Antidepressant Treatment Outcomes of Psychogenic Movement Disorder

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Background: Psychogenic movement disorder (PMD) is a subtype of conversion disorder. We describe the outcomes of a series of PMD patients following antidepressant treatment.

Method: Twenty-three outpatients with chronic PMD, diagnosed using Fahn and Williams' criteria, underwent psychiatric assessment. The patients were referred for assessment and management from January 2003 to July 2004. Fifteen agreed to be treated with antidepressants. Patients received citalopram or paroxetine; those who did not respond after 4 weeks of taking an optimal dose were switched to venlafaxine. Concurrently, 3 had supportive psychotherapy, and 1 had family intervention. Assessments included the DSM-IV–based Mini-International Neuropsychiatric Interview and scales measuring depression, anxiety, and motor and global severity.

Results: Eighteen patients (78%) had at least 1 Axis I diagnosis in addition to the somatoform diagnosis, and 3 (13%) had somatization disorder. Five (22%) had previous psychiatric contact. Nine (39%) had previously been treated with antidepressants, but only 4 (17%) had adequate trials. No significant differences existed in patient characteristics between treated and untreated groups. Among treated patients, Montgomery-Asberg Depression Rating Scale scores improved from baseline ($p < .01$). Two treated subgroups were identified: 10 patients (67%) had primary conversion disorder, of whom 8 had marked motor and global improvements with 7 complete remissions, and 5 (33%) had primary hypochondriasis, somatization disorder, or probable factitious disorder/malingering, of whom none improved. All of the patients with primary conversion disorder had a current or previous depressive or anxiety disorder compared with 40% ($N = 2$) of the patients with additional somatoform diagnoses.

Discussion: Our preliminary findings suggest that chronic PMD with primary conversion symptoms and with recent or current depression or anxiety may respond to antidepressants. Further well-designed studies, now under way, are required to confirm these findings.

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Psychogenic movement disorder (PMD) is a diagnosis characterized by movement symptoms (such as tremor, dystonia, myoclonus, parkinsonism, and gait disorders) without an underlying neurologic cause.[1,2] PMD is a subtype of conversion disorder,[1] a broader term defined in the *Diagnostic and Statistical Manual of Mental Disorders*, Fourth Edition (DSM-IV),[3] as unexplained neurologic symptoms related to underlying psychological factors.

Psychogenic movement disorder is diagnosed in 2% to 3% of patients in movement disorder clinics.[1] The prognostic outcome in chronic PMD (> 3 months) is commonly described as poor in the literature, with less than 45% of patients seeing improvement or remission of their symptoms.[4] The poor prognosis for PMD patients is consistent with the poor prognosis generally reported for chronic conversion disorder patients.[5,6]

The criteria for conversion disorder require an association with an underlying psychological cause (criterion B).[3] Criterion B (i.e., determining an associated nonconscious psychological association) has been criticized for its subjective nature, bringing into question the validity of this criterion and limiting systematic study of the disorder.[7,8] Furthermore, additional psychological issues are found to be associated in only half of patients with conversion symptoms.[9] Similar to the widely used diagnoses of conversion disorder and non-epileptic seizures,[10] the diagnosis of PMD has not been systematically validated but is utilized by neurologists and psychiatrists treating patients with PMD.[2]

The diagnosis of PMD as defined by Williams et al.[1] is subdivided into 4 levels dependent on the diagnostic certainty. The "clinically definite" group includes the first 2 categories of diagnosis, which are (1) documented: symptoms persistently relieved by psychotherapy, suggestion,

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or placebo or asymptomatic when observed surreptitiously and (2) clinically established: symptoms inconsistent over time or incongruous with a classic movement disorder along with supportive evidence of definite psychogenic neurologic signs, multiple somatizations, obvious psychiatric disturbances, disappearance of movement with distraction, or excessive slowness of movement. The other 2 categories are (3) possible and (4) probable.[1]

The only controlled treatment trials to date dealing with conversion disorder have involved treatment with hypnosis and neuroleptics.[11–13] The treatment literature has focused primarily on psychotherapy and rehabilitation for patients with conversion disorder and is generally limited by small sample sizes, heterogeneous cohorts, and a lack of controls. The mix of acute and chronic patients and the presence of different conversion subtypes in conversion disorder study populations confound the results of most studies. Antidepressants have been reported to be effective in functional somatic syndromes (for example, disorders such as irritable bowel syndrome and fibromyalgia) and somatization disorder dissociated from a current depression or anxiety disorder.[14,15] Antidepressants are used frequently in the conversion disorder[16,17] and PMD[1] populations, presumably targeting underlying depressive symptoms. However, there are no published studies investigating the efficacy of antidepressants in conversion disorder.

We describe the outcomes of a series of PMD patients after antidepressant treatment.

METHOD

Patients with a diagnosis of PMD according to Fahn and Williams' criteria[1] were included in this study. Exclusion criteria included symptoms that were the result of elaboration of the patient's underlying primary neurologic disorder and medical or neurologic issues that contributed to the patient's movement symptoms. The patients were referred from a movement disorders clinic at the Toronto Western Hospital (Toronto, Ontario, Canada). The clinic has a similar proportion of PMD referrals (2%–3%) as other movement disorder clinics.

These referrals and subsequent treatment arose out of routine clinical practice. Patients were advised that the antidepressants would be used to control their comorbid depressive, anxiety, or subsyndromal psychiatric symptoms and agreed to the use of antidepressants. Patients were told that the psychiatric symptoms may improve but that the effect on motor symptoms was not known, and potential side effects were explained. The patients were told that the diagnosis of PMD was not related to a neurologic diagnosis and that the treatment would consist primarily of psychiatric interventions.

Citalopram or paroxetine was administered orally starting at 10 mg per day; dosage was increased as needed to a maximum of 40 mg per day. Outcomes were assessed at 8 weeks after onset of treatment; patients who did not respond to citalopram or paroxetine after 4 weeks of taking an optimal dose were switched to venlafaxine starting at 37.5 mg per day administered orally and increased as needed to a maximum of 300 mg per day. Assessments were performed at the beginning and at a mean of 3.1 months (SD = 1.2) after the initiation of treatment.

Psychiatric diagnoses were based on the DSM-IV and made on the basis of both an unstructured psychiatric interview and a semistructured interview using the Mini-International Neuropsychiatric Interview (MINI).[18] The MINI is a shortened version of the Structured Clinical Interview for DSM-IV Axis I Disorders. To allow for the assessment of comorbidities, Axis I diagnoses found in the MINI (which does not include the somatoform disorder diagnoses) were made using a non-hierarchical approach. Somatization disorder and hypochondriasis fulfilling DSM-IV criteria[3] were separately diagnosed.

Possible factitious disorder and malingering were diagnosed when there were clear and significant discrepancies in the patient or collateral history. In all observed instances of possible factitious disorder and malingering, significant external gains existed and symptoms resolved prior to referral for a second opinion regarding the underlying etiology of the patient's symptoms. As a clear diagnosis of voluntary production of symptoms could not be made on the initial assessment, these patients were included in the treatment study; over the course of the treatment, the nature of the underlying symptoms became more apparent.

Depression was assessed with the clinician-rated Montgomery-Asberg Depression Rating Scale (MADRS).[19] Anxiety severity was assessed using the patient-rated Beck Anxiety Inventory (BAI).[20] The Clinical Global Impressions-Severity of Illness scale (CGI-S) and -Change scale (CGI-C), focusing separately on motor and global outcomes, were used.[21] The CGI-S is a clinician-rated 7-point scale with "normal" scored as 1 and "among the most extremely ill" scored as 7. As motor and functional improvement may be dissociated, the 2 parameters were assessed separately. These general instruments were utilized as there are no published scales for the severity of PMD. The CGI-C is a clinician-rated scale from 1 to 7 that assesses the change in degree of illness in relation to the original assessment. "Marked improvement" is scored as 1, "much improvement" as 2, "minimal improvement" as 3, "no change" as 4, and "very much worse" as 7. The previous and current use of antidepressants (type, dose, and duration) were reviewed through patient interview, collateral information, and pharmacy record review.

The patients' mean age, duration of illness, and depression scores were compared between the following groups: treated patients, treated patients with conversion disorder only (vs. those with additional somatoform disorders), and untreated patients. The comparison was made using the

Table 1. Characteristics of Patients With Psychogenic Movement Disorder (PMD)

Characteristic	Total (N = 23)	Untreated (N = 8)	Treated		
			Overall (N = 15)	Primary PMD (N = 10)	PMD Plus Other Somatoform Diagnosis (N = 5)
Age, mean (SD), y	47.9 (14.7)	43.5 (13.5)	50.2 (15.7)	54.5 (15.8)	41.8 (6.7)
Gender, N, female:male	18:5	6:2	12:3	7:3	5:0
Marital status, N, married/single	16/7	6/2	10/5	8/2	2/3
Previous psychiatric contact, N (%)	5 (22)	2 (25)	3 (20)	2 (20)	1 (20)
Duration of illness, mean (SD), mo	62.1 (56.7)	73.0 (65.0)	56.3 (50.8)	54.5 (15.8)	41.8 (6.7)

Table 2. Psychiatric Diagnoses and Outcomes of Patients With Psychogenic Movement Disorder (PMD) Treated With Antidepressants

Characteristic	Total (N = 23)	Untreated (N = 8)	Treated		
			Overall (N = 15)	Primary PMD (N = 10)	PMD Plus Other Somatoform Diagnosis (N = 5)
Lifetime and current psychiatric diagnoses, N (%)[a]					
> 2	6 (26)	3 (38)	3 (20)	3 (30)	0 (0)
≥ 1	18 (78)	6 (75)	12 (80)	10 (100)	2 (40)
None	5 (22)	2 (25)	3 (20)	0 (0)	3 (60)
Baseline scores, mean (SD)					
MADRS	12.5 (8.6)	16 (10)	10.6 (6.9)	12.0 (6.9)	7.8 (6.0)
BAI	14.4 (10.4)	8.4 (4.2)	14.8 (11.2)	18.7 (11.9)	8.8 (5.0)
Final scores, mean (SD)					
MADRS			5.2 (3.0)*	4.5 (1.6)	6.6 (4.3)
CGI-C motor			2.3 (1.4)	1.5 (1.0)	4 (0)
CGI-C global			2.4 (1.5)	1.6 (1.2)	4 (0)
Remission of motor symptoms, N (%)			7 (47)	7 (70)	0 (0)

[a]Mini-International Neuropsychiatric Inventory (does not include somatoform disorders, factitious disorder, or malingering).
*$p < .01$ compared with baseline scores.
Abbreviations: BAI = Beck Anxiety Inventory, CGI-C = Clinical Global Impressions-Change scale, MADRS = Montgomery-Asberg Depression Rating Scale.

nonparametric Mann-Whitney test for 2 unmatched samples with Bonferroni correction for multiple comparisons. Significance was assigned if the corrected p value was less than .05.

RESULTS

Twenty-three patients who fulfilled the criteria for "clinically definite" PMD[1] were consecutively referred for outpatient psychiatric assessment and management from January 2003 to July 2004. Four additional patients who had contributory medical or neurologic issues were not included in this group of 23 patients.

Fifteen patients underwent treatment. Eight patients did not undergo treatment due to distance (N = 2), preexisting management by a psychiatrist (N = 2), and reluctance to undergo any form of psychiatric management including psychotherapy or psychotropic treatment (N = 4). The reasons cited for refusing psychiatric treatment were that the patient was planning to pursue naturopathic therapies or that the patient did not believe he or she had a psychogenic disorder. One patient elected to continue psychotherapy with a current psychiatrist.

Table 1 outlines the demographic and background data of the compared groups. The psychogenic movement symptoms included resting, postural, and action tremor (N = 12); fluctuating symptoms of dystonia (N = 3); gait abnormalities (N = 2); tics (N = 1); myoclonus (N = 1), tremor and gait abnormalities (N = 2); tremor and dystonia (N = 1); and tremor and myoclonus (N = 1). The symptoms were observed during both the neurologic and the psychiatric assessments. During the psychiatric assessment, all patients had an examination focusing on their physical symptoms to confirm both diagnostic features and severity of symptoms.

There were no statistically significant differences in age, duration of illness, or depression rating scale scores between the primary PMD and the PMD plus other somatoform diagnoses groups or the treated and the untreated groups.

Table 2 provides details on psychiatric diagnoses for the compared groups. The MADRS and CGI-C for motor and global outcomes were repeated at a mean of 3.1 months (SD = 1.2) following treatment. The MADRS score was improved from 10.6 (SD = 6.9) to 5.2 (SD = 3.0) following antidepressant treatment ($p < .01$). The patients' illnesses ranged from mild to moderate severity; the mean CGI-S score was 3.1 (SD = 1.5). Table 3 provides details on the types of psychiatric diagnoses documented for all of the patients in the study.

Table 3. Current Psychiatric Diagnoses in 23 Psychogenic Movement Disorder Patients

Diagnosis[a]	N (%)
Major depressive disorder[b]	8 (34.8)
Psychotic disorder	0 (0)
Bipolar disorder	0 (0)
Any anxiety disorder	12 (52.2)
Obsessive-compulsive disorder	1 (4.3)
Generalized anxiety disorder	11 (47.8)
Panic disorder	2 (8.7)
Posttraumatic stress disorder	1 (4.3)
Social phobia	1 (4.3)
Depression and anxiety	7 (30.4)
Substance disorder	0 (0)

[a]Mini-International Neuropsychiatric Inventory (does not include somatoform disorders, factitious disorder, or malingering).
[b]Fourteen patients (60.9%) had a lifetime diagnosis of major depressive disorder.

Eighteen patients (78%) had at least 1 previous or current Axis I diagnosis in addition to the somatoform diagnosis; 9 patients (39%) had 2 or more previous or current Axis I diagnoses in addition to the somatoform diagnosis. Somatization disorder was identified in 3 patients (13%); stressors at onset, in 12 patients (52%); onset after a motor vehicle accident, in 2 patients (9%); and an abuse history, in 2 patients (9%).

Five patients (22%) had previous psychiatric contact. At baseline, 9 patients (39%) were either previously or currently on treatment with antidepressants. The indication for previous antidepressant use was commonly reported to be depression, but not all patients were questioned regarding antidepressant indication. One patient currently on treatment with antidepressants was not included in the trial given the patient's current contact with a psychiatrist. The psychiatrist was contacted to suggest further optimization of the patient's antidepressant regimen targeting ongoing depressive symptoms. On review of the antidepressant history, we found that only 4 patients (17%) had an adequate trial (an adequate dose for at least 6 weeks' duration) of antidepressants.

Two subgroups of treated patients were identified: 10 patients (67%) had primary conversion symptoms ("primary PMD"), and 5 patients (33%) were diagnosed with primary hypochondriasis (N = 1), somatization disorder (N = 2), or possible factitious disorder/malingering (N = 2) ("PMD plus other somatoform diagnosis"). The second group of patients was suspected of having these diagnoses at baseline assessment. Eight (80%) of the primary PMD patients had "marked" improvements on both motor and global outcomes; 7 of these patients attained complete remission. In contrast, none of the patients with PMD plus other somatoform diagnoses had improvements.

Concurrently, 3 patients had supportive psychotherapy, and 1 patient had family intervention. All primary PMD patients had current or previous depressive or anxiety disorders, whereas only 2 (40%) of the 5 patients with PMD plus other somatoform diagnoses had either a current or previous depressive or anxiety disorder. Three of the improved primary PMD patients did not have a current depression or anxiety diagnosis and had low baseline depression (MADRS score < 7) and anxiety (BAI score < 10) scores. However, these 3 patients also had each suffered recent depressive episodes.

Four primary PMD patients (40%) experienced treatment failure with their first trial of an antidepressant and were switched to a second antidepressant. The duration of this first trial in these 4 patients was a mean of 1.6 months (SD = 0.6). The mean final daily dose of citalopram or paroxetine was 32.2 mg (SD = 6.3), and the mean final daily dose of venlafaxine was 182.1 mg (SD = 97.0). The duration of the trial for the patients who did not improve on antidepressant treatment was 2.1 months (SD = 0.5).

Following the antidepressant trial, 7 of the 15 treated patients (4 with primary PMD and 3 with PMD plus other somatoform diagnoses) were subsequently referred for either group or individual cognitive-behavioral therapy or insight-oriented psychotherapy to manage other psychological issues or residual symptoms.

The 10 patients with primary PMD were reassessed at a mean of 16.2 months (SD = 6.3) after the start of the initial antidepressant trial. Eight patients continued on treatment with antidepressants. The patient with no improvement had been referred for further psychotherapy and rehabilitation; the symptoms were unchanged. The patient with mild improvement had been referred for further pain management with marked improvement of her symptoms; antidepressants were discontinued after 6 months with ongoing remission. Of the other 8 patients, 4 attempted to decrease or discontinue the antidepressants on their own with a subsequent relapse of their movement symptoms and improvements of their movement symptoms on returning to the higher dose of antidepressant. The 4 patients had associated either depressive or anxiety symptoms with the relapse; these symptoms were not quantified. A fifth patient decreased the antidepressant dose, with a relapse of depressive symptoms without the movement symptoms. Following completion of the trial, 2 patients who had minor relapses in the context of stressors were referred for group cognitive-behavioral therapy. One patient had insight-oriented psychotherapy for long-term issues. Functional outcomes of these 10 patients from the time of treatment to reassessment at 16.2 months ranged from continuing with full-time work (N =1), changing from part-time to full-time work (N = 2), changing from short-term disability to full-time work (N = 1), changing from leave of absence from school to return to college (N = 1), continuing as a homemaker or retired (N = 4), and continuing on disability for alternate reasons (N = 1).

DISCUSSION

There are several weaknesses inherent in this study, including its small sample size, the lack of a control group, the use of a nonblinded assessment, the occurrence of possible placebo effects, and the potentially confounding effect of supportive psychotherapy.

That this study represents patients who are agreeable to psychiatric treatment may also represent a selection bias. In this regard, the willingness to accept a psychological explanation for the conversion symptoms has been associated with a better prognosis.[4] The subspecialized nature of the clinic, the confirmation of the diagnosis by a secondary or tertiary opinion, and the immediate referral to a psychiatrist attached to the movement disorders clinic with assessment within a month (thus reinforcing the psychological nature of the diagnosis) may have contributed as nonspecific confounders to the outcomes. However, the patients studied had long, unremitting courses of illness with multiple previous contacts with health care providers. Many of these patients had previously been told of the functional nature of their diagnoses. Chronic conversion disorder is furthermore also commonly associated with a poor prognosis.[5,6]

The lack of a consistent weekly psychotherapeutic intervention at the time of the antidepressant trial argues against a significant contribution from psychotherapy. Despite the potential confounds of the study, these results are in keeping with the observation of antidepressant efficacy in randomized controlled trials of functional somatic syndromes and somatization disorder.[14,15] Antidepressants are also commonly utilized in this patient population.[1,16,17] However, given the nature of this study, these findings should be considered preliminary. Further well-designed studies, particularly to control for a placebo effect, are necessary to confirm these findings.

The results of the present study suggest that treatment with antidepressants can have marked positive effects on motor, psychiatric, and global outcomes in PMD outpatients with primary conversion symptoms and comorbid current or recent depression or anxiety disorders. Duration of symptoms was not associated with treatment outcomes. The positive outcomes reported in this study (especially as compared with less positive outcomes reported in the literature) may be partly attributable to a cohort effect. The patients in this study include only outpatients with primarily mild-to-moderate (mean CGI-S score of 3.6) homogeneous symptoms and limited previous contact with psychiatry. In contrast, studies in the literature are typically based in centers subspecializing in conversion disorder or somatoform disorders and often include populations with mixed symptoms, inpatients, and most likely a more treatment-refractory population, with limited generalizability of the results. The population in this present study is representative of a tertiary movement disorders clinic.

Two subgroups of treated patients were identified, 1 with primary PMD (or conversion symptoms) and 1 with PMD plus other somatoform diagnoses. The study does not address whether the latter subgroup may have had conversion symptoms as the primary diagnosis at the onset of illness. Notably, patients with somatization disorder, factitious disorder, or malingering would not have been expected to improve with such an intervention. The patient with hypochondriasis was subsequently referred for cognitive-behavioral therapy.

Within the treated primary PMD group, although 3 patients did not have current psychiatric diagnoses and had low depression and anxiety scale scores, it is notable that these 3 patients had experienced recent episodes of depression or anxiety. As such, the conversion motor symptoms in these 3 patients may still be associated with residual or subsyndromal depressive symptoms.[22] All of the treated primary PMD group had either a current or previous depression or anxiety disorder, in comparison to 40% of the treated group with PMD and other somatoform diagnoses. This finding is in keeping with the prognostic factors associated with conversion disorder. The presence of a comorbid depression or anxiety disorder has been found to be a positive prognostic factor for the outcome of conversion symptoms.[23] However, the exact relationship between depression and somatoform disorders is not clear, as the response of the somatic symptoms to antidepressants has also been observed in patients with functional somatic syndromes without comorbid depression.[15]

Twenty-two percent of the patients had previous psychiatric contact, with 39% having had previous antidepressant trials. However, in less than half of these patients were these antidepressant trials of adequate duration or dose. During the study itself, 40% of the treated conversion disorder patients experienced treatment failure with the initial SSRI antidepressant but responded to a second antidepressant with both serotonergic and noradrenergic effects. Notably, venlafaxine, a dual reuptake inhibitor, has been suggested to be more efficacious in treating patients with more severe depression[24] (for review, see Kienke and Rosenbaum[24]) and in maintaining remission[25]; however, a placebo response to a second medication also cannot be ruled out. Although many of these patients may have had previous exposure to antidepressants, these findings suggest that suboptimal trials are not uncommon and optimization of treatment will improve outcomes. The limited contact of these patients with psychiatrists is remarkable in the context of their lengthy illness and emphasizes the necessity of appropriate diagnosis and referrals for treatment. The depression rating scale scores of the untreated group were not statistically different from those of the treated group, suggesting that the untreated patients would most likely also benefit from antidepressant treatment.

The study suggests that antidepressant treatment trials should be considered in the treatment armamentarium for

PMD patients with conversion symptoms and comorbid current or recent depression or anxiety. Further psychotherapy including cognitive-behavioral and insight-oriented psychotherapy was provided to a proportion of the patients after the trial concluded. Given the nature of this disorder and the likely pathophysiologic heterogeneity, psychotropic, psychotherapeutic, and social interventions will most likely be necessary.[17] Such management is not dissimilar to the management of other chronic psychiatric illnesses including chronic depression. Determining predictive factors of response to differing interventions will be necessary. More detailed discussions of the potential mechanism of action of antidepressants on PMD symptoms and the potential relationship between depression and PMD can be found in references 26 and 27. Further larger, well-designed controlled studies with long-term outcomes, which are now under way in nonepileptic seizures, are needed to confirm these findings.

Drug names: citalopram (Celexa and others), paroxetine (Paxil, Pexeva, and others), venlafaxine (Effexor).

REFERENCES

1. Williams DT, Ford B, Fahn S. Phenomenology and psychopathology related to psychogenic movement disorders. Adv Neurol 1995;65: 231–257
2. Stone J, Carson A, Sharpe M. Functional symptoms and signs in neurology: assessment and diagnosis. J Neurol Neurosurg Psychiatry 2005;76 (suppl 1):2–12
3. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition. Washington, DC: American Psychiatric Association; 1994
4. 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
5. Stone J, Sharpe M, Rothwell PM, et al. The 12 year prognosis of unilateral functional weakness and sensory disturbance. J Neurol Neurosurg Psychiatry 2003;74:591–596
6. Ron MA. The prognosis of hysteria/somatization disorder. In: Halligan PW, Bass C, Marshall JC, eds. Contemporary Approaches to the Study of Hysteria. New York, NY: Oxford University Press; 2001
7. Krem MM. Motor conversion disorders reviewed from a neuropsychiatric perspective. J Clin Psychiatry 2004;65:783–790
8. Ron M. Explaining the unexplained: understanding hysteria. Brain 2001; 124:1065–1066
9. Cloninger C. The origins of DSM and ICD criteria for conversion and somatization disorders. In: Halligan PW, Bass C, Marshall JC, eds. Contemporary Approaches to the Study of Hysteria. New York, NY: Oxford University Press; 2001
10. Rowan AJ. Diagnosis of non-epileptic seizures. In: Gates JR, Rowan AJ, eds. Non-Epileptic Seizures. 2nd ed. Boston, Mass: Butterworth-Heinemann; 2000:15–30
11. 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
12. Moene FC, Spinhoven P, Hoogduin KA, et al. A randomised controlled clinical trial on the additional effect of hypnosis in a comprehensive treatment programme for in-patients with conversion disorder of the motor type. Psychother Psychosom 2002;71:66–76
13. Rampello L, Raffaele R, Nicoletti G, et al. Hysterical neurosis of the conversion type: therapeutic activity of neuroleptics with different hyperprolactinemic potency. Neuropsychobiology 1996;33:186–188
14. Menza M, Lauritano M, Allen L, et al. Treatment of somatization disorder with nefazodone: a prospective, open-label study. Ann Clin Psychiatry 2001;13:153–158
15. O'Malley PG, Jackson JL, Santoro J, et al. Antidepressant therapy for unexplained symptoms and symptom syndromes. J Fam Pract 1999; 48:980–990
16. Hurwitz TA. Somatization and conversion disorder. Can J Psychiatry 2004;49:172–178
17. Stone J, Carson A, Sharpe M. Functional symptoms in neurology: management. J Neurol Neurosurg Psychiatry 2005;76(suppl 1):13–21
18. Sheehan DV, Lecrubier Y, Sheehan KH, et al. The Mini-International Neuropsychiatric Interview (M.I.N.I.): the development and validation of a structured diagnostic psychiatric interview for DSM-IV and ICD-10. J Clin Psychiatry 1998;59(suppl 20):22–33
19. Davidson J, Turnbull CD, Strickland R, et al. The Montgomery-Asberg Depression Scale: reliability and validity. Acta Psychiatr Scand 1986;73:544–548
20. Beck AT, Epstein N, Brown G, et al. An inventory for measuring clinical anxiety: psychometric properties. J Consult Clin Psychol 1988;56:893–897
21. Guy W. ECDEU Assessment Manual for Psychopharmacology-Revised. US Dept Health, Education, and Welfare publication (ADM) 76-338. Rockville, Md: National Institute of Mental Health; 1976
22. Judd LL, Akiskal HS, Maser JD, et al. A prospective 12-year study of subsyndromal and syndromal depressive symptoms in unipolar major depressive disorders. Arch Gen Psychiatry 1998;55:694–700
23. Crimlisk HL, Bhatia K, Cope H, et al. Slater revisited: 6 year follow up study of patients with medically unexplained motor symptoms. BMJ 1998;316:582–586
24. Kienke AS, Rosenbaum JF. Efficacy of venlafaxine in the treatment of severe depression. Depress Anxiety 2000;12(suppl 1):50–54
25. Mallick R, Chen J, Entsuah AR, et al. Depression-free days as a summary measure of the temporal pattern of response and remission in the treatment of major depression: a comparison of venlafaxine, selective serotonin reuptake inhibitors, and placebo. J Clin Psychiatry 2003;64:321–330
26. Voon V. Treatment of psychogenic movement disorder: psychotropic medications. In: Hallett M, Fahn S, Jankovic J, et al, eds. Psychogenic Movement Disorders, Neurology and Neuropsychiatry. Neurology Reference Series. Philadelphia, Pa: Lippincott Williams and Wilkins; 2005:302–310
27. Voon V, Hallett M. Depression. In: Hallett M, Fahn S, Jankovic J, et al, eds. Psychogenic Movement Disorders, Neurology and Neuropsychiatry. Neurology Reference Series. Philadelphia, Pa: Lippincott Williams and Wilkins; 2005:148–153

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