The neuropsychiatry of conversion disorder

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Purpose of review

During the past two decades there has been a relative surge of interest in conversion disorder, and a multitude of studies have emerged on the subject. With continuing developments in neuroscience – mainly brain imaging – new applications to conversion disorder are being reported every year.

Recent findings

Diagnosis continues to represent a challenge, although neurological signs are increasingly being developed and validated to assist with this. Psychiatric co-morbidity diagnosed according to operational criteria is common. Brain imaging studies have brought some clues to understanding the pathophysiology of conversion disorder.

Summary

Evidence-based medicine requires reliable diagnostic criteria, and attempts have recently been made to validate some of the well known neurological signs of conversion disorder. From a psychiatric point of view, there is a need for greater understanding of the aetiology and mechanisms underlying conversion disorder and its relationship to other psychiatric disorders. Although advances have been made both in diagnostic methods and in the groundwork for a neurobiological model, no clear rationale for treatment is yet available and further research is strongly needed.

Keywords

clinical presentation, conversion disorder, hysteria, neurobiological correlates, psychogenic

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Introduction

Since its peak at the turn of the 20th century, there has been a steady decline in the medical community's interest in conversion disorder to a point that the disease itself was thought to have waned [1]. In the past decade, however, such interest has undergone a revival. It has been established that conversion disorder remains common, and disabling [2], while advances in neuroscience have given hope for new insight into its biological mechanisms. There have been several studies aimed at refining the diagnosis and understanding the pathophysiology, which we shall review here.

A problematic diagnosis

Conversion disorder is a psychiatric diagnosis but rarely presents to psychiatrists. The presenting complaint is typically of 'one or more symptoms or deficits affecting voluntary motor or sensory function' [criterion A in the Diagnostic and Statistical Manual of Mental Disorder (DSM)-IV] and therefore usually presents to a neurologist, where it represents a diagnostic challenge. The full diagnosis, however, requires an 'associated psychological factor' (criterion B) and most neurologists do not feel comfortable making such an association, since they usually do not explore the psychological aspects in sufficient depth in their evaluation. Their preliminary diagnosis of 'medically unexplained symptoms' may lead patients either to view their doctor to be incompetent or to view their disease as a rare condition that needs further investigation.

The full diagnosis usually requires two specialists, both a psychiatrist and a neurologist, and therefore needs good collaboration between them in the multiple-step process: careful history taking and physical examination by the neurologist, referral to the psychiatrist, and finally, optimally, a clear joint explanation to the patient. This collaboration is sometimes jeopardized by the fear of misdiagnosis; the psychiatrist has to trust the neurologist that organic disorders explaining the symptoms have been entirely excluded. It was previously understood that conversion disorder often represented missed organic conditions and that, at follow up, such a disorder would be found in up to one-third of patients [3]. A thorough meta-analysis [4], however, recently established that misdiagnosis rates are around 4% in conversion disorder – no more

frequent than in diseases such as schizophrenia or motor neurone disease.

Another difficulty in establishing a diagnosis of conversion disorder lies in the lack of consensus concerning the psychological mechanism of criterion B. This is illustrated by the different classification categories in DSM-IV and International Classification of Disease-10. In the former, conversion disorder is grouped with the somatoform disorders and has been classified as such mainly because of the presence of a symptom mimicking a medical disorder, but also because the mechanism thought to be involved is a 'symbolic resolution of an unconscious psychological conflict', 'converting' the stressor into a physical symptom. In the latter, conversion disorder is included with the dissociative disorders, implying that the production of the symptoms is the result of 'a complete or partial loss of the normal integration between memories of the past, awareness of identity and immediate sensations and control of bodily movement'. It also implies that a satisfactory psychological explanation can be given for the symptom, when finding an associated psychological factor, even if denied by the individual. The debate concerning which definition is more adequate is still active [5•], and will probably go on until 2011, when the new DSM-V and International Classification of Disease-11 classifications are due.

The neurological diagnosis

The neurological presentation of conversion disorder has been described for centuries but, with the development of evidence-based medicine and more sophisticated investigations, there have been attempts to establish the neurological part of the diagnosis on more than a clinical picture and the overall impression of the physician. Some attempts to validate clinical signs are described below.

For motor symptoms, the diagnosis relies on signs such as inconsistency (e.g. the patient displays a leg monoplegia when tested on the bed examination, but is then able to stand on one foot to put his trousers back on) and give-way weakness. Although widely known and used by the experienced neurologist, these signs may not reliably exclude neurological disease. In a study on 30 patients admitted for organic neurological disease [6], seven alleged features of conversion disorder have been tested, including give-way weakness, which was found in 30% of cases. The Hoover's sign (involuntary extension of a pseudo-paralysed leg when the 'good leg' is flexing against resistance) is a good test of motor conversion disorder, which was not examined in the Gould study. A quantitative evaluation of the Hoover's sign with computerized myometry has shown a significant difference between organic paralysis and pseudo-paralysis [7], although its reliability has not been validated in its qualitative form, as used in the clinic setting.

The diagnosis of psychogenic movement disorders relies mostly on the observation by an experienced movement disorder specialist, and Fahn and Williams [8] developed clinical diagnostic criteria for psychogenic dystonia that were then extended to all psychogenic movement disorders. Those criteria have been recently modified and their validity established [9]. Despite some methodological weaknesses remaining (such as the retrospective design), their preliminary results showed good specificity (96%) and sensitivity (97%). Psychogenic tremor has recently been the subject of a separate validation [10]. Its diagnosis rests on the presence of variability in frequency and amplitude, distractibility and entrainment. The clinical relevance of those signs was assessed by means of blinded ratings of video recording of 12 patients with psychogenic tremor and 33 patients with essential tremor. The finding of distractibility (by contralateral alternate finger tapping) brought a specificity of 72.7% and a specificity of 73.3%. Surprisingly, entrainment did not help to differentiate psychogenic tremor from essential tremor, and neither did suggestibility (by hyperventilation and/or tuning fork).

There are no good validated clinical signs to distinguish sensory loss of psychogenic or organic origin and the clinician usually relies on discrepancies (e.g. a patient with complete leg anaesthesia and absent proprioception should not demonstrate a normal Romberg sign, and one would expect some cutaneous lesions, like in a patient with diabetes) or nonorganic distribution (a hemi-leg territory, for example, not corresponding to any radicular, truncal or central distribution).

'La belle indifference' was a term used by Freud to refer to the apparent indifference and cheerfulness he observed in one of his patients with hysteria, and has since been accepted as a clinical sign suggesting conversion disorder. A recent systematic study [11•] examined the validity of 'la belle indifference', reviewing 11 studies published between 1965 and 2006. The median frequency of 'la belle indifference' in 356 pooled patients with conversion disorder was 21% (range 0–5%) and in 157 patients with organic disease was 29% (range 0–60%). Among the only four controlled studies available, one demonstrated a significant association with hysteria, whereas the three others found no difference between groups. Before drawing a conclusion that this sign is not useful in conversion disorder, it must be outlined that important methodological issues were noted with no blinded study design and only two gave a description of what they meant by 'la belle indifference'. This sign currently has no clear definition and should not be used to

differentiate organic from functional symptoms without further study.

Psychogenic gait disorders can be recognized by specific patterns such as 'walking on ice' or noneconomic postures (eccentric placement of the centre of gravity, which puts extra demands on balance and strength systems, like walking with knees flexed), but no definite signs have been formally tested. A recent article [12] mentions a potential new sign that would benefit from further assessment: the 'chair sign', where patients with psychogenic gait disorder could propel a chair when sitting on it better than control individuals with organic gait disorders.

The finding of closed eyes during a seizure brings a positive predictive value of 0.94 (sensitivity 96% and specificity 98%) for psychogenic nonepileptic seizures (PNESs) [13••]. Other clinical signs, although not validated, are considered strong indicators of PNESs: pelvic thrusting, opisthotonic arching, side-to-side head shaking, asynchronous movements, stuttering, weeping, gradual onset and termination. When PNESs are suspected, the use of 24-h video electroencephalogram monitoring [14] is the most accurate test, with an up to 73% success rate in obtaining a diagnosis [15]. If the pretest suspicion is high, based on the presence of the aforementioned signs, a short-term video electroencephalogram with induction (either saline injection, hyperventilation or photic drive) can also have a high yield (66%) [16]. PNES is one of the few conversion disorder syndromes for which the diagnosis is aided by a simple blood test: serum prolactin measurement 10–20 min after the event. The American Academy of Neurology [17] has revealed level B evidence for this test to differentiate PNES from generalized or complex seizures (60% and 46% sensitivity, respectively, and 96% specificity for both). The test, however, does not differentiate PNES from syncopes, as all organic 'loss of consciousness' might produce prolactin. The presence of stertorous breathing has recently been demonstrated [18,19•] as very useful in distinguishing seizures from PNES, when reported by the medical staff.

While reporting statistically validated signs, we cannot resist mentioning the Teddy bear sign: the presence of a stuffed animal brought by the patient to the electroencephalogram-monitoring unit was studied in 903 patients [20], and yielded a positive predictive value for PNES of 87%, with a high specificity (93%) but low sensitivity (5.2%).

Even though such objective signs can help the clinician in reaching a diagnosis, just as all other medical conditions, it is the general clinical picture, the experience of the clinician and an acceptance that they will make mistakes from time to time that lead to the lowest rate of misdiagnosis possible.

The psychiatric diagnosis

From a psychiatric point of view, it has been shown that patients with conversion disorder often have associated mental illnesses, mostly depression and anxiety disorders. Fink *et al.* [21] reported 23% of patients with conversion disorder had associated phobia, anxiety or panic attacks, and Sar *et al.* [22] reported 50% anxiety, 42% phobia, 34% obsessive–compulsive disorder, 10% panic attacks, 71% affective disorder and 34% depression among patients with conversion disorder. In patients with psychogenic nonepileptic seizures [23,24], researchers found 47–57% had associated depression, 39–47% anxiety, 35–49% posttraumatic disorder, 62% personality disorder, and no psychotic disorders. Hence multiple psychiatric co-morbidities are common.

Since the Freudian model emphasized trauma and sexual abuse, several studies have examined the association of history of abuse with conversion disorder, and found between 28% and 44% of patients reported a history of physical abuse and 24–26% reported sexual abuse [22,25]. In a controlled study of 30 patients with motor conversion disorder [26], however, only 3% of patients reported childhood abuse, which was not significantly different from control individuals. It may be that abuse is more frequent in PNES than in other clinical presentations of conversion disorder [27], with up to 67% of those patients reporting past physical or sexual abuse [24].

Traumatic life events were associated with conversion disorder even before Freud but a precise quantification of such an association is lacking, mostly because the study of life events and trauma is complicated by the unreliability of self-report. In the conversion model, a patient with conversion disorder represses the traumatic event, and therefore would not consider the event relevant when asked to report it, and may not even 'remember' it. A controlled study of 50 patients [28] did not find an increased number of stressful life events in patients with conversion disorder compared with patients with affective disorder but did demonstrate a correlation between the reported unpleasantness of events and the severity of the symptom. The methodology of the study was not ideal, with the events in question being based on a self-report questionnaire. Another controlled study on 30 consecutive patients [29] did show a significantly higher number of life events in the preceding year of the symptom onset in the conversion disorder group. In this study, the bias of the self-report might have been minimized by the design of a semi-structured interview aimed at exploring five domains of life events. It has also been argued that life events should be independent of the illness. For example, significant harm to her child would be considered a stressful life event to a mother and might be thought responsible for her symptoms; however, the fact that the mother

already had some mild symptoms at the time might lead to her neglecting the child, and thus the life event may be a consequence of the disease and not an independent cause. The Life Event and Difficulties Schedule [30] puts great emphasis on independence in order to avoid the problems of reverse causality, bias and confounding, and would be an important advance in exploring the stressors for patients with conversion disorder [31].

Neurobiological correlates

Conversion disorder presents with neurological symptoms and signs that are unexplained by neurological disease. How then are the symptoms and signs produced, if they are not feigned? In the 1960s, electrophysiological studies suggested a central corticofugal inhibition of afferent stimuli as responsible for hysterical sensory loss, since evoked potentials showed abnormalities that disappeared when tested again after resolution of the symptoms' [32], with similar results replicated in two patients in 2004 [33]. In the 1990s, further evidence of central inhibition came from a single-photon emission computed tomography [34] study in a woman with sensorimotor hemisyndrome, which demonstrated hypoperfusion in the contralateral parietal region and increased perfusion in the frontal region. This pattern was supported by a positron emission tomography study of a patient with hemiparesis [35], where again increased activity was found in the frontal region with activation of the anterior cingulate and orbitofrontal cortex. The authors hypothesized that frontal regions were inhibiting the motor and premotor areas when the patient tried to move the affected limb. In 2001, Vuilleumier and colleagues [36] performed a larger sample single-photon emission computed tomography study on seven patients with sensorimotor conversion disorder, when they were symptomatic and when a subset of them recovered. Reduced blood flow in the subcortical structures (contralateral thalamus, putamen and caudate) was found when a vibration stimulus (which normally activates both sensory and motor cortices) was applied to the affected limb. The pattern normalized in those patients who subsequently recovered. This study shed further light on central inhibitor mechanisms, involving not only cortical areas but also corticosubcortical circuits.

The central inhibition hypothesis does not address the question as to whether this inhibition is 'voluntary' or 'involuntary'. A few studies have tried to distinguish the two, by comparing conversion disorder patients with feigners. Spence and colleagues [37] demonstrated a hypoactivation of the left dorsolateral prefrontal cortex during a motor task in three patients with conversion disorder irrespective of the lateralization of the symptoms (two patients with left-sided paresis, one with right-sided paresis), which was found neither in feigners nor in control individuals, implicating the left dorsolateral prefrontal cortex in altered volition in conversion disorder. Another study [38] compared individuals hypnotized to have a subjectively real paralysis with individuals feigning paralysis. The former demonstrated a contralateral activation of the orbitofrontal region and cerebellum and an ipsilateral activation of the thalamus and putamen. More recently, a functional magnetic resonance imaging study [39] in four patients with motor symptoms, compared with four control individuals feigning a motor deficit, implicated a network including the putamen and the lingual gyri bilaterally, the left inferior frontal gyrus, left insula and right middle frontal and orbitofrontal cortices underlying conversion disorder.

Overall, these imaging studies suggest that complex brain mechanisms are involved in preventing normal cortical activity; however, no consistent model has yet emerged. In part, this is because of the small samples included and the heterogeneity of the symptoms examined.

Apart from functional imaging, studies of patients with cerebral lesions have implicated brain dysfunction in the symptoms of conversion disorder. For example, Eames [40] reported that a large number of patients (32%) with hypoxia (and thus basal ganglia lesions) displayed conversion disorder symptoms. Drake [41] reported five cases of hysteria in patients with left cerebral hemisphere lesions.

A structural neuroimaging study [42] suggested there may be macroscopic abnormalities in the brains of patients with conversion disorder after all, at least at the group level. The study found a significant reduction in the volume of the basal ganglia in 10 patients with conversion disorder, compared with control individuals, although no correlations with laterality or clinical variables were presented.

Freudian model

Even though, as discussed above, no definite pathophysiological model can explain conversion disorder, there is good evidence that neural circuits are involved in this pathology. Should we then conclude that hysteria is a neurological disease after all? Hence, is the 'associated psychological factor' a coincidence or even a consequence of that particular brain disease? What about the Freudian model of emotion 'converted' into somatic symptoms?

A recent study [43•] demonstrated that negative emotion (recall of a traumatic event) was linked to conversion disorder symptoms. When examined in a functional magnetic resonance imaging scanner, a woman with a right hemiparesis showed increased activity in her right medial temporal lobe (including the amygdala, involved in emotional recall) and concurrent deactivation of her

contralateral (left) primary motor cortex when cued to remember a key traumatic event linked to the onset of her symptoms. Further studies on larger samples might help us understand the link between psychological stressors and the development of conversion disorder using a cognitive model of memory suppression.

Therapeutic approach

To build a rationale for treatment, a theoretical aetiology is required. Unfortunately, even though there are promising clues, as described above, no model, and no treatment, has been clearly established. A recent Cochrane review [44] looked at the available treatments for PNES and found only three relevant studies, two on hypnosis and one on paradoxical therapy, which were insufficient to draw any firm conclusion. There also have been claims made for repetitive transcranial magnetic stimulation [45], physiotherapy [46], antidepressants [47], cognitive behavioural therapy [48], paradoxical injunction [49] (favouring situations, during therapy sessions, in which patients might aggravate their symptoms, such as thinking of a traumatic event) and a strategic behavioural approach [50] (patients are assured that full recovery after an inpatient physiotherapy programme would be the proof of an organic disease whereas no improvement would imply a psychiatric cause for the symptom). A recent review [51•] of 34 available randomized control trials of treatments in somatoform disorders (with only three specific to conversion disorder) showed some evidence for cognitive behavioural therapy with no evidence of a positive effect of antidepressant medication.

Conclusion

Over the past 10 years there has been a resurgence in research into conversion disorder. This research, mostly utilizing brain imaging, has given some clues to the complex mechanisms involved. Understanding the pathophysiology of this disorder, however, remains a challenge, with the broad range of symptoms making large-scale studies difficult. The lack of a clear conceptual model hampers the development of therapies. Recent evidence distinguishing feigning from conversion disorder, however, and pointing to a cerebral inhibiting process, may help patients and their carers accept that they suffer from a 'real disease', and remove some of the stigma of 'hysteria'. In the future, further development of neuropsychological models that reconcile the 'psychosocial' and the 'biological' may lead to more specific and effective therapy.

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Additional references related to this topic can also be found in the Current World Literature section in this issue (p. 314).

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