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## Functional Gait Disorders: Clinical presentations, Phenotypes and Implications for treatment

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### ABSTRACT

**Background:** Functional Gait Disorders (FGD) are a common presentation of motor-Functional Neurological Disorders (motor-FND) that affect walking ability.

**Aim:** To provide a narrative review of the current literature on FGD.

**Methods:** A narrative overview of published literature was undertaken, based on a systematic search of relevant databases, authoritative texts and citation tracking.

**Results:** FGD is multidimensional and disabling, with numerous phenotypes described in the literature, including 'knee buckling,' 'astasia-abasia' and 'excessive slowness.' Motor symptoms such as weakness or tremor, and non-motor symptoms, such as pain and fatigue may contribute to the disability and distress in FGD. Phenotypic features and clinical signs are seen in FGD that demonstrate inconsistency and incongruity with structural disease. A limited number of treatment studies have specifically focussed on FGD, however, reporting of outcomes from motor-FND cohorts has demonstrated short and long-term improvements in walking ability through multidisciplinary rehabilitation.

**Conclusions:** The relative contribution of motor and non-motor symptoms in FGD remains unknown, but it is likely that non-motor symptoms increase the illness burden and should be considered during assessment and treatment. Recommended treatment for FGD involves multidisciplinary rehabilitation, but optimum treatment elements are yet to be determined.

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functional neurological disorders; functional gait disorders; clinical presentations; phenotypes; treatment

### Introduction

Functional neurological disorders (FND) are characterized by sensory, motor and cognitive symptoms that are unexplained by neuropathology (1). People with motor-FND present with abnormalities of motor function, such as weakness or tremor, which when impacting gait can be specifically described as functional gait disorders (FGD) (2). Functional gait disorders are common in outpatient settings, where they have been reported in up to 40% of people with motor-FND (3,4). Presentations of FGD have been long described in medical history, including Charcot's works from the 19<sup>th</sup> century (5), and the descriptions of shell shock from the first world war (6). There has been a resurgence of interest in the field over the past two decades with particular advancements in both clinical and research domains across diagnosis, etiology and treatment (7,8). Descriptions of FGD in the literature have evolved over time with reported phenotypes including 'knee buckling,' 'astasia-abasia' and 'excessive slowness' amongst others. Functional gait disorders have been reported to occur in isolation, or as a combined presentation of impaired gait alongside other symptoms, such as functional tremor or dystonia (9). They may also co-occur with other neurological conditions such as brain injury or multiple sclerosis (10,11). Presentations of FGD are understood to be part of the wider spectrum of FND

symptoms, that also includes associated non-motor symptoms such as pain and fatigue.

This paper will provide an overview of the existing literature on FGD, including etiology, clinical presentation, phenotypes, diagnosis and treatment.

### Methodology

A narrative review of published literature on FGD was undertaken. Papers included in this review were identified by searches of relevant databases (PubMed, CINAHL, Science Direct, Cochrane Library, Web of Science and Elsevier). In addition, a review of authoritative texts, reference checking and citation tracking took place. The following search terms were used: "functional gait disorder"; "functional neurological disorder"; "functional movement disorder"; "functional motor disorder"; "psychogenic motor disorder"; "psychogenic movement disorder"; "conversion disorder"; "psychogenic gait"; and "hysterical gait." From the resultant articles, findings relating to FGD were synthesized and presented in this review.

### Etiology and mechanism

The etiology of motor-FND is usually understood using a biopsychosocial model, where individuals have different

predisposing and precipitating factors for developing symptoms, which are maintained by perpetuating factors (12). Each of these etiological factors may be considered in terms of biological, psychological or social domains. Examples of predisposing factors could include biological vulnerabilities in the nervous system, emotional disturbance or adverse life events (12). In this context, the presence of neurological disease or injury can be considered a predisposing risk factor for developing FND (13). Precipitating events may include injury, illness, dissociation, trauma, or other physical or psychological events (13–16). For example, functional symptoms can occur following a mild traumatic brain injury (17). Perpetuating factors may include learnt habitual movements, illness beliefs and social factors (12). This model allows for the integration of both physical and psychological factors when accounting for symptoms, without emphasis on psychological factors, which were previously considered a requirement for diagnosis (18).

The mechanism for symptoms of FND has been suggested to follow a hierarchical Bayesian model of altered higher-level feed-forward control with impaired intentional movement and sensory processing. During normal movement, our nervous system predicts a certain response from the intended action, and the prediction error which arises can be explained as the difference between what you expect to sense and what you sense (15). The Bayesian model proposes that the aim of normal movement is to minimize the prediction errors at each level of control (19). Predictions of the sensory consequences of intended movement occur at a high level in the cortical hierarchy and are transmitted down the descending motor pathways, producing movements that follow these prediction errors (15). Theoretically, in motor-FND, an abnormal

*prior* expectation occurs in an intermediate motor area, which is given excessive attention and precision, that leads to a prediction error, that is corrected through symptom production (e.g., added movements such as tremor or dystonia) (15).

The altered prior expectation can follow certain beliefs and events. For example, beliefs about illness can occur following a physical injury (20), health scares in the media (21), or concern over inheritable family illness (22). Motor-FND may occur when this belief is combined with a precipitating event, such as a painful injury to a limb resulting in functional weakness (23).

These models provide a theoretical basis for the etiology and mechanisms of FGD, but whether there are additional factors contributing to symptoms unique to FGD remains unknown.

### Clinical presentation

Functional gait disorders are diverse and may vary over time in their clinical presentation. People with FGD usually present with motor and non-motor symptoms and most have other comorbid conditions, making it a complex and multidimensional disorder.

### Motor symptoms

Common functional motor symptoms include weakness, tremor, dystonia and myoclonus (Figure 1), that may be present in people with FGD (24). It is clear that these motor symptoms will contribute to the disruption of normal gait kinetics and kinematics. Motor symptoms have been found to persist in the long term, as shown in a 14-year-long case-control study with

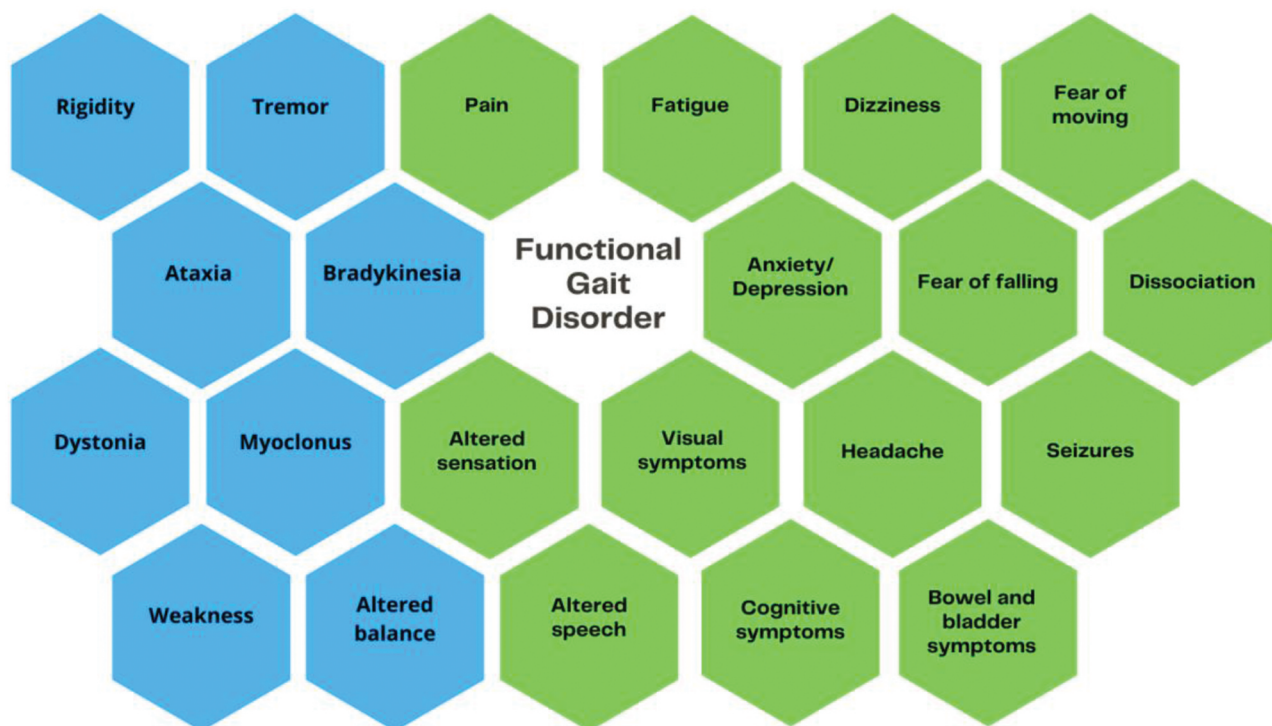


Figure 1. Reported functional motor symptoms (blue) and non-motor symptoms (green) that occur in motor-FND that may contribute to presentations of FGD.

76 participants with functional weakness, where complete resolution occurred in 20% of their participants, improvement in 31% and worsened or remained stable in 49% (25).

### Non-motor symptoms

Non-motor symptoms are common in people with motor-FND, including those with FGD 26 (Figure 1). Tinazzi et al. (27) investigated the clinical correlates of motor-FND in a cohort of 410 patients. The most common non-motor symptoms were anxiety (52.1%), fatigue (45.1%), and pain (41.9%). Chronic pain was also shown to be highly prevalent in patients attending specialist FND clinics, affecting 56% and 79% in a Canadian and United Kingdom clinic-cohort respectively (28). Other symptoms found by Tinazzi et al. 27 included somatosensory symptoms (25.3%), functional visual symptoms (11.4%) and cognitive symptoms (10.9%). Each of these symptoms may have a direct impact on walking ability.

Two recent studies have explored the impact of non-motor symptoms in people with motor-FND. One study with 61 participants reported that health-related quality of life scores negatively correlated with depression, anxiety and pain, with no correlation found between health-related quality of life and motor symptom severity (26). Similarly, in 181 participants with motor-FND, Gelauff et al. (24) reported that quality of life was negatively associated with fatigue and depression but not self-rated motor symptom severity. These findings indicate a multifaceted interplay of FND symptoms and suggest that the non-motor symptoms may have a greater impact on quality of life than motor symptoms.

Other non-motor features that may be associated with FGD include fear of falling, kinesiophobia and dizziness. Fear of falling is a common feature in the community-dwelling elderly, especially following falls (29). The term “cautious gait” describes a response to perceived disequilibrium or a postural threat (30) and is associated with an increased risk of falling (31). Fear of falling may be an important consideration during assessment and treatment in FGD. In persistent pain cohorts, kinesiophobia is defined as fear of pain associated with movement, leading to avoidant disuse and central sensitization of pain (32). Given the high incidence of persistent pain in people with FND, it is possible that kinesiophobia may occur in some cases of FGD. Another non-motor symptom that can impact gait is functional dizziness (33), but is often not considered in classical descriptions of FGD. Functional dizziness (also known as persistent postural-perceptual dizziness) commonly follows vestibular disorders, such as benign paroxysmal positional vertigo or vestibular neuritis (33,34).

Further research into the prevalence, severity and impact of motor and non-motor symptoms in FGD may help to inform the etiology and mechanisms to support targeted assessment and individualized treatment.

### Functional gait disorder phenotypes and classification

Over the years many subtypes of FGD have been described. The term “astasia-abasia,” first used in the nineteenth century, is an early description of a functional gait disorders, with

astasia relating to the inability to stand upright and independently, and abasia denoting the inability to walk in a coordinated manner (35). Charcot described clinical observations of “dragging gait” in patients with functional paralysis where the affected leg was dragged behind with the forefoot in contact with the ground (36,37). This phenotype is still reported today (6), which suggests the stability of this phenotype over time.

A range of FGD phenotypes have been described over the years and there have been attempts to develop classification systems with various objectives, such as reporting characteristics, supporting diagnosis or to provide phenomenological classifications.

Tinazzi et al. (38) investigated 109 participants with FGD and reported “slow gait” (n = 43, 39.4%), “astasia-abasia” (n = 26, 23.8%), and “knee buckling” (n = 24, 22%) as the most common phenotypes. Lempert et al. (39) classified the gait disorder in 37 patients and found that 97% of their sample could be categorized into one of six groups (momentary fluctuation of gait and stance, excessive slowness, psychogenic rhombeg, uneconomic postures, walking on ice, and sudden buckling of knees without falls). Jordbru et al. (40) further developed this work by testing the inter-rater reliability of these phenotypes in a sample of 30 patients with FGD. Good inter-rater reliability and agreement was found using the three most common phenotypes in their sample (limping/dragging of one leg, walking on ice/slow gait, and truncal ataxia/imbalance).

Nonnekes et al. (41) developed a sign-based approach to support the diagnosis of FGD using clinical features that demonstrate inconsistencies and incongruencies with neurological disease. The authors suggest that seven broad categories capture the diverse clinical spectrum of FGD (ataxic gait, spastic gait, weak gait, antalgic gait, parkinsonian gait, hemiparetic gait, and dystonic gait). Table 1 includes a summary of the reported phenotypes of FGD.

Functional gait disorders are difficult to categorize because of their complexity, variability and heterogeneity, as highlighted by the broad range of presentations reported in Table 1. However, difficulty with classification is not unique to FGD. In dystonia, phenotypic categorization has proven to be challenging due to a large degree of variability among presentations, resulting in different methods of classification based on etiology, age at onset or body distribution (44). Classification of FGD into distinct subtypes may have its limitations, as features can be heterogenous, however, gait analysis and validation of phenotypes in a large cohort may inform a system that supports treatment planning.

### Terminology and diagnosis

Terminology used to describe FND has evolved over the editions of the International Classification of Diseases (ICD) and the Diagnostic and Statistical Manual of Mental Disorders (DSM). Having previously been known as ‘hysteria,’ it came to be referred as ‘Conversion disorder’ (DSM-4) or ‘Dissociative (conversion) disorder’ (ICD-10), which evolved to ‘Functional neurological symptom disorder (Conversion disorder)’ in DSM-5TR, and ‘Dissociative neurological



**Table 1.** A summary of the reported phenotypes of functional gait disorders.

Phenotypes	Description
<i>Psychogenic Rhombberg</i>	Patients may fall toward or away from the tester Large amplitude body sway, building up after a silent latency of a few seconds Improvement of postural balance when the patients are distracted (39)
<i>Uneconomic postures</i>	High muscle energy demands in order to maintain difficult postures with an eccentric displacement of the center of gravity or flexed knees and hips (39)
<i>Walking on ice</i>	Cautious, wide-based steps with decreased stride length, rigidity in knees and ankles, shuffling of the feet (39)
<i>Sudden buckling of knees without falling</i>	Intermittent sudden buckling of the knees associated with bracing or activating anti-gravity muscles before touching the ground (39)
<i>Excessive slowness/cautious</i>	Slow motion gait. Slowed speed of every step and may have simultaneous innervation of antagonist muscles and abduction of the arms (39,42)
<i>Dragging of one foot</i>	Monoplegic gait with normal swing phase on the unaffected side. The forefoot of the dragging leg is often kept in contact with the floor in all of the phases of gait cycle. The leg and/or the foot is often externally or medially rotated with clear compensatory effort to propel the leg forwards during swing phase, with an extended hip and/or knee (39,40,43)
<i>Truncal imbalance</i>	Instability of the trunk in stance and gait, with side-to-side swaying and flailing of the arms. Stepping often occurs to correct balance (40,41)
<i>Functional Dystonic gait</i>	Abnormal posturing of the leg or trunk during gait cycle. Fixed plantar flexion and inversion posturing of the foot is often seen. Inconsistencies may include disappearance of abnormal posturing in different positions (41)
<i>Functional Tremulous gait</i>	Gait characterized by tremor in stance or swing phases affecting lower limbs or trunk. Tremor may change in different postures (42,43)
<i>Tightrope walking</i>	Exaggerated truncal sway while maintaining a narrow base, with legs appearing to follow a tightrope, truncal instability with good targeting of nearby walls or furniture (43)
<i>Neurological disease mimics</i>	<i>Camptocormic:</i> Abnormal flexion of the trunk in standing position that worsens during walking (43) <i>Sensory ataxic:</i> Broad-based gait, with feet dropping down clumsily during initial stance. The person tends to look at their feet throughout the gait cycle (43) <i>Choreoballistic:</i> Involuntary flinging of the limbs or trunk, which results in severe, uncontrollable flailing (43) <i>Stiff-man syndrome/robotic:</i> Involuntary activation of axial agonist and antagonistic muscles impacting postural control (43) <i>Ataxic gait:</i> Variability in base of support or inability to walk in a straight line, often with excessive arm movements (41) <i>Spastic (scissoring) gait:</i> Legs cross the midline during swing phase despite no adductor spasticity on testing and normal leg reflexes. Scissoring gait can improve when walking backwards (41,42) <i>Trendelenburg (weak) gait:</i> Inconsistent waddling gait (41) <i>Parkinsonian gait:</i> Bradykinetic gait with inconsistent and distractible freezing, tremor and rigidity (41,42) <i>Hemiparetic gait:</i> Dragging of one leg through the swing phase, variability during gait and no spasticity found on testing. Give-way weakness with positive hoover/abductor sign can be found (41) <i>Footdrop:</i> Dropping of the forefoot at initial swing or mid-swing (43)

Adapted from Lempert et al., 1991 (39), Jordbru et al., 2012 (40), Nonnekes et al., 2020 (41), Fung 2016 (43), Baizabal-Carvallo et al., 2020 (42)

symptom disorder' in ICD-11 (45,46). The term 'functional' has become preferred among neurologists (47,48) and people diagnosed with the condition (49).

The diagnosis of FND is usually made by neurologists, especially when motor symptoms are the dominant presentation (43,50). Psychiatrists may have a role in diagnosis, especially for psychological formulation (51). Where possible, the diagnosis should be made based on the identification of positive clinical signs, such as Hoover's sign for functional weakness, as well as inconsistency of the presentation or incongruency with structural disease (38,52). Examples of inconsistency include a disparity between gait patterns in different environments; variability of symptoms over short periods of time; sudden changes in the frequency or amplitude of a tremor; and a difference between clinical assessment and function, for instance, an inability to access movement during a formal assessment that returns to normal during spontaneous movement (53–55). An example of incongruency is a delayed

onset of motor symptoms following minor injury (41). Information from the subjective history provides supporting evidence for the diagnosis, such as transitory episodes of spontaneous remission (41).

The diagnosis can be difficult to distinguish from other conditions, such as movement disorders, because the phenotypes can be similar. Additionally, FGD can coexist with other neurological disease, such as brain injury (11), multiple sclerosis (10) or parkinsonism (38). It can often be pertinent to make two diagnoses in these cases (56). Eames (11), for example, identified that 54 patients from a cohort of 167 (32.3%) with brain injury developed functional symptoms. Associations were found in those with diffuse forms of brain injury, such as hypoxia, and the author also identified a higher incidence of extrapyramidal disorders in those with functional symptoms. Stone and colleagues (10) found that 11.9% of patients in their cohort, with a confirmed neurological diagnosis, also had symptoms that were 'somewhat' or 'not at all' explained by

Table 2. Multidisciplinary treatment studies in motor-FND (including FGD).

Trial	Study design	Cohort	Symptom duration	Treatment (type/setting)	Outcome measures	Main outcomes
Petrochilos et al 2020 (64)	Prospective cohort study	Motor-FND n = 78	Mean of 6.5 years	MDT program, day therapy 2 days per week for 5 weeks Individual treatment sessions of CBT (× 9), physiotherapy (× 9), OT (× 9), consultant neuropsychiatry sessions (× 3) and a family session.	<ul style="list-style-type: none"> <li>● PHQ15</li> <li>● PHQ9</li> <li>● GAD7</li> <li>● SPIN</li> <li>● RSE</li> <li>● HONOS</li> <li>● WSAS</li> <li>● COPM</li> <li>● EQ-5D-5 L</li> <li>● CGI</li> </ul>	<ul style="list-style-type: none"> <li>● At 6-month review post-program improvements were seen in Somatic symptoms (PHQ15)</li> <li>● Depression (PHQ9)</li> <li>● Anxiety (GAD7)</li> <li>● Health and social functioning (HONOS)</li> <li>● Functionality (COPM), health status (EQ-5D-5 L)</li> <li>● Patient-rated clinical global improvement (CGI)</li> </ul>
Demartini et al 2014 (65)	Prospective cohort study	Motor-FND n = 66	Mean of 4.8 years	4-week specialized inpatient program 5-days per week of therapy input Cognitive behavioral therapy, occupational therapy, physiotherapy and neuropsychiatry	<ul style="list-style-type: none"> <li>● HoNOS</li> <li>● COPM</li> <li>● FQ</li> <li>● PHQ15</li> <li>● CNSQ</li> <li>● IPS</li> <li>● HADS</li> <li>● IPQ-R</li> <li>● CGI</li> </ul>	<ul style="list-style-type: none"> <li>● At discharge 66.2% patients rated their general health as "better" or "much better" on the CGI and 75.0% rated their main symptoms as "better" or "much better" on the IPS</li> <li>● 12-month follow up showed maintained improvements on the CGI</li> </ul>
Jordbru et al 2014 (63)	RCT	Exclusively FGD n = 60	Mean of 9 months	3-week inpatient rehabilitation program including adapted physical activity within a cognitive behavioral framework, reinforcing normal gait	<ul style="list-style-type: none"> <li>● FIM</li> <li>● FMS</li> <li>● SF-12</li> </ul>	<ul style="list-style-type: none"> <li>● The mean difference between treatment and no treatment was 6.9 FMS units (p &lt; 0.001, 95% CI 5.5–8.3)</li> <li>● The mean difference between treatment and no treatment was 8.4 FIM units (p &lt; 0.001, 95% CI 5.2–11.7)</li> <li>● The mean difference in SF-Physical score for treatment vs no treatment was 11.7 units (p &lt; 0.001, 95% CI 7.2–16.1)</li> </ul>
Mccormack et al 2013 (66)	Retrospective study	Motor-FND n = 33	Mean of 48 months	Inpatient treatment in a neuropsychiatry unit Input from neuropsychiatrist, psychologist, physiotherapist and occupational therapist individualized to the patients	<ul style="list-style-type: none"> <li>● Mobility</li> <li>● ADLs</li> <li>● MRS</li> </ul>	<ul style="list-style-type: none"> <li>● Data from admission and discharge was extracted and compared</li> <li>● The proportion of patients progressing from mobilizing in a wheelchair to unaided or with the aid of a stick/crutches increased from 33.3% (n = 11) to 72.7% (n = 24)</li> <li>● The proportion of those who were wheelchair dependent fell from 57.6% (n = 19) to 15.2% (n = 5)</li> <li>● Cases also showed a statistically significant improvement in MRS score from admission (mean 3.64, SD 0.86, range 2–5) to discharge (mean 2.82, SD 0.85, range 2–5); p &lt; 0.001.</li> </ul>
Czarnecki et al 2012 (67)	Historical-cohort-study	Motor-FND n = 60	Median duration of 17 months	Outpatient, 5-day intensive rehabilitation program based on the concept of motor reprogramming following a comprehensive diagnostic neurological evaluation, including psychiatric/psychological assessment.	<ul style="list-style-type: none"> <li>● Patient- and physician-rated improvements (scale 0–4)</li> <li>● Self-rated level of disability</li> </ul>	<ul style="list-style-type: none"> <li>● The end-of-treatment rating was classified as markedly improved or completely normal/in remission ("good outcome") in 73.3% based on physician assessment and 68.8% by patient assessment.</li> <li>● Self-assigned level of disability differed significantly between groups (p = 0.019), with 62.4% of treated patients rating their disability level as "mild" or "none," compared to 43.8% of controls.</li> <li>● Follow up at 25 months indicated that 60.4% participants were markedly improved or almost completely normal/in remission, compared to 21.9% of controls (p &lt; 0.001)</li> </ul>

MDT: Multidisciplinary team; CBT: Cognitive behavioral therapy; PHQ15: Patient health questionnaire 15; PHQ9: Patient health questionnaire 9; GAD7: Generalized Anxiety Disorder Assessment 7; SPIN: Social Phobia Inventory; RSE: Rosenberg Self-Esteem Scale; HONOS: Health of the Nation Outcome Scales; WSAS: Work and social adjustment scale; COPM: Canadian Occupational Performance Measure; CGI: Clinical global impression scale; FQ: Fear Questionnaire; CNSQ: The Common Neurological Symptom Questionnaire; IPS: Improvement in presenting symptom; HADS: Hospital anxiety and depression scale; IPQ-R: The Revised Illness Perception Questionnaire; FIM: Functional independence measure; FMS: Functional mobility scale; SF-12: 12-Item Short Form Survey; ADLs: Activities of daily living; MRS: Modified Rankin Scale

the neurological disease, effectively describing concurrent FND and structural disease. Owing to this concurrence, the differentiation of symptoms can be challenging, especially in settings where clinicians' knowledge of and training in FND is limited. However, literature describing validated positive clinic signs for FND has been reported to support the diagnosis (52,57). Additionally, investigations including neuroimaging may be important to rule out other potential causes for symptoms, alongside a thorough neurological examination.

People with FND may initially voice disbelief in the diagnosis, which may be related to stigma or an expectation that an alternative explanation for their symptoms may appear over time. However, misdiagnosis rates are low when the diagnosis is made in a tertiary setting (58). Helping patients understand their diagnosis is an important first step in treatment as acceptance is associated with improved prognosis (48) and greater benefit from treatment (59). Patients may be more accepting of the diagnosis if it is communicated clearly, including an explanation of how the diagnosis was made based on positive clinical signs (60).

## Treatment

Consensus from the experts in the field recommend multidisciplinary treatment for motor-FND, including FGD, which includes input from physicians, physiotherapists, occupational therapists and psychologists, based on a biopsychosocial framework (61,62). Studies have reported favorable short- and long-term outcomes following therapy for people with motor-FND, but few trials have focussed specifically on those with FGD.

Jordbru et al. (63) completed the only randomized trial explicitly investigating rehabilitation for FGD. The study randomized sixty people to a three-week inpatient multidisciplinary rehabilitation program or to a waiting list control group. The treatment was described as adapted physical activity within a cognitive behavioral framework. Significant between-group improvements were reported immediately after treatment in the Functional Mobility Scale, Functional Independence Measure and the Physical Domain of the SF-12. Benefits from treatment were mostly maintained at 12-month follow up, with some loss of treatment effect in measures of mental health.

Table 2 provides an outline of the results from multidisciplinary intervention programs that have focussed on outcomes in motor-FND, which includes participants with FGD. Positive outcomes were reported in all studies, with most adopting small cohort designs, some with long-term follow up. Consistent themes are evident that can be applied to treatment of FGD including 1) multidisciplinary interventions, 2) motor retraining, 3) goal setting with a graded approach, and 4) an individualized treatment tailored to the patients' needs.

Physiotherapy is an integral part of the rehabilitation of gait in people with FGD. Nielsen et al (68) conducted a pilot randomized study of specialist physiotherapy for motor-FMD. Participants were randomized to the treatment group ( $n = 30$ , specialized physiotherapy) or control group ( $n = 30$ , treatment as usual). Results indicated high acceptability of the treatment and no adverse events, with 72% of the treatment group rating their symptoms as improved at 6 months, compared to 18% in the control group, and moderate to large treatment effect

across a range of outcomes, including the physical domains of the Short Form-36 (Cohen's  $d = 0.46-0.79$ ). Consequently, a powered randomized controlled trial is underway (69).

Owing to the multidimensional nature of FGD and the contribution of non-motor symptoms, the involvement of other multidisciplinary disciplines is an essential part of the treating team. Occupational therapists are key members of the treating team involved with people with FGD, often addressing both motor and non-motor symptoms and the impact on independence in daily function. Consensus expert recommendations for occupational therapy have been described, which are supported by evidence from multidisciplinary treatment trials (70,71). Similarly, psychologists are integral to the treating team and provide psychotherapy to address FND symptoms, as well as comorbid mental illness, such as depression and anxiety, which commonly occur in this population (72). A recent systematic review of psychotherapy treatment for adults with FND indicated that both cognitive behavioral therapy and psychodynamic therapy were potentially effective treatments, although further controlled trials and long-term follow-up are needed (73). Ideally, this interdisciplinary care is best provided within the context of a specialty FND service, either in hospital-based or community-based settings, with leadership and care coordination from a rehabilitation physician, including communication with the patient's community-based primary care giver. However, these specialty services are rare and many challenges impact how this treatment is delivered, including limitations around resources and clinicians' knowledge of FND (28). The rehabilitation of people with FND may occur more commonly in typical neurorehabilitation settings. It is vital to recognize that FND may not respond to typical approaches of treatment, and that treatment modifications are needed to address mechanistic drivers of these symptoms, such as attention (i.e., the reversibility of symptoms with diverted attention) but also psychological factors such as anxiety. It is for these reasons that improved awareness of the assessment and management of FND amongst clinicians in these treatment settings is vital to improved patient outcomes.

This review found there is a growing evidence base for the treatment of FGD, but there is a lack of well-powered randomized controlled trials. More research is needed to determine the optimal treatment parameters for FGD, including the type of therapy, dosage, setting and intensity.

## Conclusion

Functional gait disorders are multidimensional and disabling, with numerous phenotypes described in the literature. Both motor and non-motor symptoms contribute to FGD, but their relative contribution needs further investigation. Non-motor symptoms have been shown to be associated with increased illness burden and should be carefully considered during assessment and treatment. The current recommended treatment for FGD involves multidisciplinary rehabilitation, but optimum treatment elements are yet to be determined. Future research should focus on further characterization of the motor and non-motor symptoms in FGD and their impact on quality of life, gait and participation, to inform future treatment studies.

## Key points

- Diagnosis of FGD should be based on the clinical examination identifying positive clinical signs of FND (e.g., Hoover's sign for functional weakness) and symptoms that are inconsistent and incongruent with structural disease.
- Functional gait disorders may occur alongside other neurological diseases, such as brain injury. Up to 12% of patients with neurological disease may have functional neurological symptoms.
- The aetiology of FND is best understood using a biopsychosocial model that considers predisposing, precipitating and perpetuating factors.
- Non-motor symptoms are common in people with functional gait disorders, and these may account for a greater proportion of the experienced disability and distress than motor symptoms. These symptoms should be considered during assessment and treatment.
- Multidisciplinary rehabilitation is recommended for people with FGD.

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