A practical review of functional neurological disorder (FND) for the general physician

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We present a practical overview of functional neurological disorder (FND), its epidemiology, assessment and diagnosis, diagnostic pitfalls, treatment, aetiology and mechanism. We present an update on functional limb weakness, tremor, dystonia and other abnormal movements, dissociative seizures, functional cognitive symptoms and urinary retention, and ‘scan-negative’ cauda equina syndrome. The diagnosis of FND should rest on clear positive evidence, typically from a combination of physical signs on examination or the nature of seizures. In treatment of FND, clear communication of the diagnosis and the involvement of the multidisciplinary team is beneficial. We recommend that patients with FND are referred to specialists with expertise in neurological diagnosis. FND is a common presentation in emergency and acute medical settings and there are many practical elements to making a positive diagnosis and communication which are useful for all physicians to be familiar with.

KEYWORDS: functional neurological disorder, conversion disorder, dissociative seizures, treatment

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Introduction

Functional neurological disorder (FND) describing motor and sensory symptoms (such as paralysis, tremor and seizures), that are genuinely experienced and related to a functional rather than a structural disorder, is perhaps one of the oldest and most recognisable of the functional disorders. Its other names (conversion disorder, psychogenic disorder and, in the past, hysteria) indicate what a large historical burden the disorder bears.

In the last 10–15 years, there has been a renaissance of interest in this clinical area. Where neurology was once guilty of ignoring its functional disorders more than most specialties, the FND research community is now flourishing with a new international FND society (www.fndsociety.org) which is multidisciplinary and multiperspective. FND is finding its way into mainstream neurology training curricula and textbooks as a disorder at the interface between neurology and psychiatry.

Several developments have helped with this process. At the bedside, there has been a rediscovery of positive clinical signs of FND, mostly long known about but previously kept hidden in neurological textbooks and curricula. Videotelemetry electroencephalography (EEG) and high-resolution structural neuroimaging has made it clear that some people with severely disabling disorders do not have structural pathology. Functional neuroimaging and neurophysiology have helped to show that people with these disorders do have something wrong with the functioning of their brain which looks different to feigning. Lastly, neurologists are now much more diverse, and arguably less willing to take the often dismissive ‘I have excluded disease – it’s not my problem,’ approach popular in the past.

Here, we present a practical overview of FND, its diagnosis, treatment, aetiology and mechanism. We recommend that patients with FND are referred to specialists with expertise in neurological diagnosis. There are many pitfalls to diagnosis. Nonetheless, FND is a common presentation in emergency and acute medical settings and there are many practical elements to making a positive diagnosis and communication which are useful for all physicians to be familiar with.

Epidemiology of FND

FND is a common cause of disability and distress, especially in neurological practice. Functional disorders represent the second commonest reason to see a neurologist after headache. More tightly defined FND still accounts for at least 5%–10% of new neurological consultations. Estimates of incidence are conservatively 12 per 100,000 per year. Based on this, around 8,000 new diagnoses of FND are made per year in the UK and around 50,000–100,000 people have it in the community. FND disproportionately affects women (around 3:1) although, as age of onset increases, the proportion of men affected increases. Incident cases demonstrate that FND can occur across all ages, from young children (although it is rare before 10 years old) up to patients in their 80s.
FND symptoms are associated with high levels of physical disability, equivalent to people with multiple sclerosis or epilepsy, and even higher frequencies of psychological comorbidities than these disorders. Other functional disorders (such as irritable bowel syndrome and chronic pain syndromes) are common. Comorbid neurological conditions occur in approximately 20% of cases; greater than would be expected by chance. Studies of prognosis from secondary care show that, in the majority of cases, symptoms are persistent for many years and it’s hard to predict outcome. These don’t include transient symptoms in primary care or emergency settings.

There is a high associated healthcare utilisation cost with FND, the estimated total annual healthcare cost of FND, narrowly defined, in a recent study from the USA equates to $900 million. FND accounts for a significant proportion of outpatient attendance at neurology clinics.

In clinical practice, there is a fear of misdiagnosis of FND, however, studies have repeatedly demonstrated low rates of misdiagnosis. In a large cohort study of 1,030 patients with functional disorder diagnoses from a neurology clinic sample, only four patients had acquired a new neurological diagnosis that better explained their presentation after 18 months of follow-up. Twice as many patients were misdiagnosed in the opposite direction. This should not lead to complacency, however, in clinical practice, FND is often comorbid with other conditions especially neurological ones.

Assessment and diagnosis: general principles

A therapeutic assessment for someone with probable FND may include making a thorough list of presenting symptoms, focusing on the mechanism of onset (looking especially for pathophysiological triggers such as migraine, acute pain, panic disorder, episodic dissociation, infection or drug side effects). It’s helpful to ask patients about fatigue, sleep disturbance, pain and concentration symptoms which are present in the majority and may determine disability and quality of life more than the neurological symptom. People with FND have often been through negative experiences of healthcare including being disbelieved. It’s often useful to spend time hearing about those experiences, finding out what the patient, and those around them, think would be most helpful now and whether they have strong views about the diagnosis.

The diagnosis of FND should always rest on clear positive evidence of the diagnosis, typically from a combination of physical signs on examination or the nature of seizures described later. If that isn’t present, think again.

Diagnostic pitfalls

Clinicians often diagnose FND for the wrong reasons. Some of the commonest diagnostic errors we encounter follow.

Failure to consider comorbidity of another medical condition

Even if the diagnosis of FND is clear, always ask yourself whether it’s possible this is a patient with FND and another condition, possibly in a prodromal state (for example, functional tremor may occur in the early stages of Parkinson’s disease or functional/ dissociative seizures may occur simultaneously with syncope or epilepsy).

Reliance on unusual clinical features

There are many odd and unusual symptoms of other neurological conditions (such as geste antagoniste in dystonia, brief frontal lobe seizures with retained awareness and bicycling movements, ability to suppress movements in tics, and ability to self-induce epileptic seizures). The take home message is don’t diagnose FND because a presentation is unusual – there must be clear FND signs.

Diagnosis based on psychiatric features / recent stress

FND should not be diagnosed just because the patient has psychological comorbidity or recent stress. People with FND do tend to have, on average, more physical and psychological symptoms than equivalent disease controls. They are also more likely to have had adverse experiences both in childhood and more recently (odds ratio ∼2–4). However, all of these clinical features may occur in other disorders (such as multiple sclerosis or epilepsy) which are also associated with higher rates of psychiatric comorbidity. Migraine is a disorder also associated with increased adverse childhood experience in which neuroimaging is also usually normal. Conversely, the diagnosis of FND is often delayed in people without psychiatric comorbidity. FND seems to be especially rare in people with psychosis or where the presenting symptom is psychological.

Reliance on normal investigations

Many neurological conditions may have normal structural imaging.

Misinterpretation of abnormal investigations

Incidental findings are common on brain magnetic resonance imaging (MRI), and disc disease is present in most people after the age of 40 years. Even EEG and cerebrospinal fluid oligoclonal bands can be false positive in some individuals.

Functional limb weakness, tremor, dystonia and other abnormal movements

Limb weakness in FND is most commonly unilateral. It has a sudden onset in more than half of cases which often leads to presentations to stroke services. In one study of London hyperacute stroke units, it was one of the most common stroke mimics accounting for 8% of all stroke presentations.

Patients with functional limb weakness typically report a feeling that the limb doesn’t belong to them and sensory disturbance: the whole limb is commonly affected. Diagnosis, however, should be made on the basis of the pattern of weakness and, most importantly, evidence of internal inconsistency between voluntary movements (impaired) and automatic movements (preserved). Hoover’s sign and hip abductor sign are the most reliable (Fig 1).

Other clinical features support the diagnosis of functional limb weakness: a global pattern of weakness affecting flexors and extensors equally (normally, the flexors in the arms are stronger than in the legs and the extensors in the legs are stronger than the arms; it would be very unusual to have another pathological process disease which selectively affected the strongest muscle
groups), and collapsing weakness (but beware of pain or difficulty understanding instructions).

Functional tremor should be considered when there is a history of variable frequency (not amplitude) tremor which changes dramatically during externally cued rhythmic movements. For arm tremor, ask the patient to copy a rhythmical movement made between finger and thumb using their better hand. If the tremor in their other hand stops, entrains to the same rhythm or the patient has difficulty copying the movement, then consider functional tremor. For leg tremor, ask the patient to copy foot tapping, and for neck tremor, ask them to follow movements of your hand with their tongue. Tremor that increases or moves to a different body part when the arm is immobilised or pauses briefly during ballistic movements is also a clue. Most movement disorders worsen with stress and disappear with sleep, so don’t use those features to make a diagnosis.

Functional dystonia typically presents with a fixed abnormal posture, rather than other forms of dystonia which is usually mobile. Functional dystonia usually involves inverted or plantarflexed ankle, or flexion of fingers (Fig 2). This presentation of FND is often associated with severe disability, and pain is common. It overlapps clinically with complex regional pain syndrome, the motor and sensory features of which share the same characteristic as FND.14

Another common type of functional dystonia affects the face. This presentation, which is especially common in stroke settings can be recognised by the presence of jaw deviation to one side, usually with contraction of platysma. The lip curls downward (or sometimes upward) giving the appearance of facial weakness. There may also be contraction of orbicularis oculi leading to clinical queries about ptosis. The eyebrow is usually lowered on the affected side.15

Functional gait disorders, jerky movement disorders and tics can also occur as part of motor FND and are described in more detail elsewhere.

Dissociative/functional seizures

Dissociative or functional seizures are paroxysmal events often superficially resembling epileptic seizures or syncope. These are common presentations not only in neurology but also in emergency and general medicine departments.16 They are associated with poorer quality of life than those with epileptic seizures. Twenty per cent of patients have a comorbid diagnosis of dissociative seizures and epileptic seizures.9

In the clinical setting, dissociative seizures are best recognised using a combination of positive semiological features. Signs that suggest dissociative seizures include a longer duration (>90 seconds), fluctuating course, asynchronous movements, side-to-side head or body movement, closed eyes and memory of the event.17 Signs that suggest epileptic seizures include post-ictal confusion and stertorous breathing.17 Contrary to many people’s teaching, a history of urinary incontinence and physical injury are poor distinguishers. Daily seizures or a high frequency of hospital admissions with seizure activity is commonly found in dissociative seizures. In addition, dissociative events may occur during recovery from general anaesthetic. Around one-third of episodes look more like syncope and present to cardiology services. Here, frequent events where the patient suddenly falls down and lies still with their eyes closed for more than a minute should be a strong red flag for the diagnosis.

Recent studies of these events have shown that these events share many characteristics of panic attacks. There is often a brief prodrome of escalating severe symptoms, with autonomic arousal. The seizure can be seen as an involuntary, learned, brain ‘reflex’ which gets rid of these sensations which are usually experienced as unpleasant although not always fearful.18

The longer duration of events in dissociative seizures places these patients at risk of being mismanaged as status epilepticus. Treating dissociative seizures with antiepileptic medications can cause iatrogenic harm, delay correct management and may even exacerbate their events. Improvement with treatment does not differentiate dissociative from epileptic seizures as 40% of patients with dissociative seizures reported an improvement in their seizures using antiepileptic medications.19

Eye-witness reports from both lay and medical witnesses are notoriously unreliable.20 A recording from a mobile phone may be sufficient but recording a usual attack using video EEG may be necessary. Some types of epileptic seizures (for example frontal lobe seizures) may have a normal ictal EEG, and if the attack didn’t occur during the recording, the investigation may be of limited value. Importantly, it’s ok to say, ‘I’m not sure,’ and re-evaluate
seizure semiology at each visit looking for positive signs of dissociative or epileptic seizures.

Functional cognitive symptoms

Many patients with FND complain of problems with memory and concentration alongside their other symptoms. These cognitive symptoms are typically suggestive of attentional deficit, such as lapses in concentration during conversations or reading, walking into rooms and forgetting what one has gone in for, ‘gaps’ spent on autopilot when going about normal activities, blocks for overlearned information (PINs and passwords), mild subjective word-finding difficulties and an unpleasant experience of excessive cognitive effort and inefficiency are also common.

In those with functional motor disorder, seizures or dizziness, these cognitive problems can be understood as a consequence of diversion of attention towards other symptoms. However, metacognitive error is also often prominent in the form of catastrophic negative self-evaluation of one’s own cognitive performance. Teodoro and colleagues suggest a unifying model of cognitive symptoms in a range of cognitive disorders incorporating disruptions of attention and expectation in line with predictive theories of functional disorder mechanism.21

The last 5 years has seen increasing recognition of functional cognitive disorder, generally without other functional symptoms, as a common differential of degenerative brain diseases (such as Alzheimer’s disease in those presenting to memory or neurology clinics with complaints of memory difficulties).22–26 A recent review suggested that up to 24% attending memory clinics may have functional cognitive disorders.22

Recently proposed diagnostic criteria define functional cognitive disorder on the basis of positive features of internal inconsistency.27 Cognitive testing has poor specificity in this group of patients who may excel in tests despite disabling symptoms, or who may perform in mild or even severe impairment ranges despite good occupational function. In contrast, examination of behaviour and language during the consultation seems effective in discriminating functional from neurodegenerative cognitive symptoms: those with functional symptoms are more likely to attend alone, to be distressed about their symptoms and to give richer and more specific accounts of memory failures than those with neurodegenerative disease.26

There remains an absence of evidence to support any specific treatment approach for functional cognitive disorders. Clinical experience suggests that a cognitive behavioural approach may be helpful: helping patients to contextualise memory lapses that fall within normal, and tackling unhelpful avoidance and excessive, and even counterproductive, use of cognitive safety strategies.27

Treating comorbid depression and anxiety is also important as these may either drive or exacerbate cognitive symptoms.

Urinary retention and ‘scan-negative’ cauda equina syndrome

Urinary retention was thought to be one of the possible symptoms of FND by Charcot as long ago as in the 1880s. In the 1980s, urethral sphincter electromyography in women with urinary retention presumed to be related to a functional disorder were found to demonstrate abnormal decelerating bursts and complex repetitive discharges suggesting a neuromuscular disorder.28

Later work found these changes in asymptomatic women who did not develop urological retention even on 10-year follow-up.29

The status of chronic urinary retention as a type of FND remains uncertain. Women with this disorder have a high prevalence of other FND symptoms and higher than expected use of opiates due to chronic pain, which in turn may cause retention.30 Some causes of chronic intermittent urinary retention can be treated purely by cognitive behavioural therapy. Paruresis, a little researched condition which may affect up to 7% of the male population, occurs when a person is unable to urinate when others are present, such as in a public toilet.31 Patients’ social, work and romantic lives can be significantly affected.

Urinary retention also occurs as an acute phenomenon in cauda equina syndrome. Around two-thirds of patients presenting with symptoms of sphincter dysfunction with or without leg weakness and pain have normal imaging or lumbar root changes that don’t explain retention.32,33 Patients with negative scans were found to commonly have positive features of FND, such as Hoover’s sign. Factors such as pain, medications (particularly opiates) and pre-existing bladder disorders may all explain these common neurosurgical presentations.

Other FND Symptoms

There are many other FND symptoms which are described elsewhere: speech problems (such as dysarthria, mutism or foreign accent syndrome); sensory symptoms (including numbness or positive sensory symptoms especially with motor symptoms); visual loss or diplopia; hearing loss or sensitivity; and globus.34,35 Persistent postural perceptual dizziness (PPPD) describes chronic dizziness as part of a functional disorder. It has specific diagnostic criteria and clinicians should be wary of diagnosing it too easily in anyone with persistent dizziness.36 FND does not encompass pain or fatigue; although those symptoms are very common in combination with FND and almost certainly have shared mechanisms in the nervous system.

New advances in mechanism and aetiology

The renaissance in interest in FND over the last 15–20 years has included new and multiple perspectives in mechanism. What these have in common is a recognition that, at some level, the brain must be involved in FND. Clinicians and patients, especially if working with dualistic ideas of mind and brain, often see neural and psychological mechanisms in competition. The increasing consensus from this field and many others related to functional disorders is to see them as mutually complementary with explanatory power at different levels of brain, personal and societal functioning.

A brain network disorder

Functional neuroimaging studies have suggested changes in several types of brain networks. In one study, patients experiencing functional tremor had hypoactivation of the right temporoparietal junction compared with when the same patients were making a voluntary tremor.37 This area appears to be an important node in a network important in the body’s sense of ‘agency’ (ie ‘it was me that made that movement’). Other studies have found abnormally strong connections between networks related to emotion and motor networks, disorders of attentional systems and motor planning systems.38 Activations look different, and more complex than individuals feigning similar symptoms.
A disorder of predictive processing

In phantom limb syndrome, an individual senses the presence of a limb that is not there. In functional leg weakness, the patient experiences the opposite, they don’t properly sense a limb that is there. The predictive processing / active inference model of the brain is a powerful construct that suggests the brain anticipates and predicts motor and sensory experience, constantly updating those predictions with new information. In phantom limb syndrome, the prediction that the leg is ‘still there’ is not updated by the new sensory input. In functional leg weakness, there is another brain prediction, that the leg is ‘not there’, which again overrides and fails to be updated by the sensory input that indicates that it is there. 38

Linking brain abnormalities to psychological models

Previous models of FND placed a lot of emphasis on prior traumatic events as causative agents without really explaining how they might cause a weak leg or seizures. Modern cognitive behavioural theories integrate these brain-based theories looking at predisposing, precipitating and perpetuating factors. 39 40 Studies show how commonly its events experienced in the body (such as pain, injury, migraine or panic) rather than in the emotions which help determine the site and nature of symptoms, perhaps partly via an abnormality of predictive processing. 40 There are parallels here with irritable bowel syndrome after gastrointestinal infection, or prolonged ‘post-concussion’ symptoms lasting years after a minor knock to the head. Psychodynamic models and those based on attachment theory also still have relevance for many patients. 41 The key thing is not to assume that ‘one size fits all’.

Investigation

Comorbidities are common in FND. Always consider what additional neurological or general medical condition may be present and investigate appropriately. For example, we would typically arrange MRI of the brain and whole spine for a patient with functional leg weakness even when the physical signs point only to FND. Consider whether FND may be occurring in the early stages of a disorder like Parkinson’s disease and follow the patient. There is good evidence that anticipating normal results of investigations with patients helps reduce anxiety while waiting for outcomes. This approach also allows earlier discussion and treatment of an FND diagnosis.

Treatment

Many patients with FND have negative experiences of healthcare arising from a combination of poor knowledge and also a tendency for healthcare professionals to implicitly, and sometimes explicitly, disbelieve, blame and humiliate patients with these disorders. 42 43 The patient with FND has been, and still is, often subject to an approach that there is ‘nothing wrong’, accusations that their symptoms are voluntarily produced, or that the symptoms are not within the domain of the physician, and just need to be solved by a psychiatric formulation and discovery of the ‘root cause’ (when such an outcome is actually infrequent). In contrast, the approach to treatment of FND building up over the last 10–15 years reverts to a more standard multidisciplinary approach involving the following ingredients.

Communication of the diagnosis

As with other conditions, FND should be explained by what it is (ie you have FND) rather than by what it is not. Positive features of the diagnosis can be shared with patients. These in turn help to explain the underlying mechanisms of the condition, show that FND is a ‘rule in’ not a ‘rule out’ condition and, in the case of some signs such as Hoover’s sign, how there is a potential for reversibility. Table 1 contains some ideas for communicating with patients with FND and their friends and family both about the diagnosis and also its treatment. Many of these simply replicate what is done for any condition seen by physicians.

We would also suggest supplementing this information with a copy of a clinic letter, printed information and links to other resources (for example www.neurosymptoms.org, www.fndhope.org or www.fndaction.org.uk). Note however, that information alone is not a treatment in itself, it’s just the starting point for treatment. A recent randomised controlled trial (RCT) in the Netherlands showed no benefit to website provision in patients with motor FND. 44

Assessing response to communication of the diagnosis

Many healthcare professionals make the mistake of assuming that just because they think that they have shared the diagnosis well, the patient understands it and is therefore ready for treatment. FND is a hard condition for a patient to understand and a follow-up visit is essential to allow the patient time to digest the information, assess response to communication, as well as motivation, and decide what to do next. We ask the patient to tell us what they understand about the diagnosis and try to make it easy for them to admit when they don’t understand it, or when they fundamentally disagree. We recommend going through this step before referring for treatment.

Management of physical symptom comorbidities

Detection and management of relevant comorbidities can improve FND. Migraine and chronic daily headache are common and often overlooked. FND sometimes arises on a background of mild carpal tunnel syndrome, ulnar nerve irritation, sciatica or meralgia. Rheumatological issues, especially joint hypermobility / Ehlers–Danlos type 3, are a common comorbidity. When structural comorbidities are present then make more than one diagnosis and have an open discussion about how much these represent an obstacle to improvement. For example, if someone has multiple sclerosis and FND and it’s unclear how much of the disability relates to MS, they may have little to lose by exploring, via treatment, how much of their disorder is FND related. Chronic pain and/or fatigue are common comorbidities and, when dominant, it may be more sensible to start with a pain or fatigue management approach rather than focusing on FND symptoms, especially if milder.

Physiotherapy

A new approach to the physiotherapy of functional motor disorders has developed in parallel with the new transparent and ‘rule in’ approach to diagnosis. 45 Whereas, for a patient with stroke or MS, physiotherapy might ask the patient to focus hard on the movement or break it down into constituent parts,
Practical overview of FND

for a functional movement disorder, movements are worse with this excess attention. Patients with FND, therefore, can benefit from physiotherapy techniques which take attention away from the movement, sometimes using approaches that may seem paradoxical. For example, some patients are better at walking or running on a treadmill than walking normally. Music or using other ‘automatic’ learnt movements from dancing can help access better movement. These approaches have been turned into consensus recommendations. A pilot trial of 60 patients with symptoms for over 5 years showed that 1 week of this approach delivered intensively led to a 72% improvement in the intervention group at 6 months compared with only a 28% improvement in patients receiving the same amount of standard physiotherapy.46

Psychiatry / psychological therapy

Psychiatric or psychological assessment and formulation may reveal a much more complex picture than initially apparent. This is not about ‘reattributing’ neurological symptoms to psychological causes, but about setting them in an appropriate context which helps patients and therapists understand predisposing, precipitating and perpetuating factors. Treatment of psychological

Table 1. Examples of communication for functional neurological disorder based on clinical features and neuroscience of the disorder

<table>
<thead>
<tr>
<th>FND problem</th>
<th>Examples</th>
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<tr>
<td>Rule in diagnosis</td>
<td>‘You have FND for the following reasons: a, b and c.’</td>
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<tr>
<td>General</td>
<td>‘FND is a problem with the functioning of the nervous system. A problem with the software rather than the hardware.’ Or, for patients that aren’t as computer literate: ‘It’s like a piano that is out of tune, not broken but just not working properly.’</td>
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<tr>
<td>Overcoming dualism</td>
<td>Patient: ‘So, are you saying it’s in my mind or brain?’ Healthcare professional: ‘FND is a condition that shows that the mind and the brain are one and the same thing.’</td>
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<tr>
<td>Limb weakness</td>
<td>‘Did you see how your leg returned briefly to normal when I did that test (Hoover’s sign). That shows us that there is a problem with the way your brain is sending the signal to your leg (voluntary movement), but the automatic movements are still okay.’</td>
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<tr>
<td>Limb weakness/blindness</td>
<td>‘Have you heard of phantom limb syndrome? That’s when someone has an amputation, but their brain still thinks the limb is there. FND is a bit like the opposite, the leg/visual/sensation is there but the brain thinks it isn’t anymore. The map of that part of the body in the brain has gone wrong’</td>
</tr>
<tr>
<td>Weakness/movements</td>
<td>‘Functional brain scans have shown that the brain is working too hard in FND. Normally we shouldn’t have to think about how to move our arms or our legs. As soon as our brains start to work on this too hard it goes wrong. It’s similar to thinking about your feet when you are climbing upstairs, or trying too hard to fall asleep at night.’</td>
</tr>
<tr>
<td>Seizures</td>
<td>‘Functional seizures are when the brain goes into a trance-like state called “dissociation” suddenly, all by itself. This is the medical word for being cut off or distant from your surroundings. That’s a bit like the feeling you have just before your seizures sometimes. We think it does this as a “reflex” response – sometimes to get rid of a horrible feeling that many people report just before. After a while, it will often happen for no reason and when people are most relaxed.’</td>
</tr>
<tr>
<td>Dystonia</td>
<td>‘Your brain thinks that the foot is straight even though it’s turned inwards. That’s why it’s hard for you to keep it in a straight position.’</td>
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<tr>
<td>Associated pain</td>
<td>‘Chronic pain is usually due to an “increased volume knob” in the pain pathways throughout the nervous system, but especially the brain. This is called “central sensitisation” and, like FND, is also a problem with abnormal nervous system functioning.’</td>
</tr>
<tr>
<td>Prognosis</td>
<td>‘This is not an easy problem to put right, but it does have the potential to improve and many people do make a good recovery.’</td>
</tr>
<tr>
<td>Physiotherapy</td>
<td>‘Physiotherapy can help “retrain” the brain in FND. It works best when we can use those principles of distraction that I showed you. A physiotherapist may ask you to try to speed up the movement or do it in an unusual way, to music or in a mirror. Somewhere in your brain we think the automatic movements are in there, and we need to coax them out.’</td>
</tr>
<tr>
<td>Psychiatry/psychology</td>
<td>‘It’s common in FND for people to have problems like anxiety and depression. This can be a consequence of having the symptoms but, in many, it is already there for other reasons. FND symptoms make people fearful of falling and being injured and of being embarrassed. For some, there are things that have happened which may explain why your brain is vulnerable to going wrong in this way and could be worth exploring. I think a psychiatric/psychological assessment could be helpful. What do you think?’</td>
</tr>
</tbody>
</table>

= occupational therapy and speech and language therapy also may be important; FND = functional neurological disorder.
comorbidities when present (such as anxiety, panic disorder, depression and post-traumatic stress disorder) may be essential before other therapy can begin. When present, personality traits (such as emotional instability, in turn often linked to previous trauma) may need to be understood and managed as part of treatment.

Psychological therapy is the current treatment of choice for functional/dissociative seizures. A cognitive behavioural approach uses a model similar to treatment of panic disorder with grounding techniques and education early on, and later techniques to overcome ‘safety behaviours’ and avoidance triggered by the experience of seizures. A recent large RCT in 368 patients with functional seizures did not demonstrate improvement in seizure frequency at 12 months compared with highly supportive psychiatric care alone, although it was superior in a range of secondary outcomes. Psychodynamic approaches may be more suitable for some, especially individuals with trauma histories.

Occupational therapy

Occupational therapists (OTs) have a skillset which is a good fit for FND, allowing a focus on day-to-day activity rather than impairment and which crosses physical/psychological divides. Recently consensus recommendations have been developed by OTs working frequently with FND. These are especially important when considering how to use disability aids and adaptations in a way that promotes recovery but also respects the nature and time course of the patient’s illness.

Speech and language therapy

When delivered by someone with an interest in FND, speech and language therapy can yield positive, sometimes curative, results.

Medication

At present, the best evidence for medication relates to common comorbidities of FND rather than the motor and sensory symptoms of FND itself. In practice, many patients with FND come to clinic overmedicated and benefit from targeted reduction of medications, especially opiates, gabapentinoids and benzodiazepines.

Hypnosis and other

Hypnotherapy has an evidence base for motor FND. Other treatments (such as inpatient rehabilitation and therapeutic sedation) are also described.

Key practice implications

If you learnt about FND 20 years ago, think again – it has undergone a significant change since then. There remain many clinical ‘myths’ still in common day-to-day practice which are highlighted in Table 2.

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Conflicts of interest

Jon Stone runs a free self-help website (neurosymptoms.org) mentioned in this article. Jon Stone, Alan Carson and Laura

| Table 2. Myths about functional neurological disorder, old and new; adapted from Lidstone et al

<table>
<thead>
<tr>
<th>Myths</th>
<th>Newer research-based ideas</th>
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<tr>
<td>FND is a diagnosis of exclusion</td>
<td>The diagnosis of FND should be ‘ruled in’ based on the presence of positive signs.</td>
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<tr>
<td>Patients have either FND or another neurological disorder</td>
<td>FND commonly co-occurs with other neurological disorders.</td>
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<tr>
<td>A bizarre presentation indicates FND</td>
<td>A bizarre presentation does not equate to a diagnosis of FND.</td>
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<tr>
<td>Different phenotypes of FND indicate different disorders</td>
<td>Functional symptoms are often part of a broader FND syndrome including pain, fatigue and cognitive symptoms.</td>
</tr>
<tr>
<td>FND symptoms are voluntary</td>
<td>FND symptoms are involuntary; patients are not ‘putting them on’ and feigning is rare.</td>
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<tr>
<td>There is no role for investigations in the diagnosis of FND</td>
<td>Investigations can be useful to identify comorbid neurological conditions, diagnose phenotypically.</td>
</tr>
<tr>
<td>There is less harm in missing a diagnosis of FND than missing another neurological disease</td>
<td>FND is not misdiagnosed more than other conditions. Erroneously diagnosing FND as another neurological condition can be as harmful as the reverse</td>
</tr>
<tr>
<td>FND is exclusively a psychological problem caused by psychological factors</td>
<td>Psychological factors are one of many possible risk factors for FND and should not be considered the sole aetiological cause.</td>
</tr>
<tr>
<td>The prognosis of FND is usually good</td>
<td>Patients with FND are as disabled and have as impaired a quality of life as patients with other neurological conditions.</td>
</tr>
<tr>
<td>The treatment of FND is solely referral to a psychologist or psychiatrist</td>
<td>FND treatment is individualised and involves careful explanation, and combinations of physical and psychological rehabilitation.</td>
</tr>
</tbody>
</table>

FND = functional neurological disorder.
McWhirter carry out expert witness work which involves patients with FND.

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