



# Diagnosis and treatment of psychogenic parkinsonism

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Received 1 July 2011  
Revised 9 July 2011  
Accepted 20 July 2011  
Published Online First  
20 September 2011

## ABSTRACT

Parkinson's disease (PD) is the most common movement disorder encountered in movement disorder clinics but psychogenic parkinsonism (PP) is relatively rare. Based on a study of 32 patients, coupled with a comprehensive review of the literature, the diagnosis, clinical phenomenology, epidemiology, natural history, imaging and treatment, this article provides a critical review of PP. In addition to other PD-like symptoms, patients with PP often exhibit abrupt onset, typically in response to a stressful event, followed by a fluctuating course, early disability, bilateral shaking and slowness, non-decremental slowness when performing repetitive movements, voluntary resistance against passive movement without cogwheel rigidity, distractibility, generalised and 'give-way' weakness, stuttering speech, bizarre gait and a variety of behavioural, sexual and other motor and non-motor symptoms. Levodopa related motor complications do not occur even in the setting of chronic, high dose therapy. In some cases, differentiation between PD and PP is very difficult, and in those cases the response to placebo may be helpful. A comprehensive, multidisciplinary assessment of patients with PP, when combined with insight oriented psychotherapy, physiotherapy and treatment of comorbid depression, is often helpful, although the prognosis for this group of disorders remains relatively poor.

## INTRODUCTION AND BACKGROUND

Psychogenic movement disorders (PMDs) are a group of heterogeneous disturbances manifested by deliberate slowness or a variety of other motor abnormalities, such as shaking, jerking, spasmodic and other, often bizarre, movements, postures or gaits that cannot be explained by organic conditions, frequently occurring in association with underlying psychological or psychiatric disturbances.<sup>1–5</sup> Although different psychiatric diagnoses have been proposed, most patients with PMDs have somatoform or conversion disorders, presumably without conscious awareness of the underlying mechanism, but in some cases the motor abnormalities represent factitious disorders or malingering, in which the abnormal movements are purposefully feigned. Psychological or physical stress often plays a role in precipitating and maintaining the movement disorder, even though specific acute or chronic stressors are not always initially identifiable by the patient, partly because of lack of insight or denial. While the term 'functional' is occasionally used to describe this group of disorders, the term implies normal function rather than dysfunction and, therefore, the term 'psychogenic' seems more appropriate. This term is increasingly being adopted

by other disciplines, including psychiatry. As an example, the term 'psychogenic seizure' has now largely replaced the traditional term 'pseudoseizure'. This review focuses on psychogenic parkinsonism (PP), manifested chiefly by slowness of movement and other features commonly associated with Parkinson's disease (PD).<sup>4</sup>

## EPIDEMIOLOGY

The epidemiology of PMDs has not been well studied, largely because of lack of consensus on diagnostic criteria, the use of different methodologies to ascertain cases and the frequent coexistence of organic movement disorder. The reported estimates of prevalence of PMDs have ranged between 1% and 9% of the general population, about 10% (range 1.7–25%) of which have been categorised as PP.<sup>5–9</sup> PMDs have been estimated to account for 3.6% of all patients seen in a movement disorders clinic over a 71 month period.<sup>10</sup> Out of 530 patients diagnoses with PMD, 17 (3.2%) had predominantly parkinsonism, 211 (39.8%) tremor, 215 (40.6%) dystonia, 91 (17.0%) myoclonus, 23 (4.3%) tics, eight (1.5%) dyskinesia and three (0.6%) had psychogenic chorea,<sup>11</sup> but many patients exhibited a combination of different PMDs.<sup>10</sup> The observed exponential increase in the incidence of PMDs in specialty clinics<sup>7</sup> has been partly attributed to improved diagnostic skills by neurologists and other physicians in recognising typical movement disorders, thus more and more patients referred to movement disorders clinics have atypical disorders, many of which are psychogenic.

## DIAGNOSIS AND CLINICAL FEATURES

The diagnosis of PP is not always easy, but a thorough clinical history and a detailed physical examination by an experienced clinician, focusing on not just excluding organic parkinsonism but also to elicit features supporting psychogenic aetiology, are critical. In the absence of a reliable diagnostic marker, differentiation between PD and PP depends on recognition of the characteristic phenomenology and an interpretation of historical and clinical features by a clinician experienced in movement disorders. The diagnosis of PMDs should not be regarded as a diagnosis of exclusion but should be based on positive clinical criteria, partly supported by neuropsychological, neurophysiological and imaging studies. At the Baylor College of Medicine Parkinson's Disease and Movement Disorders Clinic, we use the following criteria for the diagnosis of documented or clinically established PP.<sup>12 13</sup> The presence of at least five of the following characteristic features, one of

which must be (1) or (2), was used in arriving at the diagnosis of PP: (1) slow, deliberate, effortful (associated with grimacing or sighing) rapid succession movements without decrementing amplitude, but normal speed of movement when distracted; (2) abnormal response to postural stability testing; patients may have exaggerated, bizarre responses to minimal perturbations on testing of postural instability, which may include the flailing of arms and reeling backwards without falling; (3) tremor of an abrupt onset, typically involving the dominant hand; (4) tremor with a variable frequency, direction and anatomical distribution and reduced amplitude or speed with distraction; (5) tremor that spreads to other body parts when the affected limb is immobilised; (6) active resistance against passive movement; true 'cogwheel' rigidity may not be present and if present may decrease with distraction; (7) stuttering, whispering, gibberish speech; and (8) other psychogenic features, such as 'give-way' weakness, non-anatomical sensory loss and other PMDs.

Using the above criteria, we enrolled 32 patients with PP, 17 (53%) women, with a mean age at diagnosis of  $48 \pm 8.6$  years and mean duration of symptoms of  $5.24 \pm 1.2$  years<sup>13</sup> (table 1). A precipitating event was identified in 56% of our patients and included job related stress in 11 (34%), personal life stress in four (13%) and physical trauma in four (13%); 13 (41%) had a combination of multiple stressors. The majority of our patients (56%) had a history of comorbid psychiatric disorder, with depression being the most common. Mean educational level was  $14 \pm 2.5$  years; 13% were employed in the healthcare field. Twenty-one (66%) of the patients were married, 20 (63%) were on disability and seven (22%) had pending litigation. A family history of tremor or parkinsonism was present in nine (28%) patients. Half of our PP patients underwent a placebo challenge with carbidopa (n=12), tuning fork (n=2) or saline injections (n=2). The improvement after placebo, based on a 'blinded' review of videos, was rated as 1 or 2 (marked or moderate improvement) in four (40%) patients and 3 (mild improvement) in six (60%) patients. At a follow-up evaluation, 3–6 months after the initial pre- and post-placebo video session, 60% of the patients had had complete resolution of their symptoms.

This largest reported series of PP highlights the most characteristic features of this psychogenic disorder, including lack of male preponderance (in contrast with typical male preponderance in organic PD), frequent precipitating events and psychiatric comorbidities, early disability and atypical features on examination which were incongruous with organic PD (table 1). For example, in contrast with tremor in patients with organic PD, the tremor in PP often starts not in the hand, as is typical of PD, but may present as a unilateral leg tremor.<sup>18</sup> Furthermore, tremor associated with PP usually does not disappear with movement of the limb. Similar to psychogenic tremor without parkinsonism,<sup>19</sup> in addition to marked distractibility, the

frequency of tremor in PP varies in rhythmicity and direction of oscillation. Rigidity, if present, is often associated with active resistance against passive movement and there is usually no cogwheeling. While slowness of movement (bradykinesia) is present in almost all patients with PP, there is usually no decrementing amplitude on rapid succession movements, typically seen in PD. In one study involving 195 PMD patients, the finger tapping task scores were significantly lower in the PMD group compared with the other diagnostic groups (PD, dystonia, essential tremor and 130 normal adults).<sup>20</sup> Patients with PP often demonstrate slow and deliberate movement when asked to perform a particular task but are able to function normally when distracted or when they do not think they are being observed. For example, they can dress and perform other activities of daily living without any perceptible slowness. The handwriting is often laboured and irregular but without the typical micrographia. On a pull test, the patient's response is often inconsistent, manifested either by minimal displacement or by an extreme response associated with flinging of the arms and reeling backward, but never falling. When asked to walk fast or to run, the gait often becomes stiffer and the short stride is maintained, but there is no freezing. Some patients with PP also manifest features of psychogenic gait, including bouncing, buckling of the knees and atasia-abasia.<sup>21</sup> Speech in a patient with PP often becomes stuttering, 'baby-like' or demonstrating a foreign accent. If the patient has 'levodopa related dyskinesia' the hyperkinetic movement is often bizarre and incongruous with typical levodopa induced stereotypy, chorea or dystonia.

Although the diagnosis of PP can be usually established on the basis of atypical history and phenomenology, some doubts and uncertainties may remain. In such cases the judicious use of placebo, neurophysiological studies or functional imaging may be used to enhance or supplement the diagnostic acumen.<sup>13 19 22–25</sup> Many patients with PP have been previously diagnosed with PD and have used dopaminergic drugs in the treatment of PD or are already taking levodopa/carbidopa or dopamine agonists. In patients already treated with other dopaminergics, we often discuss with them that in order to have a better understanding of their parkinsonism we need to examine them at least 24 h off levodopa/carbidopa. In patients never previously treated with dopaminergic drugs or in those who have discontinued their treatment, we sometimes administer a 25 mg tablet of carbidopa and observe their response. If the administration of carbidopa, an inert placebo as it does not cross the blood–brain barrier, results in motor improvement, it may be used as supportive evidence for the diagnosis of PP. Indeed, in many of our cases, carbidopa was associated with robust improvement in parkinsonian and other symptoms.<sup>13</sup> Disclosure to these patients that the carbidopa acted as an inert placebo often frames the discussion of the diagnosis of PP and of the need to avoid potentially harmful pharmacotherapy.

**Table 1** Review of the literature on psychogenic parkinsonism

Author	Year	No of subjects	Mean age (years)	Movement duration (years)	Abrupt onset (%)	Sex (F:M)
Lang <i>et al</i> <sup>5</sup>	1995	14	48	5.3	79	7:7
Factor <i>et al</i> <sup>6</sup>	1998	2	53.5	6.0	100	1:1
Booij <i>et al</i> <sup>14</sup>	2001	4	51	Unknown	NA	NA
Gaig <i>et al</i> <sup>15</sup>	2006	8	53.7	6.1	55	5:4
Benaderette <i>et al</i> <sup>16</sup>	2006	9	49.9	4.7	44	4:5
Felicio <i>et al</i> <sup>17</sup>	2010	15	37.4	3.9	NA	9:6
Jankovic and Hunter <sup>13</sup>	2012	32	48	5.2	84	17:15

The documented response to placebo may be helpful not only in differentiating between organic parkinsonism and PP, but also in improving patients' insights into the nature of their disorder and in planning treatment strategy. Although we recognise the controversial nature of placebos in the diagnosis of PMDs, when fully transparent, the approach is often beneficial in the management of patients with PMDs, including PP. A recent review on the role of placebos or suggestion in PMD concluded that suggestion is "ethically justifiable" for patients with PMD and that "When issues of choice, consent, deceit, disclosure and decision making are analysed from the perspective of an ethics of care, we see that suggestion may enhance patient autonomy and does not violate the trust between doctors and their patients".<sup>25</sup> Furthermore, placebos have been used in clinical practice and in research not only to help establish the diagnosis but also to determine the magnitude of a therapeutic effect in double blind, placebo controlled trials.<sup>22 26 27</sup>

There is growing evidence that placebos may not necessarily be completely inert but that they induce changes in brain function that are distinct from those associated with antidepressant medication.<sup>7 28</sup> Using quantitative electroencephalographic techniques, prefrontal neurophysiological changes have been found in patients administered placebo, and these changes may be predictive of subsequent response to antidepressant medications.<sup>28</sup> Positron emission tomography studies have shown that dopamine is released in response to a placebo and that the placebo effect may be related to dopamine mediated reward mechanism.<sup>29–33</sup> The placebo induced changes in striatal raclopride binding in patients with idiopathic PD were similar to those obtained after therapeutic doses of levodopa or apomorphine and the estimated amount of dopamine release was greater in those who perceived the placebo effect than those who did not.<sup>34</sup> Thus the dopamine released within the ventral striatum is more related to the expectation of a reward rather than to the reward itself.

## IMAGING

In some cases, the use of imaging techniques, such as demonstration of reduced [18F]-fluorodopa (FDOPA) uptake on positron emission tomography or reduced dopamine transporter density (DAT) by single photon emission CT (SPECT) may be helpful in differentiating PD from PP.<sup>14–17 35 36</sup> Several studies have found that 10–15% of patients with clinically diagnosed PD have normal FDOPA or  $\beta$ -CIT SPECT scans without evidence of dopaminergic deficit (SWEDD),<sup>17 37–39</sup> some of whom have been later found to have PP. One of our patients with clinically diagnosed PD was disqualified from the Parkinson's Progression Markers Initiative because of SWEDD and another patient initially enrolled in The Parkinson's Research Examination of CEP-1347 Trial (PRECEPT) was later found to have PP and SWEDD, as indicated by normal DAT density on  $\beta$ -CIT SPECT.<sup>39</sup> Both patients initially had the classic features of PD, but later developed irregular, intermittent, distractible tremor and other features that were incongruent with organic parkinsonism and indicative of PP. Thus in cases where PP is suspected, these imaging studies designed to investigate the integrity of the nigrostriatal system, combined with neurophysiological techniques, can be useful in discriminating PP from PD. These studies have been useful also in identifying possible PP patients who are being considered candidates for deep brain stimulation or have had an atypical response to deep brain stimulation.<sup>40</sup>

Functional MRI imaging of PMDs has provided important insights into possible pathophysiological mechanisms of this

group of disorders and may be used as a tool to address the question of whether or not the conversion movements are self-generated.<sup>41</sup> Whether 3.0 T MRI and high resolution diffusion tensor imaging, recently reported to differentiate between early PD and healthy controls,<sup>42</sup> can be used to differentiate between PD and PP remains to be determined.

## NATURAL HISTORY AND PROGNOSIS

The long term prognosis of PMDs, including PP, is usually poor and the adverse impact of these disorders on quality of life is often similar to that of organic, neurodegenerative PD.<sup>43</sup> Patients with PMD often place high demands on available healthcare resources and many undergo unnecessary, expensive diagnostic tests, surgical interventions and other potentially life threatening procedures. As a result, this group of disorders has been referred to as a 'crisis for neurology'.<sup>44</sup>

There is a paucity of data on the long term prognosis of PMDs, especially PP. After a mean duration of follow-up of  $3.4 \pm 2.8$  years, an improvement in symptoms was noted in 56.6% of patients (22.1% were worse and 21.3% remained the same) with PMD initially evaluated at the Baylor College of Medicine Movement Disorders Clinic.<sup>10</sup> Factors associated with good outcome included positive attitude towards treatment prescribed by the physician, shorter duration of illness, stronger physical health and positive social life perceptions. This is similar to a permanent meaningful benefit in 52% of patients in the Columbia series<sup>12</sup> and resolution of symptoms in 60% of patients followed at Baylor<sup>13</sup>, but the reported prognosis is considerably poorer in another study which found that only 35% of PMD patients had resolution of symptoms over a 6 year period.<sup>6</sup>

Prognosis appears to be better in children with PMDs.<sup>45 46</sup> In one study, 50% of children remitted and 40% returned to a normal school life.<sup>46</sup> As with adults, children who remitted were treated early, usually in their first year after symptom onset, whereas those without improvement had been symptomatic for many years. Several studies have shown that children may remain disabled for several years, particularly if the PMD is not addressed early.

## TREATMENT

Treatment of PMD starts with honest and tactful discussion of the diagnosis. A sympathetic, compassionate approach generally leads to a trusting patient–physician relationship. Steps must be taken to avoid confrontational, punitive or an adversarial approach. While the diagnosis should be made by the neurologist, psychiatrist may be helpful in the future management of the patient. Once the diagnosis of PP is established, patients should be warned against further unnecessary investigations, procedures and treatments. It is beyond the scope of this review to discuss in detail therapeutic strategies in the treatment of PP and the reader is referred to other reviews.<sup>47</sup>

A brief presentation of possible psychodynamic factors and an introduction to an insight oriented psychotherapy may be appropriate at the initial encounter but more involved psychiatric treatment or referral for psychotherapy should be delayed until the follow-up visit. The treatment approach must be individualised and tailored to the specific case. Although stress often plays a role in triggering PP, it is important to note that some organic parkinsonian disorders, such as rapid onset dystonia–parkinsonism and dopa responsive dystonia,<sup>48 49</sup> can also be triggered by stress and may be mistaken for a PMD. Stress management, including biofeedback, cognitive behavioural therapy, hypnosis and other techniques designed to reduce

the effects of stress on the body should be discussed with all PP patients. Physical therapy designed to relax the agonist muscles and to facilitate a full range of motion may help prevent contractures. Overall, a multidisciplinary approach, involving a neurologist, psychiatrist or behavioural psychologist and physical therapist most likely yields the best therapeutic results. Further prospective studies are needed to evaluate the therapeutic options in these patients. As depression and anxiety, whether acknowledged by the patient or not, often accompany PP, antidepressants may be prescribed. In addition to their beneficial effects on mood, antidepressants may also improve associated anxiety and insomnia, although their added role as placebo therapy cannot be excluded. Finally, treatment of factitious disorders, including Munchausen's syndrome, as well as any legal issues also need to be addressed.

**Competing interests** None.

**Provenance and peer review** Commissioned; not externally peer reviewed.

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