Psychogenic Movement Disorders

Janis M. Miyasaki, Daniel S. Sa, Nestor Galvez-Jimenez, Anthony E. Lang

ABSTRACT: Psychogenic movement disorders (PMD) are challenging to diagnose and to treat. Since the nineteenth century, PMDs were recognized and described in painstaking detail. In the modern neurology clinic, PMDs may comprise 2-25% of the patient population. Recognition of the various types of PMDs, differentiation from organic illness and an approach to PMDs are described in this article.

RÉSUMÉ: Troubles du mouvement d'origine psychogène. Les désordres du mouvement d'origine psychogène posent un défi diagnostique et sont difficiles à traiter. Ces désordres sont connus et décrits en détail depuis le dix-neuvième siècle. Dans une clinique de neurologie moderne, ces désordres peuvent atteindre de 2 à 25% des patients. L'identification des différents types de désordres du mouvement d'origine psychogène, les caractéristiques qui les différentient des maladies organiques et la prise en charge de ces désordres sont décrites dans cet article.

Can. J. Neurol. Sci. 2003; 30: Suppl. 1 – S94-S100

Recognition of psychogenic movement disorders (PMD) has a long and distinguished history in neurology, beginning with Charcot in the nineteenth century.[1] His interest in hysteria led to theories that women, working-men,[2] children[3] and effeminate men[4] commonly manifested hysterical neurologic deficits. In addition to displaying these patients to medical staff and lay persons, Charcot employed suggestion and hypnosis in their treatment[5] and claimed excellent success.

Later, Gowers was moved to write, "there are few organic diseases of the brain that the great mimetic neurosis may not simulate. (Therefore) we must never infer that this is the primary disease until we have searched for, and excluded, the symptoms of organic disease".[6] Regrettably, this admonition resulted in exhaustive investigations for many patients with psychogenic disorders.

Perhaps the most elegant descriptions of psychogenic movement disorders belongs to Sir Henry Head.[7] In 1922, he wrote the following description of psychogenic dystonia "… any attempt to break down a spasm of this kind, to open the closed hand, or to straighten the flexed knee, meets with intense resistance … resistance may be experienced not only in pushing the head towards the normal shoulder, but also in moving it farther in the direction of the affected side". In describing psychogenic ataxia, Head described finger to nose testing showing past-pointing to the same side of the head but, if the head was pushed in the direction of the past-pointed finger so as to make contact with it, the affected limb would deviate even further away from the head.[7] Examinations of such patients led Head to conclude that "hysteria is sometimes said to imitate organic affections; but this is a highly misleading statement. The mimicry can only deceive an observer ignorant of the signs of hysteria or content with perfunctory examination".[7]

However, the risk of wrongly labeling someone with neurologic disability is equal to misdiagnosing organic illness as psychogenic. For example, focal dystonias such as torticollis (turning the head away from conflict) and writer's cramp (sexual overtones caused by the pen ejaculating ink) were labeled hysterical, yet today, are accepted as organic illness.

Neurologists must tread through this diagnostic minefield. We will discuss the epidemiology of PMD, clinical features, common PMDs and classification of PMD.

Epidemiology

Estimates of PMD in neurology clinics range from 2.6%[8] to 25%.[9] The higher figure likely represents an over-estimate since the patients were referred to a tertiary centre. At the Movement Disorders Clinic of the Toronto Western Hospital, 64 patients were diagnosed with PMD between July 2000 and May 2002. The predominant types of PMD are as follows: tremor (32.8%), dystonia (25%), myoclonus (25%), parkinsonism (6.1%) and gait disorder (10.9%). Forty-nine of the patients (76.5%) were women.

A series from the Columbia-Presbyterian Medical Center,[10] described 131 patients with PMD. Referral bias (their site is also a dystonia research center) resulted in the following distribution of PMD: dystonia (53%), tremor (13%), gait disturbances (9%), psychogenic myoclonus (2%), blepharospasm and facial movements (2%), parkinsonism (1.9%), tics (1.3%) and stiff person syndrome (0.6%). Paroxysmal dyskinesias/shaking and

From the Morton and Gloria Shulman Movement Disorders Centre, Toronto Western Hospital, Department of Medicine (Neurology), University of Toronto, Toronto, ON, Canada (JMM, DSS, AEL); and the Cleveland Clinic Florida, Weston, Florida, USA (NG-J)

Reprint requests to: Janis M. Miyasaki, Toronto Western Hospital, 11-MP, 399 Bathurst Street, Toronto, ON M5T2S8 Canada

undifferentiated movements comprised the remainder of their cases (9%).

Co-existing psychiatric diagnoses are common among these patients. Feinstein and colleagues[11] (from our unit in Toronto) interviewed 42 patients with a diagnosis of documented or clinically established PMD. Anxiety (38.1%) and major depression (19.1%) were common. Other lifetime psychiatric diagnoses included adjustment disorder (9.5%), schizoaffective disorder (2.4%), bipolar disorder (2.4%) and alcohol or sedative abuse (2.4% for each).

One can, therefore, develop a profile of the typical PMD patient. Ford and colleagues[12] describe such a patient as young (mean age 36 years), female, of average or above average intelligence with a mean duration of symptoms of five years, unable to work and on disability. The psychiatric diagnoses given to these patients included conversion disorder (75%), somatization disorder (12.5%), factitious disorder (8%) and malingering (4%). Other secondary diagnoses were present with dysthymia (67%) being most common.

Table 1: General clues suggesting that a movement disorder may be psychogenic [10,14,15]

A) Historical

1. Abrupt onset.
2. Static course.
3. Spontaneous remissions (inconsistency over time).
4. Obvious psychiatric disturbance.
5. Multiple somatizations.
6. Employed in a health profession.
7. Pending litigation or compensation.
8. Presence of secondary gain.
9. Young female.

B) Clinical

1. Inconsistent character of the movement (amplitude, frequency, distribution, selective disability).
2. Paroxysmal movement disorder.
3. Movements increase with attention or decrease with distraction.
4. Ability to trigger or relieve the abnormal movements with unusual or nonphysiological interventions (e.g. trigger points on the body, tuning fork).
5. False weakness.
6. False sensory complaints.
7. Self-inflicted injuries.
8. Deliberate slowness of movements.
9. Functional disability out of proportion to exam findings.
10. Movement abnormality that is bizarre, multiple or difficult to classify.

C) Therapeutic responses

1. Unresponsive to appropriate medications.
2. Response to placebos.
3. Remission with psychotherapy.

Table 2: Clinical features of organic dystonias which sometimes encourage a misdiagnosis of a psychogenic disorder. [39,40,41]

1. The movements in dystonic syndromes can be quite varied including prolonged spasms, sinuous writhing, brief myoclonic jerks, slow rhythmical movements and faster tremors.
2. Dystonia can remit in up to 20% of patients especially in those with cervical dystonia (spasmodic torticollis).
3. Patients with idiopathic torsion dystonia have no other neurological deficits and normal ancillary investigations.
4. Dystonia can be task-specific (i.e., writer's cramp) or may be purely action-induced (e.g., foot dystonia when walking forwards but not backwards or oromandibular dystonia only when attempting to speak or, alternatively, only on eating).
5. Occasional patients experience dystonia at rest with improvement on action ("paradoxical dystonia").
6. Dystonia can be relieved by "sensory tricks" (geste antagoniste). The best known of these occur in patients with cervical dystonia where light touch or pressure very often will correct the abnormal head position. Many other examples are seen in a variety of dystonias.
7. Organic dystonia can be relieved by relaxation and hypnosis and is typically worsened by emotional stress.
8. Dystonia can be paroxysmal (e.g., paroxysmal kinesigenic choreoathetosis, paroxysmal nonkinesigenic choreoathetosis) or can show diurnal variation, as seen most prominently in dopa-responsive-dystonia.

Review of psychiatric diagnoses

In assessing and diagnosing those with PMD, knowledge of terminology and diagnostic criteria by the American Psychiatric Association's Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM IV)[13] is essential. We will discuss somatoform disorders, factitious disorders and the histrionic personality.

Somatoform disorders are characterized by the "presence of physical symptoms that suggest a general medical condition and are not fully explained by a general medical condition, by the direct effects of a substance or by another mental disorder." Impairment in social, occupational or other areas of functioning or significant distress must be present. The pertinent subdivisions of somatoform disorders to this discussion include somatization disorder and conversion disorder. Somatization disorder consists of recurring, multiple, clinically significant somatic complaints (Table 1). The complaints must result in medical treatment or significant impairment. Somatic complaints must begin before the age of 30 years and continue over a period of several years. Historical details include pain to over four sites, gastrointestinal symptoms, sexual or reproductive symptoms other than pain and pseudoneurologic symptoms. Symptoms are not feigned or intentionally produced as in malingering. In conversion disorder, the main symptoms or deficit involves voluntary motor or sensory function (Table 2). Psychological factors are associated with the deficit based on initiation or

https://doi.org/10.1017/S0317167100003292 Published online by Cambridge University Press

Table 3: Differences between "Causalgia-Dystonia" and Primary Torsion Dystonia *

Causalgia-dystonia	Idiopathic dystonia
Clear preponderance of women	No preponderance of women
No family history	Positive family history not uncommon
Painful (causalgia)	Usually painless
Vasomotor, sudomotor and trophic changes	Such changes not seen
Fixed spasm	Mobile spasms
Contractures common and early	Contractures uncommon and late
No geste antagoniste	Geste frequent
No improvement with sleep	Sleep often improves
Poor response to botulinum toxin and other therapy	Often responds to botulinum toxin, others
Onset in leg in adult	Adult leg onset very rare
Rapid spread	Slow progression

*Adapted from Bhatia [25]

Table 4: French-school classification of hysterical gait disorders at the turn of the century.

Charcot and Tourette	Roussy and Lhermitte
Astasia-abasia	Astasia-abasia
Paralytic	Pseudotabetic
Ataxic	Pseudopolyneuritic
Choreiform	Tight-rope walker
Trepidant	Robot
Habit limping	
Choreic	
Knock-kneed	
As on a sticky surface	
As through water	

(Adapted from Keane after Southard and Roussy and Lhermitte) [30]

exacerbation of the symptom coincident with conflict or other stressors. The symptoms are not intentionally produced or feigned as in malingering. Note that the criteria for conversion disorder does not have an age limit for onset. Typically, resolution is rapid, however recurrence may occur in up to 25%.[13]

Factitious disorders are characterized by physical or psychological symptoms that are intentionally produced or feigned in order to assume the sick role (Table 3). In other words, the motivation is psychological. In malingering, the symptoms are intentionally produced for an external gain (avoiding work, compensation, and more favourable living arrangements) (Table 4).

A personality disorder may predispose to PMD. For example, the histrionic personality disorder is characterized by pervasive and excessive emotionality and attention-seeking behaviour. They are superficially charming and flirtatious but need to be the centre of attention. They are highly suggestible and theatrical. Other personality disorders such as borderline personality or antisocial personality may be present. The diagnosis of personality disorders requires determination of an enduring pattern of inner experience and behaviour that deviates markedly from the expectations of the individual's culture and is pervasive, inflexible and stable over time. Establishing a diagnosis of a personality disorder is best left to psychiatrists.

HISTORICALAND CLINICAL FEATURES OF PMD

Just as a profile of the typical PMD patient may be derived, red flags to suggest the diagnosis of PMD are recognized.[10,14,15] Abrupt onset, especially with maximal severity at onset followed by a static course, is not typical of organic movement disorders. However, rapid onset dystonia-parkinsonism, Wilson's disease and post-lesion movements can have abrupt onset or very rapid deterioration. Paroxysmal movements and spontaneous complete remissions are also common in PMD although these can be features of organic disorders as well. The presence of psychiatric disturbance or multiple somatizations should lead one towards a diagnosis of PMD as does the presence of secondary gain or pending litigation or compensation. Due to familiarity with organic illness, many movement disorders neurologists find that PMD commonly occurs in health professionals or allied health professionals. Other historical details including unresponsiveness to appropriate medications (for example the patient with parkinsonism who does not respond to high doses of levodopa) or remission with psychotherapy are suggestive of PMD.

As Head wrote in 1922,[7] keen observation and thorough knowledge of organic illness is essential to the diagnosis of PMD. In typical movement disorders, the characterization of the movement (distribution, amplitude, frequency, and direction) is consistent. Distracting manoeuvres tend to change PMD. Marked decrease or complete abolition of PMD may occur with such distraction. In organic illness, distraction typically increases the involuntary movement. Exceptions to this rule include tics, stereotypies and tardive akathisia. In some cases, entrainment occurs, where the "affected" limb takes on a new frequency similar to that of a complex task performed by the normal limb. This feature is especially useful in psychogenic tremor. Psychogenic movement disorders may be triggered or relieved by trigger point or nonphysiological interventions. Again, caution must be exercised, since sensory tricks ("geste antagoniste") relieve organic dystonia. The "company" that the movement keeps can also be a clue to PMD. For example, false weakness or sensory complaints, self-inflicted injuries, deliberate, excruciating slowness and bizarre, multiple movements would increase the likelihood of PMD.

As noted above, there are exceptions to every "rule". As Gowers[16] noted, "hysteria, it must be remembered, is common not only as an isolated, but also as a conjoined, morbid state and that hysteria can be the consequence of organic disease". More recently, Ranawaya et al[17] reported six patients with organic movement disorders combined with PMD. In keeping with the criteria for somatoform illness, all patients felt the PMD was impairing. Significantly, all patients felt that the PMD was more disabling than their pre-existing organic movement disorder.

https://doi.org/10.1017/S0317167100003292 Published online by Cambridge University Press

Specific psychogenic movement disorders

Psychogenic dystonia

Dystonia is a syndrome dominated by sustained muscle contractions, often causing twisting and repetitive movements that result in abnormal or dystonic postures.[18] In cases of organic dystonia, typically there is gradual onset with activity that progresses to presence at rest. Involvement is rarely in the lower limb when dystonia begins in adult life. Remarkably, pain is not the primary feature despite extremely contorted postures. Fahn and Williams[10] and Lang[19] described series of patients with psychogenic dystonia. The majority of patients had onset of dystonia at rest and more commonly involved the lower limb. Generalized dystonia was much higher than expected. Associated features in their patients included give-way weakness/paralysis, nonanatomic sensory changes, excessive slowness, marked resistance to passive movements and multiple somatizations. Muscle atrophy or joint contractures may be present in PMD. In these instances, they represent secondary changes due to lack of use or prolonged maintenance of a tonic posture.

Controversy surrounds the issue of peripheral trauma and abnormal postures. Reports of case series of dystonia following peripheral nerve injury are abundant.[20-23] The only case-control study, however, failed to show an association between idiopathic dystonia and a history of previous injury.[24] Review of the above reports reveals a clinical picture that differs significantly from idiopathic torsion dystonia. Thus, the term "causalgia-dystonia" was coined by Bhatia.[25] He described fixed spasm with early appearance of contractures, rapid onset, severe pain and vasomotor, sudomotor and trophic changes. His patients did not report sleep benefit, geste antagoniste and did not respond to botulinum toxin. None of his patients had a positive family history of dystonia. Women predominated in this group. Despite significant disability, none of these patients had overt nerve damage.[26]

At Toronto Western Hospital, a series of 13 patients with involvement of the neck and shoulder following minor injury revealed similar characteristics. Fixed postures occurred after trivial injury, frequently work-related and involving the neck or shoulder area. The posture developed to its maximum within two weeks of the inciting event. Pain predominated. The majority of patients were involved with litigation and/or compensation matters. Minnesota Multiphasic Personality Inventory suggested conversion disorders in all 13. Sodium amytal interview resulted in improvement of pain and posture in most patients. Other associated features included false weakness, nonanatomic sensory loss, distractibility, improvement with sham botulinum toxin injections, symmetrical neck tan and surreptitious observation of a symptom-free period. Previous physical examinations commented on muscle hypertrophy, however during amytal interview or general anesthesia, only one patient had true muscle hypertrophy. These features suggest a non-organic basis for the postures. Therefore, we suggest that true peripheral injury induced dystonia is rare and patients should not be labeled as such without substantial clinical evidence.

Psychogenic tremor

Tremor is an involuntary, rhythmic, sinusoidal movement due in part to regular rhythmical contractions of reciprocally innervated muscles.[18] Our centre reported a series of patients with psychogenic tremor.[27] These patients experienced abrupt onset with immediate or virtually immediate maximal severity. Tremor typically followed a known trivial precipitant. Tremor persisted at rest, with postural maintenance and during action. The only organic tremor to exhibit such features is a cerebellar outflow tremor, as seen in multiple sclerosis, ischemic infarction of the posterior fossa or cerebellar degeneration. In contrast to this so-called Holme's tremor, the amplitude of psychogenic tremor remains roughly the same rather than increasing from rest to postural maintenance and further still with action. Complete suppression with distraction was often present. Alternatively, the tremor would entrain with distracting manoeuvres. Forcefully restraining the limb often caused tremor to appear in a previously unaffected limb, head or trunk.

Electrophysiological studies may be helpful in the diagnosis of psychogenic tremor. The consistency of frequency and distractibility can be documented. Co-activation or increased amplitude with weighting of the limb frequently occurs in psychogenic tremor.[28]

Psychogenic gait disorders

Psychogenic gait disorders are common, accounting for 1.5% to 26% of patients with neurologic complaints.[29,30] A series from the Munich University Clinic involved videotape analysis to develop diagnostic criteria for psychogenic gait.[29] Important features included hesitation (16.2%), excessive slowness of movements (35%), fluctuations in gait impairment with "uneconomic" postures and wasting of muscle energy (51%), "walking on ice" gait pattern (30%) and a "psychogenic Romberg" test (32%). If one or more of the features were present, the diagnosis of psychogenic gait disturbance could be made on phenomenological grounds alone with over 90% certainty. Other features included knee buckling without falls, astasia and vertical shaking tremor, suffering or strained facial expression with moaning, mannered posturing of hands and grasping of the leg along with hyperventilation. Other neurologic signs included false weakness, scissoring, flailing of the arms, bizarre tremor, pseudoataxia and voice abnormalities.

Psychogenic myoclonus

Myoclonus is brief, shock-like muscle contractions (positive myoclonus) or sudden lapses in tone (negative myoclonus) as seen in asterixis. Psychogenic myoclonus is common comprising 11%[31] to a high of 25% at Toronto Western Hospital of all psychogenic movement disorders.

A series of 18 patients was reported by Monday and Jankovic.[32] Women predominated and the average age was 42 years. Eighty-three percent had a precipitating event and 61% reported sudden onset or onset over several days in 38%. Stress worsened myoclonus in these patients. The distribution of myoclonus was segmental in 55% followed by generalized in 39% and focal in 5% of patients. Myoclonus was present at rest in all and exacerbated by movement. Other neurological findings included tremor, focal dystonia and gait abnormalities. These associated signs were felt to be psychogenic in origin. Formal neuropsychological testing revealed psychiatric pathology in 61%, however 55% of patients had overt psychiatric disturbances prior to the onset of myoclonus. The majority of patients available for follow-up reported improvement. A significant percentage

https://doi.org/10.1017/S0317167100003292 Published online by Cambridge University Press

reported worsening of myoclonus. The authors found distractibility was helpful in establishing the diagnosis.

An objective evaluation of myoclonus can be invaluable. Electrophysiology of myoclonus should reveal brief (cortical origin – EMG burst of 10-50 ms,[33] brainstem – EMG burst >100 ms[33] and reflex myoclonus – short latency to EMG burst of 60-70 mg[34]). Propriospinal myoclonus has latency to onset outside these ranges since it is a slower, polysynaptic response. Patients with psychogenic myoclonus show variable and inconsistent muscle activation patterns and stimulus-induced responses habituate as seen in normal startle response.[35] The readiness potential or bereitschaftspotential is typically not seen in psychogenic myoclonus.[36] In psychogenic myoclonus, the presence of a bereitschaftspotential strongly suggests a voluntary movement. Timing the onset of movement from the time to stimulus is also helpful. In psychogenic myoclonus, the latency falls within the range of voluntary reaction time (100-120 ms, P. Ashby, personal communication).

These physiologic studies are also useful in startle. Startle diseases or hyperekplexia has a rostral caudal progression similar to that seen in reticular reflex myoclonus or reflex myoclonus of brainstem origin.[35] Patients have an exaggerated response and lack habituation. In normal physiologic startle, there may be a varying pattern of muscle contraction and habituation occurs after an exaggerated startle response.

Psychogenic parkinsonism

A profile of psychogenic parkinsonism arises from a report by three tertiary referral centres.[37] Men and women were equally represented. The average age was 47 years, younger than organic Parkinson's disease. Seventy-one percent of patients noted sudden onset of symptoms after a work-related injury or motor vehicle accident. The majority of patients presented with bilateral symptoms (57%). Tremor was present in 85% with the majority having characteristic features of psychogenic tremor. The tremor persisted in postures and with action, lacking the dampening with adopting a new posture or with movement in organic parkinsonism. Excessive slowness was noted, but fatigue with decremental amplitude and arrest in ongoing movement were lacking. Rigidity was similar to voluntary resistance or difficulty relaxing and would decrease with performance of synkinetic movements of an opposite limb. In Parkinson disease, this manoeuvre accentuates rigidity. Gait had a variety of bizarre or atypical features including antalgia. Postural stability assessment using the pull-test elicited an exaggerated or extreme response. Other features of the response to the pull-test were helpful. One patient whose "affected" arm was slow and stiff and held tightly at the side while walking and running, flailed both arms rapidly upwards when tested for postural stability. Imaging of the dopamine system using PET or SPECT may provide useful supportive evidence for a diagnosis of psychogenic parkinsonism in difficult or questionable cases.

Establishing the diagnosis

Psychogenic movement disorders are a diagnostic challenge. An experienced neurologist or movement disorders specialist should make the diagnosis since comparison to the organic counterpart is essential. Therefore, the physician must be familiar with the entire field of movement disorders and recent literature. In selected cases, admission to hospital allows for continuous observation and/or video monitoring. Appropriate tests to rule out organic disease, especially routine imaging, should be undertaken.[10] This also convinces the patient that the physician takes the physical complaints seriously and does not dismiss them. Ethical, legal and physician-patient relationship issues make the use of placebo controversial. Therefore, the use of placebo should be limited to patients in whom there remains diagnostic uncertainty despite a full clinical assessment and investigations. Once the diagnosis is established, testing should not continue to avoid the sense of diagnostic uncertainty. It is important to emphasize that the diagnosis should rest on positive clinical criteria applied by an experienced physician. This should not be considered a diagnosis of exclusion made only after every imaginable test is performed and all conceivable organic diseases eliminated.

Apsychiatrist should participate in therapy and be involved in establishing the diagnosis. However, many patients may not have overt psychopathology even after an extended assessment. It should be emphasized that a neurologist with experience in the field of movement disorders makes the diagnosis of PMD. The diagnosis should not be made or refuted by a psychiatrist. It is extremely counter-productive in the management of these patients when, as often occurs, a psychiatrist gives a patient "psychiatric clearance" and insisting that an organic basis must be present.

The errors in diagnosis of PMD include misdiagnosing PMD when there is an organic basis or PMD misdiagnosed as organic. As a result of either type of error, the patient does not receive the appropriate therapy or may be subjected to invasive procedures.

Categories of diagnostic certainty

Fahn and Williams[10] outlined the levels of certainty for the diagnosis of PMD. In documented PMD, the movements must be persistently relieved by psychotherapy, suggestion, administration of placebos or the patient is witnessed as being free of symptoms when left alone unobserved. Clinically established PMD is inconsistent over time or incongruent with the classical definition of movement disorders. The patient must show additional features that suggest psychogenicity such as other neurological signs that are definitely psychogenic. Probable PMD includes movement disorders that are inconsistent or incongruent with classical definitions but without other features suggesting psychogenicity. Alternatively, the movement disorder is consistent with organic disease but either accompanying neurological signs are definitely psychogenic or multiple somatizations are present. When obvious emotional disturbance accompanies a movement disorder that is consistent with a known organic disease, the appropriate diagnosis is possible PMD. Considerable caution must be exercised in diagnosis and management of patients in the second category of probable PMD and those with possible PMD given the common occurrence of psychological or behaviour difficulties in patients with a variety of organic movement disorders.

Approach to therapy of PMD

The manner of disclosure of the diagnosis of PMD has equal importance to the preceding investigations. Empathy and a non-judgmental manner are essential. Ford, Williams and Fahn[12]

https://doi.org/10.1017/S0317167100003292 Published online by Cambridge University Press

formally stated that the use of a neurobiological explanation for the patient's symptoms helps in establishing trust, acceptance and understanding of their diagnosis and recovery of the symptoms. The nature of the movement disorder should be confirmed (i.e., the patient is told he/she has a form of dystonia, tremor, myoclonus) but that the problem is not due to a severe or permanent structural brain disease. Psychiatric consultation may be introduced as a means of coping with the disability. For receptive patients, discussing the powerful interaction between the mind and physical state in illnesses such as hypertension, coronary artery disease and increased risk of cancer may increase the acceptance of a psychological contribution to the movement disorder.

Prognosis

The prognosis for functional recovery is often poor. Given the significant disability experienced by these patients, there is an urgent need for more research into various treatment modalities. At Toronto Western Hospital, a subset of our PMD patients was examined in an ambulatory care setting (J. Fine, A. Nieves and A. Lang, unpublished observations). Twenty-three patients were identified. These patients generally had an established movement disorder for some time before their assessment in our centre. All continued to manifest PMD with a mean duration of symptoms of 8.6+/-8.5 years. Significant disability persisted with 82.6% reporting impairment in activities of daily living. By contrast, patients with a conversion disorder with recent onset often have a good prognosis.[38] Condition upon discharge predicted the likelihood of recovery with 96% of patients experiencing improvement in hospital returning to premorbid function. Other features associated with good outcome include a clear emotional trigger or precipitant and lack of longstanding psychopathology.[38]

The rapid settlement of litigation or compensation may improve the chance of recovery since unresolved economic issues provide an "incentive" for ongoing PMD even in patients with somatoform or conversion disorders. Factitious disorders and malingering respond poorly and unpredictably. The latter is not considered a psychiatric illness and these individuals may be investigated and successfully "caught out" by detectives working for insurance agencies.

Ford, Williams and Fahn[12] state that even "…patients with many years of established psychogenic symptomatology were able to make full recoveries". However, their treatment consisted of prolonged hospitalization with intensive physical and psychiatric therapy. Long-term follow-up after discharge was not available.

Conclusions

Neurology is prone to pitfalls in diagnosis of psychogenic disorders more than any other internal medicine specialty due to our lack of biologic markers of illness. Imaging often does not disclose alterations in function. More than any other specialty, we rely upon the art of medicine – careful, some would say obsessive, history taking and observation. This is especially required with PMDs. Psychogenic movement disorder is a further challenge since it calls upon an exhaustive knowledge of organic illness and its many manifestations. Establishing the diagnosis is only the beginning since successful outcome for these patients depends on empathy on the part of the physician and receptiveness in the patient. Further knowledge about the long-term outcome of these patients and a better approach to treatment is mandatory in view of their poor prognosis and the level of disability they cause.

References

1. Charcot J-M. Hystero-epilepsy: a young woman with a convulsive attack in the auditorium. In: Goetz CG, (Ed.). Charcot The Clinician. The Tuesday Lessons. New York: Raven Press, 1987: 102-122.
2. Freud S. Charcot. In: Sutherland JD, (Ed.). Collected Papers – Vol 1. London: The Hogarth Press, 1957: 9-23.
3. Guillain G. J.M. Charcot, 1825-1893, His Life – His Work. New York: Paul B. Hoeber, Inc., 1959.
4. Havens LL. Charcot and hysteria. J Nerv Ment Dis 1966; 141:505-516.
5. Lecrubier Y. Images in psychiatry – Jean-Martin Charcot, 1825-1893. Am J Psychiatry 1995; 151:1:121.
6. Gowers WR. A Manual of Diseases of the Nervous System. Philadelphia: Blakiston, 1888.
7. Head H. The diagnosis of hysteria. BMJ 1922; 1:827-829.
8. Snyder S, Strain JJ. Somatoform disorders in the general hospital inpatient setting. Gen Hosp Psychiatry 1989; 11(4):288-293.
9. Schepank H, Hilpert H, Honmann H, et al. The Mannheim Cohort Project – prevalence of psychogenic diseaes in cities. Z Psychosom Med Psychoanal 1984; 30(1):43-61.
10. Fahn S, Williams PJ. Psychogenic dystonia. Adv Neurol 1988; 50:431-455.
11. Feinstein A, Stergiopoulos V, Fine J, Lang AE. Psychiatric outcome in patients with a psychogenic movement disorder: a prospective study. Neuropsychiatry Neuropsychol Behav Neurol 2001; 14(3):169-176.
12. Ford B, Williams DT, Fahn S. Treatment of psychogenic movement disorders. In: Kurlan R, (Ed.). Treatment of Movement Disorders. J.B. Lippincott Company, 1995: 475-485.
13. American Psychiatric Association. Diagnostic and statistical manual of mental disorders, 4th edition (DSM-IV), Washington. American Psychiatric Assoc 1994.
14. Barclay CL, Lang AE. Other secondary dystonias. In: Tsui J, Calne DB, (Eds.) Handbook of Dystonia. New York: Marcel Dekker, 1995: 267-305.
15. Koller W. Movement Disorders: Which Ones are Real? Malingering and Conversion Reactions. Washington, D.C.: American Academy of Neurology Annual Meeting, 1994: #222-3-225-25.
16. Gowers WR. Epilepsy and Other Chronic Convulsive Diseases: Their Causes, Symptoms & Treatment. New York: Dover Publications, Inc., 1964.
17. Ranawaya R, Riley D, Lang AE. Psychogenic dyskinesias in patients with organic movement disorders. Mov Disord 1990; 5:127-133.
18. Weiner WJ, Lang AE. Movement Disorders – A Comprehensive Survey. New York: Futura Publishing, 1989.
19. Lang AE. Psychogenic dystonia: a review of 18 cases. Can J Neurol Sci 1995; 22:136-143.
20. Marsden CD, Obeso JA, Traub MM, et al. Muscle spasms associated with Sudeck's atrophy after injury. BMJ 1984; 288:173-176.
21. Schott JD. Induction of involuntary movements by peripheral trauma: an analogy with causalgia. Lancet 1986; 2:712-715.
22. Schott GD. The relationship of peripheral trauma and pain to dystonia. J Neurol Neurosurg Psychiatry 1985; 48:698-701.
23. Jankovic J, Van Der Linden C. Dystonia and tremor induced by peripheral trauma: predisposing factors. J Neurol Neurosurg Psychiatry 1988; 51:1512-1519.
24. Fletcher NA, Harding AE, Marsden CD. A case-control study of idiopathic torsion dystonia. Mov Disord 1991; 6:304-309.
25. Eidelberg D, Takikawa S, Wilhelmsen K, et al. Positron emission tomographic findings in Filipino X-linked dystonia-parkinsonism. Ann Neurol 1993; 34:185-191.

https://doi.org/10.1017/S0317167100003292 Published online by Cambridge University Press

26. Bhatia KP, Bhatt MH, Marsden CD. The causalgia-dystonia syndrome. Brain 1993; 116:843-851.
27. Kim YJ, Pakiam AS, Lang AE. Historical and clinical features of psychogenic tremor: a review of 70 cases. Can J Neurol Sci 1999; 26(3):190-195.
28. Deuschl G, Köster B, Lücking CH, Scheidt C. Diagnostic and pathophysiological aspects of psychogenic tremors. Mov Disord 1998; 13(2):294-302.
29. Lempert T, Brandt T, Dieterich M, Huppert D. How to identify psychogenic disorders of stance and gait. J Neurol 1991; 238:140-146.
30. Keane JR. Hysterical gait disorders: 60 cases. Neurology 1989; 39:586-589.
31. Williams DT, Ford B, Fahn S. Phenomenology and psychopathology related to psychogenic movement disorders. In: Weiner WJ, Lang AE, (Eds.) Behavioural Neurology in Movement Disorders. New York: Raven Press, 1994: 231-257.
32. Monday K, Jankovic J. Psychogenic myoclonus. Neurology 1993; 43:349-352.
33. Obeso JA, Artieda J, Martinez-Lage JM. The physiology of myoclonus in man. In: Quinn NP, Jenner PG, (Eds.) Disorders of Movement. Clinical, Pharmacological and Physiological Aspects. London: Academic Press, 1989: 437-444.
34. Hallett M, Chadwick D, Marsden CD. Cortical reflex myoclonus. Neurology 1979; 29:1107-1125.
35. Thompson PD, Colebatch JG, Brown P, et al. Voluntary stimulus-sensitive jerks and jumps mimicking myoclonus or pathological startle syndromes. Mov Disord 1992; 7:257-262.
36. Terada K, Ikeda A, Van Ness PC, et al. Presence of bereitschafts-potential preceding psychogenic myoclonus: clinical application of jerk-locked back averaging. J Neurol Neurosurg Psychiatry 1995; 58:745-747.
37. Lang AE, Koller WC, Fahn S. Psychogenic parkinsonism. Arch Neurol 1995; 52:802-810.
38. Couprie W, Wijdicks EFM, Rooijmans HGM, vanGijn J. Outcome in conversion disorder: a follow up study. J Neurol Neurosurg Psychiatry 1995; 58:750-752.
39. Verma A, Berger JR, Bowen BC, Sanchez-Ramos J. Reversible parkinsonian syndrome complicating cysticercus midbrain encephalitis. Mov Disord 1995; 10:215-219.
40. Herz E. Dystonia II. Clinical classification. Arch Neurol Psychiat (Chicago) 1944; 51:319-355.
41. Marsden CD, Harrison MJG. Idiopathic torsion dystonia (dystonia musculorum deformans). A review of forty-two patients. Brain 1974; 97:793-810.

https://doi.org/10.1017/S0317167100003292 Published online by Cambridge University Press