

Psychogenic Movement Disorders

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1. Introduction

Abnormal movements and postures resulting from primary psychiatric disease are a diagnostic dilemma because all types of movement disorders may be mimicked by a psychogenic disease, including akinetic-rigid and hyperkinetic disorders, with the latter more frequent, particularly tremor, myoclonus, and dystonia (Williams et al., 2005; Reich, 2006).

Psychogenic movement disorders (PMDs), are a valuable model for all medically unexplained symptoms and raise arduous challenges for diagnosis and treatment indicating our restricted understanding of the true pathogenesis that causes them. A multiplicity of terms such as “hysterical conversion”, “functional”, “psychosomatic”, “neuropsychiatric”, “dissociative motor disorders”, and so on, have been applied to describe neurological symptoms that cannot be attribute to any known organic disease (Mace & Trimble, 1991; Lang, 2006). The term “psychogenic” is the commonest in the movement disorder literature, but there is no unanimity whether it reflects the precise nature of a syndrome containing both neurologic and psychiatric components.

By the late-19th century, psychoanalytic theory ruled medical reasoning about these symptoms. Originally referring to these disorders as hysteria, neuropsychiatrists began illustrating the various clinical phenomenological aspects of such disorders. Paralysis, tremors, convulsions and sensory alterations were identified as sometimes being due to hysteria. Subsequently, different etiologies of dystonia, tremor, myoclonus and other movement disorders were recognized. Over the years, newer clinical criteria, laboratory investigations, particularly neurophysiological findings, and improved neuroimaging have provided significant insights about the psychogenicity of the diagnosis. However, a misdiagnosis is possible either on patients originally believed to have a conversion disorder or because PMD was never considered on differential diagnosis (Rosebush & Mazurek, 2006; Lang & Gupta, 2009).

The pathophysiology of PMDs are not yet well known, but functional brain imaging studies combined with other neurophysiologic techniques are starting to help understand them (Stone & Carson, 2011). These studies promise an understanding of these symptoms in parallel neurologic and psychiatric ways.

The diagnosis of a psychogenic movement disorder is often difficult and the level of diagnostics that the clinician has for PMD varies remarkably, depending on the clinical feature of the movement disorders and the accompanying signs and symptoms. PMDs are

classified by the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV) of the American Psychiatric Association as conversion disorder of motor subtype and must be differentiated from factitious disorder and malingering, in which the abnormal movements are purposefully forged. Since most patients with conversion symptoms are found to have “no psychiatric disease” by the psychiatric and “no neurologic disease” by the neurologist, a multidisciplinary treatment approach, including the movement disorders neurologist, the consulting psychiatrist, and frequently a physical therapist, is crucial in obtaining symptom remission in many subjects (Williams et al., 2005; Stone et al., 2010).

Although evidence for treatment of PMDs are lacking and is mainly based on case control, retrospective, or case report studies, the therapeutic process starts with the diagnosis and the explanation of the symptoms to the patient. To date, the treatment for each patient with PMDs is individualized and may include psychotherapeutic interventions, behavioral therapy, hypnosis, pharmacotherapy, physical therapy, and other approach.

Physicians should not underestimate the importance of distress and disability that subjects with these symptoms suffer. Failure to diagnose a PMDs inevitably delays treatment and may perpetuate a patient’s situation of disability (Williams et al., 2005). In addition, patients with somatisation had approximately twice the medical care utilisation and twice the annual medical care costs of non-somatizing patients and spend 1.3 to 3.9 days in bed per month compared to an average of one day or less for patients with major medical problems (Smith et al., 1986; Barsky et al., 2005). The current chapter reviews empirical evidence concerning clinical manifestations of PMDs and summarizes how PMDs are currently diagnosed, investigated and treated.

2. Epidemiology and risk factors

Although psychogenic neurological symptoms are common and account for 1–9% of neurological symptoms observed in the general population (Lempert et al., 1990; Factor et al., 1995), the following caveats should be borne in mind: (i) available reports result from tertiary movement disorders centers, and therefore it is difficult to valuate how common these disorders are in the general population or even what is the prevalence respect to all conversion disorders, (ii) the insufficiency of confirmatory diagnostic testing prejudices case definition even when clinical satisfactory criteria are applied and (iii) several clinical settings and situations (e.g., neurology or psychiatry in- or outpatient service, emergency room or general practitioner cases, chronic or refractory cases, etc.) may confound case ascertainment (Lang, 2006). In the only series dividing medically unexplained motor symptoms into “absence of motor function” and “presence of abnormal motor activity”, 48% of the patients had index symptoms in the former category, while 52% had symptoms, such as tremor, dystonia or ataxia (Crimlisk et al., 1998).

PMDs also can occur over a wide range of ages from teenage to the mid seventies (Deuschl et al., 1998; Feinstein et al., 2001). The mean age at onset described in several case series on these disorders ranges between 37 years and 50 years and women are predominantly affected with a range of 61–87% (Hinson et al., 2005; Factor et al., 1995).

There are no data on racial distribution in the published research, however a trans-cultural comparison between patients with these disorders in the USA, in Spain, and in Brazil showed essentially similar demographic and clinical characteristics by ethnic origin (Cubo et al., 2005; Munhoz et al., 2010). Nearly 15% of patients with a psychogenic movement disorder have also an underlying organic movement disorder (Ranawaya et al., 1990).

Curiously, PMDs are frequently seen in subjects employed in health care professions or allied health care professionals possibly due to the exposure to disease and those who have witnessed the organic form of the movement disorder (e.g. Parkinson's disease) in other relatives (Miyasaki et al., 2003;)

More frequently, PMDs are encountered in the context of a second coexisting psychiatric illness. Feinstein and colleagues followed 88 patients suffering from psychogenic movement disorders, over an average of 3 years. 42 patients agreed to undergo a structured psychiatric interview. The most common life time prevalence rates were found for depression (42%), anxiety disorders (62%), a combination of depression and anxiety (29%) and conversion disorder (95%). Personality disorder (antisocial, borderline, dependent, avoidant or a mixture of those) was diagnosed in 42% of patients tested (Feinstein et al., 2001). Among the a series of 127 patients with psychogenic tremor, depression (51%) and anxiety (31%) were the most common psychiatric co-morbidities (Jankovic & Thomas, 2006).

Risk factors for these disorders include: history of sexual abuse or rape, physical trauma, previous surgery, major stressful life experiences (Williams et al., 2005; Feinstein et al., 2001). Many patients with functional neurologic symptoms report just as much physical disability and are more distressed than patients with neurologic disease. Furthermore, subjects with these symptoms are more likely to be out of work because of ill health than the general population (Stone et al., 2011).

3. Pathophysiology

In reviewing the history of psychogenic neurologic disorders, hysterical paralysis and sensory loss have been known presumably back in antiquity (Ng BY, 1999) and have been debated before the late 19th century, when Charcot, Janet, Breuer, and Freud gave a systematic psychological account of these phenomena. (Charcot, 1889; Janet, 1907; Freud, 1910). From then on, there has been a large body of literature regarding the possible psychogenic, psychoanalytical, cultural and biological mechanisms underlying PMDs.

According to Janet, traumatic events can cause a functional separation (dissociation) of structures of memory, identity, insight, and perception of the environment from conscious awareness. As a result, unexplained symptoms emerge from the activation of these dissociated structures (Janet, 1889). Patients suffering from non-organic motor symptoms are singularly susceptible to hypnosis, an inducible state of dissociation.

This theory was later developed by Breuer and Freud, who considered dissociation a psychological defence mechanism rather than a disorganizing phenomenon: the mental conflict is partially or completely resolved by the expression of physical symptoms, which is called primary gain. According to this psychodynamic model, dissociation preserves the subject from the invalidating affect associated with remembrances of trauma by its "conversion" in to somatic symptoms and once these latter have developed, may confer further advantages to the patient (attention, solicitude, social interaction, etc.) or "secondary gains" (Breuer & Freud, 1895).

In contemporary psychiatry, dissociative symptoms and dissociation as a mechanism are taken to point to a role traumatic events in pathogenesis. Various factors may provide to the pathway to conversion disorder, perhaps comprehending, concurrent somatic illness in adolescence or adulthood, parental illness in the subject childhood, and early illness. In particular physical or sexual abuse in childhood have considered key factors in the generation of vulnerability to unexplained somatic symptoms (Ovsiew, 2006).

The definite nature of emotional disorders responsible of psychogenic disorders, and their functional consequences on neural systems in the brain, still remain largely unknown.

In the closing decades of the 19th century, researchers have progressively been looking for organic correlates of PMDs and the neural mechanism perspective became more approved with the advent of novel functional neuroimaging methods. These new approaches have now allowed to detect in vivo regionally specific changes in cerebral blood flow and task-related changes in the attempt to identify specific neural correlates associated with conversion symptoms, that is the “dynamic lesion” that Charcot considered responsible for the neurological signs he observed in patients affected by “hysterical movement disorders”.

PMDs evoke notable interest because, as protean disorders of willed action or intention, the underlying mechanism are supposed to be the result of unconscious processes, a sort of impairment to the volition system, once any demonstrative organic or feigning dysfunctions has been excluded (Fink, 2006). Psychogenic movements may be voluntary or involuntary. Factitious disorder and malingering describe when the disorder is voluntary, and the patients are lying. On the other hand, most patients with PMDs have a conversion aetiology and manifest movements that look voluntary, even if patients declare that the movements are involuntary. By a physiological point of view we can not tell the difference between voluntary and involuntary, but we know (most of time) that both are preceded by a normal “readiness potential” or so called *Bereitschaftspotential*, a manifestation of cortical contribution to the pre-motor planning of volitional movement (Shibasaki & Hallet, 2006) and share cortical structures that are involved in movement planning and execution.

In a go-nogo task, while a patient underwent functional magnetic resonance imaging (fMRI), Cojan et al. demonstrated that distinct inhibitory mechanism are implicated in simulation and conversion disorders and that conversion symptoms do not act through cognitive inhibitory circuits, but involve selective activations in midline brain regions associated with self-related representations and emotion regulation (Cojan, 2009). Other neuroimaging results have shown increase activation in limbic regions, such as orbitofrontal or cingulate cortex during conversion symptoms affecting different motor or sensory modalities (Vuilleumier, 2005, Nowak, 2009). A recent single photon emission computed tomography (SPECT) study by Czarnecki et al. suggested that the prominent hypoperfusion of the prefrontal and anterior cingulate cortex in patients with psychogenic tremor likely indicates deactivation of the anterior portion of the default mode network (the baseline state of the brain that deactivates during goal-directed activity), which could prove to be a peculiar marker of PMDs. Moreover, this study reported resting hyperperfusion in left insula and left inferior frontal gyrus in psychogenic tremor that may also prove to be a disease characteristic (Czarnecki et al. 2011).

Voon and colleagues used functional magnetic resonance imaging (fMRI) to study patients with psychogenic tremor who could voluntarily mimic their tremor and showed hypoperfusion of the right temporal parietal junction only during involuntary movements. The authors speculate that this hypoactivity may reflect the lack of an appropriate sensory prediction signal, being the right temporal parietal junction implicated as a general comparator of internal predictions with actual events. This suggests a loss of self-agency awareness that made the movements feel involuntary (Voon et al., 2010).

There may be a pathologic unconscious influence on movement production associated with a disconnection between movement production and sense of volition (Hallet, 2010). Besides, earliest affective or stress-related factors, neuropsychological and psychosocial processes, perhaps involved primitive reflexive mechanisms of protection and alertness that are not

fully independent of conscious control (Vuilleumier, 2005). This prominent evidence in favour of multi causes of PMDs requires a multifaceted approach integrating innovative neuroimaging and neurophysiological techniques with social, psychological and psychodynamic theories.

4. Clinical manifestations and diagnostic clues

The diagnosis of PMDs remains a fascinating and challenging dilemma in both clinical neurology and psychiatry. It should not be considered as a diagnosis of exclusion but should be established on positive clinical criteria to determine whether abnormal movements are produced by organic disease, psychiatric disorder, or both (Jankovic & Thomas, 2006). Taking into account that unnecessary investigations should be prevented, more notable evidence is required before a diagnosis of psychogenic disorder can be confirmed. Some studies, including exhaustive neurologic assessments and modern diagnostic techniques, have shown that a misdiagnosis is possible on long-term follow-up of patients initially diagnosed with a conversion disorder and later identified as having an organic disorder (Moene et al., 2000; Lang, 2006; Lang & Gupta, 2009). Otherwise, failure to make the diagnosis arises because PMDs were seldom contemplated in the differential diagnosis, especially in patients who have a coexistent neurologic disease, such as neurodegenerative or demyelinating disorder or epilepsy. Coexistent organic neurologic disease was present in 37% of patients with psychogenic tremor followed for over 3 years (Jankovic et al., 2004). Additionally, the problem is exacerbated by a tendency among physicians to be concerned about missing an “organic” diagnosis in order to relieve the patient from a “functional” one, even if the latter is treatable and the former is not (Rosebush & Mazurek, 2006). Another troubling side of these disorders is the reluctance of many physicians to put their judgments and conclusions into a transparent discussion with the patient. So an ambivalent communication wanes to deliver the presumed diagnosis in real terms and running the risk that patients continue to explore for months or years further opinion through “doctor shopping”. (Friedman & LaFrance WC, 2010).

Because many patients or family members of patients with PMDs are strongly reluctant to the diagnosis not explained organically and may resistant to a psychiatric referral, a multidisciplinary approach, including the general or movement disorders neurologist and the consulting psychiatrist is essential. The role of the neurologist is primary in determining whether there is an underline neurologic disorder and whether it could explain the clinical picture. Hardly mental health professionals undertake a treatment of such patients without the neurologic diagnosis has been either established or dismissed (Feinstein et al., 2001; Williams et al., 2005).

Fahn and Williams developed four degrees of certainty for the diagnosis (Tab. 1) of psychogenic dystonia, which are commonly applied in clinical practice and research to all PMDs (Fahn & Williams, 1988). Shill and Gerber formulated further criteria with a denomination of “clinically proven PMDs” which requires remission when the patient is unobserved or with psychotherapy or when there is a Bereitschaftspotential on electroencephalography (for myoclonus only). Moreover, they added further criteria of PMDs to include excessive pain or fatigue and previous disease exposure (Shill & Gerber, 2006). Developing this idea, Hallett proposed that a new designation of “laboratory proven PMDs” could be considered (Peckham & Hallett, 2009). A recent study demonstrated that

the finger tapping test may provide an objective tool to aid the clinical diagnostic criteria set by Fahn and Williams for identifying patients with PMDs (Criswell et al., 2010).

Documented psychogenic

Movements are persistently relieved by psychotherapy or psychological suggestion or with the administration of placebos. If the patient is observed to be symptom free when left alone, this may also be documented as psychogenic; however, this feature is usually indicative of malingering or factitious disorder.

Clinically Established Psychogenic

Inconsistent or incongruent with classical dystonia (on examination, the patient is unable to move the limbs but is able to dress herself in daily life). In addition, one or all of the following is highly suggestive: other neurologic signs present that are psychogenic (self-inflicted injuries, false weakness, false sensory findings), an obvious psychiatric disturbance is present, and multiple somatizations are present.

Probable Psychogenic

Movements are inconsistent or incongruent, but there are no other features (as above) to further support the diagnosis. Movements are consistent with organic dystonia, but there are other features on examination to suggest psychogenicity (self-inflicted injuries, false weakness, false sensory findings). Multiple somatizations are present, but movements are consistent with organic dystonia.

Possible Psychogenic

An obvious emotional disturbance is present, but movements are consistent with organic dystonia.

Table 1. The classification of psychogenic movement disorders (Williams & Fahn, 1995)

An additional support, that seems appropriate to capture the complexity of PMDs and that can be used to assess PMDs and test the efficacy of intervention strategies is the Psychogenic Movement Disorder Rating Scale (PMDRS). This clinimetric assessment describes and quantifies the complicated phenomenology of PMDs, and provides the following six types of information: movement phenomenology, anatomic distribution and severity of abnormal movements, duration of abnormal movements, assessment of two functions (gait and speech), impairment-based incapacitation by abnormal movement or function, and total severity score (Hinson et al., 2005).

It should be emphasized that observation and examination are the most important tools for the physician looking for inconsistency of movements. The first trace of psychogenicity in a patient presenting with such abnormal motor activity can be obtained by history (Table 2.A). This may comprise psychiatric history, childhood history, personality factors, drug experience, recent personal and family life events, stressful situations or work-related injury, litigation or compensation pending, personal encounter or knowledge of similar disorders serving as a “model” and possible secondary gain (Bhatia & Schneider, 2007; Nowak & Fink, 2009).

In general, the manner of onset characterizes the clinical presentation of PMDs (Table 2.B): symptoms appear abruptly, frequently in the context of precipitating factors and, the highest disability and severity are reached quickly (Feinstein et al., 2001). Important specifics of PMDs are an inconsistent character of movement (unusual presentation in amplitude, frequency, distribution), and they may increase with attention or decrease with distraction (Miyasaki, 2003). A deliberate slowness of movement is incongruent with an

organic movement disorders, as well as simultaneous occurrence of variegated abnormal movements and disfunctions, and peculiarly, patients may seem to struggle and put in more effort than needed to complete the task (Hinson & Blacke Haren 2006; Bhatia & Schneider 2007). Often, this is manifest by sighing, grimacing, and using their whole body to do a movement. The movements themselves may appear bizarre and should be incongruous with a known movement disorder.

There are controversial points of view whether there is a place for placebo in management of PMDs, as it reflects ethical evaluations and can infringe the relationship between physician and patient. Although a response to placebo of a movement disorder is seriously supportive of a diagnosis of PMDs (Espay et al., 2009). Equally, spontaneous resolution and improvement of unexplained symptoms with psychiatric evaluation or psychotherapy are highly suggestive of psychogenic aetiology of them (Fahn, 1994).

Diagnostic testing should be used primarily to give further support to the underlying clinical suspicion that it is psychogenic. Routine blood test including haematology, thyroid function, renal and liver function, and evaluation for Wilson's disease may be helpful. Magnetic resonance imaging can be helpful for excluding an underlying structural, vascular or demyelinating lesion, particularly if the abnormal movement is unilateral or asymmetrical. Neurophysiology studies to evaluate tremor and myoclonus can aid in the diagnosis of PMDs. Dopamine transporter (DAT) SPECT and Fluorodopa (18F-dopa) PET scans have been proven quite helpful in distinguishing psychogenic parkinsonism from Parkinson's disease or essential tremor (Kagi et al., 2010; Czarnecki et al. 2011).

The role of the consulting psychiatrist is to interpret the psychopathology present, ascertain its relevance to the presenting PMDs symptoms and establish a positive rapport with the patient. If this appears feasible, the psychiatrist will then begin the treatment course, with adequate collaborative support from neurologist (Williams et al., 2005).

However, psychiatric aspects and categorical differentiations, which are discussed in detail elsewhere in this book, also apply to PMDs. In brief, the psychiatric examination includes research of individual psychodynamics and significant environmental events as well as a complete multiaxial delineation of specific psychopathology according to the official psychiatric classification system such as the Diagnostic and Statistical Manual of Mental Disorders, fourth Edition (DSM-IV). The primary psychopathology underlying PMDs can be divided into two categorical diagnostic subgroups: somatoform disorders on the one hand, and factitious disorders and malingering on the other. The first category includes conversion disorder and somatization disorder. Conversion disorder is likely the most common mechanism of PMDs and is defined by the DSM-IV criteria as a disorder including one or more symptoms, that are not the result of a neurological disorder, affecting voluntary motor or sensory function that suggest a medical condition and is associated with psychological factors.

The primary psychiatric diagnosis varies: most cases are considered to be conversion disorders, in which the problem is generated by an unconscious mechanism, but infrequently some are factitious disorders or malingering, in which the abnormal movements are purposefully forged. Factitious disorders include intentional production of physical or psychological symptoms, where the goal is to assume the role of a patient and external incentives, such as economic gain or avoiding legal responsibility, are absent. In malingering, the symptoms can also be physical or psychological, but the individual is consciously aware of external pragmatic incentives, such as gaining financial compensation, acquiring drugs, avoiding work or school, et cetera, and when the external incentives are

removed, the symptoms resolve. Beyond a categorical diagnostic classification, it is often very intricate, especially initially, to face with a patient having PMDs and make a differential diagnosis among somatoform disorders, factitious disorders, and malingering. Simplification may arise however, in the course further and exhaustive evaluation, by reinforcing the agreement between physician and patient and ensuring patient's confidence in the treatment plan (Williams et al., 2006). Moreover, most patients with PMDs have a coexisting variety of different psychiatric disturbances such as dysthymia, major depression, anxiety, adjustment disorders, obsessive-compulsive disorder, panic attacks, bipolar disorders and others (Williams et al., 2005; Bhatia & Schneider, 2007). Yet, it is important to add that many organic movement disorders have an important incidence of the above-named psychiatric diagnoses (Reich, 2006). Finally, the common occurrence of movement disorders complicating primary mental illness and their treatment makes it important for psychiatrists to be able to recognize the various movement disorders, some of which have singular phenomenology such as tardive akathisia, tardive dystonia, tourettism and some unusual form of parkinsonism or tremor (Factor et al., 2005).

A) Historical

1. **Abrupt onset**
2. **Static course**
3. **Spontaneous remission**
4. **Precipitated by minor trauma**
5. **Obvious psychiatric disturbance**
6. **Multiple somatization**
7. **Employed in health profession**
8. **Pending litigation or compensation**
9. **Presence of secondary gain**
10. **Young age (female>male) Inconsistent character of movement (amplitude, frequency, distribution, selective ability)**

B) Clinical

1. **Paroxysmal movement disorder**
2. **Movements increase with attention or decrease with distraction**
3. **Ability to trigger or relieve the abnormal movements with unusual or non physiological interventions (e.g. trigger points on the body, tuning fork)**
4. **False weakness**
5. **False sensory complaints**
6. **Self-inflicted injuries**
7. **Deliberate slowness of movements**
8. **Functional disability out of proportion to exam findings**
9. **Movement abnormality that is bizarre, multiple or difficult to classify Unresponsive to appropriate medications**

C) Therapeutic responses

1. **Response to placebos**
2. **Unresponsive to appropriate medication**
3. **Remission with psychotherapy**

Table 2. General clues suggesting that a movement disorder may be psychogenic (Miyasaki et al., 2003; Lang, 2006)

4.1 Types of psychogenic movement disorders

4.1.1 Psychogenic tremor

Although many patients has a mixture of different movement disorders, psychogenic tremor is the prevalent movement disorder, up to 55%, of all PMDs. Clinical sites affected include the hand (84%), the leg (28%), and generalized body tremor 20% (Thomas & Jankovic 2004). Clinically, absence of finger tremor can be a positive diagnostic sign for psychogenic tremor (Jankovic & Thomas, 2006; Bhatia & Schneider, 2007) and attempting to immobilize the affected limb often makes a functional tremor worse, as well as loading the limb with weights tends to make the tremor worse, whereas organic tremor usually improves with this operation.

The diagnosis may be supported by the “coactivation sign”. As in the testing procedure for rigidity, the physician feels the increased muscle tone in the tremulous extremity in both directions and this cogwheel-like resistance is strictly related to tremor or if the patient can be made to relax completely (Deuschl et al. 1998). The technique of back-averaging electroencephalographic activity preceding the electromyographic ones, can be useful to detect premovement potential in subjects with psychogenic tremor, absent in organic involuntary movement (Brown & Thompson, 2001). Zeuner et al. using accelerometry to measure frequency changes during tapping showed that in contrast to parkinsonian and essential tremor, patients with psychogenic tremor revealed larger tremor frequency changes and marked variability in tapping (Zeuner et al., 2003). Entrainment of tremulous movements of different body parts into a single rhythm has been used clinically as a means of distinguishing these tremor forms. If functional tremor involves more than one limb, it usually has the same frequency. On the other hand, organic tremor usually has slightly different frequencies in different body parts. A quantified electrophysiological entrainment test performed on accelerometer or surface EMG tremor signals may provide supportive evidence of a functional tremor (McAuley & Rothwell 2004). Recently, Czarnecki and colleagues revealed distinct patterns of cerebral perfusion, during rest and motor task, as measured by SPECT that distinguish psychogenic tremor from essential tremor and controls (Czarnecki et al. 2011).

4.1.2 Psychogenic dystonia

Dystonia exemplifies one of the longest history of misdiagnosis: for many centuries it was considered a psychogenic condition, then, after torsion dystonia was accepted as an organic entity in the early-20th century and different aetiologies of this condition were recognized, it was thought that psychogenic dystonia rarely occurred (Fahn, 2006). In general, psychogenic dystonia represents only approximately 5% of subjects with dystonia, but in most centers, it is the second most commonly encountered among PMDs and accounts for 20% to 50% of cases (Miyasaki et al., 2003). Psychogenic dystonia, classified as a secondary dystonia, largely remains a clinical diagnosis and there are no physiologic tests available that distinguish a psychogenic aetiology from an organic form (Peckham & Hallett, 2009).

Psychogenic dystonia may not occur with the typical variability and distractibility of other PMDs and presents with fixed dystonic postures (Fig. 1) without return to the neutral position at rest from the beginning. Leg involvement is uncommon in adult-onset organic dystonia, as well as lack sensory tricks or relief by certain inexplicable trick action, and presence of severe pain suggest psychogenic dystonia, even if not in a specific way (Schrag, 2006).

Controversial patients are those with dystonia developing within hours or days after a minor injury, with a fixed dystonic posture and severe pain. This kind of dystonia may be associated with features of complex regional pain syndrome type I. A study by Schrag and co-workers show that a substantial proportion of patients with fixed dystonia clearly fulfils criteria for a psychogenic dystonia (37%) or somatization disorder (29%). Although fixed dystonia sometimes developed in patients in whom a diagnosis of somatization disorder had already been made, a history of somatization was often unrecognized and, in many cases, only became evident after examination of primary care records (Schrag et al., 2004; Miyasaki et al., 2003). Various features of complex regional pain syndrome-related fixed dystonia suggest abnormal regulation of inhibitory interneuronal mechanisms at the brainstem or spinal cord level and impairment central synaptic reorganisation due to an interaction between neuroplastic activities and anomalous environmental necessities (Munts & Koehler, 2010).



Fig. 1. A fixed dystonic posture of psychogenic origin

4.1.3 Psychogenic parkinsonism

Psychogenic parkinsonism is a rare syndrome accounting for 0.17–0.5% of all parkinsonism cases and representing nearly 10% of PMDs (Factor et al., 1995; Benaderette et al., 2006). In this disorder, atypical tremor occurs in conjunction with extremely slow movements that are often accompanied by grimacing, sighing, or whole-body movements when patients do a simple motor task. Common characteristics of organic parkinsonism, such as hypomimia, decreased blink rate, axial rigidity, and “cogwheel” phenomenon are usually absent in psychogenic parkinsonism. On postural stability testing, patients may have bizarre responses including flailing of the arms and reeling backward without falling (Thomas & Jankovic, 2004). Additionally, patients with psychogenic parkinsonism may also suffer from depression, which can cause psychomotor retardation, a clinical condition which may be difficult to distinguish from the bradyphrenia associated to Parkinson’s disease (Morgan &

Sethi, 2006). It should be also remarked that parkinsonism or akinetic-rigid syndrome not uncommonly occur in the setting of major psychiatric disease and exposure to pharmacological agents.

Electrophysiology studies can be supportive in distinguishing a Parkinson's psychogenic tremor from other forms of tremor. Functional neuroimaging can be helpful in confirming a diagnosis of psychogenic parkinsonism. Loss of dopamine nerve function seen in organic parkinsonism can be measured by decreases in dopamine transporter density or presynaptic dopamine deficiency (I 123 B-CIT) on single positron emission computed tomography (SPECT) and Fluorodopa positron emission tomography (F-DOPA-PET). In psychogenic parkinsonism, these features are absent, but keeping in mind that other conditions, i.e. drug induced parkinsonism, dopa responsive dystonia-parkinsonism, have normal SPECT or F-DOPA-PET scans (Benaderette et al., 2006; Scherfler et al., 2006).

Table 3 summarizes the differences of clinical findings in Psychogenic parkinsonism, Drug-induced parkinsonism and Parkinson Disease.

	Psychogenic Parkinsonism	Drug-Induced Parkinsonism	Parkinson Disease
<i>Onset</i>	Abrupt, varied age of onset	Bilateral and symmetric, more common in the elderly	Gradual, unilateral or asymmetric, typically in the 6 th or 7 th decade
<i>Cours</i>	Usually static with maximum disability early, condition may abruptly or gradually remit	Acute or subacute	Insidious, slowly progressive
<i>Tremor type</i>	Unilateral or bilateral, rest, postural, action; usually involving the dominant hand, varying frequency and amplitude, spreads when immobilizing the affected limb	Not always present, bilateral, symmetric, postural or rest	Unilateral or asymmetric at rest, 4-6 Hz, worsens with distraction
<i>Bradykinesia</i>	Extremely slow movement often with fatigue, arrest or decrement; grimacing, sighing or whole body movements when performing simple task; normal speed of movements when not being examined	Often the earliest and commonest manifestation, facial hypomimia	Slowing of rapid repetitive movements without fatigue, arrest or decrement

<i>Rigidity</i>	Cogwheel absent, voluntary resistance which may decrease with distraction	Often uniform, inconstant cogwheel rigidity	Cogwheel rigidity
<i>Postural instability</i>	Impaired early, may have exaggerated or bizarre response to minimal backwards	Mild stooping, decreased arm swing	Impaired in moderate to advanced disease, retropulsion on pull test
<i>Speech</i>	Stuttering, bizarre dysarthria, distractible	Mild stuttering	Hypophonic, stuttering, tachyphemia
<i>Motor fluctuation</i>	Very rare complaint of “extra movements” on levodopa, no dyskinesias after long-term levodopa treatment	Absent	Dyskinesias in half of patients on levodopa after 5-7 years, on-off fluctuations
<i>Psychiatric history</i>	Previous conversion disorders, somatisation, factitious disorder, anxiety, and depression	Relevant, particularly schizophrenia but also depression	Depression may precede diagnosis
<i>Medication response</i>	Usually unresponsive to multiple medical trials	Responds well to anticholinergic drugs, remittance within weeks or months when withdrawal of offending drug	Responds well to levodopa and dopamine agonist
<i>Nonmotor problems</i>	Sexual dysfunction and sleep disturbances occur	Possible swallowing less than normal, hallucination, rare constipation	Dysautonomia, constipation, sexual dysfunction, sleep problem, hallucination

Table 3. Clinical features of Psychogenic parkinsonism, Drug-induced parkinsonism and Parkinson Disease. Adapted from Morgan & Sethi, 2006.

4.1.4 Psychogenic gait

Abnormal gaits frequently occur in the setting of major psychiatric disease and represent 8% to 10% of all PMDs (Sudarsky, 2006). Psychogenic gait disorders are characterized by

exaggerated effort or fatigue with grimaces, excessive slowness, convulsive shaking, often with knee buckling especially when the patient has unilateral functional weakness, astasia-abasia, arms are outstretched like a tightrope walker (Fig. 2), unusual uneconomic posture and bizarre movements (Bhatia, 2001; Baik & Lang, 2007). Patients with psychogenic require further strength and balance than an indifferent gait and they seem to be frightened of falling, and this gait allows them to be closer to the floor (Stone & Carson, 2010). The movement disorder is commonly accompanied by other psychogenic neurological symptoms, such as false weakness or sensory findings, or by excessive pain and tenderness (Thomas & Jankovic, 2004). Okun et al. described 9 consecutive patients who presented with a psychogenic gait disorder who underwent "chair testing." Each patient was asked to walk 20-30 feet forward and backward toward the examiner. Patients were then asked to sit in a swivel chair with wheels and to propel the chair forward and backward. Compared with their walking, 8 of the 9 patients in the psychogenic group performed well on the chair test, showing improved ability to propel a chair forward when seated. By contrast, all 9 control patients with nonpsychogenic gait problems, performed equally when walking or propelling utilizing the chair (Okun et al., 2007).



Fig. 2. Tightrope walking: the patient walks very slowly on a broad base with his arms extended.

4.1.5 Psychogenic myoclonus

Myoclonus account for 10% to 20% of PMDs. The clinical features of psychogenic myoclonus usually includes segmental or generalizes jerking, occurring at rest and during movement, commonly changing pattern, frequency, amplitude and anatomic distribution and may be stimulus sensitive (Monday & Jankovic 1993; Williams et al., 1995).

Neurophysiologic methods are particularly useful in distinguishing between voluntary jerking and cortical or brainstem myoclonus (Brown & Thomson, 2001). Organic myoclonus is characterized by burst length of less than 70 ms, and jerks lasting longer than that are suggestive of a psychogenic etiology. Functional myoclonus is often associated with a Bereitschaftspotential before the movement, which requires recording multiple events using an electroencephalogram and back averaging according to an electromyogram. In the absence of a Bereitschaftspotential, it is not possible to exclude a psychogenic etiology, as the Bereitschaftspotential can be absent in normal subjects (Brown & Thomson, 2001; Peckham & Hallett, 2009). Interestingly, in a recent case series of 35 consecutive patients with jerks of the trunk referred as possible prorioisinal myoclonus, 34 patients showed features suggestive of a psychogenic origin even in the presence of a classic polymyography pattern or in the absence of a Bereitschaftspotential (van der Salm et al., 2010).

5. Treatment

There is no consensus even among the experts about the best treatment approach to patients with PMDs. Therefore, a common agreement is that treatment begins when the physician has made the diagnosis and mostly depending on the way of explaining PMDs to the patient, as well as a very close working relationship between neurologist, consulting psychiatrist, and frequently physical therapist, is crucial in obtaining symptom remission in many subjects. Table 4 emphasizes, by the acronym form THERAPIST, the essential basis for a treatment process.

Terminology must engage and not alienate the patient
Hear out the patient with interest, compassion, and empathy (and patience)
Explain the diagnosis and the mechanism of symptoms
Reassure that there is no evidence of neurologic damage
Address psychosocial and family issues
Prognosis is likely favorable, the patient has the potential to recovery fully
Individualize the therapy and customize it
Self-help is a crucial part of getting better
Treat concurrent psychiatric and medical illness (if present)

Table 4. Nine essential steps for an approach to management of patients with PMDs.

The objective of effective treatment is not only to provide symptoms remission in the short term, but to evaluate the causes that produced the heterogeneous symptomatology and to assess feasible strategies to remove them.

The issue about the terminology to use for the diagnosis is unresolved. Some authors find that “functional” disorder is an accurate term, which describe a disorder of the way the brain is working. Others find “psychogenic” an acceptable term with vagueness implication. Still others think that the latter arouses too much a “crazy” condition in the patient and family’s mind and prefer the more broad word “neuropsychiatric” (Williams et al., 2005; Stone & Carson, 2010). In any case, whatever term is used it is important to find an explanatory language that engages the patient and gives a scenario within which to understand the disorder. In this regard, a self-rating approach reported that 49% of patients

attributed a favorable outcome to a physician's described treatment (Jankovic & Thomas, 2006).

It is very important to reassure the patient early on, for example emphasizing that this is an "involuntary" condition and is most likely the result of an impairment of neural pathways. Another option is to explain that some of the symptoms are stress-related symptoms, pointing out that stress is a common cause of many physical afflictions. A sincere, supportive, hopeful and, professional manner of approach will allow to understand and, at the same time, have patients understand what the movement disorder means, what is its functions, why and when it evolved. Some experts and the authors also suggest an active physiotherapy program, from the beginning, in order to desensitized the stress-induced contraction that generate the anomalous muscle jerks. Overall, this aspect of the treatment may corroborate the "physical" dimension of the disorder and may allow decreasing the symptom without activating psychological defence mechanisms (Williams et al., 2006; Rosebush & Mazurek, 2006). Besides, the physiotherapist frequently recognizes the fears and unhelpful preconceptions which patients have, and can stimulate and compliment patients in their activity in a much more confidential mode than the allied psychotherapist.

Medication treatment can be initiated and the choice of a particular drug depends on the accompanying psychiatric or medical conditions. In authors experience these most frequently include anxiety, depression, insomnia and headache, and a low dose of tricyclic antidepressants or benzodiazepine can help with symptoms of pain and muscle tension.

Large randomized studies in patients with PMDs are lacking, and evidence for treatment is largely based on retrospective, case control, and case report studies. Several other clinical trials, not specifically designed for PMDs but for other forms of conversion or somatoform disorders, are treated more in detail elsewhere in this book.

In a study by Voon and co-workers, 23 patients were identified with PMDs, and 15 patients agreed to be treated with antidepressant drugs. Of the 15 patients, 10 were diagnosed with primary PMDs, and the remaining 5 were diagnosed with PMDs and another somatoform disorder. Patients were treated with either citalopram or paroxetine. Those who did not respond were switched to venlafaxine. Of the primary PMDs patients, 80% (8 patients) had marked improvement, and 7 patients had complete remission. None of the 5 patients with PMDs and other somatoform disorders improved. (Voon, & Lang, 2005).

An open-label trial of somatisation disorder studied the efficacy of nefazodone in patients with and without comorbid depression and showed improvement in clinical global impression and functioning in 73% of patients (Menza et al., 2001).

In a study by Rampello and colleagues, 18 patients were treated (6 with haloperidol and 12 with sulpiride). The latter group showed remarkable improvement in 8 patients, partial improvement in 2 patients, and no improvement in 1 patient. The haloperidol group showed 1 patient with remarkable improvement, 3 with partial improvement, and 2 with no improvement. This study showed a possible positive correlation between dopamine blockade, drug-induced plasma prolactin concentration, and improvement in a patient's conversion symptoms.

Hinson and colleagues recruited ten patients with PMDs for a single-blind clinical trial to receive 12 weeks of treatment with outpatient psychodynamic psychotherapy and use of antidepressants or anxiolytic drugs, depending on comorbid psychiatric diagnosis. The movement disorder was videotaped before and after treatment and rated in a random order by a rater unaware of treatment allocation using PMDRS. All patients were diagnosed with conversion disorder. Nine of ten recruited patients completed the study. Total mean PMDRS

and total mean PMDRS function scores improved with psychotherapeutic intervention. There were significant treatment effects in Hamilton depression scores, Beck anxiety scores, and global assessment of function.

Shapiro and Teasell described a case series of 39 consecutive patients with conversion disorder who were told that they had a musculoskeletal problem that could resolve completely if they had an organic etiology. If the patients did not improve after 4 weeks, then they were told that it was a psychiatric condition, and the treatment would be modified to help them improve completely. If they did not improve, then they were given a final diagnosis of conversion disorder, and they were told that they could not improve because of an unconscious need to remain disabled. In 8 of 9 patients with acute conversion disorder (symptoms <2 months), the treatment was successful. In 1 of 28 chronic (>6 months duration) patients, behavioral treatment was successful (Shapiro & Teasell, 2004). In a randomised controlled clinical trial, Moene and colleagues assigned 48 patients to receive either hypnosis or a control intervention consisting of generic elements of psychotherapy. Outcome measures were a video rating scale for motor conversion symptoms, the symptom checklist-90, and elements of the international classification of impairments, disabilities, and handicaps. Independent of the treatment condition, 65% of patients showed substantial improvement at post-treatment assessment and 84% at 6-month follow-up, which suggests that both psychotherapy and hypnosis have a role in the treatment of conversion disorder.

A meta-analysis of studies of cognitive behavioral therapy for various somatisation syndromes showed a definite or possible treatment effect of cognitive behavioral therapy in 71 of patients (Kroenke et al., 2000). Randomized controlled studies support the efficacy of individual cognitive behavioral therapy for the treatment of hypochondriasis, body dysmorphic disorder, and undifferentiated somatoform disorders including medically unexplained symptoms, chronic fatigue syndrome, and noncardiac chest pain (Looper & Kirmayer, 2002; Allen et al., 2006).

In a single-blind study, 16 patients with PMDs completed a thrice-weekly, 12-weeks mild walking program. Assessments included DSM-IV interview, PMDRS, Beck Anxiety Inventory, Hamilton Depression Scale, V02 Max, and body mass index. A comparison of all measures taken at study onset and after completing the exercise program indicates statistically significant improvements. We observed a relevant improvement in 10 of 16 patients (62%). The mean difference for the primary outcome (PMDRS total) corresponded to about 70%. Compliance was good, and there were no adverse effects. This study provides preliminary evidence for regular low-medium intensity exercise as a safe, adequate, and pleasing intervention for PMD. (Dallocchio et al., 2010).

A retrospective study was performed in 10 patients with psychogenic gait. Patients were treated with physical therapy, occupational therapy, and recreational therapy, and psychological interventions were used in appropriate cases. All patients were able to ambulate normally before discharge (Speed, 1996).

A successful management of the three cases described in another report involved a combination of behavioral modification and physical therapy interventions. Abnormal movement patterns were ignored, and correct movement patterns were reinforced using feedback and praise. All three patients showed complete resolution of their symptoms (Ness, 2007).

There is one published abstract report where EMG biofeedback as used as a treatment for psychogenic tremor; this open label study estimated the effectiveness of biofeedback therapy and found improvement in 60% of 15 subjects (Levy et al., 2006). One case report

described a dramatic response to acupuncture in a patient with chronic, treatment-resistant PMD (Van Nuenen et al. 2007).

A preliminary experience with the application of transcranial magnetic stimulation to achieve symptom relief in psychogenic tremor showed its effectiveness in conversion disorder of motor subtype. In a group of 8 patients, 4 responded, 2 showed temporary improvement, and 2 did not respond. (Dafotakis et al., 2008). In a case report, a patient with psychogenic dysphonia was reported to have a dramatic improvement after 2 sessions of repetitive transcranial magnetic stimulation over the prefrontal cortex (Chastan et al., 2009).

6. Prognosis

The outcome of patients with PMDs is variable, and several elements that influence recovery have been described. These include the nature, chronicity, seriousness of the veiled psychopathology, the influence of external factors, the attitude of the patient, the capability of the patient's support system, as well as the modalities and the effectiveness of treatment. Predominantly, data are available on the outcome of conversion disorders in general.

Williams et al. found a permanent benefit in 52% of 131 patients, with complete, considerable and moderate relief in 25%, 21%, and 8% respectively, after a follow-up of an average of 1,8 years. They found that age, gender, intelligence, chronicity of illness, and types of symptoms had no influence on the outcome. In another longitudinal study of 228 patients with PMDs improvement symptoms was noticed in 56% of patients, 21% reported no change, and 22% were worse after an average duration of 3.4 years' follow-up. In this study, poor prognostic factors were inconsistent movements, dissatisfaction with the physician, long duration of illness, positive history of smoking, and suggestibility. Good prognostic factors were good physical health, positive social life, patients' perception of receiving effective treatment by the physician, elimination of a stressor, comorbid diagnosis of anxiety, and attribution of a specific medication (Thomas et al., 2006). A follow up report compared 66 patients with PMDs to 704 with Parkinson's disease and showed comparable levels of disability and physical quality of life, increased psychiatric comorbidity and more severe mental health disorders, even if patients with PMDs were 20 years younger and had a shorter pathological condition (Anderson et al., 2007).

Feinstein et al. reported persistence of abnormal movements in 90% of 88 patients followed up for an average of 3,2 years. Poor outcome was associated with psychiatric, long duration of symptoms, and insidious onset of symptoms (Feinstein, (2001). Much higher rates of improvement was reported in another longitudinal study of 127 patients with psychogenic tremor followed for at least 3 years, 55% reported improvement in tremor. Dissatisfaction with the physician was identified as the stronger prognostic risk factor of poor long term outcome; good prognostic factors were physician's prescribed treatment, elimination of stressor, specific medication, stress management, biofeedback, and psychotherapy (Jankovic et al., (2004). Other authors described the presenting features and long-term outcomes of 33 patients with electrophysiologically-confirmed psychogenic tremor by a follow-up questionnaire. After a median follow-up of 3.2 years, 64% of patients valued their disability as moderate severe, 27% had complete resolution of symptoms, and 9% reported mild unchanged symptoms. Of the patients who had resolution of symptoms, in 15% the resolution occurred spontaneously and in 12% it occurred after an intervention (1 with an antidepressant, 1 with psychology/rehabilitation, 1 with hypnotherapy, 1 with behavioral therapy). (McKeon et al., (2008). In a follow study involving 64 patients affected by

medically unexplained movement disorders, 28% showed complete resolution of symptoms, 20% improved, 14% remained unchanged, and 38% worsened after 6 years of follow-up (Crimlisk et al., 1998). Finally, other data showed that 83% of 42 patients with functional weakness or sensory symptoms, who have been investigated as inpatients, have symptoms and disability after a median of 12 years following initial assessment. In this study patients with only sensory symptoms and signs at presentation had significantly better outcome in terms of higher physical functioning, social functioning, and pain than patients with any symptoms or signs of weakness, a higher age of onset predicted lower physical functioning at follow up (Stone et al., 2003). If untreated, PMDs are inclined to become chronic, and follow-up data in several studies demonstrate 65-95% of patients are left with a high level of disability (Factor et al., 1995; Williams et al., 2005), undoubtedly asserting the necessity for an effective early intervention to convert the “sick role” of the patient and return to the suitable level of function as quickly as possible.

7. Conclusion

PMDs are important and underdiagnosed cause of major neurologic disability. Signs and symptoms must be interpreted in the overall clinical and psychological context. Neurophysiological and imaging findings may provide important understanding and confirmation of the diagnosis, but some cases pose a arduous challenge to both neurologists and psychiatrists. An adequate explanation of the symptoms to patients is a prerequisite to successful further treatment (Stone & Carson, 2010; Friedman & LaFrance, 2010). To date, the treatment for each patient with PMDs is individualized and may include psychotherapeutic interventions, behavioral therapy, pharmacotherapy, physical therapy, hypnosis and others. Recovery is sometimes delayed and can take place over the course of months and several patients are left with a high level of disability, but a supportive, nonjudgmental, and persistent multidisciplinary approach can divert the illness course to an excellent clinical outcome (Rosebush & Mazurek, 2006). Further researches are required, not only to improve the understanding and management of these heterogeneous diseases, but also for reconsidering conversion disorder terminology and positive rather than negative diagnostic criteria.

8. References

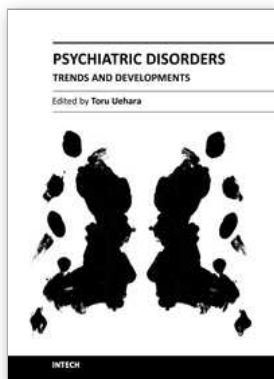
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Due to their prevalence, pervasiveness and burden inflicted on men and women of today, psychiatric disorders are considered as one of the most important, severe and painful illnesses. This impairment of cognitive, emotional, or behavioural functioning is in some cases tragic. Aside from knowing the physical organic factors, such as infections, endocrinal illnesses or head injuries, the aetiology of psychiatric disorders has remained a mystery. However, recent advances in psychiatry and neuroscience have been successful in discovering subsequent pathophysiology and reaching associated bio-psycho-social factors. This book consists of recent trends and developments in psychiatry from all over the world, presented in the form of multifarious and comprehensive articles. The first two sections of the book are reserved for articles on schizophrenia and depression, two major illnesses present in this field. The third section of the book is reserved for addiction psychiatry, related not only to socio-cultural but also biological alterations. The last section of the book, titled Biological Neuropsychiatry, consists of three topics - updated molecular biology, fundamental neuroscience and clinical neuropsychiatric conditions. Doubtlessly, this book will be fruitful for future developments and collaboration in world psychiatry.

How to reference

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