Psychogenic movement disorders
Amitabh Gupta and Anthony E. Lang

Introduction
Psychogenic movement disorders (PMDs) are movement disorders that result from a psychological or psychiatric rather than neurological disturbance. The primary psychiatric diagnosis varies; most cases are considered to be conversion disorders, in which the problem is caused by an unconscious mechanism, but infrequently some are factitious disorders or malingering, in which the abnormal movements are purposefully feigned. Traditionally, PMDs represented a diagnosis of exclusion. This perception was fuelled by the observation that PMDs can mimic various organic diseases, sometimes with confounding test results [1], and by studies and experiences of high false positive rates [2–4]. Over the years, stricter clinical criteria, improved imaging, and investigational advances have allowed the diagnosis of PMDs to be made more comfortably [5], and organic movements are far less often misdiagnosed as psychogenic. Here, we review the literature of the past 2 years and summarize how PMDs are currently diagnosed, investigated, and treated.

Purpose of review
This review summarizes the progress made in the area of psychogenic movement disorders (PMDs) over the past 2 years, and a simplified classification of diagnostic certainty is proposed that incorporates electrophysiological assessment.

Recent findings
Functional magnetic resonance imaging studies have demonstrated altered blood flow in conversion disorders that may reflect changes in synaptic activity. Electrophysiological testing shows limitations in distinguishing between psychogenic and organic propriospinal myoclonus and dystonia. Recent evidence cautions against the uncritical acceptance of all cases of posttraumatic myoclonus and ‘jumpy stump’ as being organic in nature. ‘Essential palatal tremor’ is recognized as a rather heterogeneous group of tremors that includes psychogenic tremor. Two recent studies evaluating the long-term prognosis of psychogenic tremor differ in the degree of unfavorable outcome. Different groups of PMDs might have distinctive gait characteristics with prognostic, diagnostic, or therapeutic value. Two recent reviews provide comprehensive information on the understudied area of PMDs in children.

Summary
The diagnosis of PMDs should not be regarded as a diagnosis of exclusion. Careful clinical assessment is critical, and imaging or electrophysiological studies may provide important insights and confirmation of the diagnosis though some cases remain challenging and current assessments fail to provide needed clarification. Treatment is often delayed, contributing to a largely unfavorable long-term outcome. Well designed randomized control trials that validate and compare therapeutic options are urgently required.

Keywords
children, diagnosis, investigations, movement disorders, psychogenic, treatment

Diagnosis
Although no single clinical finding is pathognomonic for PMDs, several features are quite helpful. The clinical aspects of specific types of PMDs (i.e. tremors, myoclonus, dystonia, Parkinsonism, gait disorders) have been reviewed at length elsewhere [6–8]. Table 1 [8,9] provides a list of typical historical and clinical clues to the diagnosis.

Over a decade ago, criteria were proposed for various levels of diagnostic certainty of PMDs [10]. With the subsequent progress made in establishing clinical characteristics and investigational methods, it appears timely to propose modifications to the diagnostic classification, including the incorporation of electrophysiological
sprain and headaches. It has been shown that FES in patients with movement disorders can be used to suppress akathisia.

**Functional MRI (fMRI) imaging of conversion disorders**

May be extremely useful in distinguishing psychogenic dysfunction from organic disease. Functional MRI (fMRI) of the brain has provided interesting insights into the condition.

**Nuclear imaging**

May be normal in some early cases of Parkinson's disease, but show diminished signal in Parkinson's disease. PET studies have shown that cerebral activity is changed in MCD, comparison to organic weakness requires elucidation. Similarly, in patients with a sensory conversion disorder, fMRI has demonstrated that vibratory stimulation of the affected limb fails to activate the contralateral cortical sensory area. This result supports the notion that clinical deficits in this psychiatric condition are associated with real changes in blood flow that indicate reduced cortical responsiveness. The mechanism underlying these changes is not well understood, but cortical activation was shown to be restored with bilateral stimulation, possibly acting as a ‘distractor’ to reverse inhibition.

Patients with motor conversion disorders (MCDs) have been shown to activate the motor cortex in a pattern that differs from controls simulating weakness (17**). Although this suggests that cerebral activity is changed in MCD, comparison to organic weakness requires elucidation. Similarly, in patients with a sensory conversion disorder, fMRI has demonstrated that vibratory stimulation of the affected limb fails to activate the contralateral cortical sensory area. This result supports the notion that clinical deficits in this psychiatric condition are associated with real changes in blood flow that indicate reduced cortical responsiveness. The mechanism underlying these changes is not well understood, but cortical activation was shown to be restored with bilateral stimulation (18), possibly acting as a ‘distractor’ to reverse inhibition.

Previous studies have established the utility of specialized electrophysiological techniques in aiding or confirming the diagnosis of certain PMDs, particularly psychogenic tremor and psychogenic myoclonus. These studies have been reviewed elsewhere (11,19*). Recent electrophysiological studies have better delineated PMDs. Compared with controls, PMD patients exhibited an excessive affective response to the startle eye blink reflex (21). When pictures invoking either positive or negative affective states were shown at the time of eliciting the eye blink startle, reflex potentiation was seen in both conditions, in contrast to the normal inhibition with the negative affective state seen in...
controls. It remains to be seen whether this result can separate PMDs from organic disease, particularly in patients with underlying concurrent psychopathology. Central motor conduction is typically normal in patients with MCD. Using transcranial magnetic stimulation (TMS) in such patients, Liepert et al. [22] found that motor threshold, short and long interval intracortical inhibition (SICI and LICI), and intracortical facilitation (ICF) were similar to that in controls, indicating unchanged baseline cortical excitability. When movements were imagined, however, cortical excitability was decreased in the affected limb of MCD patients but increased in the unaffected limb as it was in the limbs of healthy individuals. Further confirmatory studies are required. Theoretically, this decreased cortical excitability with motor imagery might be able to separate conversion disorder from factitious disorder or malingering.

Some electrophysiological testing fails to distinguish PMDs from organic movement disorders, indicating complex overlapping neuronal mechanisms and the importance of careful clinical assessment. Electrophysiological findings are very similar between simulated propriospinal myoclonus (PSM) and the organic counterpart [23]. Aside from a generally longer electromyographic burst duration observed in controls purposefully simulating PSM, a fixed pattern of muscle recruitment, synchronous activation of agonist and antagonist, electromyographic burst duration less than 1000 ms, and slow conduction in the spinal cord (5–15 m/s) have been shown in both groups. In a recent case of confirmed psychogenic PSM following eye surgery [24\*], electrophysiological analysis demonstrated slow conduction, short burst duration, consistent caudal muscle activation, and absence of premovement potentials, with only some variability in muscle activation possibly suggesting a PMD. Another example in which PMDs and the organic counterpart may not be differentiated with electrophysiological studies is dystonia. TMS has shown increased cortical excitability (decreased SICI, LICI, ICF) in both groups [25], suggesting that this abnormality can occur as a consequence of the dystonic postures or, alternatively, it may be an ‘endophenotype’ that predisposes to the dystonia in both organic and psychogenic cases. A distinction between these two possibilities was not possible, as the unaffected side was not investigated. Avanzino et al. [26\*\*] analyzed both sides with TMS and obtained similar results, supporting the conclusion that the cortical hyperexcitability may reflect a predisposing ‘endophenotypic trait’ for dystonia in either condition [27], although transcallosal or ipsilateral descending influences from the involved hemisphere could still result in these changes being secondary to the postures. Future investigation searching for improved electrophysiological approaches could provide great value, as clinical distinction between organic and psychogenic dystonia can be extremely challenging [28,29\*].

**Posttraumatic movement disorders**

Posttraumatic movement disorders are a source of considerable controversy. Complex regional pain syndrome (CRPS) type I typically following minor injury may be associated with fixed dystonia, myoclonus, and tremor. Some authors have provided considerable evidence in favor of a psychogenic cause [30,31], whereas others favor an ‘organic’ explanation [32]. Munts et al. [33] recently presented the electrophysiological characterization of myoclonus associated with CRPS. However, it was subsequently argued that their findings of burst duration length of more than 70 ms, variability in burst characteristics, side-to-side coherence, and entrainment were strongly supportive of a psychogenic cause of the movements [34]. The ‘jumpy stump’ (an uncommon but widely

### Table 2 Diagnostic classification of psychogenic movement disorders

<table>
<thead>
<tr>
<th>Traditional</th>
<th>Proposed revision</th>
</tr>
</thead>
<tbody>
<tr>
<td>Classification of degrees of certainty in diagnosis*</td>
<td>Classification of degrees of certainty in diagnosis</td>
</tr>
<tr>
<td>1. Documented†</td>
<td>1. Documented (as in original)</td>
</tr>
<tr>
<td>Remittance with suggestion, physiotherapy, psychotherapy, placebos, ‘while unobserved’</td>
<td>2a. Clinically established plus other features (as in original)</td>
</tr>
<tr>
<td>2. Clinically established‡</td>
<td>2b. Clinically established minus other features</td>
</tr>
<tr>
<td>Inconsistent over time/incongruent with clinical condition + other manifestations: other ‘false’ signs, multiple somatizations, obvious psychiatric disturbance</td>
<td>Unequivocal clinical features incompatible with organic disease with no features suggesting another underlying neurological or psychiatric problem</td>
</tr>
<tr>
<td>3. Probable</td>
<td>1 + 2a + 2b = Clinically Definite</td>
</tr>
<tr>
<td>Inconsistent/incongruent – no other features</td>
<td>3. Laboratory-supported definite</td>
</tr>
<tr>
<td>Consistent/congruent + ‘false’ neurological signs‡</td>
<td>Electrophysiological evidence proving a psychogenic movement disorder (primarily in cases of psychogenic tremor and psychogenic myoclonus)</td>
</tr>
<tr>
<td>Consistent/congruent + multiple somatizations‡</td>
<td></td>
</tr>
<tr>
<td>4. Possible§</td>
<td></td>
</tr>
<tr>
<td>Consistent/congruent + obvious emotional disturbance§</td>
<td></td>
</tr>
</tbody>
</table>

*Adapted from [12].
†Subsequently, Fahn and his coauthors [10] proposed combining categories 1 + 2 under ‘Clinically Definite’.
‡We proposed to reclassify these patients under ‘Possible’.
§We also questioned the utility of retaining the ‘Possible’ category as this generally represents patients with organic movement disorders with additional psychiatric problems rather than a true ‘Possible psychogenic movement disorder’ [11].
Psychogenic movement disorders: a review

Psychogenic tremor

Psychogenic tremor is a heterogeneous disorder, encompassing a wide range of clinical presentations, which can be challenging to distinguish from organic tremors. It is characterized by repetitive, involuntary movements that do not follow the rules of anatomy and physiology. This movement disorder is often associated with psychological distress, such as anxiety or depression, and is often triggered by stress or anxiety. It can be severe and disabling, and the diagnosis can be difficult to make due to the overlap with organic tremors.

Psychogenic gait disorders

Psychogenic gait disorders are another type of psychogenic movement disorder, characterized by disturbances in walking, standing, or sitting. These disorders can range from mild difficulties to severe incapacitation, and they can be challenging to differentiate from organic gait disorders. The diagnosis of psychogenic gait disorders is often based on the patient's history, physical examination, and ruling out organic causes. It can be difficult to make the diagnosis, and the treatment is often supportive care.
patients); for example, patients with lower body dystonia might show false positive results if they were able to use the chair as a geste antagoniste, and patients with ‘gait apraxia’ who can perform normal bicycling leg movements might also fair better in the chair than on standing.

Baik and Lang [47] assessed gait abnormalities in a large group of patients with PMDs. When patients were subdivided into those with more generalized PMDs that also compromised the gait and those with a pure psychogenic gait disorder, the mixed PMD group most frequently showed slowness of gait (followed by dystonic gait), whereas the pure gait group most commonly displayed buckling of knees (followed by astasia–abasia). This study provides an incentive to assess whether gait differences among PMD subpopulations have diagnostic, predictive, or therapeutic value and whether these differences reflect distinct pathophysiological processes.

**Psychogenic movement disorders in children**

PMDs in children have been addressed by recent reviews [48*,49*]. Uncommon before the age of 10 years, clinical clues in children are derived from the adult literature. As in adults, there is female sex predominance, they comprise approximately 3% of children visiting movement disorders clinics, and the distribution of psychiatric diagnosis shows conversion disorder in up to 80%, followed by somatization disorders (10–20%) and factitious disorders (<5%). Quite in contrast to the adults, malingering was not reported. Similar to adults [10], dystonia and tremor were the most common clinical phenotypes, followed remotely by gait disorders. In addition, dystonia was fixed in most cases and usually preceded by minor physical trauma.

Some differences are noteworthy. Although in adults the nondominant limb may be most often affected (except for tremor in psychogenic Parkinsonism), children more frequently have PMDs in their dominant limb. It has been suggested that this may reflect incomplete hemispherical lateralization [49*]. Although coexisting organic neurological disease is well recognized in patients with psychogenic neurological complaints, associated organic movement disorders are rare in children with PMDs [49*] in contrast to estimates in adults, which range between 10 and 25%. Similarly, psychiatric diseases in children appear to be less common, as most studies report 10% comorbidity, compared with the 40% rate quoted for adults with PMDs. However, Ferrara and Jankovic [48*] reported a 50% comorbidity rate (anxiety, depression, irritability) and a 40% rate of perfectionistic personality, a common trait in patients with conversion disorders. Lastly, psychogenic dystonia is less easily and comfortably diagnosed in children, given the spectrum of organic dystonia that results from genetic mutations, neurometabolic diseases, and other causes.

**Treatment**

A delay in diagnosing PMDs should be avoided at all costs. Failure to do so often results in multiple referrals, repeated unnecessary diagnostic tests, unjustified and potentially harmful treatments including medication trials and even surgeries, and the perpetuation of the belief of underlying organic illness. This also delays the initiation of appropriate treatment (though, as discussed below, treatment is often very different), which reduces efficacy, particularly if treatment is started 6–12 months after onset of the movement disorder. To maximize treatment compliance, it should be acknowledged that the patient has a movement disorder (i.e. a form of tremor, myoclonus, or dystonia) and a biological explanation provided. Lastly, despite the various treatments applied, evidence-based data are limited, and prospective double-blinded studies are urgently required.

Therapy is best administered in multimodal fashion. Psychotherapy [50], cognitive behavioral therapy, rehabilitation [51,52], antidepressants [53], and hypnosis [54] have had variable success. Monthly sessions of acupuncture produced normalization for only 4–5 days in a patient with psychogenic jerking movements [55]. She was wheelchair bound, with symptoms present for over a decade. This effect may have simply been because of a reduction of anxiety or some other form of placebo effect. Recently, a 17-year-old boy with psychogenic aphonia for 20 months who had failed speech therapy recovered completely following two sessions of repetitive TMS [56*]. Low-frequency stimulation was used, rather than the high-frequency pulses usually applied to psychogenic limb paralysis intended to directly activate the primary motor areas. Functional imaging data in psychogenic paralysis have shown decreased activity in the primary motor cortex and increased activity in prefrontal cortex. Thus, low-frequency stimulation may have inhibited the overactive prefrontal areas resulting in disinhibition of the primary motor cortex. Target choice might be critical; low-frequency stimulation resulted in rapid recovery, whereas high-frequency treatment takes many weeks and produces variable success. In light of the strong potential for a placebo effect, a controlled trial is clearly needed to demonstrate reproducibility, taking into account the well-established effect of patient anticipation on treatment outcomes [57].

Data on long-term prognosis are scarce, but most studies point to significant impact on quality of life. Anderson et al. [58] compared 66 patients with PMDs to 704 with Parkinson’s disease and found increased psychiatric comorbidity, more severe mental health disturbances, and very similar levels of disability and physical quality of life, despite the fact that patients with PMDs were 20 years younger, had shorter disease duration (4 versus
7 years), and were compared to a Parkinson’s disease population with a 30% prevalence of motor fluctuators. Prognosis appears to be better in children with PMDs. In one study [49*], 50% of children remitted, 40% returned to normal school life, whereas 20% experienced only partial improvement or remained disabled. As with adults, children who remitted were treated in their first year after symptom onset, whereas those without improvement had been symptomatic for many years [49*]. Remission was found to occur most often with tremor, which may relate to early visits to the specialist, whereas children with dystonia often did not improve. Recent studies have shown that many children remain disabled for several years; the long-term prognosis of these children requires careful assessment.

**Conclusion**

PMDs can be diagnosed with reasonable certainty in many cases, after limited ancillary testing is obtained. Sometimes the diagnosis can be made with certainty on the first clinical assessment and, at other times, comes only after repeated careful evaluations and the exclusion of other possible causes. The importance of special electrophysiological analysis has been repeatedly emphasized, including in recent studies; however, availability of expertise in this area may limit its broader application. Critical is the realization that exclusively psychogenic or organic findings may not always be available, but it is the constellation and pattern of findings that leads to the recognition of PMDs. Given the significant impact on quality of life, future work should focus on assisting more definitive early diagnosis, a better understanding of the true pathogenesis of these disorders (including whether there are unique differences or whether they are similar to one another and to other somatoform disorders), and finally on treatment trials that are tailored to providing aggressive therapeutic intervention early on in the disease state and to patients with well established disabling symptoms.

**References and recommended reading**

Papers of particular interest, published within the annual period of review, have been highlighted as:
- of special interest
- **of outstanding interest

Additional references related to this topic can also be found in the Current World Literature section in this issue (p. 448).


17. Stone J, Zeman A, Simonetto E, et al. fMRI in patients with motor conversion syndrome and controls with simulated weakness. Psychosom Med 2007; 69:961–969. This article demonstrates changes in cerebral blood flow between patients with MCD and healthy controls that simulate weakness. As this finding emphasizes real changes in psychogenic patients that likely reflect altered synaptic activity, further studies with this methodology may help to distinguish conversion syndromes from malingering or factitious disorders in challenging cases.
20. This article documents that though the presence of finger tremor usually strongly indicates an organic nature, finger tremor has also been observed in patients with otherwise convincing psychogenic pattern.
26. This report indicates that electrophysiological testing may not be able to differentiate psychogenic from organic PSM, placing emphasis on clinical acumen. Perhaps surprisingly, though confirmation of a PMD was clinically not difficult in this case, several electrophysiological features seen in organic PSM were recorded from this patient (though the recording conditions for the absent Bereitschaftspotential were not described).

This article and [25] provide a detailed electrophysiologic analysis of patients with organic and psychogenic dystonia and demonstrate the difficulties and limitations of such assessment in distinguishing between both groups.
Movement disorders


43 McKeon A, Ahlkog KE, Bower JH, et al. Psychogenic tremor: long term progression in patients with electrophysiologically-confirmed disease. Mov Disord 2009; 24:72–76. This article and [44] provide important insight into the long-term prognosis of psychogenic tremor. As they differ in their prognostic outlook, based on the experiences in the respective movement disorder clinics, they provide an opportunity to identify reasons for this difference, with the prospect of improving long-term outcome in psychogenic tremor.


