Functional Neurologic Symptom Disorder:

Definition, Etiology, Symptomatology, and Treatment

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Neuropsychiatric disorders exist at the bridge between severe neurological and psychiatric impairment. Recognized as one of the most widely occurring psychopathologies within this category is Functional Neurologic Symptom Disorder (FNSD), also referred to as Conversion Disorder (American Psychiatric Association [APA], 2022). FNSD is a significantly debilitating disorder characterized by various somatoform symptoms (e.g., weakness, paralysis, speech impairment, seizures) that are not consistent with any known medical or neurological conditions (APA, 2022). While the available literature is markedly heterogeneous, the prevalence of FNSD is estimated by Finkelstein et al. (2024) at 50 to 1,600 cases per 100,000 and incidence at 10-22 cases per 100,000. Further variance in incidence is noted in pediatric populations. This heterogeneity is likely due to methodological limitations encountered by the contributing researchers, such as reliance on indirect extrapolation methods for incidence and prevalence rates. Furthermore, the data in question are derived from studies spanning populations of differing geographical, sociocultural, and socioeconomic statuses. Regardless, these data, while broad in their scope, are highly indicative of their prominent occurrence across populations at both ends of the spectrum. The ensuing paper will encompass a comprehensive review of all aspects of FNSD, covering its clinical definition, frequently presenting symptomatology, prevailing etiological hypotheses, and treatment/prevention plans.

Clinical Definition

The DSM-5-TR (APA, 2022) requires that the affected individual meet four core criteria to qualify for a formal diagnosis of FNSD. Criterion A mandates the occurrence of “one or more symptoms of altered voluntary motor or sensory function,” the phenotypic presentations of

which are discussed in the subsequent section on symptomatology (p. 360). Criterion B is the most distinguishing of the four, and requires that the presentation of the individual in question be incompatible with any other symptomatologically similar medical or neurological conditions. An example of this would be a patient presenting with functional limb paralysis in their left leg exhibiting Hoover's sign, wherein weakness in the affected limb is momentarily resolved upon the execution of a counter-movement in the healthy limb (in this case, the *right* leg). The weakness under these circumstances can consequently be attributed to a psychogenic origin rather than an organic one. Criterion C functions in tandem with Criterion B by necessitating that positive clinical findings are not better explained by another established medical or neurological condition. If an individual presents with symptoms commonly ascribed to carpal tunnel syndrome (CTS) and a nerve conduction study in the affected extremity confirms the compression of the median nerve, a diagnosis of CTS would supersede FNSD. Criterion D, being the most standardized among all psychopathological diagnoses, requires that the symptoms or deficits cause clinically significant levels of "distress or impairment in social, occupational, or other important areas of functioning or warrants medical evaluation" (pp. 360-361). These four criteria work in conjunction to provide a general clinical framework for the diagnosis of FNSD. Beyond the four main diagnostic criteria outlined by the DSM-5-TR, further diagnostic specifiers regarding the nature, duration (i.e., acute or persistent), and context of the symptoms (i.e., in the presence or absence of a psychological stressor) are required to make a clinical diagnosis.

Symptomatology

Within any given diagnosis, many symptom permutations may exist, allowing a wide variety of patient presentations. Among other psychiatric illnesses, FNSD is unique for its significant phenotypic heterogeneity. Commonly observed symptoms of FNSD can generally be

divided into two distinct clinical domains: altered voluntary motor function and altered sensory function.

Altered Voluntary Motor Function

Altered voluntary motor function represents the most clinically prominent symptom domain of FNSD (Park, 2024). Common functional motor symptoms include limb weakness or paralysis, tremors, dystonia, speech impairments, and attacks resembling epileptic seizures (PNES), among others (APA, 2022). Park (2024) draws from a meta-analysis of 4,905 individual case studies to explore the occurrence of these various motor symptoms in Functional Movement Disorder (FMD), a subtype of FNSD that encompasses the presence of specifically motor-related symptoms. Around 3% to 8% of visits to movement disorder (MD) clinics are comprised of FMD cases. Multiple FMD phenotypes are observed, with the highest prevalence being that of functional tremors in 43% to 61% of observed cases. Having mixed symptoms was noted as being the second-highest occurring phenotypic expression of FMD, with functional Parkinsonism being the least common (Park, 2024).

The development and use of complex, structured frameworks are required for clinicians to assess the vast phenotypic heterogeneity of FNSD. To navigate this, functional motor symptoms are further bifurcated into positive and negative motor symptoms, where “negative” denotes the inhibition or deficit of certain voluntary actions and “positive” denotes the superimposition of an involuntary movement, not consciously suppressable by the affected individual (Mavroudis et al., 2025). After identifying over 60 signs of functional neurological disorder (FND) subtypes, specific bedside tests were developed and discussed in detail by Mavroudis et al. (2025) to address both positive and negative symptom domains.

Positive Motor Symptoms

Other functional positive motor symptoms include tremors, gait disorders, nonepileptic seizures, and myoclonus (sudden, involuntary muscle jerks) (Mavroudis et al., 2025). However, among all observed cases of FNDs where positive motor symptoms are present, functional tremors occur the most frequently (Park, 2024). A technique called the tremor entrainment test is used to diagnose functional tremors. In this test, the patient is asked to mimic a rhythmic tapping pattern in the healthy hand. The observation of the affected hand matching the new voluntary rhythm or being temporarily suppressed altogether indicates psychogenic origins for the tremor (Mavroudis et al., 2025).

Negative Motor Symptoms

Primary examples of negative functional motor symptoms include limb weakness or paralysis (Mavroudis et al., 2025). Specific tests, such as Hoover's sign, have been presented by Mavroudis et al. (2025) to differentiate functional weakness from organic paresis. Hoover's sign, being one of the most commonly administered bedside tests for functional weakness, entails an assessment of the strength of hip extension in the supine position, wherein flexion of the contralateral hip against resistance causes involuntary extension of the weak or paralyzed leg. Hoover's sign, among other mechanically similar indicators like the Abductor sign, allows clinicians to discern symptoms of psychogenic origin from those of organic origin with an increased degree of certainty.

Altered Sensory Function

Functional sensory symptoms/deficits exist as the second core domain of impairment caused by FNSD (APA, 2022). Within this category, somatic/tactile symptoms and special sensory symptoms represent the two sensory symptom subtypes (APA, 2022). Across all FNDs, sensory symptoms are noted as co-occurring with motor symptoms in 96% of observed cases, as

per the findings of Nielsen et al. (2026). Functional anesthesia, or the complete lack of feeling, is identified in 27% of cases, and 19% of cases have documented the more extreme sensation of functional asomatognosia (feeling complete limb absence or “death”). Sensory symptoms go beyond loss or deficits in feeling, as 88% of documented cases also experienced the positive symptom of comorbid pain.

Special sensory symptoms impair at the level of conscious perception rather than somatotopic representation, like tactile sensory symptoms. Some prominent examples of functional special sensory symptoms are olfactory loss, deafness, and vision loss. In Functional Vision Loss (FVL), the affected individual experiences sudden-onset blindness or tunnel vision (Mavroudis et al., 2025). Just as in the case of functional motor symptoms, clinicians can administer specialized bedside tests to assess the origin of various sensory-related symptoms. In the case of FVL, when presented with a mirror, the patient may report total functional blindness while simultaneously maintaining eye contact with their reflection or even going so far as to groom themselves (Mavroudis et al., 2025). This is a clear indication that the visual cortex is still functioning as intended, despite the reported blindness. Another frequently administered test involves obstacle navigation, where, assuming the reported symptoms do indeed lack any kind of organic neuro-ophthalmologic pathology, the patient can navigate around obstacles in the clinic despite claiming not to observe them (Mavroudis et al., 2025).

Etiological Hypotheses

Contemporary research into the etiology of FNSD has largely refuted former hypotheses rooted in psychoanalytical theory (Raynor & Baslet, 2021). Modern perspectives take a more multidisciplinary approach, concentrating on neuroimaging, cognitive science, and the

environment as primary risk factors. Four prevailing hypothetical models regarding the etiology of FNSD have been identified (Fobian & Elliott, 2019).

Neurobiological Model

The Neurobiological Model relies on fMRI evidence of interactions between the neuroanatomical structures that regulate emotion and the structures that regulate motor function (Fobian & Elliott, 2019). Originally proposed by Voon et al. (2011, as cited in Fobian & Elliott, 2019), the model specifically proposes that FNSD is caused by miscommunication between the limbic-motor system and prefrontal cortex. The fMRI studies analyzed by Fobian and Elliott (2019) demonstrated that presenting participants with fearful and happy facial expressions and also subjecting them to recollection of "stressful life events" generated increased connectivity between the right supplementary motor area and the right amygdala (p. 11). The Neurobiological Model posits that this interaction may be the core mechanism behind FNSD development, wherein "abnormal functional connectivity" between these neuroanatomical structures stimulates excess emotional arousal in the amygdala and triggers a previously mapped motor representation through the supplementary motor area (Fobian & Elliott, 2019, p. 12). Fobian and Elliott (2019) further connected this to the "decreased sense of agency" reported by patients with FMD over their own actions in another study, where hypoactivity was observed in the right temporoparietal junction (p. 10). The overall poor functional connectivity between the limbic regions, sensorimotor cortices, and the right temporoparietal junction suggests the perception of actions to be involuntary despite using pathways dedicated to voluntary motor control.

Bayesian Account

The Bayesian account is a neurobiologically aligned model that posits functional symptoms observed in FNSD are caused and sustained by "actions based on inference"

encompassing the affected individual's preconceived notions about symptoms, "past emotional and illness experiences," and an intense focus on their own body (Fobian & Elliott, 2019, p. 12). This model is conceptually associated with a theoretical framework called active inference, in which past experiences act as the core sources of prediction and explanation for sensory inputs (Fobian & Elliott, 2019). The inferences held by patients with FNSD, or "jumping to conclusions" bias, are viewed as a highly plausible risk factor for the brain inappropriately updating its active inference (Fobian & Elliott, 2019, p. 10). Anticipation of symptoms through Bayesian inference supplants sensory input and causes the feeling of symptoms being involuntary (Raynor & Baslet, 2021). Consequently, dysfunctional inference outside of conscious control directly leads to the functional symptoms occurring in FNSD (Fobian & Elliott, 2019, p. 12).

Cognitive Conceptual Model

The Cognitive Conceptual Model is similar to the Bayesian account in its core assertion that faulty inference is the catalyst for functional symptoms (Fobian & Elliott, 2019). In this case, the inference involves the entirety of an individual's "health scaffold," encompassing personal and parental health experiences and anxieties, both witnessed and experienced, as well as their cultural beliefs (Fobian & Elliott, 2019, p. 13). Where the Bayesian account used active inference, the Cognitive Conceptual Model branches off and uses a framework proposed by Brown (2004, as cited in Fobian & Elliott, 2019, p. 12) called the "cognitive model of unexplained illness." In this framework, internal and external influence from the experience or witness of health-related situations creates "rogue representations" that lead to a misinterpretation of physical symptoms as a genuine medical condition (as cited in Fobian & Elliott, 2019, p. 12). Subsequently, their anxiety and/or anticipation of said symptoms increases

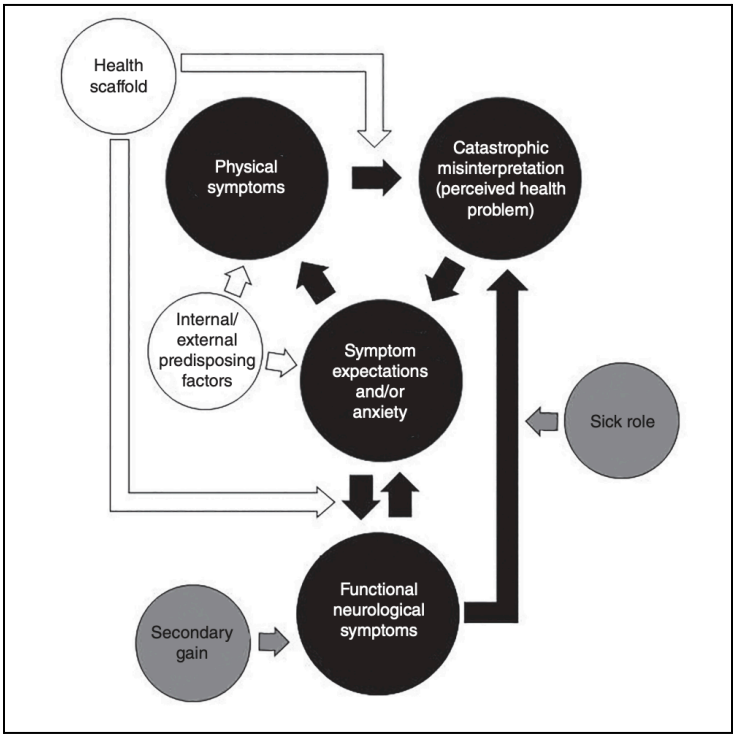
and can manifest reflexively as functional neurological behavior if it has been classically conditioned into their health scaffold (Fobian & Elliott, 2019, p. 14).

Integrated Biopsychosocial Model

The Integrated Biopsychosocial Model, proposed by Fobian and Elliott (2019), draws the most from all previous contemporary models, while simultaneously offering its own unique explanations for its origin, and attempts to blend their concepts together to represent FNSD as a multifaceted diagnosis. Their model (see Figure 1) describes predisposing factors in white, main model pathways in black, and reinforcing factors in gray.

Figure 1

The Integrated Biopsychosocial Model



Note. From "A review of functional neurological symptom disorder etiology and the integrated etiological summary model," by A. D. Fobian and L. Elliott, 2019, *Journal of Psychiatry and Neuroscience*, *44*(1), p. 13 (https://doi.org/10.1503/jpn.170190). Copyright 2019 by Joule Inc.

Fobian and Elliott (2019) argue that FNSD, being heterogeneous as it is among patient profiles, likely has a wide variety of predisposing factors contributing to the expression of similar functional symptoms. Some predisposing factors include aspects of both the shared and nonshared environment from an early age, traumatic experiences, and medical history, among many others. Reinforcers, such as the "sick role" represented in Figure 1, perpetuate the functional symptoms of FNSD after its onset (Fobian & Elliott, 2019, pp. 13-14). Staying at home, abstinence from responsibilities, and overutilization of medical facilities by the caretaking party are all prominent reinforcers proposed by the Integrated Biopsychosocial Model. Secondary gain similarly reinforces FNSD symptoms through operant conditioning by both negative (e.g., "decreased aversive responsibilities") and positive reinforcement (e.g., "increased attention from family and friends") (Fobian & Elliott, 2019, p. 14).

Treatment and Prevention

Since FNSD is an incredibly multifaceted diagnosis, treatment methods must integrate multiple approaches to be clinically effective (Park, 2024). Frequent checkups with both a psychiatrist and a neurologist are required to gauge the progress of the diagnosis. Beyond this, since no widely accepted scientific finding firmly establishes FNSD as a structural issue, pharmacological treatment has not been created or approved. Instead, psychotherapeutic modalities are the core basis of a strong treatment plan (Raynor & Baslet, 2021). The most common modalities for the treatment of FNSD are Cognitive Behavioral Therapy (CBT) and Psychodynamic Therapy. CBT, as a kind of manualized psychotherapy, is particularly helpful for the encouragement it provides to track symptoms as they occur in certain contexts so the patient can attain a great deal of understanding of "situations that create vulnerability towards symptom expression" (Raynor & Baslet, 2021, p. 6). Psychodynamic therapy for the treatment of FNSD

places a greater emphasis on recognizing early traumas through the creation of a personal narrative. The overall premise entails “greater awareness of ongoing psychic conflicts..." (Raynor & Baslet, 2021, p. 4).

There is an apparent lack of recognized preventative measures for FNSD (Fobian & Elliott, 2019). Many of the predisposing factors described by the aforementioned models are not under an individual’s control, and even the inference-based models are not combated by sincere educational efforts since these predictive processes and faulty inferences are not within the domain of conscious control. Previously mapped motor representations cannot be educated away, and so prevention must be operationally limited to the prevention of *relapse*. An important note for relapse prevention in FNSD is that, despite the extinguishment of the conditioned response, the symptoms are never truly “unlearned.” New learned behaviors get stored with previous learning, and thus have the propensity to relapse within a certain context (Fobian & Elliott, 2019). For efforts to prevent such relapse, various methods like extinction techniques (e.g., “exposure with response prevention”) have been proposed (Fobian & Elliott, 2019, p. 18).

Conclusion

FNSD has evolved dramatically from being viewed as a mystery to being recognized as a multifaceted culmination of reinforcing and precipitating social and cognitive factors (Raynor & Baslet, 2021). The sheer heterogeneity of this condition necessitates a complex multidisciplinary approach so that diagnosis and treatment are not impeded for patients (Fobian & Elliott, 2019). Bridging the contemporary models of its etiology with relevant treatment frameworks is the necessary pathway for effectively and efficiently addressing its needs (Raynor & Baslet, 2021). More than this, it will provide physicians with the additional knowledge necessary to provide

patients with a new level of understanding that hopefully breeds willingness to pursue long-term treatment (Fobian & Elliott, 2019).

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