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Abstract

Functional Neurological Disorder (FND) is one of the commonest reasons that people seek help from a neurologist and is for many people a life-long cause of disability and impaired quality of life. Although the evidence base regarding FND pathophysiology, treatment and service development of has grown substantially in recent years, a persistent ambivalence remains amongst health professionals and others as to the veracity of symptoms in those with FND, and whether they are not, in the end, just the same as feigned or malingered symptoms. Here, we provide our perspective on the range of evidence available which in our view provides a clear separation between FND and feigning and malingering. We hope this will provide a further important step forward in the clinical and academic approach to people with FND, leading to improved attitudes, knowledge, treatments, care pathways and outcomes.

[H1] Introduction

Functional neurological disorder (FND) describes motor and/or sensory symptoms that arise from the voluntary motor or somatosensory nervous system and are experienced as

involuntary. FND is a disabling condition and is the second commonest reason for an outpatient neurology consultation after headache disorders[1]. In contrast to the optimism often expressed by clinicians when delivering the diagnosis, follow-up studies show that individuals with FND have high levels of persistent symptoms that substantially impair their quality of life and participation in society[2]. Healthcare costs associated with FND are high, as are the associated costs of social care, indirect costs from lost earnings, and provision of unpaid care by family members[3].

Our understanding of clinical, pathophysiological and treatment aspects of FND has progressed substantially over the past two decades. However, two common concerns make clinicians wary of the diagnosis. First, in our experience, concerns persist about the possibility of misdiagnosis. Although misdiagnosis of FND can happen (as for all medical diagnoses), the evidence suggests that the diagnosis of FND is stable over time and that change in diagnosis is unusual and no more common than for other neurological or psychiatric disorders[4,5]. The second common concern of clinicians, which we consider to be logically incompatible with the first, is whether the symptoms of FND are genuinely experienced or are to some extent voluntary. For example, in studies of both neurologists and psychiatrists, a considerable proportion of clinicians believed that feigning is inevitably intertwined with, or is the same thing as, FND[6–8]. For example, in a questionnaire study of over 350 neurologists, 58% thought that there was an overlap between FND and feigning with 13% indicating that FND and feigning were simply subsets of the same phenomenon[7]. The techniques used to provide a positive diagnosis of FND such as Hoover's sign **[G]** or the tremor entrainment test **[G]** demonstrate that the capacity for normal function is present, indicating that that symptoms arise as a problem with accessing or controlling the body normally**.** However, this observation

also raises the question of whether or not the symptoms displayed by someone with suspected FND are voluntary.

Here, we present clinical, epidemiological and experimental evidence that directly addresses this difficult but fundamental issue. We seek to understand how likely it is that wilful and conscious fabrication or exaggeration of symptoms can explain the available data.

[H1] Clinical and epidemiological data

[H2] Historical consistency

FND is not a new diagnosis, and reports of individuals with problems consistent with FND appear in some of the earliest medical documents. For example, Thomas Sydenham, one of the most prominent physicians in 17th Century England wrote extensively on the topic of hysteria (an historical term for FND), noting it as one of the commonest illnesses and that it could occur in both men and women[9]. Following the great interest in hysteria in 19th century France by Pierre Janet, Paul Briquet, Jean-Martin Charcot and latterly Sigmund Freud we have a wealth of careful clinical descriptions of individuals with FND, which are remarkably consistent with presentations of FND in the 21st Century[10]. Examples of this consistency include the dragging gait of functional weakness, the fixed postures of the hands and feet seen in functional dystonia, the appearance of functional facial dystonia[11], and the semiology of convulsive and akinetic attacks in functional seizures (Table 1)[12]. Physicians in the 19th century noted how often FND was triggered by physical injury, transient dissociative experiences and in some cases emotional stress[72]. A similar range of triggering events is observed in contemporary samples of individuals with FND[12].

[H2] Cross-cultural consistency

Contemporary reports provide evidence of cross-cultural consistency in FND phenomenology. For example, in cohorts of people with functional movement disorders recruited from Spain and North America, the proportions of different movement disorders are remarkably similar, with action tremor being most common (48% of both cohorts), dystonia second (28% in US cohort, 27% in Spanish cohort) and myoclonus third (12% in US cohort, 17% in Spanish cohort)[13]. Studies of seizure semiology also show similar patterns in other geographical cohorts[14]. More generally, studies from diverse global locations such as Tanzania, Pakistan, Turkey, Iran and China all describe similar phenomenology of FND presentations to those reported in the European and North American literature. Attributions of symptoms vary considerably by culture, but the basic semiology seems to be similar[15–19]. These data suggest that as well as consistency over time, FND phenomenology is consistent across cultures as would be expected of a typical illness.

[H2] Subjective experiences

An alternative view of similar objective semiology between different cohorts around the world and across time would be that feigned symptoms might also have historical and geographical consistency. However, evidence also indicates that the subjective experiences reported by individuals with FND are highly consistent. For example, the symptoms reported prior to, during, and after functional seizures tend to be consistent across individuals. In a study of 354 participants with functional seizures or epilepsy, the presence of six or more panic attack symptoms identified functional seizures with a sensitivity of 60% and a specificity of 79%[20]. Self-report questionnaires based on this work correctly classified 91% of individuals with epilepsy versus those with syncope, 94% of those with functional seizures versus those

with syncope and 77% of those with epilepsy versus functional seizures[21]. In contrast, another study found that healthy volunteers asked to feign a seizure share some of the clinical characteristics of functional seizures but do not report these subjective experiences[22].

In the 19th century, people with functional motor disorder were also noted to have a range of subjective experiences[10]. For example, in 1907, Pierre Janet found that individuals with functional limb weakness often had impaired mental imagery and disrupted ownership of the affected limb[88], something that is often found in contemporary cohorts[23].

[H2] Comorbidities

Individuals with FND also have a consistent pattern of comorbidities. Pain, fatigue and cognitive symptoms occur in most people with functional motor symptoms, and evidence indicates that they have a larger effect on disability and quality of life than the primary FND symptoms[24]. FND is also often present alongside other comorbid somatic symptoms and diagnoses including fibromyalgia, irritable bowel syndrome and other primary chronic pain syndromes[25,26]. Psychiatric comorbidities, including depression, post-traumatic stress disorder and obsessive-compulsive personality traits, are also consistently more common in FND than in other neurological conditions[27]. In summary, FND seems to be similar to many other disorders in terms of level of inter-individual consistency in phenomenology and comorbidities as well as consistency across cultures and across history.

[H2] Interaction with investigations

In our experience, people with FND consistently demonstrate behaviour that would be surprising for a population group who were feigning symptoms. They seek repeated investigations for their symptoms, the results of which are consistently normal unless a

comorbid neurological condition is present. For example, a systematic review of 32 studies that used suggestive seizure induction while recording an EEG found that between 30 and 100% of individuals with functional seizures had an event in response to verbal suggestion[28]. These events occurred even though participants were aware that functional seizures are associated with a normal EEG, and even when the suggestion protocol was transparent and non-deceptive. Similarly, in a retrospective study of individuals eventually diagnosed with FND admitted to an in-patient unit for prolonged concurrent video and EEG recording to diagnose their seizures, the majority experienced a functional seizure within the first 48 hours[29]. Evidence also indicates that with the advent of video equipped smart phones, video footage sent by patients to treating clinicians can be used to aid the diagnosis of functional seizures in the majority of cases[30,31]. Contrary to these observations, we would expect individuals with proven factitious disorder **[G]** and malingering **[G]** to typically avoid such investigations, and not submit videos of their seizures for review, as such investigations can disprove the presence of a neurological disease.

[H2] Prognosis and treatment response

Long-term follow-up studies of individuals with FND typically report persistence of symptoms for many years[73]. In one prospective study of functional limb weakness, 80% of participants still reported the symptom at follow–up, which was an average of 14 years after baseline assessment[32]. In our opinion, malingering or simulation of limb weakness might have some benefit in the short term but we consider it an implausible explanation for 14 years of symptoms, especially when other symptoms such as pain, depression or fatigue are arguably much easier to maintain as a feigned symptom.

We would not expect simulated symptoms to improve with treatment, and in particular we would not expect them to improve more with treatment designed specifically for functional neurological symptoms than with a superficially similar treatment or in a randomized clinical trial that uses an active placebo. One randomized controlled trial[33] compared 29 participants with functional movement disorders who received a 5-day specialist physiotherapist intervention based on a specific mechanistic model of FND with 28 participants with functional movement disorder who received treatment as normal with a community neurophysiotherapy team (the control group). At 6 months after pre-treatment baseline assessment, 72% of the intervention group rated their symptoms as improved, compared with 18% in the control group. A moderate to large treatment effect was reported across a range of outcomes, and clinicians blinded to the treatment group rated those participants who had undergone the intervention as having a substantial improvement after 6 months.

In the CODES trial, 368 people with functional seizures were randomly assigned to receive standardised neuropsychiatric treatment with or without an additional cognitive behavioural therapy protocol designed specifically to help with functional seizures[35]. Both groups had an average of 3 years of seizures and had a rough halving in the number of seizures (the primary outcome) over the 12-month period following treatment. However, a number of secondary outcomes, including work and social adjustment score, longest interval seizure free and seizure bothersomeness, were improved in the cognitive behavioural therapy group compared with the control group. At the level of the individual patient, any treatment option could provide a ‘face-saving’ opportunity for those feigning their symptoms to change their symptom performance. However, in our opinion, treatment effects such as those described above, which were observed in a large population of individuals who have had symptoms for years, are much more difficult to explain as ‘face-saving’. More importantly, this theory would

not explain why individuals with FND respond better to the interventions of interest than to active control treatments, particularly in trials such as the physiotherapy trial described above, where the number of treatments were similar[33], or in semi-blinded or fully-blinded settings, for example, in placebo-controlled trials of serotonergic medication[36]. We argue, therefore, that consistency of response to the treatment interventions described above provides evidence against simulation as an explanation for symptoms in people with FND.

[H1] Experimental data

In addition to the epidemiological and clinical evidence described above, experimental studies provide powerful evidence for why individuals with FND are not feigning their symptoms. These studies have identified biomarkers of FND that are based on functional imaging, psychophysical or physiological approaches.

[H2] Task activation studies

Several functional imaging and neurophysiological studies have used an experimental design where people with FND are compared with healthy people who have been asked to feign similar symptoms[37]. The majority of these studies have been small, but they have reported differences between the two groups. In one example of such a study, researchers asked a single individual with unilateral functional paralysis to perform a go/no-go task **[G]** while undergoing fMRI[38]. In this individual, preparatory activation was seen in right motor areas (relating to the affected left hand) but was accompanied by activation in ventromedial prefrontal areas, which could reflect attentional diversion to the self as well as emotional processing. In contrast, healthy controls asked to feign showed similar patterns of brain activation during go trials for their feigned weak hand as for no-go trials in their normal hand,

suggesting a more direct cognitive inhibition of movement underlying the feigned weakness.

Perhaps more persuasively, eight participants who had intermittent functional tremor were scanned using fMRI in the situation where they had their involuntarily experienced functional tremor, and also at a time when they did not have functional tremor and were asked instead to mimic their tremor[39]. A comparison of these two conditions revealed a hypoactivation of the right temporoparietal junction during the functional tremor compared with the feigned tremor. This area is an important node in the network that mediates a sense of agency. This task therefore provides a direct comparison of an FND symptom with its feigned counterpart. Using a within-subject design helps to control for many additional factors that might confound studies with healthy controls

In a neurophysiological study, an EEG pre-movement potential known as contingent negative variation was recorded during a pre-cued reaction time task in 6 participants with unilateral functional weakness, 24 healthy participants who were asked to feign weakness and 12 healthy participants not asked to feign weakness[40]. In the group with FND and the feigning controls, movements of the affected had were slower and had less power than movements of the unaffected hand; the degree of difference between the two hands was similar in both groups. Individuals with FND had significantly suppressed amplitude of contingent negative variation when their weak hand was cued compared with when their normal hand was cued, but in the feigning controls the amplitude was similar to controls not asked to feign**.** This finding suggests that a specific inhibitory process relating to normal movement preparation is present in the group with functional limb weakness but not in people asked to feign, despite the similarity of the movement deficits observed in the two groups. Corroborating these findings, abnormal contingent negative variation was also reported in another study of people

with functional motor symptoms, and reduction in this abnormality after treatment correlated with the degree of clinical improvement[41].

Taken together, these findings indicate differences in experimental correlates of neural function between people with FND and healthy controls being asked to feign. However, the use of volunteers feigning symptoms as a comparator group has shortcomings. First, the feigning of symptoms over a prolonged period, as could be suspected in individuals with possible FND, might be associated with different patterns of neural activation than the feigning of symptoms over a few hours in a research study. Second, participants who have been instructed to feign might use strategies to imagine and express their symptoms that are different to those used by people who successfully deceive observers. This difference in strategy could give rise to different patterns of brain activation in 'healthy' feigners versus 'pathological' feigners. Third, there is no 'cost' to healthy study participants failing to feign symptoms in a believable manner, whereas a patient who is deliberately feigning symptoms risks being discovered; this difference could influence patterns of behaviour and therefore neural correlates. An ideal, though likely infeasible study would be to compare participants with FND with participants who have admitted feigning neurological symptoms on the tasks described above. In this regard, we consider the within-subject feigning study, described above, to be particularly powerful, especially as the clinical and kinematic properties of the tremor produced during feigning and functional tremor were identical.

[H2] Other activation studies

Because of the shortcomings of the task activations studies described above, other paradigms have been incorporated into neuroimaging studies of individuals with FND. These include studies in which participants either watch or imagine movements, thus generating an internal

motor representation of the observed action. This methodology can be used to study the generation and preparation of action, because the brain regions activated by imagined and executed movements overlap. The approach can also indirectly assess neural functioning as it applies to planning, preparation and execution of motor control.

In one study, decreased brain activity during observation of hand movements was observed in four participants with functional limb weakness compared with seven healthy controls, but crucially this decrease was only observed in motor areas contralateral to the functionally paralysed limb[42]. As the authors of the study noted, this kind of passive stimulation is independent of the participant's motivation and cooperation, and therefore difficult to feign, and is consistent with the existence of a specific pathophysiological problem with the higher sensorimotor control of the weak limb. Another study used a motor imagery task in which participants had to judge as fast as possible the laterality of a visually presented rotated hand stimulus[43]. To perform this task participants must therefore implicitly rotate the hand presented to them. In participants with unilateral functional paralysis, greater ventromedial prefrontal and superior temporal cortical activation was observed in the affected side compared with the unaffected side, which suggests heightened self-monitoring of the affected side. The time taken for mental rotation did not differ significantly between the affected and unaffected hand, emphasising that the differences in brain activation were not a result of differences in effort or task performance**.**

In another study, seven participants with unilateral functional motor and sensory loss received passive, vibratory stimulation of both hands while undergoing single photon emission computed tomography (SPECT) to assess blood flow to the brain[44]. Passive vibration is known to activate both sensory and motor areas through proprioceptive pathways

including primary and secondary cortex, premotor areas, and subcortical areas. The use of a vibration paradigm avoids the confounding effects of unreliable or variable task performance. In this study, SPECT signal was reduced in the basal ganglia and thalamus contralateral to the motor and functional loss, compared with those same regions on the ipsilateral side. This difference between hemispheres disappeared after successful treatment of the motor and sensory symptoms, supporting the robustness of the study findings. Activity in the contralateral basal ganglia and thalamus also correlated with changes in the connectivity between these regions and inferior and ventromedial prefrontal areas of the same hemisphere. These findings were therefore consistent with inhibition of sensorimotor pathways while patients were symptomatic, which improved in conjunction with symptom improvement.

[H2] Psychophysics and psychophysiology

Some studies have used psychophysical **[G]** and psychophysiological **[G]** assessments to investigate FND. In these assessments, the pattern of deficit observed in participants with FND, arguably, cannot be explained by systematic manipulation or feigning (Fig. 1).

[H3] Action–effect binding

The Libet clock paradigm assesses action–effect binding. In this paradigm, participants use a rotating clock to judge the time of their own voluntary key presses (action) and a subsequent auditory tone (effect). In healthy participants, an effect following a voluntary action is typically reported as occurring earlier, and the preceding action later, than in trials of only key presses or tones. This subjective contraction of time between an action and its effect only occurs if the participant feels that they are the agent responsible for the action. In a study of

participants with a functional movement disorder a reduction in this binding effect compared with healthy participants was observed, which implies that abnormalities in the conscious experience of action underlie functional movement disorder[45,46]. Given the implicit nature of the measure of agency used in this study, feigning would be an unlikely explanation for the results.

The Libet task was combined with fMRI in a study that replicated the above finding that the perception of intention to move and the movement itself were indistinguishable in participants with FND, compared to healthy participants who perceived intention to move earlier than the actual movement[47]. In addition, this study showed that attention towards intention to move was associated with lower right inferior parietal cortical activity in participants with FND than in healthy participants. Crucially, the reporting of the time of intention was more precise or associated with a smaller standard deviation in participants with FND than in healthy participants, which refutes the hypothesis that participants with FND might be feigning, or that the findings might be related to non-specific attentional deficits.

[H3] Sensory attenuation

Sensory attenuation is a phenomenon whereby the intensity of sensation caused by self-generated movements is reduced compared with that caused by movements that are not self-generated[48]. This phenomenon can be understood as a decrease in the attentional gain of the sensory consequences of one's own actions compared with sensory phenomena that occur as a result of external or involuntary stimuli. Under the feed-forward model **[G]** of movement generation, this reduction in gain is necessary to allow movement to occur. The experience of sensory attenuation is thought to be important in labelling movements as self-generated,

and a loss of sensory attenuation has been associated with a loss of agency for movement in people with schizophrenia[49]. Experimentally, sensory attenuation has been most commonly explored using the force-matching paradigm[48]. In this paradigm, participants are asked to match a force delivered to their finger, either by pressing directly on their own finger with the other hand, or by operating a joystick that, via a non-linear transform, causes a robot arm to press down on their finger. Healthy participants consistently generate more force than required when directly pressing on their finger compared with using the joystick. The excess force exerted in the first condition is thought to reflect sensory attenuation of the sensory consequences of self-generated movements, something not present in the second condition, where the non-linear transform between movement and sensation disrupts the sense of agency[48].

A study of 14 participants with functional movement disorders used the force-matching paradigm and found that these participants had a loss of sensory attenuation in the self-condition and were consistently more accurate at force matching than healthy participants[50]. This result seems to be an unusual if not impossible one to feign. In participants with functional movement disorders, the variability or standard deviation of force matching was much smaller than in healthy participants and accuracy of force matching showed a linear dose–response relationship, which again would be very hard to replicate with feigning. It seems highly unlikely that patients would be aware of which condition to manipulate, and it would in any case be very difficult to deliberately override a physiological bias towards less accurate force-matching in the self-condition. One remaining criticism of force-matching paradigms is that attentional differences between the groups might confound the results. However, another study identified a reduction in sensory attenuation in participants with FND

as measured by sensory evoked potentials (SEPs) at the onset of self-paced movement[51], a finding which cannot be explained by volitional attentional differences.

[H3] Temporal discrimination of stimuli

One study explored temporal discrimination in a group of 36 participants with functional movement disorders and 36 healthy participants[52]. Participants had to judge whether they could feel one or two electrical stimuli, which were administered to one finger. The interval between the stimuli was varied, allowing the calculation of a temporal discrimination threshold **[G]**, with further metrics such as reaction time allowing the assessment of other aspects of the decision-making process. The responses of individuals with functional movement disorders were less accurate and less sensitive to changes in stimulus than the responses of healthy participants. Reaction times were also significantly slower in the group with functional movement disorders and this metric did not improve in response to increasing the interval between stimuli**.** However, importantly, when looking at participants with equivalent levels of response accuracy, reaction time differed only slightly between participants with functional movement disorders and healthy participants. This observation suggests that response uncertainty was the main driver of the prolongation of reaction time, as opposed to effort. Analysis of these data using a drift diffusion model **[G]** revealed that the mechanism behind these shifts in performance in functional disorders was a significant reduction in drift rate, that is, an impairment in the quality of the information that drives decision making processes**.**

In a similar vein, one study of participants with functional gait disorder used a stationary or moving sled, kinematic gait measures (trunk displacement, step timing, gait velocity), and EMG recording to investigate gait learning [53]. Gait and EMG data indicated that, although

participants with FND and healthy participants have similarly normal adaptive gait learning, only the participants with FND had enhanced locomotor aftereffects [G], which indicate a tendency to perpetuate learned motor programmes. Both of the temporal discrimination studies discussed here identified specific patterns of deficit, which seem difficult to feign.

One alternative explanation for the findings of psychophysical and psychophysiological assessment in participants with FND is that chronic feigning of symptoms gives rise to a particular pattern of performance in these tests, for example, force matching or temporal discrimination, that is different from healthy people. However, we are struck by the normal performance in the tasks we have described above — for example, in the accuracy of force matching in the "external" condition in the force-matching task, or reaction time at similar levels of response accuracy in the temporal discrimination task — which in our opinion is not consistent with a person feigning symptoms during these experimental paradigms.

[H1] Models of brain function

Having discussed the experimental evidence, we now discuss a theoretical, but important, argument that FND is different from feigning. Active inference models of brain function account for conscious perception and action via a process of integration between incoming sensory data and "top down" predictions about those data (Box 1). Given our current knowledge and understanding of brain function, particularly with regard to these models, we consider that it is entirely expected that FND should exist; we outline our logic below.

First, there is a system that, when functioning correctly, mediates the experience of our bodies and the world, our conscious sense of presence and agency and allows us to exert control over our environment through action. This system must be capable of malfunction. If

malfunction of this system occurs, then it could disrupt perceptual experience, sense of presence and agency and motor function; however, the symptoms would not be expected to follow the patterns observed in recognised structural disease or dysfunction that occurs outside this system. The symptoms would also not be associated with objective pathology within other (neural) systems. Instead, the symptoms would follow patterns appropriate to the system that has malfunctioned, for example, to present primarily during attentional activity and to disappear with distraction, to conform to high-level beliefs or internal symptom models about nervous system dysfunction, and to be changed dramatically by expectation or suggestion. Furthermore, the personal experience of having dysfunction within this system would be one of unwilled loss of control and access over the body. Given the integration of processing of bodily visceral signals into this system (interoception), it would be expected that there would be an interaction between functional neurological symptoms and illnesses associated with abnormal processing of such signals and emotional dysregulation, for example, neurodevelopmental disorders, environmental stressors (including trauma) and psychiatric diagnoses such as anxiety disorder, personality disorder and post-traumatic stress disorder.

[H1] Detection of feigning

If feigning is sometimes confused with FND then we should consider how clinicians can detect it. Here, we address concepts around performance validity testing, how this relates to clinical features of FND and which features should be given weighting when considering feigning.

[H2] Performance validity testing

Performance validity tests (PVTs) were developed to aid the assessment of cognitive difficulties in individuals who are litigating to obtain compensation after traumatic brain injury. The original aim of these tests was to distinguish 'genuine' cognitive impairment from so-called 'exaggerated' symptoms in individuals who were malingering or had factitious disorders. These tests have now been applied in other contexts, including in the assessment of functional neurological disorders in a non-litigatory context. Failure in a performance validity test (PVT) is generally defined as scoring below a specific threshold, but this definition can be operationalised in different ways[56]. First, it might mean that an individual has responded below chance level. This result suggests a deliberate selection of incorrect answers, thus providing the strongest evidence for symptom exaggeration. However, this result still does not resolve whether deliberate choice occurred with conscious reflection or not. Alternatively, failure could be construed as 'inadequate effort' on a PVT, which has been defined as abnormally poor performances on easy test items combined with chance-level performance on difficult items, and by performances on easy items that are worse than one would expect to see in mild-to-moderate structural neurological pathology [56]. The use of the term "effort" in this context has been criticised as substantial cognitive effort is arguably required to deliberately select incorrect answers or otherwise to deliberately feign poor performance[57].

PVTs are now recognised as an essential part of neuropsychometric assessment[58]. Poor performance, when present, tends to invalidate much of the rest of the assessment. A meta-analysis of PVTs performed in a range of neurological conditions, including FND, reported failure on single tests across conditions, especially in people with dementia and moderate-to-severe TBI[59]. Although some people with FND do fail PVTs the evidence does not suggest that they fail more than people with other disorders. These data argue that the phenomenon of

poor performance on neuropsychological testing is generic in a wide variety of neurological and psychiatrically defined conditions and not specific to FND.

We recognise that symptom validity testing (SVT) is also commonly recommended for use alongside PVT in the detection of malingering[60,61]. However, in our opinion, caution needs to be taken when interpreting some SVT results. Some symptom validity tests (SVTs) have a similar premise to PVTs; however, other SVTs are based on the premise that if an individual claims to have symptoms after a particular proposed mechanism of brain injury or damage (for example traumatic brain injury) that are not generally complained of by people who are known to have definitely suffered that type of brain injury or damage, this is evidence of non-credible performance and by implication, feigning. We disagree that this is inevitably the case. In our view, if an individual fails an SVT — for example, after a minor head injury — the complained of symptoms are unlikely to be a result of structural brain injury but it does not follow that they are "non-credible". Indeed, the symptoms might be entirely compatible with the diagnosis of FND, which can often occur in the context of triggering "health events" such as accidents and injuries. The tendency to automatically interpret abnormal performance on this type of SVT as evidence of feigning is similar to studies that have considered the diagnosis of factitious disorder as proven when the only evidence is that an individual has symptoms that are incongruent with typical disease processes and has normal results on the relevant investigations[62]. However, in the absence of other evidence, such an individual would fulfil the Diagnostic and Statistical Manual of Mental disorders, 5th Edition criteria for FND[74].

The clinical implications of the type of positive signs used to diagnose FND, such as Hoover's sign and the tremor entrainment sign are important to consider in this context. Failure of voluntary hip extension in Hoover's sign, with normal automatic movement when flexing the

contralateral hip, could also be read as a performance validity failure. Indeed, these positive signs of FND do not, on their own, distinguish FND from feigning. Therefore, we need to be very careful in how we interpret and even label 'poor performance' and not to necessarily equate it with feigning.

[H2] Reliability of reported experience

We do not have space for a detailed discussion of how to identify feigning and malingering in individuals with possible FND but, in broad terms, the diagnosis of FND does rely on the clinician accepting at face value the reported experience of the individual. Therefore, evidence that brings the reliability of that reported experience into serious doubt should be a red flag for feigning or wilful exaggeration[63,64]. Such evidence would include a major discrepancy between reported and observed function. Someone with FND should have a good idea of what they can and cannot do, for example, they should know if they can sometimes walk and sometimes cannot. On occasion, their movement might be better than they are aware of, but that should not impair their knowledge of how they can use their movement, for example, to stand or walk. Subjective reports from the individual should be broadly consistent with documented history, for example, medical records or witness statements. Similarly, repeated and serious evidence of deception of others, including clinicians, if clearly substantiated such as by surveillance or social media, should call in to question an individual's reliability.

The presence of a medicolegal claim or other clear source of material benefit creates conditions in which some individuals might be motivated to report symptoms less reliably, but are not, in themselves evidence of feigning. Furthermore, in our experience, evidence of discrepancies between a patient's self-reported symptoms and their self-reported day-to-day

function — for example, someone who reports ’10 out of 10 pain’ but is able to go shopping — is common in many settings in neurological practice and not just FND. This situation indicates difficulty in scaling the severity of symptoms but not absence of the symptoms reported.

Feigning does occur in clinical practice, but how commonly it occurs is still unclear. Estimates are often drawn from non-typical practice, for example, in a forensic or medicolegal setting where estimates of feigning in neuropsychological assessments range from 15–40%[65–67]. Clinicians might fear missing a diagnosis of factitious disorder, or not identifying a patient who is malingering, as they do not wish to be deceived. However, assuming feigning when symptoms are genuine causes direct harm through stigma and alienation from health services as well as indirect harm by preventing access to potentially beneficial treatment.

[H2] Overlap between feigning and FND

The evidence presented above does not imply that people with FND never feign or exaggerate symptoms. Feigning and wilful exaggeration occur in some people with FND, as indeed they do in some people with other causes of illness. One hypothesis attempts to resolve the issue of feigning and FND by suggesting that there is a spectrum of wilfulness, a concept that tends to suggest that most people with FND are feigning, or have feigned in the past, to some degree[75]. Instead, we argue that conflating the diagnosis of FND with feigning or malingering is incorrect — the two are categorically different. Therefore, if individuals with FND happen to feign or exaggerate symptoms, this is an additional phenomenon and not the same as FND. As one example of this, evidence of the personal experience of people with factitious disorder indicates that these individuals not only consciously produce their symptoms, but also have

conscious and self-aware motivations as to their behaviour[76]. In our opinion, this characteristic is categorically different from the mechanism and personal experience of FND.

Human beings commonly use deception as part of normal communication, and often for personal gain. For example, 72% of healthcare workers questioned after returning from a "sick day" admitted that they were not sick[68], a third of National Institutes of Health funded scientists admitted to some form of fabrication, plagiarism or falsification in their work[69], and most people are said to lie about twice a day[70]. Therefore, such behaviour is expected to also be present in people who are ill, perhaps especially when there are external drivers to such behaviour, for example, in the context of litigation and in a forensic setting. Amplification of symptom report might be particularly likely if individuals sense that they are not being taken seriously.

This feeling of not being taken seriously might be particularly relevant for people with FND. Evidence indicates that, compared with other conditions, health professionals have less knowledge about FND and poorer attitudes towards people with the disorder[77, 78, 79]. Furthermore, the existence of structured care pathways and access to suitable rehabilitative treatments for FND are generally lacking[71]. Indeed, people with FND often report receiving a diagnostic explanation that leaves them feeling that symptoms are not real[80]. This experience, in combination with an approach to management that offers no support or treatment, would tend to lead to an understandable, logical change in behaviour to seek help repeatedly and to maximise the expression of symptoms to try to get understanding and help. Thus, the styles of behaviour that might lead to accusation of feigning in some people with FND might, at least in part, be innate and understandable responses to external stimuli, rather than internally driven choices.

[H1] Conclusions

The issue of feigning looms large over people with FND in neurological practice and has been a major barrier to diagnosis and treatment. Here we have assembled a wide variety of evidence (summarised in table 2) that we feel supports the conclusion that FND is a disorder which is categorically separate from feigning, exaggeration and malingering. Furthermore, FND is a disorder that we argue ought to exist based on active inference models of brain function. The evidence presented here adds to existing data on the high prevalence, health and social economic cost, poor prognosis and severely impaired quality of life that relates to the diagnosis of FND [2-4]. It also underpins the clinical and ethical call[71] for provision of improved access to existing evidence-based management and treatment for people with FND as well as investment in research to improve our understanding of FND aetiology, pathophysiology and therapeutics.

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Table 1 | Historical consistency of typical FND presentations

FND presentation	Functional leg weakness	Functional dystonia	Functional facial dystonia	Functional seizures
Clinical features	Dragging gait with hip externally rotated	Clenched fist sometimes with wrist flexion	Orbicularis oculis contraction and lowered eyebrow	'Arc-de-cercle' appearance
19th century	[NRNEUROL-Edwards-t1]	[NRNEUROL-Edwards-t2]	[NRNEUROL-Edwards-t3]	[NRNEUROL-Edwards-t4]
21st century	[NRNEUROL-Edwards-t5]	[NRNEUROL-Edwards-t6]	[NRNEUROL-Edwards-t7]	[NRNEUROL-Edwards-t8]

FND, functional neurological disorder. Top row of images, from left to right: adapted from ref[81], adapted from ref[89], adapted from ref[84][THIS IS A SENSITIVE IMAGE: USE APPROPRIATE XML CODING], adapted from ref[86][THIS IS A SENSITIVE IMAGE: USE APPROPRIATE XML CODING]. Bottom row of images, from left to right: adapted from ref[90], adapted from ref[83], adapted from ref[85][THIS IS A SENSITIVE IMAGE: USE APPROPRIATE XML CODING], adapted from ref[87][THIS IS A SENSITIVE IMAGE: USE APPROPRIATE XML CODING]

Table 2. Clinical and epidemiological evidence arguing against FND being a result of feigning

Consistent clinical features in FND	Example	If FND was simulated	Historical consistency	Geographical consistency
Objective symptom patterns on observation and examination	Dragging leg, curled fingers, typical facial spasm, Hoover's sign (involuntary extension of weak leg when flexing the contralateral leg against resistance), seizure semiology	Could occur, although differences between these symptoms and the symptoms observed in healthy controls asked to simulate have been identified[6,7]	Yes	Yes
Subjective experiences	Somatic and dissociative symptoms before and during a seizure Loss of mental imagery of a limb	Unlikely to occur. Hard to research. Not observed in healthy controls asked to simulate symptoms[7]	Yes	Yes
Comorbid somatic symptoms[a]	Pain, fatigue, cognitive symptoms	If someone has so many disabling symptoms already, it does not seem logical to simulate additional ones	Yes	Yes
Comorbid mental health symptoms[a]	Anxiety, depression, OCD	Given the stigma associated with mental health symptoms it would be unexpected for patients to also simulate these symptoms in addition to FND	Yes	Yes
Predisposing factors	Other functional disorders Adverse childhood experience(s)	In our experience, patients typically don't connect their different functional disorders, and it would therefore be unusual for someone to feign multiple functional disorders. We have also observed that adverse experiences, such as mental health comorbidity are often hidden	Yes	Yes
Precipitating factors	Physical injury, dissociative experiences, other medical illness	Would not be expected to have such striking similarity between patients. Hard to research if wanting to feign symptoms and signs.	Yes	Yes
Interaction with investigation	Has event during EEG even with an open transparent explanation	Allowing an event to be captured would serve no clear purpose.	Yes	Yes
Response to treatment studies	Differential improvement in RCT	Treatment would not be expected to improve simulated symptoms, Improvement of simulated symptoms would not be expected to occur in interventions based on proposed pathophysiology of FND compared with a control intervention of similar intensity	NA	NA

[a]Not always present in FND. EEG, electroencephalogram; FND, functional neurological disorder; NA, no available historical or geographically distributed evidence on differential improvements in controlled trials; OCD, obsessive compulsive disorder; RCT, randomized control trial

Figure 1 | A range of functional neuroimaging and neurophysiological evidence supporting FND as a disorder distinct from feigning.

a| Hypoactivation (red) of right temporoparietal junction observed in functional tremor compared with voluntary tremor, detected with functional MRI. **b |** Single photon emission computed tomography showing recovery of hypoactivation in thalamus, caudate and putamen corresponding with clinical recovery in an individual with functional neurological disorder (FND). **c |** Increased precision in Libet Clock task in individuals with FND compared with healthy individuals. M (movement) refers to the perceived time the participant made a voluntary movement. W (will) refers to the perceived time the participant felt the intention to move. **d |** Sensory attenuation testing shows greater task precision in individuals with FND compared with healthy controls. **e |** A specific pattern of response indicating poor evidence accumulation in individuals with functional motor disorder (FMD) during temporal discrimination testing. **f |** Broken escalator phenomenon shows similar motor learning but abnormal persistence of locomotor after effects in individuals with FND compared with healthy individuals. N, Newtons

Part a adapted from ref.[39]. Part b adapted from ref.[44] and ref.[90]. Part c adapted from ref.[45]. Part d adapted from ref.[50]. Part e adapted from ref.[52]. Part f adapted from ref.[53].

Box 1. The active inference model of brain function

A neural system underpins the conscious experience of our body and the world[54]. The multilayered anatomical organisation of the nervous system, with reciprocal connections between layers, provides the physical basis for theories that our conscious experience relates to “message passing” between hierarchical levels of processing where predictions (based on prior experience) about expected sensory inputs are “tested against” actual sensory inputs[54].

According to these theories, perceptual experience arises from the collision of these two sets of data. Crucially, the strength or "precision" of these two data sets is not fixed, but is instead in a constant state of flux. This variation in precision results in the possibility of major deviance between objective sensory data and perceptual experience. The attention (or salience) network is likely to have a major role in biasing specific "channels" of sensory input or feedback, thus acting as a gain or "volume" function. Interoception, the conscious and preconscious experience of the viscera of the body, which is amongst other things a fundamental quanta of emotion and emotional regulation, is also underpinned by a similar system[55].

Glossary

Psychophysical: study of the interaction between physical stimuli and their psychological correlates

Psychophysiological: study of the relationship between physiological phenomena and psychological phenomena.

Feigning: the deliberate and voluntary production of physical symptoms and signs in order to deceive.

Factitious Disorder: deceptive falsification of physical or psychological symptoms or signs, or self-induced injury or illness in the absence of an external reward, but typically to receive healthcare, or care from others.

Malingering: deceptive falsification of physical or psychological symptoms for external reward and is not a diagnostic category in the Diagnostic and Statistical Manual of Mental disorders, 5th Edition

Go/no-go task: an experimental paradigm in which participants are given a cue to get ready to move and then are given either a “go” cue, where they should move or a “no-go” cue which means that they should withhold the prepared movement.

Feed-forward model: a model of movement and sensation that relates to predictions about expected movement and sensation, which are integrated with feed-back from actual sensory input.

Temporal discrimination threshold: the minimum time between two sensory stimuli that a person can perceive.

Drift diffusion model: a way of modelling the cognitive processes involved in a two-choice decision task.

Hoover’s sign: A sign of functional leg weakness where voluntary hip extension is weak when tested directly but returns to normal power when the same movement is triggered by contralateral hip flexion.

Tremor entrainment test: A test for functional tremor where the frequency of the tremor changes to match the frequency of an externally paced tapping movement.

Locomotor aftereffects: A change in gait pattern that is triggered by exposure to a stimulus such as walking on split belt treadmill with each leg going at a different speed, which then lasts after the stimulus is withdrawn.

In this Perspective, Mark Edwards and colleagues present their opinion that functional neurological disorder is categorically different from feigning and malingering. They discuss clinical, epidemiological and experimental evidence in support of this view.