

## 16 Psychogenic Movement Disorders

*Dr M Edwards*

*Sobell Department of Motor Neuroscience and Movement Disorders, Institute of Neurology, UCL, Queen Square, London*

### **Introduction**

Psychogenic disorders are common throughout neurological practice, and psychogenic movement disorders (PMD) form a notable proportion of this case load. These disorders can be difficult to diagnose with confidence, but there are a number of clinical findings and targeted investigations that can aid diagnosis. Evidence regarding the best approach to treatment is still lacking, but early intervention appears to be helpful in preventing the development of chronic symptoms.

### **Definitions**

One of the first issues to consider is the lack of consensus on what to call psychogenic movement disorders. We have a number of words at our disposal, from “hysteria” (with all its unfortunate connotations regarding the source of these problems being the uterus), to functional, supratentorial, non-organic, and the latest contender: medically unexplained symptoms. We know already what patients think about these words, thanks to Stone et al (2002), and their study of “the number needed to offend”. Patients attending a neurology clinic were asked “if your doctor said you had X, would you think he was suggesting that you were (or had) Y. Words which patients felt meant that the doctor was suggesting that they were “putting it on”, “mad”, or that “symptoms were all in the mind” were judged offensive. Of the commonly used euphemisms for psychogenic illness, “functional” came out best of all, with “hysteria” and “medically unexplained symptoms” causing almost the same amount of “offence”. The word “psychogenic” was not specifically tested in this piece of research, although the word “psychosomatic” was, and scored badly.

Within the movement disorders medical community, “psychogenic movement disorders” is the most commonly used phrase, and this is what will be used in this chapter. However, it does pre-suppose an aetiology that may not be correct, and is likely to be offensive to some patients. The word “functional” is too broad a term for some doctors to accept, but it is perhaps a more appropriate word to use with patients when discussing the diagnosis.

### **Diagnostic Criteria**

In broad terms, PMD are disorders of movement (usually involving extra movement, but also a reduction of movement, particularly when associated with an abnormal posture – fixed dystonia), which are incongruent with typical movement disorders, where no organic cause can be found, and where there may be co-existent psychological disturbance. This definition, such as it is, is rather tautological and is certainly open to misinterpretation. More formal diagnostic criteria have been suggested by Fahn and Williams (1988), and although they too are somewhat problematic to apply, do seem to have reasonable specificity and sensitivity.

The Fahn Williams criteria divide PMD into four categories:

- **Documented:** persistent relief by psychotherapy, suggestion or placebo has been demonstrated, which may be helped by physiotherapy, or the patient was seen without the movement disorder when believing him- or herself unobserved
- **Clinically established:** the movement disorder is incongruent with a classical movement disorder or there are inconsistencies in the examination, plus at least one of the following three: other psychogenic signs, multiple somatisations, or an obvious psychiatric disturbance
- **Probable:** the movement disorder is incongruent or inconsistent with typical movement disorder, or there are psychogenic signs or multiple somatisations
- **Possible:** evidence of an emotional disturbance.

In practice it is fairly unusual to see a patient with a “documented” PMD – most patients will fall into the “clinically established” or “probable” category. The category of “possible” PMD is really too vague to be of much clinical use. As an aid to apply these criteria, the following additional definitions are given:

*Features that may be incongruent with a classical movement disorder include:* paroxysmal symptoms, abrupt onset, distractibility, astasia-abasia gait, entrainment of tremor to the frequency of repetitive movements, fixed dystonic postures, atypical stimulus sensitivity.

*Other psychogenic signs include:* “give-way” weakness, “false” weakness e.g. positive Hoover’s sign, non-organic patterns of sensory loss, distractibility, non-epileptic attacks.

*Somatizations* refer to multiple somatic symptoms (e.g. pain, gastrointestinal symptoms, sexual symptoms) where symptoms cannot be explained by a known medical condition, or where the complaints are in excess of those that would be expected from the history, physical examination and investigations.

### ***Psychiatric Diagnoses in PMD***

Two broad psychiatric diagnoses are often used (rather loosely in some cases) to describe the psychiatric disturbance that commonly occurs in PMD: conversion disorder and somatization disorder. Conversion disorder is characterised by the close temporal relationship of an unpleasant event with the development of one or more physical symptoms. Patients with conversion disorder should not have a long history of multiple physical complaints, and resolution of the physical symptom is often swift. DSM IV criteria for the diagnosis of conversion disorder are given in Table 1. Somatization disorder is characterised by a long history (often extending back into late childhood or early teenage years) of multiple physical complaints affecting many different systems. DSM IV criteria for the diagnosis of somatization disorder are given in Table 2. Both these disorders are thought of as “unconscious” thus differentiating them from factitious disorder (deliberate production of symptoms for psychological gain) and malingering (where symptoms are deliberately produced for external/material gain). Other patients with psychogenic illnesses may not fulfil full criteria for conversion or somatization disorders, but may have some features of these conditions, or may have other psychiatric illness such as depression, anxiety, obsessive compulsive disorder or psychosis. In those with somatization disorder, great care should be taken with accepting the patient’s description of previous illnesses; such descriptions are frequently incorrect or embellished, and confirmation of the nature of any previous illness and treatment should be made directly with previous treating health professionals.

***Table 1 DSM IV-TR Criteria for Conversion Disorder***

The patient has one or more symptoms or deficits affecting the senses or voluntary movement that suggest a neurological or general medical disorder.
The onset or worsening of the symptoms was preceded by conflicts or stressors in the patient’s life.
The symptoms are not faked or produced intentionally.
The symptom cannot be fully explained as the result of a general medical disorder, substance intake, or a behavior related to the patient's culture.
The symptom is severe enough to interfere with the patient's schooling, employment, or social relationships, or is serious enough to require a medical evaluation.
The symptom is not limited to pain or sexual dysfunction, does not occur only in the context of somatization disorder, and is not better accounted for by another mental disorder.
DSM-IV-TR divides conversion disorder into four subtypes: conversion disorder with motor symptom or deficit, conversion disorder with sensory symptom or deficit, conversion disorder with seizures, conversion disorder with mixed presentation.

**Table 2 DSM IV-TR Criteria for Somatization Disorder**

The individual must have a history of multiple physical complaints that began before age 30 and that continued for several years. These symptoms must cause significant impairment to social, occupational or other areas of functioning, and/or lead the patient to seek medical treatment.
The individual must report a history of pain affecting at least four different parts or functions of the body. Examples include headaches, back, joint, chest or abdominal pain, or pain during menstruation or sexual intercourse.
A history of at least two gastrointestinal symptoms, such as nausea, bloating, vomiting, diarrhoea, or food intolerance must be reported.
There must be a history of at least one sexual or reproductive symptom, such as lack of interest in sex, problems achieving erection or ejaculation, irregular menstrual periods, excessive menstrual bleeding, or vomiting throughout pregnancy.
One symptom must mimic a neurological condition. Examples include weakness, paralysis, problems with balance or coordination, seizures, hallucinations, loss of sensations such as touch, seeing, hearing, tasting, smelling-or difficulty swallowing or speaking, or amnesia and loss of consciousness. Pseudo-neurologic symptoms like these are the primary characteristics of another somatoform disorder known as “conversion disorder” (see above).

### ***How Common are PMD?***

Psychogenic illness in general is very common, accounting for as much as 20% of all consultations in primary and secondary care. PMD form a small proportion of the burden of psychogenic medical problems in general, and in general movement disorder clinics PMD account for about 2-5% of consultations. In tertiary movement disorder clinics such patients are much more common, accounting for 20-30% of consultations.

In terms of presentation, the commonest PMDs seen are psychogenic tremor and psychogenic dystonia, between them accounting for about 70% of all patients. Psychogenic myoclonus and gait disturbance account for most of the remaining cases. Psychogenic parkinsonism is rare, although Parkinson’s disease is often considered as a possible differential diagnosis in patients with unexplained tremor.

### ***An Approach to Patients with PMD***

#### ***Machine-Gunner or Sceptic?***

So how should one approach the patient with a possible psychogenic movement disorder? Perhaps one should exhaustively investigate such patients for every possible organic illness, however remote the likelihood. However, this “machine gun” approach to investigation can be directly harmful to a patient with psychogenic movement disorder. Exhaustive (and often invasive) testing is time-consuming, carries risks, and tends to reinforce the idea in the mind of the patient that the condition must be an organic one. Testing of this sort will often throw up spurious abnormal results, which may be latched onto by the patient as further evidence of organicity of the condition. The time delay involved in this kind of approach also fails the patient by delaying the onset of appropriate psychiatric treatment, and generates considerable uncertainty.

So, perhaps one should immediately assume that if the patient has symptoms that do not fit with a pattern that one has seen before, that the cause must be psychogenic. This “sceptical” approach is equally flawed. The history of movement disorders is littered with organic conditions previously labelled as psychogenic. Patients with cervical dystonia were said to be manifesting their psychological stress by “turning away” from their problems. Patients with writer’s cramp were said to be manifesting inner sexual conflict by being unable to hold phallic shaped object (a pen!). Just because the patient presents with an unusual set of symptoms and signs does not mean that the condition is psychogenic.

Clearly, there is a middle ground between these two approaches. This rational approach is primarily based on a thorough knowledge and familiarity with the range of organic movement disorders and their presentation. This provides a firm base from which to identify patients with symptoms and signs

that do not fit recognized patterns of organic movement disorders, and therefore where a psychogenic disorder needs to be considered in the differential diagnosis. At this stage, targeted and limited investigations that have value in distinguishing psychogenic from organic movement disorders should be performed, and in many cases this can lead to sufficient diagnostic certainty.

### **Elliot Slater and Long-Term Follow-up of Psychogenic Illness**

In 1965 Elliot Slater published two influential papers regarding 10 years follow-up on patients diagnosed with psychogenic neurological symptoms. Fifty percent of patients were said to have developed clear cut psychiatric or organic neurological conditions during follow-up. This study encouraged the reluctance of many clinicians to diagnose psychogenic illness, for fear of missing an underlying organic diagnosis.

However, Slater relied on telephone interview with patients as a method of discovering if a new organic diagnosis had been made, and this is notoriously unreliable in patients with psychogenic illness. The issue was revisited in 1998 in a six year follow up of 73 patients diagnosed with psychogenic neurological symptoms, with face-to-face interview and analysis of GP and hospital records conducted to determine if an organic diagnosis had been made that explained the original symptoms. Only 3 cases were found to have organic diagnoses that explained their original symptoms at follow up. The message from this and other follow-up studies of patients diagnosed in the modern era with psychogenic neurological problems is that mis-diagnosis is not as common as may be feared, and in the right circumstances the diagnosis of PMD can be made confidently.

### ***Medical History***

The history of both current complaint and past medical history may be revealing. While most organic movement disorders (with the exception of some secondary movement disorders, e.g. due to vascular lesions, and rare disorders such as rapid-onset dystonia-parkinsonism) have an insidious onset with gradual progression to maximum severity, PMD characteristically have an abrupt onset with rapid progression to maximum severity. Previous episodes of a movement disorder in the same or another limb may have occurred with complete or partial *remissions*, which occur rarely in organic movement disorders (although partial remissions can be seen in cervical dystonia), and *paroxysmal exacerbations* are common. In addition, organic movement disorders are usually consistent over time with little change in the phenotype (although they may progress). The phenotype of the PMD, on the other hand, may have been *inconsistent* over time, with a complete change in the nature of the abnormal movement. There may also have been *other somatisations*, putting the current presentation in the context of a wider somatoform illness. This may not be the first episode of somatisation, and previous unexplained medical symptoms are frequently unearthed when a careful history is taken. These may include other “functional” syndromes, such as fibromyalgia, atypical chest pain, or irritable bowel syndromes (Wessely et al, 1999) or other medically unexplained symptoms, which may have resulted in a number of investigations and treatments, including operations (Cohen et al, 1953). For example, inflammation may have been absent following an appendectomy for severe abdominal pain, or an episode of unexplained prolonged fatigue may have occurred previously. While patients with somatoform illness often report a number of previous diagnoses or complaints, their somatoform nature however often only becomes apparent when specifically sought in questions about the outcome of investigations to the patient or their general practitioner (Schrag et al, 2004).

Other pertinent information in the illness history includes the frequency of general practice attendances (the average number of annual GP attendance per year in the UK is 4 for men and 5 for women) and the frequency of requests for referral for a specialist opinion, which can be an indicator for the diagnosis.

The history may also be informative in other respects. It may reveal *abnormal illness behaviour*, for example non-compliance with treatment, “splitting” behaviour among the health professionals involved in their care, or “doctor-shopping”. *Litigation or a compensation claim* may represent a maintaining factor or there may be obvious *secondary gain*. There may have been an obvious *psychological stressor* before the onset of the PMD, suggesting a diagnosis of conversion disorder, or *psychological trauma* in the past history. However, this type of information should be treated with

caution as psychological conflicts are common in the population, and the coincidence between past psychological trauma and the presentation may be spurious.

## ***Examination***

The physical examination concentrates on four aspects:

1) *Absence of “hard” neurological signs.* A diagnosis of a PMD should not be made in the presence of hard neurological signs. However, as mentioned above, psychogenic overlay may exist comorbidly with an underlying organic illness. This may have a variety of reasons, including the patient’s wish to demonstrate to the doctor the extent of their problem, e.g. in patients referred for stereotactic surgery for undoubted Parkinson’s disease, or when patients have had previous experience with doctors who were unconvinced of the seriousness of their problem. In addition, pseudo-neurological signs are not uncommon, including pseudo-clonus, increased reflexes in a rigidly held limb, or pseudo-Babinski (often as a delayed, prolonged plantar extension), which can mislead the examiner. Care is required in the interpretation of such findings, but recognizing the possibility of a pseudo-neurological sign will facilitate the recognition of a psychogenic disorder.

2) *The presence of other non-organic signs,* such as non-organic weakness, non-anatomical sensory loss or excessive startle response. In movement disorders, extreme slowness may be seen which is, unlike bradykinesia, not fatiguing and without a decrement in the amplitude of the movement. There may be consistent past-pointing in an otherwise normal (sometimes excessively slow) finger nose test, and other tasks may simply not be completed, e.g. stopping two inches early in the finger-nose-test. The most useful sign is probably Hoover’s sign, which has been shown to have acceptable sensitivity and specificity (Ziv et al, 1998). This is performed by assessing the power of extension of a leg that is otherwise paralysed when the other, “good” leg is flexed. Caveats apply to all non-organic signs. For example, give-way weakness may be seen if the movement causes pain, and sensory disturbance not following a nerve or radicular distribution is common in Parkinson’s disease and often predates the onset of motor symptoms. In addition, classical signs such as midline splitting, splitting of vibration sense, and la belle indifference have poor sensitivity and specificity and are therefore of limited value in assessing these patients (Stone et al, 2002).

3) *Psychogenic signs.* There are a number of specific, positive features, which suggest a diagnosis of a psychogenic movement disorder. These include fluctuations during the examination, particularly an increase with attention and suggestion and decrease with distraction; the ability to trigger movements with unusual or non-physiological interventions (e.g. trigger points on the body); incongruence with the presentation of a recognized organic movement disorders; discrepancy between objective signs and disability (e.g. a patient with mild unilateral weakness who is bed or wheelchair bound); and discrepancy of symptoms and investigations excluding a pathophysiological correlate e.g. normal sensory evoked potentials in a patient reporting total loss of sensation in a limb. However, the only reliable feature that allows making a confident diagnosis of a PMD is a marked and persistent improvement with psychotherapy, placebo or suggestion. It is important that this response is marked and sustained as placebo effect is well recognized to improve movement disorders transiently in up to 30% (Goetz et al, 2002).

4) *Scars from multiple operations or self-inflicted injuries.* The physical examination may reveal multiple scars from multiple previous operations, which may be due to previous abnormal illness behaviour or somatisation, or self-inflicted injury, suggesting an underlying psychiatric disturbance.

**Table 3 Features in history and examination that can be helpful in the diagnosis of PMD**

<b>History</b>
Sudden onset of symptoms with rapid progression
Waxing and waning of symptoms with sudden remissions and reappearances of symptoms, often in different body parts.
Paroxysmal exacerbations of symptoms, particularly provoked by psychological stress.
Multiple additional neurological and systemic symptoms.
Overt symptoms of psychiatric disease such as depression or anxiety.
Presence of an identifiable psychological precipitating event to the emergence of symptoms or the worsening of symptoms.
<b>Examination</b>
Resolution or diminution of symptoms with distraction.
Exacerbation of symptoms when the affected body part is examined.
Improvement of symptoms with suggestion (for example with a non-physiological manoeuvre such as placing a vibrating tuning fork on the patient's forehead with prior suggestion that it might reduce symptoms).
"Give-way" weakness of the limbs
Non-organic patterns of sensory disturbance
Non-organic patterns of speech disturbance.
Excessive response to startle.
Disability out of proportion to examination findings
Non-organic gait disturbance (see below)

### ***Gait Disturbance in PMD***

Gait disturbance is a common accompaniment to PMD. It is often the single most useful clinical feature that discriminates between psychogenic and organic movement disorders, and a thorough assessment of gait is essential. A variety of gait problems are seen, alone or in combination, including:

- *Astasia/Abasia* (also known as "tightrope walker gait" or "skating on ice gait"): Here, the patient dramatically veers from side to side when walking, often waving the arms at the same time. Patients seem all the time to be about to lose their balance, but tend not to. In fact, such gait demonstrates very good balance, as the patient is able to shift their centre of gravity quickly from side to side without falling
- *Narrow base*: In contrast to many other patients with poor balance, patients with psychogenic gait disturbance tend to walk with a narrow, rather than a broad base
- *Hesitation*
- *Dramatic response to Romberg's test and tests of postural stability*
- *Excessive slowness*

### ***Specific Clinical Aspects of PMD***

As well as the features mentioned above, there are aspects of the clinical examination that can be helpful in the diagnosis of PMD which depend on the movement disorder that is present:

#### **Psychogenic Tremor**

- Present at rest, posture and on action
- Variable amplitude and frequency
- Entrain with movement of another limb
- Distractible
- Worsens when limb is examined.

### **Psychogenic Dystonia**

- Often a precipitating factor e.g. minor trauma
- Unusual distribution of dystonia given the age at onset (e.g. generalized dystonia in an adult)
- Fixed postures rather than the typical mobile postures of organic dystonia
- Severe pain
- Absence of task/position specificity
- Absence of sensory geste
- Poor response to botulinum toxin.

### **Psychogenic Myoclonus**

- Dramatic stimulus-sensitivity of jerks (although stimulus-sensitivity can be seen in organic myoclonus)
- Variability in distribution of jerks from day to day.

### **Psychogenic Parkinsonism**

- Tremor is often a prominent feature, and has features typical of other psychogenic tremors
- “Rigidity” has the characteristics of voluntary stiffness, and the resistance often changes depending on how fast the limb is moved
- Although movements may be slow, the progressive fatiguing of movement seen with organic akinesia is usually absent. Movements are often extremely slow when the patient is being examined, but less so when they are distracted
- Symptoms are usually symmetrical
- Gait is often bizarre, with slowness and unsteadiness combined. Reduction in arm swing may occur, but usually because the arms are held tightly at the sides
- Testing of postural stability often leads to dramatic loss of balance and falls

### ***Useful Bedside Tests in PMD***

There are some bedside tests that can be helpful in the assessment of patients with a suspected PMD. The usefulness of these tests depends very much on the type of movement disorder. They are most useful for patients with tremors and jerks, and least helpful for patients with fixed abnormal postures.

**Distractibility:** Ask the patient to close their eyes and perform a cognitive task (e.g. serial subtraction of seven from 100 with the answers said out loud). This tends to reduce the amplitude and frequency of psychogenic tremor, but tends to exacerbate organic tremor. In patients with psychogenic myoclonus the number of jerks also tends to reduce during such tasks. Cognitive tasks will tend to increase rigidity in organic parkinsonism, but may reduce rigidity in psychogenic parkinsonism.

**Entrainment:** Ask the patient to make a rhythmic movement with an unaffected limb at a different frequency to the tremor. In psychogenic tremor, this tends to cause the tremor to change. It may stop, become intermittent, or adapt (entrain) to the new frequency.

**Loading:** The majority of organic tremors improve with loading of the affected limb. If patients with psychogenic tremor of the arm are given weights to hold, their tremor usually worsens.

**Restraint:** In patients with psychogenic tremor or myoclonus, if the shaking limb is deliberately restrained by the examiner, the tremor tends to worsen and spread to other parts of the body.

### ***Specific Investigations for PMD***

There are some specific tests that can be helpful in the positive diagnosis of PMD. As with the bedside tests listed above, these tests are most helpful in patients with tremors or jerks, and least helpful in patients with fixed abnormal postures. Some of the electrophysiological tests described below are time consuming and/or need specific neurophysiological expertise.

**Electromyography:** Organic myoclonic jerks are typically caused by very brief (<50ms) bursts of muscle activity (although brainstem myoclonus can be of longer duration). It is not possible to

consistently voluntarily produce an EMG burst of less than about 50-75ms, and therefore a finding of bursts shorter than this supports the diagnosis of organic myoclonus.

In patients with tremor, electromyography can be used to assess tremor frequency, and, more accurately than at the bedside, to assess the impact on tremor frequency of tapping at a different frequency with another limb.

**Pre-movement EEG potentials:** Prior to normal voluntary movement, a slow rising wave is seen in the EEG called the *bereitschaftspotential* (BP). This can be looked for by performing an EEG during EMG recording of a number of jerks. The EEG trace shortly before each jerk is then selected and averaged, and this may reveal the presence of a BP in psychogenic myoclonus. If jerks are too frequent (>1 every 4-5 seconds), then it will be difficult to record a BP.

**“Back-averaging” of jerks:** In cortical myoclonus, jerks will be preceded by a cortical discharge, which can be recorded on EEG. A single discharge will be impossible to pick out amongst the background EEG activity. Therefore EEG is performed during EMG recording of a number of jerks. The EEG trace shortly before each jerk is then selected and averaged, and this will have the effect of revealing any underlying cortical discharge. The finding of such a cortical discharge is clearly incompatible with psychogenic myoclonus. However, the absence of such a discharge does not mean that the jerks are due to psychogenic myoclonus, as organic subcortical/brainstem myoclonus is not associated with cortical discharges. Note that it will be impossible to perform back-averaging if the jerks are very infrequent.

**Dopamine transporter imaging (DAT):** In particular patients with suspected psychogenic tremor and/or parkinsonism, DAT imaging can be helpful. A normal DAT scan effectively rules out the diagnosis of Parkinson’s disease and many atypical parkinsonian conditions such as progressive supranuclear palsy or multiple system atrophy. However, such scans are normal in many organic conditions causing tremor (e.g. essential tremor, dystonic tremor) and parkinsonism (e.g. dopa-responsive dystonia, drug-induced parkinsonism).

## ***Management***

Although patients with psychogenic movement disorders are not suffering from a life threatening illness, they are still experiencing subjectively compelling symptoms, often causing considerable distress and functional disability. Their management requires no less attention as symptoms due to organic causes. It is however important to recognise the potential dangers of unnecessary investigations and treatments once a diagnosis of PMD has been made. Therefore, once organic disease has been excluded, further unnecessary referrals, admissions to hospitals, investigations and treatments should be avoided. For patients with prolonged illness and non-acceptance of the diagnosis this may be difficult to achieve, and close cooperation between GPs, neurologists and psychiatrists may be required. On the other hand, it is important to bear in mind that unlike many neurological conditions this is a potentially treatable disorder and can be successfully managed.

### ***Breaking the News***

It is not sufficient for the clinician to reach the diagnosis of a psychogenic movement disorder and then to fail to communicate this to the patient. Particularly in the days before letters were routinely copied to patients, it was not uncommon to find that patients with PMD (and other psychogenic neurological problems) were fobbed off with a vague explanation for their symptoms and the rather frightening news that despite the symptoms all the tests were normal (leaving the patient with the impression that something must have been missed as how can the tests be normal if the symptoms are still there). There would then be a rather caustic letter to the GP with an undertone of a clear clinical impression of a lack of moral fibre on the part of the patient.

There are many factors that make clinicians unwilling to discuss the diagnosis. Patients with psychogenic illness often have a rather confrontational approach to the consultation, and may have a history of making complaints against health care professionals. Such patients often have fixed ideas regarding the organicity of their symptoms, and seem unlikely to accept a psychogenic explanation. Patients often have a very complex and established role based on their illness, often involving family

members and social services, and so the situation just seems too difficult to unravel. The clinician may feel that no treatment is available, and no hope of recovery, therefore there is no reason to give the diagnosis. Consultations with those with PMD are often very long, and the clinician may feel that there is simply no time left to embark on a long discussion regarding psychogenic illness. The clinician may feel uncertain of the diagnosis.

However, some PMD are treatable, and in those who fail to improve or who do not accept they have a PMD, a clear diagnosis greatly assists the process of “containment”.

The best approach is to make a positive diagnosis of a functional disorder, and communicate this clearly to the patient. This has the advantage of providing a clear answer to the question both the patient and the referring doctor have i.e. “what is wrong with me/ my patient?” This is better than a perhaps more typical explanation that “all the tests are normal, so it’s nothing to worry about”. This latter explanation fails to appreciate that the symptoms are very real to the patient, and the fact that the doctor “can’t find anything wrong”, is in fact a source of stress, not reassurance.

Most patients are very familiar with the concept of interaction between body and mind: indeed the concept that “stress” causes physical symptoms is part of common public medical understanding. This understanding can form the basis for an explanation that symptoms are unconsciously produced and are not deliberate or “faked”. It is important to give a clear statement that no further tests are necessary to establish the diagnosis, and that symptoms can get better.

### ***Treatment***

Depending on the type of PMD, associated disability, psychiatric co-morbidity, and patient expectations, some helpful management strategies include:

- Addressing the symptoms themselves - physiotherapy, hydrotherapy, occupational therapy, speech therapy, and other therapies may be particularly useful with loss of function. They also allow for recovery without a “loss of face” and can aid the relearning of lost functions
- Maintaining preserved function to avoid further functional loss e.g. by physiotherapy
- Addressing the distress caused by the symptoms, e.g. cognitive-behavioural therapy, biofeedback, TENS or pain management techniques (if pain is present)
- “De-medicalisation” – although it is appropriate to address the symptoms pragmatically, efforts should be made to try to reduce and/or withdraw medications, and to avoid other hospital appointments.
- Antidepressants should be used when symptoms are related to depression or anxiety, or depression or anxiety developed as a consequence; in some cases, anti-depressant medication may be useful in the treatment of symptoms not obviously related to an anxiety or affective disorder. If antidepressants are to be used, it is better to use one with minimal somatic side effects, and thus an SSRI is often preferred
- Psychological therapy is normally the preferred treatment approach, often using Cognitive-Behavioural Therapy (CBT). Cognitive therapy addresses how an individual’s thoughts, beliefs and expectations influence the occurrence and experience of unexplained symptoms. Behaviour therapy is used to identify behaviours that may be contributing to the maintenance of symptoms. CBT often involves training patients in psychological techniques that help them to change the way they think about and react to illness and physical symptoms. Psychotherapy is often used to explore and address feelings and conflicts that are distressing. It may be particularly useful in cases where there are emotional difficulties or traumatic events in the past. The choice of psychological therapy will normally be determined by a psychiatrist following referral
- Inpatient treatment, although of unproven efficacy, can sometimes be helpful in carefully selected patients. Such treatment usually involves a multi-disciplinary approach with graded physiotherapy and occupational therapy combined with psychotherapy and withdrawal from

unnecessary medication. Certainly this approach can be successful, but the relapse rate after discharge is unknown.

### ***Who Should Manage the Patient?***

The primary care physician has the most important role in the management of patients with a PMD. However, particularly in severe and persistent cases, GPs may need considerable support from the neurologist and/or psychiatrist. Infrequent, regular follow-ups by the same neurologist can provide important and consistent care, and help avoid further unhelpful referrals, starting new rounds of investigations and potentially harmful treatments.

### ***Prognosis***

There are few follow-up studies of PMD. The overall prognosis is often considered poor with persistence of the PMD over many years and a high rate of associated psychopathology (Lang, 1995; Feinstein et al, 2001). However, centres that can provide intensive treatment programs have reported considerably better outcome (Fahn and Williams, 1995). The prognosis of PMD is better in cases where there are few and mild symptoms with an acute onset, particularly if symptoms are preceded by traumatic or stressful events and if the duration of symptoms is short. The prognosis also appears to be better in younger patients with shorter duration of symptoms than in older patients with more chronic symptoms (Feinstein et al, 2001). At the opposite end of the spectrum, there are patients who continue to experience symptoms over time and often develop new symptoms related to other parts of their body. For them, it is particularly important to avoid unnecessary investigations and treatment, as this could lead to considerable secondary damage. Some of these patients represent a considerable therapeutic challenge and may be helped by specialised centres with multidisciplinary input from psychiatrists, neurologists and therapists that are experienced in the management of these disorders.

### ***Conclusions***

PMD are complex disorders which form part of the large burden of psychogenic neurological problems. They present particular difficulties due to the lack of diagnostic tests for many organic movement disorders, and the diverse nature of organic movement disorders. However, armed with an understanding of the typical range of presentations of organic movement disorders, and common features of PMD on history, examination and investigation, clinicians should be able to make a positive diagnosis of PMD. Evidence to support particular treatment approaches is still lacking, but a pragmatic approach to symptom control combined with a clear positive diagnosis and regular follow-up is a rational strategy.

### ***Further Reading***

- Brown P, Thompson PD. Electrophysiological aids to the diagnosis of psychogenic jerks, spasms, and tremor. *Mov Disord.* 2001;16:595-9.
- Deuschl G, Koster B, Lucking CH, Scheidt C. Diagnostic and pathophysiological aspects of psychogenic tremors. *Mov Disord.* 1998 Mar;13(2):294-302.
- Fahn S, Bressman SB, Marsden CD. Classification of dystonia. *Adv Neurol* 1998; 78: 1-10
- Fahn S, Williams DT. Psychogenic dystonia. *Adv Neurol.* 1988; 50: 431-55.
- Farmer SF, Sheean GL, Mayston MJ, Rothwell JC, Marsden CD, Conway BA, Halliday DM, Rosenberg JR, Stephens JA. Abnormal motor unit synchronization of antagonist muscles underlies pathological co-contraction in upper limb dystonia. *Brain.* 1998;121:801-14.
- Feinstein A, Stergiopoulos V, Fine J, Lang AE. Psychiatric outcome in patients with a psychogenic movement disorder: a prospective study. *Neuropsychiatry Neuropsychol Behav Neurol.* 2001;14:169-76.
- Francis P, Baker GA. Non-epileptic attack disorder (NEAD): a comprehensive review. *Seizure.* 1999;8:53-61.
- Goetz CG, Leurgans S, Raman R; Parkinson Study Group. Placebo-associated improvements in motor function: comparison of subjective and objective sections of the UPDRS in early Parkinson's disease.
- Hallett M. Dystonia: abnormal movements result from loss of inhibition. *Adv Neurol.* 2004;94:1-9.
- Lempert T, Brandt T, Dieterich M, Huppert D. How to identify psychogenic disorders of stance and gait. A video study in 37 patients. *J Neurol.* 1991;238:140-6
- Mai FM, Merskey H. Briquet's Treatise on hysteria. A synopsis and commentary. *Arch Gen Psychiatry.* 1980;37:1401-5.
- Miyasaki JM, Sa DS, Galvez-Jimenez N, Lang AE. Psychogenic movement disorders. *Can J Neurol Sci.* 2003 Mar;30 Suppl 1:S94-100.

- Schrag A, RJ Brown RJ, Trimble MR. The reliability of self-reported diagnoses in patients with neurologically unexplained symptoms. *J Neurol Neurosurg Psychiatry*. 2004 Apr;75(4):608-11.
- Stone J, Wojcik W, Durrance D, Carson A, Lewis S, MacKenzie L, Warlow CP, Sharpe M. What should we say to patients with symptoms unexplained by disease? The "number needed to offend". *BMJ*. 2002;325:1449-50.
- Stone J, Zeman A, Sharpe M. Functional weakness and sensory disturbance. *J Neurol Neurosurg Psychiatry*. 2002;73:241-5
- Tijssen MA, Marsden JF, Brown P. Frequency analysis of EMG activity in patients with idiopathic torticollis. *Brain*. 2000;123:677-86.
- Wessely S, Nimnuan C, Sharpe M. Functional somatic syndromes: one or many? *Lancet* 1999;354:936-9
- Williams DT, Ford B, Fahn S. Phenomenology and psychopathology related to psychogenic movement disorders. *Adv Neurol*. 1995;65:231-57.
- Ziv I, Djaldetti R, Zoldan Y, Avraham M, Melamed E. Diagnosis of "non-organic" limb paresis by a novel objective motor assessment: the quantitative Hoover's test. *J Neurol*. 1998;245:797-802.