Functional (psychogenic) movement disorders: merging mind and brain

Mark J Edwards, Kailash P Bhatia

Functional (psychogenic) movement disorders (FMD) are part of the wide spectrum of functional neurological disorders, which together account for over 16% of patients referred to neurology clinics. FMD have been described as a “crisis for neurology” and cause major challenges in terms of diagnosis and treatment. As with other functional disorders, a key issue is the absence of pathophysiological understanding. There has been an influential historical emphasis on causation by emotional trauma, which is not supported by epidemiological studies. The similarity between physical signs in functional disorders and those that occur in feigned illness has also raised important challenges for pathophysiological understanding and has challenged health professionals’ attitudes toward patients with these disorders. However, physical signs and selected investigations can help clinicians to reach a positive diagnosis, and modern pathophysiological research is showing an appreciation of the importance of both physical and psychological factors in FMD.

Introduction

Functional (psychogenic) movement disorders (FMD) are part of the spectrum of functional neurological disorders, some of the most prevalent disorders seen in neurological practice.1 In common with other functional disorders, there is an absence of appropriate health-service provision and research interest for FMD, despite their prevalence. These disorders occupy a grey area between neurology and psychiatry—often with neither specialist group willing to take charge—which has resulted in what has been described in relation to FMD as a “crisis for neurology”.2

There are three rationales behind this Review. First, there have been notable developments in diagnostic techniques, pathophysiological understanding, and treatments in FMD, which together represent a substantial advance in knowledge. Second, we wish to highlight an important shift that has taken place in approaches to functional disorders in general: the historically influential explanation for symptoms triggered by emotional trauma (and the research and treatment agendas that emerge from this explanation) has been challenged. Third, because of the enormous health-care and social-care costs associated with functional symptoms such as FMD, health professionals and medical scientists need to take an active interest in keeping up to date with best practice in diagnosis and management. FMD have traditionally been thought of as the most difficult of the functional neurological disorders to diagnose and manage, but we will show that they need not always carry such a reputation.

Terminology and definition

When experts cannot agree on a unified terminology for a disorder, there is likely to be a fundamental problem with understanding the underlying pathophysiology. This difficulty in understanding is certainly present for psychogenic disorders, including FMD, for which there are many descriptive terms to choose from (panel I). The choice of term is not a trivial issue, because it directly affects case definition, diagnosis, treatment, research agenda, and explanations of illness that we give to patients.

Some terms, such as psychogenic, conversion, or somatization, directly suggest that the cause of physical symptoms is psychologically mediated. Conversion and somatisation are operationalised diagnoses that specifically need the presence of a psychological triggering factor and exclusion of feigning. However, for most movement disorder clinicians, the presence of a psychological triggering factor is not a requirement for diagnosing a patient with FMD,3 and the difficulties of routinely excluding feigning in clinical practice are complex.4 We also show later that recent epidemiological studies question the relevance of psychological triggers in most patients with FMD.5 However, other terms also have their difficulties. For example, does a disorder that is medically unexplained simply mean that we have not yet found the medical explanation, and with advancement of medical knowledge it will become a medically explained disorder? What level of medical explanation do we need for a disorder to be medically explained? The term hysteria comes with substantial historical baggage, but some movement disorder specialists, including the most eminent of recent times, David Marsden, have argued passionately that the term should be retained.6 The term functional also has a long and distinguished neurological history, but some argue that it has lost its meaning over time and is now too broad a term to be helpful.7

Patients are directly affected by the diagnostic labels we give them. Stone and colleagues8 explored this issue with unselected neurology outpatients and found that many terms were judged by patients as suggesting that the doctor thought their symptoms were “put on” or “all in the mind”. Hysteria came out badly on this assessment, but so did the term medically unexplained. The term psychogenic was not specifically assessed, but somatic was and was rated negatively. Functional was the term most acceptable to patients.
We use the term functional in this Review. The origins and chequered history of this term have been discussed in detail by Trimble.7 He argues that the original physiological use of the term as a disturbance of functioning of the nervous system where the cause has yet to be defined has value, in contrast to its use as a “polite eponym” for a psychiatric disorder.7 We accept that there are difficulties in using this term as a replacement for other terms such as psychogenic. Although in our view this term accurately reflects the state of the evidence regarding the pathophysiology of psychogenic disorders, this use does also mean that the word functional is used to apply to the functional disturbance that occurs in this patient group only, and not in patients with, for example, headache. Unfortunately, this debate cannot be solved in this Review, and we recognise the insufficiency of present terminological options. In clinical practice, we use the term functional, because it is the term most acceptable to patients and does not presuppose a cause for symptoms that is unproven. We specifically define this disorder by its clinical appearance, rather than by any causative speculation as a movement disorder that is significantly altered by distraction or non-physiological manoeuvres (including dramatic placebo response) and that is clinically incongruent with movement disorders known to be caused by neurological disease.

Epidemiology, quality of life, and cost
The subject of this Review represents an important issue because of its prevalence and effect on quality of life and health-care economics. FMD are part of the wide spectrum of functional or psychogenic neurological symptoms, which together account for 16% of new patients attending neurology outpatients’ clinics.1 Accurate estimates of prevalence of FMD are hampered by case definition and the setting of the clinic from which cases are ascertained, and range between 2 and 20% of patients attending movement disorder clinics.9,10 These disorders cause an impairment in quality of life that is similar to, and in some aspects worse than, that experienced by patients with Parkinson’s disease.36 No studies have specifically addressed the economic burden of FMD but, given the level of disability reported by patients in the long term (see Prognosis section), there are probably substantial associated health and social care costs. In a large study of patients with functional neurological symptoms (n=1144) who were followed up for 1 year, at least 50% had stopped working and more than one-quarter were receiving illness-related financial benefits;38 the economic burden for those with FMD is unlikely to differ from this. UK estimates for the yearly costs associated with working-age patients with “medically unexplained symptoms” are approximately £18 billion,39 slightly more than the annual cost associated with dementia for patients of all ages in the UK.39 Women are more often affected by FMD than men, and mean age at onset in different studies ranges from 37 to 50 years.39 FMD are also reported, but not commonly, in children40 and in the elderly.41,42

Clinical features
Several historical features and examination findings are commonly noted in patients with FMD regardless of the movement disorder phenomenology. These features are not diagnostic of FMD, but can be helpful as part of the diagnostic process. Patients often describe the sudden onset of symptoms, which might be precipitated by a physical event (eg, injury or illness).35 Symptoms can rapidly progress to become severe—a pattern that is unlike the slow progressive course of most movement disorders.35 Patients might report marked variability in symptom severity, including complete remissions and sudden recurrences. The phenomenology of the movement disorder might shift over time. Patients might have a history of other functional symptoms. Neurological signs apart from

Panel 1: Terms commonly used to describe psychogenic disorders and their implications

<table>
<thead>
<tr>
<th>Psychogenic</th>
<th>Conversion disorder</th>
<th>Somatisation disorder</th>
<th>Medically unexplained symptoms</th>
<th>Non-organic</th>
</tr>
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<tbody>
<tr>
<td>Suggests psychological causation</td>
<td>Operationalised within DSM: requires an identified psychological triggering factor for diagnosis</td>
<td>Operationalised within DSM: requires presence of multiple physical symptoms including one conversion neurological symptom</td>
<td>Suggests that a medical explanation might one day be apparent</td>
<td>Defines the condition by what it is not; the term organic is itself not well defined</td>
</tr>
<tr>
<td>Broad term suggesting a functional rather than a structural deficit, which could apply to several neurological disorders not regarded as psychogenic but where structural pathology is absent, eg, migraine</td>
<td>Historical term that carries substantial stigma in society and implies a link between symptoms and the uterus</td>
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the movement disorder can be consistent with functional illness, for example Hoover’s sign (leg paresis), give-way weakness, and non-anatomical patterns of sensory loss. The co-occurrence of functional and organic movement disorders might be expected, given the common co-occurrence of other functional disorders with organic disorders, for example epilepsy and non-epileptic seizures. However, hard evidence for the prevalence of this phenomenon is limited.

Functional tremor

Functional tremor (FT) is the commonest presentation of FMD, accounting for at least 50% of patients. Commonly, the historical features are as outlined earlier, with sudden onset, variability in severity with remissions, and variability in the body part affected. Most patients have a tremor that is present (or at least can be present at different times) at rest, in posture, and during action, which is an unusual pattern for organic tremor. Tremor can occur in any body part: the hands and arms are most frequently involved, but tremor of the head, legs, or even palate can also occur. By contrast with patients with organic tremor, patients with FT often direct clear visual attention towards their affected limb during examination.

The key clinical feature that helps to differentiate FT from organic tremor is that FT changes with the level of attention towards the affected limb. This can be appreciated during history taking as fluctuation in tremor severity (or even presence) while the patient’s attention is engaged. Conversely, FT commonly worsens during examination. Specific examination manoeuvres can be used to distract attention away from the tremoring limb; some formal assessments of specificity and sensitivity have been done on these manoeuvres clinically, and more extensively with electrophysiological tremor recordings. Thus, tremor may change with cognitive distraction tasks or tapping at different frequencies (it may entrain to the tapping frequency, shift in frequency, or stop altogether; or the patient might inexplicably be unable to undertake the required tapping movement correctly); pause with ballistic movement of the other limb (figure 1); or paradoxically worsen with loading. The specificity and sensitivity of some of these tasks have been investigated (without tremor recordings) in patients with FT compared with those with essential tremor; tapping tasks had the highest sensitivity and specificity (72.7% and 73.3%, respectively). In a head-to-head study, we compared these techniques with tremor recordings in FT and a

Figure 1: Tremor recordings in a patient with functional tremor

Tri-axial accelerometry recordings from an accelerometer attached to the tremoring hand (top three traces) and to the unaffected hand (bottom three traces). The patient is undertaking rapid reaching movements to a target with the unaffected hand, which produces brief pauses in the tremor in the other hand. Acc=acceleration. X=x axis. Y=y axis. Z=z axis. R=right. L=left.
range of organic tremor disorders (Parkinson’s disease, essential tremor, dystonic tremor, and neuropathic tremor). We found that no single measure had sufficient specificity and sensitivity to differentiate FT from organic tremor. This finding is probably due to the different mechanisms for tremor generation in FT, with some patients generating tremor primarily by co-contraction, which is not readily distractible by tapping tasks. Cognitive distracter tasks were poor discriminators between organic tremor and FT. A cutoff score was devised by combining several of these measures, which, if validated in a prospective study, could provide a laboratory-supported level of diagnostic certainty (table).

**Functional dystonia**

Functional dystonia is the second most common presentation in patients with FMD. There are substantial differences of opinion between experts regarding the diagnosis of functional dystonia. These differences are not helped by the history of dystonia in general: patients now classified as having organic dystonia were, until the 1980s, commonly classified as having hysteria. Advances in genetics have led to recognition of the phenotypes of primary idiopathic dystonia, which have typical ages of onset, courses, and distributions of dystonia. For example, DYT1 gene-related primary dystonia starts before age 25 years, often affects the legs at onset, and can spread over a few years after onset to cause generalised dystonia. By contrast, late-onset primary dystonia affects the cranio-cervical region (spasmodic torticollis is the most common form) and tends to remain focal. This identification of distinct phenotypes has made easier the recognition of secondary dystonic (including functional) disorders, which have presentations incongruous with primary dystonia phenotypes.

Patients with functional dystonia typically present with fixed abnormal postures accompanied by severe pain similar to that noted in chronic regional pain syndrome type 1 (CRPS1). Most patients with functional dystonia are young women and the usual trigger is a minor peripheral injury, but the disorder is sometimes spontaneous. Such patients (who might also be classed as having “causalgia-dystonia” or “tonic dystonia of chronic regional pain”) may experience spread of symptoms to other body parts without further injury. Limbs are usually involved, but fixed dystonia affecting the neck or jaw has also been reported.

Physical examination manoeuvres can be used to show with certainty whether attention is playing a key part in symptom generation in functional tremor; however, to show the same level of certainty in fixed dystonia is difficult. One might argue that this difficulty occurs because fixed dystonia is not a functional disorder, but to state that maintenance of postures does not need a similar level of attention as maintenance of tremor would also be reasonable. However, in some patients a brief give way of muscle activity in the affected limb will be felt when the patient is distracted. In support of the functional label for fixed dystonia, symptoms may resolve with multidisciplinary rehabilitation with an emphasis on cognitive-behavioural therapy, the disorder may co-exist with other more clearly defined psychogenic disorders, and marked (curative) placebo responses have been reported. However, some, but not all, research electrophysiological tests suggest similarities between patients with fixed dystonia and those with organic dystonia, although these tests are all subject to confounding from muscle activity, attention, and anxiety. Maintenance of a fixed posture has been hypothesised to produce secondary changes in central body schema, and these changes might contribute to pain and other unusual features, such as the seeking of limb amputation by some patients.

**Functional myoclonus**

Functional myoclonus is reported in about 20% of patients with FMD. As might be expected in patients with intermittent movements, distractibility can be difficult to demonstrate on examination. Electrophysiological tests can therefore be of substantial diagnostic help to clinicians. Simple electromyography (EMG) recordings can be used to assess EMG burst duration: consistent EMG bursts of less than 75 ms do not occur in functional myoclonus. However, the converse is not true, because some forms of organic myoclonus are associated with long-duration EMG bursts. Electrophysiological features associated with cortical myoclonus (giant somatosensory evoked potentials, electroencephalogram [EEG] spike 20 ms...
before jerks) would not be expected in functional myoclonus. The most useful diagnostic test in patients with suitable symptoms (see below) is EEG–EMG back-averaging—a method for assessing cortical activity shortly before movement (figure 2). In healthy people undertaking a self-paced voluntary movement, a slow rising potential is seen in the EEG starting about 1·5 s before movement and peaking just before movement: this is the pre-movement potential, or Bereitschaftspotential. This potential can be recorded in patients with functional myoclonus and is not seen in people with organic myoclonus. There are technical limitations to this test: at least 30 jerks need to be recorded, so patients must have a reasonable number of jerks within the recording time; and pre-movement potentials are often difficult to record in patients with very rapid jerks (more than one every 3–5 s), although distractibility is often easy to indentify in such patients clinically. Pre-movement potentials have been reported in patients with tics, but not consistently. Two groups have independently reported that most patients diagnosed with idiopathic spinal segmental or propriospinal myoclonus (the latter characterised by flexion jerks of the abdomen) have pre-movement potentials before jerks and are therefore best characterised as functional.

**Other functional movement disorders**

Pure functional gait disturbance accounts for about 6% of patients with functional movement disorders, but an abnormal gait is a common feature in patients with other FMD. Various gait patterns are described, but a key feature of most of these patterns is that the patient does not seem to adapt to the gait problem they complain of in an optimum way. For example, patients who complain of unsteadiness might walk with a narrow base or might adopt uneconomic postures, which are apparently compensatory for the gait disturbance but would seem objectively to make it worse. Some patients have objectively very good balance while subjectively complaining of poor balance; such patients shift their centre of gravity by pivoting from side to side at the waist on a narrow base without falling, thus showing excellent balance. This pattern has been termed the “walking on ice” gait. Another common pattern of functional gait disturbance is a monophasic dragging gait, where the affected leg is dragged behind the patient, typically with the medial surface of the foot in contact with the floor and the leg externally rotated. This is quite different from the circumducting gait typically seen in patients with organic hemiplegia. So-called bizarre patterns of gait are seen in organic movement disorders such as Huntington’s disease and generalised dystonia, and care needs to be taken in reaching a diagnosis with regard to unusual gait disturbance.

Functional parkinsonism, chorea, and tics are rarely reported. Most patients diagnosed with functional parkinsonism actually have a functional resting tremor rather than other features (such as slowness of movement) that mimic parkinsonism. Dopamine transporter scans can be helpful to a limited extent if diagnostic uncertainty exists. Dopamine transporter scans are normal in patients with functional parkinsonism but also in organic parkinsonism due to postsynaptic dopaminergic deficit, such as drug-induced parkinsonism. Paroxysmal functional movement disorders are rarely reported but do occur. They may be under-recognised because patients might instead be diagnosed with functional non-epileptic seizures. Clinicians need to be familiar with the range of triggers, attack durations, and attack frequencies that occur in organic paroxysmal movement disorders to help them to differentiate patients with functional attacks with confidence and to exclude epilepsy by EEG measurement during an attack if necessary. There is no substitute for seeing an attack, and the video facility available on many modern mobile phones makes it easier for patients’ relatives to record an attack for viewing by the physician.

**Diagnostic criteria**

We emphasised earlier that the diagnosis of FMD should as much as possible be a positive diagnosis. It should not be a diagnosis of exclusion, nor a diagnosis...
made on the basis of co-existence of a movement disorder with psychological disturbance. Co-existent psychological disturbance is common throughout organic neurological disease and is not an adequate symptom on its own to diagnose a psychogenic disorder.\(^6\)

Operationalised diagnostic criteria for functional movement disorders include the Fahn-Williams criteria\(^7\) (the most widely used), the Shill-Gerber criteria,\(^8\) and a recent revision of the Fahn-Williams criteria proposed by Gupta and Lang (panel 2).\(^9\) All these criteria have as a key element a gradation of certainty of diagnosis; for example, in the Fahn-Williams criteria the gradation is documented, clinically established, probable, and possible. Various alterations to the Fahn-Williams criteria have been suggested, including a merging of documented and clinically established categories into one category of clinically definite; the removal of the possible category; and the addition of laboratory tests to produce a category of laboratory supported.\(^10\) The Shill-Gerber criteria have been criticised for being so heavily weighted towards historical information that the diagnosis of FMD could possibly be made with little reference to the clinical characteristics of the movement disorder.\(^11\) These criteria also place weight on the notion of disease modelling, in which experience of a disease in a family member, acquaintance, or via work provides a model for patients to produce functional symptoms. This notion is difficult to investigate (eg, the quantification of all potential disease models to which a person has been exposed would seem to be very difficult), and therefore its place in diagnostic criteria seems questionable according to the available evidence.

The Fahn-Williams and Shill-Gerber criteria have recently been subjected to assessment of inter-rater reliability.\(^12\) There was only poor (Shill-Gerber) to moderate (Fahn-Williams) inter-rater reliability for probable and possible categories, with good agreement for the clinically definite category.

### Pathophysiology

The earlier discussion of terminology shows a historical emphasis on psychological causation in FMD, as with other functional disorders. Psychiatric formulations based on late 19th and early 20th century concepts of conversion, somatisation, and dissociation still form the basis for psychiatric diagnoses in these disorders and, by extension, ideas regarding pathophysiology.\(^13\) However, patients with psychogenic disorders in general, including those with FMD, do not have the expected rates of psychological trauma, either at the onset of physical symptoms or in the past.\(^14\) This finding presents a problem for those who emphasise such factors as important pathophysiologically. This problem has traditionally been solved, in a rather circular argument, by suggesting that recall of such life events is repressed by patients and therefore is not available for report. Although this repression may occur in some patients, the suggestion is largely untestable.

### Panel 2: Fahn-Williams and Gupta-Lang criteria for diagnosis of psychogenic movement disorders

**Fahn-Williams criteria**\(^47\)

**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 himself 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 disorders or there are psychogenic signs or multiple somatisations.

**Possible**

Evidence of an emotional disturbance.

**Laboratory supported definite**

Not included in this classification.

**Gupta and Lang proposed revisions**\(^48\)

**Clinically definite**

Includes Fahn-Williams documented and clinically established categories, and also includes movement disorders that are incongruent with a classical movement disorder or for which there are inconsistencies in the examination, without the need for the additional presence of psychogenic signs, multiple somatisations, or an obvious psychiatric disturbance.

**Probable**

Not included in this classification.

**Possible**

Gupta and Lang question the utility of this category. They suggest it could be used to include those with movement disorders congruent or consistent with a classical movement disorder but where there are additional psychogenic signs, somatisations, or evidence of emotional disturbance. However, they suggest that this category may then include patients who are different pathophysiologically from those with true psychogenic movement disorders.

**Laboratory supported definite**

Presence of data from electrophysiological tests that prove the presence of a psychogenic movement disorder (primarily evidence of pre-movement potentials before jerks or data from tremor studies).
The key clinical feature that separates patients with FMD from those with organic movement disorders is that the movements have features that one would usually associate with voluntary movement (distractibility, resolution with placebo, and presence of pre-movement potentials), but patients report them as being involuntary and not under their control. There seem to be just two logical explanations for this feature: either movements are deliberately feigned or there must be a brain mechanism that allows voluntary movement to occur but to be experienced subjectively as involuntary. Understanding this mechanism would seem to be key to understanding the development of symptoms and their treatment.

Although study of subjective experience of movement might seem impossible, cognitive neuroscience has revealed the existence of mechanisms within the brain that confer a sense of intention and a sense of agency to movement, and examples of organic brain disease in which such processes are disrupted. Functional imaging recorded in patients during an episode of functional tremor or when the same patients were voluntarily mimicking their tremor showed hypoactivation of the temporoparietal junction during the psychogenic tremor. This area is thought to be an important comparator region, comparing actual with predicted sensory feedback. The suggestion is that hypoactivity might represent a failure to match the actual and predicted sensory feedback, producing a feeling of involuntariness associated with movement. Linked with this finding, we have reported that patients with functional tremor do not have the normal sense of intention that is associated with voluntary movement. Another functional imaging study in FMD noted abnormally strong amygdala–supplementary motor area connectivity when patients were presented with emotionally valent stimuli and abnormally weak supplementary motor area–prefrontal cortex connectivity in a reaction time task. A hypothesis arising from this work and a further functional imaging study in FMD is that emotionally arousing events might trigger movement controlled by the supplementary motor area that is functionally disconnected from top-down control by the prefrontal cortex.

In a recent study that compared patients with functional tremor to those with organic tremor, we compared self-completed diaries in which patients rated the amount of the waking day they felt they had tremor with the results of continuous tremor recordings from a wrist-worn actigraph. Patients with organic tremor tended to over-rate their tremor (by about 20%) in diaries compared with the tremor watch recordings. Patients with functional tremor over-rated their tremor by a significantly higher amount (more than 65%; p=0.0001), and had on average only 30 min of tremor per day. We have interpreted this finding within a Bayesian framework as a dominance of prior expectancy over sensory data.

These studies all provide results that would be unexpected in patients feigning symptoms, although they do not amount to an aid to diagnose feigning of symptoms. However, these studies do provide examples of research in functional disorders that look beyond the rigid framework that has provided a causal model for symptoms on the basis of emotional trauma alone. There has been a wider rebalancing of attitudes toward functional disorders, so that they are considered within a biopsychosocial model, not just a psychosocial one. This change in attitude might prompt a search for psychological factors of causative importance that are not solely related to emotional trauma. This type of search has been underway for some decades in other disorders (eg, schizophrenia) once regarded as mental disorders but in which great importance is now given to understanding the biological basis of the disorder. The old dichotomy between mental and brain disorders has increasingly been swept away by the progress of cognitive neuroscience and, although long overdue, this process is now affecting views of functional neurological disorders. To regard FMD and other functional disorders as just brain disorders would also be incorrect, and so a combined approach is necessary that integrates societal and psychological factors with our present knowledge of the biology of brain function. This process might not just lead to better understanding of FMD, but might also improve our understanding of the human brain.

Management

There are limited studies available on which to base management decisions in FMD. It seems reasonable to presume that treatment of FMD can be informed by data regarding treatment of other functional neurological conditions, in particular those that involve motor symptoms.

In our view, the most important first steps in a successful treatment approach are effective communication of the diagnosis and the provision to patients and their families of a rational model within which to understand the physical symptoms. In light of the earlier discussion regarding diagnosis, we emphasise the positive ways in which the diagnosis has been made rather than falling back on explanations based on normal test results. We try to introduce the role of psychological factors in their proper context and do not insist on extensive exploration for underlying psychological trauma. Patients with FMD are vulnerable to unscrupulous medical and health practitioners, particularly over the internet. There are some useful web resources that can help to support understanding of the diagnosis for patients with functional symptoms, and we direct patients towards these.
There is no evidence to support the use of drugs traditionally used for the treatment of organic movement disorders in patients with FMD. Medical and surgical interventions are often harmful to patients with FMD,\(^{30,35}\) and part of successful treatment is removal of unnecessary medications and avoidance of unnecessary tests and surgical treatments. The only exception to this is provided by studies of intrathecal baclofen in patients with fixed dystonia and CRPS1, but these results carry important caveats. An initial controlled study in a small group of patients gave impressive results,\(^{62}\) but a follow-up study of a larger group of patients\(^{41}\) found treatment-related complications to be high, although a beneficial effect was seen in many patients. The difficulty with both studies is that the systemic effects of intrathecal baclofen cannot be adequately controlled, and therefore patients are systematically unmasked to the intervention. We would urge caution with the use of this invasive treatment given evidence of dramatic placebo response of patients with fixed dystonia to other treatments.\(^{31}\) We have highlighted earlier the difference of specialist opinion regarding the nature of the disorder in patients with fixed dystonia. Despite this difference in opinion, there is no need to delay effective management, because delay is associated with worse outcome and, in some patients, the development of irreversible contractures.\(^{31}\) The key component of treatment is rapid early mobilisation with suitable holistic management of pain (with emphasis on techniques used in CRPS1 such as desensitisation). Surgical intervention and prolonged immobilisation should be avoided.

There is some evidence that psychological intervention, in the form of either psychodynamic psychotherapy\(^{44}\) or more pragmatic symptom-based cognitive-behavioural therapy,\(^{65}\) might be helpful for patients with functional motor symptoms, including FMD. These techniques are only applicable to those patients who accept that psychological or behavioural interventions are valid methods of treatment for their physical symptoms. Likewise, there is evidence that a subset of patients who are willing to take antidepressants (in this study, those diagnosed with primary conversion disorder and not those with somatisation disorder) can benefit from this treatment.\(^{46}\)

Physical rehabilitation has face validity as a treatment to manage motor symptoms, but there are few trials upon which to base opinion. There is evidence that a multidisciplinary approach combining physical and psychological treatment can be effective for some patients.\(^{46}\) This intensive (often inpatient) treatment is expensive and will always have limited availability. In a retrospective, case-control study, a brief (5 day) intensive inpatient physical therapy programme produced major self-rated improvement in symptoms in more than 60% of participants (n=48) compared with 22% of control individuals (n=32) after 2 years of follow-up.\(^{47}\) This study used an explanatory model of symptoms that was deliberately physical (abnormal motor learning) and, although psychological factors were addressed, the focus was maintained on physical symptoms and treatment. Benefit has also been reported from a simple 12-week programme of supervised low-to-medium intensity walking in patients with FMD.\(^{44}\) Such findings, if confirmed by further studies, suggest that physical interventions (perhaps combined with symptom-focused cognitive-behavioural techniques)\(^{46}\) may provide an effective and acceptable means of symptom management.

Placebo interventions can have strong effects in patients with FMD,\(^{46}\) but evidence for long-term benefit is absent and the ethics of such treatments are hotly debated.\(^{49}\) In this vein, transcranial magnetic stimulation has been reported to be of benefit in patients with FT, with investigators suggesting a possible real effect of stimulation.\(^{50}\) However, the unmasked nature of the intervention makes a placebo effect likely.

**Prognosis**

Long-term follow-up studies are confounded by the manner in which cases are diagnosed—typically by tertiary movement disorder clinics where patients with brief transient symptoms will be missed. In these studies, about half of patients report some improvement in symptoms at long-term (3–5 years) follow-up, although most patients remain out of work due to illness.\(^{70,72}\) Good prognostic features include a short duration of illness, perception by the patient of effective management by the clinician, and the presence of depression or anxiety (which is therefore amenable to psychiatric treatment).\(^{71,72}\)

**Future work**

This Review reports several areas in which evidence-based knowledge is limited. With specific reference to FMD, we wish to highlight the following areas and important questions.

**Diagnostic tests and criteria**

The discussion of FT shows how clinical (and simple electrophysiological) tests can be used to make a positive diagnosis. This process urgently needs to be extended to other movement disorders, in particular to patients with abnormal postures. If successful, use of this process could lead to new diagnostic criteria, which would be based on these positive clinical features and rely less on associations with psychological factors or with the notion of (unspecified) incongruity with organic movement disorders.

**Research**

By contrast with some functional disorders in which symptoms are subjective (pain, sensory loss, or disturbance), functional motor disorders such as FMD provide
We have described here how the correct diagnosis of FMD should rely on positive clinical characteristics and not on the presence of psychological trauma. The historical emphasis on psychological trauma as a triggering factor has perhaps skewed research agendas and neurological interest in these patients, and has certainly alienated many patients who cannot believe that their physical symptoms are related to psychological trauma. We do not aim to minimise the importance of psychological factors (anxiety, depression, arousal, and attention) in such patients, but rather to point out that a dogmatic and relentless search for a clear triggering psychological trauma may be misguided and unhelpful.

One additional benefit of rebalancing the approach to FMD and functional disorders in general is that it might allow us to reconsider some of the symptoms that are present in our patients with organic neurological disorders. Any practising neurologist would recognise that patients with the same organic disease of apparently similar severity manifest symptoms in differing ways, which can have a dramatic effect on disability and quality of life. This phenomenon, often called functional overlay, is, we would suggest, often ignored as a non-symptom that interferes with the neurological management of patients. However, understanding the pathophysiology of this overlay and knowing how to treat it—knowledge that is likely to come from research into pure functional disorders—could be of substantial benefit to patients with organic disease. The common occurrence of physical triggering events such as illness or injury in patients with pure functional symptoms is itself a pointer towards an important overlap between organic and non-organic illness.\(^2\)

Although we agree that FMD and other functional disorders do represent a crisis for neurology, it is not an unsolvable one. We believe that now is the time for the movement disorder and wider neurological community, in cooperation with psychiatry, psychology, and physical therapy services, to lead the search for solutions.

**Conclusions**

We have described here how the correct diagnosis of FMD should rely on positive clinical characteristics and not on the presence of psychological trauma. The historical emphasis on psychological trauma as a triggering factor has perhaps skewed research agendas and neurological interest in these patients, and has certainly alienated many patients who cannot believe that their physical symptoms are related to psychological trauma. We do not aim to minimise the importance of psychological factors (anxiety, depression, arousal, and attention) in such patients, but rather to

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**Search strategy and selection criteria**

For the purposes of this Review, we searched Medline between 1975 and December, 2011, for articles with the keywords “psychogenic”, “functional”, “conversion”, “movement disorder”, “parkinsonism”, “tremor”, “dystonia”, “myoclonus”, “chorea”, “tics”, and “gait”. We selected papers relevant to diagnosis, treatment, and pathophysiology.

Researchers with a measurable entity that reflects the underlying symptom. We suggest, therefore, that patients with functional motor disorders, in particular FMD, are the natural group to include in future research studies. Research studies such as those reviewed herein show that such patients can participate in research and that informative results can be obtained.

**Treatment**

There is a clear and urgent need for treatment studies in FMD and other functional neurological disorders. The acceptability of treatment approaches and the availability of those who might deliver treatment should be considered when planning clinical trials. In this regard, patients with FMD may not accept that there is an important psychological dimension to their symptoms, and therefore they might be less likely to accept treatments based solely on psychotherapy or cognitive-behavioural therapy. However, research on symptom-focused cognitive-behavioural therapy approaches and simple physical interventions point towards workable interventions which, if given early in the course of the illness, could produce benefit in these patients.

**Education**

None of the aforementioned suggested changes is likely to happen unless concerted efforts are made to increase interest and knowledge about FMD among movement disorder specialists. Through this process, patients will be most likely to receive early positive diagnoses, avoid iatrogenic harm by unnecessary investigations and treatments, benefit from world-class research, and receive effective treatment in a timely manner.

**Conclusions**

We have described here how the correct diagnosis of FMD should rely on positive clinical characteristics and not on the presence of psychological trauma. The historical emphasis on psychological trauma as a triggering factor has perhaps skewed research agendas and neurological interest in these patients, and has certainly alienated many patients who cannot believe that their physical symptoms are related to psychological trauma. We do not aim to minimise the importance of psychological factors (anxiety, depression, arousal, and attention) in such patients, but rather to point out that a dogmatic and relentless search for a clear triggering psychological trauma may be misguided and unhelpful.

One additional benefit of rebalancing the approach to FMD and functional disorders in general is that it might allow us to reconsider some of the symptoms that are present in our patients with organic neurological disorders. Any practising neurologist would recognise that patients with the same organic disease of apparently similar severity manifest symptoms in differing ways, which can have a dramatic effect on disability and quality of life. This phenomenon, often called functional overlay, is, we would suggest, often ignored as a non-symptom that interferes with the neurological management of patients. However, understanding the pathophysiology of this overlay and knowing how to treat it—knowledge that is likely to come from research into pure functional disorders—could be of substantial benefit to patients with organic disease. The common occurrence of physical triggering events such as illness or injury in patients with pure functional symptoms is itself a pointer towards an important overlap between organic and non-organic illness.\(^2\)

Although we agree that FMD and other functional disorders do represent a crisis for neurology, it is not an unsolvable one. We believe that now is the time for the movement disorder and wider neurological community, in cooperation with psychiatry, psychology, and physical therapy services, to lead the search for solutions.

**Contributors**

MJE and KPB generated an outline for the paper. MJE wrote the first draft and MJE and KPB revised this draft.

**Conflicts of interest**

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