Challenges to the Diagnosis of Functional Neurological Disorder: Feigning, Intentionality, and Responsibility

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Abstract The diagnosis of Functional Neurological Disorder (FND) requires differentiation from other neurologic diseases/syndromes, and from the comparatively rare diagnosis of feigning (Malingering and Factitious Disorder). Analyzing the process of diagnosing FND reveals a necessary element of presumption, which I propose underlies some of the uncertainty, discomfort, and stigma associated with this diagnosis. A conflict between the neurologist’s natural social cognition and professional judgement (cognitive dissonance) can be understood by applying a framework originally designed for the determination of moral responsibility. Understanding the source of this cognitive dissonance may effect its alleviation, and in turn, allow more compassionate treatment of patients with FND.

Keywords Functional neurological disorder · Intentionality · Intention-attribution · Reasons-responsiveness · Reactive-attitudes

Introduction

In 2018, at their annual meeting, the American Academy of Neurology hosted a morning plenary session that was framed as a debate. The resolution: should Functional Neurological Disorder be cared for by psychiatrists, or neurologists? This resolution was grounded in 150 years of history, during which neurology and psychiatry diverged from a common field, concerned with both the neurologic system and the emergent psychology of the mind. Objective clinical tools – the neurologic examination, electrophysiologic recording, and neuroimaging among them – developed, which could reliably identify discrete or diffuse neuropathologies and correlate their associated symptoms. This so called “organic” neurologic disease was differentiated from a second category – disease not easily associated with a specific lesion or pathology. This second category has had many names over time: hysteria, psychosomatic disease, and most recently Functional Neurological Disorder (FND).

FND is defined in the Diagnostic and Statistical Manual of Mental Disorders (DSM-V) as altered voluntary motor or sensory function, with clinical findings providing evidence of “incompatibility between the symptom and recognized neurologic or medical conditions” [1]. This definition lays bare the fact that as neurologic diseases are “recognized”, the clinical boundaries around FND are necessarily redrawn. Indeed as clinical neurology and neuroscience advanced, the conception of “lesion” expanded, diseases were redefined, and the category of FND necessarily shrank. Epilepsy, which at one time was felt to be psychological or even spiritual in origin, was defined by its electrographic correlates; so too...
Dystonia was thought to be entirely psychogenic until Oppenheim’s initial recognition of an inherited form of the disease in 1911. Recent research into FND has a similar goal: to ground the disorder in a neuro-pathophysiologic framework and thus squarely in the domain of the clinical neurologist.

Neurologists however, have generally accepted FND as neurologic based on the presence of neurologic symptoms, even as the field concurrently works to define the neuropathology. During the clinical encounter though, when faced with unexplained symptoms and undifferentiated disease, the diagnosis of FND does not require an understanding of hypothesized pathophysiology. Instead, the neurologist follows a pragmatic process that is engrained during training and reinforced in clinical practice.

In the following essay I outline the process of diagnosing FND, including a problematic element of presumptive diagnosis. I then introduce a few concepts from the psychological and philosophical literature to create a model of FND diagnosis, which I propose is inevitably accompanied by “cognitive dissonance” – the simultaneous adoption of multiple incongruous beliefs.

The General Process of Diagnosing FND

Diagnosis of FND requires differentiation from both (1) other neurologic diseases, of which FND can be a mimic, and (2) feigned illness (Malingering and Factitious Disorder).

The process of identifying and distinguishing FND from other neurologic disease involves both systematic and heuristic methods. The systematic methods encompass the practice of clinical neurology, but as directed to FND this can be summarized as identifying “positive” and “negative” features. “Positive” features are those which are characteristic of FND: i.e. variable or waxing-and-waning course, examination findings such as distractibility and variability (of weakness or tremor for example), “give-way” weakness, and Hoover’s sign [2]. “Negative” features are those which are not characteristic of FND and so point away from the diagnosis. These include a history of gradual or insidious progression, any objective abnormality on the neurologic examination related to the patient’s complaint (such as hypo/hyper-reflexia), and relevant abnormalities on neuroimaging or other diagnostic testing. Various heuristic methods are often used by experienced neurologists, the most common of which is pattern-recognition/representativeness [3]: identifying that the patient’s symptoms do not look like another neurologic disease or syndrome which has been seen by the neurologist in the past. Heuristics are highly prone to bias but can be valuable components of the diagnostic process when combined with systematic methods.¹

The second, more difficult task, is to differentiate FND from feigned illness: Malingering, and Factitious Disorder. These two disorders are defined by the presence of direct benefits from seeking medical care, called “gain”. Two types of gain – “primary” and “secondary” – are formally differentiated based on their incentives. In primary gain, the incentives are internalized: comfort from anxiety for example, or relief of guilt, or satisfaction of a compulsion. In secondary gain, incentives are typically externalized: shelter, or potential for financial compensation, or material evidence for legal actions in the form of medical records. Feigned illness is thus separated into two diagnoses based on the predominant type of gain: “Factitious Disorder” when the gain is primary, and “Malingering” when the gain is secondary. However, a patient’s motivation is individualized, subjective, and often driven by both internalized and externalized incentives and disincentives. The distinction between Malingering and Factitious Disorder is therefore fuzzy, and one or both diagnoses may be appropriate at different times.

One method of differentiating FND and feigned illness is the identification of gain during the clinical encounter. A pattern of seeking medical care, often supported by inexplicable laboratory findings and paraphernalia of tampering (syringes, hidden medications) is evidence of primary gain (Factitious Disorder), but this often requires some knowledge of the patient’s behavior over time. In Factitious Disorder involving feigned neurologic symptoms specifically, objective evidence is often absent, and most cases are

¹ Rare, underdiagnosed, and new/emerging diseases are particularly prone to diagnostic error and are sometimes misdiagnosed as FND. Minimizing this type of error is one of the strongest arguments for research into the development of biomarkers and objective ancillary tests to aid in the diagnosis of FND.
determined through “detection of previous history… use of false names, false histories, and peregrination” [4]. Without this historical evidence the distinction between Factitious Disorder and FND is very difficult. Malingering can likewise be detected based on a past or present interest in secondary gain, though this is often covert, and discovery may require longitudinal research or special investigations by insurance or legal professionals [5]. The ethical cost of these considerations is also high because professional and ethical standards dictate that physicians trust their patients unless there is specific reason to suspect deception [6–8]. It has also been theorized that feigned illness may sometimes evolve, through a process of self-deception, into symptoms of FND, which makes a clinical differentiation based on present or past gain all the more difficult [9].

A second method of differentiating these conditions could be to decide if the symptoms are involuntary (as in FND) or voluntary (as in feigned illness). Voluntary action can be thought of as action that is “driven by an agent” and not manipulated by some external force, or accidental, or coerced [10]. Any neuropathology could act as an external force, including an infarction, focal brain injury, or neurodegeneration. Absent identifiable neuropathology, the question of whether the movement is “driven by an agent” is the same as whether to attribute intentionality to the patient’s symptom. Unfortunately neurologists have no tools to aid in this objective intention-attribution. Clinicians are not trained in the identification of intentionality, nor can physical or psychological examination techniques reliably assess for presence of intention. While some studies have shown measurable differences in sensory processing in patients with FND (temporal binding and sensory attenuation), only in cases of tremor or myoclonus does ancillary testing have the capability to distinguish functional symptoms, and then only by quantifying properties of the movements themselves, rather than by identifying signatures of intentionality [11, 12]. We could simply ask the patient if they are acting intentionally, though this “reverting to naïve reliance on subjective reports” would fail to identify patients with feigned illness [13]. Moreover, it has been shown that patients with unexplained symptoms are more likely to report inconsistent and arguably deceptive histories, supporting a role for some inquiry, confirmation, and judgement in obtaining an accurate history. For these reasons intention-attribution is difficult to purposefully achieve.

Intention-attribution also has moral consequences which bias the decision. Malingering should be construed as immoral, and Factitious Disorder, while perhaps not immoral if driven entirely by compulsion, often has moral implications insofar as it usually stems from voluntary behavior. Physicians are trained to remain morally neutral on the actions and decisions of their patients, focusing on health outcomes rather than moral consequence. The negative moral associations of feigned illness therefore draw neurologists away from these diagnoses. Simply put, without evidence, it is unethical and unprofessional to call a patient a liar.

At this point then, we can see that while neurologists may have the ability to differentiate other neurologic disease from FND, the distinction of FND from feigned illness is difficult or impossible in the absence of direct evidence of primary or secondary gain (which is often unavailable) or a method of assessing intentionality (which is always unavailable). The only course at this point, and the one taken in most cases, is a presumptive diagnosis of FND.

**Psychological Consequences**

The necessary presumptive diagnosis of FND has consequences for the neurologist. The first and most unfortunate is the discomfort of uncertainty. This is apparent in qualitative studies of neurologists, with statements such as.

“Is this person malingering? Are they putting it on for my benefit? I have great difficulty sorting that one out.”

“The conversion is not manipulation, supposedly; it’s not somebody who is malingering, supposedly; though I find it very difficult to draw the line very clearly between the two”

demonstrating the challenge [6]. Neurologists sometimes feel as if the patient is acting intentionally and therefore deceiving them, even after rationally determining this not to be the case, vis-à-vis a diagnosis of FND. Indeed, often neurologists understand and characterize FND symptoms as deception, although most simultaneously accept the
unintentional nature of their patients’ symptoms [6].

This then is the root source of cognitive dissonance, but how can it be resolved? In general, cognitive dissonance is relieved through adoption of beliefs which resolve the internal conflict, and in this case, through a better understanding of the causes of the cognitive dissonance in the deliberative and implicit processes of intention-attribution.

**Deliberative Intentionality:**
**Reasons-Responsiveness**

The question of how we decide whether symptoms are voluntary, or in other words whether a patient’s symptoms are intentional, is what Harry Frankfurt called the “problem of action”: distinguishing “between the bodily movements that he [or she] makes, and those that occur without his [or her] making them” [15].

Frankfurt argues that intentional movements are those conducted “under the person’s guidance”, and that we perceive intentionality when we observe or detect

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2 Deception in this framework is communicative; it originates with the patient but emerges from characteristics of the interaction between the doctor and patient. In the performance of the doctor-patient relationship for example, both parties behave according to expected cultural norms, rather than being strictly truthful. Kanaan has explored various lines of evidence that patients with FND are deceptive (their motivations for deception, the apparent voluntariness of symptoms, the idea of simulation or “pantomime”) and offers a discussion of whether these deceptions can and should be classified as lying based on philosophical and behavioral evidence [14]. As Kanaan says, “all patients with hysteria will lie from time to time, for they are human.” An explanation that he does not consider is that the deception can be entirely internal – self deception. Assuming the self-deception is complete, any external communication resulting from the self-lie is not deceptive. The patient’s belief in their own condition is complete enough that the communication with their neurologist is a truthful reflection of their beliefs. The advantage of both frameworks is that they allow for the presence of deception without assigning further intentionality to the expression of symptoms or the consequences. As discussed by Merckelbach and Merten, intentionally deceptive malingering might sometimes evolve into unintentional (possibly self-deceptive) FND, further emphasizing that deception can take many forms, independent of intentionality [9].

3 Although the word “action” suggests positive symptoms (i.e. tremor), I am also referring to negative symptoms (weakness, sensory loss) which in their expression do require action. Feigned weakness for example being produced through the action of feigning weakness.

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Might positive-findings of FND be our method of detection? In identifying a positive Hoover’s sign, or the entrainment of a tremor, the neurologist is detecting that the motor system is capable of modifying action: a paretic limb can move under certain conditions, and a tremor frequency can be entrained to that of another movement (respectively). However, in these cases the patient is not necessary acting to contract the paralyzed hip flexor, nor to change the frequency of their tremor. Indeed these positive-findings are most useful precisely because they are involuntary. Therefore they do not speak to the presence or absence of intentionality. We are back to square one.

Another possibility is that we attribute intentionality through some kind of deliberative cognitive process. Fischer and Ravizza draw out a model of this deliberation in their discussion of moral responsibility. They propose that agents are morally responsible for their actions when those actions are reasons-responsive. They outline multiple subtypes of reasons-responsiveness, but the one most relevant to a discussion of intentionality is the easiest to fulfill: “weak” reasons-responsiveness. Roughly speaking, an agent’s actions are weakly reasons-responsive when:

“there exist[s] some possible scenario (or possible world) in which there is a sufficient reason to do otherwise, the agent recognizes this reason, and the agent does otherwise for that reason.” [16]

In other words, if a patient is capable of modifying their actions in response to reasons (the types of reasons which have typically guided their actions in the past) then they are responsible for these actions.
For their purposes of discussing moral responsibility, Fischer and Ravizza assume that “the appropriate relationship between reasons and subsequent behavior” exists, one which has three steps: (1) “recognition” of appropriate reasons to modify behavior (2) “reactivity”, or the process of being influenced by these reasons, and (3) “translating” reasons into action through the motor system. Fischer and Ravizza thus assume that intentionality (recognition, reactivity, translating) is intact, and can then question moral responsibility. However, the same arguments can be used to assess intentionality itself when the ability to “translate” reasons is in question. For our purposes we need not bother with the extent of receptivity or reactivity: it matters only that they be present to some degree in some cases. Take for example a patient with paralysis of the right arm, perhaps due to stroke or perhaps due to FND. The arm is pinched by an examiner, but still does not move. The patient fails to respond to the reason of “avoid pain” due to an inability to translate reasons into this specific action. However, the patient is certainly able to recognize, react, and translate avoidance of pain into movements of their other limbs.4 Because the patient has intentionality in a general sense, we can apply the framework of reasons-responsiveness to assess the intentionality of specific symptoms.

Appealing to a method of assessing moral responsibility might seem a step too far here. It is true that questions of moral responsibility, of being blame-worthy, are tied to outcomes in a way that intentionality is not. Imagine a woman Jane, who we find strolling down the street with her friend. Noticing a mosquito on her arm, Jane swiftly moves to swat it with her other hand. In so doing, Jane’s arm strikes her friend, who again careens. The arm moves swiftly and strikes her friend, who again careens. The cyclist crashes. Holding the mechanism of her movement constant (the stroke and resultant ballismus), it is impossible to conceive of any reason that would compel Jane to act differently. She would certainly be able to appropriately recognize and be influenced by reasons to not fling her arm (not wanting to cause pain or injury for example), though a discrete brain-lesion has interfered with her ability to translate these reasons into modified action. In this clear case, assessment of reasons-responsiveness is not necessary for a discussion of diagnosis (because we have examination and neuroimaging evidence of a lesion). Neither would it seem necessary for a discussion of moral responsibility. Intuitively, the ballismus is something that happened to Jane, not something that she did (or was done under her guidance). However, our concern is with the fuzzy territory wherein symptoms can exist on a spectrum of intentionality. FND is certainly in this territory, but so too is another disorder which is more accepted as purely “organic”: tics.

Imagine now that Jane has a tic disorder. The urge to tic is usually suppressible (indeed this suppressibility helps to define the diagnosis), but at some times the urge becomes so strong as to be considered clinically insuppressible. Now consider that in expressing her insuppressible urge to tic, Jane flings out one arm, which again strikes her unlucky friend, who

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4 One might invoke another positive-finding of FND here, the “arm-drop test”, wherein a paralyzed limb is dropped above a patients face and allowed to fall. If the arm avoids the face, presumably for the reason of avoiding pain, is the patient reasons-responsive and therefore acting intentionally? Whether or not the patient is consciously acting to avoid the face is uncertain, and so again this positive finding does not demand we attribute intentionality.
again careens into the passing cyclist. In this case, Jane can again recognize and be influenced by reasons. The question arises of whether she is unable to translate reasons into action due to her disorder, and the answer here is not quite straightforward. It is not self-evident from the diagnosis of tics, or the established neuropathology of tics, that Jane would be unable to translate reasons into action. In the majority of cases, tics remain suppressible: patients can translate reasons to not tic into modified action (in this case, inaction). However, in some cases, patients tell us that tics are insuppressible. Neurologists trust this report and verify through observation that tics sometimes persist despite many objective and subjective reasons that they should be suppressed. This lack of reasons-responsiveness is critical to how we treat the movements: both how we might ascribe a moral responsibility, but also (much more practically for neurologists), what medications or therapies to recommend to the patient.5

Under this deliberative model then, the neurologist need only gather a history which demonstrates a lack of reasons-responsiveness. The report of the patient contributes reasons to change the action, to which the patient’s symptoms run counter. “I want to move, so that I can get back to work” says the patient with weakness, “however, I am unable”. In the absence of a psychiatric or neurologic diagnosis to suggest a general difficulty with recognition or reactivity, this lack of reasons-responsiveness suggests an issue with translation, and thus an absence of intentionality, consistent with FND [16]. In practice, simply by presenting with a desire to address neurologic symptoms based on demonstrable reasons, and the persistence of these symptoms despite these reasons, patients fail to demonstrate reasons-responsiveness.

This lack of reasons-responsiveness can be a method of withdrawing (or not attributing) intentionality in theory, but in practice it is difficult to argue that neurologists are conducting these sorts of cognitively demanding analyses implicitly. We do not routinely present counter-factuals or lie to patients to determine if “there exist some possible scenario (or possible world)” in which the symptoms are modified. Something else must be happening more automatically. An adequate explanation of this process must involve a method of determining intentionality more implicitly.

### Implicit Intentionality: Social Cognition

Theories of social cognition can provide a reasonable account of this implicit determination. Bertram Malle and colleagues define intentionality as “the property of actions that make ordinary people and scholars alike call them purposeful, meant, or done intentionally” [17]. Under this view, intentionality is not an objective property of agents, but instead is an explanatory tool in human social cognition. We assign intentionality when we perceive that another person can understand their own reasons, and that their thinking is rational [18], judged against a normative standard of behavior in society [19].6 These theories still necessitate some small degree of rational deliberation, however even this may not be necessary. P.F. Strawson illustrates that implicit emotional responses to others are critical to our theory of mind [20]. When we observe others performing actions, we experience a reactive emotional response (or “reactive-attitude”), such as resentment for a negative action, or gratitude for positive actions. Strawson argues that our experience of these reactive-attitudes is responsible for our attribution of intentionality – our belief that the agent is acting with free-will. In cases where the agent is constrained, or compelled, or incapable of acting otherwise, the reactive-attitudes are absent and we feel that the agent is not acting intentionally. In a few circumstances – mental illness for example, or in young children – we naturally counter our reactive-attitudes and adopt an objective attitude. We may also

5 In the case of insuppressible tics, movement-disorders neurologists have invented a term (“unvoluntary”) to describe this fuzzy territory between voluntary (intentional) and involuntary (unintentional). This word is of course meaningless. The very fact of its creation and use reflects the difficulty that we face when diagnosis relies on the attribution of intentionality. This is a common problem for tics and for FND, and a model of reasons-responsiveness can be useful in both cases.

6 Fischer and Ravizza also address these ideas of normativity, describing a moderate reasons-responsiveness which forms the meat around their arguments. Under moderate reasons-responsiveness, a morally-responsible agent’s reasons are not “unusual” or “incoherent”, and therefore are based on a normative social and cultural standard which might not apply to patients with mental illness, certain cognitive deficits, or to children.
decide to do this consciously, deploying this cognitive “resource” for practical purposes, such as in a therapeutic relationship [21].

How does this all play out when a neurologist evaluates a patient with FND? First, through the collection of history, performance of an examination, and collection of ancillary data, the neurologist uncovers evidence that the relevant neurologic system (i.e. the motor system in a patient with weakness) is in fact intact. The implicit response to this disagreement, the reactive-attitude, is perhaps mistrust, or some degree of resentment with having been “deceived”. These reactive-attitudes lead directly to the impression that the patient is acting under their own guidance: that they possess intentionality. However, the neurologist further recognizes that these emotions run counter to their professional training, and that the conclusions ignore the potential diagnosis of FND. Lacking further means to determine if the patient is acting intentionally (see “The General Process of Diagnosing FND” section), they deploy a cognitive “resource” to objectify and thereby withdraw intentionality. But what is the overriding factor here that has allowed them to use their resource and objectify? Simply the diagnosis of FND. Unfortunately, we now have a tautology: FND is defined by lack of intentionality, but now apparently lack of intentionality is established by the diagnosis of FND. Not only is there no way to definitively conclude that patients are acting unintentionally (“The General Process of Diagnosing FND” section), but now also no overriding factor to justify adopting an objective attitude. Thus, a combination of innate social cognition, and professional reasoning leads to a logical inconsistency. How can we proceed?

I propose that the deliberative model of reasons-responsiveness is the method used by neurologists diagnosing FND. Absent a rational way to determine intentionality, and faced with reactive-attitudes, we fall back to the history provided by the patient. Is there “sufficient reason to do otherwise?” Does the patient “recognize this reason” and then do they modify their behavior? If not, the diagnosis of FND is made simply by recognizing that the patient is unable to translate reasons into action, that they are unable to overcome their symptoms. The deliberative model of reasons-responsiveness accounts for the importance of our impression of the patient and their history, while the implicit model of reactive-attitudes accounts for the tendency to ascribe some degree of intentionality to these patients. When combined, these theories uncover a conflict between our innate and professional cognition. It is this conflict that produces the cognitive dissonance in neurologists treating FND.

The source of cognitive dissonance now being made explicit, it should be emphasized that differentiating feigned illness and FND remains difficult and cannot be done by either deliberate or implicit intention attribution. Absent external evidence or a reliable neurologic biomarker of feigning, a clinical observer does not have access to the internal motivational states of the patient. Some persistent cognitive dissonance is thus inevitable in diagnosis of FND.

**Conclusion**

In the midst of the COVID-19 vaccination campaign, there arose a phenomenon fueled by social media platforms, of vaccine-related FND and functional “TikTok-Tics” [22]. Given this surge in FND, the intense social and political environment, and the strong disagreements between pro- and anti-vaccination communities, reasonable questions arose: what are the motivations of these afflicted persons? How many of them might be “putting it on”? Is FND a legitimate diagnosis? Neurologists have had these same questions for years, and the answers are not simple.

For the neurologist, a diagnosis of FND requires a determination of intentionality which at first glance seems difficult or impossible, necessitating a presumptive diagnosis which leads to discomfort and uncertainty. However, when we analyze this process from a philosophical and psychological standpoint, we can show that the diagnosis of FND leads to an inevitable cognitive dissonance that emerges not merely from the patient’s presentation, but also the neurologist’s implicit and deliberate cognition. This can be understood, and so overcome. It need not prevent us from affording trust in the patient and their subjective experience.

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