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Functional movement disorder and functional seizures: What have we learned from different subtypes of functional neurological disorders?

Sushma Kola [a], Kathrin LaFaver [a,b,*]

[a] Northwestern University Feinberg School of Medicine, Department of Neurology, Chicago, IL, United States
[b] Saratoga Hospital Medical Group - Neurology, Saratoga Springs, NY, United States

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ABSTRACT

The objective of this paper is to compare and contrast FMD and FS, and highlight important differences in etiology and the clinical approach towards these two entities. While patients with FMD often experience abnormal movements on a daily basis, FS is characterized by paroxysmal events. Both patient populations share psychiatric and environmental comorbidities, but patients with FS may have increased anxiety and neuroticism and a higher percentage of childhood trauma. Functional MRI scans have demonstrated impaired executive control over motor behavior in both groups. FMD responds well to multidisciplinary rehabilitation-oriented treatment, while psychotherapy remains the mainstay of treatment for FS. For practicing clinicians, recognizing commonalities and differences in patients with FMD and FS is important to develop the most appropriate treatment plan.

Contents

* Corresponding author at: Saratoga Hospital Medical Group – Neurology, 6 Care Ln, Saratoga Springs, NY, 12866, United States.
E-mail address: klafaver@saratogahospital.org (K. LaFaver).

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1. Case vignettes

1.1. Functional movement disorder

Sarah, a 45-year-old married woman and mother of two children, presented for neurological evaluation with concerns about leg weakness, abnormal gait, and balance problems. About six months before, she noted intermittent buckling of her left knee when running, and soon had switch to walks instead, which became shorter and shorter due to her increasing unsteadiness and easy fatigability. During the visit with her neurologist, she reported a high level of stress related to her work in retail with a difficult to please boss, a son with autism, and becoming progressively responsible for looking after her aging mother. Running, which she had done for many years to stay fit and release stress, had increasingly felt like a luxury that she no longer had time for. She also reported an old knee injury in her left leg many years ago. On examination of her strength, a positive Hoover's sign was noted, and her gait showed prominent left knee buckling, dragging the left forefoot, and swaying towards the examiner on Romberg's testing with eyes open and closed. A diagnosis of FMD was made and explained to Sarah using a biopsychosocial disease model. It was felt that her previous physical injury, her increased level of stress, and a behavioral pattern of putting her own needs behind those of others, played a role in symptom development and maintenance. A treatment plan was made that included working with a physical therapist and seeing a psychologist for cognitive behavioral therapy. Over the course of the next two months, Sarah was able to normalize her gait, regained control over her legs, and learned to identify thoughts and behavioral patterns that worked against her. Specifically, she was able to reduce her work hours and hire a family friend to help in the care of her mother. Several therapy sessions included her husband so he could understand the nature of her conditions and ways to support her. On follow-up three months later, she continued to do well, although it remained necessary for her to continue with physical therapy exercises and scheduled rest times on busy days.

1.2. Functional seizures

Tonya, a 22-year-old college student, was seen in the emergency room (ER) after a witnessed convulsive event. She had been in her dorm room studying and was about to lay down to rest because she had been feeling unwell. Her roommate observed her head started shaking from side to side, eyes forcefully closed, and extremities flailing from side to side. She called 911 and the spell continued for about 20 min, until she was given lorazepam in the ER. She had vague recollection of the event the next day but continued to feel foggy for 1–2 days, and had a headache. Over the following week, she had two additional spells similar to the first one, both occurring in her dorm room. Neurological exam in between spells was normal, and she had a normal MRI brain and routine EEG. Of note, she had a prior history of traumatic brain injury related to an abusive relationship as a teenager. She had been under increased stress over the past several weeks due to upcoming exams and poor school performance in the last semester. Due to a suspected diagnosis of functional seizures, she was admitted for video-EEG monitoring and a spell was captured, but did not show epileptiform activity. A diagnosis of functional seizures was explained to her, and she was referred to a psychologist experienced in the treatment of functional seizures. She learned several techniques to ground herself when she felt an episode coming on, how to recognize trigger factors early, and get support from her friends to avoid additional ER visits. Over the next few weeks, she had several additional spells with mild twitching in her arms, but no further events with loss of consciousness. She continued working with a psychologist and was started on treatment for previously undiagnosed anxiety.

2. Introduction

Functional neurologic disorder (FND) is a common and often disabling condition, attributed to complex biopsychosocial risk factors leading to dysfunction in the brain's signaling pathways. FND affects the way individuals interpret and interact with the world around them. Although prevalence data is difficult to ascertain, it is estimated that up to one-third of patients seeking evaluation by a neurologist suffer from functional neurological symptoms, associated with high costs for healthcare systems [1–5].

FND can manifest as a wide variety of phenotypes and cause significant disability. The most common presentations in clinical neurology include functional movement disorders (FMD) and FS, also known as psychogenic nonepileptic seizures (PNES). FMD is defined by the existence of abnormal, involuntary, hypo- or hyperkinetic movements that are incongruent with known pathology [6]. FS, on the other hand, are paroxysmal events that may resemble epileptic seizures without having ictal correlates on electroencephalogram (EEG) [7]. The objective of this paper is to 1) compare and contrast FMD and FS, and 2) review best practices to guide clinicians in recognizing and managing these conditions.

3. Clinical presentation

Symptoms of FND can be intermittent, fluctuate in severity, or become chronic. Patients with FMD may present with paroxysmal or continuous abnormal movements, and the phenomenology is broad. Common hyperkinetic movements include tics, myoclonus, chorea, dystonia, and tremor, whereas functional slowness, paralysis, or impaired gait are hypokinetic in nature [6]. FMD has several characteristics that can help clue in a physician to the correct diagnosis. Diagnosis relies on the neurological examination demonstrating inconsistency, variability, and incongruence. During the exam, movements may increase with attention or decrease with distraction, and tremor is non-rhythmic. Historically, the onset is often abrupt, patients may be asymptomatic between flares, and movements may be treatment-resistant. However, it is important to note that while these historical features can be supportive, they are neither necessary nor sufficient for diagnosis [8].

While patients with FMD are more likely to have chronic, disabling movements, those with FS experience paroxysmal, self-limited events. The semiology of these events can be described as alterations in physical or cognitive function, behaviors, sensations, or level of awareness. Patients may display jerking movements, repetitive eye blinking or rolling, grunting, paresthesias, or unresponsiveness [1]. In contrast to epileptic seizures, there is no post-ictal phase and patients generally return to their neurologic baseline quickly [9]. FS are similar to FMD in that episodes can be triggered by prompting or aborted with distraction, patients are neurologically intact between incidents, and attacks do not respond to antiseizure therapies [1,10].

4. Biopsychosocial model

Although the clinical presentations differ in patients with FMD and FS, both these subtypes of FND are presumed to share similar risk factors. Historically, the primary risk factors for FND were thought to be comorbid psychiatric disease, history of childhood abuse, and female gender [11]. Recently, however, environmental factors such as physical neglect have been found to play a role [12]. It can also be helpful to inquire about chronic pain, fatigue,

Table 1
Diagnostic criteria stratified by clinical confidence for functional movement disorders (FMD) vs. functional seizures (FS).

	Functional Movement Disorders	Functional Seizures
Documented	Patients who have complete resolution of FMD following a non-physiologic intervention (e.g., applying a vibrating tuning fork to the forehead).	Neurologist witnessed event showing typical FS semiology while on ictal video-EEG without EEG correlate.
Clinically Established	Inconsistent over time and incongruent with the broad phenotypic presentation of movement disorders.	Neurologist witnessed event showing typical FS semiology while not on EEG, no epileptiform activity on ictal EEG.

somatization, or unemployment, which are also commonly found in both patient populations [13–15].

It is not definitively known what causes FND-prone patients to manifest as FND versus FS. Some believe that differences in personality and life experiences may influence phenotype. Studies have shown that patients with FS have higher anxiety and neuroticism [16]. They also have an increased prevalence of childhood physical or sexual abuse, neglect, and borderline personality disorder when compared to those with FMD. In addition, their most disturbing trauma occurs at a younger age in patients with FS, likely accounting for their younger age at presentation [17]. Prevalence of mood disorders, gender breakdown, educational level, and employment status were not significantly different between both patient populations [16]. A detailed discussion describing the unifying and contrasting imaging features of FND follows in section 6.1 Pathophysiology.

5. Diagnostic evaluation

Gone are the days when FND was thought to be exclusively psychological and the diagnosis could only be made after a battery of normal investigations. It is now widely acknowledged that FND is not a diagnosis of exclusion, but rather a disorder of nervous system functioning with specific clinical features. Though many patients have psychological risk factors, some do not [18]. Traditional tests such as MRIs and EEGs are usually normal, and this emphasizes the importance of the physical exam [6]. The DSM-5, in fact, requires the presence of key signs to diagnose FND. A previous review described the positive clinical signs of functional weakness, sensory disturbances, and gait disorders. The authors found that Hoover sign, abductor sign, abductor finger sign, co-contraction, midline splitting, non-anatomical sensory loss, dragging monoplegic gait, and the chair test have been validated [19].

FMD are variable and incongruent with the traditional definitions of movement disorders. Thus, it is crucial to highlight FMD phenomenology and characteristics when making a diagnosis. Distractibility and fatigability on examination are common. FMD can present with a wide variety of phenomenology, such as tremor, dystonia, myoclonus, chorea, tics, ataxia, parkinsonism, or gait disorders [8]. Oftentimes, these manifestations exist in combination and afflict several body parts simultaneously. Recent developments in clinical neurology and neurophysiology have sparked the creation of diagnostic criteria for FMD. The criteria listed in Table 1 aid in the diagnosis of FMD and are stratified by clinical confidence [20]. Tremor is the most prevalent FMD, followed by dystonia. Functional tremor most frequently affects the upper extremities. Distractibility, entrainment, and co-activation should clue in the physician [21]. Blepharospasm, focal limb dystonia, and abductor laryngeal dystonia are the most common forms of functional dystonia. Functional chorea, ataxia, and parkinsonism are rare [22].

A diagnosis of FS should be considered when seizures are long in duration, inconsistent, have changing semiologies, can be recalled, are preceded by emotional stress, and when response to treatment is nonexistent, short-lived, or paradoxical [9,23]. Of note, these patients tend to have more frequent seizures and seizure-related hospitalizations, and may have a seizure in the physician's presence [1,23]. Non-epileptic status can also occur, as in the patient presented in the introductory vignette. The gold standard for diagnosis of FS is video-EEG that captures a typical event without electrographic correlate before, during, or after [7,23]. Similarly to a diagnosis of FMD, FS have levels of diagnostic confidence (Table 1) [10].

6. Pathophysiology

Recent advancements demonstrate abnormalities on functional and structural imaging in FND. Studies on FMD have looked at changes seen on functional MRI (fMRI), SPECT, and PET scans. With respect to fMRI in FMD, several studies have been done. One group found that patients with functional tremor had right temporoparietal junction hypoactivation and decreased connectivity to other cortices when compared to their volitional reproductions of the same movement [24]. A second study discovered that FMD patients had increased amygdalar activation to both positive and negative stimuli, suggesting increased functional connectivity of their amygdala and greater influence of emotions on motor behavior and planning [25]. Another group found that FMD patients had decreased left supplemental motor area activation associated with greater right amygdala, left anterior insula, and bilateral posterior cingulate cortex activation, possibly signifying decreased executive control over motor behavior [26]. When using SPECT imaging in FMD, patients with functional tremor had increased cerebral blood flow in the left insula and inferior frontal gyrus at rest. During their tremor, there was deceased bilateral ventromedial prefrontal cortex activation and increased right cerebellar hemisphere activation, suggesting impaired emotional regulation, awareness, and motor behavior [27].

Studies have also found structural abnormalities in the limbic system in patients with FMD. Mauer et al (2018) identified increased left amygdalar, left striatal, fusiform gyrus, cerebellar, and bilateral thalamic grey matter volumes when compared to controls. Left sensorimotor cortical volumes were reduced. Childhood trauma burden and depression positively correlated with left caudate and cerebellar tonsil volumes. Anxiety was inversely associated with left fusiform gyrus volumes [28]. A different study conversely found that patients with functional tremor had decreased left caudate and right postcentral grey matter volumes [29]. In FMD patients with functional dystonia, patients with mobile functional dystonia displayed decreased grey matter volumes compared to controls in the left nucleus accumbens, putamen, thalamus, and bilateral caudate. Voxel-based white matter analyses in the same study exhibited decreased fractional anisotropy in the corpus callosum, corticospinal tract, anterior thalamus, cingulate, and brainstem [30]. The above multimodality neuroimaging studies in FMD point to abnormal structure and pathophysiology disrupting the neurological circuitry responsible for proper emotional, cognitive, and motor control [31].

When it comes to FS, both structural and functional abnormalities have also been seen on neuroimaging. Two studies using fMRI demonstrated that patients with PNES had increased connectivity at baseline between the areas of the brain responsible for emotional processing, executive control, and movement [32,33]. A third study found that PNES patients had abnormal neurologic circuitry

on fMRI in regions involved in emotional, subcortical, and sensorimotor control [34].

Structural differences in FS have been studied quantitatively using voxel-based morphometry and cortical thickness analysis. Patients with FS had decreased volumes of grey matter and thinning in the right anterior cingulate cortex, supplementary motor cortex, and bilateral cerebellum, as well as and frontal, precentral, and paracentral gyri [35]. In summary, the above studies suggest that functional and structural alterations exist in patients with FS, and these findings could lead to further understanding of the underlying pathophysiology.

7. Treatment

Historically, FND was shown to have a poor long-term prognosis, with many patients demonstrating ongoing disability even after a decade [36–38]. Over the last decade, numerous studies have emphasized the potential for reversibility of FND with early diagnosis, education, and patient-tailored treatments [18]. In view of the complex presentation and comorbidities present, treatment should start with education of the patient and family about the diagnosis within a biopsychosocial framework. A multidisciplinary team-based approach to FND treatment is often paramount, which may include a neurologist, psychiatrist, psychologist, physiatrist, physical, occupational, and/or speech therapist, and clinical social worker. This approach has led to the formation of a few successful FND treatment centers across the United States [39]. Nonetheless, unique challenges exist in FND treatment. There is often high stigma associated with the diagnosis, misperception by the general public and healthcare providers that patients are malingering, lack of clarity regarding who should plan and oversee treatment, and poor access to treatment resources across the globe [36].

To help combat some of these challenges, an effective treatment strategy should begin with a clear and caring explanation of the positive clinical features that led to the FND diagnosis. Researchers have shown that patients with FS who do not receive feedback or intervention after a negative video EEG do not improve. Conversely, patients who receive a correct diagnosis via therapeutic communication can experience significant amelioration of symptoms [40]. Understanding their diagnosis helps patients realize that their condition is real, common, and treatable, which in turn empowers them to fully engage with the therapeutic process. Motivational interviewing has been shown to be helpful in patients with FS to improve adherence and therapy outcomes [41].

For patients with FMD, physical therapy (PT) from a therapist knowledgeable about FND is beneficial. The program involves treatments that focus on motor retraining, allowing patients to relearn normal movement patterns [11,18,42]. There were a few randomized control trials (RCT) for FMD, but all have had small sample sizes. The interventions studied include outpatient PT, inpatient rehabilitation, hypnosis, cognitive behavioral therapy (CBT) with physical therapy, CBT-based guided self-help, and interdisciplinary psychotherapy [18,43–49].

In the last decade, great advancements have led to validated treatments for patients with FS. CBT is the most widely used therapy, with clinical trials showing improvements in quality of life but no improvement in seizure frequency [50]. CBT helps patients understand their seizures, recognize preceding warning signs, and learn ways to regain control. Some patients may benefit from looking introspectively at their thoughts, feelings, and experiences that could have contributed to their symptoms [51]. Even patients without comorbid mood disorders can regain confidence through psychological therapies. Other advances include an open label study of sertraline followed by an RCT [52]. The combination of sertraline and CBT has successfully reduced functional seizures, and also improved patients' comorbidities, quality of life, and daily functioning [51,53]. Other treatment modalities such as short-term psychodynamic psychotherapy have demonstrated moderate to large improvements in several outcome domains, but access to therapists trained in this modality remains a treatment obstacle [54]. A recent pilot study showed excellent benefit in children with FS from retraining and control therapy (ReACT), using principles of habit reversal therapy [55]. A Cochrane review published in 2021 by the ILAE concluded that psychological interventions provide moderate improvement in health related quality of life for adults and adolescents with FS [56]. It also should be stressed that it is very important to make an emergency plan for patients with FS and instruct families in providing adequate safety for patients experiencing seizure-like symptoms in the home setting to avoid unnecessary hospitalizations and iatrogenic harm, such as when FS are mistakenly treated for presumptive status epilepticus.

Despite the advances made in treatment of FND, several questions remain in regards to optimal treatment setting, duration, frequency, and intensity of interventions. Furthermore, there are preliminary studies showing a potential benefit from neuromodulation techniques for treatment of FND such as transmagnetic stimulation which need to be explored further before wide application can be recommended [49,57].

8. Discussion

FND are common and encompass up to 30% of patients in outpatient neurology clinics. About 5% of patients in Movement Disorder Clinics have a functional disorder, and up to 30% of patients in Epilepsy Monitoring Units are diagnosed with functional seizures [4]. Despite a high prevalence in both inpatient and outpatient settings, many neurologists remain unfamiliar with the diagnosis and treatment of FND [58].

Similarities and differences exist between FMD and FS. FMD may present with continuous abnormal movements, while FS are self-limited. Both are often abrupt in onset, distractible, and medication-resistant. Shared risk factors include a history of psychiatric disease, abuse, trauma, and female gender. In addition, both patient populations have a higher prevalence chronic pain, fatigue, and somatization. Neuroimaging studies have demonstrated abnormal neurologic circuitry in regions involved in cognitive control over motor behavior in both FMD and FS. Despite the above similarities, treatment strategies differ. Multidisciplinary treatment is most effective for FMD, and psychotherapy and psychosocial support currently standard of care for FS.

Both FMD and FS represent different phenotypes under the umbrella diagnosis of FND. FND often causes chronic disability, is afflicted with a lack of public awareness and research efforts, and leads to a large economic burden on health care systems [5]. Unique challenges include the high stigma associated with the diagnosis, misperception by the general public and healthcare providers that patients are malingering, and poor access to treatment resources.

For the practicing clinician, developing a standard approach towards delivering the diagnosis of FND and providing educational resources to patients and families, developing a treatment plan and providing long-term follow-up for patients to oversee treatment outcomes and help with course corrections could be an important step towards improving care for patients with both FMD and FS [59].

In order to advance the field of FND further, several questions still need to be answered: What determines the phenomenology of FND in an individual? What is the ideal treatment setting? What is the optimal duration and intensity of therapy? What is the role of neuromodulation in FND? The rising recognition of FND as neu-

ropsychiatric disorders worthwhile of investigation and insights gained through neuroimaging research has stimulated multiple collaborative efforts among researchers and clinicians to find answers to complex questions of genetic, psychosocial and environmental factors involved in the pathophysiology of FND. Examples of such collaborations include the International FMD study group (https://www.movementdisorders.org/MDS/About/Committees–Other-Groups/Study-Groups/FunctionalMD-Study-Group.htm) and the International League against Epilepsy Psychology Task Force [56].

Such international collaborations and task forces are setting standards for diagnosis and treatment, textbooks are being published, and national foundations are increasingly providing financial support for FND research. On the treatment side, more efforts are still needed to form multidisciplinary teams to provide optimal treatment resources for patients with FND and to offer hope to a group of patients who have been misunderstood and stigmatized for far too long.

Declarations of interest

None. SK and KL have nothing to disclose.

Ethics

The manuscript is a review and did not include any research subjects. No IRB or informed consent was needed.

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References

[1] Paola LD et al. Psychogenic nonepileptic seizures and psychogenic movement disorders: two sides of the same coin? Arq Neuropsiquiatr 2014;72(10):793–802.
[2] Lempert T et al. Psychogenic disorders in neurology: frequency and clinical spectrum. Acta Neurol Scand 1990;82(5):335–40.
[3] Stone J et al. Symptoms 'unexplained by organic disease' in 1144 new neurology out-patients: how often does the diagnosis change at follow-up? Brain 2009;132(Pt 10):2878–88.
[4] Stone J et al. Who is referred to neurology clinics?–the diagnoses made in 3781 new patients. Clin Neurol Neurosurg 2010;112(9):747–51.
[5] Stephen CD et al. Assessment of emergency department and inpatient use and costs in adult and pediatric functional neurological disorders. JAMA Neurol 2021;78(1):88–101.
[6] Espay AJ, Lang AE. Phenotype-specific diagnosis of functional (psychogenic) movement disorders. Curr Neurol Neurosci Rep 2015;15(6):32.
[7] Asadi-Pooya AA et al. Terminology for psychogenic nonepileptic seizures: making the case for "functional seizures". Epilepsy Behav 2020;104:106895.
[8] Factor SA, Podskalny GD, Molho ES. Psychogenic movement disorders: frequency, clinical profile, and characteristics. J Neurol Neurosurg Psychiatry 1995;59(4):406–12.
[9] Asadi-Pooya AA, Sperling MR. Epidemiology of psychogenic nonepileptic seizures. Epilepsy Behav 2015;46:60–5.
[10] LaFrance WC et al. Minimum requirements for the diagnosis of psychogenic nonepileptic seizures: a staged approach: a report from the International League Against Epilepsy Nonepileptic Seizures Task Force. Epilepsia 2013;54(11):2005–18.
[11] Thomas M, Jankovic J. Psychogenic movement disorders: diagnosis and management. CNS Drugs 2004;18(7):437–52.
[12] Kranick S et al. Psychopathology and psychogenic movement disorders. Mov Disord 2011;26(10):1844–50.
[13] Hopp JL et al. Psychogenic seizures and psychogenic movement disorders: are they the same patients? Epilepsy Behav 2012;25(4):666–9.
[14] Driver-Dunckley E et al. Comparison of psychogenic movement disorders and psychogenic nonepileptic seizures: is phenotype clinically important? Psychosomatics 2011;52(4):337–45.
[15] Grimaldi I et al. Anxiety and depression in psychogenic movement disorder and non-epileptic seizures: a prospective comparative study. Rev Neurol (Paris) 2010;166(5):515–22.
[16] Ekanayake V et al. Personality traits in psychogenic nonepileptic seizures (PNES) and psychogenic movement disorder (PMD): Neuroticism and perfectionism. J Psychosom Res 2017;97:23–9.
[17] Stone J, Sharpe M, Binzer M. Motor conversion symptoms and pseudoseizures: a comparison of clinical characteristics. Psychosomatics 2004;45(6):492–9.
[18] Espay AJ et al. Current concepts in diagnosis and treatment of functional neurological disorders. JAMA Neurol 2018;75(9):1132. https://doi.org/10.1001/jamaneurol.2018.1264.
[19] Daum C 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.
[20] Fahn S, Williams DT. Psychogenic dystonia. Adv Neurol 1988;50:431–55.
[21] Bhatia KP, Schneider SA. Psychogenic tremor and related disorders. J Neurol 2007;254(5):569–74.
[22] Munhoz RP et al. Cross-cultural influences on psychogenic movement disorders - a comparative review with a Brazilian series of 83 cases. Clin Neurol Neurosurg 2011;113(2):115–8.
[23] Brown RJ et al. Psychogenic nonepileptic seizures. Epilepsy Behav 2011;22(1):85–93.
[24] Voon V et al. The involuntary nature of conversion disorder. Neurology 2010;74(3):223–8.
[25] Voon V et al. Emotional stimuli and motor conversion disorder. Brain 2010;133(5):1526–36.
[26] Voon V et al. Aberrant supplementary motor complex and limbic activity during motor preparation in motor conversion disorder. Mov Disord 2011;26(13):2396–403.
[27] Czarnecki K et al. SPECT perfusion patterns distinguish psychogenic from essential tremor. Parkinsonism Relat Disord 2011;17(5):328–32.
[28] Maurer CW et al. Gray matter differences in patients with functional movement disorders. Neurology 2018;91(20):e1870–9.
[29] Espay AJ et al. Impaired emotion processing in functional (psychogenic) tremor: a functional magnetic resonance imaging study. Neuroimage Clin 2018;17:179–87.
[30] Tomic A et al. Are there two different forms of functional dystonia? A multimodal brain structural MRI study. Mol Psychiatry 2020;25(12):3350–9.
[31] Perez DL et al. Neuroimaging in functional neurological disorder: state of the field and research agenda. Neuroimage Clin 2021;30:102623.
[32] van der Kruijs SJ et al. Functional connectivity of dissociation in patients with psychogenic non-epileptic seizures. J Neurol Neurosurg Psychiatry 2012;83(3):239–47.
[33] van der Kruijs SJ et al. Resting-state networks and dissociation in psychogenic non-epileptic seizures. J Psychiatr Res 2014;54:126–33.
[34] Ding JR et al. Altered functional and structural connectivity networks in psychogenic non-epileptic seizures. PLoS ONE 2013;8(5):e63850.
[35] Labate A et al. Neuroanatomic correlates of psychogenic nonepileptic seizures: a cortical thickness and VBM study. Epilepsia 2012;53(2):377–85.
[36] Adams C et al. You've made the diagnosis of functional neurological disorder: now what? Pract Neurol 2018;18(4):323–30.
[37] Gelauff J et al. The prognosis of functional (psychogenic) motor symptoms: a systematic review. J Neurol Neurosurg Psychiatry 2014;85(2):220–6.
[38] Stone J et al. The 12 year prognosis of unilateral functional weakness and sensory disturbance. J Neurol Neurosurg Psychiatry 2003;74(5):591–6.
[39] LaFaver, K., et al., Treatment of functional neurological disorder: current state, future directions, and a research agenda. CNS Spectr, 2020: p. 1-7.
[40] Gedzelman ER, LaRoche SM. Long-term video EEG monitoring for diagnosis of psychogenic nonepileptic seizures. Neuropsychiatr Dis Treat 2014;10:1979–86.
[41] Tolchin B et al. Motivational interviewing techniques to improve psychotherapy adherence and outcomes for patients with psychogenic nonepileptic seizures. J Neuropsychiatry Clin Neurosci 2020;32(2):125–31.
[42] Czarnecki K et al. Functional movement disorders: successful treatment with a physical therapy rehabilitation protocol. Parkinsonism Relat Disord 2012;18(3):247–51.
[43] Nielsen G et al. Randomised feasibility study of physiotherapy for patients with functional motor symptoms. J Neurol Neurosurg Psychiatry 2017;88(6):484–90.
[44] Jordbru AA et al. Psychogenic gait disorder: a randomized controlled trial of physical rehabilitation with one-year follow-up. J Rehabil Med 2014;46(2):181–7.
[45] Moene FC et al. A randomized controlled clinical trial of a hypnosis-based treatment for patients with conversion disorder, motor type. Int J Clin Exp Hypn 2003;51(1):29–50.
[46] Dallocchio C et al. Cognitive behavioural therapy and adjunctive physical activity for functional movement disorders (conversion disorder): a pilot, single-blinded, randomized study. Psychother Psychosom 2016;85(6):381–3.
[47] Sharpe M et al. Guided self-help for functional (psychogenic) symptoms: a randomized controlled efficacy trial. Neurology 2011;77(6):564–72.
[48] Hubschmid M et al. Efficacy of brief interdisciplinary psychotherapeutic intervention for motor conversion disorder and nonepileptic attacks. Gen Hosp Psychiatry 2015;37(5):448–55.
[49] Pick S et al. Trial Of Neurostimulation In Conversion Symptoms (TONICS): a feasibility randomised controlled trial of transcranial magnetic stimulation for functional limb weakness. BMJ Open 2020;10(10):e037198.
[50] Goldstein LH et al. Cognitive-behavioral therapy for psychogenic nonepileptic seizures: a pilot RCT. Neurology 2010;74(24):1986–94.

[51] LaFrance WC et al. Cognitive behavioral therapy for psychogenic nonepileptic seizures. Epilepsy Behav 2009;14(4):591–6.
[52] LaFrance WC et al. Nonepileptic seizures treatment workshop summary. Epilepsy Behav 2006;8(3):451–61.
[53] LaFrance WC et al. Multicenter pilot treatment trial for psychogenic nonepileptic seizures: a randomized clinical trial. JAMA Psychiatry 2014;71(9):997–1005.
[54] Abbass A et al. Short-term psychodynamic psychotherapy for functional somatic disorders: a systematic review and meta-analysis of within-treatment effects. J Psychosom Res 2021;145:110473.
[55] Fobian AD, Long DM, Szaflarski JP. Retraining and control therapy for pediatric psychogenic non-epileptic seizures. Ann Clin Transl Neurol 2020;7(8):1410–9.
[56] Michaelis R et al. Cochrane systematic review and meta-analysis of the impact of psychological treatment on health-related quality of life in people with epilepsy: an update by the ILAE Psychology Task Force, highlighting methodological changes. Epileptic Disord 2021.
[57] Garcin B et al. Impact of transcranial magnetic stimulation on functional movement disorders: cortical modulation or a behavioral effect? Front Neurol 2017;8:338.
[58] LaFaver K et al. Opinions and clinical practices related to diagnosing and managing functional (psychogenic) movement disorders: changes in the last decade. Eur J Neurol 2020;27(6):975–84.
[59] Perez DL, Haller AL, Espay AJ. Should neurologists diagnose and manage functional neurologic disorders? It is complicated. Neurol Clin Pract 2019;9(2):165–7.