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
Adapted from [8].

twisting facial movements that move mouth to one side or the other (organic dystonia of the facial muscles usually does not pull the mouth sidewise).

unless repeated reversals with placebo are documented when symptom otherwise is frequent and attacks are prolonged.

with central nervous system circuitry [9].

Functional MRI (fMRI) imaging of conversion disorders Parkinsonism from Parkinson's disease.

may be extremely useful in distinguishing psychogenic Parkinsonism from Parkinson's disease.

and nuclear imaging [15]. As these methods detect the degree of viable dopaminergic neurons of the substantia nigra (PET) or [13,14], and, recently also, 123I-Isoflupane SPECT [15]. As these methods detect the degree of viable dopaminergic neurons of the substantia nigra (PET) or their respective synaptic termini in the striatum (SPECT) [13,14], and, recently also, 123I-Isoflupane SPECT [15]. As these methods detect the degree of viable dopaminergic neurons of the substantia nigra (PET) or their respective synaptic termini in the striatum (SPECT), scan results are normal in psychogenic cases but show diminished signal in Parkinson’s disease. Although it has been suggested that nuclear imaging may be normal in some early cases of Parkinson’s disease, recent evidence suggests that most of these cases are likely falsely classified [16]. Therefore, nuclear imaging may be extremely useful in distinguishing psychogenic Parkinsonism from Parkinson’s disease.

Table 1 Clues suggesting psychogenic cause

<table>
<thead>
<tr>
<th>Historical</th>
<th>General examination</th>
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<tbody>
<tr>
<td>Abrupt onset (symptoms often maximal at that time)</td>
<td>Movement inconsistent</td>
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<tr>
<td>Static course</td>
<td></td>
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<tr>
<td>Spontaneous remissions/cures</td>
<td>Variability over time (frequency, amplitude, direction/distribution of movement)</td>
</tr>
<tr>
<td>Paroxysmal symptoms (generally nonkinesigenic)b</td>
<td>Distractibility reduces or resolvesa, attention increases movement</td>
</tr>
<tr>
<td>Psychiatric comorbiditiesc</td>
<td>Selective disability</td>
</tr>
<tr>
<td>Secondary gain (often not apparent)</td>
<td>Entrainment (especially with tremor)</td>
</tr>
<tr>
<td>Risk factors for conversion disorderd</td>
<td>Movement incongruent with organic movement disorders</td>
</tr>
<tr>
<td>Psychological stressorsm</td>
<td>Mixed (often bizarre) movement disorders</td>
</tr>
<tr>
<td>Multiple somatisations/undiagnosed conditions</td>
<td>Paroxysmal attacks (including pseudoseizures)</td>
</tr>
<tr>
<td>Employed in allied health professions (infrequent)</td>
<td>Precipitated paroxysms (often suggestible/startle)</td>
</tr>
<tr>
<td></td>
<td>Suggestibilityf</td>
</tr>
<tr>
<td></td>
<td>Effortful production or deliberate slowness (without fatiguing) of movement</td>
</tr>
<tr>
<td></td>
<td>Self-inflicted injury (caution: tic disorders)</td>
</tr>
<tr>
<td></td>
<td>Delayed and excessive startle response to a stimulus</td>
</tr>
<tr>
<td></td>
<td>Burst of verbal gibberish or stuttering speechg</td>
</tr>
<tr>
<td></td>
<td>False (give-away) weakness</td>
</tr>
<tr>
<td></td>
<td>Nonanatomical sensory loss or spread of movement</td>
</tr>
<tr>
<td></td>
<td>Certain types of abnormal movements common in individuals with PMDsah</td>
</tr>
<tr>
<td></td>
<td>Functional disability out of proportion to examination findings</td>
</tr>
</tbody>
</table>

a Distracontibility should be tested both with mental and motor tasks. Although most often organic movement disorders are not suppressed, organic tics or akathisia can be suppressible, and recently it was shown that diaphragmatic tremor was suppressed by simple motor tasks, perhaps by interference with central nervous system circuitry [9].

b Separation from organic paroxysmal dyskinesias can be challenging, particularly if they occur infrequently with prolonged symptom-free periods.

c Psychiatric diseases can also coincide with organic illness or present as part of the organic movement disorder.

(d) Sexual and physical abuse, trauma.

* Often initiated by injury (often minor) or motor vehicle accident associated with litigation or compensation.

f Application of pressure with finger or tuning fork may reduce symptom. With paroxysmal symptom, suggestibility and placebo trial may not be helpful, unless repeated reversals with placebo are documented when symptom otherwise is frequent and attacks are prolonged.

g Particularly if the entire word is repeated (typically broken up into syllables, each repeated), rather than the initial syllable.

h Such movements include dystonia that begins as a fixed posture (particularly if abrupt onset, painful, and early contractures are seen); bizarre gait; twisting facial movements that move mouth to one side or the other (organic dystonia of the facial muscles usually does not pull the mouth sidewise).

Adapted from [8].

Investigations

Nuclear imaging, in contrast to magnetic resonance imaging (MRI), has proven quite helpful in distinguishing psychogenic Parkinsonism from Parkinson’s disease. Well established methods include fluorodopa positron emission tomography (FDOPA-PET), βCIT single photon emission computed tomography (SPECT) [13,14], and, recently also, 123I-Isoflupane SPECT [15]. As these methods detect the degree of viable dopaminergic neurons of the substantia nigra (PET) or their respective synaptic termini in the striatum (SPECT), scan results are normal in psychogenic cases but show diminished signal in Parkinson’s disease. Although it has been suggested that nuclear imaging may be normal in some early cases of Parkinson’s disease, recent evidence suggests that most of these cases are likely falsely classified [16]. Therefore, nuclear imaging may be extremely useful in distinguishing psychogenic Parkinsonism from Parkinson’s disease.

Functional MRI (fMRI) imaging of conversion disorders has provided interesting insights into the condition.

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 [11,19,20]. 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 separately distinguish 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

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**Table 2 Diagnostic classification of psychogenic movement disorders**

<table>
<thead>
<tr>
<th>Traditional</th>
<th>Proposed revision</th>
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<tbody>
<tr>
<td><strong>Classification of degrees of certainty in diagnosis</strong></td>
<td><strong>Classification of degrees of certainty in diagnosis</strong></td>
</tr>
<tr>
<td>1. Documented</td>
<td>1. Documented (as in original)</td>
</tr>
<tr>
<td>Remittance with suggestion, psychotherapy, psychotherapy, placebo, ‘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>

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*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].
acknowledged ‘peripheral’ movement disorder developing in an amputated limb) has been considered another example of an organic posttraumatic movement disorder. Zadikoff et al. [35] recently presented a case of psychogenic ‘jumpy stump’, in which paroxysmal rhythmic jerking movements of the stump were associated with palpable muscle activation in the proximal limb. Electrophysiological analysis confirmed reciprocal contraction of hamstrings and quadriceps at 8 Hz, increased EMG activity with stump restraint (indicating voluntary effort against resistance), variability, and distractibility with coherence. This case cautions against the uncritical application of the diagnosis of ‘jumpy stump’ as organic, but the true prevalence of psychogenic movements in such patients remains unclear without further systematic study.

Psychogenic tremors
Psychogenic tremor can be remarkably variable in its presentation, as supported by recent case reports [19], as well as severe and disabling. Recent experience suggests that the diagnosis should be considered in patients failing deep brain stimulation surgery for tremor [36]. In recent years, it has become clear that some patients who would have been previously diagnosed as ‘essential palatal tremor’ have a psychogenic cause. Recognizing the heterogeneous nature of ‘essential palatal tremor’, including a PMD, a learned movement, and tics, Zadikoff et al. [37] proposed the term ‘isolated palatal tremor’ to avoid confusion with other more homogeneous disorders such as essential tremor and essential myoclonus (now myoclonus dystonia). Psychogenic palatal tremor may or may not have extrapalatal movements. As in other forms of psychogenic tremor, psychogenic palatal tremor is typically variable in frequency (often changes in response to challenging motor tasks), and it may be entrained and suppressed by distracting maneuvers. By definition, the MRI is always normal [38]. One patient with a 2 Hz palatal tremor restricted to the posterior soft palate determined to be psychogenic even had a sensory trick that suppressed the tremor [39]. Finally, psychogenic suprathyroid neck tremor was recently reported; this appeared suddenly, was irregular, disappeared with open mouth or with distraction, and resolved with placebo injection [40].

Investigations of how to better identify psychogenic tremor are ongoing. Kenney et al. [41] studied what features might distinguish psychogenic tremor from essential tremor, given that sensitivity and specificity of psychogenic clues generally lack systematic evaluation in this context. Psychogenic tremor was differentiated by negative family history, sudden onset, spontaneous remission, shorter duration of tremor, suggestibility, and distractibility. Curiously, entrainment was not seen often in either tremor type. The authors’ claim that the results have predictive value is limited by the fact that essential tremor and psychogenic tremor were diagnosed prior to assessment of distinctive parameters, and sample size was small. In addition, the method of evaluating entrainment (10 s of wrist extension and flexion in the unaffected arm) is probably not an adequate assessment of this feature. Kumru et al. [42] assessed whether dual task interference (difficulties with carrying out tasks simultaneously with both arms) could distinguish psychogenic tremor from Parkinsonian rest tremor and essential tremor. Only psychogenic tremor patients showed delayed reaction time when they performed a simple reaction time task to a visual stimulus with their nontremulous side when tremor was present compared with when it was not. It is unclear whether this method is superior to classic distraction paradigms (with respect to ease of testing, reliability, sensitivity, and specificity) and it requires that the tremor be intermittent rather than persistent.

Evaluation of long-term prognosis of psychogenic tremor has proven difficult, as underscored by two recent studies with a similar follow-up period (3 years). Earlier investigations have reported tremor remission rates of only 20%. McKeon et al. [43] arrived at a similar pessimistic outlook, finding a 65% disability rate on patient-reported disability scales. In contrast, Jankovic et al. [44] reported a better prognosis with a 60% improvement rate. However, they included any degree of improvement, as measured on a global rating scale, their patients had a shorter duration of symptoms at the time of diagnosis (0.9 years versus 1.5 years in the McKeon study), and they had a greater proportion of patients enrolled in treatment. This study also lacked a prospective follow-up design and confirmatory electrophysiological assessment of psychogenic tremor used in the McKeon report. Nonetheless, favorable prognostic signs were determined, which included the patient’s perception of effective treatment by the physician, elimination of stressors, and compliance with the treatment regimen. No statistical difference in long-term outcome was found between patients involved and not involved in litigation.

Psychogenic gait disorders
Given the variable presentation of psychogenic gait [45], formal tests definitively identifying this condition would be very helpful but have yet to be developed. On the basis of Paul Blocq’s original description of an astasia–abasia patient who effectively propelled a chair while being seated, Okun et al. [46] assessed whether this ‘chair test’ could distinguish psychogenic from organic gait. Eight of nine patients with psychogenic gait moved much better sitting in a chair than when walking in the upright position, whereas nine organic gait disorder patients performed similarly in both conditions. Although promising, test sensitivity and specificity need to be assessed with a larger sample size and a more diversified control group (the control group had seven Parkinson’s disease...
neurometabolic diseases, and other causes.

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.

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 hemispheric 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 perfectionism 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).


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.


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.


This report indicates that electrophysiological testing may not always be able to differentiate psychogenic from organic PSM, placing emphasis on clinical acumen. Perhaps surprisingly, though confirmation of a PSM was clinically not difficult in this case, several electrophysiological features seen in organic PSM were also recorded from this patient (though the recording conditions for the absent Bereitschaftspotential were not described).


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


Secondary contractures are usually observed with organic disease. However, this pediatric case illustrates that they can also be seen with PMDs.


This article [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.


These two recent reviews [48, 49] constitute a comprehensive analysis of PMDs in children. They provide not only valuable data on epidemiology and long-term prognosis but also on similarities to and differences from PMDs in the adult population. The comparison with adult PMDs is particularly well addressed in the discussion section of the Schwingenschuh article.


This report underscores the potential of TMS as a therapeutic modality, indicating that benefit can be achieved in a psychogenic patient who has longstanding symptoms that were resistant to other classic treatment approaches.
