Functional movement disorders

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Purpose of review
The review highlights the clinical presentation of functional movement disorders (FMDs) and presents current evidence on bedside signs and paraclinical tests to differentiate them from other neurological disorders.

Recent findings
FMDs are diagnosed by the presence of positive clinical signs as emphasized in the new Diagnostic and Statistical Manual of Mental Disorders-5 classification criteria. Bedside signs are numerous, and a subset of them has been validated in controlled studies. This review summarizes evidence from the literature on specificity and sensibility of positive clinical signs for FMDs. The value of rule-in paraclinical tests to confirm the diagnosis is also presented. Recent developments in neuroscience with pathophysiological mechanisms and current treatment strategies are also discussed.

Summary
FMDs represent a field of neurology that is currently rapidly growing in terms of research. Clinicians should be aware that highly reliable signs exist for the diagnosis and that early multidisciplinary treatment should be offered.

Keywords
functional movement disorders, pathophysiology, positive clinical signs, treatment

INTRODUCTION

Functional movement disorders (FMDs) are defined as abnormal involuntary movements that are incongruent with a known neurologic cause and neuroanatomy. Already known in ancient Greece, they were considered as a form of hysteria, with symptoms due to a wandering uterus. The subsequent term 'conversion' expressed Sigmund Freud's theory that unconscious intrapsychic conflicts were converted into neurologic physical symptoms. In line with this view of a causal psychological factor, the term 'psychogenic' has then been widely used. New nomenclature uses the term 'functional disorder' [1], which is less stigmatizing and offensive [2] and by consequence better accepted by patients. As negative signs of neurologic disease do not exclude an organic cause, diagnosis should rely on the presence of positive clinical characteristics as emphasized in the new DMS-5 criteria. This review will focus on this diagnostic aspect by presenting current evidence on positive clinical signs for FMDs. Physiopathological mechanisms and current treatment strategies will be discussed.

DIAGNOSIS

The previous version of the Diagnostic and Statistical Manual of Mental Disorders (DSM)-IV defined conversion disorder as the association of a functional, medically unexplained, neurological symptom (criterion A) with a psychological stressor (criterion B).

The new DSM-5 version, published in May 2013, insists on the importance to make a 'positive' diagnosis of conversion disorder and to put less weight on the associated psychological factors. A new criterion (B) requires that 'clinical findings provide evidence for incompatibility between the symptom and recognized neurological or medical conditions' [3,4] (American Psychiatric Association, 2013). The way to provide the evidence for incompatibility is to use bedside tests [5] or paraclinical tests [6] that are specific and reliable for functional neurological disorder (FND) [7,8]. Earlier diagnostic criteria for functional/psychogenic movement disorders, such as the original [9] and modified [10] Fahn and Williams criteria, also emphasized incongruence and incompatibility with a known neurological condition. The

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KEY POINTS

- FMDs are defined as abnormal involuntary movements that are incongruent with a known neurologic cause and neuroanatomy.

- Diagnosis should rely on the presence of positive clinical characteristics.

- Psychopathology is not always evident but psychological factors are important risk factors and/or maintaining factors.

- Functional movement disorders must be distinguished from simulating.

- Cerebral dysfunction can be objectified by fMRI.

- Therapeutic approach should be multidisciplinary, involving both physical therapy and psychiatric care.

advantage of the new DSM-5 classification is that it covers not only movement disorder presentation but also represents a generic classification with sub-types according to the symptom [e.g. F44.4 for motor FND (weakness or abnormal movement), F44.5 for attacks or seizure].

CLINICAL EVALUATION

Anamnesis revealing mismatch between impairment and disability, and also history of marked variability of symptoms or exacerbations with sustained spontaneous remissions can suggest a functional origin. Anamnesis also provides a good opportunity to start the examination by observing the symptom's variations and distractibility during spontaneous speech and behavior, as it is a typical functional sign [11].

Dystonia

Functional dystonia makes up to nearly a third of all functional phenotypes [12]. Fluctuations in severity and variation in tone with passive manipulation, as observed in other functional disorders as reliable positive signs, are not very helpful in differentiating dystonia, as they are often also present in organic dystonia [13]. Inconsistency in topographic localization and severity over time are more useful [12]. Sudden onset and a fixed posture at rest, which offers marked resistance to passive manipulation, fixed postures at onset, prompt resolution immediately after botulinum toxin injections, and variable generalization to the rest of the body with intermittent episodes of exacerbation are typical features of functional dystonia [14,15] (Table 1).

Torticollis

Classical phenotype is the painful torticollis with sudden onset often after trivial trauma [16,17]. Often, predominant laterocollis with ipsilateral shoulder elevation and contralateral shoulder depression is seen. Contralateral shoulder depression and resistance to passive manipulation are only seen in functional cervical dystonia and therefore a good sign to distinguishing from its organic counterpart.

Foot dystonia

In adults, foot dystonia at rest (nonexercise-induced, nonparoxysmal) is most often functional, commonly with a plantar flexion an inversion of the foot [17]. An unusual variant is the functional 'striatal toe' with a fixed first-toe extension and second to fifth-toe flexion. Extension of first toe is typically resistant to forced flexion but undergoes spontaneous flexion when the examiner extends the second to fifth toes [18].

Hand dystonia

Functional dystonia of the hand typically involves the second to fifth fingers with relative or complete sparing of the thumb, and, in some instances, the index finger, thus preserving the important pincer function of the hand [17].

Facial movement disorders

Functional facial movement disorders involve the lips (60.7%), eyelids (50.8%), perinasal region (16.4%), and forehead (9.8%) [19], resembling blepharospasm, hemifacial spasm, or oromandibular dystonia [20▪▪].

Typical sign for functional blepharospasm is the contraction of corrugator (forehead frown) and procerus muscles (wrinkles the skin overlying the nasal radix) in the absence of spasm of the orbicularis oculi, resulting in narrowing of the eyelid fissure, depression of the eyebrows, but without spasm of orbicularis oculi [21].

The so-called Babinski 'other' sign only seen in organic blepharospasm (100% specificity) [22,23] is characterized by an elevation of frontalis on the same side as orbicularis oculi involvement (narrowing of eyelid fissure and ipsilateral rising of the eyebrows during eyelid spasms). On the contrary, functional patients with asymmetric spasm of orbicularis oculi will display rising of eyebrow contralateral to the closing eye [19].

Inability to open the eyes, resembling 'eyelid-opening apraxia,' can be seen [24] and is characterized in FND by variable resistance to passive opening of eyelids (Table 1).

Table 1. Clinical features of functional movement disorders (FMDs)

	Features suggesting FMD	Features suggesting organic disorder	Complementary examinations
Dystonia	Inconsistency in localization and severity over time	Geste antagoniste	
	Sudden onset	Progressive onset	
	Fixed posture at rest		
	Fixed postures at onset		
	Prompt resolution after botox/suggestion		
Torticollis	Predominant laterocollis with ipsilateral shoulder elevation and contralateral shoulder depression		
	Prompt resolution after botox		
Foot dystonia	Nonexercise-induced		
	Nonparoxysmal		
Hand dystonia	Spared pincer function (dystonia Dig. II–IV)	Involvement Dig I–II	
Facial movement disorders	Eyebrow rising contralateral to closed eye	Babinskis 'other' sign: narrowing of eyelid fissure and ipsilateral frowning/elevation of the eyebrows during eyelid spasms	
	Lip-pulling sign	Tongue deviation ipsilateral to facial weakness	
	Resistance to passive lid opening	Persistence during sleep	
	Tongue deviation contralateral to facial weakness		
	Absence during sleep		
Tic	Adult onset, absence of tics in childhood	Perceived as intentional movement (release inner tension)	
	Lack of premonitory sensations,	Premonitory sensation	
	Inability to suppress movements		
	Perceived as involuntary		
Tremor	Distractibility	Constant frequency-peak	ENMG
	Variability (amplitude/frequency, localization, direction)	Rhythmic, nondistractible	
	Entrainment	Cave: irregular dystonic tremor	
	Whack a mole sign		
Parkinsonism	Bradykinesia without decrement in amplitude	Cogwheel, increasing with distraction	DAT scan
	Resistance without cogwheel, decreasing with distraction maneuvers	Bradykinesia with decrement in amplitude	
	Tremor variable, diminishing during walking	Re-emergent tremor	
		Tremor increasing during walking	
Myoclonus	Axial myoclonus	Short lasting	ENMG: inconsistent pattern, >700 ms
	Too slow/complex for organic myoclonus		EEG: Bereitschaftspotential (Backaveraging)
	Spinal myoclonus with coexistence of facial movements/vocalizations		
	Distractibility, entrainment		
Gait	Uneconomic postures: knee buckling, astasia-abasia, 'tightrope walking', 'walking on ice'	Enhancement with distraction	
	Exaggerated compensatory maneuvers		
	Huffing and puffing		
	Improvement with distraction		

DAT, Dopamine transporter scan; EEG, Electroencephalogram; ENMG, Electroneuromyography.

The most common pattern of facial FMDs consists of tonic, lateral, and/or downward protrusion of one side of the lower lip with ipsilateral jaw deviation (84.3%), called 'lip-pulling sign' [19].

The tongue may be involved, with 'wrong-way' tongue deviation (contralateral to the facial weakness) [25].

Palatal tremor can also be seen in FND, with the key features of entrainment and distractibility [26,27].

Functional facial spasms typically disappear during sleep [19,28] when it can persist in up to 80% of patients with hemifacial spasm [29].

Tremor

Functional tremor is the most frequent presentation of FMD [30]. The tapping task for distraction is the most reliable positive sign for detection of functional tremor (sensitivity and specificity both 73%) [31]. With distraction (during anamnesis, mental calculation, examination of other body part), functional tremor typically dramatically improves, or changes in frequency and/or amplitude (sensitivity of 58% and specificity of 84%) [31]. By performing a finger-tapping task at a given frequency with the contralateral hand or another body part, tremor may demonstrate entrainment (tremor overtakes the frequency of the contralateral movements), another clinical hallmark of functional tremor [32]. Whereas organic tremors can also have variable amplitude influenced by the level of anxiety/exercise/position, variability in frequency and direction (changing from pronation/supination to flexion/extension) is typical for functional tremor. When restricting a tremulous limb, the tremor may suddenly spread to another limb, so called 'whack-a-mole' sign. [33]. In case of a tremor irregular in rhythm and direction, differential diagnosis of a dystonic tremor should be considered [32].

Parkinsonism

Tremor in functional parkinsonism often affects the dominant hand and is mostly equally present during rest, posture, and action, opposed to the rest tremor in Parkinson's disease, which classically decreases with action. Further difference is the absence of a brief pause in tremor when assuming an outstretched posture of the hands, so-called re-emergent tremor classically seen in PD [34]. The tremor is distractible and diminishing while walking, in contrasts with the rest tremor in PD, which classically enhances when walking. Repetitive movements may be slow, but true bradykinesia with decrement in amplitude or arrests in

movements is not seen [35]. When assessing tone, there is active resistance without cogwheeling, decreasing with distraction maneuvers – the opposite of what occurs in PD [36].

Myoclonus

After functional tremor and dystonia, functional myoclonus and jerks represent the third most common diagnosis of FMDs [37]. Axial jerks are more likely to represent functional jerks (58% of propriospinal myoclonus) [38,39]. Arguments in favor of functional propriospinal myoclonus are [1] previous somatizations, [2] coexistence of facial involvement (do not occur with a spinal origin), [3] normal imaging of the spinal axis [4], and presence of Bereitschaftspotential (cave: latter can be absent or not recordable) or inconsistent electromyogram pattern [39–41]. Functional jerks are distractible, may show entrainment [3], and are often too slow or too complex to be organic myoclonus. When there is stimulus sensitivity, delay is long and variable from the stimulus.

Gait

Abnormal gait is a common feature in patients, with 5.7% of FMDs presenting isolated gait disorders and 36.6% mixed movement disorder with gait disorders [42]. In pure functional gait, 'knee-buckling' is the most common pattern, followed by astasia-abasia [43]. Functional impairment of equilibrium is often accompanied with exaggerated compensatory maneuvers such as putting the arms out ('tightrope walking'), or marked reduction in the step height and stride length ('walking on ice') [44]. These following signs have a high specificity (between 94 and 100%): momentary fluctuations of stance and gait, excessive slowness or hesitation, psychogenic Romberg test, uneconomic postures, 'walking on ice,' and sudden buckling of the knees [8,45]. 'Huffing and puffing' signs (huffing, grimacing, and breath holding while walking) were present in 44% of patients with functional gait disorders, but minimal or absent in organic gait disorder, yielding 89–100% specificity [46]. Sway may improve in functional disorders rather than worsen with mental distraction [47]. Other inconsistency is when patients complain of poor balance or falls, with preserved or even skillful balance control to maintain uneconomic postures [45,48] or continuous exaggerated truncal sway (astasia-abasia) (Table 1).

Tic

Functional tics are a rather rare, particularly challenging because typical features of FMDs (distractibility,

suggestibility) are also characteristic of organic tics [49,50]. Hints for their diagnosis are: adult onset, no premonitory sensations, no tics in childhood, negative family history, inability to suppress movements (patients with tics can usually suppress their tics for short periods), and coexistence with other FNDs [49,51]. Tics are usually experienced as intentional movements performed in order to relieve inner tension, whereas functional tics are perceived as involuntary [51,52]. Premonitory sensation is considered a hallmark feature of organic tics, and it is reported in about 90% of patient (although also reported in patients with presumed functional tics) [39,52], but larger studies are needed [49,51] to have definite views on this issue. (Table 1)

COMPLEMENTARY EXAMINATIONS

Electroneuromyography

Electroneuromyography is especially helpful for tremor analysis and discrimination of different types of myoclonus or dystonia.

Tremor recording by electromyography (EMG) and accelerometer (recording the movement, tremor frequency and amplitude) has a good sensitivity (89.5%) and specificity (95.9%), and also interrater reliability for differential diagnosis of functional versus organic tremor [53""]. Test battery consisted of the following: tremor recording at rest, posture (with and without loading), action, while performing tapping tasks (1, 3, and 5 Hz), and while performing ballistic movements with the less-affected hand). Tonic muscular co-activation, intermanual coherence of tremor frequency, response to contralateral ballistic movement (pause of tremor), loading (increased tremor frequency), and correct tapping performance to a given frequency were regarded as positive functional signs.

Assessment of jerks is made by surface EMG, conventional electroencephalogram (EEG), Jerk-Locked-Back-Averaging (JLBA), somato-sensory evoked potentials (SEP), and C-reflex studies [54]. The duration of EMG bursts is below 75 ms in organic and above 75 ms in psychogenic myoclonus. EEG helps in identifying the cortical origin of a myoclonic jerks and its temporal relationship with the cortical activity [54]. Presence of Bereitschaftspotenzial [negative cortical potential, maximal amplitude over the central areas (Cz), starting 2000–1000 ms prior to the jerk, associated with self-initiated movement] in EEG strongly suggests functional myoclonus [40,41]. Inconsistent muscular recruitment pattern in EMG with entrainment, distractibility, and stimulus sensitivity can be found [55].

In functional blepharospasm, the blink reflex (paired supraorbital nerve stimuli) exhibits a normal recovery cycle (R2) [56], unlike organic blepharospasm (abnormal R2).

In fixed-foot dystonia, an abnormal pattern of co-contraction of antagonist muscles preceding the movement can only be observed in functional foot dystonia [57].

Ultrasound

The use of ultrasound can be recommended [58] as a noninvasive, convenient diagnostic tool for further studies of diaphragmatic myoclonus. Ultrasound can evidence distractibility and entrainment of functional diaphragmatic myoclonus on real-time video [58].

Dopamine transporter scan

Dopamine transporter single-photon emission computed tomography (DAT-SPECT) can be useful in distinction between functional parkinsonism and Parkinson's disease. Normal DAT-SPECT does not distinguish functional tics from benign tremor disorders (essential or dystonic tremor) or from nondegenerative parkinsonian disorders (drug-induced or vascular parkinsonism) [59].

PHYSIOPATHOLOGY

With the new DSM-5 classification, diagnosis of FMD can be made without an identified psychological triggering factor [60,61]. Whereas a recent study confirmed higher rates of childhood trauma in FMD patients compared to healthy volunteers [62], psychopathology is not always evident; a substantial proportion of patients (from 0 to 86%) do not report having experienced traumatic events in their history [63]. If psychological factors alone are not sufficient to explain the etiology of FMD, they are still important risk factors and/or maintaining factors [60,61].

Evidence suggesting abnormal emotional regulation in FND was gathered with increased amygdalar [64] and periaqueductal gray (PAG) activity [64] in patients during negative emotion stimuli. Lack of habituation in amygdalar activity over time suggests a general hyperarousal state in FND. Patients may be more prone than healthy controls to automatic motor defense behavior, such as freeze response, mediated by PAG abnormal activity, because this region is known to be implicated in the freeze response in both animal [65] and human [66,67""] studies.

There is also evidence of alteration in autonomic nerve system function in FMD: decreased vagally mediated heart rate variability was observed in FMD

patients, both [68▪] adults and children [69], resulting in potentially inadequate protection from sympathetic stressors in these patients [68▪].

Abnormal limbic–motor interaction is suspected to play a role in the generation of motor symptoms; an fMRI study looking at implicit emotion processing [64] found a greater connectivity in FMD patients between the amygdala and the supplementary motor area (SMA), whereas another found this same hyperconnectivity during the recall of autobiographical traumatic events [70]. Additional finding from this study was increased dorsolateral prefrontal cortex activity and concomitant reduced hippocampal activity in patients during recall of the trauma, which is a pattern involved in active forgetting of unwanted memories [70,71], congruent with Freud's repression theory.

Distinctive changes in brain activation were found when comparing functional symptoms to fake symptoms (voluntarily produced by simulators/feigners) [72–76]. Compared to healthy controls and actors feigning weakness, conversion patients showed decreased dorsolateral prefrontal cortical activity, and also reduced activation of the contralateral parietal region and increased perfusion in the frontal region. The authors hypothesized that frontal regions were inhibiting the motor and premotor areas when the patient tried to move their affected limb, as if the 'center of volition' was malfunctioning. This fitted with a view developed long ago by James Paget: the patient says 'I cannot,' it looks like 'I will not,' but it is 'I cannot will' [77]. It is important to note that symptoms are perceived as involuntary by the patient and by consequence FMDs must be distinguished from simulating.

Comparing patients when they had their involuntary functional tics to a period where they had a voluntary tremor (order to reproduce their tremor intentionally), fMRI demonstrated a reduced brain activity in the right temporo-parietal junction (TPJ), a key area involved in integrating complex sensory signals [78,79]. The right TPJ plays an important role in the sense of agency, which is the sense that we are the actors of our own actions. The TPJ acts as a comparator between the feed-forward (intended movement) and the feedback processes (feedback of the effectuated movement); when feed-forward and feedback processes match, the sense of agency is reached. The reduced TPJ activity in functional patients suggests that patients are no longer able to perceive their movement as voluntary.

TREATMENT AND OUTCOME

Treatment begins with communication of the diagnosis to the patient. Use of the term 'functional disorder' is recommended, as it is descriptive (disorder of function of nervous system, no underlying structural abnormality) and nonjudgmental [2,80]. It should be emphasized that symptoms are real, are not imagined or simulated, and are potentially reversible [81▪▪]. The pathological role of attention toward movement should be explained and distraction techniques (e.g. music, talking, or altered gait patterns) can be taught [82▪▪] with the aim to reinstate automatic neurologic control and redirect unhelpful movement-focused attention [83]. Further medical information on the disorder can be provided by indicating a website (www.neurosymptoms.org) by a neurologist and translated in many languages and self-help guides [84] can be provided.

The therapeutic approach should be multidisciplinary [85] involving the general practitioner, the neurologist, the physiotherapist, and when relevant, the psychiatrist, speech therapist, occupational therapist, and social worker.

Physiotherapy has a key role in allowing patients to better understand the illness and its potentially reversibility (demonstration that normal movement can occur), and to retrain movement with diverted attention [82▪▪]. TENS (transcutaneous electrical nerve stimulation, producing a tingling sensation) has been tested as a potential treatment for patients with functional sensory loss to improve sensory awareness [82▪▪,86]. Physiotherapy showed improvement of symptoms in 55% of patients at 3 months of follow-up [87]. Another randomized controlled study of physical rehabilitation in FMD reported symptom improvement in 72% of the intervention group (only 18% of the control group) [88▪].

Accompanying symptoms are frequent (pain, fatigue, difficulties in concentration) and should be explained to the patient [82▪▪].

Patients with FNDs often have more than one symptom and or may develop other functional disorders later during follow-up. [89]. Receiving health-related benefits has a negative effect on outcome [90]. Longer duration of symptoms seems to correlate with negative outcome [91]. Early diagnosis is important to prevent chronification. Psychiatric comorbidity was found to be an inconsistent predictor of poor outcome [92]. Data concerning outcome are variable. The largest prospective follow-up study in mixed FNDs (cohort study of 716 patients followed up over a 1-year in Scotland) [90], outcome was poor with 67% of the patients having unchanged symptoms or worse.

Psychiatric evaluation is important, as many FMD patients also suffer from psychiatric comorbidities such as anxiety and/or depression, but often only accepted with a good alliance with the patient [93].

A few studies evaluated transcranial magnetic stimulation (TMS) as a potential treatment for FND. One recent controlled trial [94] randomized patients with paralysis of at least one hand to either active (subthreshold TMS) or placebo treatment (stimulation of the underlying scalp) before switching to the other treatment (active/placebo) 2 months later. Significantly, larger median increase in muscular strength after active compared to sham TMS (24% versus 6%; $P < 0.04$) was observed. The mechanism by which TMS could work is, however, still unclear as placebo effect, suggestion, and neuromodulation [95] are all possibilities; further studies are still needed.

CONCLUSION

Functional movement disorders are frequent and diagnosed by clinical positives signs. Psychological factors alone are not sufficient to explain the etiology of FMD, but are important risk factors and/or maintaining factors. Approach should be multidisciplinary. In the future, the development of specialized centres, with both in-patient and out-patient treatment plans, should be a priority for these complex patients.

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- ■ of special interest
- ■■ of outstanding interest

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