

Dissociative neurological symptom disorder

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Dissociative Neurological Symptom Disorder (Functional Neurological Symptom Disorder)

Primary Disciplinary Field(s): Neurology, Psychiatry, Clinical Psychology

1. Core Definition and Relationship to FND

The term **Dissociative Neurological Symptom Disorder** is used conceptually and clinically by some researchers to highlight the prominent role of dissociation--a disruption of integrated consciousness, memory, identity, or perception--in the presentation of symptoms traditionally classified as Functional Neurological Symptom Disorder (FND) or **Conversion Disorder** (CD). FND is characterized by one or more symptoms of altered voluntary motor or sensory function (e.g., weakness, paralysis, seizures, blindness) that cannot be explained by recognized neurological disease or organic pathology.

The symptoms experienced by individuals with this condition are genuinely involuntary and often profoundly disabling. They are understood to arise from a complex, reversible problem in central nervous system functioning, rather than structural damage. The conceptual linkage to dissociation emphasizes that the neurological symptoms themselves may represent a form of somatoform dissociation, where a disruption in the sense of agency or sensorimotor processing manifests physically, particularly in individuals with a history of trauma or overwhelming psychological distress.

2. Etymology and Historical Evolution

The history of this disorder is long and complex, reflecting shifting views on the mind-body relationship. Symptoms resembling FND were historically grouped under the label of "hysteria," famously studied by neurologist Jean-Martin Charcot at the Salpêtrière Hospital in the late 19th century. Charcot's work, which included using hypnosis to induce and remove symptoms, demonstrated their psychological modifiability and laid the groundwork for distinguishing these presentations from organic neurological conditions.

Charcot's student, Pierre Janet, proposed that **dissociation**--a splitting of consciousness often related to trauma--was central to hysteria, suggesting that segregated traumatic memories manifested as physical symptoms. Simultaneously, Sigmund Freud, who also studied with Charcot, developed the concept of **conversion**, suggesting that repressed emotional conflicts were unconsciously "converted" into physical symptoms as a defense mechanism. This psychoanalytic heritage led to the inclusion of "Conversion Disorder" in the early editions of the Diagnostic and Statistical Manual of Mental Disorders (DSM).

A significant modern shift occurred with the publication of the DSM-5 (2013), where the name was

formally updated to Functional Neurological Symptom Disorder (Conversion Disorder). This change highlights the focus on problems in nervous system **functioning** rather than relying solely on the psychoanalytic theory of conversion. Crucially, the DSM-5 removed the requirement that the symptom must be linked to a psychological stressor, emphasizing instead the requirement of **positive clinical signs** of inconsistency (Criterion B) for diagnosis, moving the condition toward a mechanism-based understanding rooted in clinical neuroscience.

3. Key Clinical Characteristics and Symptomatology

FND manifests through a diverse array of neurological symptoms that mimic organic disease but display features (such as inconsistency, incongruity, and distractibility) inconsistent with established anatomical or physiological patterns. Onset is often abrupt, sometimes following a stressor or minor injury.

The major categories of symptoms include:

Motor Symptoms: These are highly prevalent, often presenting as functional weakness, paralysis, or abnormal movements. Functional weakness may involve "giving way" or demonstrate positive signs like **Hoover's Sign** (weakness of hip extension improves when the contralateral leg flexes against resistance).

Functional Movement Disorders: Include functional tremor (often variable and entrainable by movements of the contralateral limb), functional dystonia, and functional gait disorders (e.g., astasia-abasia), which lack the typical rhythmicity or distribution of organic counterparts.

Sensory Symptoms: Altered or absent sensation (numbness, anesthesia) is common. Functional sensory loss typically has sharp boundaries that do not follow dermatomes or peripheral nerve distributions (e.g., a "glove" or "stocking" pattern). Functional blindness or deafness may also occur, often inconsistent with objective testing results.

Non-Epileptic Seizures (NES) or Attacks: Also known as Psychogenic Non-Epileptic Seizures (PNES) or **Dissociative Seizures**, these episodes resemble epileptic seizures but are not caused by abnormal cortical electrical discharges. Key features suggesting NES include gradual onset/offset, prolonged duration, asynchronous movements, or memory recall for the event. Video-EEG monitoring is the gold standard for differentiation.

Speech and Swallowing Symptoms: This category includes functional dysphonia (whispering or strained voice, often with normal cough), functional dysarthria, or functional dysphagia (difficulty swallowing).

Cognitive Symptoms: Though not primary criteria for motor/sensory FND, complaints of "brain fog," memory difficulties, and concentration problems are common comorbidities.

4. Etiology: The Dissociative and Biopsychosocial Framework

FND is best understood through a **biopsychosocial framework**, where predisposing vulnerabilities, precipitating triggers, and perpetuating factors dynamically interact to disrupt brain function related to sensorimotor control and self-agency.

Psychological and Dissociative Factors: A strong association exists between FND and psychosocial stressors, particularly a history of trauma (especially childhood abuse or neglect) and high levels of comorbidity with Post-Traumatic Stress Disorder (PTSD) and dissociative disorders. The **Dissociative Neurological Symptom Disorder** terminology emphasizes the hypothesis that dissociative mechanisms might disrupt the integrated perception of one's body and the sense of agency (the feeling of being in control of one's actions), leading to the physical manifestation of the symptom.

Neurobiological Factors: Neuroimaging studies confirm that FND involves alterations in brain **function** and **connectivity**, not structural damage. Key findings indicate altered interactions between brain regions governing emotion and motor control. For instance, increased activity in limbic areas (like the amygdala, involved in threat detection) may inappropriately influence or inhibit motor planning areas (like the supplementary motor area), suggesting that emotional circuits interfere with voluntary control. Research also points to impaired sense of agency and abnormalities in attentional networks.

Cognitive Factors: Maladaptive illness beliefs, heightened self-focused attention, and catastrophic interpretations of bodily sensations perpetuate the condition. The predictive coding model suggests that strong, top-down predictions about a bodily state (e.g., "my leg is weak"), potentially driven by anxiety or past experience, may override actual sensory inputs, resulting in the functional symptom.

5. Diagnostic Criteria and Positive Signs

Diagnosis requires recognition of **positive clinical signs** of inconsistency, moving beyond simply excluding other neurological diseases. The DSM-5 criteria (Code 300.11) are:

Criterion A: One or more symptoms of altered voluntary motor or sensory function.

Criterion B: Clinical findings provide evidence of **incompatibility** between the symptom and recognized neurological or medical conditions.

Criterion C: The symptom or deficit is not better explained by another medical or mental disorder.

Criterion D: The symptom or deficit causes clinically significant distress or impairment.

A comprehensive neurological examination specifically seeks positive signs of inconsistency, which are highly characteristic of FND:

Hoover's Sign: Demonstrating intact involuntary activation of the pyramidal tract; voluntary hip extension weakness improves significantly when the patient is asked to flex the contralateral hip against resistance.

Tremor Entrainment: The frequency of a functional tremor changes to match or become phase-locked with voluntary rhythmic movements performed by the unaffected limb.

Give-Way Weakness: Sudden, non-physiological collapse of muscle force during strength testing.

Inconsistent Sensory Findings: Sensory loss with clear midline boundaries or non-anatomical "glove" patterns.

Characteristic Features of Non-Epileptic Seizures: Asynchronous limb movements, resistance to eye opening during the attack, or prolonged duration (>2-3 minutes).

6. Treatment and Multidisciplinary Management

Optimal management requires a collaborative, multidisciplinary approach involving neurologists, psychiatrists, psychologists, and physical therapists. The initial step is **confident and compassionate communication** of the positive diagnosis, validating the reality of the symptoms while explaining the functional (software) nature of the problem.

Specialized Physical Rehabilitation: For motor symptoms, specialized physical therapy (PT) or occupational therapy (OT) is crucial. This differs from standard rehabilitation by focusing on shifting attention away from the affected body part, demonstrating normal capacity, and graded retraining of movement patterns using behavioral techniques.

Psychotherapy: Psychotherapy is a cornerstone, particularly targeting psychological factors, emotional processing difficulties, and trauma. **Cognitive Behavioral Therapy (CBT)**, adapted for FND, focuses on modifying maladaptive illness beliefs, reducing avoidance behaviors, and managing stress. For patients with significant trauma, Eye Movement Desensitization and Reprocessing (EMDR) or psychodynamic approaches may be used to process underlying conflicts and traumatic memories linked to symptom onset.

Pharmacotherapy: Medications are generally used only to treat comorbid psychiatric conditions, such as depression or severe anxiety, which significantly impact FND severity and prognosis.

7. Stigma and Prognosis

Individuals with FND frequently face significant stigma, stemming from historical misconceptions that the condition is "imagined" or feigned. This stigma, often manifested by disbelief or dismissal from healthcare professionals and the public, leads to diagnostic delays, psychological distress, and reluctance to engage in essential treatment. Addressing stigma through education and using validating language (emphasizing that FND is a legitimate disorder of brain function) is critical for improving care.

The prognosis is highly variable. Generally, a **shorter duration of symptoms** before diagnosis and subsequent treatment initiation is associated with a better outcome. Patients who accept the diagnosis and actively participate in multidisciplinary treatment show the highest rates of improvement. While a significant proportion (approximately 40-60% in specialized programs) achieve substantial improvement or recovery in the short term, long-term outcomes are mixed, with many experiencing persistent residual symptoms or relapses, especially if severe psychiatric comorbidities (like PTSD or chronic pain) are present. The goal of treatment is often focused on improving overall functioning and quality of life, even if complete symptom remission is not achieved.

Further Reading

American Psychiatric Association. (2013). Diagnostic and Statistical Manual of Mental Disorders (5th ed.).

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FND Hope International. (Patient advocacy and support source)

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