Psychogenic movement disorders: frequency, clinical profile, and characteristics

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Abstract
Of 842 consecutive patients with movement disorders seen over a 71 month period, 28 (3.3%) were diagnosed as having a documented or clinically established psychogenic movement disorder. Tremor was most common (50%) followed by dystonia, myoclonus, and parkinsonism. Clinical descriptions of various types are reviewed. Clinical characteristics common in these patients included distractability (86%), abrupt onset (54%), and selective disabilities (39%). Distractability seems to be most important in tremor and least important in dystonia. Other diagnostic clues included entrapment of tremor to the frequency of repetitive movements of another limb, fatigue of tremor, stimulus sensitivity, and previous history of psychogenic illness. On average, 71% had other psychogenic features. Over 60% had a clear history of a precipitating event and secondary gain and 50% had a psychiatric diagnosis (usually depression). Twenty five per cent of patients presented with combined psychogenic movement disorder and organic movement disorder; 35% resolved and this subgroup had a shorter duration of disease than those who are unresolved. Psychogenic movement disorder represents an uncommon diagnosis among patients with movement disorders. The ability to make a diagnosis rests on the presence of a multitude of clinical clues and therapeutic action should be taken as early as possible.

Keywords: psychogenic movement disorders; tremor; dystonia; myoclonus; parkinsonism

Psychogenic movement disorders represent a substantive diagnostic challenge for clinicians. The term “psychogenic” has been coined to include movement disorders due to somatoform disorder, factitious disorder, and malingering (all of which may be impossible to differentiate) and to distinguish these syndromes from organic diseases. The frequency of psychogenic movement disorders in relation to organic movement disorders is not known but is thought to be low. Unlike epilepsy in which EEG can assist in the diagnosis of pseudoseizures, movement disorders can usually only be approached from a clinical standpoint. There have been only a few reports primarily on psychogenic movement disorder in the medical literature. They have included case reports or series of cases specifically dealing with tremor, dystonia, myoclonus, and parkinsonism. Which of these is most common has been a matter of debate. These reports have provided descriptions of clinical characteristics, both historical and by physical examination, which may lead to an index of suspicion which, in turn, will enhance diagnostic capability. Despite this information, neurologists, including movement disorder specialists, remain tentative about making a diagnosis of psychogenic movement disorder.

The purpose of this study is to provide additional data on psychogenic movement disorders to highlight the important clinical clues which may enhance our ability to make a correct diagnosis. Specifically, we studied frequency, profile of types of psychogenic movement disorders seen, and the frequency of coexistence with organic movement disorders in a movement disorder clinic. We also studied the frequency with which clinical features previously thought common in psychogenic movement disorder occurred, and their possible specificity to different types. Additional clinical characteristics which have not been emphasised in existing publications were also examined. Finally, we attempted to track the outcome of these patients and consider how this information can be used when a neurologist is presented with a patient with a psychogenic movement disorder. This paper is an update of data previously reported in abstract form.

Methods
We performed a retrospective evaluation of psychogenic movement disorders in a movement disorder clinic using a computerised database, record review, and videotape review (when available). Patients with a diagnosis of psychogenic movement disorder (made prospectively by at least one of the authors) were selected from the database generated during the first 71 months of our movement disorder clinic. To that point 842 consecutive patients with movement disorders were seen. The records and videotapes of those patients diagnosed as having psychogenic movement disorder were reviewed in detail and only those patients in whom we confirmed a high level of clinical probability were included.
The movement disorders were categorised in accordance with the organic phenomenon which they most closely resembled (tremor, dystonia, etc). For a diagnosis of psychogenic movement disorder to be made movements were required to be inconsistent over time or incongruent with the classic disease. Clinical features of patients with psychogenic movement disorders described in previous studies were used as guides to the diagnosis and they included abrupt onset, cessation of movement with distraction, multiple somatisations, selective disabilities, lack of response to typical pharmacological treatments, placebo response, spontaneous remissions and occurrence of previous psychogenic illness. The frequency of these features in psychogenic movement disorders was examined. We also sought to define any other features which might represent further clues to the diagnosis of psychogenic movement disorder. Previously reported descriptive characteristics of psychogenic tremor, dystonia, myoclonus, and parkinsonism were used in the same manner. We diagnosed patients as psychogenic parkinsonism instead of psychogenic tremor because they presented with a diagnosis of Parkinson’s disease and were treated with levodopa. All patients were thoroughly investigated and secondary causes were ruled out as far as possible before a diagnosis was accepted.

Once the initial diagnosis of psychogenic movement disorder was confirmed by record and videotape review, the patients were classified according to the Fahn and Williams classification of certainty of diagnosis from its original use in describing patients with psychogenic dystonia. Documented psychogenic movement disorders are described as involuntary movements that are persistently relieved by psychotherapy, suggestion (including physical therapy), placebos, or the patient is witnessed as being free of the involuntary movements when supposedly unobserved. Clinically established psychogenic movement disorders are defined by abnormalities which are inconsistent or incongruent with classic descriptions of recognised movement disorders in association with the presence of other psychogenic signs on neurological examination, multiple somatisations, or obvious psychiatric disturbance. Cases of probable and possible psychogenic movement disorder were excluded from this study.

In addition to assessing frequency of psychogenic movement disorders and other factors described above, we also examined the frequency of other important associated features. These included presence of a precipitating event, secondary gain, history of prior psychiatric disease, other non-organic findings on examination, and coexistence with organic movement disorders. To examine outcome of patients not seen in our clinic in the past year and whose movement disorder was previously unresolved, we attempted to contact the referring physicians by telephone and recorded information or received updated office records.

**Results**

**FREQUENCY**

Twenty eight of 842 consecutive patients with movement disorders (3-3%) were identified as having a documented (nine) or clinically established (19) psychogenic movement disorder (table 1). Seventeen (61%) were women. Mean age was 50 (range 17-83) years. Mean duration of illness at the time of initial evaluation was 33 months (range one week to 12 years). The patients saw one to 15 (average three) physicians for their illness before arriving in our clinic. Twenty one of the 28 patients allowed us to videotape at least one examination.

**PROFILE OF TYPE**

Psychogenic tremor was the most often encountered psychogenic movement disorder with 14 patients (50%) given this diagnosis. Psychogenic dystonia was diagnosed in five (18%), myoclonus in four (14%), and parkinsonism in two (7%). One additional patient had a combination of tremor and dystonia, one had psychogenic tongue movements after accidental exposure to a dopamine antagonist, and one had unclassifiable foot and toe movements. Table 1 lists the anatomical distribution of movements for each patient.

The referral pattern was such that 11 of the 14 patients with psychogenic tremor were referred because of tremor. This represented 11-3% of the tremor referrals to our clinic. The five patients with dystonias represented 2-9% of all patients with dystonia referred. The two parkinsonian patients made up only 0-5% of all patients presenting with parkinsonism and the three patients with myoclonus presenting primarily for their jerky movements made up 30% of myoclonus referrals.

**CLINICAL CHARACTERISTICS**

The patients diagnosed in this study had many of the clinical features shown previously to be suggestive of psychogenic movement disorder. Improvement or disappearance of the movement disorder on distraction was obvious in 22 of 28 (86%) patients. A history of abrupt onset was evident in 15 (54%) patients, selective disabilities in 11 (39%), and multiple somatisations in 10 (36%). There were other, less common, features which we found to strongly support the diagnosis in some patients. Of 11 patients with tremor whose videotapes were available for review, three had their tremor entrained to the frequency of movements in the opposite limb, such as finger tapping. Fatigue occurred in three patients with tremor. In four patients there was evidence (past medical records) of a previous psychogenic neurological event. Two patients had complete control of their tremors with medication not typically used for this purpose; acetaminophen with codeine and methocarbamol (patients 3 and 7). One other unusual feature was stimulus sensitivity of the psychogenic movement disorder. Four patients had an increase or onset of the movement disorder with stimuli; two had escalation of tremors with touch or passive movements,
<table>
<thead>
<tr>
<th>Patient</th>
<th>Ages</th>
<th>Sex</th>
<th>Psychogenic movement</th>
<th>Additional psychogenic features</th>
<th>Genuine movement disorder</th>
<th>Psychiatric diagnosis</th>
<th>Duration of psychogenic movement</th>
<th>Identified potential for patient gain</th>
<th>Precipitating event</th>
<th>Outcome</th>
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<tbody>
<tr>
<td>1</td>
<td>31/F</td>
<td></td>
<td>Tremor and dystonia, four extremities</td>
<td>Give way weakness, gait disorder, non-physiological pattern of sensory loss, slow movement</td>
<td>None</td>
<td>Depression, history of suicide attempts, alcohol abuse</td>
<td>17 months</td>
<td>Workers compensation claim</td>
<td>Fell off ladder at work</td>
<td>Resolved spontaneously</td>
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<tr>
<td>2</td>
<td>17/F</td>
<td></td>
<td>Right leg tremor</td>
<td>Right foot dystonia</td>
<td>None</td>
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<tr>
<td>3</td>
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<td></td>
<td>Tremor, both legs</td>
<td>Total loss of body sensation, give way weakness</td>
<td>None</td>
<td>Depression</td>
<td>12 months</td>
<td>None identified</td>
<td>None identified</td>
<td>Unresolved</td>
</tr>
<tr>
<td>4</td>
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<td>Tremor, both arms</td>
<td>Vibration lost on one side of forehead, slow movement, polyposis, gait disorder</td>
<td>None</td>
<td>Depression</td>
<td>39 months</td>
<td>Workers compensation claim</td>
<td>Exposure to polyvinyl-alcohol and trichloroethylene</td>
<td>Unresolved</td>
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<tr>
<td>5</td>
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<td></td>
<td>Tremor, trunk and arms with standing</td>
<td>Gait ataxia and false reposition</td>
<td>None</td>
<td>Depression</td>
<td>144 months</td>
<td>To gain the attention of family members</td>
<td>Movement disorder began after death of spouse</td>
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<tr>
<td>6</td>
<td>68/M</td>
<td></td>
<td>Myoclonic jerks, both legs</td>
<td>Non-physiological sensory loss</td>
<td>None</td>
<td>Depression</td>
<td>60 months</td>
<td>Personal injury claim</td>
<td>Motor vehicle accident</td>
<td>Resolved after claim settled</td>
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<tr>
<td>7</td>
<td>23/F</td>
<td></td>
<td>Left arm tremor</td>
<td>None</td>
<td>None</td>
<td>Depression</td>
<td>4 months</td>
<td>Personal injury claim</td>
<td>Motor vehicle accident</td>
<td>Unresolved</td>
</tr>
<tr>
<td>8</td>
<td>57/F</td>
<td></td>
<td>Unclassifiable toe movements</td>
<td>Retropulsion, shuffling steps</td>
<td>None</td>
<td>Depression</td>
<td>24 months</td>
<td>Disability claim</td>
<td>Back injury while lifting at work</td>
<td>Unresolved</td>
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<tr>
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<td>Myoclonic jerks, both legs</td>
<td>None</td>
<td>None</td>
<td>Depression</td>
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<td>Unresolved</td>
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<tr>
<td>10</td>
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<td>Tremor, both arms</td>
<td>Left arm tremor</td>
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<td>48 months</td>
<td>None identified</td>
<td>None identified</td>
<td>Unresolved</td>
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<tr>
<td>11</td>
<td>57/M</td>
<td></td>
<td>Essential tremor</td>
<td>None</td>
<td>None</td>
<td>Depression</td>
<td>60 months</td>
<td>Workers compensation claim</td>
<td>Movement disorder claimed to be part of work related carpal tunnel syndrome</td>
<td>Unresolved</td>
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<tr>
<td>12</td>
<td>58/F</td>
<td></td>
<td>Head tremor</td>
<td>Give way weakness, false reposition</td>
<td>None</td>
<td>Depression</td>
<td>72 months</td>
<td>Unemployment benefits</td>
<td>None identified</td>
<td>Unresolved</td>
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<tr>
<td>13</td>
<td>52/M</td>
<td></td>
<td>Parkinsonism</td>
<td>Give way weakness</td>
<td>None</td>
<td>Post-traumatic stress disorder, past suicide attempts, depression</td>
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<td>None identified</td>
<td>None identified</td>
<td>Unresolved</td>
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<tr>
<td>14</td>
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<td>Give way weakness, slow movement, non-physiological sensory loss</td>
<td>None</td>
<td>Depression</td>
<td>3 months</td>
<td>Personal injury claim</td>
<td>None identified</td>
<td>Unresolved</td>
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<td>15</td>
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<td>Generalised myoclonic jerks, legs, arm</td>
<td>Gait ataxia, eyelid fluttering</td>
<td>None</td>
<td>Depression</td>
<td>10 months</td>
<td>Family attention claim</td>
<td>None identified</td>
<td>Resolved</td>
</tr>
<tr>
<td>16</td>
<td>59/M</td>
<td></td>
<td>Tremor, legs, head</td>
<td>None</td>
<td>None</td>
<td>Depression</td>
<td>60 months</td>
<td>None identified</td>
<td>None identified</td>
<td>Resolved with P/T and psychological counselling</td>
</tr>
<tr>
<td>17</td>
<td>48/M</td>
<td></td>
<td>Jerking head movements</td>
<td>None</td>
<td>None</td>
<td>Depression</td>
<td>3 months</td>
<td>Paranoid schizophrenia</td>
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<td>Give way weakness, slow movement</td>
<td>None</td>
<td>Depression</td>
<td>2 weeks</td>
<td>None identified</td>
<td>None identified</td>
<td>Resolved with suggestion</td>
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<tr>
<td>19</td>
<td>45/M</td>
<td></td>
<td>Left foot dystonia</td>
<td>None</td>
<td>None</td>
<td>Depression</td>
<td>12 months</td>
<td>None identified</td>
<td>None identified</td>
<td>Worsening medical condition</td>
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<tr>
<td>20</td>
<td>55/F</td>
<td></td>
<td>Myoclonic jerks of legs</td>
<td>None</td>
<td>None</td>
<td>Depression</td>
<td>7 months</td>
<td>None identified</td>
<td>None identified</td>
<td>Unresolved</td>
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<tr>
<td>21</td>
<td>49/M</td>
<td></td>
<td>Tremor, both hands</td>
<td>None</td>
<td>None</td>
<td>Depression</td>
<td>1 week</td>
<td>Parkinson's disease</td>
<td>None identified</td>
<td>Resolved with suggestion</td>
</tr>
<tr>
<td>22</td>
<td>22/F</td>
<td></td>
<td>Paroxysmal generalised dystonia</td>
<td>Positive Hoover's sign, None</td>
<td>Essential tremor</td>
<td>Depression</td>
<td>24 months</td>
<td>To gain the attention of spouse</td>
<td>None identified</td>
<td>Unresolved</td>
</tr>
<tr>
<td>23</td>
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<td></td>
<td>Tremor, index finger left hand</td>
<td>None</td>
<td>None</td>
<td>Depression</td>
<td>9 months</td>
<td>Anxiety/panic disorder</td>
<td>None identified</td>
<td>Unresolved</td>
</tr>
<tr>
<td>24</td>
<td>34/F</td>
<td></td>
<td>Left foot dystonia</td>
<td>None</td>
<td>None</td>
<td>Depression</td>
<td>20 months</td>
<td>Workers compensation claim</td>
<td>Hand caught in a bus door while lifting at work, injured back</td>
<td>Unresolved</td>
</tr>
<tr>
<td>25</td>
<td>42/F</td>
<td></td>
<td>Blepharospasm, dysphonia</td>
<td>None</td>
<td>None</td>
<td>Depression</td>
<td>48 months</td>
<td>Disability</td>
<td>None</td>
<td>Unresolved</td>
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<tr>
<td>26</td>
<td>55/F</td>
<td></td>
<td>Parkinsonism</td>
<td>None</td>
<td>None</td>
<td>Depression</td>
<td>12 months</td>
<td>None identified</td>
<td>None identified</td>
<td>Unresolved</td>
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<tr>
<td>27</td>
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<td></td>
<td>Head tremor</td>
<td>None</td>
<td>None</td>
<td>Depression</td>
<td>12 months</td>
<td>Psychogenic</td>
<td>None identified</td>
<td>Unresolved</td>
</tr>
<tr>
<td>28</td>
<td>38/F</td>
<td></td>
<td>Tongue movements</td>
<td>None</td>
<td>None</td>
<td>Depression</td>
<td>55 months</td>
<td>Litigation</td>
<td>None identified</td>
<td>Unresolved</td>
</tr>
</tbody>
</table>

Table 1: Clinical summary
one patient with paroxysmal dystonia had onset with a loud noise, and one patient with tremor had enhancement with both types of stimuli.

OTHER ASSOCIATED FEATURES
A precipitating event was described in 17 patients (61%). Work related injuries were most prevalent (seven patients) and they included back injury with lifting, a short fall from a ladder, and chemical exposure. Table 1 lists other precipitants. A definite secondary gain was evident in 18 (64%) patients (table 1). Of these, seven patients submitted workers’ compensation claims, five submitted personal injury claims, one was pursuing litigation against a pharmacy which erroneously dispensed a wrong medication for nausea, and one patient had onset of his foot dystonia in court to avoid prosecution. Fourteen (50%) patients had been previously diagnosed with psychiatric disorders (table 1); nine with depression (three with a history of suicide attempts), five with alcohol misuse, three with anxiety disorders, two with post-traumatic stress disorder, and two with paranoid schizophrenia.

Other psychogenic features were found on neurological examination in 20 (71%) patients (table 1). False weakness including give way and a positive Hoover’s sign was most common (12 patients). Non-physiological sensory loss was seen in 11 patients and included stripes of sensory loss and unilateral loss of vibratory sense on the forehead. Gait disorders with false retropulsion were present in 10 and slow movements (not true bradykinesia) in six. This was associated with extreme effort and exhaustion on a background of normal speed of movement with spontaneously performed tasks. Three others had polyopia (seeing three or more images), false arm drifts were seen in two, and a slow halting voice in one. Of the 20 patients with these findings, seven had one psychogenic feature, seven others had two, one patient had three, and five patients had four or more.

Seven patients with psychogenic movement disorders (25%) had coexisting organic movement disorders (patients 2, 5, 12, 17, 21, 23, and 28; table 1). In these patients the psychogenic movement disorder was of a distinct character, time of onset, and level of disability separate from their organic disease. If the psychogenic movement disorder occurred in the same location as the organic movement disorder, it was of a different character; however, similar types could occur in a different body part.

OUTCOME
The outcome was deciphered through our continued care of the patients or from assistance from referring physician. In eight patients the outcome was unknown because they had been lost to follow up. Ten patients resolved; five spontaneously (three with suggestion), two after psychological treatment and physical treatment, one after placebo (only one of five patients receiving placebo in the office or hospital responded), one after the psychosis improved, and one after settlement of the personal injury claim. Ten patients remained unresolved. Mean duration of illness at the time of initial visit was 22 months for those who had resolved and 42 months for those who had not.

Discussion
In this study, we diagnosed 28 patients seen in the movement disorder clinic over a 71 month period as having psychogenic movement disorders. Although the diagnosis can be difficult and at times unsettling because of fear of misdiagnosis, we considered our diagnoses to be secure. In all patients the movement disorders were inconsistent or incongruent with classic organic syndromes and all patients had associated historical, clinical, and behavioural
features which seem to be typical of psychogenic movement disorders. Our experience supports the idea that many psychogenic movement disorders are clinically recognizable and definable disorders. The diagnosis may be difficult, however, as clinical heterogeneity exists and should be left to neurologists with considerable experience in diagnosing classic organic disorders.

The frequency of 3-3% for psychogenic movement disorders in our movement disorder population is similar to that reported by Fahn who indicated that 2-1% of his population of 3700 patients with idiopathic movement disorders were given that diagnosis. These estimates in a movement disorder clinic are based on a biased referral pattern with the most unusual cases being seen in a subspecialty clinic. It would indicate, however, that psychogenic movement disorder is very unusual in general neurological practice and that such a diagnosis should be made with extreme caution. These estimates are low when compared with the 9% figure for psychogenic neurological disorders in an outpatient general neuroradiology population and the 8% to 20% figures given for pseudoseizures in an intractable seizure population. Because the patient profile (distribution and frequency of various types) in our clinic is similar to that of Seimers and Reddy in Indianapolis, we believe that a similar frequency of psychogenic movement disorders will be found in other movement disorder clinics, especially those established in the 1980s.

The most common type of psychogenic movement disorder diagnosed in our clinic was psychogenic tremor, making up 50% of the patients. This finding is opposed to that of Monday and Jankovic who indicated that myoclonus was the most frequent. Our results support the statement of Fahn which stated that “shaking movements” were the most common type encountered.

In our population a substantial percentage of patients referred for tremor had psychogenic tremor (11-3%). We suspect that this high number is based, in part, on the fact that subspecialty clinics such as ours see mainly patients with essential and other tremor types, who are the most difficult to treat. The actual frequency of psychogenic tremor among patients with tremor overall is probably much lower. Psychogenic dystonia was diagnosed in 2-9% of patients referred for dystonia (idiopathic or secondary) and this result was similar to the 2-1% reported by Fahn. The rare occurrence of psychogenic parkinsonism (0-5%) is also in agreement with estimates made by Fahn (0-2%).

The clinical characteristics of the psychogenic movement disorders in our patients (Table 2) were typical of psychogenic movement disorder described elsewhere. Distractability of the patients was most common. Only one patient (seven) with psychogenic tremor did not have a change in tremor with distraction; a finding similar to that of Koller et al. This feature seems to be most important in making a diagnosis of psychogenic tremor and not very important in psychogenic dystonia. Three of the patients seen in this study with psychogenic dystonia were not distractable. Another feature specific to psychogenic tremor is the finding that the tremor could become entrained to the frequency of a repetitive movement of another limb, a finding previously described in relation to the tremor of psychogenic parkinsonism. Also, in three patients with psychogenic tremor, we noted clear signs of fatigue, including diaphoresis and breathlessness, and the movements would sometimes cease. After a brief rest they would start again. Tremors of essential tremor and Parkinson’s disease do not result in fatigue. All the other common features of psychogenic movement disorder seen in this study were present across all the types of movements. There were other clinical features which we found to be helpful clues in the diagnosis of psychogenic movement disorder which have not been emphasized in existing reports. Stimulus sensitivity of movements occurred in three patients with tremor and one with paroxysmal dystonia. Paroxysmal dystonic choreoathetosis is not associated with triggers and tremor of Parkinson’s disease is not stimulus sensitive. This characteristic, although not often discussed, seems to be fairly common as it has been noted in other studies. Six patients with psychogenic myoclonus were found to be noise sensitive and four others were light sensitive. One patient with psychogenic dystonia and jerky movements was sensitive to tapping on the buttock and Walters and Henning described a patient with post-traumatic stress disorder and psychogenic tremor induced by noise. Four patients had a history of prior psychogenic neurological disorder (not a movement disorder). Finally, the point has been made that patients with psychogenic movement disorders do not respond to frequently used and effective medications; however, response to medications not typically used in organic movement disorder occurred in three of our patients (Nos 3, 7). These additional characteristics could be helpful in the differential diagnosis of psychogenic movement disorders.

Our results also emphasized the frequent association of psychogenic movement disorder with other features seen on neurological examination and medical history which represent important diagnostic clues. First is the presence of other psychogenic features seen in nearly 75% of our patients. The most common were false weakness, non-physiological sensory loss, and gait disorders. Nearly half of the patients had two or more of these features and we think that this further strengthens the likelihood of a diagnosis of psychogenic movement disorder. By history, all previously published studies have indicated an association with prior diagnosis of psychiatric disease, particularly depression, and our study confirms this. We also examined two patients with paranoid schizophrenia and psychogenic movement disorder, a circumstance not often
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Discussed in the medical literature. Patient No 27 was particularly interesting because he claimed that his head tremor was being caused by "Satan". The tremor disappeared when his psychosis remitted. Other important diagnostic clues for psychogenic movement disorder include the presence of a clear precipitating event and secondary gain both of which were found in over 60% of our cases. Woolsey suggested that patients with spontaneous recovery from psychogenic disorders were more likely to have a clear precipitating event, but, we saw no such relation. He also noted that secondary gain would lead to fixed hysterical symptoms; however, five of 18 patients with secondary gain in this study improved or resolved, indicating that this is not necessarily the case.

The descriptive characteristics of the movement disorders seen in our patients were typical of previous reports, with some variations. Koller et al described the typical picture of psychogenic movement disorder as starting bilaterally, having complex characteristics (resting, postural, and kinetic), with variation in amplitude, frequency, and direction. This was typical of our cases of limb tremor, except that 30% were unilateral at onset and remained that way. Head tremor occurred at a similar frequency to the previous study. With regard to dystonia, Fahn and Williams reported that nearly half of their 21 patients were adults with abrupt onset, fixed foot dystonia which occurred at inception of the disorder or shortly thereafter. Lang also described lower extremity involvement in 12 of his 18 patients with psychogenic dystonia with many having fixed postures. One third of the cases of Fahn and Williams had non-familial, paroxysmal, non-kinetic dystonia with frequent prolonged attacks. In fact, Fahn has indicated that psychogenic movement disorder is the most common diagnosis in patients with paroxysmal dystonia. Two of our patients (Nos 19, 24) presented with foot dystonia and one (No 22) with paroxysmal dystonia; all would be typical for a psychogenic diagnosis. We also saw two patients with psychogenic blepharospasm. One had a sudden onset of eyelid fluttering and closure on the left which spread to the right. It stopped with distraction and worsened with eye movement testing. She had an associated psychogenic gait and a history of depression. The other patient had increased blinking, eyelid closure, and fluttering. It was associated with many remissions, six other psychogenic features on examination, and a history of two previous psychogenic neurological disorders.

The entire clinical picture resolved spontaneously. Although there were no cases of psychogenic blepharospasm in the large series of Fahn and Williams or Lang, this phenomenon has been recognized and presented in case reports. The general characteristics of psychogenic blepharospasm bear a strong resemblance to other psychogenic movement disorders. There is often a sudden onset in association with a stressful situation. The blepharospasm may be episodic in nature with spontaneous remissions and other characteristics not typical of essential blepharospasm or Meige's syndrome. Resolution occurs suddenly and there may be associated psychiatric disease and secondary gain. One patient presented with tremor and dystonia, a combination found in seven of the patients reported by Fahn and Williams, and five reported by Lang. In psychogenic myoclonus the movements are slow and sustained instead of the fast ones seen in organic disorders. Our patients were typical and also showed variations in frequency, amplitude, and direction and one patient made grunting sounds with each jerk.

Psychogenic parkinsonism was recently described by Lang et al in their study of 14 patients from three large movement disorder centres. These patients present with a constellation of signs and symptoms including tremor (with all the features of psychogenic tremor), slowness without fatigue or dystonia, pronkike, and dystonia. There was an unusual gait disorder with a bizarre response to postural stability testing. The rigidity is usually without cogwheeling and decreases with distraction or activating synkinetic movements in the opposite limb (rigidity in Parkinson's disease usually responds in the opposite manner). With walking patients may have decreased arm swing but the arm may be held tightly to the side or cradled in front. With retropulsion testing a slight push to the shoulders results in an almost catastrophic response with falling of the arms and reeling backwards without actually falling. Our two patients (Nos 13 and 26) had all these characteristics. In addition, neither had a masked face. One had give way weakness on examination and both had a history of psychiatric disease and a parent with Parkinson's disease. The onset in one was after giving long term care to two sickly parents, both, as in the series of Lang et al, gave a history of abrupt onset severe disease without progression, and both presented with a diagnosis of Parkinson's disease. One patient with a 10 year history presented on levodopa and did not worsen but levodopa was stopped. One unusual patient (No 28) presented to us with a presumptive diagnosis of tardive dyskinesia but was later diagnosed as having psychogenic movement disorder. This patient was prescribed the antiemetic promethazine, but received prochlorperazine from the pharmacy. After four doses of 25 mg an acute dystonic reaction developed. This was treated in a local emergency room with diphenhydramine and cleared. Since that event numerous complaints emerged including tongue movements. She described them as tongue thrusting but on examination various movements were seen including up and down and side to side. The movements disappeared with distraction, were inconsistent over time, and were non-stereotypic and thus, were incongruent with typical orobuccal-lingual dyskinesia. She also was found to have several other psychogenic features on examination, a history of psychiatric disease, and multiple somatisations.
type of psychogenic movement disorder has not been previously described.

The coexistence of psychogenic movement disorder and organic movement disorders has been described in six patients by Ranawaya et al. Fahn and Williams also reported a patient with both organic and psychogenic dystonia, Monday and Jankovic indicated that three of their patients with psychogenic myoclonus had essential tremor, and pseudo-tics usually occur in patients with Tourette's syndrome. Our results suggest that this combination occurs in about 25% of patients with psychogenic movement disorders. This estimate is somewhat higher than the 10–15% previously reported and similar to the estimated frequency of combined pseudoseizures and epileptic seizures. In our patients the most common organic disorders were dystonia (three patients) and essential tremor (two patients) and the most common psychogenic movement disorder was tremor (five patients). Ranawaya et al had essentially the same experience. These authors indicated that the organic disorder was usually present years before the onset of psychogenic movement disorder and that the psychogenic movements generally occurred in the same or contiguous location as the organic features and we concur with these findings. The possibility of an underlying organic movement disorder should always be considered when a patient presents with a possible psychogenic movement disorder.

In attempting to explore the outcomes of our patients eight were completely lost to follow-up. Of the 20 remaining, 10 were unresolved despite therapeutic attempts. This finding, in conjunction with the difficulty in documenting cases, underlines the point made by Fahn that "symptoms will disappear only when the patient is ready to give them up". An important point brought out by our outcome results was that those patients with a shorter duration of psychogenic movement disorder seem more likely to resolve. This finding is supported by the work of Lempert et al in which most patients with psychogenic disorders for two weeks or less resolved whereas most patients with symptoms for longer than six months did not. Fahn and Williams found similar trends, but indicate that long duration does not necessarily preclude response, only that those with a shorter duration of disease have a better prognosis.

This emphasises the need for the neurologist to reach an early definitive diagnosis. If facilities are available, one should initiate the treatment plan recently outlined by Fahn as early as possible.

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