a stressor preceding onset of physical symptoms has been removed from the DSM-5. In the absence of an established therapeutic

relationship, as would be typical in the ED, we suggest avoiding routinely questioning patients about past trauma. While it is a risk factor for FND, occurring in 10% to 30%, diagnosis should not be based on its presence or absence, and harm can be done by bringing this up with patients if they are not prepared to talk about it.

In gathering the history, care should be given, as always, to taking the patient's symptoms seriously. Practically, this can include making statements indicating that these symptoms are familiar, that this is a real problem, and that you believe them.[30] It is important to ask about the amount of disability the symptoms are causing for the patient on a day-to-day basis.[31]

Functional limb weakness

Functional limb weakness is one of the most common presentations of FND to the ED[2] and can present similarly to a variety of structural disorders including stroke and demyelinating lesions. About half of patients with functional limb weakness will present with acute onset of symptoms.[32] One or any combination of limbs can be affected, although unilateral symptoms are the most common.[20,33] Often when there is only one limb that feels weak, subtle weakness will also be found in the other ipsilateral "normal" feeling limb on examination.[34]

Patients may subjectively note that their limb feels heavy, like it is "not there," or "not a part" of them.[34] If the upper limb is affected, patients may report frequently dropping things. If the lower limb is affected, patients may drag their leg behind them,[20,35] or find their knee giving way leading to falls.[34] Sensory symptoms, in conjunction with weakness, are very common.[34]

A variety of clinical signs have been studied to aid in diagnosis of functional limb weakness (Table 1, Figures 2 and 3). Current data regarding sensitivity and specificity of clinical signs are limited, and need to be interpreted with caution. For example, specificity of Hoover's sign has been reported as 100% in two studies[36,37] but

FIGURE 2 Hip abductor and Hoover's sign of functional leg weakness. Top left: Hip abductor sign – weak left hip abduction. Top right: Hip abductor sign – strength in left hip returns to normal with abduction of right hip. Bottom left: Hoover's sign – weak left hip extension. Bottom right: Hoover's sign – strength in left hip extension returns to normal with right hip flexion.

was infrequently present in patients with structural neurological disease in another.[20] Similarly, drift without pronation as a sign of functional arm weakness has a reported high specificity of 93% to 95%.[38] However, most providers would agree that this can be seen in clinical practice in a variety of non-FND patients. Caution in interpretation should be taken when only one positive sign is present, when they are only mildly positive, or when there is significant pain. Patients with neglect or apraxia may also have falsely positive signs.[34] We present the reliability of these signs in Table 1 as a composite of the data available and author consensus based on clinical experience.

How do I know it's not a stroke?

Stroke and transient ischemic attacks, as well as other stroke mimics, will necessarily be on the differential for acute onset neurological symptoms, and typical stroke protocol should be followed in the initial workup of these patients. Data from a systematic review and a meta-analysis show that FND represents between 7% and 15% of stroke mimics, making it only slightly less common than stroke mimics related to migraine or seizure.[12,13] If the diagnosis remains uncertain, patients can usually be treated safely with tPA: the rate of symptomatic intracerebral hemorrhage in stroke mimics is 0% to 0.5%, with systemic hemorrhage and angioedema being similarly rare.[46–50] Other potential harms of giving tPA to a patient with FND include increased cost, with one study showing a median excess cost for stroke mimics given tPA to be over $5,000 USD per admission[51] as well as a potential for adverse psychological impact. On balance, it is likely best to err on the side of overtreating, rather than undertreating, with tPA in cases of uncertainty when patients otherwise meet criteria for thrombolysis.

Functional sensory loss

Sensory symptoms in FND range from pain or a "pins and needles" sensation, to heaviness or numbness.[52,53] It may be useful to look for motor signs of FND, such as a Hoover's sign, because these often occur in

FIGURE 3 Platysma sign of functional facial spasm, dragging monoplegic gait of functional leg weakness. Top left and top right: Platysma overactivation causing appearance of facial droop, with return of normal strength when asked to show teeth. Bottom left: Dragging monoplegic leg.

conjunction with sensory changes and can help put the sensory symptoms in a broader clinical context.[54] Sensory testing on examination is necessarily subjective and prone to bias, on the part of both the patient and the examiner.[55] The clinical signs for functional sensory loss have not been found to be reliable in terms of differentiating from structural sensory loss.[53] For example, reliability for splitting of vibration sense across the sternum or forehead varies widely across studies, ranging from 50% to 95% for sensitivity and 14% to 88% for specificity.[38]

Functional seizures

Functional seizures are perhaps the most well studied of all functional disorders, and several attempts have been made to determine the reliability of various distinguishing features from epilepsy. Patients often report warning symptoms of autonomic arousal prior to the event.[56–60] They may also report dissociation—a feeling that the world or their body is disconnected from them[56,61,62] (a note of caution: symptoms of autonomic arousal and dissociation can also precede focal onset seizures as well as syncopal episodes).

A detailed history from the patient and any witnesses to the event should be taken, going over any warning symptoms, ictal features, and postictal state. Examining any video the patient or their family members have of similar events can help greatly with diagnosis.[63] Table 2 lists selective features that have been shown to be useful in differentiating between functional and epileptic seizures. The sum of the clinical signs and history, rather than one clinical sign provided, should be taken as a whole to determine whether the episode is likely a functional seizure.[64] We strongly discourage maneuvers that may harm an individual, such as dropping the patient's arm on to their face. These tests are diagnostically unhelpful because they will often be negative in dissociative states, even when the patient is able to experience them. For a patient in a persistent unrousable state, to assess responsiveness, a high-pitched tuning fork applied to the nostrils is a kinder and more effective stimulus.[65]

TABLE 2 Clinical features distinguishing functional from epileptic seizures[38,72-74]

Clinical sign	Notes	Reliability[a]
Highly suggestive of functional seizures		
Closed eyelids during ictal peak	Patients may actively resist eyelid opening.	+++
Prolonged duration	Most epileptic seizures will stop spontaneously in 2 min or less. Particularly useful if it resolves spontaneously after prolonged duration, without significant postictal period. Caution: patients with status epilepticus will have prolonged seizure activity.	++
Fluctuating course	Movements may wax and wane in intensity or stop and start.	++
Ictal awareness/memory of seizure	Only relevant for generalized seizures (abnormal movements of all four limbs). Caution: frontal lobe seizures can involve bizarre movements with retained awareness. Loss of awareness is standard for most functional seizures.	++
Ictal/postictal weeping	Relatively specific for functional seizures, although low sensitivity. May also have other signs of emotional distress.	++
Asynchronous limb movements	Caution: can also be present in frontal lobe seizures.	++
Side to side head shaking	May rarely be present in epileptic seizures. Good differentiator for generalized shaking events only.	++
Response to stimuli during ictal period	Only applies to generalized shaking attacks.	++
Highly suggestive of epileptic seizures		
Figure of four sign	One arm flexed at elbow, other arm extended at the elbow, usually present just before secondary generalization.	+++
Guttural cry/scream	During tonic phase, typically at seizure onset.	++
Prolonged rigid phase with cessation of respiration	Based on authors' experience.	++
Postictal stertorous breathing	Low-pitched sound from back of throat, like sound from nasal congestion or snoring.	+++
Unhelpful features common to both		
Tongue biting Injury (although severe burns and shoulder dislocation should prompt consideration of epilepsy) Urinary incontinence Attack appearing from sleep/no witnesses to seizure Presence of aura or postictal confusion Breath holding High serum lactate after an event[71]		

+++ = highly reliable; ++ = reliable; + = suggestive

[a]Reliability determined based on available clinical data[73,75–77] and author consensus.

While the majority of functional seizures are convulsive, thrashing, or tremulous events, about 30% of patients will have events that resemble syncope, in which they fall down, are still, and are unresponsive.[38] For these types of events, a phenotype of sudden collapse to the ground, with eyes closed, and documentation of 2 or more minutes of loss of consciousness is highly specific for a functional disorder etiology.[38,66,67]

Research on biomarkers to differentiate functional from epileptic seizures has thus far not proven helpful. Serum lactate and prolactin levels may be raised in epileptic seizures compared to functional seizures, but levels are highly dependent on timing in relation to the seizure and can be elevated in functional seizures.[68–70] For example, one study asking participants to feign a seizure demonstrated an increase in lactate levels from baseline.[71] Similarly, elevation of creatine kinase or white blood cell count, while possibly more common after an epileptic seizure in comparison to a functional seizure, are nonspecific and should not be relied upon for diagnosis.[68]

Functional movement disorders

Functional movement disorders are the second most common cause of acute movement disorders presenting to the ED.[78] The primary characteristics of functional movement disorders are that they diminish or resolve with distraction and/or entrain (change frequency to match that of other motor tasks).[79–81] Movements may be sudden in onset and have spontaneous remissions. The affected body part may change over time. Do not assume that just because the movement appears to be "bizarre" that it relates to a functional disorder. Many movement disorders can appear strange, such as task-specific dystonia or stiff person syndrome, emphasizing the need for a neurologist to usually be involved in making a diagnosis.

In the case of functional tremor, it may be present at rest, with sustained postures, or on action: look for variability in frequency, rhythm, and axis or direction (but not amplitude, because this can vary in a number of tremor etiologies).[82] Improvement with distraction may be seen while taking a history or may require the examiner to ask the patient to perform other motor tasks with a nonaffected body part.[79] Entrainment can be demonstrated by asking the patient to copy a rhythmic movement with an unaffected limb, such as finger tapping.[83] In functional tremor, tremor will either improve or change to match the frequency of the voluntary movement or the patient will have trouble copying the movement.

FND and suspected cauda equina syndrome

Over 50% of patients presenting with cauda equina syndrome (CES) will have normal imaging ("scan-negative CES").[84,85] Recent studies have pointed to a high frequency of associated FND symptoms and signs, especially lower-limb weakness FND signs, in these patients.[86] Patients with scan-positive CES are more likely to have diminished or absent ankle jerks than scan-negative patients (78% vs. 12%). Abnormal anal sphincter tone on digital rectal examination and high postvoid residual volume (200 or 500 cc) have *not* been shown to be clinically useful differentiators.[87] Ultimately, given the potential morbidity of CES, no historical features or clinical signs remove the need for urgent neuroimaging. If imaging fails to identify a structural etiology, however, then discussing FND as a possible contributor to symptoms may be appropriate.

Diagnostic pitfalls

The diagnosis of possible or likely FND should usually be made on the basis of positive clinical features, usually from the physical examination (including seizure semiology), not from the clinical history. Table 3, adapted from Stone 2013,[74] addresses some common misconceptions that may unduly sway a physician toward or away from a diagnosis of FND.

Psychiatric comorbidity

Many patients with FND have a comorbid psychiatric disorder, such as depression or anxiety, which can complicate their presentation to the ED. Rates of depression among patients with FND are likely between 20% and 40%,[89–91] and rates of anxiety around 40%.[92] Rates of psychiatric comorbidity are higher in FND patients (two-thirds to three-quarters of patients) than in other neurology patients with similar levels of disability (one-half to two-thirds of patients).[20,90,91,93]

TABLE 3 FND diagnostic pitfalls[74]

1. Presence of psychiatric comorbidity: A diagnosis of FND should not be based on the patient having a psychiatric disorder such as anxiety, depression, or a personality disorder.
2. Failure to consider structural disease comorbidity: One of the commonest risk factors for FND is the presence of minor or major disease comorbidity such as multiple sclerosis, stroke, or epilepsy. Therefore, even in a patient with clear FND, always consider whether they may have an *additional* medical or neurological condition.
3. Putting too much weight on the presence or absence of "stress": A diagnosis of FND should not be based on the presence of an obvious life event or stressor nor should it be discarded due to lack of recent stress. Similarly, just because the patient attributes their symptoms to stress does not mean this is the case.
4. La belle indifférence: i.e., the patient seemingly not caring about their symptoms is not a reliable marker for FND and occurs just as commonly in structural disorders.[88]
5. The patient is not a young female: FND should not be excluded based on demographics. Patients can be male or female, young or elderly, and from diverse socioeconomic backgrounds.
6. The patient seems too "normal": patients with FND may be nice, normal people too!

Adapted by permission from BMJ Publishing Group Limited from Stone J, Reuber M, Carson A. Functional symptoms in neurology: mimics and chameleons. Pract Neurol. 2013;13(2):104–113.[74]

Abbreviation: FND, functional neurological disorder.

Comorbid personality disorder may also be present in patients with FND at rates increased from those in the general population.[94] Despite the higher rate of psychiatric disorders in the FND population, not all FND patients have a psychiatric diagnosis (indeed, up to one-third may not). As such, psychiatric comorbidity is best seen as a risk factor, rather than a causative factor, for FND. In patients who do present with clear psychiatric symptoms, ensuring that these are optimally managed is often necessary for patients to engage meaningfully in therapy for FND symptoms.

DEALING WITH DOUBT: IS MY FND PATIENT FAKING HIS OR HER SYMPTOMS?

In the ED setting, perhaps more than any other, the issue arises as to whether someone with clinical features of FND really does have a genuinely experienced condition or whether they could be feigning symptoms for attention or other reward. Many patients report psychologically and sometimes physically harmful experiences in EDs from health care professionals including not being believed, being laughed at,[95] unnecessarily painful procedures during presentations with altered states of awareness, and clinicians jumping to conclusions about potential psychiatric causes.

There is a range of evidence to support what patients with FND tell us, which is that they really do experience the neurological symptoms with which they present. This includes similar presentations and symptom clusters around the world and across history, persistent symptoms at long-term follow-up studies, evidence from functional neuroimaging and neurophysiological studies with findings that are different between FND and feigning, and positive responses in randomized controlled trials. One cannot prove that someone is not feigning, and exaggeration can occur in all medical conditions, often to convince skeptical doctors. Evidence of feigning should come from evidence of lying or finding a marked discrepancy between what the patient says they can do and what they are seen to do. This is not the same as observing variability that the patient is aware of. Frank deception remains rare, and the error of considering that someone is feigning when they are not is one that every doctor should strive to avoid.

INVESTIGATIONS

In the ED, an important focus is to rule out diagnoses with a high chance of immediate morbidity. In addition, the presence of positive clinical signs of FND does not exclude the presence of a concomitant neurological condition. Consequently, we recommend a low threshold to investigate patients in the ED—especially in patients with unclear diagnoses, acute focal neurological presentations, and seizures. Moreover, investigations should be done selectively according to the presenting symptom and guided by a thorough physical examination. Many symptoms that go along with FND, such as a fatigue, can be due to many causes and should be investigated appropriately. Patients may benefit from investigations being done all at once at the outset and not in a prolonged, serial or repetitious way,[96] which typically reinforces the idea that their doctors do not know what the problem is. There are many neurological disorders with normal investigations and doing tests is not the way to achieve a positive FND diagnosis. Tests that are ordered to "reassure" the patient often do not. In a randomized controlled trial of 150 patients with chronic daily headache, investigators found that patients receiving neuroimaging had no difference in anxiety scores at 1 year compared to those who had not undergone neuroimaging.[97]

MANAGEMENT

Recognition of FND is one of the first challenges, especially in "acute stroke" or "status epilepticus" presentations. Generally, we recommend involving a clinician with expertise in neurological diagnosis because there are many pitfalls in the diagnosis of FND, most importantly failure to recognize another comorbid neurological/medical condition. Nonetheless, the ED physician can make and communicate a suspected FND diagnosis and is often involved in seeing people with an established diagnosis of FND from a previous encounter, where diagnostic conversations still need to occur.

The pillars of managing suspected FND in the ED include:

1. Effective and therapeutic disclosure of the possible/likely diagnosis.
2. Avoidance of iatrogenic harm.
3. Appropriate referral for follow-up care.

The first step in management of FND is to provide patients with a name for their likely or suspected diagnosis. While it is important to address specific illness concerns, avoid only telling them what it is not and discuss FND as a possible or likely diagnosis. Although this sounds obvious, often patients are told what has been ruled out or are presented with a possible risk factor for their symptoms, such as stress, without actually being told what the problem is, leaving them with a sense that the diagnosis is still unknown and that they remain a medical mystery. Providing patients with a diagnosis of possible or likely FND is the first step in management, and this can be therapeutic in and of itself when done well.[30]

The diagnosis of possible or likely FND can be delivered in the same manner as diagnosing any other condition (Table 4). The clinician should explain to the patient the name of the diagnosis and how the diagnosis was made and provide some basics regarding pathophysiology. In explaining how the diagnosis was made, it is often useful to demonstrate to the patient any positive physical signs on their examination, such as a Hoover's sign.[98] In the case of functional seizures, review semiological features that are strongly suggestive of FND rather than focusing on why it is not epilepsy. Any specific concerns the patients may have had about alternative diagnoses should be addressed. In explaining pathophysiology, it can be effective to use analogies, such as comparing the brain to

TABLE 4 Key elements to include and to avoid in discussing a possible diagnosis of FND

Do include	Avoid
• The name of the diagnosis • How the diagnosis was made (including sharing positive diagnostic signs) • A brief explanation of pathophysiology • Tell the patient their symptoms are real and not imagined • Emphasize that these symptoms are common • Emphasize that symptoms are potentially reversible and therefore could improve • Offer further resources to learn more	• Only an explanation of what they do *not* have • Attributing symptoms to psychological problems or stress • Saying or inferring that this is "imagined," "all in their head," or voluntary in some way • Misattribution of symptoms • Using negative investigations as evidence of the diagnosis

Abbreviation: FND, functional neurological disorder.

TABLE 5 Examples of ways to explain the diagnosis of possible FND

"You likely have functional neurological disorder, or FND, which is causing your weakness. I can see from your examination that your nervous system is not damaged; however, it's struggling in getting its messages through.
Can you see how the more you try, the worse your leg weakness gets, but when you are focused on your other leg it works much better? [demonstrate Hoover sign]
What this tells me is that your brain is having difficulty sending messages to your leg, but that improves when you are distracted.
It's like the opposite of phantom limb pain. Your brain thinks the leg isn't there even though it is.
It shows us that there is no damage to your nervous system and the problem is potentially reversible."
"Seizures/attacks in FND are caused by a 'trance-like' state in the brain called dissociation. The brain shuts itself down temporarily, often in response to a 'red-alert' state and this becomes a reflex or habit, which is why it keeps happening."

Abbreviation: FND, functional neurological disorder.

a computer and explaining that FND is "software problem" of the brain (Table 5).

Referral

Assessment by a neurologist is usually necessary to confirm the diagnosis, arrange therapy, and identify any concurrent neurological disorders. Once the diagnosis of FND is confirmed by a neurologist, typical avenues for treatment include physiotherapy or psychological therapy.[27,99] There is increasing evidence of effectiveness of these approaches, which should ideally be delivered in a multidisciplinary team.[100] Therapies for FND have become much more tailored in recent years. Consensus recommendations for physiotherapy have been tested with promising results in randomized clinical trials for patients with motor FND.[27,101–104] Psychological therapy is the treatment of choice for functional seizures, where treatment has similarities to the management of panic attacks.[105] Psychiatric assessment is often important to provide a more detailed formulation and assessment of common comorbidities including anxiety, panic disorder, and depression.

CONCLUSION

Functional neurological disorder is a disabling and distressing condition that commonly presents to the ED and can take many forms. As a first point of contact, emergency physicians are well positioned to suspect the diagnosis of functional neurological disorder. The diagnosis of functional neurological disorder is based on identifying positive diagnostic phenomena that typically indicate a disorder of voluntary but not automatic movement or have other characteristic features. The treatment of functional neurological disorder begins in the ED by disclosing the potential diagnosis to patients in a clear manner, providing a brief explanation for why this diagnosis is suspected, and referring on to neurology for further treatment.

CONFLICT OF INTEREST

The authors have no potential conflicts to disclose.

AUTHOR CONTRIBUTIONS

Sara A. Finkelstein, Miguel A. Cortel-LeBlanc, Achelle Cortel-LeBlanc, and Jon Stone conceived and designed the content of the manuscript. Sara A. Finkelstein and Miguel A. Cortel-LeBlanc drafted the manuscript, and all authors contributed substantially to its revision. All authors approved the final version of the manuscript. Sara A. Finkelstein takes responsibility for the paper as a whole.

ORCID

Sara A. Finkelstein https://orcid.org/0000-0003-3578-9593
Miguel A. Cortel-LeBlanc https://orcid.org/0000-0002-9209-9401
Achelle Cortel-LeBlanc https://orcid.org/0000-0002-3263-5751
Jon Stone https://orcid.org/0000-0001-9829-8092

REFERENCES

1. American Psychiatric Association. *Diagnostic and statistical manual of mental disorders*. 5th ed. Arlington, VA: American Psychiatric Association; 2013.
2. Dula DJ, DeNaples L. Emergency department presentation of patients with conversion disorder. *Acad Emerg Med*. 1995;2(2):120–123.
3. Merkler AE, Parikh NS, Chaudhry S, et al. Hospital revisit rate after a diagnosis of conversion disorder. *J Neurol Neurosurg Psychiatry*. 2016;87(4):363–366.

4. Gelauff J, Stone J, Edwards M, Carson A. The prognosis of functional (psychogenic) motor symptoms: a systematic review. *J Neurol Neurosurg Psychiatry*. 2014;85(2):220–226.
5. Stephen CD, Fung V, Lungu CI, Espay AJ. Assessment of emergency department and inpatient use and costs in adult and pediatric functional neurological disorders. *JAMA Neurol*. 2021;78(1):88–101.
6. Williams ER, Guthrie E, Mackway-Jones K, et al. Psychiatric status, somatisation, and health care utilization of frequent attenders at the emergency department: a comparison with routine attenders. *J Psychosom Res*. 2001;50(3):161–167.
7. Beharry J, Palmer D, Wu T, et al. Functional neurological disorders presenting as emergencies to secondary care. *Eur J Neurol*. 2021;28(5):1441–1445.
8. Dickson JM, Dudhill H, Shewan J, Mason S, Grünewald RA, Reuber M. Cross-sectional study of the hospital management of adult patients with a suspected seizure (EPIC2). *BMJ Open*. 2017;7(7):e015696.
9. Walker MC, Howard RS, Smith SJ, Miller DH, Shorvon SD, Hirsch NP. Diagnosis and treatment of status epilepticus on a neurological intensive care unit. *QJM*. 1996;89(12):913–920.
10. Reuber M, Mitchell AJ, Elger CE, et al. Clinical significance of recurrent psychogenic nonepileptic seizure status. *J Neurol*. 2003;250(11):1355–1362.
11. Popkirov S, Stone J, Buchan AM. Functional neurological disorder: a common and treatable stroke mimic. *Stroke*. 2020;51(5):1629–1635.
12. Gibson LM, Whiteley W. The differential diagnosis of suspected stroke: a systematic review. *J R Coll Physicians Edinb*. 2013;43(2):114–118.
13. Jones AT, O'Connell NK, David AS. Epidemiology of functional stroke mimic patients: a systematic review and meta-analysis. *Eur J Neurol*. 2020;27(1):18–26.
14. Barsky AJ, Orav EJ, Bates DW. Somatization increases medical utilization and costs independent of psychiatric and medical comorbidity. *Arch Gen Psychiatry*. 2005;62(8):903–910.
15. Moeller JJ, Kurniawan J, Gubitz GJ, Ross JA, Bhan V. Diagnostic accuracy of neurological problems in the emergency department. *Can J Neurol Sci*. 2008;35(3):335–341.
16. Stone J, Zeman A, Simonotto E, et al. FMRI in patients with motor conversion symptoms and controls with simulated weakness. *Psychosom Med*. 2007;69(9):961–969.
17. Cojan Y, Waber L, Schwartz S, Rossier L, Forster A, Vuilleumier P. The brain under self-control: modulation of inhibitory and monitoring cortical networks during hypnotic paralysis. *Neuron*. 2009;62(6):862–875.
18. Van BM, De Jong BM, Gieteling EW, Renken R, Leenders KL. Abnormal parietal function in conversion paresis. *PLoS One*. 2011;6(10):e25918.
19. Voon V, Gallea C, Hattori N, Bruno M, Ekanayake V, Hallett M. The involuntary nature of conversion disorder. *Neurology*. 2010;74(3):223–228.
20. Stone J, Warlow C, Sharpe M. The symptom of functional weakness: a controlled study of 107 patients. *Brain*. 2010;133(Pt 5):1537–1551.
21. Stone J, Carson A, Aditya H, et al. The role of physical injury in motor and sensory conversion symptoms: a systematic and narrative review. *J Psychosom Res*. 2009;66(5):383–390.
22. Schrag A, Trimble M, Quinn N, Bhatia K. The syndrome of fixed dystonia: an evaluation of 103 patients. *Brain*. 2004;127(Pt 10):2360–2372.
23. Voon V, Cavanna AE, Coburn K, Sampson S, Reeve A, Curt LW. Functional neuroanatomy and neurophysiology of functional neurological disorders (conversion disorder). *J Neuropsychiatry Clin Neurosci*. 2016;28(3):168–90.
24. Macerollo A, Chen JC, Parees I, et al. Abnormal movement-related suppression of sensory evoked potentials in upper limb dystonia. *Eur J Neurol*. 2016;23(3):562–568.
25. Parees I, Saifee TA, Kassavetis P, et al. Believing is perceiving: mismatch between self-report and actigraphy in psychogenic tremor. *Brain*. 2012;135(1):117–123.
26. Edwards MJ, Adams RA, Brown H, et al. A Bayesian account of "hysteria". *Brain*. 2012;135(Pt 11):3495–3512.
27. Nielsen G, Stone J, Matthews A, et al. Physiotherapy for functional motor disorders: a consensus recommendation. *J Neurol Neurosurg Psychiatry*. 2015;86(10):1113–1119.
28. Espay AJ, Edwards MJ, Oggioni GD, et al. Tremor retrainment as therapeutic strategy in psychogenic (functional) tremor. *Park Relat Disord*. 2014;20(6):647–650.
29. Carson A, Hallett M, Stone J. Assessment of patients with functional neurologic disorders. In: Hallett M, Stone J, Carson A, eds. *Handbook of clinical neurology*. Vol. 139. Amsterdam: Elsevier B.V.; 2016:169–188.
30. Stone J, Carson A, Hallett M. Explanation as treatment for functional neurologic disorders. In: Hallett M, Stone J, Carson A, eds. *Handbook of clinical neurology*. Vol. 139. Amsterdam: Elsevier B.V.; 2016:543–553.
31. Stone J, Carson A, Sharpe M. Functional symptoms and signs in neurology: assessment and diagnosis. *J Neurol Neurosurg Psychiatry*. 2005;76:i2–i12.
32. Stone J, Warlow C, Sharpe M. Functional weakness: clues to mechanism from the nature of onset. *J Neurol Neurosurg Psychiatry*. 2011;83(1):67–69.
33. Gargalas S, Weeks R, Khan-Bourne N, et al. Incidence and outcome of functional stroke mimics admitted to a hyperacute stroke unit. *J Neurol Neurosurg Psychiatry*. 2017;88(1):2–6.
34. Stone J, Aybek S. Functional limb weakness and paralysis. In: Hallet M, Stone J, Carson A, eds. *Handbook of clinical neurology*. Vol. 139. Amsterdam: Elsevier B.V.; 2016:213–228.
35. Daum C, Gheorghita F, Spatola M, et al. Interobserver agreement and validity of bedside "positive signs" for functional weakness, sensory and gait disorders in conversion disorder: a pilot study. *J Neurol Neurosurg Psychiatry*. 2015;86(4):425–30.
36. McWhirter L, Stone J, Sandercock P, Whiteley W. Hoover's sign for the diagnosis of functional weakness: a prospective unblinded cohort study in patients with suspected stroke. *J Psychosom Res*. 2011;71(6):384–386.
37. Sonoo M. Abductor sign: a reliable new sign to detect unilateral non-organic paresis of the lower limb. *J Neurol Neurosurg Psychiatry*. 2004;75(1):121–125.
38. Gasca-Salas C, Lang AE. Neurologic diagnostic criteria for functional neurologic disorders. In: Hallett M, Stone J, Carson A, eds. *Handbook of clinical neurology*. Vol. 139. Amsterdam: Elsevier B.V.; 2016:193–212.
39. Ziv I, Djaldetti R, Zoldan Y, Avraham M, Melamed E. Diagnosis of "non-organic" limb paresis by a novel objective motor assessment: the quantitative Hoover's test. *J Neurol*. 1998;245(12):797–802.
40. Fasano A, Valadas A, Bhatia KP, et al. Psychogenic facial movement disorders: clinical features and associated conditions. *Mov Disord*. 2012;27(12):1544–1551.
41. Gould R, Miller BL, Goldberg MA, Benson DF. The validity of hysterical signs and symptoms. *J Nerv Ment Dis*. 1986;174(10):593–597.
42. Rolak LA. Psychogenic sensory loss. *J Nerv Ment Dis*. 1988;176(11):686–687.
43. Daum C, Aybek S. Validity of the "Drift without pronation" sign in conversion disorder. *BMC Neurol*. 2013;13(1):31.
44. Koehler PJ. Freud's comparative study of hysterical and organic paralyses: how Charcot's assignment turned out. *Arch Neurol*. 2003;60(11):1646–1650.
45. Chabrol H, Peresson G, Clanet M. Lack of specificity of the traditional criteria of conversion disorders. *Eur Psychiatry*. 1995;10:317–319.
46. Chernyshev OY, Martin-Schild S, Albright KC, et al. Safety of tPA in stroke mimics and neuroimaging-negative cerebral ischemia. *Neurology*. 2010;74(17):1340–1345.

47. Ali-Ahmed F, Federspiel JJ, Liang LI, et al. Intravenous tissue plasminogen activator in stroke mimics: findings from the get with the guidelines-stroke registry. *Circ Cardiovasc Qual Outcomes*. 2019;12(8):e005609.
48. Kostulas N, Larsson M, Kall TB, Von Euler M, Nathanson D. Safety of thrombolysis in stroke mimics: an observational cohort study from an urban teaching hospital in Sweden. *BMJ Open*. 2017;7(10):e016311.
49. Sivakumaran P, Gill D, Mahir G, Baheerathan A, Kar A. A retrospective cohort study on the use of intravenous thrombolysis in stroke mimics. *J Stroke Cerebrovasc Dis*. 2016;25(5):1057–1061.
50. Tsivgoulis G, Zand R, Katsanos AH, et al. Safety of intravenous thrombolysis in stroke mimics. *Stroke*. 2015;46(5):1281–1287.
51. Goyal N, Male S, Al Wafai A, Bellamkonda S, Zand R. Cost burden of stroke mimics and transient ischemic attack after intravenous tissue plasminogen activator treatment. *J Stroke Cerebrovasc Dis*. 2015;24(4):828–833.
52. Toth C. Hemisensory syndrome is associated with a low diagnostic yield and a nearly uniform benign prognosis. *J Neurol Neurosurg Psychiatry*. 2003;74(8):1113–1116.
53. Stone J, Vermeulen M. Functional sensory symptoms. In: Hallett M, Stone J, Carson A, eds. *Handbook of clinical neurology*. Vol. 139. Amsterdam: Elsevier B.V.; 2016:271–281.
54. Stone J, Sharpe M, Rothwell PM, Warlow CP. The 12 year prognosis of unilateral functional weakness and sensory disturbance. *J Neurol Neurosurg Psychiatry*. 2003;74(5):591–596.
55. Lindley RI, Warlow CP, Wardlaw JM, Dennis MS, Slattery J, Sandercock PA. Interobserver reliability of a clinical classification of acute cerebral infarction. *Stroke* 1993;24(12):1801–1804.
56. Reuber M, Jamnadas-Khoda J, Broadhurst M, et al. Psychogenic nonepileptic seizure manifestations reported by patients and witnesses. *Epilepsia*. 2011;52(11):2028–2035.
57. Vein AM, Djukova GM, Vorobieva OV. Is panic attack a mask of psychogenic seizures?–A comparative analysis of phenomenology of psychogenic seizures and panic attacks. *Funct Neurol*. 1994;9(3):153–159.
58. Hendrickson R, Popescu A, Dixit R, Ghearing G, Bagic A. Panic attack symptoms differentiate patients with epilepsy from those with psychogenic nonepileptic spells (PNES). *Epilepsy Behav*. 2014;37:210–214.
59. Galimberti CA, Teresa Ratti M, Murelli R, Marchioni E, Manni R, Tartara A. Patients with psychogenic nonepileptic seizures, alone or epilepsy-associated, share a psychological profile distinct from that of epilepsy patients. *J Neurol*. 2003;250(3):338–346.
60. Witgert ME, Wheless JW, Breier JI. Frequency of panic symptoms in psychogenic nonepileptic seizures. *Epilepsy Behav*. 2005;6(2):174–178.
61. Reuber M, Rawlings GH. Nonepileptic seizures – subjective phenomena. In: Hallett M, Stone J, Carson A, eds. *Handbook of clinical neurology*. Vol. 139. Amsterdam: Elsevier B.V.; 2016:283–296.
62. Goldstein LH, Mellers JD. Ictal symptoms of anxiety, avoidance behaviour, and dissociation in patients with dissociative seizures. *J Neurol Neurosurg Psychiatry*. 2006;77(5):616–621.
63. Tatum WO, Hirsch LJ, Gelfand MA, et al. Assessment of the predictive value of outpatient smartphone videos for diagnosis of epileptic seizures. *JAMA Neurol*. 2020;77(5):593–600.
64. Goldstein LH, Mellers JD. Recent developments in our understanding of the semiology and treatment of psychogenic nonepileptic seizures. *Curr Neurol Neurosci Rep*. 2012;12(4):436–444.
65. Ludwig L, McWhirter L, Williams S, Derry C, Stone J. Functional coma. In: Hallett M, Stone J, Carson A, eds. *Handbook of clinical neurology*. Vol. 139. Amsterdam: Elsevier B.V.; 2016:313–327.
66. Blad H, Lamberts RJ, Van DJ, Thijs RD. Tilt-induced vasovagal syncope and psychogenic pseudosyncope. *Neurology*. 2015;85(23):2006–2010.
67. Tannemaat MR, Van Niekerk J, Reijntjes RH, Thijs RD, Sutton R, Van Dijk JG. The semiology of tilt-induced psychogenic pseudosyncope. *Neurology*. 2013;81(8):752–758.
68. Sundararajan T, Tesar GE, Jimenez XF. Biomarkers in the diagnosis and study of psychogenic nonepileptic seizures: a systematic review. *Seizure Eur J Epilepsy*. 2016;35:11–22.
69. Doğan EA, Ünal A, Ünal A, Erdoğan Ç. Clinical utility of serum lactate levels for differential diagnosis of generalized tonic–clonic seizures from psychogenic nonepileptic seizures and syncope. *Epilepsy Behav*. 2017;75:13–17.
70. Matz O, Zdebik C, Zechbauer S, et al. Lactate as a diagnostic marker in transient loss of consciousness. *Seizure*. 2016;40:71–75.
71. Lou IA, Jensen ME, Lindelof M. Plasma-lactate levels in simulated seizures - an observational study. *Seizure*. 2020;76:47–49.
72. Lafrance WC, Ranieri R, Blum AS. Nonepileptic seizures – objective phenomena. In: Hallett M, Stone J, Carson A, eds. *Handbook of clinical neurology*. Vol. 139. Amsterdam: Elsevier B.V.; 2016:297–304.
73. Avbersek A, Sisodiya S. Does the primary literature provide support for clinical signs used to distinguish psychogenic nonepileptic seizures from epileptic seizures? *J Neurol Neurosurg Psychiatry*. 2010;81(7):719–725.
74. Stone J, Reuber M, Carson A. Functional symptoms in neurology: mimics and chameleons. *Pract Neurol*. 2013;13(2):104–113.
75. Syed TU, LaFrance WC, Kahriman ES, et al. Can semiology predict psychogenic nonepileptic seizures? A prospective study. *Ann Neurol*. 2011;69(6):997–1004.
76. Mostacci B, Bisulli F, Alvisi L, Licchetta L, Baruzzi A, Tinuper P. Ictal characteristics of psychogenic nonepileptic seizures : what we have learned from video/EEG recordings — a literature review. *Epilepsy Behav*. 2011;22(2):144–153.
77. Dhiman V, Sinha S, Rawat VS, Harish T, Chaturvedi SK, Satishchandra P. Semiological characteristics of adults with psychogenic nonepileptic seizures (PNESs): an attempt towards a new classification. *Epilepsy Behav*. 2013;27(3):427–432.
78. Dallocchio C, Matinella A, Arbasino C, et al. Movement disorders in emergency settings: a prospective study. *Neurol Sci* 2019;40(1):133–138.
79. Thenganatt MA, Jankovic J. Psychogenic tremor: a video guide to its distinguishing features. *Tremor Other Hyperkinet Mov (N Y)*. 2014;4:253.
80. Kenney C, Diamond A, Mejia N, Davidson A, Hunter C, Jankovic J. Distinguishing psychogenic and essential tremor. *J Neurol Sci*. 2007;263(1–2):94–99.
81. Gupta A, Lang AE. Psychogenic movement disorders. *Curr Opin Neurol*. 2009;22(4):430–436.
82. Barbey A, Aybek S. Functional movement disorders. *Curr Opin Neurol*. 2017;30(4):427–434.
83. Roper LS, Saifee TA, Parees I, Rickards H, Edwards MJ. How to use the entrainment test in the diagnosis of functional tremor. *Pract Neurol*. 2013;13(6):396–398.
84. Rooney A, Statham PF, Stone J. Cauda equina syndrome with normal MR imaging. *J Neurol*. 2009;256(5):721–725.
85. Bell DA, Collie D, Statham PF. Cauda equina syndrome - what is the correlation between clinical assessment and MRI scanning? *Br J Neurosurg*. 2007;21(2):201–203.
86. Hoeritzauer I, Pronin S, Carson A, Statham P, Demetriades AK, Stone J. The clinical features and outcome of scan-negative and scan-positive cases in suspected cauda equina syndrome: a retrospective study of 276 patients. *J Neurol*. 2018;265(12):2916–2926.
87. Hoeritzauer I, Carson A, Statham P, et al. Scan-negative cauda equina syndrome: a prospective cohort study. *Neurology*. 2021;96(3):e433–e447.
88. Stone J, Smyth R, Carson A, Warlow C, Sharpe M. La belle indifférence in conversion symptoms and hysteria: systematic review. *Br J Psychiatry*. 2006;188(3):204–209.

89. Crimlisk HL, Bhatia K, Cope H, David A, Marsden CD, Ron MA. Slater revisited: 6 year follow up study of patients with medically unexplained motor symptoms. *BMJ*. 1998;316(7131):582–586.
90. Carson AJ, Ringbauer B, Stone J, McKenzie L, Warlow C, Sharpe M. Do medically unexplained symptoms matter? A prospective cohort study of 300 new referrals to neurology outpatient clinics. *J Neurol Neurosurg Psychiatry*. 2000;68(2):207–210.
91. Carson A, Stone J, Hibberd C, et al. Disability, distress and unemployment in neurology outpatients with symptoms "unexplained by organic disease". *J Neurol Neurosurg Psychiatry*. 2011;82(7):810–813.
92. Feinstein A, Stergiopoulos V, Fine J, Lang AE. Psychiatric outcome in patients with a psychogenic movement disorder: a prospective study. *Neuropsychiatry Neuropsychol Behav Neurol*. 2001;14(3):169–176.
93. Diprose W, Sundram F, Menkes DB. Psychiatric comorbidity in psychogenic nonepileptic seizures compared with epilepsy. *Epilepsy Behav*. 2016;56:123–130.
94. Carson A, Lehn A. Epidemiology. In: Hallett M, Stone J, Carson A, eds. *Handbook of clinical neurology*. Vol. 139. Amsterdam: Elsevier B.V.; 2016:47–60.
95. Tolchin B, Baslet G, Dworetzky B. Psychogenic seizures and medical humor: jokes as a damaging defense. *Epilepsy Behav*. 2016;64(Pt A):26–28.
96. Wei D, Garlinghouse M, Li W, Swingle N, Samson KK, Taraschenko O. Utilization of brain imaging in evaluating patients with psychogenic nonepileptic spells. *Epilepsy Behav*. 2018;85:177–182.
97. Howard L, Wessely S, Leese M, et al. Are investigations anxiolytic or anxiogenic? A randomised controlled trial of neuroimaging to provide reassurance in chronic daily headache. *J Neurol Neurosurg Psychiatry*. 2005;76(11):1558–1564.
98. Sethi NK, Stone J, Edwards MJ. Trick or treat? Showing patients with functional (psychogenic) motor symptoms their physical signs. *Neurology*. 2013;80(9):869.
99. Sharpe M, Walker J, Williams C, et al. Guided self-help for functional (psychogenic) symptoms. *Neurology*. 2011;77(6):564–572.
100. Espay AJ, Aybek S, Carson A, et al. Current concepts in diagnosis and treatment of functional neurological disorders. *JAMA Neurol*. 2018;75(9):1132–1141.
101. Nielsen G, Buszewicz M, Stevenson F, et al. Randomised feasibility study of physiotherapy for patients with functional motor symptoms. *J Neurol Neurosurg Psychiatry*. 2017;88(6):484–490.
102. Jordbru AA, Smedstad LM, Klungsoyr O, Martinsen EW. Psychogenic gait: a randomized controlled trial on effect on rehabilitation. *J Rehabil Med*. 2014;46(2):181–187.
103. McCormack R, Moriarty J, Mellers JD, et al. Specialist inpatient treatment for severe motor conversion disorder: a retrospective comparative study. *J Neurol Neurosurg Psychiatry*. 2014;85(8):893–898.
104. Maggio JB, Ospina JP, Callahan J, Hunt AL, Stephen CD, Perez DL. Outpatient physical therapy for functional neurological disorder: a preliminary feasibility and naturalistic outcome study in a U.S. cohort. *J Neuropsychiatry Clin Neurosci*. 2020;32(1):85–89.
105. Goldstein LH, Robinson EJ, Mellers JD, et al. Cognitive behavioural therapy for adults with dissociative seizures (CODES): a pragmatic, multicentre, randomised controlled trial. *Lancet Psychiatry*. 2020;7(6):491–505.

How to cite this article: Finkelstein SA, Cortel-LeBlanc MA, Cortel-LeBlanc A, Stone J. Functional neurological disorder in the emergency department. *Acad Emerg Med*. 2021;28:685–696. https://doi.org/10.1111/acem.14263

PennState Health
Emergency Medicine
Residency Program Director

Penn State Health Milton S. Hershey Medical Center is seeking an Emergency Medicine Residency Program Director to join our exceptional academic team located in Hershey, PA. This is an excellent opportunity to join an outstanding academic program with a national reputation and inpact the lives of our future Emergency Medicine physicians.

What We're Offering:

- Competitive salary and benefits
- Sign-On Bonus
- Relocation Assistance
- Leadership for Emergency Medicine Residency Program
- Comprehensive benefit and retirement options

What We're Seeking:

- MD, DO, or foreign equivalent
- BC/BE by ABEM or ABOEM
- Leadership experience
- Outstanding patient care qualities
- Ability to work collaboratively within a diverse academic and clinical environment

FOR MORE INFORMATION PLEASE CONTACT:

Heather Peffley, PHR CPRP
Physician Recruiter
Penn State Health

Email: hpeffley@pennstatehealth.psu.edu
Website: careers.pennstatehealth.org

What the Area Offers:

Located in a safe family-friendly setting, Hershey, PA, our local neighborhoods boast a reasonable cost of living whether you prefer a more suburban setting or thriving city rich in theater, arts, and culture. Known as the home of the Hershey chocolate bar, Hershey's community is rich in history and offers an abundant range of outdoor activities, arts, and diverse experiences. We're conveniently located within a short distance to major cities such as Philadelphia, Pittsburgh, NYC, Baltimore, and Washington DC.