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Functional movement disorders

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Abstract :

Functional movement disorders (FMD) represent a complex and disabling entity characterized by a broad range of clinical symptoms not explained by a classical neurological disease. In 2013, the Diagnostic and Statistical Manual of Mental Disorders (DSM-5) added a clinical criterion based on incongruence and inconsistency, supported by recent literature highlighting the role of "positive clinical signs". These clinical signs allow a "rule-in" procedure in making a diagnosis of FMD so that the diagnosis is no longer a "rule-out" or "by default" diagnosis made after exclusion of other neurological conditions. This review summarizes current evidence on common clinical features and highlights bedside signs in FMD, such as tremor, dystonia, myoclonus and parkinsonism. Tics, chorea and hemiballism are also briefly discussed.

Keywords: Functional neurological disorder, Functional movement disorder, Psychogenic, Conversion disorder.

Abbreviations

FND: functional neurological disorder, FMD: functional movement disorder

1. Introduction:

(FMD) are frequent and disabling. They include mainly tremor, dystonia, myoclonus and parkinsonism and belong to a larger entity called functional neurological disorders (FND). FND comprise neurological symptoms that cannot be explained by a classical neurological disease. Consequently, FND are embodied by a broad phenomenological spectrum that encompasses non-epileptic seizures, sensorimotor deficits, gait abnormalities as well as abnormal movements. Importantly, FND are involuntary by nature and must be distinguished from factitious disorders and malingering [1]. However, clinicians use a variety of terms, including “psychogenic”, “conversive”, “dissociative” or “hysteria”, thus maintaining some stigma. In the latest version of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5) [2], the term FND is used in the interests of standardization. It refers to pathophysiological mechanisms highlighted by recent neuro-imaging findings supporting a dysfunction in complex neural networks [3,4]. The term functional is thus now added to the term “conversion” based on Sigmund Freud’s theories (the symptom represented a conversion of an intra-psychic conflict into a physical sign) [5]. Furthermore, DSM diagnostic criteria for FND have been modified, with removal of the previously required association with a psychological precipitant, which may be difficult to investigate in clinical practice and is rather unspecific [6]. Simulation was also formally discarded, since a diagnosis of certainty could only be based on either confession by the patient, or evidence of feigning, which are both extremely rare in clinical practice [6]. An important step forward is the integration of “clinical findings providing evidence of incompatibility between the symptom and recognized neurological or medical conditions”. Indeed, recent literature has focused on the so-called “positive clinical signs” such as Hoover’s sign in functional paresis, or closed eyes during

non-epileptic seizure. Some of these signs especially sensorimotor showed an excellent specificity (92-100%) but a low sensibility (8-100%) with acceptable inter-observer agreement [7,8]. Therefore, the diagnosis of FND includes positive criteria and is no longer a purely exclusionary approach. Some practitioners have even proposed to show these signs to patients, in order to facilitate understanding and acceptation of the diagnosis [9].

The aim of the present article is to review "positive clinical signs" in FMD. We focus on tremor, dystonia, myoclonus and Parkinsonism, and briefly discuss tic, chorea and hemiballism. We do not discuss gait disorders, because although they can be considered as FMD, they may also belong to other forms of FND such as sensorimotor deficit.

2. Epidemiology

The prevalence of FND in neurological inpatients is consistently reported to be between 1 and 9%, while incidence is between 4 and 12 per 100,000 population per year [10–12]. Medically unexplained symptoms represent 30% of neurological consultations and often have a chronic course [13,14]. Symptoms have been reported to persist or worsen in 39% during follow up of 7.4 years, according to the type of symptom [15]. In Neurology departments specialized in abnormal movement, the prevalence of FMD ranged from 3.3 to 3.6%, with 40.6 to 50% of patients having tremor, 17.2 to 18% dystonia, 4.3 to 14% myoclonus and 7 to 39.8% Parkinsonism [16–18]. It is probably under estimated in the elderly [19]. Mean age at FMD onset is 40 to 50 years with preponderance of female patients (2.5 females for 1 male) [16,17]. In the setting of FND, compared to the general population, psychiatric comorbidity have been reported to be frequent; up to 43% having major depression, 61% anxiety disorder (both in 28% of cases) and 45% personality disorder [20]. Overall, prevalence of psychiatric comorbidity can reach 89% of FND and it is frequently associated with sexual abuse, physical negligence and self-harm behaviour [21]. Furthermore, childhood trauma and recent life

events seem to play a role in the occurrence and severity of the disorder [22]. Generally, FMDs are considered sporadic disorders but family history is described and may be an additional risk factor [23]. Few studies have focused on differences between cultures and only one has compared American and Spanish populations, and found no significant differences (e.g. in terms of symptoms, age, or sex ratio) [24].

3. Common clinical characteristics :

FMD share some common characteristics (see Table 1), which were the basis for the diagnostic criteria developed by Fahn and Williams in 1988, defining four categories, namely documented, clinically established, clinically probable and clinically possible [25]. These criteria were subsequently updated in 2009 by Gupta and Lang, who proposed a simplified classification of diagnostic certainty incorporating electrophysiological assessment [26,27]. Classically, onset of FMD is sudden and often with a precipitating trigger [28]. The course of the disorder includes periods of remission (which may be complete) and is variable over time, with worsening and sometimes even a change in the nature of the disorder [29]. A “selective” handicap for a certain type of activity is often present, reflecting discordance with the physical examination of the patient and the socio-professional impact of the disorder. In general, FMD are inconsistent, with variability, distractibility and the possible presence of entrainment (this sign is discussed in further detail below) [30]. Recently, the “whack-a-mole” sign has been described, whereby, if the involuntary movements of one body part are suppressed by holding it, then the involuntary movements will migrate to other limbs, and possibly even occur with greater magnitude [31]. FMD present some discordances with abnormal movements of organic origin that are established and responsive to suggestibility, either by administration of a placebo or other means. The association of different types of FMD is possible and FMD

may also be associated with other types of FND. The following sections describe the clinical signs specific to each type of FMD (see Table 2).

3.1. Tremor:

Tremor is defined as a rhythmic and oscillatory movement of a body part resulting from synchronised alternating contraction of agonist and antagonist muscles. Functional tremor is the most common FMD. By contrast with organic tremor, functional tremor often displays wide variability in terms of amplitude, frequency, or direction, and may be present at rest, during action or on posture [32]. Functional tremor mainly affects the upper limbs, although the fingers are usually spared. The following is a (non-exhaustive) list of useful clinical tests to diagnose functional tremor:

- Entrainment: This is likely the most useful clinical test to confirm the functional origin of tremor. The exact definition of the entrainment test varies across studies, and may sometimes be confused with distractibility. Basically, the subject is asked to perform a voluntary, repetitive movement with the contralateral limb at a given frequency (for example, tapping thumb and index, moving the tongue from one side to the other, flexion-extension of the wrist, or auditory stimuli). The affected limb should be in the position where the tremor is most prominent, and the examiner should do the movement to set the imposed frequency, varying the tapping speed. The objective is to check whether the tremor takes up the frequency of the unaffected body part, and secondly, whether the patient has any difficulty following the requested rhythm with the healthy limb. Some authors have suggested that the test is positive if there is a change in tremor frequency (acceleration or slowing) or a pause [33] ; however, this more close resembles distractibility than “pure” entrainment. The entrainment test is based on the fact that it is difficult for a normal or functional subject to

generate and maintain two different rapid rhythms for a prolonged period. In organic tremor, the generation of the movement is independent [34]. The sensitivity of the entrainment test is low (from 8.3 to 16%, and up to 39% with electrophysiological assessment), but specificity is high (from 75 to 95%) [35,36].

- Distractibility: This test consists in diverting the subject's attention away from the tremor by asking them to perform different manoeuvers; for example, counting backwards from 7 to 0, performing ballistic movements with the contralateral limb, or repeatedly touching the thumb with the second, fifth then third fingers as fast as possible [35]. In functional tremor, the tremor should diminish or stop altogether during these manoeuvers. Distractibility tests are based on the same principle as the entrainment test, namely that it is extremely difficult to generate and maintain two different rhythms concurrently. Sensitivity of distractibility testing is quite high (between 58 and 73%), as is specificity (73 to 84%) [35].

- Suggestibility: This consists in inducing a change in the symptom through the suggestion of ideas. For example, in their study, Kenney et al [35] explained to patients that hyperventilating had been shown to increase tremor, or the application of a vibrating source to the body had been shown to improve tremor greatly. Then, they asked to the patient to hyperventilate for 10 seconds, or applied a vibrating tuning fork to the patient's forehead until vibration stopped spontaneously. The prevalence of this sign is 33% [37], with a reported sensitivity of 42 to 50%, and specificity of 82 to 88% [35].

- Coactivation: Coactivation can be tested in the same way as rigidity, namely by having the patient perform slow arrhythmic, passive movements of one or two joints. In functional tremor there might be an increased baseline tone which will thus fluctuate or even disappear with relaxation, as will the tremor itself [32]. No study has evaluated the sensitivity

or specificity of this sign. Raethjen et al [38] compared accelerometry and electromyography (EMG) recordings in 15 patients with functional tremor manifesting in both hands. They reported that 7/15 patients showed significant coherency between the two hands, while the remaining 8 patients maintained independent oscillations. Those who had coherency between hands had a tremor below 6 Hz, which is close to "voluntary" tremor. In addition, this is coherent with the fact that it is impossible to voluntarily produce two oscillations of different frequencies. Conversely, those without coherency had oscillations in the 7-10 Hz band, which is a frequency similar to that of physiological tremor. This is the result of co-contraction and reflects hyperexcitability of motor neurons [39].

3.2. Dystonia :

Dystonia is defined as a sustained or intermittent muscle contractions causing abnormal movements, postures or both. It is typically patterned and twisting, influenced par volontary action and associated with overflow muscle activation [40]. The origin of dystonia was long considered to be functional but there is now a distinction between organic dystonias (with some genetic forms now better characterized) and functional dystonia [41].

The distinction is still difficult but some clinical features can be of help; a functional origin of dystonia is suspected when it is fixed at onset, whereas organic dystonia is often mobile at the initial phase. Sudden onset precipited by a minor physical trauma is strongly suggestive of functional origin [42]. Functional dystonia is suspected in the presence of an atypical localisation and age of onset, for example adult onset foot dystonia [25]. Functional dystonia spreads to other parts of the body (head, neck and trunk) with episodic exacerbation and might show an exaggerated and rapid improvement after botulinum toxin injections. Different phenotypes have been described according to the fixed or paroxysmal characteristics and the body's part affected [43]. The following are other important clinical clues (see Figure 1):

- Fixed dystonia : When dystonic posture reduce joint mobility; the absence of sensory tricks orients towards functional dystonia. Sensory tricks are voluntary manoeuvres that reduce the severity of dystonia. They can be observed in focal or generalized dystonia, and in primary or secondary dystonia, and may take several forms (for example, in focal cervical dystonia, the patient may hold their head with their hand) [44]. These manoeuvers are usually absent in functional dystonia [45]. Furthermore patients present exaggerated pain during passive movements, as well as an active resistance [42].

- Paroxysmal dystonia : Defined by a sudden self-limited episodes of dystonia, it functional origin can be pointed out by an important variability between episodes especially for duration and phenomenology. Adulte age onset and atypical trigger may be helpful such as medical examination worsening symptomatology. Its were highlighted by Ganos et al [46] who described a group of functional paroxysmal movement disorders including dystonia, tremor, jerks and complex generalised movements.

- Body distribution : head, neck, upper limbs and especially lower limbs can be affected in functional dystonia.

Commonly for the lower limbs, there is a plantar flexion with inversion of the foot. The "psychogenic toe" sign has been described by Espay et al [47] in focal dystonia of the big toe. A noteworthy feature is the presence of passive spontaneous plantar flexion of the big toe on forced dorsiflexion of the second to fifth toes, which is observed in functional dystonia but not in organic (striatal) toe. This indicates preserved physiological synkinetic movements of coordination (as in the Hoover sign for paresis of the leg), whereas these signs are absent in abnormal movement (paralysis) that is not of functional origin.

Concerning upper limbs, fixed wrist and finger flexion with relative sparing of the thumb and index fingers are common, preserving pincer function [48].

For the neck, controlateral lowering shoulder and an elevation of ispilateral shoulder relative to a laterocolis is typical in functional cervical dystonia.

Functional facial dystonia very often involves the lower part of the face, with unilateral contraction of the orbicularis muscle of the mouth downwards, with deviation of the ipsilateral cheek [49,50]. This sign is called the "lip-pulling test" and may be accompanied by ipsilateral involvement of the platysma [51]. In the hemifacial spasm; frontalis and orbicularis oculis have an ispilateral contraction leading to an eyebrown elevation and eye closure called "other Babinski sign" [52]. In functional dystonia, the eyebrown is raised controlateraly to the closed eye [51]. This can be accompagnied by a fluctuant resistance to passive opening eyelids. Futhermore, tongue may be helpful deviating in the "wrong way" towards the affected side in case of functional hemispam [51].

3.3. Parkinsonism:

Parkinsonism is characterised by bradykinesia in combination with at least one of rest tremor or rigidity [53,54]. Functional parkinsonism is difficult to diagnose but Lafaver et al [55] proposed definite criteria based on examination findings like slowness and variable resistance supported by laboratory examination. The following clinical clues can help establishing the diagnosis [56–59] :

- Tremor: Tremor is present not only at rest, but also during action and on posture. Many of the characteristics are shared with functional tremor (see above), notably distractibility. Extended to the leg, tremor suggests functional parkinsonism [59]. Classically, tremor in Parkinson's disease temporarily abates with a change of posture, and reappears

maintaining an attitude ("re-emergent tremor") [60]; these features are not observed in tremor of functional origin.

- Rigidity: The patient presents increased tone characterised by active and variable resistance against passive movement, which is amenable to distractibility. "Cogwheel" rigidity is uncommon in patients with functional parkinsonism.

- Hypokinesia: Patients present excessively slow, deliberate movements, often associated with grimacing, tiredness or sighing called the "huffing and puffing sign" [61]. During repetitive movements, the slowness is not progressive or accompanied by decrementing amplitude, a phenomenon known as the sequence effect and which is classically observed in Parkinson's disease.

- Postural/gait instability: Arm-swing in a patient with functional symptoms is diminished (mainly on the most affected side), with a stiff, extended arm, which is different from Parkinson's disease, where the arm is usually slightly flexed. When asked to run, there is no increase in arm-swing, contrary to parkinsonian patients. Postural stability testing (the pull test) often yields bizarre, atypical reactions. The patient may make exaggerated, inappropriate movements in an attempt to regain balance, e.g. wave or fling their arms and flail or reel backwards, without actually falling. In case of associated gait impairment, the"chair test" can be useful confronting abnormal ambulation with an easy propulsion when the patient is sitting on a swivel chair with wheels [62].

3.4. Myoclonus:

Myoclonus is defined as brief, sudden and involuntary contraction or inhibition of a muscle, generally caused by the central nervous system. Features that suggest a functional origin include variability or incoherence in amplitude, frequency and topographic distribution [63,64]. Generally, the movement is more complex and prolonged and can occur at rest, during action, or, like organic myoclonus, may be reflex-induced. In this latter case, a long latency between the reflex movement and the myoclonus could be one of the clinical signs specific to functional origin, but no study has examined this point specifically. One of the most demonstrative example is functional axial jerk mimicking propriospinal myoclonus. The majority of "propriospinal myoclonus" adressed in a tertiary referral center finally has been reported to be functional [65]. Recently Van der salm et al [65,66] proposed diagnostic criteria for functional axial jerk including anamnestic, clinical, neuroimaging and electrophysiological criteria. For instance facial movements or vocalizations associated with axial jerks argue in favor of functional myoclonus [65, 67].

3.5. Tics, chorea and hemiballism:

Regarding tics, there is a paucity data with only a small number of subjects studied to date. A functional origin is the observed etiology in 10% of all tics [68]. Clinical clues include adult onset, non-stereotypical phenomenology, absence of premonitory sensation, inability to transiently suppress the tics, and prominent distractibility [69]. Demartini et al [70] also add to this definition the absence of pali-, echo- and copro- phenomena, and the fact that the tic can disrupt an ongoing action, termed "blocking tics", which is rare in organic tics. Tic is felt as intentional movement in order to suppress inner tension whereas functional tic is experienced totaly as involontary [71–73]. Head is less commonly affected and absence of rostro-caudal distibution is seen in functional tics. More recently Ganos et al [74] question

some functional clinical clues such as adult onset (6 patients developed symptoms under 18 years-old) or sensory premonition (present in 9/13 patients). Finally, distinction between organic or functional tics is challenging because of sharing commun features. Diagnosis should be based more on a combination of symptoms. For chorea and hemiballism, in addition to the characteristics shared with other functional paroxysmal movement disorders, there are no other specific clinical signs yet reported to suggest a functional origin [46].

4. Conclusion :

In the era of technical progress in medicine, huge advances have been made in diagnostic procedures in domains like genetics, electrophysiology and imaging. This is of tremendous help but should not lead to a devaluation of the importance of the clinical examination. In particular, in the field of functional neurological disorders, the semiology and clinical examination remains of paramount importance and represents a reliable way to establish a diagnosis. Future multimodal diagnostic algorythms, combining semiology, electrophysiology imaging and biomarkers might increase the diagnostic certainty, which will only improve clinical care and research opportunities in this field. Currently, testing these positive signs at the bedside not only improves the diagnosis certainty but also allows a good communication with the patient, which is a very important first step towards treatment [9,75].

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Table 1 - Anamnestic clues suggesting functional movement disorders, adapted from [26,29].

Sudden onset (maximal at the beginning)
Waxing and waning course (spontaneous remission)
Paroxysmal nature
Migration around the body
Association or change of type of FMD over time
Association with other FND
Psychiatric comorbidity
Physical or psychological trauma (during childhood or/and recently)

FMD : Functional movement disorders; FND : functional neurological disorders

Table 2 - Features suggesting functional movement disorder.		
Tremor	Variability in amplitude,frequence and direction Fingers usually spared Entrainment Distractibility Suggestibility Coactivation “Wach-a-mole” sign	
Dystonia	Absence of sensory tricks Exaggerated pain Passive resistance Worsening symptomatology during examination Atypical trigger	Foot Plantar flexion with inversion of the foot “psychogenic toe” Hand Sparing of the thumb and index fingers Neck Controlateral lowering shoulder and an elevation of ispilateral shoulder relative to a laterocolis Head/cranial Lower part of the face Lip-pulling test Ipsilateral involvement of the platysma Eyebrown is raised controlateraly to the closed eye. Fluctuant resistance to passive opening eyelids « wrong way » tongue deviation.
Parkinsonism	Variable tremor (located in the leg) Active and variable resistance against passive movement without cogwheel rigidity Excessive slowness without sequence effect Huffing and puffing sign Running do not increase arm-swing Atypical reactions in postural stability test Chair test	
Myoclonus	Prolonged and complex movement Long latency between reflex movement and stimulus Association with facial movements or vocalizations	
Tic	Inability to transiently suppress it Absence of premonitory sensation Distractibility Blocking tics Unintentional feeling	

Figure 1 - Clinical clues suggesting functional dystonia.