



Information and guidance for medical professionals

[A summary of Functional Neurological Disorder]

This sheet is for information purposes only, and is designed to provide medical professionals with up-to-date and accurate information about the condition Functional Neurological Disorder (FND).

What you need to know

Functional Neurological Disorder (FND) is a condition that arises from a problem with how the brain and nervous system sends and receives signals, rather than an identifiable disease process.

Symptoms may appear similar to neurological conditions such as Multiple Sclerosis, Parkinson's disease and epilepsy, and result in similar levels of disability and distress. However, the underlying cause is different.

Diagnostic Symptoms may include:

- Movement and other motor symptoms such as tremors, limb weakness, episodes of paralysis, altered gait, muscle spasms or fixed joints.
- Sensory symptoms such as altered sensation or visual disturbances.
- Seizures which resemble those associated with epilepsy or syncope.

Hypersensitivity
Drop attacks Tremors
Dystonia Fleeting sensations
Stroke-like symptoms Dissociation
Walking difficulties Spasms
Loss of bladder / bowel function
Cog fog Chronic pain
Limb weakness Speech impairment
Dizziness Anxiety Fatigue
Seizures Depression
Paralysis Stress
Myoclonus

Other symptoms may co-exist that are associated with the nervous system, and associated with poor health in general, such as pain, fatigue, memory and other cognitive symptoms, or mental health difficulties.

Mechanisms and aetiology of FND

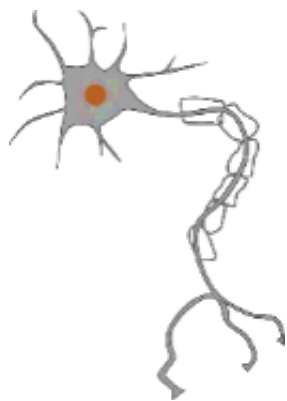
Historically FND has been viewed as resulting purely from psychological and emotional trauma, however recent neuroscientific research, including the use of fMRI brain imaging, has revealed these models to be outdated and simplistic. This has led researchers in the field to now identify FND as a '**brain network disorder**' rather than relying on previous purely stress-based models such as 'conversion disorder', 'psychogenic', 'somatisation' etc.

For example 'functional' imaging (fMRI) brain scans, as opposed to 'structural' scans routinely done as clinical investigations, have revealed disruptions to many key brain areas, including the amygdala, insular cortex, and temporo parietal junction. ^{(1) (2) (3)}

Structural MRI scans have also revealed alterations at the group level between FND patients and control groups in both grey and white matter volumes. ^{(4) (5)}

Given FND affects brain areas that are responsible for multiple functions such as motor movement, pain processing, emotional processing and self agency, it is clear dualistic models that separate illness into categories of either physical or psychological are no longer adequate. FND presents a paradigm shift in our understanding of how the brain works because it lies between the fault lines of mind and body, neurology and neuro psychiatry, and for this reason FND is best viewed within the framework of the biopsychosocial model.

FND – An error of predictive processing



Modern neuroscience views the brain as a ‘predictive’ organ, and recent evidence ⁽⁶⁾ supports the view that FND is a result of problems with the pre-conscious phases of motor planning, disrupted by the brain’s abnormal involuntary predictions about movement ⁽⁷⁾. These take place at very low levels of the nervous system beneath conscious awareness. The brain relies on predictions of what it will be able to see and do based on previous internal models, prioritising them over incoming sensory input.

For example, in the movement disorder subtype of FND this results in a mismatch between instructions from the brain to move a limb and the sensory feedback from doing so. In this view FND is now often conceived as an ‘error of predictive processing’ caused by a disruption of the functioning of brain networks.

How psychological is FND?

FND can be triggered by physical trauma such as an accident or head injury, and can also be triggered by psychological trauma. Having a pre-existing neurological condition is also a strong risk factor for developing FND. In addition to this there are a proportion of patients who have no physical or psychological trauma suggesting genetic risk factors.

Historically medical professionals have often confused risk factors with root cause; psychological stress and trauma are certainly risk factors for triggering FND but not ‘necessary or sufficient’ to cause the disorder. Like most illnesses the reasons why patients may develop FND are complex and heterogeneous.

“Functional Neurological Disorder is often explained to patients as a psychological reaction due to past trauma, or as symptoms due to stress. These explanations usually fail and result in patients feeling alienated, stigmatised and not believed. The main reason for the failure of such explanations is that they take a potential risk factor and turn it into the cause of the problem.” – Professor Mark Edwards, Professor of Neurology, Kings College, London.

At present the DSM-5 definition of FND requires the presence of positive diagnostic features, which means FND is not a diagnosis of exclusion but a ‘rule in’ diagnosis. Previously one of the diagnostic requirements for FND was a recent psychological stressor, however this was recently removed in recognition that many FND patients do not have any identifiable psychological stressor.

“Psychological trauma is to FND what smoking is to stroke, a risk factor but not the cause.” – Professor Jon Stone, Consultant Neurologist and Honorary Senior Lecturer at the dept. of Clinical Neurosciences, University of Edinburgh

How is a positive diagnosis of FND made?

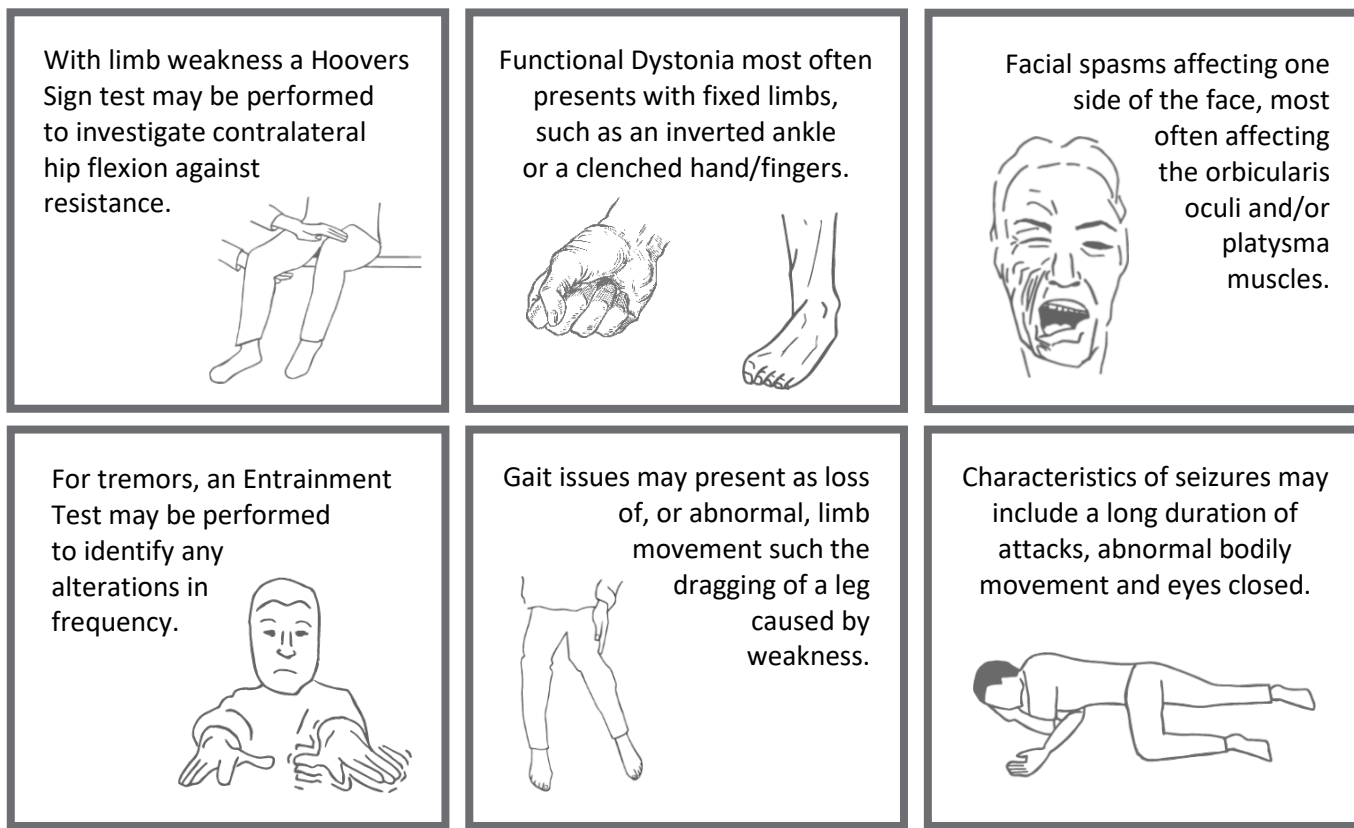
While it is reasonable to raise the possibility of an FND diagnosis with the patient, patients with a suspected diagnosis should be referred to a Neurologist for a specialist assessment. A diagnosis of FND should be based on positive clinical features.

Patients with FND often have many complex symptoms, so it is important to ask about motor and sensory symptoms, fatigue, pain, sleep disturbance, memory and dissociative symptoms. FND often co-occurs with other conditions, most commonly neurological illnesses, and under these circumstances may be referred to as ‘functional overlay’.

Despite the lack of NICE Guidelines for FND at this present time, the clinical pathway for diagnosis should start with a Neurologist making a rule-in diagnosis.

Clinical features/diagnostic signs

A neurologist will diagnosis FND on positive clinical features. Below are some examples of presentations and tests that may be undertaken.



How should a diagnosis be explained to the patient?

Given the complexity of FND it is often not an easy diagnosis for patients to receive. It is important to explain that FND is a problem with 'the way the brain and nervous system send and receive signals' and that it is a 'brain network disorder', meaning 'different parts of the brain are not communicating as they should be'.

Make it clear there can be many contributing factors to developing FND. While psychological factors are important for some patients, for others they will not be relevant.

Reassure the patient that there are evidence-based treatments for FND, and because it is predominantly a 'signalling problem' and not a disease process, positive outcomes can be achieved.

How is FND treated?

Treatment plans must be tailored to suit the person's individual need, it is important that collaborative care is accessible. Specialist neuro-physiotherapy, Occupational Therapy, Speech and Language Therapy, and psychological therapies (for example CBT), are all treatment modalities that can be used to 'retrain the brain'. In some cases, particularly where the condition is severe, these can be given together in a multidisciplinary day hospital or inpatient programme. Common comorbidities such as pain, fatigue, PTSD, depression, and anxiety need to be assessed for and managed as they would be in other conditions.

It is recognised that the sooner treatment is started the better chance a person has of recovery/symptom management. Treatment outcomes are variable, not all people can improve, but evidence from randomised trials indicate that appropriate treatment can be highly effective for some.

What we would like medical staff to do

Take the time to learn about the condition. People who feel that they are being listened to, by someone who has a current and up to date understanding of FND, will feel much less anxious and more willing to engage.

Understand that the person is in no way consciously controlling their symptoms. Offer compassionate support to avoid people not seeking medical care because of their fear of not being believed.

If a person presents with a new symptom, strike a balance between automatically assuming it is related to FND and recognising an individual's vulnerabilities.

Do not rush to blame symptoms on stress, anxiety, or depression. These problems may not be present at all, may occur as a consequence of a bewildering and currently stigmatised diagnosis, or a trigger of symptoms. Psychological factors and treatment remain important for some people, but an insistent focus on this is akin to a narrow focus on smoking in patients with stroke, in some it is relevant in others it is not.

Despite the fact a patient may have an FND diagnosis, any new symptoms should be fully investigated to avoid delay diagnosing other comorbidities. Always be willing to consider whether patients with symptoms of FND may also have a pathophysiological disease, and be willing to make other diagnosis' if appropriate. It can have a detrimental effect on a person's health if other conditions are present and treatable.

Provide and coordinate continuous care and support whilst a person remains symptomatic, inclusive of primary health, MDT's and social care.

Useful resources:

- Stone J, Burton C, Carson A. **Recognising and explaining functional neurological disorder**. *BMJ*. 2020 Oct 21;371:m3745. doi: 10.1136/bmj.m3745. PMID: 33087335.
- Bennett K, Diamond C, Hoeritzauer I, Gardiner P, McWhirter L, Carson A, Stone J. **A practical review of functional neurological disorder (FND) for the general physician**. *Clin Med (Lond)*. 2021 Jan;21(1):28-36. doi: 10.7861/clinmed.2020-0987. PMID: 33479065; PMCID: PMC7850207.
- Gilmour, G.S., Nielsen, G., Teodoro, T. *et al.* **Management of functional neurological disorder**. *J Neurol* **267**, 2164–2172 (2020). <https://doi.org/10.1007/s00415-020-09772-w>
- www.neurosymbols.org
- www.fndaction.org.uk

Citations:

- (1) Diez I, Ortiz-Terán L, Williams B, et al. **Corticolimbic fast-tracking: enhanced multimodal integration in functional neurological disorder**. *Journal of Neurology, Neurosurgery & Psychiatry* 2019;90:929-938.
- (2) Voon V, Cavanna AE, et al. **Functional Neuroanatomy and Neurophysiology of Functional Neurological Disorders (Conversion Disorder)**. *J Neuropsychiatry Clin Neurosci*. 2016 Summer;28(3):168-90. doi: 10.1176/appi.neuropsych.14090217. Epub 2016 Feb 22. PMID: 26900733.
- (3) Maurer CW, LaFaver K, Ameli R, Epstein SA, Hallett M, Horovitz SG. **Impaired self-agency in functional movement disorders: A resting-state fMRI study**. *Neurology*. 2016 Aug 9;87(6):564-70. doi: 10.1212/WNL.0000000000002940. Epub 2016 Jul 6. PMID: 27385746; PMCID: PMC4977370.
- (4) Diez, I., Williams, B., Kubicki, M., Makris, N., & Perez, D. (2021). **Reduced limbic microstructural integrity in functional neurological disorder**. *Psychological Medicine*, 51(3), 485-493. doi:10.1017/S0033291719003386
- (5) Maurer CW, LaFaver K, Limachia GS, Capitan G, Ameli R, Sinclair S, Epstein SA, Hallett M, Horovitz SG. **Gray matter differences in patients with functional movement disorders**. *Neurology*. 2018 Nov 13;91(20):e1870-e1879. doi: 10.1212/WNL.00000000000006514. Epub 2018 Oct 10. PMID: 30305450; PMCID: PMC6260194.
- (6) David L. Perez, Timothy R. Nicholson, et al. **Neuroimaging in Functional Neurological Disorder: State of the Field and Research Agenda**, *NeuroImage: Clinical*, Volume 30, 2021, 102623, ISSN 2213-1582, <https://doi.org/10.1016/j.nicl.2021.102623>.
- (7) Edwards MJ, Adams RA, Brown H, Pareés I, Friston KJ. **A Bayesian account of 'hysteria'**. *Brain*. 2012 Nov;135(Pt 11):3495-512. doi: 10.1093/brain/aws129. Epub 2012 May 28. PMID: 22641838; PMCID: PMC3501967.