

Functional neurological disorder: new subtypes and shared mechanisms

Mark Hallett, Selma Aybek, Barbara A Dworetzky, Laura McWhirter, Jeffrey P Staab, Jon Stone



Functional neurological disorder is common in neurological practice. A new approach to the positive diagnosis of this disorder focuses on recognisable patterns of genuinely experienced symptoms and signs that show variability within the same task and between different tasks over time. Psychological stressors are common risk factors for functional neurological disorder, but are often absent. Four entities—functional seizures, functional movement disorders, persistent perceptual postural dizziness, and functional cognitive disorder—show similarities in aetiology and pathophysiology and are variants of a disorder at the interface between neurology and psychiatry. All four entities have distinctive features and can be diagnosed with the support of clinical neurophysiological studies and other biomarkers. The pathophysiology of functional neurological disorder includes overactivity of the limbic system, the development of an internal symptom model as part of a predictive coding framework, and dysfunction of brain networks that gives movement the sense of voluntariness. Evidence supports tailored multidisciplinary treatment that can involve physical and psychological therapy approaches.

Introduction

The term functional neurological disorder conveys a condition in which the primary pathophysiological processes are alterations in functioning of brain networks rather than abnormalities of brain structures. Although long recognised, this disorder was largely neglected in medical teaching and by health-care workers from the mid to late 20th century, and was given various diagnostic labels including conversion, psychogenic, and dissociative disorders. Psychiatric diagnostic criteria are outlined in the fifth edition (text revision) of the *Diagnostic and Statistical Manual of Mental Disorders*, in which functional neurological disorder is referred to as functional neurological symptom disorder (conversion disorder). Diagnostic criteria have also been established in partnership by neurologists, psychiatrists, and other health-care professionals.^{1,2-4} In this Review, we define the term functional neurological disorder to denote clinical syndromes consisting of symptoms and signs of genuinely experienced alterations in motor, sensory, or cognitive performance, which are distressing or impairing, and manifest one or more patterns of deficits that are consistent predominantly with dysfunction of the nervous system, and show variability in performance within the same task and between different tasks.

This Review covers the most common presentations of functional neurological disorder, namely functional seizures (also called dissociative or psychogenic non-epileptic seizures) and functional movement disorders, including paresis. Chronic dizziness and cognitive dysfunction (as part of a functional disorder) have occupied an uncertain place in relation to functional neurological disorder, and we also cover them in this Review. We discuss possible shared mechanisms and distinguishing processes of these four entities, and note that individuals with functional neurological disorder often have more than one subtype, or alternate from one to another over time. Other frequent manifestations are somatosensory or visual symptoms and speech disorders, which we exclude from

this Review due to space constraints. Functional disorders have been defined in other medical specialties (eg, irritable bowel syndrome in gastroenterology, fibromyalgia in rheumatology, and painful bladder syndrome in urology). Comorbidity of functional disorders with each other, including functional neurological disorder, is common, which suggests either shared risk factors or a tendency for one disorder to lead to others over time. The relationship of other functional disorders to functional neurological disorder is uncertain, except for some shared predisposing and precipitating factors that we discuss in this Review.

We include evidence from neuroimaging, neurophysiological, and genetic studies, and consider new concepts of voluntary motor and sensory control to explain the unique alterations in neurological functioning that identify functional neurological disorder as a bona fide condition. Finally, we discuss improved diagnostic techniques, potential biomarkers, and emerging evidence for improved treatments based on a better understanding of mechanisms.

Epidemiology

Functional neurological disorder has been reported in people aged 4–94 years.^{5,6} There is a striking female preponderance (60–80%), although the gender gap is narrower in early and late life.^{7,8} Functional movement disorders have a lower female predominance than functional seizures, which might be due to interactions of age and trauma history with sex. The mean age of onset of functional movement disorders is in the late 30s⁷ versus functional seizures in the late 20s.⁸

Global and historical data suggest that functional neurological disorder has a similar prevalence (cases per population) across geographical regions⁹ and eras,¹⁰ although reported data are of poor quality due to inherent limitations in available sources and study designs. Moreover, the spectrum and nature of symptoms reported by individuals with functional neurological disorder (eg, seizures and paralysis) are similar in Brazil, Europe, India,

Lancet Neurol 2022; 21: 537–50

Published Online

April 14, 2022

[https://doi.org/10.1016/S1474-4422\(21\)00422-1](https://doi.org/10.1016/S1474-4422(21)00422-1)

51474-4422(21)00422-1

This online publication has been corrected. The corrected version first appeared at thelancet.com on May 11, 2022

See [Comment](#) page 499

Human Motor Control Section, National Institute of Neurological Disorders and Stroke, National Institutes of Health, Bethesda, MD, USA (Prof M Hallett MD); Psychosomatic Medicine Unit, Neurology Department, Bern University Hospital Inselspital, Bern, Switzerland (S Aybek MD); Department of Neurology, Brigham and Women's Hospital, Harvard Medical School, Boston, MA, USA (Prof B A Dworetzky MD); Centre for Clinical Brain Sciences, University of Edinburgh, Edinburgh, UK (L McWhirter PhD, Prof J Stone PhD); Department of Psychiatry and Psychology, and Department of Otorhinolaryngology - Head and Neck Surgery, Mayo Clinic, Rochester, MN, USA (Prof J P Staab MD)

Correspondence to: Dr Mark Hallett, Human Motor Control Section, National Institute of Neurological Disorders and Stroke, National Institutes of Health, Bethesda, MD 20892, USA hallettm@ninds.nih.gov

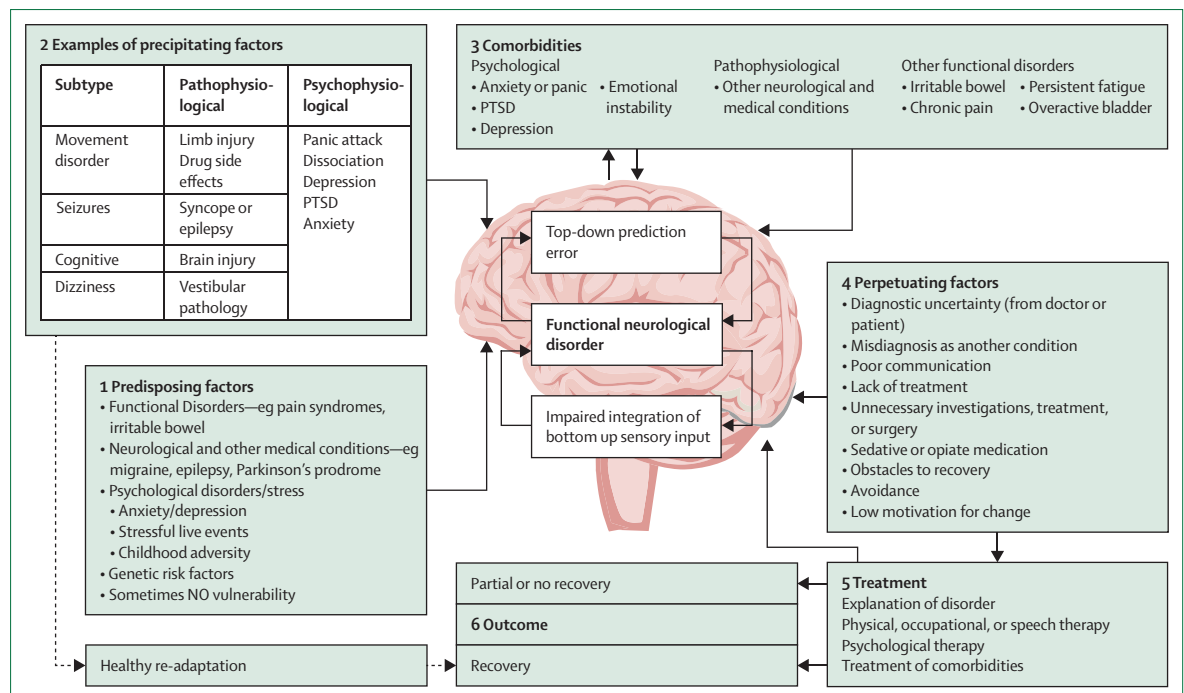


Figure 1: Pathophysiological and psychophysiological events that might trigger functional neurological disorder

The aetiology of functional neurological disorder depends on predisposing, precipitating, and perpetuating factors that affect the neural mechanisms of the disorder. The dotted line indicates that in most individuals the presence of these factors does not lead to functional neurological disorder. PTSD=post-traumatic stress disorder.

North America, and South Korea. Functional neurological disorder is the second most common reason for a new outpatient neurological consultation.¹¹ Population studies suggest a prevalence of 50–100 cases in 100 000,^{12–14} which would correspond to up to 300 000 people in the USA, for example. Levels of disability,¹¹ caregiver burden, and adverse effects on quality of life¹⁵ are comparable with those seen in other neurological conditions. Health-care costs for inpatient treatment of adults with functional neurological disorder in the USA exceed US\$1 billion annually.¹⁶ Access to diagnosis and treatment is problematic,¹⁷ because there are too few health-care workers who have knowledge about the disorder.¹⁸ Furthermore, the experience of the diagnosis is often stigmatising. Physicians often wrongly equate functional neurological disorder with feigning (ie, malingering).¹⁸

Predisposition, precipitants, perpetuants, and outcomes

Functional neurological disorder is multifactorial in origin (figure 1). Risk factors for the disorder in adults include exposure to psychological stressors and a history of childhood adversity, particularly neglect, with odds ratios averaging about 3–4.¹⁹ However, in many published studies, such events were not recorded in over 50% of people. The reported female preponderance in functional neurological disorder might be partly attributable to the higher exposure of females to childhood adversity,²⁰ although other disorders (eg, multiple sclerosis) are also

more common in women, and women are more likely to present to health services generally.²¹ Risk factors in children can include family dysfunction, bullying, perceived peer pressure, and (in some cases) abuse.²² Functional neurological disorder frequently coexists with depression, anxiety, post-traumatic stress disorder, and cluster B personality traits.^{23,24} Other functional somatic disorders, including chronic pain and irritable bowel syndrome are also common, which suggests common risk factors or mechanisms.^{25–27} Fatigue and pain have been shown to have more effect on quality of life than the symptoms of functional neurological disorder at a group level.^{28,15} Psychiatric comorbidities are not unique to functional neurological disorder, and are common in structural neurological disorders and other types of functional disorders.

Predisposition to functional neurological disorder probably includes genetic factors. In a study of 18 single nucleotide polymorphisms from 14 candidate genes among 69 patients with functional movement disorder,²⁹ the 703G→T polymorphism of the *TPH2* gene significantly predicted clinical manifestations and alterations in neurocircuitry. Compared with GG homozygotes, carriers of a T allele (including both GT heterozygotes and TT homozygotes) were younger at symptom onset, showed a greater interactive effect with childhood trauma in predicting symptom severity, and had less connectivity between the right amygdala and middle frontal gyrus. Epigenetic processes, such as methylation, could underlie

associations between genes, physiological reactivity, and environmental exposures (including childhood adversity). For example, individuals with functional neurological disorder might have less resilient responses to stress, as indicated by decreased 24-h heart rate variability, than healthy individuals of the same age and sex.³⁰ A pilot study of 15 patients with functional neurological disorder found increased methylation of the gene encoding the oxytocin receptor (*OXTR*), which plays a role in regulating responsivity to stress.³¹

Many clinicians associate functional neurological disorder exclusively with psychiatric morbidity, but observations beginning in the 19th century already found that structural illnesses and injuries can also predispose to or precipitate functional neurological disorder. For example, epilepsy can predate functional seizures,³² and migraine can trigger functional limb weakness.²⁶ Physical injury, acute illness, and drug side-effects appear to precipitate functional neurological disorder by causing novel nociceptive or unexpected sensory experiences.²⁶ Some neurological disorders are more likely than others to trigger comorbid functional neurological disorder.²⁶ For example, Parkinson's disease is more likely to be a precipitant of functional neurological disorder than Alzheimer's disease,³³ perhaps due to specific effects on motor-linked networks.

Clinicians often fear making diagnostic errors in patients who present with functional neurological disorder, but its misdiagnosis in neurological clinics is no more common than an incorrect diagnosis of other neurological and psychiatric disorders.³⁴ In fact, missing the diagnosis of functional neurological disorder could be a more frequent occurrence than making an erroneous diagnosis of functional neurological disorder.^{35,36}

The prognosis of functional neurological disorder is usually poorer than most clinicians expect. In a systematic review of 24 studies of functional motor disorders, corresponding to a total of 2069 patients, 40% of patients were unchanged (ie, they stayed the same) or had worse functional motor symptoms after a mean of 7 years, and only 20% of patients went into remission.³⁷ The frequency of remission of functional seizures (40–50%) is on average higher than that for other subtypes of functional neurological disorder, but considerable variability exists between studies.³⁷ Mortality is also higher in patients with functional seizures compared with those without functional seizures (eg, 2–3 fold increase), although the reasons for this greater mortality are unclear.³⁸

Functional seizures

Individuals with functional seizures, which are also known as psychogenic non-epileptic seizures or dissociative seizures, have episodes that resemble epilepsy or syncope. Making an accurate diagnosis can be a challenge because signs are transient and complete histories might only be available from witnesses, meaning that the diagnosis is usually delayed (table).^{39–53}

No one sign is pathognomonic of functional seizures. Studies have highlighted an overlap between preictal panic-like symptoms or signs and functional seizures,⁵² in which dissociation might occur in response to autonomic arousal and be perpetuated by classic and operant conditioning. In qualitative conversation analysis of seizure descriptions, functional seizures were distinguished from epilepsy with high sensitivity and specificity.⁴⁸ A machine learning algorithm processing clusters of responses to questionnaires from patients and observers showed promise in distinguishing epilepsy from syncope, but this algorithm is not yet ready for diagnosing individual patients.⁵⁴

The gold standard for diagnosis of functional seizures is use of video EEG (vEEG) recordings to assess the patient's typical episodes.⁵⁵ Some patients will have events during an outpatient EEG. Most (81%) patients who need long-term EEG monitoring will have events within 2 days.⁵⁶ Signs that have high sensitivity or specificity for functional seizures are long duration of events, fluctuating asynchronous limb or side-to-side

See Online for video

	Established diagnostic features	New diagnostic features*
Functional seizures		
Seizures	Eyes closed; prolonged attacks; hyperventilation; awareness during generalised shaking; ictal or post-ictal weeping	Suggestive seizure induction; ⁴⁸ qualitative conversation analysis; ⁴⁹ use of smartphone video; ⁵⁰ wrist-worn accelerometers; ⁵¹ post-ictal plasma proteins ⁵²
Functional movement disorder		
Tremor	Tremor entrainment or cessation to externally cued rhythm; variability of frequency and amplitude of tremors	"Whack-a-mole" sign: holding down a tremulous body part induces tremor in another body part; ⁴⁰ coherence between antagonist muscles measured with standard coherence or wavelets ⁴¹
Dystonia	Fixed inverted or plantar flexed ankle; fixed clenched fist	Dystonia of the face: downward lip pulling, orbicularis oculi spasm, platysma spasm; ⁴² sustained facial movement to evoke a spasm; ⁴² functional hemifacial spasm lacks the "other Babinski sign" (ie, raising of eyebrow on affected side)
Gait and balance	Variability of gait performance; gait performance shows excellent balance; "walking-on-ice" gait, dragging monoplegic gait, or knee-buckling gait	Classification of gait types into seven types: ataxic, spastic, weak gait, antalgic, parkinsonian, hemiparetic, and dystonic; ⁴³ "huffing and puffing" sign: huffing, grunting, grimacing, and breath holding after small amounts of exercise; ⁴⁰ posturographic improvement with distraction (guessing numbers written on back or cognitive task) ⁴⁴
Jerks or myoclonus	Truncal jerking, especially with facial movement†; positive Bereitschaftspotential before movement using back averaging	Increased startle; ⁴⁵ event related desynchronisation using back averaging ⁴⁴
Limb weakness and generic motor dysfunction	Hoover's sign; hip abductor sign; drift without pronation	Absence of amplitude suppression of median nerve somatosensory evoked potential; ⁴⁴ decreased prepulse inhibition of the blink reflex by stimulation of the index finger; ⁴⁶ absence of contingent negative variation in reaction time task ⁴⁷

*Described in the past 10 years. †The diagnosis of functional jerks can be difficult, but 104 (58%) of 179 patients with truncal myoclonus had functional neurological disorder in one series.⁵³

Table: Positive diagnostic features and biomarkers of functional seizures and functional movement disorder

head movements, pelvic thrusting, ictal eye closure, ictal crying, post-ictal memory recall,⁵⁵ and peri-ictal responsiveness³⁷ (video 1) The results of vEEG require interpretation by a skilled assessor because the vEEG can be normal in some types of focal epilepsy (eg, hypermotor frontal lobe seizures). In addition, interictal non-epileptiform abnormalities that are seen on vEEG in people with functional seizures are commonly misinterpreted by non-experts as epilepsy.⁵⁸

A drawback of vEEG monitoring is that equivocal or uneventful monitoring can lead to an indeterminate diagnosis. Also, in some settings, patients might not have access to specialised monitoring units. In these cases, a diagnosis can be made using the criteria established by the International League Against Epilepsy, for a less-than-certain diagnosis.¹ Video-only recordings interpreted by experts, including videos captured using a smartphone,⁴⁹ have shown acceptable reliability compared with vEEG.⁵⁹ Ambulatory EEG can also be useful when routine EEG is equivocal or when medication withdrawal is not needed.⁶⁰ In a prospective study, suggestive seizure induction (using non-deceptive and consented procedures) avoided the need for long-lasting and expensive vEEG in 8 (21%) of 39 patients with functional seizures, and decreased indeterminate testing in 5 (13%) of 38 patients.⁴⁷

Research on biomarkers to differentiate epileptic seizures from functional seizures is ongoing, but these new biomarkers are currently less reliable for diagnosis than patient history, risk factors, and ictal data. Prolactin and lactate concentrations in blood can be elevated after epileptic seizures, but a sample needs to be obtained within 1–2 h of the event. These concentrations can be within the normal range after focal seizures, and sometimes can be abnormal after functional seizures, so their use is not recommended.⁶¹ A diagnostic accuracy study of four plasma proteins sampled within 24 h of a seizure in 137 patients showed promising results for distinguishing functional seizures from epileptic seizures.⁵¹ Wrist-worn accelerometers⁵⁰ and surface electromyography⁶² both independently distinguished convulsive epileptic seizures from functional seizures with moderate sensitivity and specificity. Neuropsychological assessment does not distinguish functional seizures from epilepsy but can be helpful with other aspects, such as highlighting areas of poor cognitive functioning.⁵⁵

Functional movement disorders

Functional movement disorders can manifest with any type of abnormal movement.⁶³ Most case series have found that tremor is the most frequent abnormal movement (video 2), followed by dystonia,²⁵ myoclonus,⁴⁴ and gait disorders.⁴² Less common syndromes are parkinsonism,³³ tics,⁶⁴ stereotypy,⁶⁵ facial movements such as hemifacial spasms,⁴¹ and chorea. In a multicentre study of 410 patients with functional movement disorders, 188 (46%) had a mixture of movement disorder types.²⁷

Paresis and paralysis merit inclusion among manifestations of functional movement disorders because they commonly overlap with hyperkinetic movements and share similar pathophysiological alterations of higher control of brain networks involved in voluntary movement. In functional movement disorders, disruptions of voluntary movement contrast with preserved habitual or reflexive movements of the same body part, which is helpful, for example, in distinguishing functional movement disorders from isolated dystonia. Other common features of functional movement disorders included sudden onset of symptoms (in 71% of patients), high rates of comorbidity with anxiety (52%), fatigue (45%), and pain (42%).²⁷ Sudden onset of limb weakness is a common stroke mimic, after migraine and epilepsy. In a single-centre, retrospective study of 1165 people who were admitted for acute stroke-like presentations, 98 (8%) had functional neurological disorder, 163 (14%) had other medical conditions, and 904 (78%) had stroke.¹⁴

Diagnosis of functional movement disorders rests on key elements of clinical history and characteristic signs during examination (table). Although emphasis has been placed on examination, historical features can provide guidance for differential diagnosis. In a single-centre, retrospective study of 874 patients with hyperkinetic movement disorders, a model, which used an algorithm of historical features, had a discriminative ability of 91% to classify functional movement disorder.⁶⁶ The features that were more common included abrupt onset of symptoms, fluctuations of symptom severity during the day, a waxing and waning longitudinal course, pain or fatigue, and positive psychiatric features. In a single-centre, retrospective study of 99 patients with functional dystonia, an algorithm of similar historical features had good specificity (89%) and sensitivity (92%) against other types of dystonia.⁶⁷

A patient's movement patterns can be diagnostic (table).⁶³ Variability in patterns of movement can be detected by comparing movements initiated with the patient's full attention, movements performed under distraction, habitual movements (eg, shifting position in a chair), and movements induced by tendon reflexes. Various tests might show these differences, such as the Hoover's sign or the hip abductor sign of functional leg weakness, and the entrainment test for tremor.⁶³ Techniques of suggestibility and deception, such as the use of deceptive placebo, counter the principles of a patient's autonomy, unless undertaken after informed consent, and add little information to well gathered histories and non-deceptive examinations.

Clinical neurophysiological tests can aid diagnosis of functional movement disorders,⁶⁸ and are often specific to the movement disorder type.^{63,43} New methods might prove to be useful if validated (table). Functional neuroimaging and structural neuroimaging studies have identified group differences between patients with

Panel 1: Diagnostic criteria for persistent postural perceptual dizziness and functional cognitive disorder

These criteria for persistent postural perceptual dizziness were established by the Bárány Society in 2017.² Criteria for functional cognitive disorder have been proposed.⁴

Persistent postural perceptual dizziness

- One or more symptoms of dizziness, unsteadiness, or non-spinning vertigo on most days for at least 3 months
 - Symptoms last for prolonged (hours-long) periods of time but may wax and wane in severity
 - Symptoms or signs need not be present continuously throughout the entire day
 - Persistent symptoms occur without a specific stimulus, but are exacerbated by three factors: upright posture, active or passive motion without regard to direction or position, and exposure to moving visual stimuli or complex visual patterns
- The disorder is triggered by events that cause vertigo, unsteadiness, dizziness, or problems with balance, including acute, episodic, or chronic vestibular syndromes, other neurological or medical illnesses, and psychological distress
 - When triggered by an acute or episodic event, symptoms settle into a long-lasting, waxing and waning pattern that persists as the event resolves; they may occur intermittently at first, and then consolidate into a persistent course
 - When triggered by a chronic event, symptoms may develop slowly at first and worsen gradually
- Symptoms cause significant distress or functional impairment

- Symptoms are not better accounted for by another disease or disorder

Functional cognitive disorder

- One or more symptoms or signs of impaired cognitive function
- Clinical evidence of internal inconsistency
 - The ability to perform a task well in a particular cognitive domain at specific times, but with significantly impaired ability shown in the same domain, particularly when the task is the focus of attention
 - Not simply fluctuation over time
 - Consider red flags* for other cognitive disorders, which may present with features of inconsistency or fluctuation
- Symptoms or signs that are not better explained by another medical or psychiatric disorder, although comorbid disorders can occur
- Symptoms or signs that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning, or that warrant medical evaluation

*Red flags include: (1) inconsistency between cognitive domains (eg, impaired single word vs sentence comprehension in semantic dementia), difficulties relating to visual comprehension (eg, posterior cortical atrophy) that can produce effects similar to internal inconsistency (eg, the reverse size effect), effects of apathy or low mood, intact implicit memory with defective conscious memory (eg, Korsakoff syndrome); and (2) variability over time can occur in other disorders such as Lewy body dementia, delirium, and obstructive sleep apnoea.

functional movement disorders and comparison groups (ie, healthy individuals), but these differences cannot be used diagnostically for individual patients.⁴

Persistent postural perceptual dizziness

The Bárány Society (The International Society for Neuro-otology) defined the functional vestibular disorder of persistent postural perceptual dizziness for the International Classification of Vestibular Disorders (panel 1).^{2,4} Key symptoms of persistent postural perceptual dizziness are chronic dizziness, unsteadiness, and swaying or rocking (non-spinning) vertigo, which are exacerbated by the patient's own movements and exposure to visually complex or motion-rich environments (panel 2). The symptoms and syndromes of phobic postural vertigo, space motion discomfort, visual vertigo, and chronic subjective dizziness, going back more than 150 years, preceded and informed the diagnostic criteria of persistent postural perceptual dizziness.²

Studies from hospital-based neurology clinics found the prevalence of persistent postural perceptual dizziness to be 20% among all patients with vestibular symptoms, rising to 40% in a dedicated dizziness centre.⁶⁹ The median values for patient age in these

studies were 53, 55, and 59, with a two-to-one female predominance. Illnesses precipitating persistent postural perceptual dizziness in the patients documented in these studies included structural vestibular conditions, such as benign paroxysmal positional vertigo and unilateral peripheral vestibulopathies (23–25% of patients); vestibular migraine (11–20%); panic disorders (15%) and generalised anxiety disorders (15%); mild traumatic brain injuries (3–15%); stroke (2%); dysautonomia (1–7%); and other medical conditions (3–6%).⁶⁹

Investigations of patients with persistent postural perceptual dizziness and the four clinical entities that preceded it suggest that neuroticism could predispose to the condition. High levels of body vigilance (ie, conscious attention to internal cues) at the time of precipitating events (eg, illnesses) might initiate persistent postural perceptual dizziness, leading to functional shifts in stance and gait (eg, stiffened postural control, widened base of support, shorter stride length).⁶⁹ Patients with persistent postural perceptual dizziness also overly rely on visual inputs versus vestibular and somatosensory inputs (ie, they have visual dependence), making them susceptible to degradation of dynamic visual acuity when exposed to moving visual stimuli.⁶⁹ Perceptual and reflexive responses can dissociate, with

Panel 2: Case studies of functional neurological disorder

A patient with persistent postural perceptual dizziness

A 24-year-old woman presented with a history of persistent dizziness for the previous 2 years. She had an initial presentation of continuous spinning vertigo, nausea, and pyrexia and could hardly get out of bed for a week. The patient was diagnosed with vestibular neuronitis and given an expectation that she would recover over a period of days to weeks. During her recovery, the complaint of spinning vertigo changed to a non-specific feeling of dizziness that the patient found hard to describe. The feeling was no longer a spinning sensation but a feeling as if she was swaying or in motion, which was present mainly on standing and walking. Every now and then, the patient also felt as if she was floating and people seemed far away, which she found frightening. The patient described sensitivity to objects moving in her environment when she was still. It was difficult for her to use a computer or be in busy environments, such as supermarkets. The patient often thought about the possibility of falling; she had fallen once but felt that she had experienced some so-called near misses at times. Embarrassment at the thought of falling outside made her anxious, and she avoided busy places and going outside as much as possible. Over a 12-month period, the patient reported that the dizziness had started to take over her life. She developed symptoms of fatigue and poor concentration and had periods off work. She was desperate for an answer to what was causing the dizziness, and wondered why no one could tell her what was wrong and how to get better. Findings on neurological examination, including vestibular testing, were normal.

During Romberg's test, the patient reported a marked sense of sway, but no objective increase in sway was reported. Clinical assessment led to a diagnosis of persistent postural perceptual dizziness, according to Bárány Society criteria.² The initial triggering event was probable vestibular neuronitis with comorbidities of anxiety and dissociative symptoms. Treatment with a detailed explanation, vestibular habituation exercises, and cognitive behavioural therapy directed specifically towards the dizziness gradually improved the patient's symptoms over a 6-month period, such that they were only intermittent. The patient was able to resume social and work activities.

A patient with functional cognitive disorder

A 63-year-old woman presented to a neurology clinic with symptoms of memory loss. She was concerned that she could have Alzheimer's disease, like her mother. The patient had first realised that something might be wrong about 3 months previously, when she was at the bank machine and had forgotten her PIN code, which had never happened before. The patient reported that she lost focus during conversations and sometimes could not remember things that her husband insisted he had already told her. She began to use lists, notes, and reminders on her mobile phone, feeling sure that she would miss an appointment if she did not do these things.

The patient reported that, every day, she would walk into a room and forget what she was there for. Although she had never got lost when out of the house, the patient started to worry that this might happen. Her husband said that only he would drive the car, and they should only go out together. After letting a saucepan boil dry one evening, the patient stopped cooking and started to buy microwave meals so it would not happen again. She had trouble getting to sleep and would lie awake for hours worrying about the things she had to do the next day, and about how her family would manage as her dementia got worse.

The patient performed well on bedside cognitive testing, but she did not feel reassured by this result and believed something was wrong with her memory. The diagnosis of functional cognitive disorder was explained. The patient did not have a major depressive or anxiety disorder but had developed poor confidence in her memory, and she had developed abnormal attentional focus on memory lapses. Education (such as about the frequency of normal memory lapses in the general population), encouragement to rely on her memory more, and an altered focus on times that her memory was working very well began to persuade the patient about the diagnosis. The patient also became more able to control her anxiety about her memory. She stopped using notes and lists, and despite making some mistakes with shopping, the patient reported not nearly as many mistakes as expected. Although the patient still has concerns about getting dementia in the future, she has started seeing her friends again, and has stopped planning for the worst scenario.

thresholds for conscious detection of rotary stimuli reduced by more than half, whereas vestibular-ocular reflex responses remain unchanged.⁷⁰ Spatial navigation and internal representations of the body in space can be impaired.⁷¹

Functional cognitive disorder

Cognitive symptoms have traditionally been excluded from definitions of functional neurological disorder, but research from the past 5 years has highlighted positively identifiable symptoms and signs of the condition. Consortium diagnostic criteria, published in 2020, defined functional cognitive disorder as a condition of cognitive

symptoms with clear evidence of internal inconsistency, not better explained by another disorder, and causing distress or impairment, or warranting medical evaluation (panel 1).⁴ Similar to other functional neurological disorders, internal inconsistency refers to differences between occupational functioning and observed performance during interview, and variable patterns within cognitive tests. Analysis of the patient's behaviour and language during a consultation helps to discriminate functional cognitive disorder from neurodegenerative disorders. Patients with functional cognitive symptoms typically give detailed and specific descriptions of episodes of memory failure, are more likely to attend a

consultation alone than are patients with neurodegenerative disorders, and are more concerned about their symptoms than other people around them (panel 2).^{72,73} Performance validity tests within cognitive batteries might not be as helpful as previously thought for diagnosing functional cognitive disorders. Tests applied on their own are often positive in conditions such as epilepsy and mild cognitive impairment.⁷⁴

Functional cognitive disorder accounts for symptoms in a proportion of patients with subjective cognitive decline or mild cognitive impairment who do not go on to develop dementia. A meta-analysis of 12 003 patients attending memory clinics found that 2832 (24%) received descriptive diagnoses consistent with functional cognitive disorder.⁷⁵

Functional cognitive disorder can also coexist with structural and metabolic causes of cognitive decline. The relation can be comorbid, with both disorders contributing to disability or change over time, with anxiety-related prodromal functional symptoms predating onset of dementia. In some cases, a disease biomarker such as amyloid β detected by an amyloid PET scan could prove to be a false-positive finding, with the patient never progressing to degenerative illness.

Abnormal metacognition (ie, awareness and understanding of one's own thought processes) is a prominent feature in people with functional cognitive disorder.⁷⁶ Individuals with functional cognitive disorder describe cognitive failures (eg, walking into a room and not knowing why they are there) experienced by healthy people in everyday life.⁷⁷ They might perceive themselves as having a severe impairment despite good performance on cognitive tests or in occupational settings. Some patients consider that they had an above average memory in the past, although it remains unclear whether this inclination is a risk factor or consequence of the functional neurological disorder.

Various subtypes of functional cognitive disorder have been proposed,⁷⁵ including isolated cognitive symptoms, illness anxiety about dementia, cognitive symptoms in mixed functional neurological disorder,⁷⁸ and dissociative amnesia. A subtype of cognitive symptoms caused by anxiety or depressive disorders has been suggested, but anxiety and depression could also be viewed as comorbidities that exacerbate cognitive symptoms.⁷⁹

Distinguishing functional neurological disorder from feigning

Unfortunately, patients with functional neurological disorder are frequently suspected of feigning symptoms by clinicians,⁸⁰ and this scepticism has led to substantial stigma surrounding this condition.¹⁸ Suspicion about feigning arises because functional neurological disorder is a disorder of voluntary movement, in which performance changes with attention, and symptoms and disability might be variable. Currently, no data are available to suggest that feigning is more of a concern

with functional neurological disorder than with other neurological conditions that have symptoms such as pain or fatigue, or for conditions with psychiatric symptoms such as depression or anxiety.⁷⁷ The voluntary-automatic dissociation seen in patients with functional neurological disorder also exists during specific circumstances in other neurological conditions, such as stroke and Parkinson's disease (eg, automatic but impaired voluntary smiling in oropercular syndrome⁸¹).

Feigning is often divided into factitious disorder (wilfully simulating symptoms for medical care) or malingering (simulating symptoms for other gain). Both subtypes of feigning require evidence of conscious deception and action for identification.⁸² Evidence of feigning primarily involves a major discrepancy between reported and observed activity (ie, what the person says they can do vs what they are seen to do), such as someone claiming they cannot walk who is seen walking their dog. However, discrepancies between reported symptoms and observed daily activity, or variability in performance, that the patient reports usually do not provide evidence of feigning. For example, someone might say that their leg is always weak to some extent, and be in a wheelchair at an appointment, but might also state that at other times their leg weakness improves enough to allow them to walk the dog.

An expanding range of evidence supports functional neurological disorder as a genuinely experienced disorder. The observable manifestations of functional neurological disorder, aetiological risk factors, and coexistence with other medical and psychiatric conditions are similar around the world and throughout history, allowing for cultural differences in attributions of illness. Neurophysiological studies, including those looking at event-related potentials and sensory attenuation,^{83,84} and functional neuroimaging studies^{85–88} have identified differences between functional neurological disorder and feigning. Differential positive treatment responses obtained from randomised controlled trials of therapeutic interventions designed specifically for patients with functional neurological disorder^{89,90} are hard to explain if such studies were contaminated by the inclusion of individuals feigning illness.

Pathophysiology

The fundamental pathophysiology of functional seizures, functional movement disorders, persistent perceptual postural dizziness, and functional cognitive disorder can be considered in a similar way—as a dysfunction of sensory processing, motor or thought output, or both. Functional movement disorders, functional seizures, and functional cognitive disorder have prominent output abnormalities, whereas persistent postural perceptual dizziness has both sensory and motor dysfunction. Our understanding of the model is best explained in terms of movement, so we will describe it that way.

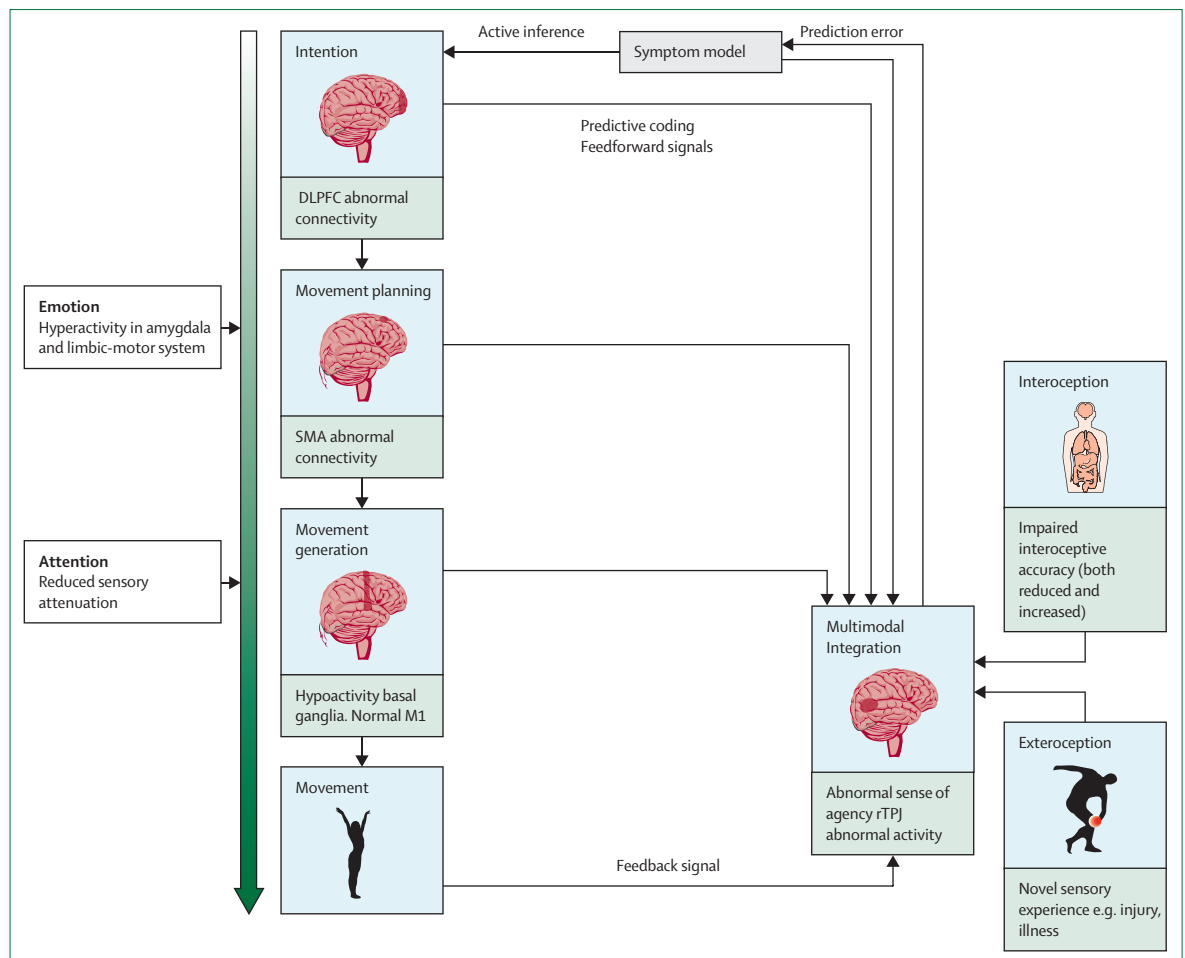


Figure 2: Neural mechanisms of functional neurological disorder

This scheme relates to functional movement disorder, but its principles are applicable to all functional neurological disorder entities. Movements are generated by the motor cortex after planning and preparation in the SMA. Planning and preparation of movement produce feedforward signals to be compared with feedback from interoceptive and external signals after action. If the signals do not match, movement will not be appreciated as voluntary. The brain has a model of the body and world that adds predictive coding to this multimodal integration. Feedback signals that do not match predictive coding create prediction error, which modifies the model so that predictive coding matches subsequent feedback. In functional neurological disorder, it is hypothesised that prediction error is not accurately updated, perpetuating dysfunction. The green arrow shows that there are multiple general influences on motor generation, including emotion and attention. DLPFC=dorsolateral prefrontal cortex. SMA=supplementary motor area. M1=primary motor area. rTPJ=right temporoparietal junction.

A key element of functional neurological disorder is partial loss of voluntary control over the body. Patients report feeling that they are not in control of their abnormal movements. This symptom can be understood in the frame of predictive coding models about how the brain generates the sense that the individual is the agent of what happens in their body, particularly when generating movements (figure 2). When a movement is planned, a motor command is sent to the motor cortex, which will execute the movement. In parallel, a feedforward signal goes to the so-called agency network (an important hub is the right temporoparietal junction). Once the movement is executed, feedback information goes to the agency network and a comparison between feedforward and feedback data occurs. When there is a good match in this comparison, the sense of agency arises.⁶³ A similar model has been developed to explain functional seizures and

functional somatic symptoms in general.^{91,92} Many studies converge to a mechanism of abnormal sense of agency, with neuroimaging studies pointing to abnormal activation of the right temporoparietal junction.^{63,43} An uncontrolled pilot study noted clinical improvement after non-invasive stimulation of the right temporoparietal junction in seven patients with functional seizures.⁹³

In functional neurological disorder, evidence points to an overweighting of the feedforward message under the influence of previous expectations, attention, and emotion (figure 2). A cognitive bias (ie, the tendency to jump to conclusions) found in patients with functional neurological disorder supports this hypothesis. Patients tend to favour their past expectation of an outcome over objective data regarding the future outcome.⁹⁴ An electrophysiological experiment provided evidence for slow sensory information processing in patients with functional

Panel 3: Diagnostic and treatment pitfalls in functional neurological disorder

Diagnostic pitfalls

Common reasons for a wrong diagnosis

- Jumping to conclusions based on a patient's psychological history or psychological stress
- Failure to consider an additional medical or neurological cause
- Not basing diagnosis on the presence of positive diagnostic signs (ie, features typical of functional neurological disorder)
- Diagnosis based on "bizarre" presentation alone
- Reliance on usual (ie, laboratory testing with results in the normal range) investigations

Common reasons for missing a diagnosis

- Absence of psychological risk factors or stress in the patient
- Failure of the clinician to consider functional neurological disorder in the presence of other medical or neurological conditions
- Insufficient knowledge of the clinician of positive features of functional neurological disorder
- Patient is a male old adult (the disorder is commonly overdiagnosed in young women and underdiagnosed in older men)
- Placing too much importance on incidental imaging findings (eg, white matter lesions)

Treatment pitfalls

The clinician should

- Explain the diagnosis to the patient on the basis of positive clinical features of functional neurological disorder
- When possible, show the patient positive clinical signs supporting the diagnosis and explain signs to the patient's family and friends
- Check and consolidate the patient's understanding of the diagnosis, consider copying correspondence to patients and signpost patients to online information and support organisations (eg, neurosymptoms.org, fndhope.org, fndaction.org.uk)

- Encourage early and active goal-directed rehabilitation and engage family and friends with that process
- Refer the patient for appropriate therapies (eg, physiotherapy, psychotherapy, speech-language therapy, or occupational therapy)
- Treat comorbidities (eg, depression, anxiety disorders [including post-traumatic stress disorder], or sleep disorders), and refer to psychiatry if necessary
- Review medication regimens; opiates, benzodiazepines, and other sedatives can worsen symptoms of functional neurological disorder
- Connect with, and train, other professionals to prevent the patient undergoing unnecessary and potentially harmful investigations or treatments

The clinician should not

- Make a diagnosis of functional neurological disorder on the basis of normal radiological or laboratory diagnostics
- Frame the patient's diagnosis as a medical mystery
- Highlight risk factors (eg, stress, psychological) when discussing possible causes
- Provide written information or signpost to online information without also providing treatment or referring the patient for further treatment
- Encourage unrealistic expectations; improvement is a gradual active process, and many patients do not improve
- Neglect to treat comorbid psychiatric disorders
- Withdraw medications suddenly or without explaining the reason
- Assume that any new symptoms are attributable to functional neurological disorder; the disorder could be comorbid with or precede other neurological disorders; new signs should be assessed on their own merits

Some common areas in which errors occur in the diagnosis and treatment of functional neurological disorder.^{34,103–105}

neurological disorder, suggesting a reduced attention allocation to objective body signals,⁹⁵ which could explain the shift towards an overemphasised feedforward signal. In patients with chronic functional dystonia, increased pain tolerance has been found, despite a normal pain threshold, indicating a dissociation between the discriminative and affective components of pain.⁹⁶ It can be postulated that attention also plays a role in filtering the feedback signal during multimodal integration, including emotional salience. Abnormal increased attention to the symptom also explains why explicit or deliberate movements (eg, lifting the leg during examination) are harder to execute than implicit or automatic movements, because a different motor programme is involved.^{63,97}

Increased activity in the limbic system seen on neuroimaging (ie, functional MRI) during motor and emotional tasks,⁹⁸ increased concentrations of cortisol, and

variability in heart rate⁹⁹ all point to abnormal stress reactivity and regulation of emotion.⁹⁸ These abnormalities can be important precipitating and perpetuating factors.

Findings in functional seizures and persistent postural perceptual dizziness, although less extensively studied, suggest similar abnormalities in agency and emotional networks that have been seen in functional neurological disorder. Functional neuroimaging studies in people with functional seizures highlight increased connectivity between the insular, motor, and parietal areas.¹⁰⁰ Small studies of ictal SPECT in patients with functional seizures highlighted abnormalities of agency and limbic networks.¹⁰¹ Changes in persistent postural perceptual dizziness are more nuanced. Activity and connectivity in vestibular cortical areas, including the posterior insula and parietal operculum, were decreased in individuals with persistent postural perceptual dizziness compared with those

without this condition, whereas connectivity between prefrontal and primary visual cortices was increased in proportion to symptom severity in individuals with this condition, an association modulated by anxiety and neuroticism.⁷¹

Several studies have shown brain anatomical differences (in grey matter and basal ganglia volumes) in patients with functional neurological disorder.¹⁰² Machine-learning paradigms have categorised individuals with functional neurological disorder and healthy controls with good accuracy, both with resting state functional and structural neuroimaging.⁸⁵ Correlation between structural changes and clinical data (ie, symptom severity, emotion, mood, dissociation, sex, and childhood trauma⁸⁵) strongly suggests a link between symptoms of functional neurological disorder and anatomical differences, but longitudinal studies are needed to address causality.

Treatment

Effective treatment for all types of functional neurological disorder begins by establishing a two-way communication between clinician and patient to improve understanding and engage patients in their own treatment (panel 3).^{34,103–105} Neurologists have historically avoided taking responsibility for the treatment of people with functional neurological disorder, although these clinicians are often the most appropriate experts to engage patients in treatment.¹⁰⁵ Explaining the diagnosis with clarity using the principles of a diagnosis of inclusion is a key step in treatment.¹⁰⁶ Patients should be provided with information, although a randomised trial showed that being provided with online information alone was insufficient to improve outcome.¹⁰⁷

Individualised treatment for patients with functional neurological disorder has gained support over a purely psychological therapeutic approach.^{34,108,109} A common theme of a multidisciplinary approach is that treatment is based on the new, mechanistic understanding of functional neurological disorder symptoms, and not just the individual's aetiological risk factors. For example, therapy for functional seizures now commonly focuses on the psychophysiological mechanism of the seizure events. Therapy for functional motor disorders builds on differences between automatic and voluntary movement that form the diagnosis and uses these differences in physical therapy. Clinical experience suggests that treatment for coexisting conditions (eg, migraine, orthostatic intolerance, anxiety, or depression) is also important.

Good evidence suggests that new approaches to physiotherapy, which are especially modified to the condition, can be used for the treatment of functional motor disorders.³⁴ For example, someone with stroke might be encouraged to focus on their affected limb during physiotherapy, whereas in functional motor disorder, explicit use of distraction and preserved automatic movements can be used to retrain aberrant motor function. In a randomised study in people with

functional motor disorders, 29 patients received a specific new physical therapy intervention and 28 patients received a similar number of treatment sessions of conventional physiotherapy (ie, treatment as usual).⁸⁹ At 6 months, 21 (72%) patients in the new physiotherapy group rated their symptoms as improved compared with 5 (18%) patients in the conventional physiotherapy group. A physical therapy programme can be managed by telemedicine and still be successful.¹¹⁰ In persistent postural perceptual dizziness, studies of vestibular rehabilitation¹¹¹ have reported reductions in patients' sensitivity to motion and dizziness. Occupational therapy can also have an important role, and consensus recommendations have been published.¹¹² However, occupational therapy has generally been only research as part of multidisciplinary treatment.¹⁰⁹

Various forms of psychotherapy have been shown to reduce morbidity in patients with functional seizures, but none are effective across all aspects of this illness. A systematic review found eight randomised controlled trials and 11 case series with medium sized benefits from psychotherapy for physical symptoms, mental health, wellbeing, function, and resource use.¹¹³ In the past decade, several randomised controlled trials have shown benefit from cognitive behavioural therapy (CBT)-informed psychotherapy over usual care for reduction of seizure frequency over periods of up to 6 months.¹¹³ In the largest randomised controlled trial, 368 patients with functional seizures received either CBT and standardised therapy or standardised therapy alone.⁹⁰ Both groups showed reductions in seizure frequency but no change in frequency of seizures was seen from adding CBT to standardised therapy. CBT did, however, lead to improvement in eight of 13 secondary outcomes, such as general functioning, distress, somatic symptoms, and duration of seizure freedom.⁹⁰ Other types of psychotherapy (eg, mindfulness-based psychotherapy, psychodynamic psychotherapy, and prolonged exposure therapy for individuals with concomitant post-traumatic stress disorder^{114,115}) have shown promising outcomes for patients with functional seizures in, mostly, small studies. Self-help treatments for stress reduction¹¹⁶ are safe and acceptable to patients with functional seizures, and they might be helpful for patients with scarce access to treatment. Evidence for the use of psychotherapy for patients with functional movement disorders is scant,¹¹³ and to our knowledge only one study on psychotherapy for functional cognitive disorder has been published.¹¹⁷ In patients with persistent postural perceptual dizziness, CBT enhanced the response to sertraline in a single-site, randomised, parallel arm trial,¹¹⁸ and acceptance and commitment therapy produced significant benefits when combined with vestibular rehabilitation.¹¹⁹

Up to now, trials of psychopharmacological treatments have been small and findings inconclusive for seizure and motor symptoms in functional neurological disorder, although treatment of comorbidity is less controversial.¹²⁰

For treatment of persistent postural perceptual dizziness, eight uncontrolled trials (totalling more than 300 patients) found that selective serotonin reuptake inhibitors and serotonin norepinephrine reuptake inhibitors reduced the mean severity of persistent postural perceptual dizziness or previous syndromes of persistent dizziness by 50% in 6–12 weeks.⁶⁹ For functional seizures, removing any antiseizure medications that are not treating common comorbidities (eg, anxiety, depression, or epilepsy) has been shown to be helpful and safe.¹²¹

Repetitive transcranial magnetic stimulation,¹²² including intermittent theta burst stimulation,¹²³ has had mixed results in functional motor disorders. A large placebo-controlled trial of botulinum toxin therapy for tremulous and jerky functional motor disorders showed no benefit, although both groups (botulinum toxin vs placebo) improved.¹²⁴ Treatments such as hypnotherapy, therapeutic sedation, and intensive inpatient treatment could help some patients.¹²⁵

Conclusions and future directions

Since 1968, when DSM-II was published, functional neurological disorder has been considered to comprise conditions involving the voluntary motor and sensory nervous system. Functional seizures are paroxysmal motor events, which is a strong reason to discuss them in this Review alongside functional motor disorders.²⁷ We also included in our Review two common functional neurological disorder subtypes involving dizziness and cognition, to explore their overlap with functional seizures and functional motor disorders (figure 1). We have not included sensory disorders (eg, functional anaesthesia or blindness) or other motor disorders (eg, speech and swallowing disorders), which have their own diagnostic features, but are based on similar principles to those we have discussed.

A striking feature of many functional disorders is that they are triggered by, and coexist with, recognised pathophysiological events. Functional motor disorders and functional seizures are often triggered by injury or other neurological disease. For persistent postural perceptual dizziness, an initial, usually vestibular, event is part of the definition. Functional cognitive disorder commonly follows mild traumatic brain injury, after the point when natural recovery might have been expected to occur. Psychological factors (eg, adversity, personality traits, or psychiatric disorder) might be relevant as predisposing, precipitating, or perpetuating factors at all stages of functional neurological disorder (figure 1). However, contrary to previous conversion disorder models, they are not a prerequisite. The strength of these triggering pathophysiological events, which themselves often shape the symptom of the subsequent functional disorder, could partly explain why there is such heterogeneity in the risk for functional neurological disorder.

The pathophysiology of functional neurological disorder overlaps in all its variants (figure 2). In all functional

Search strategy and selection criteria

References included in this Review were identified by searches of PubMed between Jan 1, 2016, to Nov 30, 2021, and from the references of relevant articles. The following search terms were used: “functional neurological disorder”; “conversion disorder”; “functional movement disorder”; “psychogenic movement disorder”; “psychogenic”, “dissociative” or “nonepileptic”, and “seizure”, “attack”, or “spell”; “persistent postural perceptual dizziness” and “functional cognitive disorder”. There were no language restrictions. The final reference list was generated on the basis of relevance to the topics covered in this Review.

neurological disorder entities, the patient develops an abnormal internal model of brain and body function. The model confirms and reinforces the abnormal function. The underlying push towards the abnormal model comes from a physical, cognitive, or emotional experience that cannot be subsumed into normal function. Functional neurological disorder is arguably what might be expected to happen when predictive processing in the brain goes awry.

Commonalities exist in the best therapeutic approaches for all these disorders. Physical therapy in functional motor disorders promotes automatic movements over abnormal overlearned impaired voluntary ones. In persistent postural perceptual dizziness, habituation exercises aim to desensitise a similarly aberrant sensory and motor system, in which there is an overlearned abnormal attentional spotlight on the symptom of dizziness. Similarly, psychotherapies, such as CBT for functional neurological disorder, aim to modify the various inputs that might contribute to the disorder, including interoception, attentional style, cognition, emotion, and psychological comorbidities.

Clearly, there are important differences between these diverse symptoms. Seizures are paroxysmal, whereas persistent postural perceptual dizziness and functional motor disorders are typically continuous and are arguably as different as panic disorder and depression. As with panic disorder and depression, functional motor disorders and persistent postural perceptual dizziness often coexist, and treatment typically improves with the understanding of their overlap.

The new principle of making a diagnosis of inclusion on the basis of clinical signs relates to increased understanding of pathophysiology and treatment, and allows diagnosis in people with other neurological conditions. Establishing reliability in the positive diagnosis of persistent postural perceptual dizziness and functional cognitive disorder will be especially important when evaluating their relevance to syndromes that are still to be clarified, such as the Havana syndrome and long COVID. With increasing availability of evidence-based treatment, the diagnosis of functional neurological disorder should be a process of looking for

potentially reversible causes of disability and distress, regardless of whether an individual has abnormalities on conventional laboratory or radiological testing.

Contributors

All authors contributed equally to the literature selection, conceptualisation, writing, and revision of this Review.

Declaration of interests

JS reports royalties from UpToDate for articles on functional neurological disorder and runs a free self-help website for people with functional neurological disorder. JS also carries out independent expert medicolegal work including in relation to functional neurological disorder, and is on the medical advisory board for FND Hope and FND Action. BAD receives royalties from Oxford University Press on her book *Psychogenic Nonepileptic Seizures: Toward the Integration of Care*. LM reports expert witness work in personal injury and negligence cases including functional neurological disorder. All other authors declare no competing interests.

Acknowledgments

MH is supported by the National Institute of Neurological Disorders and Stroke Intramural Program. BAD is supported by the Andrew J Trustey Research Fund. JS is supported by a National Health Service Scotland Research Career Fellowship. JPS is supported by a grant (W81XWH1810760) from the US Army Medical Research and Development Command via the Congressionally Directed Medical Research Program. SA is supported by a grant from the Swiss National Science Foundation (PP00P3_176985). LM is supported by a Baillie Gifford Clinical Research Fellowship and the NHS Scotland Chief Scientist's Office. We thank Stoyan Popkirov for the graphic concept in figure 1.

References

- LaFrance WC, Baker GA, Duncan R, Goldstein LH, Reuber M. 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**: 2005–18.
- Staab JP, Eckhardt-Henn A, Horii A, et al. Diagnostic criteria for persistent postural-perceptual dizziness (PPPD): consensus document of the committee for the classification of vestibular disorders of the barany society. *J Vestib Res Equilib Orientat* 2017; **27**: 191–208.
- Gupta A, Lang AE. Psychogenic movement disorders. *Curr Opin Neurol* 2009; **22**: 430–36.
- Ball HA, McWhirter L, Ballard C, et al. Functional cognitive disorder: dementia's blind spot. *Brain* 2020; **143**: 2895–903.
- Harris SR. Psychogenic movement disorders in children and adolescents: an update. *Eur J Pediatr* 2019; **178**: 581–85.
- Chouksey A, Pandey S. Functional movement disorders in elderly. *Tremor Other Hyperkinet Mov* 2019; **9**: 1–6.
- Lidstone SC, Costa-Parke M, Robinson EJ, Ercoli T, Stone J. Functional movement disorder gender, age and phenotype study: a systematic review and individual patient meta-analysis of 4905 cases. *J Neurol Neurosurg Psychiatry* 2022; published online Feb 22. <https://doi.org/10.1136/jnnp-2021-328462>.
- Goldstein LH, Robinson EJ, Reuber M, et al. Characteristics of 698 patients with dissociative seizures: a UK multicenter study. *Epilepsia* 2019; **60**: 2182–93.
- Kanemoto K, LaFrance WC, Duncan R, et al. PNES around the world: where we are now and how we can close the diagnosis and treatment gaps—an ILAE PNES Task Force report. *Epilepsia Open* 2017; **2**: 307–16.
- Stone J. Neurologic approaches to hysteria, psychogenic and functional disorders from the late 19th century onwards. *Handb Clin Neurol* 2016; **139**: 25–36.
- Carson A, Lehn A. Epidemiology. In: Hallett M, Stone J, Carson A, eds. *Handbook of Clinical Neurology Vol 139: Functional Neurologic Disorders*. Amsterdam: Elsevier, 2016: 47–60.
- Villagrán A, Eldøen G, Hofoss D, Ingvar M, Duncan R. Incidence and prevalence of psychogenic nonepileptic seizures in a Norwegian county: a 10-year population-based study. *Epilepsia* 2021; **62**: 1528–35.
- Duncan R, Razvi S, Mulhern S. Newly presenting psychogenic nonepileptic seizures: incidence, population characteristics, and early outcome from a prospective audit of a first seizure clinic. *Epilepsy Behav* 2011; **20**: 308–11.
- 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**: 2–6.
- Jones B, Reuber M, Norman P. Correlates of health-related quality of life in adults with psychogenic nonepileptic seizures: a systematic review. *Epilepsia* 2016; **57**: 171–81.
- 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**: 88–101.
- Hingray C, El-Hage W, Duncan R, et al. Access to diagnostic and therapeutic facilities for psychogenic nonepileptic seizures: an international survey by the ILAE PNES Task Force. *Epilepsia* 2018; **59**: 203–14.
- MacDuffie KE, Grubbs L, Best T, et al. Stigma and functional neurological disorder: a research agenda targeting the clinical encounter. *CNS Spectr* 2020; published online Dec 3. <https://doi.org/10.1017/S1092852920002084>.
- Ludwig L, Pasman JA, Nicholson T, et al. Stressful life events and maltreatment in conversion (functional neurological) disorder: systematic review and meta-analysis of case-control studies. *Lancet Psychiatry* 2018; **5**: 307–20.
- Kletenik I, Holden SK, Sillau SH, et al. Gender disparity and abuse in functional movement disorders: a multi-center case-control study. *J Neurol* 2022; published online Jan 31. <https://doi.org/10.1007/s00415-021-10943-6>.
- Hobbs FDR, Bankhead C, Mukhtar T, et al. Clinical workload in UK primary care: a retrospective analysis of 100 million consultations in England, 2007–14. *Lancet* 2016; **387**: 2323–30.
- Watson C, Sivaswamy L, Agarwal R, Du W, Agarwal R. Functional neurologic symptom disorder in children: clinical features, diagnostic investigations, and outcomes at a tertiary care children's hospital. *J Child Neurol* 2019; **34**: 325–31.
- Brown RJ, Reuber M. Psychological and psychiatric aspects of psychogenic non-epileptic seizures (PNES): a systematic review. *Clin Psychol Rev* 2016; **45**: 157–82.
- Kranick S, Ekanayake V, Martinez V, Ameli R, Hallett M, Voon V. Psychopathology and psychogenic movement disorders. *Mov Disord Off J Mov Disord Soc* 2011; **26**: 1844–50.
- Frucht L, Perez DL, Callahan J, et al. Functional dystonia: differentiation from primary dystonia and multidisciplinary treatments. *Front Neurol* 2021; **11**: 1–25.
- Tinazzi M, Geroi C, Erro R, et al. Functional motor disorders associated with other neurological diseases: beyond the boundaries of “organic” neurology. *Eur J Neurol* 2021; **28**: 1752–58.
- Tinazzi M, Morgante F, Marcuzzo E, et al. Clinical correlates of functional motor disorders: an Italian multicenter study. *Mov Disord Clin Pract* 2020; **7**: 920–29.
- Věchetová G, Slovák M, Kemlink D, et al. The impact of non-motor symptoms on the health-related quality of life in patients with functional movement disorders. *J Psychosom Res* 2018; **115**: 32–37.
- Spagnolo PA, Norato G, Maurer CW, et al. Effects of TPH2 gene variation and childhood trauma on the clinical and circuit-level phenotype of functional movement disorders. *J Neurol Neurosurg Psychiatry* 2020; **91**: 814–21.
- Maurer CW, Liu VD, LaFaver K, et al. Impaired resting vagal tone in patients with functional movement disorders. *Park Relat Disord* 2016; **30**: 18–22.
- Apazoglou K, Adouan W, Aubry J-M, Dayer A, Aybek S. Increased methylation of the oxytocin receptor gene in motor functional neurological disorder: a preliminary study. *J Neurol Neurosurg Psychiatry* 2018; **89**: 552–54.
- Kutlubaev MA, Xu Y, Hackett ML, Stone J. Dual diagnosis of epilepsy and psychogenic nonepileptic seizures: systematic review and meta-analysis of frequency, correlates, and outcomes. *Epilepsy Behav* 2018; **89**: 70–78.
- Ambar Akkaoui M, Geoffroy PA, Roze E, Degos B, Garcin B. Functional motor symptoms in Parkinson's disease and functional parkinsonism: a systematic review. *J Neuropsychiatry Clin Neurosci* 2020; **32**: 4–13.

- 34 Espay AJ, Aybek S, Carson A, et al. Current concepts in diagnosis and treatment of functional neurological disorders. *JAMA Neurol* 2018; **75**: 1132.
- 35 Xu Y, Nguyen D, Mohamed A, et al. Frequency of a false positive diagnosis of epilepsy: a systematic review of observational studies. *Seizure* 2016; **41**: 167–74.
- 36 Walzl D, Carson AJ, Stone J. The misdiagnosis of functional disorders as other neurological conditions. *J Neurol* 2019; **266**: 2018–26.
- 37 Gelauff J, Stone J. Prognosis of functional neurologic disorders. *Handb Clin Neurol* 2016; **139**: 523–41.
- 38 Nightscales R, McCartney L, Auvrez C, et al. Mortality in patients with psychogenic nonepileptic seizures. *Neurology* 2020; **95**: e643–52.
- 39 Gilmour GS, MacIsaac R, Subotic A, Wiebe S, Josephson CB. Diagnostic accuracy of clinical signs and symptoms for psychogenic nonepileptic attacks versus epileptic seizures: a systematic review and meta-analysis. *Epilepsy Behav* 2021; **121**: 108030.
- 40 Kramer G, Van der Stouwe AMM, Maurits NM, Tijssen MAJ, Elting JWJ. Wavelet coherence analysis: a new approach to distinguishing organic and functional tremor types. *Clin Neurophysiol* 2018; **129**: 13–20.
- 41 Stone J, Hoeritzauer I, Tesolin L, Carson A. Functional movement disorders of the face: a historical review and case series. *J Neurol Sci* 2018; **395**: 35–40.
- 42 Nonnekes J, Růžicka E, Serranová T, Reich SG, Bloem BR, Hallett M. Functional gait disorders. *Neurology* 2020; **94**: 1093–99.
- 43 Thomsen BLC, Teodoro T, Edwards MJ. Biomarkers in functional movement disorders: a systematic review. *J Neurol Neurosurg Psychiatry* 2020; **91**: 1261–69.
- 44 Dreissen YEM, Cath DC, Tijssen MAJ. Functional jerks, tics, and paroxysmal movement disorders. In: Hallett M, Stone J, Carson A, eds. *Handbook of clinical neurology vol 139: functional neurologic disorders*. Amsterdam: Elsevier, 2016: 247–58.
- 45 Hanzlíková Z, Kofler M, Slovák M, et al. Prepulse inhibition of the blink reflex is abnormal in functional movement disorders. *Mov Disord* 2019; **34**: 1022–30.
- 46 Teodoro T, Koreki A, Meppelink AM, et al. Contingent negative variation: a biomarker of abnormal attention in functional movement disorders. *Eur J Neurol* 2020; **27**: 985–94.
- 47 Popkirov S, Grönheit W, Jungilligens J, Wehner T, Schlegel U, Wellmer J. Suggestive seizure induction for inpatients with suspected psychogenic nonepileptic seizures. *Epilepsia* 2020; **61**: 1931–38.
- 48 Jenkins L, Cosgrove J, Chappell P, Kheder A, Sokhi D, Reuber M. Neurologists can identify diagnostic linguistic features during routine seizure clinic interactions: results of a one-day teaching intervention. *Epilepsy Behav* 2016; **64**: 257–61.
- 49 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**: 593.
- 50 Kusmakar S, Karmakar C, Yan B, et al. Novel features for capturing temporal variations of rhythmic limb movement to distinguish convulsive epileptic and psychogenic nonepileptic seizures. *Epilepsia* 2018; **60**: 165–74.
- 51 Gledhill JM, Brand EJ, Pollard JR, St. Clair RD, Wallach TM, Crino PB. Association of epileptic and nonepileptic seizures and changes in circulating plasma proteins linked to neuroinflammation. *Neurology* 2021; **96**: e1443–52.
- 52 Indranada AM, Mullen SA, Duncan R, Berlowitz DJ, Kanaan RAA. The association of panic and hyperventilation with psychogenic non-epileptic seizures: a systematic review and meta-analysis. *Seizure* 2018; **59**: 108–15.
- 53 van der Salm SM, Erro R, Cordivari C, et al. Propriospinal myoclonus: clinical reappraisal and review of literature. *Neurology* 2014; **83**: 1862–70.
- 54 Wardrope A, Jamnadas-Khoda J, Broadhurst M, et al. Machine learning as a diagnostic decision aid for patients with transient loss of consciousness. *Neurol Clin Pract* 2020; **10**: 96–105.
- 55 Baslet G, Bajestan SN, Aybek S, et al. Evidence-based practice for the clinical assessment of psychogenic nonepileptic seizures: a report from the American Neuropsychiatric Association Committee on Research. *J Neuropsychiatry Clin Neurosci* 2021; **33**: 27–42.
- 56 Zanzmera P, Sharma A, Bhatt K, et al. Can short-term video-EEG substitute long-term video-EEG monitoring in psychogenic nonepileptic seizures? A prospective observational study. *Epilepsy Behav* 2019; **94**: 258–63.
- 57 Wardrope A, Wong S, McLaughlan J, Wolfe M, Oto M, Reuber M. Peri-ictal responsiveness to the social environment is greater in psychogenic nonepileptic than epileptic seizures. *Epilepsia* 2020; **61**: 758–65.
- 58 Amin U, Benbadis SR. The role of EEG in the erroneous diagnosis of epilepsy. *J Clin Neurophysiol* 2019; **36**: 294–97.
- 59 Gasparini S, Beghi E, Ferlazzo E, et al. Management of psychogenic non-epileptic seizures: a multidisciplinary approach. *Eur J Neurol* 2019; **26**: 205–15.
- 60 Syed TU, LaFrance WC, Lodenkemper T, et al. Outcome of ambulatory video-EEG monitoring in a ~10,000 patient nationwide cohort. *Seizure* 2019; **66**: 104–11.
- 61 Sundararajan T, Tesar GE, Jimenez XF. Biomarkers in the diagnosis and study of psychogenic nonepileptic seizures: a systematic review. *Seizure* 2016; **35**: 11–22.
- 62 Husain AM, Towne AR, Chen DK, Whitmire LE, Voyles SR, Cardenas DP. Differentiation of epileptic and psychogenic nonepileptic seizures using single-channel surface electromyography. *J Clin Neurophysiol* 2021; **38**: 432–38.
- 63 Perez DL, Edwards MJ, Nielsen G, Kozłowska K, Hallett M, LaFrance, Jr WC. Decade of progress in motor functional neurological disorder: continuing the momentum. *J Neurol Neurosurg Psychiatry* 2021; **92**: 668–77.
- 64 Ganos C, Martino D, Espay AJ, Lang AE, Bhatia KP, Edwards MJ. Tics and functional tic-like movements: can we tell them apart? *Neurology* 2019; **93**: 750–58.
- 65 Baizabal-Carvallo JF, Jankovic J. Functional (psychogenic) stereotypies. *J Neurol* 2017; **264**: 1482–87.
- 66 Lagrand T, Tuitert I, Klamer M, et al. Functional or not functional; that's the question. *Eur J Neurol* 2021; **28**: 33–39.
- 67 Stephen CD, Perez DL, Chibnik LB, Sharma N. Functional dystonia: a case-control study and risk prediction algorithm. *Ann Clin Transl Neurol* 2021; **8**: 732–48.
- 68 Jackson L, Klassen BT, Hassan A, et al. Utility of tremor electrophysiology studies. *Clin Park Relat Disord* 2021; **5**: 100108.
- 69 Staab JP. Persistent postural-perceptual dizziness. *Semin Neurol* 2020; **40**: 130–37.
- 70 Wurthmann S, Holle D, Obermann M, et al. Reduced vestibular perception thresholds in persistent postural-perceptual dizziness: a cross-sectional study. *BMC Neurol* 2021; **21**: 394.
- 71 Indovina I, Passamonti L, Mucci V, Chiarella G, Lacquaniti F, Staab JP. Brain correlates of persistent postural-perceptual dizziness: a review of neuroimaging studies. *J Clin Med* 2021; **10**: 4274.
- 72 Reuber M, Blackburn DJ, Elsey C, et al. An interactional profile to assist the differential diagnosis of neurodegenerative and functional memory disorders. *Alzheimer Dis Assoc Disord* 2018; **32**: 197–206.
- 73 Williamson J, Larner A. Attended with and head-turning sign can be clinical markers of cognitive impairment in older adults. *Int Psychogeriatrics* 2018; **20**: 1569.
- 74 McWhirter L, Ritchie CW, Stone J, Carson A. Performance validity test failure in clinical populations—a systematic review. *J Neurol Neurosurg Psychiatry* 2020; **91**: 945–52.
- 75 McWhirter L, Ritchie C, Stone J, Carson A. Functional cognitive disorders: a systematic review. *Lancet Psychiatry* 2020; **7**: 191–207.
- 76 Bhome R, McWilliams A, Huntley JD, Fleming SM, Howard RJ. Metacognition in functional cognitive disorder—a potential mechanism and treatment target. *Cogn Neuropsychiatry* 2019; **24**: 311–21.
- 77 McWhirter L, King L, McClure E, Ritchie C, Stone J, Carson A. The frequency and framing of cognitive lapses in healthy adults. *CNS Spectr* 2021; published online Jan 22. <https://doi.org/10.1017/S1092852920002096>.
- 78 Teodoro T, Edwards MJ, Isaacs JD. A unifying theory for cognitive abnormalities in functional neurological disorders, fibromyalgia and chronic fatigue syndrome: systematic review. *J Neurol Neurosurg Psychiatry* 2018; **89**: 1308–19.
- 79 Bhome R, Huntley JD, Price G, Howard RJ. Clinical presentation and neuropsychological profiles of functional cognitive disorder patients with and without co-morbid depression. *Cogn Neuropsychiatry* 2019; **24**: 152–64.

- 80 Kanaan RA, Armstrong D, Wessely SC. Neurologists' understanding and management of conversion disorder. *J Neurol Neurosurg Psychiatry* 2011; **82**: 961–66.
- 81 Mantyh WG, Chandregowda A, Fulgham JR, Flemming KD, Laughlin RS, Jones DT. Teaching video neuroimages: Foix-Chavany-Marie syndrome. *Neurology* 2019; **92**: e2620–21.
- 82 Bass C, Halligan P. Factitious disorders and malingering in relation to functional neurologic disorders. In: Hallett M, Stone J, Carson A, eds. *Handbook of clinical neurology vol 139: functional neurologic disorders*. Amsterdam: Elsevier, 2016: 509–20.
- 83 Pareés I, Brown H, Nuruki A, et al. Loss of sensory attenuation in patients with functional (psychogenic) movement disorders. *Brain* 2014; **137**: 2916–21.
- 84 Stone J. Unfeignable biomarkers in functional neurological disorder: drifting back to Pierre Janet. *Brain* 2020; **143**: 393–95.
- 85 Perez DL, Nicholson TR, Asadi-Pooya AA, et al. Neuroimaging in functional neurological disorder: state of the field and research agenda. *Neuroimage Clin* 2021; **30**: 102623.
- 86 Cojan Y, Waber L, Carruzzo A, Vuilleumier P. Motor inhibition in hysterical conversion paralysis. *Neuroimage* 2009; **47**: 1026–37.
- 87 Hassa T, De Jel E, Tiescher O, Schmidt R, Schoenfeld MA. Functional networks of motor inhibition in conversion disorder patients and feigning subjects. *Neuroimage Clin* 2016; **11**: 719–27.
- 88 Voon V, Gallea C, Hattori N, Bruno M, Ekanayake V, Hallett M. The involuntary nature of conversion disorder. *Neurology* 2010; **74**: 223–28.
- 89 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**: 484–90.
- 90 Goldstein LH, Robinson EJ, Mellers JDC, et al. Cognitive behavioural therapy for adults with dissociative seizures (CODES): a pragmatic, multicentre, randomised controlled trial. *Lancet Psychiatry* 2020; **7**: 491–505.
- 91 Van den Bergh O, Witthöft M, Petersen S, Brown RJ. Symptoms and the body: taking the inferential leap. *Neurosci Biobehav Rev* 2017; **74**: 185–203.
- 92 Brown RJ, Reuber M. Towards an integrative theory of psychogenic non-epileptic seizures (PNES). *Clin Psychol Rev* 2016; **47**: 55–70.
- 93 Peterson KT, Kosior R, Meek BP, Ng M, Perez DL, Modirrousta M. Right temporoparietal junction transcranial magnetic stimulation in the treatment of psychogenic nonepileptic seizures: a case series. *Psychosomatics* 2018; **59**: 601–06.
- 94 Spagnolo PA, Garvey M, Hallett M. A dimensional approach to functional movement disorders: heresy or opportunity. *Neurosci Biobehav Rev* 2021; **127**: 25–36.
- 95 Sadnicka A, Daum C, Meppelink A-M, Manohar S, Edwards M. Reduced drift rate: a biomarker of impaired information processing in functional movement disorders. *Brain* 2020; **143**: 674–83.
- 96 Morgante F, Matinella A, Andrenelli E, et al. Pain processing in functional and idiopathic dystonia: an exploratory study. *Mov Disord* 2018; **33**: 1340–48.
- 97 Huys A-CML, Haggard P, Bhatia KP, Edwards MJ. Misdirected attentional focus in functional tremor. *Brain* 2021; **144**: 3436–50.
- 98 Baizabal-carvalho JF, Hallett M, Jankovic J. Pathogenesis and pathophysiology of functional (psychogenic) movement disorders. *Neurobiol Dis* 2019; **127**: 32–44.
- 99 Sundararajan T, Tesar GE, Jimenez XF. Biomarkers in the diagnosis and study of psychogenic nonepileptic seizures: a systematic review. *Seizure* 2016; **35**: 11–22.
- 100 Foroughi AA, Nazeri M, Asadi-Pooya AA. Brain connectivity abnormalities in patients with functional (psychogenic nonepileptic) seizures: a systematic review. *Seizure* 2020; **81**: 269–75.
- 101 Gallucci-Neto J, Brunoni AR, Ono CR, Fiore LA, Martins Castro LH, Marchetti RL. Ictal SPECT in psychogenic nonepileptic and epileptic seizures. *J Acad Consult Psychiatry* 2021; **62**: 29–37.
- 102 Bègue I, Adams C, Stone J, Perez DL. Structural alterations in functional neurological disorder and related conditions: a software and hardware problem? *Neuroimage Clin* 2019; **22**: 101798.
- 103 Hallett M, Stone J, Carson A. *Functional neurologic disorders: handbook of clinical neurology vol 139*. Amsterdam: Elsevier, 2016.
- 104 Stone J, Reuber M, Carson A. Functional symptoms in neurology: mimics and chameleons. *Pract Neurol* 2013; **13**: 104–13.
- 105 Fend M, Williams L, Carson AJ, Stone J. The Arc de Siècle: functional neurological disorder during the 'forgotten' years of the 20th century. *Brain* 2020; **143**: 1278–84.
- 106 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: functional neurologic disorders*. Amsterdam: Elsevier, 2016: 543–53.
- 107 Gelauff JM, Rosmalen JGM, Carson A, et al. Internet-based self-help randomized trial for motor functional neurologic disorder (SHIFT). *Neurology* 2020; **95**: e1883–96.
- 108 Schmidt T, Ebersbach G, Oelsner H, et al. Evaluation of individualized multi-disciplinary inpatient treatment for functional movement disorders. *Mov Disord Clin Pract* 2021; **8**: 911–18.
- 109 Gilmour GS, Jenkins JD. Inpatient treatment of functional neurological disorder: a scoping review. *Can J Neurol Sci* 2021; **48**: 204–17.
- 110 Demartini B, Bombieri F, Goeta D, Gambini O, Ricciardi L, Tinazzi M. A physical therapy programme for functional motor symptoms: a telemedicine pilot study. *Parkinsonism Relat Disord* 2020; **76**: 108–11.
- 111 Nada EH, Ibraheem OA, Hassaan MR. Vestibular rehabilitation therapy outcomes in patients with persistent postural-perceptual dizziness. *Ann Otol Rhinol Laryngol* 2019; **128**: 323–29.
- 112 Nicholson C, Edwards MJ, Carson AJ, et al. Occupational therapy consensus recommendations for functional neurological disorder. *J Neurol Neurosurg Psychiatry* 2020; **91**: 1037–45.
- 113 Gutkin M, McLean L, Brown R, Kanaan RA. Systematic review of psychotherapy for adults with functional neurological disorder. *J Neurol Neurosurg Psychiatry* 2021; **92**: 36–44.
- 114 Carlson P, Nicholson Perry K. Psychological interventions for psychogenic non-epileptic seizures: a meta-analysis. *Seizure* 2017; **45**: 142–50.
- 115 Myers L, Vaidya-Mathur U, Lancman M. Prolonged exposure therapy for the treatment of patients diagnosed with psychogenic non-epileptic seizures (PNES) and post-traumatic stress disorder (PTSD). *Epilepsy Behav* 2017; **66**: 86–92.
- 116 Novakova B, Harris PR, Rawlings GH, Reuber M. Coping with stress: a pilot study of a self-help stress management intervention for patients with epileptic or psychogenic nonepileptic seizures. *Epilepsy Behav* 2019; **94**: 169–77.
- 117 Bhome R, Berry AJ, Huntley JD, Howard RJ. Interventions for subjective cognitive decline: systematic review and meta-analysis. *BMJ Open* 2018; **8**: e021610.
- 118 Yu Y-C, Xue H, Zhang Y, Zhou J. Cognitive behavior therapy as augmentation for sertraline in treating patients with persistent postural-perceptual dizziness. *Biomed Res Int* 2018; **2018**: 1–6.
- 119 Kuwabara J, Kondo M, Kabaya K, et al. Acceptance and commitment therapy combined with vestibular rehabilitation for persistent postural-perceptual dizziness: a pilot study. *Am J Otolaryngol* 2020; **41**: 102609.
- 120 Gilmour GS, Nielsen G, Teodoro T, et al. Management of functional neurological disorder. *J Neurol* 2020; **267**: 2164–72.
- 121 Oto M, Espie C, Pelosi A, Selkirk M, Duncan R. The safety of antiepileptic drug withdrawal in patients with non-epileptic seizures. *J Neurol Neurosurg Psychiatry* 2005; **76**: 1682–85.
- 122 Pick S, Hodsoll J, Stanton B, 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**: e037198.
- 123 Spagnolo PA, Parker J, Horovitz S, Hallett M. Corticolimbic modulation via intermittent theta burst stimulation as a novel treatment for functional movement disorder: a proof-of-concept study. *Brain Sci* 2021; **11**: 791.
- 124 Dreissen YEM, Dijk JM, Gelauff JM, et al. Botulinum neurotoxin treatment in jerky and tremulous functional movement disorders: a double-blind, randomised placebo-controlled trial with an open-label extension. *J Neurol Neurosurg Psychiatry* 2019; **90**: 1244–50.
- 125 Gilmour GS, Jenkins JD. Inpatient treatment of functional neurological disorder: a scoping review. *Can J Neurol Sci* 2021; **48**: 204–17.

Copyright © 2022 Elsevier Ltd. All rights reserved.