Functional neurological symptom disorder (conversion disorder) in adults: Epidemiology, pathogenesis, and prognosis

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INTRODUCTION

Functional neurological symptom disorder (conversion disorder) is characterized by neurologic symptoms such as abnormal movements, nonepileptic seizures, or weakness, which involve abnormal nervous system functioning rather than structural disease [1]. In addition, clinical findings on examination provide evidence of incompatibility between the symptoms and recognized neurologic disease. Nevertheless, functional neurological symptom disorder causes distress and/or functional impairment, and is common in clinical settings and often has a poor prognosis [2-5].

This topic reviews the epidemiology, pathogenesis, and prognosis of functional neurological symptom disorder. The terminology, diagnosis, differential diagnosis, clinical features, assessment, and treatment are discussed separately, as are specific subtypes of functional neurological symptom disorder (psychogenic nonepileptic seizures and functional movement disorders):

- (See "Functional neurological symptom disorder (conversion disorder) in adults: Terminology, diagnosis, and differential diagnosis".)
- (See "Functional neurological symptom disorder (conversion disorder) in adults: Clinical features, assessment, and comorbidity".)

- (See "Functional neurological symptom disorder (conversion disorder) in adults: Treatment".)
- (See "Psychogenic nonepileptic seizures: Etiology, clinical features, and diagnosis".)
- (See "Functional movement disorders".)

EPIDEMIOLOGY

The incidence and prevalence of functional neurological symptom disorder (conversion disorder) has been more widely studied in clinical settings than in the general population.

General population — Establishing case rates in population studies is difficult because the diagnosis is usually made in secondary care. It is thus likely that published rates underestimate the true numbers.

A review found that the estimated incidence of functional neurological symptom disorder across disparate geographical settings was 4 to 12 per 100,000 per year (0.004 to 0.012 percent), and the community prevalence was 50 per 100,000 per year (0.05 percent) [6].

Neurologic settings — The prevalence of functional neurologic symptoms in neurologic settings ranges from 9 to 16 percent, making it one of the most common disorders:

- A prospective, 15-month study of 3781 new neurology outpatients found that functional neurological symptom disorder was present in 16 percent, and was the second most common neurologic presentation [2].
- A prospective, 17-month study of 884 new neurology outpatients found that functional neurological symptom disorder was present in 15 percent, and was the third most common neurologic presentation [7].
- A retrospective, three-year study of hospitalizations on a neurology service (n = 1949) found that functional neurological symptom disorder represented 9 percent of all admissions [8].

Emergency medicine settings — A retrospective, one-year study of 213 emergency department presentations, with seizures more than five minutes long and/or more than two seizures in 24 hours (status epilepticus), found that 114 (54 percent) received a final diagnosis of functional seizures [9]. Among the 114 presentations with functional seizures, only 15 percent were initially recognized correctly.

Sociodemographic correlates

- **Age** – In a meta-analysis of patient-level data from nearly 5000 cases of functional neurological symptom disorder with abnormal movement, the mean age at onset was approximately 40 years [10]. Although functional neurological symptom disorder has been reported in patients of all ages, it is rarely diagnosed before age 10 years [11,12].

- **Sex** – Multiple studies indicate that functional neurological symptom disorder is more likely to occur in females than males [10,13-15]. As an example, a meta-analysis of nearly 5000 patients with the disorder (abnormal movement subtype) found that 73 percent were female [10]. However, in some subgroups such as older patients and patients with certain types of functional movement disorders (eg, jerky movements), males are more commonly affected.

Health care costs — A United States study of health care costs in 30 million adults in 2017 found that functional neurological symptom disorders were associated with annual costs of $1066 million for inpatient care, which were comparable to costs associated with anterior horn cell disease [15].

ETIOLOGY AND PATHOGENESIS

The etiology and pathogenesis of functional neurological symptom disorder (conversion disorder) is not fully understood, despite an increase in the number of reproducible research findings. Many biological, psychological, and social factors have been found to be more common in patients with functional neurological symptom disorder than patients with comparable symptoms due to recognized disease [16-18]. While each of these factors may play a role in predisposing patients to functional neurological symptom disorder, precipitating its onset, and/or perpetuating it once it has arisen, caution is required before assuming that these associations are causal (table 1).

Predisposing, precipitating, and perpetuating factors — Psychological factors such as stressful life events, interpersonal conflicts, and adverse childhood experience have traditionally been considered the “cause” of functional neurological symptom disorder. It was thought that these factors were converted into neurologic symptoms, hence the older name “conversion disorder.” However, patients with the disorder do not always report psychological factors, nor are they specific to functional neurological symptom disorder. In addition, “stress” is ubiquitous and adverse childhood experiences are common in the general population. Although psychological factors are often associated with functional neurological symptom disorder, this does not prove that the factors cause the disorder (correlation is not causation) [19]:

- A meta-analysis of 34 retrospective studies examined the history of stressful life events and maltreatment in patients with functional neurological symptom disorder (n >1400) and in healthy or patient controls (n >2200) [19]. Stressful life events were more common in patients with functional neurological symptom disorder than controls (odds ratio 2.8, 95% CI 1.4-6.0). In addition, maltreatment had occurred in more patients than controls:

  - Emotional neglect (49 versus 20 percent)
  - Sexual abuse (24 versus 10 percent)
  - Physical abuse (30 versus 12 percent)

  Among the 34 studies, 13 found that some patients with functional neurological symptom disorder, ranging from 14 to 70 percent, had no history of either stressful life events or any type of maltreatment.

- In a subsequent retrospective study of 322 patients with functional neurological symptom disorder (weakness or abnormal movements) and 644 psychiatric control patients, the proportion of patients with a history of childhood sexual abuse was similar in both groups (20 and 22 percent), as was the proportion with a history of physical abuse (23 and 22 percent) [13].

Pre-existing psychiatric, medical and neurologic conditions may also predispose patients to developing functional neurological symptom disorder [16]. Compared with controls with recognizable diseases, patients with functional neurological symptom disorder are more likely to have a history of pre-existing psychiatric disorders (eg, depressive, anxiety, and personality disorders), other somatic conditions (eg, pain, fatigue, and cognitive impairment) [13,20,21], or other types of preexisting functional somatic disorders, such as irritable bowel syndrome [22]. In addition, functional neurological symptom disorder is often preceded by neurologic illnesses (eg, migraine, epilepsy, peripheral nerve pathology, or stroke) that appear to precede, but do not explain the functional neurologic symptoms [23,24]. Physical injury may also precede functional neurologic symptoms [25,26]. Further, functional neurological symptom disorder is associated with lower socioeconomic status [15].

It is not clear if symptom modeling (exposure to similar symptoms in others) is a pathogenic factor. Some studies of functional neurological symptom disorder indicate it is associated with symptom modeling [27-29], but others do not [30-32]. As an example, a study of 132 patients with functional movement disorders and 148 healthy controls found that the proportion of health care workers in each group was comparable (25 and 20 percent) [33].

Other perpetuating factors — Functional neurological symptom disorder may be perpetuated if the patient is unable to understand or agree with the diagnosis. The concept that the disorder

is at least potentially reversible, and that rehabilitation may help is an essential basis for further treatment.

Clinicians may perpetuate the condition in multiple ways, including:

- Failing to give a clear explanation and positive diagnosis of the symptoms [34].
- Excessively investigating symptoms [35].
- Allowing patients to visit different specialists without anyone taking primary responsibility [36].
- Prescribing inappropriate drugs such as opiates for pain, and providing appliances such as crutches and wheelchairs at a point when these might interfere with rehabilitation [37].
- Performing unnecessary operations.
- Misattributing symptoms to recognizable disease or to irrelevant radiologic or laboratory findings (eg, age-related degenerative changes in the vertebrae) [38].

In addition, functional neurologic symptoms may be perpetuated by progressive physical deconditioning, untreated comorbid psychiatric disorders, disability-related financial benefits, and ongoing litigation [39,40]. Another potential perpetuating factor is ongoing life stress (eg, work or family) that was relieved by onset of the disorder and would probably recur if the patient was to recover. However the significance of this so-called “secondary gain” was overstated in the past [41].

Etiologic hypotheses — Different hypothetical models attempt to explain how conversion symptoms develop; the models are not mutually exclusive.

Cognitive-behavioral models — Multiple overlapping cognitive-behavioral models endeavor to explain the etiology and pathogenesis of functional neurological symptom disorder.

One cognitive-behavioral model is based upon findings that processing of perception and behavior mostly occurs outside of awareness, and proposes that functional neurologic symptoms may result from cognitive, emotional, and behavioral influences at these lower levels of processing [42]. It is suggested that patients with the disorder initially encounter (in themselves or others) a stimulus for a particular symptom, such as mild weakness from migraine. This involuntarily generates a mental representation or memory of paralysis. Excessive anxiety about becoming paralyzed, and/or hypervigilance in looking for evidence (eg,

weakness) of paralysis in oneself, may activate the mental representation to the point that it overrides sensory input and distorts awareness and behavior [43].

Another cognitive-behavioral model proposes that functional neurological symptom disorder involves dissociative experiences [44]. Dissociation is subjectively perceived as disconnection from oneself (depersonalization) or the environment (derealization). During dissociation, awareness and integration of thoughts, feelings, memories, and identity is altered, as is integration of somatic experiences and functions, and patients lose functioning of motor control or sensory awareness [45]. Dissociation may occur as a consequence of fatigue, panic attacks [46], physical injuries [25,26,47], recognizable diseases [23,48], pain [49], general anesthesia [46,50], or drug side effects [26]. According to this model, the symptom of paralysis or abnormal movement arises during the dissociated state when the patient is "depersonalized" and generally loses feelings of ownership over body movements. Attention focused upon this experience, combined with fear of what the symptom might represent (eg, stroke), may intensify and cause more "localized" depersonalization in one body part, thus prolonging the symptom. In functional seizures, prodromal symptoms of autonomic arousal may become so unbearable that patients respond by losing awareness in an apparent blackout [51-54]. Some studies of patients with functional neurological symptom disorder suggest that their explicit awareness of emotional symptoms, such as anxiety, is low, which may help explain why they are more likely to report physical but not emotional content of these events [55,56], with subsequent nonepileptic seizures occurring as a conditioned response to unpleasant thoughts, feelings, and situations, even in the absence of a prodrome. In functional limb weakness, weakness and numbness may occur as a maladaptive response in an attempt to reduce pain through dissociation [49]; this may explain the clinical overlap seen with complex regional pain syndrome [57].

Neurobiologic models — Functional neurological symptom disorder may involve abnormalities in neural networks of grey matter brain regions rather than a disturbance in one specific structure [44]. These networks are thought to include frontal (orbitofrontal and anterior cingulate cortex) and subcortical (limbic) structures that may be activated by stress and other factors, and in turn provide input to inhibitory basal ganglia-thalamocortical circuits that reduce conscious motor or sensory processing [58,59]. Functional magnetic resonance imaging (MRI) comparing patients with functional neurological symptom disorder (weakness subtype) with healthy controls during recall of traumatic events have found differences in regional brain activity (eg, prefrontal cortex and hippocampus) [60], which in other studies correlated with symptom severity [61].

In addition, there may be aberrant activity in brain networks that include the inferior parietal lobe/temporoparietal junction, which are involved in self-agency, the sense that one causes one's own actions [58,62,63]. One hypothesis is that overly sensitive amygdala responses to fear (ie, abnormal responses to stimuli, including those that are objectively neutral) lead to changes in networks mediating sensory and motor function. In the setting of abnormal self-directed attention (eg, depersonalization occurring at the time of injury or panic), these changes produce sensations or movements that are associated with an abnormal sense of self control and are interpreted as involuntary symptoms of a disease [43,44].

Neuroimaging of patients with functional neurological symptom disorder also suggests possible structural changes in the brain. As an example, structural MRI studies that compared patients with controls found evidence of altered brain structure in patients (eg, increased thalamic volume and decreased sensorimotor cortical thickness) [64,65]. However, it is not known if these differences indicate causal factors, confounding comorbidities, or sequelae of the disorder.

Other studies have examined endocrine and inflammatory profiles in patients with functional neurological symptom disorder. Although multiple studies of basal and task related cortisol levels in patients found few differences from healthy controls [66], the studies were too small to draw clear conclusions.

The concept of "active inference" suggests that the brain operates on a moment-to-moment basis by making predictions in advance rather than just sensing and responding. It has been proposed that functional neurological symptom disorder arises when patients generate incorrect inferences or predictions about motor or sensory function. As an example, a patient may have such a strong internal prediction/inference/belief that the leg is "absent" that this overrides normal sensory input suggesting otherwise. This mechanism is similar to one proposed for phantom limb syndrome, in which the brain has such a strong prediction that a leg is "present" that it overrides sensory input which should correct that prediction.

Psychodynamic models — The classic psychodynamic hypothesis, which gave rise to the name conversion disorder, is that patients experience an unconscious conflict that is converted into a somatic symptom [67-69]. The symptom serves as a defense against anxiety and distress, which are mitigated by the conflict remaining unconscious [45]. In this model, the conversion symptom symbolizes the conflict and is hypothesized to help the patient avoid an overwhelming situation [70]. The hypothesis is difficult to test scientifically, has been hard to apply clinically, and has limited empirical support [45].

Subsequent psychodynamic hypotheses emphasize abnormal interpersonal relationships that develop in the context of problematic early relationships (eg, poor parenting) or traumatic events [42]. In these models, a new conflict or traumatic event leads to recurrence of previous patterns of abnormal behavior and the development of physical symptoms. The physical symptoms are regarded as a coping response, secondary to emotional dysregulation. As an example, somatic symptoms that lead to contact with clinicians may enable patients to meet dependency needs established earlier in life.

Evidence supporting the psychodynamic model includes a retrospective study that assessed patients with functional neurological symptom disorder (n = 43) and patients with depression (n = 28) for stressors prior to symptom onset, and asked healthy controls (n = 28) about stressors in the prior two years [41]. The stressors/life events were rated for the extent to which they could be described as "escape" events," that is, events which enabled the person to escape from an aversive situation or ameliorate the stressor (eg, providing an "escape" from going to work and facing an abusive boss). The frequency of these "escape" life events in the month before symptom onset was greater for patients with functional neurological symptom disorder, than for patients with depression and healthy controls (53 versus 14 and 0 percent).

Although evidence for psychodynamic models is limited, therapy based on psychodynamic and interpersonal principles may still be useful.

PROGNOSIS

Prognosis for functional neurological symptom disorder (conversion disorder) is frequently poor. Reviews of observational studies suggest that symptoms persist or worsen in approximately 40 to 66 percent of patients [40]. As an example, a 14-year prospective study of 107 patients with functional limb weakness found that symptoms remitted in 20 percent, improved in 31 percent, persisted in 23 percent, and worsened in 26 percent [71]. Most follow-up studies have also found persistent physical symptoms (eg, fatigue and pain) and impaired quality of life [40].

In addition, prognosis may vary across subtypes of functional neurological symptom disorder:

- Sensory symptoms [72,73] may have a better prognosis than weakness/paralysis [73], dystonia [74], and tremor [4].
- The prognosis of functional seizures varies but is often poor. In a systematic review of 25 observational studies, seizure remission occurred in 40 percent or fewer patients [40].

Baseline predictors of outcome in patients with functional neurological symptom disorder vary among studies, and much of what is known comes from studies that included not only patients with functional neurological symptom disorder, but other somatic symptom and related disorders as well. In addition, these baseline factors are not consistently associated with outcome across multiple studies and do not enable clinicians to accurately predict outcome for a specific patient.

Across different studies with follow-up ranging from 8 months to 14 years, prognostic factors included the following (table 2) [4,23,39,40,44,71,73,75-81]:

- Factors associated with a positive outcome:
  - Onset in childhood or adolescence
  - Early diagnosis
  - Comorbid anxiety or depression
  - Subsequent change in marital status
  - Good therapeutic alliance with the clinician
  - Good response to initial treatment
- Factors associated with a poor outcome:
  - Multiple physical symptoms
  - Longer duration of symptoms (eg, 15 months)
  - Poor physical functioning
  - Comorbid personality disorder
  - Beliefs that symptoms are irreversible and caused by a disease with a known pathologic basis – Beliefs that symptoms are not potentially reversible may be a stronger predictor of poor prognosis than the number of conversion symptoms or severity of disability
  - Illness-related financial benefits

SUMMARY

- **Epidemiology** – The estimated prevalence of functional neurological symptom disorder (conversion disorder) in the general population ranges from 0.004 to 0.2 percent, and in clinical settings, from 2 to 6 percent. Sociodemographic correlates include younger age and female sex. Onset of functional neurological symptom disorder may occur at any age but is rare before age 10 years. (See 'Epidemiology' above.)

- **Etiology and pathogenesis** – The etiology and pathogenesis of functional neurological symptom disorder are not fully understood. However, many factors have been identified that may predispose patients to the disorder, may precipitate its onset, and/or perpetuate it (table 1). (See 'Etiology and pathogenesis' above.)

  - **Predisposing factors** – Pre-existing psychiatric medical and neurologic conditions may predispose patients to developing functional neurological symptom disorder. It may also be triggered by physical injury and neurologic illnesses. (See 'Predisposing, precipitating, and perpetuating factors' above.)

  - **Precipitating factors** – Psychological factors or life events such as trauma, interpersonal conflicts, and recent or old stressors may be associated with onset of the disorder. However, psychological factors are not always reported and they are not specific to functional neurological symptom disorder. In addition, many patients may not have identifiable psychological factors. (See 'Predisposing, precipitating, and perpetuating factors' above.)

  - **Perpetuating factors** – Functional neurologic symptoms may be inadvertently perpetuated by clinicians in multiple ways, such as failing to give a clear explanation and positive diagnosis, excessively investigating symptoms, misattributing symptoms to a disease or incidental radiologic or laboratory findings, prescribing inappropriate drugs, and performing unnecessary operations. (See 'Other perpetuating factors' above.)

  - **Etiologic hypotheses** – Hypothetical etiologic models that are not mutually exclusive include the following:

    - **Cognitive-behavioral models** – Cognitive-behavioral models are based upon evidence that processing of perception and behavior mostly occurs outside of awareness, selective attentional bias amplifies minor physiological stimuli and asymmetries, and that functional symptoms involve dissociative experiences and maladaptive thoughts. (See 'Cognitive-behavioral models' above.)

- **Neurobiologic models** – Functional neurological symptom disorder may involve abnormalities in neural networks of grey matter brain regions. Networks involved in attention and the brain's sense of self-agency, as well as interactions between limbic and motor networks, have been implicated. Another model suggests the disorder arises when patients generate incorrect inferences or predictions about motor or sensory function. (See 'Neurobiologic models' above.)

- **Psychodynamic models** – The classic psychodynamic model hypothesizes that patients experience an unconscious conflict that is converted into a somatic symptom. The symptom is a defense against anxiety and distress, which are reduced by the conflict remaining unconscious. Subsequent models have emphasized abnormal interpersonal relationships that develop in the context of problematic early relationships or traumatic events. (See 'Psychodynamic models' above.)

- **Prognosis** – The prognosis for functional neurological symptom disorder is generally poor. Factors associated with positive and poor outcomes are listed in the table (table 2). (See 'Prognosis' above.)

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