HHS Public Access

Author manuscript

Curr Neurol Neurosci Rep. Author manuscript; available in PMC 2016 February 09.

Published in final edited form as:

Curr Neurol Neurosci Rep. 2011 August ; 11(4): 396–403. doi:10.1007/s11910-011-0205-z.

An Update on Psychogenic Movement Disorders

Aviva Ellenstein, **Sarah M. Kranick**, and **Mark Hallett**

Human Motor Control Section, Medical Neurology Branch, National Institute of Neurological Disorders and Stroke, National Institutes of Health, Bethesda, MD, USA

Abstract

Psychogenic movement disorders (PMD) and other conversion disorders (CD) with apparent neurological signs (neurologic CD) plague patients and perplex physicians. Due to a lack of objective evidence of underlying brain lesions, CD were largely abandoned by neurologists and remained poorly understood psychiatric diagnoses throughout most of the 20th century. Modern neuroscience now supports increasingly comprehensive biological models for these complex disorders, definitively establishing their place in both neurology and psychiatry. Indeed although it is often clinically useful to distinguish a movement disorder as either “organic” or “psychogenic,” this dichotomy is difficult to defend scientifically. Here we describe the neuroimaging and neurophysiologic evidence for dysfunctional neural networks in PMD, explain the diagnostic potential of clinical neurophysiologic testing, discuss the promising if increasingly complex role of neuropsychiatric genetics, and review current treatment strategies.

Keywords

Movement Disorder; Psychogenic; Motor Conversion; Somatoform Disorder; Dissociative Disorder; Functional Disorder; Medically Unexplained; Hysterical; Neurologic Symptom; Diagnostic Technique

Introduction

Leading the establishment of modern neurology in 19th century Paris, Jean-Martin Charcot emphasized a neuroanatomic basis for *les névroses hystériques*(1, 2). This proposed etiology suffered from a lack of objective evidence in an era pre-dating clinical neurophysiology or neuroimaging and soon fell out of favor; however, his opinion that underlying functional lesions followed from traumatic events contributed to influential psychodynamic models of CD(1, 2). As the notion of a causative psychological stressor gained acceptance, CD became the purview of psychiatry and remained the subject of much theoretical discussion, if limited investigation, throughout most of the 20th century(1, 2).

corresponding author: Aviva Ellenstein, M.D. Ph.D., Human Motor Control Section, NINDS, NIH, Building 10, Room 7D42, 10 Center Drive, MSC 1428, Bethesda, MD 20892-1428, tel: 301-402-3496, fax: 301-480-2286, ellensteina@ninds.nih.gov.
Sarah M. Kranick, M.D., Human Motor Control Section, NINDS, NIH, Building 10, Room 7D42, 10 Center Drive, MSC 1428, Bethesda, MD 20892-1428, tel: 301-443-3475, fax: 301-480-2286, mattes1@ninds.nih.gov
Mark Hallett, M.D., Chief, Human Motor Control Section, NINDS, NIH, Building 10, Room 7D37, 10 Center Drive, MSC 1428, Bethesda, MD 20892-1428, tel: 301-496-9526, fax: 301-480-2286, hallettm@ninds.nih.gov

While neurologic diagnosis has improved significantly, patients with PMD remain common and poorly understood. It has been reported that PMD represent 3% of diagnoses in movement disorder clinics and up to 20% in more specialized clinics(3). PMD affects women more than men and can occur at any age, although it tends to be more prevalent in young adults than children or the elderly(4). Patients can present with any type of involuntary movement, the common forms being tremor, dystonia, myoclonus, gait, and parkinsonism, and the phenomena are often mixed and atypical(1). The notion that a movement cannot be psychogenic if it cannot be produced volitionally (e.g., by medical professionals evaluating the patient) is false: PMD are manifestations of dysfunctional motor control networks that can sometimes produce bizarre movements that cannot be mimicked. Evolving nomenclature ranging from abandoned ancient perceptions about the wandering uterus (hysteria) to clinical phenomenology (dynamic or functional) to suspected psychological mechanism (conversion or dissociation) to presumed cause (psychogenic or medically unexplained) complicates discussion with patients and among medical professionals. Due to response to psychotherapy and common psychiatric co-morbidities, psychological dysfunction is believed to underlie PMD pathogenesis, and “PMD” is the currently prevailing term in neurology literature. Although one study showed that patients prefer the descriptive term “functional,”(5) most reject or are reluctant to accept this diagnosis or recommended treatment(3). The differential diagnosis of PMD includes factitious disorder and malingering, in which abnormal movements are produced voluntarily; however, such cases are rare.

Neurologic diagnostic criteria for PMD emphasize using positive clinical findings such as inconsistency and distractibility(6, 7). They specifically avoid the diagnosis-of-exclusion approach, although advise that PMD and other neurologic diagnoses may co-exist, and do not require identification of a preceding life event. Unfortunately, psychiatric diagnostic criteria for CD do require identification of an antecedent stressor, which is often not made in the neurologist’s office(8). Thus, psychiatrists may not recognize the diagnosis or know how to approach patients referred with PMD(9). Even when the diagnosis is both established and accepted, treatment is commonly unsuccessful. It is not surprising that patients frequently end up moving from doctor to doctor, seeking answers we are challenged to provide, hindering recovery, and increasing cost(3).

Modern neuroscience now supports Charcot’s prescient teaching, and leading models of PMD and CD describe a shift from purely psychodynamic toward neurobiological mechanisms(10-12). The neurology community has demonstrated renewed interest in understanding and treating PMD and other CD(4, 13, 14), and the psychiatry community is confronting the necessary challenge of re-defining CD for the fifth version of the Diagnostic and Statistical Manual of Mental Disorders(15-17).

Neuroimaging

Neuroimaging research is at the forefront of establishing neurobiological models for PMD and other neurologic CD, such as psychogenic non-epileptic seizure (PNES), which are thought to share common pathogenic mechanisms. Magnetic resonance imaging (MRI), positron emission tomography (PET), and single photon emission computed tomography

(SPECT) research is beginning to characterize dynamic pathologic changes. Studies are often limited by subject number and are difficult to compare due to differences in design, symptoms, and patient co-morbidities. Although it is unknown whether there is a biological distinction between patients who accept or reject a diagnosis of PMD, studies may also be confounded by including only those patients who presumably accept their diagnosis and have an interest in participating in research. A unified neuroanatomic basis for PMD or CD has yet to be determined; however, dysfunctional regions and networks initially related to sensorimotor control and emotion and increasingly reflecting attention and self-awareness are being identified(12, 18, 19). In comparison with these advances, clinical imaging of PMD is currently limited to a single application in distinguishing Parkinson disease and psychogenic parkinsonism through PET and SPECT.

Imaging Support for a Neurobiological Model of PMD

Avoiding problems related to movement in hyperkinetic PMD, early imaging research focused on hypokinetic PMD such as psychogenic paralysis. PET and SPECT studies with functional paradigms evaluated patients trying to move the psychogenically paralyzed limb, imagining movement of the limb, or feigning immobility of the unaffected limb and demonstrated abnormal activity in networks spanning motor and limbic areas, including the prefrontal and anterior cingulate cortices(12). The pattern of activation seen in these studies initially led to the proposal that sensorimotor areas may be actively inhibited by limbic structures, preventing the limb from moving normally. Vuilleumier noted that these results may be confounded by limbic activity associated with the stress of trying to move a subjectively paralyzed limb, or of the neuroimaging experience itself(12). Additionally the concept of limbic inhibition of motor pathways would not explain the production of hyperkinetic PMD, which not only appear to utilize similar pathways as voluntary movement but likely share a common pathogenic mechanism with hypokinetic PMD(20).

Circumventing complications of volition and motor system activation, Vuilleumier et al. performed a SPECT study of psychogenic hemisensory loss measuring blood flow during tuning fork stimulation of the affected and unaffected limbs during the active phase of the CD and following its resolution(21). They found hypoactivation of the thalamus and basal ganglia contralateral to the affected limb, which normalized with symptom resolution, implicating pathogenic striatothalamocortical circuits. Vuilleumier has proposed a primary role for basal ganglia dysfunction in PMD based on their response to motivation and environmental cues in modulating movement, which may support recent phylogenetic theories about a psychoadaptive role of CD(12).

Event-related functional MRI studies have expanded examination of psychogenic paralysis. In a study investigating motor imagery (MI), imagining or visualizing an action without actually performing it, in eight patients with unilateral conversion paralysis, de Lange et al. showed increased activation of the superior temporal gyrus and medial prefrontal cortex during MI of the affected compared to the unaffected limb(22). Although limited by the absence of healthy controls, this study demonstrated pathologically increased activity of areas thought to engage during self-reflection and disengage during performance of goal-directed tasks(22). Similarly Cojan et al. compared one patient with conversion paralysis of

the left hand to 24 healthy controls moving normally and six healthy controls feigning hand paralysis during a go-no go task(23). In comparison with healthy controls moving the left hand, the patient had increased activation in the precuneus and ventrolateral frontal gyrus during attempted movement of the affected, left hand. Because these regions have also been associated with self-representation and emotional regulation, this study supports the idea that the self may be over-represented during motor conceptualization in CD. Additionally the patient had no activation in right frontal areas during go trials for the affected, left hand, but increased activation during nogo trials for the right hand, while healthy volunteers feigning paralysis had increased activation for both tasks. These results suggest distinct mechanisms of inhibition subserve simulated and psychogenic paralysis.

The most recent contributions of neuroimaging research have shown interesting results in functional connectivity, using such analysis methods as psychophysiologic interaction (PPI) to investigate coupling of task-related state with neural activity. Cojan et al. demonstrated task-specific, increased connectivity between the motor cortex and the posterior cingulate cortex, precuneus, and ventromedial prefrontal cortex in their patient(23). In contrast, De Lange et al. performed PPI analysis focused on the prefrontal cortex and sensorimotor areas(24) and did not demonstrate connectivity between the ventromedial prefrontal cortex and the motor network during their MI task. Rather they found increased interactions between the dorsal premotor and dorsolateral prefrontal cortices, which was greatest during MI of the affected hand.

The connectivity of limbic and frontal areas in patients with PMD was further demonstrated by our group in two studies of hyperkinetic PMD using functional MRI and PPI analyses. In the first study eight patients with chronic, intermittent psychogenic tremor were asked to either “trigger” or voluntarily mimic their tremor(18). During the psychogenic tremor, there was not only decreased activation in the right temporoparietal junction (TPJ) but also decreased connectivity between this region, the sensorimotor areas, and limbic regions. Voon et al. proposed that decreased activity in the TPJ related to diminished feed-forward signals related to self-agency and motor planning, thereby reducing conscious awareness of movement generation. This intriguing concept requires further study to assess its generalizability to different manifestations of hyperkinetic PMD and other CD(25). In the second study Voon et al. presented standardized emotional stimuli to 16 patients with varying forms of hyperkinetic PMD and to healthy controls(19). Patients showed the same level of right amygdala activation for fearful faces as healthy controls but increased activation for happy faces. Moreover, this activation failed to habituate only in patients when happy faces were shown repeatedly. Greater functional connectivity was also seen in patients compared to the healthy controls between the right amygdala and right supplementary motor area during fearful or happy stimuli compared to neutral stimuli. This study provides functional neuroanatomic evidence supporting the theory that the psychological factors contribute to pathophysiology of PMD.

Dopamine Neuroimaging in Psychogenic Parkinsonism

Psychogenic parkinsonism accounts for up to 0.5% of parkinsonism(1). The possibility of a placebo effect being interpreted as a positive dopaminergic trial and committing a patient to

unnecessary and potentially harmful medication or surgery makes this rare entity an important clinical consideration in the differential diagnosis of Parkinson disease (PD). Several studies have shown promise for imaging dopamine transporters and uptake in the striatum to distinguish between “organic” and psychogenic parkinsonism by confirming a normal scan in the latter(26). There is also an increasing appreciation for coexisting psychogenic and “organic” PD, when patients should be treated for both disorders(27). Further complicating this diagnosis is the uncommon phenomenon of scans without evidence of dopaminergic deficit in patients with early or pre-clinical PD. Thus, patients with clinical psychogenic parkinsonism may need to undergo longitudinal imaging and routine examination before the sole diagnosis of PMD is established. Patients with defective enzymes in dopamine synthesis pathways, such as those with guanosine triphosphate-cyclohydrolase-1-related dopa-responsive dystonia, may demonstrate a significant improvement with dopamine administration and have normal dopamine imaging; however, if clinically indicated, genetic testing or cerebrospinal fluid analysis could establish these diagnoses(20).

Physiologic Support for a Neurobiological Model of PMD

Neurophysiologic investigations suggest that psychogenic movements utilize the same neural networks as those for voluntary movement(20). Consistent with neuroimaging findings of dysfunctional networks that link psychological factors and executive functions to sensorimotor control, the collective results of various studies utilizing electroencephalography (EEG), event-related potentials, magnetoencephalography, transcranial magnetic stimulation (TMS), action observation, and motor imagery suggest normal activation of primary sensory and motor pathways but disruptions in higher level cortical pathways, which may serve to integrate perceptions about self and motivation(12, 20, 28). Although not explicitly aimed at investigating PMD, Desmurget et al. took advantage of the rare opportunity to perform a study of direct electrical stimulation (DES) to the cortex of awake individuals during surgery(29). DES of premotor cortex caused movement without awareness, and DES of posterior parietal cortex caused current-dependent perceptions of movement intention or illusory movement. These findings provide direct evidence that discrete cortical regions are functionally responsible for movement generation, planning, or perception. Related dysfunctional networks that effect movement independent of intent likely underlie the involuntary nature of PMD.

Clinical Neurophysiologic Testing

Clinical neurophysiologic testing can assist the neurologist with unclear diagnoses of certain types of PMD. Distinguishing PMD from other movement disorders is usually accomplished by recognizing that the movements are inconsistent with “organic” movement disorders and incompatible with the neurologic examination. In some cases, such as chronic PMD where the movement may have become so over-learned that it is difficult to modulate, or in cases of mixed psychogenic and “organic” movement disorders, it may not be possible to definitively diagnosis PMD by clinical examination alone. Although the diagnosis may not be in question to the neurologist, some patients will be more accepting and likely to seek treatment if there is objective evidence from a test to support the clinical diagnosis.

The utility of clinical neurophysiologic testing for psychogenic tremor relies upon a critical feature that distinguishes it from parkinsonian, essential, physiologic, drug-induced, or other "organic" tremors: it is not driven by an independent and continuous generator and can be modulated by external stimulation. Distraction, ballistic, and entrainment maneuvers will promote cessation or frequency matching of the tremor, which is readily objectively measured by accelerometry and frequency analysis(20). Several other features such as frequency and amplitude variability, coherent frequencies in different limbs, and the agonist-antagonist co-activation at the onset of tremor can also be diagnostically useful in cases of psychogenic tremor(Table 1)(20). The neurophysiologic evaluation of psychogenic myoclonus is grounded in electromyography (EMG) and EEG, and in the case of reflex myoclonus derives from reflex latency measurement. Diagnosis is based on comparing measures of EMG burst length, antagonist muscle pattern, and back-averaged EEG potentials with normative data for epileptic and non-epileptic, "organic" myoclonus(Table 2)(20). The readiness potential or *Bereitschaftspotential* (BP) is an average of small potential changes in the EEG that precede and are time-locked by EMG to discrete movement events. Reflecting premotor cortical activity, the BP is seen with psychogenic myoclonus as well as voluntary movements. Interestingly, this result indicates that the BP does not designate voluntariness(20). A recent report demonstrated the utility of measuring blink reflex recovery to distinguish psychogenic from benign essential blepharospasm(30); however, neurophysiologic analysis of psychogenic dystonia is usually complicated by a typically fixed posture and the lack of specific features to distinguish it from "organic" dystonia(20). TMS can be used in psychogenic weakness to demonstrate normal function of the corticospinal tract.

Neuropsychiatric Genetics

To date there is no known genetic risk factor for PMD or other neurologic CD. As genetics has evolved to reveal not only the pathologic implications of single nucleotide polymorphisms but also the power of epigenetic influences, the search for multi-factorial risk factors has exploded throughout neuroscience(31-33). Leading models for neuropsychological health and disease suggest a combined influence of genetic and environmental influences, with disorders occurring only at a critical, interactive threshold(34, 35). Given this increasing appreciation for the complexity of gene-environment and gene-gene risk factors, the identification of specific genes that contribute to the biological predisposition for PMD becomes practically complicated by the need for both large patient populations with similar phenotypes as well as assessment of relevant environmental factors. Although the literature has often described early childhood trauma, particularly sexual abuse, in association with CD, influences as varied as exposures *in utero*, toxins, infections, other stressful events, parenting styles, as well as their timing and duration may be involved(34, 36).

Specific genetic risk factors for PMD almost certainly exist. Genetic risk varies widely among other psychiatric disorders, ranging from 25% for social phobias and certain personality disorders to over 80% for schizophrenia, bipolar disorder, and autism(34). The association of PMD with specific psychiatric disorders derives from several case-series and chart review studies, with sample sizes ranging from five to 127 subjects(9). The most

commonly reported axis I disorder is depression, with a prevalence ranging from 19% to 57%; however, several of these studies suffered from self-reporting bias and unstructured interviews. A prospective study evaluating PMD patients specifically for psychiatric diagnoses using structured interviews found anxiety disorders to be most prevalent at 38%(37). This finding was similar to the anti-depressant treatment study by Voon et al. in which anxiety disorder had a prevalence of 52%(38). An important starting point in our understanding of the psychological profile of patients with PMD, these studies specifically support focusing PMD genetic research efforts on factors implicated in these associated disorders.

Candidate genes with potentially pathogenic variability related to PMD range from regulators of neurotransmitters that underlie pharmacotherapy of affective disorders to neuropeptides implicated in neuroplasticity to modulators of stress responses. These include but are not limited to the serotonin transporter gene-linked polymorphic region (5-HTTLPR)(32), catechol-o-methyltransferase (COMT)(33), corticotropin releasing hormone binding protein (CRHBP)(39), a glucocorticoid receptor-regulating gene (FKBP5)(34), brain-derived neurotrophic factor (BDNF)(9, 35), neuropeptide Y (NPY)(40), and fatty acid amid hydrolase (FAAH)(31).

Treatment

PMD treatment begins with confident communication about the diagnosis and acknowledgment of its severity. The reader is encouraged to see the practical recommendations for the neurologist's approach to CD by Jon Stone for particularly important considerations about communication and management(41). An exceptionally useful and educational reference for patients is the website www.neurosymptoms.org maintained by the same author. The outcome for patients with PMD worsens with delays in diagnosis and treatment. Therefore, after the diagnosis of PMD is established, the patient should be promptly referred for psychiatric consultation, including initiation of treatment for any co-morbid psychiatric illness, and psychotherapy. Although symptom reversal may be the most immediate goal of treatment, it is important to address any underlying psychopathology that may predispose the patient for further development of CD. The mainstay of therapy for CD since the work of Sigmund Freud, psychotherapy has evolved to include several approaches beside psychoanalytic and is increasingly supported by clinical research. A variety of other treatment modalities may contribute to an effective multi-factorial approach for patients with PMD, but they have not been extensively studied. Physical therapy is widely considered to be an important adjunct to psychotherapy, providing a structured setting in which patients can realize their potential for normal movement. Anti-depressants, biofeedback, and hypnosis may also be considered on a case-by-case basis. The ethics and utility of placebo are debated. The most successful treatment regimens for CD are those carried out at specialized in-patient centers using a multi-disciplinary team. According to one report, 81% of patients treated for one week to six months at such a center had resolution of their symptoms(1). Unfortunately, this result stands in stark contrast to most studies, which report 44%-90% of patients having persistent symptoms years after presentation(1). Although the reasons for this disparity are unclear,

two likely factors are that most patients are treated in less effective out-patient settings and many patients refuse treatment.

Psychiatric consultation is imperative and integral to treatment success. The reasons for referral are twofold: (1) to provide the necessary psychological evaluation and management in this patient population with common psychiatric co-morbidities, and (2) to allow the psychiatrist to recommend the optimal form of psychotherapy. Although the immediate goal of psychotherapy is the resolution of the abnormal movements, it may also serve to improve psychological health in general as well as to prevent development of further new functional symptoms once the initial ones clear. This is not an uncommon scenario. Even when no psychiatric co-morbidity is identified, as has been repeatedly reported in PMD cohorts(9), psychotherapy might still be useful. The focus is typically not on the movement itself but on the patient's coping strategies, often related to any difficult life event but sometimes on general stress management.

Several small studies have demonstrated the efficacy of psychotherapy for PMD and other neurologic CD. Hinson et al. conducted a single-blind clinical trial in ten patients with PMD in which patients received 12 weeks of one-hour weekly psychodynamic psychotherapy(42). Patients also received anti-depressants or anxiolytics as indicated. Means for the PMD rating scale, function scores, Hamilton depression scores, and Beck anxiety scores all improved. In a study of 79 patients with medically unexplained symptoms who were randomly assigned to receive cognitive behavioral therapy (CBT) or optimum medical management, the CBT group achieved a lower intensity of physical symptoms and a higher recovery rate(43). Several studies of CBT using a manual-based therapy for PNES have demonstrated good responses(14), which was also found for a patient with PMD(44). The effective use of mindfulness-based psychotherapy for a patient with complex neurologic CD including PMD was recently reported(45). In a study comparing standard medical practice with or without CBT for PNES that demonstrated a benefit of CBT, the suicide of a subject from the CBT arm during follow-up echoes other studies of long-term prognosis and emphasizes the need for psychiatric care in the CD population(1, 46).

There is little specific evidence to support or direct pharmacotherapy for PMD. In the only prospective treatment trial of motor conversion with anti-depressants, 15 patients were treated with either citalopram or paroxetine(38). Non-responders were switched to venlafaxine. Two sub-groups were observed in the study. Ten patients were considered to have "primary psychogenic movement disorder," and all had co-morbid axis I diagnoses. The remaining five patients were labeled "psychogenic movement disorder plus other somatoform disorder" and were diagnosed with primary hypochondriasis, somatization, and probable factitious disorder or malingering; only 40% of these patients had co-morbid axis I diagnoses. Eight of ten patients in the first group had significant improvements in scales of clinical global impression, depression, and anxiety, and seven had a complete remission. Few received concurrent psychotherapy, leading the authors to conclude that this treatment effect was likely due to medication. The second group did not show improvement, raising the possibility that those patients may not only be more difficult to diagnose psychiatrically but also more difficult to treat.

The purpose of physical therapy is to provide patients with a structured program through which they can progressively experience normal movement function and realize their treatment potential. Toward this goal, neurologists may take advantage of the opportunity to educate patients that the presence of normal movements in the neurologic exam means treatment is possible. While commonly recommended, there are few studies of physical therapy in PMD or CD management. A recent study of patients with PMD treated with a mild walking program showed improvement in 10 of 16 individuals(47).

There are few studies of complementary and alternative medical treatments for PMD. One case report describes a dramatic response to acupuncture in a patient with chronic, treatment-resistant PMD(48). In a randomized controlled trial of hypnosis, 44 patients with CD motor type or somatization disorder with motor conversion symptoms either received hypnosis or were put on a waiting list(49). The hypnosis group demonstrated greater improvement for up to six months. In a published abstract EMG biofeedback was a successful treatment in nine of 15 patients with psychogenic tremor (1). There have been a few reports of treatment with TMS(50). In a study of 70 adults and children with psychogenic paralysis in which repetitive TMS was used as a diagnostic tool to demonstrate normal motor evoked potentials and in which some patients were told it could be potentially therapeutic, the testing produced a total recovery or dramatic improvement in 89% of patients, which persisted for several months in most subjects. While the use of placebo without the patient’s consent is often considered unethical, the argument has been made that physicians treating psychogenic disorders could make appropriate use of placebos in diagnosis and treatment. The high rate of placebo response in “organic” disorders makes this approach all the more complicated. The distinction between physiologic and placebo effects for alternative methods particularly in a population with neuropsychiatric disorders is often unclear, and the risk for recurrence or development of new medically unexplained symptoms remains a concern.

Conclusion

Long explained as mysterious manifestations of the dynamic unconscious, PMD are increasingly recognized as neurologic disorders. Indeed neuroimaging, neurophysiologic, and multi-factorial genetic research in related neuropsychiatric disorders are converging to support neurobiological models for PMD and related CD. In addition to future neuroimaging and neurophysiologic studies focused on particular movement phenomena, investigations also considering behavioral traits and genetic variation may support more comprehensive biological understanding of PMD. In addition, as candidate genetic risk factors emerge and genetic studies become technically easier and less costly to perform, it is important to consider the potency of gene-gene and gene-environment interactions when planning and interpreting studies of PMD risk. Neurophysiologic investigations can support the clinical diagnosis of PMD, with well-established criteria for evaluating psychogenic tremor and myoclonus.

Despite these advances in our neurobiological understanding and clinical diagnosis of PMD, treatment remains grounded in psychotherapy and is generally unsuccessful. Although psychiatric co-morbidity studies are limited, the risks associated with the frequently co-

existing affective disorders, associated implications for pharmacologic management, and utility of a comprehensive psychological evaluation in choosing psychotherapeutic modality require psychiatric referral and continued care. As neuropsychiatric genetics advances, it is expected that more individualized behavioral and medical management will lead to improved treatment of PMD. For now neurologists are obliged to educate the patients that PMD has a firm, if incompletely understood, neurologic basis and it can be treated through psychotherapy.

References

1. Hallett, M.; Fahn, S.; Jankovic, J., et al. Psychogenic Movement Disorders: Neurology and Neuropsychiatry. Lippincott Williams & Wilkins; Philadelphia, PA: 2006.
2. Bogousslavsky J. Hysteria after Charcot: back to the future. Front Neurol Neurosci. 2011; 29:137–161. [PubMed: 20938153]
3. Hallett M. Psychogenic movement disorders: a crisis for neurology. Curr Neurol Neurosci Rep. 2006; 6:269–271. [PubMed: 16822346]
4. Nowak DA, Fink GR. Psychogenic movement disorders: aetiology, phenomenology, neuroanatomical correlates and therapeutic approaches. Neuroimage. 2009; 47:1015–1025. [PubMed: 19426818]
5. Stone J, Wojcik W, Durrance D, et al. What should we say to patients with symptoms unexplained by disease? The “number needed to offend”. BMJ. 2002; 325:1449–1450. [PubMed: 12493661]
6. Fahn S, Williams DT. Psychogenic dystonia. Adv Neurol. 1988; 50:431–455. [PubMed: 3400501]
7. Williams DT, Ford B, Fahn S. Phenomenology and psychopathology related to psychogenic movement disorders. Adv Neurol. 1995; 65:231–257. [PubMed: 7872143]
8. American Psychiatric Association. American Psychiatric Association Task Force on DSM-IV. Diagnostic and statistical manual of mental disorders : DSM-IV-TR. 4th. American Psychiatric Association; Washington, DC: 2000.
9. Kranick SK, Ellenstein A, Hallett M. Psychiatric Comorbidities and Risk Factors in Psychogenic Movement Disorders: A Biopsychosocial Approach. European Neurological Journal. 2010; 2:1–7.
10. Krem MM. Motor conversion disorders reviewed from a neuropsychiatric perspective. J Clin Psychiatry. 2004; 65:783–790. [PubMed: 15291655]
11. Scott RL, Anson JG. Neural correlates of motor conversion disorder. Motor Control. 2009; 13:161–184. [PubMed: 19454778]
12•. Vuilleumier P. Laureys S, Tononi G. The Neurophysiology of Self-Awareness Disorders in Conversion Hysteria. The Neurology of Consciousness Cognitive Neuroscience and Neuropathology. 2009:282–302.Academic Press This chapter cogently discusses the neuroimaging and neurophysiologic studies of conversion hysteria in the context a developing neurobiological model that includes disrupted mechanisms of self-awareness.
13. Stone J, Carson A. Movement disorders: Psychogenic movement disorders: what do neurologists do? Nat Rev Neurol. 2009; 5:415–416. [PubMed: 19657345]
14. LaFrance WC Jr. Miller IW, Ryan CE, et al. Cognitive behavioral therapy for psychogenic nonepileptic seizures. Epilepsy Behav. 2009; 14:591–596. [PubMed: 19233313]
15. Stone J, LaFrance WC Jr. Levenson JL, et al. Issues for DSM-5: Conversion disorder. Am J Psychiatry. 167:626–627. [PubMed: 20516161]
16. Mayou R, Kirmayer LJ, Simon G, et al. Somatoform disorders: time for a new approach in DSM-V. Am J Psychiatry. 2005; 162:847–855. [PubMed: 15863783]
17. Lowe B, Mundt C, Herzog W, et al. Validity of current somatoform disorder diagnoses: perspectives for classification in DSM-V and ICD-11. Psychopathology. 2008; 41:4–9. [PubMed: 17952015]
18. Voon V, Gallea C, Hattori N, et al. The involuntary nature of conversion disorder. Neurology. 2010; 74:223–228. [PubMed: 20083798]

19. Voon V, Brezing C, Gallea C, et al. Emotional stimuli and motor conversion disorder. Brain. 2010; 133:1526–1536. [PubMed: 20371508]
20. Hallett M. Physiology of psychogenic movement disorders. J Clin Neurosci. 2010; 17:959–965. [PubMed: 20493708]
21. Vuilleumier P, Chicherio C, Assal F, et al. Functional neuroanatomical correlates of hysterical sensorimotor loss. Brain. 2001; 124:1077–1090. [PubMed: 11353724]
22. de Lange FP, Roelofs K, Toni I. Increased self-monitoring during imagined movements in conversion paralysis. Neuropsychologia. 2007; 45:2051–2058. [PubMed: 17367826]
23. Cojan Y, Waber L, Carruzzo A, et al. Motor inhibition in hysterical conversion paralysis. Neuroimage. 2009; 47:1026–1037. [PubMed: 19450695]
24. de Lange FP, Toni I, Roelofs K. Altered connectivity between prefrontal and sensorimotor cortex in conversion paralysis. Neuropsychologia. 2010; 48:1782–1788. [PubMed: 20206641]
25. Stone J, Vuilleumier P, Friedman JH. Conversion disorder: separating “how” from “why”. Neurology. 2010; 74:190–191. [PubMed: 20083794]
26. Gaig C, Marti MJ, Tolosa E, et al. 123I-Ioflupane SPECT in the diagnosis of suspected psychogenic Parkinsonism. Mov Disord. 2006; 21:1994–1998. [PubMed: 16941463]
27. Hallett M. Psychogenic Parkinsonism. J Neurol Sci. 2011 In Press.
28. Liepert J, Hassa T, Tuscher O, et al. Motor excitability during movement imagination and movement observation in psychogenic lower limb paresis. J Psychosom Res. 2010; 70:59–65. [PubMed: 21193102]
29. Desmurget M, Sirigu A. A parietal-premotor network for movement intention and motor awareness. Trends Cogn Sci. 2009; 13:411–419. [PubMed: 19748304]
30. Schwingenschuh P, Katschnig P, Edwards MJ, et al. The blink reflex recovery cycle differs between essential and presumed psychogenic blepharospasm. Neurology. 2011; 76:610–614. [PubMed: 21321334]
31. Hariri AR. The neurobiology of individual differences in complex behavioral traits. Annu Rev Neurosci. 2009; 32:225–247. [PubMed: 19400720]
32•. Caspi A, Hariri AR, Holmes A, et al. Genetic sensitivity to the environment: the case of the serotonin transporter gene and its implications for studying complex diseases and traits. Am J Psychiatry. 2010; 167:509–527. [PubMed: 20231323] Following from a detailed review of the serotonin transporter gene and stress sensitivity, the authors discuss approaches to the challenging field of gene-environment research.
33. Caspi A, Moffitt TE. Gene-environment interactions in psychiatry: joining forces with neuroscience. Nat Rev Neurosci. 2006; 7:583–590. [PubMed: 16791147]
34. Hallett, M.; Lang, AE.; Jankovic, J., et al. Psychogenic Movement Disorders & Other Conversion Disorders. Cambridge University Press; Cambridge: 2011.
35. Feder A, Nestler EJ, Charney DS. Psychobiology and molecular genetics of resilience. Nat Rev Neurosci. 2009; 10:446–457. [PubMed: 19455174]
36•. Lupien SJ, McEwen BS, Gunnar MR, et al. Effects of stress throughout the lifespan on the brain, behaviour and cognition. Nat Rev Neurosci. 2009; 10:434–445. [PubMed: 19401723] The authors synthesize the animal and human research on the age-dependent pathophysiology of stress.
37. Feinstein A, Stergiopoulos V, Fine J, et al. Psychiatric outcome in patients with a psychogenic movement disorder: a prospective study. Neuropsychiatry Neuropsychol Behav Neurol. 2001; 14:169–176. [PubMed: 11513100]
38. Voon V, Lang AE. Antidepressant treatment outcomes of psychogenic movement disorder. J Clin Psychiatry. 2005; 66:1529–1534. [PubMed: 16401153]
39. Binder EB, Owens MJ, Liu W, et al. Association of polymorphisms in genes regulating the corticotropin-releasing factor system with antidepressant treatment response. Arch Gen Psychiatry. 2010; 67:369–379. [PubMed: 20368512]
40. Zhou Z, Zhu G, Hariri AR, et al. Genetic variation in human NPY expression affects stress response and emotion. Nature. 2008; 452:997–1001. [PubMed: 18385673]

41•. Stone J. The bare essentials: Functional symptoms in neurology. Pract Neurol. 2009; 9:179–189. [PubMed: 19448064] This article presents important, practical advice for the neurologist's approach to patients with conversion disorder.

42. Hinson VK, Weinstein S, Bernard B, et al. Single-blind clinical trial of psychotherapy for treatment of psychogenic movement disorders. Parkinsonism Relat Disord. 2006; 12:177–180. [PubMed: 16364676]

43. Speckens AE, van Hemert AM, Spinhoven P, et al. Cognitive behavioural therapy for medically unexplained physical symptoms: a randomised controlled trial. BMJ. 1995; 311:1328–1332. [PubMed: 7496281]

44. LaFrance WC Jr. Friedman JH. Cognitive behavioral therapy for psychogenic movement disorder. Mov Disord. 2009; 24:1856–1857. [PubMed: 19562779]

45. Baslet G, Hill J. Case Report: Brief Mindfulness-Based Psychotherapeutic Intervention During Inpatient Hospitalization in a Patient With Conversion and Dissociation. Clinical Case Studies. 2011 In press.

46. Goldstein LH, Chalder T, Chigwedere C, et al. Cognitive-behavioral therapy for psychogenic nonepileptic seizures: a pilot RCT. Neurology. 2010; 74:1986–1994. [PubMed: 20548043]

47. Dallocchio C, Arbasino C, Klersy C, et al. The effects of physical activity on psychogenic movement disorders. Mov Disord. 2010; 25:421–425. [PubMed: 20108357]

48. Van Nuenen BF, Wohlgemuth M, Wong Chung RE, et al. Acupuncture for psychogenic movement disorders: treatment or diagnostic tool? Mov Disord. 2007; 22:1353–1355. [PubMed: 17486612]

49. Moene FC, Spinhoven P, Hoogduin KA, et al. A randomized controlled clinical trial of a hypnosis-based treatment for patients with conversion disorder, motor type. Int J Clin Exp Hypn. 2003; 51:29–50. [PubMed: 12825917]

50. Chastan N, Parain D. Psychogenic paralysis and recovery after motor cortex transcranial magnetic stimulation. Mov Disord. 2010; 25:1501–1504. [PubMed: 20568093]

Table 1

Clinical Neurophysiology of Psychogenic Tremor

Tremor Physiology	**"Organic"**	**Psychogenic**
Frequency	Characteristic	Variable
Variability	Minimal	Marked
Limb Coherence	Exceptional	Routine
Amplitude Variability	Moderate	Marked
Dual Task		
External Pacing	No Interference	Tremor Entrainment, Cessation, or Change
Ballistic Movement	No Interference	Tremor Cessation

Table 2

Clinical Neurophysiology of Psychogenic Myoclonus

Myoclonus Physiology	Epileptic	Non-Epileptic	Psychogenic
EMG Burst Length	30-50 msec	> 50 msec	>50 msec
EMG Antagonist Pattern	Synchronous	Variable	Variable
EEG Correlate	ΔV < 20 sec	None	BP

Reflex Myoclonus	"Organic"	Psychogenic
C-reflex Latency	< 50 msec	> 100 msec Variable

ΔV = voltage change, BP = *Bereitschaftspotential*