# Motor Conversion Disorders Reviewed From a Neuropsychiatric Perspective

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***Background:*** Conversion disorder is a somatoform disorder defined by the presence of pseudoneurologic symptoms relating to voluntary sensory or motor function. The correct diagnosis of conversion disorder presenting with motor symptoms is complicated by the lack of gold-standard diagnostic tests and the absence of a universally accepted set of positive diagnostic criteria. This article reviews the epidemiology, pathophysiology, presentation, differential diagnosis, treatment, and prognosis of motor conversion, placing emphasis on diagnostic validity, reliability, and utility, while evaluating the empirical evidence supporting diagnostic and treatment strategies.

***Data Sources and Study Selection:*** Literature searches were carried out in PubMed using the keywords *conversion disorder*, *motor conversion*, *dystonia*, *psychogenic*, *hysteria*, *somatization*, *motion disorder*, *movement disorder*, and *pathophysiology*. Articles and book chapters in the author's personal collection were also utilized.

***Conclusions:*** Advances in neuropsychiatric research are leading to significant improvements in the diagnosis and understanding of motor conversion disorders. Positive, objective, and quantitative diagnostic criteria show significant promise for enhancing diagnostic accuracy. Current pathophysiologic research has begun to provide mechanistic explanations for conversion symptoms, thus blurring the distinction between psychogenic and organic motor disorders.

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Conversion disorder is defined by the presence of pseudoneurologic symptoms relating to voluntary sensory or motor function and is categorized as a somatoform disorder within the framework of the *Diagnostic and Statistical Manual of Mental Disorders*, Fourth Edition (DSM-IV).[1] Somatoform disorders were recognized 4000 years ago by the ancient Egyptians, who attributed the disorder to a wandering uterus.[2] In the 19th century, Briquet, Charcot, and others identified somatoform disorders as central nervous system (CNS) phenomena.[2] The term *conversion* was applied by Freud to signify the substitution of somatic symptoms for repressed emotions.[3] Guze and coworkers[4,5] developed diagnostic criteria for somatoform disorders that created a distinction between somatization disorder and conversion disorder and laid the groundwork for a medical approach to somatoform disorders.

Motor conversion disorders are reviewed in this article from an atheoretical perspective. Conversion presenting with motor manifestations is a diagnosis that is often missed or applied incorrectly and could benefit from analysis based on the medical model. The analysis will employ an evidence-based perspective, with an emphasis on diagnostic validity, reliability, and utility for the patient. I will attempt to deconstruct the dichotomy of psychogenic versus organic motion disorders, out of which motor conversion becomes a diagnosis of exclusion, and I will review recent studies that seek neurophysiologic explanations for syndromes long considered to be idiopathic.

## METHOD

Literature searches were carried out in PubMed using the keywords *conversion disorder*, *motor conversion*, *dystonia*, *psychogenic*, *hysteria*, *somatization*, *motion disorder*, *movement disorder*, and *pathophysiology*. Articles and book chapters in the author's personal collection were also utilized.

## EPIDEMIOLOGY

Epidemiologic figures for motor-associated conversion disorders are difficult to pinpoint with accuracy. One reason is that the literature contains few studies involving large numbers of motor conversion patients. This may be



  

because motor conversion disorder patients constitute a small fraction of neurologic and psychiatric patients. In addition, discrepancies in diagnostic practices among investigators and over time have hindered the construction of a consistent epidemiologic portrait, and the selection of patients may not be representative of the general population.[6] A search of the literature identified 1 contemporary citation comparing motor conversion patients with a control group of patients with similar symptoms but a definite organic lesion.[7] A similar but older study compared conversion disorder patients to age- and sex-matched psychiatry patients without conversion disorder.[8] A more recent study examined the symptoms of a larger group of motor conversion patients, but there was no nonconversion control group.[9] The following epidemiologic portrait of motor conversion is therefore constructed from a limited number of sources.

Epidemiologic studies have fallen into 2 categories: those following the general population and those examining hospital inpatients. The total incidence of conversion disorder has been estimated to be from 2.5 to 500 per 100,000 in the general population,[6,11] with most studies estimating from 5 to 10 per 100,000.[6,10,11] Prevalence is estimated at approximately 40 per 100,000.[6] Among hospital inpatients, the incidence is 20 to 120 per 100,000[8]; from 1% to 14% of neurology and psychiatry patients experience conversion disorder.[6,8,10]

Motor conversion is estimated to occur at the rate of approximately 5 per 100,000[7] and thus manifests itself in a large proportion of all conversion cases. Women are affected more often than men, accounting for 60% to 80% of motor conversion cases.[6,7,9] The mean age of patients is 39 years.[7,9,10] Compared with the control group, patients with conversion are less likely to be high school or university graduates.[7] Conversion patients are significantly more likely to have an Axis I comorbidity, occurring in approximately one third of patients, with major depression being most common.[7,9] Axis II comorbidities are more prevalent in motor conversion patients, occurring in approximately one half of patients.[7] Mild traumatic brain injury may also predispose patients to conversion disorder.[12] Anecdotal information provides other factors associated with motor conversion, including multiple somatizations, employment in a health profession or health insurance claims, and continuing care by a devoted spouse (i.e., secondary gain).[13,14]

A more vexing epidemiologic issue is the percentage of total motor disorders that are a result of conversion. From 1% to 9% of neurology patients are conversion patients, but this may not be applicable to the entire set of patients with motion disorders.[15–18] Furthermore, lack of standardized valid diagnostic criteria for motor conversion makes it difficult to accurately specify the percentage of motion disorder patients whose symptoms are the result of conversion. However, the absence of standardized, reliable diagnostic criteria does not merely create epidemiologic ambiguities, but it also generates a serious problem for the clinician diagnosing motor conversion. Therefore, this article will pay detailed attention to the differential diagnosis of motor conversion disorders.

## PATHOPHYSIOLOGY

The pathophysiology of somatoform disorders has remained a mystery for approximately 4 millennia. Recent research has begun to unravel the pathophysiology of motor conversion disorders and has emphasized the need for approaching somatoform disorders as neurophysiologic disturbances with functional and behavioral manifestations.

Neuroimaging studies have been particularly useful in locating brain structures likely to be involved in motor conversion. Positron emission tomography (PET) of a motor conversion patient with left-sided paralysis revealed loss of activation of the right primary motor cortex. Instead, the right orbitofrontal and anterior cingulate cortex[19] were activated when the patient attempted to move the affected leg. These 2 areas therefore may have an inhibitory effect on motor cortex activation in the setting of motor conversion.[19] Interestingly, hypnotic paralysis caused an almost identical activation and deactivation pattern, suggesting that hypnotic and conversion paralysis might share common neurologic pathways.[20]

Furthermore, PET scans differentiate conversion patients from feigners. Two conversion patients with left-arm paresis had decreased blood flow to the left dorsolateral prefrontal cortex, whereas feigners showed hypofunction of the right anterior prefrontal cortex.[21] Metabolic deficits associated with conversion may not be limited to the cerebral cortex. A PET study[22] of 7 patients with unilateral sensorimotor loss showed decreased perfusion to the thalamus and basal ganglia on the side contralateral to the perceived deficit, suggesting that striatothalamocortical motor circuits also have a role in the pathogenesis of motor conversion. The same circuits are compromised in paralysis after organic neurologic damage. The perfusion asymmetries disappeared in the 4 patients scanned after recovery.[22]

An issue that remains unresolved by the above studies, which implicate different brain regions in the etiology of conversion paralysis, is whether the deficit in motor conversion is volitional or nonvolitional. The study by Vuilleumier and coworkers[22] suggests inability to generate motor programs, whereas the studies implicating cortical structures suggest interruption of normally generated motor programs.[23] Although the current studies involve a limited number of patients, they converge on a common idea: motor conversion paralysis is associated with a malfunction between the regions of the brain controlling intention and execution.[24]

Computed tomography (CT) and magnetic resonance imaging (MRI) studies of conversion patients with nonepileptic seizure activity (N = 60) demonstrate a preponderance of nondominant hemisphere lesions, compared with epileptic controls (N = 102).[25] These right-sided lesions include stroke, encephalomalacia, cortical dysplasia, severe head injury, aneurysm, arteriovenous malformation, and tumors. The findings are in agreement with the putative left-sided predominance of conversion symptoms, suggesting a link between structural abnormalities and conversion disorder.[25] However, the above lesions may directly result in hemineglect, *la belle indifference* (placid acceptance of debilitating symptoms), or other symptoms mimicking conversion disorder; in patients with such lesions, it may be difficult to distinguish whether motor symptoms are the result of conversion or the immediate result of a structural anomaly.

Hormonal influences may also be at work in the pathogenesis of conversion. Conversion patients with presenting symptoms of pseudoseizure, paraparesis, and aphonia had impaired dexamethasone suppression.[26] The apparent hypothalamic-pituitary-adrenal (HPA) impairment was thought to be a possible result of comorbid depression, although conversion patients with pseudoseizure demonstrated particularly elevated cortisol levels during pseudoseizures.[27] Unfortunately, cortisol levels in those conversion patients had poor diagnostic specificity, most likely because depression frequently accompanies conversion disorders and frequently impairs dexamethasone suppression. In addition, cortisol levels in conversion patients were not compared with levels in patients with true seizures, further limiting the power of the study. The role of the HPA axis in motor conversion, specifically, has not yet been explored, though it is likely that the molecular pathogenesis of motor conversion results from a complex interplay of metabolic, electrophysiologic, and hormonal factors.

## CLINICAL MANIFESTATIONS

Motor conversion disorders manifest themselves in a variety of ways, primarily involving major muscle groups. Weakness occurs more frequently than abnormal muscle contracture or "dystonia."[9] The presentations of muscle weakness include paralysis, paresis, and gait disturbance.[9,28] In the setting of conversion, motor paralysis can occur without atrophy despite months or years of subjective muscle weakness.[29] Dystonic presentations of motor conversion include equinovarus, spasmodic dysphonia, torticollis, torsion, tremor, and gait disturbance.[30,31] Motor conversion gait disorders do not resemble pathological gaits associated with true motor impairment.[32] Additional motor conversion symptoms such as bizarre movements, ptosis, camptocormia (static truncal flexion, often induced by sitting or standing), chorea, ataxia, globus hystericus (difficulty swallowing), and astasia-abasia (inability to stand or sit upright, but ability to move legs when lying down or sitting) have been catalogued as well.[9,32–36]

In motor conversion, the lower limbs are affected more often than the upper limbs.[9] On the other hand, several studies of the lateralization of motor conversion symptoms have not produced a conclusive agreement.[7,9,10,37–43] The majority of studies indicate that motor conversion symptoms occur in greater numbers on the left side,[7,10,37–43] although the recent detailed lateralization study of Roelofs and coworkers[9] did not indicate a left-versus-right preference.

A feature that frequently distinguishes motor conversion from nonconversion motor disorders is the presence of sensory symptoms, especially pain.[7] Thus, motor conversion may manifest as part of a mixed conversion disorder or a constellation of somatoform symptoms. However, structural lesions such as large thalamic infarcts may also result in combined motor and sensory derangements. *La belle indifference* is commonly observed, though it is not specific to motor conversion patients. More often, however, patients have a histrionic demeanor and will be reluctant to accept the results of diagnostic work-ups that fail to identify neurologic causes of their symptoms.[32,35] The average duration of symptoms is approximately 4 years.[9,44] Sudden onset, spontaneous remissions, and inconsistency of symptoms over time should create a high index of suspicion.[13] Spontaneous remission should not be regarded as a diagnostic criterion, as non–conversion motor disorders may also demonstrate appreciable rates of remission.[45–49]

## ISSUES IN DIAGNOSIS

The proper diagnosis of motor conversion disorders is complicated because the presentation frequently mimics other neurologic disorders, there are no gold-standard diagnostic tests, and there is no universally accepted set of positive diagnostic criteria. To some extent, those problems result from the subjective, symptomatic nature of the illness. Accurate diagnosis would be assisted by a medical approach emphasizing diagnostic validity, reliability, and utility. The diagnostic process may be impaired when conversion is viewed as a "psychogenic" phenomenon and is thus a diagnosis of exclusion once "organic" processes have been ruled out. Diagnosis-of-exclusion strategies are fraught with problems. Exclusionary strategies are likely to have poor diagnostic sensitivity. Furthermore, if well-understood pathophysiologic processes are excluded, it would be more logical to use the term *idiopathic* in place of *psychogenic*. Finally, a diagnosis of exclusion may lump different disease processes into the same category, one whose diagnostic utility is limited to being "not something else."

**Table 1. DSM-IV Diagnostic Criteria for Conversion Disorder[a]**

| | |
|---|---|
| A | One or more symptoms or deficits affecting voluntary motor or sensory function that suggest a neurological or other general medical condition. |
| B | Psychological factors are judged to be associated with the symptom or deficit because the initiation or exacerbation of the symptom or deficit is preceded by conflicts or other stressors. |
| C | The symptom or deficit is not intentionally produced or feigned (as in Factitious Disorder or Malingering). |
| D | The symptom or deficit cannot, after appropriate investigation, be fully explained by a general medical condition, or by the direct effects of a substance, or as a culturally sanctioned behavior or experience. |
| E | The symptom or deficit causes clinically significant distress or impairment in social, occupational, or other important areas of functioning or warrants medical evaluation. |
| F | The symptom or deficit is not limited to pain or sexual dysfunction, does not occur exclusively during the course of Somatization Disorder, and is not better accounted for by another mental disorder. |

[a]Reprinted with permission from American Psychiatric Association.[1]

**Table 2. Differential Diagnosis of Motor Conversion Disorder**

| Disease/Syndrome | Reference |
|---|---|
| Amyotrophic lateral sclerosis | Moene et al[44] |
| Creutzfeldt-Jakob disease | Solvasen et al[53] |
| Dyskinesia | Moene et al[44] |
| Dystonia | Teasell and Shapiro[54] |
| Factitious disorder | Heruti et al,[28] Teasell and Shapiro[55] |
| Guillain-Barré syndrome | Parobek[56] |
| Huntington's disease | Teasell and Shapiro[54] |
| Intracerebral hemorrhage | Glick et al[57] |
| Malignancy | Moene et al,[44] Teasell and Shapiro,[54] Glick et al[57] |
| Malingering | Heruti et al[28] |
| Multiple sclerosis | Parobek[56] |
| Multiple system atrophy | Moene et al[44] |
| Myasthenia gravis | Parobek[56] |
| Parkinson's disease | Moene et al[44] |
| Pisa syndrome | Fichtner et al[58] |
| Postencephalitis syndrome | Parobek[56] |
| Radicular syndrome | Moene et al[44] |
| Somatization disorder | Gatfield and Guze[4] |
| Spinal cord compression | Glick et al[57] |
| Systemic lupus erythematosus | Parobek[56] |
| Tourette's disorder | Kulisevsky et al[59] |
| Transverse myelitis | Teasell and Shapiro[54] |

Fahn and Williams[13,50] described an illness they called "psychogenic dystonia," involving 4 degrees of psychogenic likelihood: documented, clinically established, probable, and possible. They proposed criteria including relief of symptoms by psychotherapy or placebo, absence of signs upon surreptitious observation, inconsistency over time, incongruence with a classical movement disorder, presence of another psychiatric disturbance, multiple somatizations, and deliberate slowness of movement. The important question is whether the illness defined by these criteria has diagnostic validity. There is little empiric information on which to judge this question. However, judging diagnostic validity by the scheme of Robins and Guze,[51] the following observations can be made: there is poor delimitation from other disorders, especially malingering and somatization disorder; the criteria rely on the presence of additional, potentially unrelated, psychiatric diagnoses; the criteria are problematic largely because of their highly subjective nature.

While valid diagnoses should help predict responses to treatment, the criteria for psychogenic dystonia are predicated on response to treatment or placebo; thus, etiology becomes erroneously equated with outcome. Diagnostic reliability[52] is also compromised due to the reliance on patient-reported symptoms in lieu of diagnostic signs and tests, though this is a problem for the diagnosis of somatoform disorders in general. Furthermore, the criteria are fundamentally subjective rather than objective, focusing on the "inconsisten[cy]" or "incongruen[ce]" of symptoms.

Finally, "psychogenic dystonia" is a diagnosis of limited utility. According to Fahn,[13] the diagnosis leads to "disbelief and distrust," requires inpatient treatment, can only be made by a neurologist (not a psychiatrist), and could harm the patient and his or her family. Therefore, the diagnosis is primarily subjective in nature and does not benefit the patient. Fahn also advises the physician to inform the patient that "[p]ent-up emotions need to be expressed, and they do so by producing these abnormal movements."[13(p370)] The advice implies a Freudian bias in the approach to motor conversion.

The DSM-IV criteria for conversion disorder (Table 1) may be better oriented toward diagnostic validity, reliability, and utility.[1] Criteria A and E help define the disorder and establish diagnostic utility. Criteria C and F provide diagnostic validity and reliability by explicitly delimiting conversion from other disorders such as malingering or somatization disorder. Criterion D relies on a negative definition, but that criterion differentiates conversion from disorders arising outside the CNS. Criterion B, however, is particularly problematic: the invocation of "psychological factors" and "conflicts or other stressors" has psychoanalytic overtones; while this criterion may apply to most conversion disorder patients, it is insufficiently specific to delineate conversion from other psychiatric disorders such as malingering or somatization disorder. Thus, to a limited extent, the DSM-IV definition of motor conversion disorder suffers from lack of clarity and definition by exclusion.

A serious problem in the diagnosis of motor conversion is the significant chance that diagnosis will change with follow-up. An extensive variety of medical and psychiatric derangements constitutes the differential diagnosis of motor conversion (Table 2); that alone argues for the diagnosis and treatment of motor conversion according to the same principles used for other medical and neurologic processes. The false positive rate for motor conversion, as determined from studies as early as 1990, has

  

varied from 5% to 15%, given a follow-up time ranging from 1 to 10 years.[28,44,60–63] Moene and coworkers[44] found the general neurologic exam to be particularly useful, as it indicated 4 of the 10 false positives in their study. MRI and CT imaging also play an essential role in the elimination of false positives where the etiology is structural in nature.[57]

Positive diagnostic criteria, rather than definition by exclusion, may be used to discriminate cases of conversion from false positives. In one study, prior suspicion of a neurologic disorder, younger age at onset of symptoms, and shorter duration of symptoms contributed to distinguishing false positives from conversion cases[45]; the main drawback of the study was the small sample size (N = 10) of false positives.

Schuepbach and coworkers[64] used the following positive criteria to obtain a low rate of false positive diagnoses of conversion: symptom localization that is fuzzy or not coincidental with anatomy and physiology, symptom intensity constant and independent of voluntary movements, reaction to medication that is pharmacologically irrational, and description of symptoms characterized by affective expressions, evaluative expressions, or attempted use of medical jargon.

While those recommendations may be helpful, the study would have benefited from the testing of the above criteria on nonconversion motor patients. It also must be acknowledged that positive diagnostic symptoms such as those listed above are not pathognomonic for conversion and may occur in the setting of structural CNS disease.[65] Regarding positive criteria, the presence of so-called "classic" features such as *la belle indifference* and secondary gain should not be used as diagnostic criteria as they are highly subjective and are thus difficult to rate, unreliable, and nonspecific.[66]

Recent investigation[67–69] points toward diagnostic signs that may be useful in cases of motor conversion. Healthy individuals demonstrate a speed-for-accuracy trade-off in both real and imagined movements, which is described by Fitts' Law (the movement time to a target is a mathematical function of the distance to and the size of the target).[67] This trade-off may not be present in cases of motor conversion, especially for imagined movements, whereas individuals with structural motor deficits still conform to Fitts' Law.[68] Thus, subjects cannot anticipate the effects of an actual limb injury. Assessment of whether a patient's movements conform to Fitts' Law can be determined quantitatively, which could translate into a diagnostic tool with an objective basis. The original study involved only 1 patient with conversion disorder, so further testing with a statistically informative sample is necessary before such measurements develop diagnostic power. Careful observation may also reveal physical signs of conversion. Karnik and Hussain[69] reported a "sign" that may be useful in cases of motor conversion manifesting as paraplegia: the wheelchair-bound patient derives assistance from the lower extremities for propulsion, rather than relying exclusively on the upper extremities.

I recommend the following diagnostic proposals for motor conversion disorder: retention of DSM-IV criteria A, C, D, E, and F. Criterion B does not have an analogue in the criteria for somatization disorder and is not crucial to the diagnosis of conversion. Positive, objective criteria such as symptom location inconsistent with anatomy or physiology and symptom intensity constant and independent of voluntary movements should be considered as replacements for criterion B. Careful observation with a complete neurologic exam should always be part of the workup for conversion. And in the future, quantitative studies such as kinetic imaging tests may be developed that differentiate conversion patients from those with structural motor deficits.

## TREATMENT

Since current diagnostic standards are based almost exclusively on symptomatology, and etiology is poorly understood, treatments are aimed at regaining function. Treatments should be based on empiric study of methods that have success, rather than on psychological theories of etiology that cannot be tested. Selection of the most effective therapeutic techniques using an evidence-based perspective, although the preferable approach, is nevertheless limited by the fact that published reports of effective treatment are in the format of case studies and lack control group data. It has been proposed that the field would benefit from a large, randomized, controlled trial comparing a multidisciplinary approach involving behavioral, cognitive, and rehabilitative methods to management by current services.[70]

The majority of studies detailing approaches for treating motor conversion focus on gait abnormalities and share 3 main recommendations. First, health care providers should withdraw medical and social attention from abnormal movements or gait behavior.[28,71] This does not imply punishing abnormal movement behavior but rather accepting the patient's symptoms while encouraging physical rehabilitation.[72] The second objective involves physical and occupational therapy to retrain the patient in normal gait and movement behaviors. One study details the successful adaptation of a 4- to 6-week amputee ambulation training program, in which patients remain in a wheelchair when not in treatment sessions, thus preventing recurrence of improper gait patterns.[71] Physical therapy may also take the form of stretching exercises, as in the case of camptocormia.[33] Physical approaches that work toward achievable goals and encourage physical exercise have shown promise.[72] The third line of treatment is psychotherapeutic, targeted toward helping the patient develop appropriate methods of coping with stress[71] and

supervising reintegration into the patient's school or work.[72] Heruti and coworkers[28] also emphasize the importance of long-term follow-up. Another factor in the success of behavioral management in treatment of motor conversion is the correlation between the duration of symptoms and the duration of treatment required to eradicate them; behavioral treatment may not provide statistically long-lasting resolution of symptoms.[73]

Hypnotic and narcotherapeutic approaches have been employed as adjuncts to medical treatment. Indigenous psychotherapy, in the form of the *Main Puteri*, a shamanistic healing and exorcism ceremony, was successfully used to treat a Malay woman suffering from lower limb paraparesis.[74] Hypnosis has been employed in the treatment of conversion, but a recent randomized, controlled clinical trial[75] demonstrated no additional effect of hypnosis compared with psychotherapy alone. A more recent randomized trial[76] showed that hypnosis produced a treatment response; the control group consisted of untreated patients. Thus, hypnosis has not been proven to be superior to medical therapy or other modes of psychotherapy. Pharmacologic therapy with barbiturates such as thiopentone[77] and amobarbital[78,79] has been employed as an aid to hypnosis or psychotherapy, but the clinical evidence consists of chart reviews and case reports rather than randomized studies. Because of their more favorable adverse effect profiles, short-acting benzodiazepines have recently come into favor over barbiturates for use with hypnosis.[36] Antidepressants may also be useful adjunctive agents to alleviate comorbid mood and anxiety disorders.[80]

Electrical stimulation therapies have been utilized to a limited extent for motor conversion disorders. Electroconvulsive therapy (ECT) has been used sporadically, and clinical trial evidence is lacking. Furthermore, there are few contemporaneous reports of ECT for conversion disorder. The most recent report of successful ECT for motor conversion, a man with right-hand paralysis and disuse atrophy, dates back to 1988. One of the issues complicating ECT for conversion is that the treatments may actually be remedying underlying depression[81]; on the other hand, ECT may also hyperstimulate the motor cortex, thereby achieving restored function. High-frequency repetitive transcranial magnetic stimulation was recently reported to induce remission of conversion paralysis, although the study was a case report involving 1 patient, and there was no long-term follow-up for relapse.[82]

Placebo treatments have been utilized in the diagnosis of "psychogenic dystonia."[50] For example, patients' anticholinergic medications were switched to placebo without their knowledge to evaluate the etiology of symptoms. Placebo treatments by themselves were not successful as curative measures, as patients experienced relapses, presumably because the underlying psychiatric issues behind the dystonia remained unaddressed. The appropriateness of such treatments can only be weighed after it is determined whether placebos actually contribute usefully to diagnosis or treatment.

## PROGNOSIS

The prognoses of patients with motor conversion vary widely. Recovery may be complete or incomplete and may occur within days to months. However, longer recovery times are associated with greater residual functional deficits.[16] Factors favorably affecting prognosis include sudden onset, presence of psychological stressors prior to onset, short duration between diagnosis and treatment, high level of intelligence, absence of other psychiatric disorders, and aphonia as the presenting symptom. Factors adversely affecting prognosis include severe disability with long duration, age above 40 years, and convulsions and paralysis as presenting symptoms.[16,32,60]

## CONCLUSIONS

Advances in neuropsychiatric research are quickly eroding the black-and-white distinction between "organic" and "psychogenic" motor disorders. Pathophysiologic studies like those reviewed herein have begun to provide feasible mechanistic explanations for conversion symptoms. Processes that have been traditionally categorized as "psychogenic" are being explained by "organic" phenomena, and as future research identifies molecular causes of motor conversion disorders, those processes may become amenable to pharmacologic interventions. Thus, the paradigm for the diagnosis and treatment of conversion will eventually come full circle, becoming medical as it was during the time of the ancient Egyptians, with the notable exception that our grasp of the pathophysiology should be more accurate.

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