Progress in understanding conversion disorder

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Abstract: Conversion disorder has a history that may reach back into antiquity, and it continues to present a clinical challenge to both psychiatrists and neurologists. This article reviews the current state of knowledge surrounding the prevalence, etiology, and neurobiology of conversion disorder. There have been improvements in the accuracy of diagnosis that are possibly related to improved technologies such as neuroimaging. Once the diagnosis is made, it is important to develop a therapeutic alliance between the patient and the medical team, and where comorbid psychiatric diagnoses have been made, these need to be adequately treated. While there have been no formal trials of medication or psychoanalytic treatments in this disorder, case reports suggest that a combination of antidepressants, psychotherapy, and a multidisciplinary approach to rehabilitation may be beneficial.

Keywords: conversion disorder, hysteria, diagnosis

Introduction

"Does the body rule the mind, or does the mind rule the body? I don't know"

THE SMITHS

The relationship between mental and physical symptoms is a core tenet of psychiatry. Egyptian physicians described cases of women suffering from unspecific symptoms: one bed-bound; another who could not open her mouth; and a third who was "ill in seeing". They ascribed such symptoms to "starvation of the uterus". These cases were described by Veith (1965), although her interpretation of them as hysterical has been challenged (Gilman et al 1993; Ng 1999). According to Veith, the Greek physician Hippocrates took up this concept and coined the term "hysteria". He described an illness in which the uterus (in Greek: hystera) dries up and wanders the body in search of moisture. Symptoms would then be caused by the uterus pressing on other organs. If it had wandered as far as the cranium, for example, the symptom would be headaches.

In modern classification systems (DSM-IV and ICD-10) the term "conversion disorder" replaced "hysteria" some time ago. The core feature of conversion disorder is a deficit or distortion in neurological functioning, or symptoms suggesting a general medical condition that is not referable to an organic lesion. In both DSM-IV and ICD-10 classifications, non-organic neurological symptoms must occur in isolation for a diagnosis of conversion disorder to be made. Multiple symptoms are suggestive of somatization disorder. DSM-IV lists four subtypes of conversion disorder: motor, sensory, seizures, and mixed. Classically, presentations include motor and sensory deficits (such as hemiparesis, paraparesis, and hemisensory loss), blindness, swallowing difficulties (globus hystericus), and nonepileptic seizures. Presentations can encompass any nervous system activity that is to some degree under voluntary control and include psychogenic dementia.

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A brief history of hysteria

Prior to 1600, the hysteric affliction was linked with an illness of the uterus (Wenegrat 2001) or given metaphysical explanations such as witchcraft or demonic possession.

Indeed at witch trials, sensory lesions, which did not follow known anatomical distributions, were described as "devil's patches" and were held to be a sign that the accused had been consorting with the devil (Maldonado and Spiegel 2001).

By the early 17th century, a more "psychologically minded" explanation of hysteria was introduced. Hysteria was classified as a variety of melancholy by Robert Burton in his *Anatomy of Melancholy* (Burton 1927). Thomas Sydenham believed that both men and women could suffer from hysteria, which he considered an affliction of the mind, and the Scottish physician Robert Whytt wrote a book entitled *Nervous, Hypochondriac and Hysteric Disorders*, which classified hysteria as a nervous disorder (Whytt 1768).

This concept was further developed by Jean Martin Charcot in the 19th century at the Salpêtrière in Paris. He described a syndrome of "hysteria major" (similar to the modern condition of nonepileptic seizures) and hypnotized patients to facilitate the presentation of their symptoms. This technique influenced Freud and Breuer's (1905) publication *Studies on Hysteria*. Breuer treated a young Viennese woman who he called Anna O who suffered from convergent squint, paralyses, paresis of her neck muscles, and contractures. The patient could enter a state of autohypnosis in which, with Breuer's help, she could provide detailed accounts of the circumstances in which each individual symptom had begun. After this, that particular symptom would abate. Anna O famously named this the "talking cure" (Freud and Breuer 1974).

The term conversion derives from this psychoanalytic tradition and reflects the emergence of physical symptoms as an attempt to resolve, or to communicate, unconscious and unbearable psychic conflicts – to "convert" them from psychic symptoms to physical ones. In clinical situations, it is often difficult to pinpoint a relevant psychological stressor, and Freud's notional association of these symptoms with the repression of sexual desire remains controversial. However, Roelofs et al (2002) assessed 54 patients with conversion disorder and a comparison group of 50 patients with affective disorder and found that those with conversion disorder reported a higher incidence of physical and sexual abuse.

The epidemiology of conversion

Conversion disorder is a relatively common presentation in neurological practice, accounting for perhaps 1%–3% of diagnoses in general hospitals (Marsden 1986) and more in specialist neurological settings (Ron 1994). Carson et al (2003) found that 30% of new attendees at neurology outpatient clinics had "medically unexplained symptoms", a category that includes, but is not synonymous with, conversion disorder. When followed up 8 months later, over half were still troubled by their symptoms and had not improved. No cases developed a neurological diagnosis.

In primary care, conversion disorder is less common. Singh and Lee (1997) surveyed primary care physicians and identified 18 patients (out of a catchment population of 37 000) with conversion symptoms. They found an association with female gender and a history of childhood sexual abuse.

The neurobiology of conversion disorder

The development of functional neuroimaging techniques has provided the methodology to study the neural basis of conversion disorders. Using positron emission tomography (PET), Marshall et al (1997) found increased cerebral blood flow in the right anterior cingulate and right orbitofrontal cortex in a patient with a hysterical left hemiparesis. Spence et al (2000) found deactivation of the left dorsolateral prefrontal cortex (DLPFC) in subjects with hysterical hemiparesis, regardless of the side of the deficit. Maruff and Velakoulis (2000) compared a patient with hysterical paralysis with normal controls in generating motor imagery (performing a movement in imagination), and they found the ability to generate motor plans was preserved in the conversion patient. It is possible therefore that these alterations in frontal function are related not to a deficit within the neural network but rather to an action of the "will" in inhibiting movements.

Using SPECT scanning, Vuilleumier et al (2001) studied 7 patients with hysterical unilateral sensorimotor impairment. They showed reduced blood flow in the thalamus and basal ganglia contralateral to the deficit which resolved after recovery of the symptoms. They suggest that conversion symptoms may involve striato-thalamo-cortical circuits, which would tie in with the known roles of such circuits in the emotional moderation of motor processes. Emotional stressors could inhibit these pathways, thus impairing motor readiness and affect the quality of voluntary movements. Interestingly, these circuits are also involved in the unilateral sensorimotor neglect, which occurs after neurological lesions, and in which the affected limb is not paralyzed, but is not under voluntary control.

Striato-thalamo-cortical loops are involved in many processes, and might also plausibly underlie non-motor conversion symptoms such as memory loss.

Unlike functional imaging findings, evoked motor and somatosensory potentials have in the past been reported as normal in conversion disorders (Meyer et al 1992). However, two cases reported recently by Yazici et al (2004) showed greatly diminished somatosensory evoked responses while symptoms were present, with a return to normal after conversion symptoms had abated. Additional studies are clearly required to elicit the neural correlates of conversion.

Diagnosis of conversion disorder

The diagnosis of conversion disorder is a clinical challenge. A careful psychiatric history and examination should be taken to screen for comorbid psychiatric illness, and if identified these should be treated appropriately. The psychiatric history is required also to elucidate the onset and nature of symptoms and the presence of stressors, although in many cases a psychological reason is elusive (Ron 2001). This may be because a patient with conversion symptoms is not often able to explain psychological factors themselves – this being the reason that they need (albeit unconsciously) to express them as bodily symptoms.

Early in a patient's presentation, physical investigations may be undertaken to exclude general medical conditions. While conversion disorder should not be considered a diagnosis of exclusion, in clinical practice the patient often undergoes multiple physical examinations and investigations. A consequence of this is that patients may be given a psychiatric diagnosis as a last resort when all else has failed. The process of serial medical investigation may not allay fears, and may exacerbate anxiety in some patients.

While it has only a limited role as a diagnostic tool, historical information such as childhood experience, personality style, illness beliefs, and "secondary gain" can influence the quality of the patient's experience (Stone et al 2002). Crimlisk et al (1998) encourage doctors to make a positive diagnosis early to avoid creating uncertainty and reduce the need for costly and unnecessary investigations. The possibility of introducing a psychiatrist early in the process of diagnosis – perhaps in the neurology clinic – might be of benefit. Demonstration of the physical signs to the patient may be therapeutically helpful, and may help to explain that under certain circumstances their nervous system is capable of functioning normally. This also promotes trust in the doctor–patient relationship (Stone et al 2002).

According to DSM-IV, conversion symptoms must be of clinical significance to the patient, or of social or occupational consequence. While for some patients the benefits of the "sick role" may result in perceived significant gain (Ron 2001), the associated handicaps are often great (Vuilleumier et al 2001). Factitious disorder (the conscious production of symptoms in pursuit of medical attention) or malingering (the conscious production of symptoms and signs for financial or material gain) are distinct from conversion disorder. Stone et al (2002) suggest that physicians have a tendency to overdiagnose feigning of symptoms: in clinical practice the distinction between conversion and factitious disorders can only be considered definitive if corroborated by covert surveillance or confession.

An apparent lack of distress in the face of unpleasant symptoms or even disability – "la belle indifference" – is classically associated with conversion symptoms. This is held to be a useful diagnostic sign, but is not a common feature, and the majority of patients with conversion symptoms are in fact distressed by them (Stone et al 2002).

A core feature of the diagnosis of conversion disorder is the absence of a neurological or organic diagnosis. Slater (1965) famously undertook a 10-year follow-up study of patients diagnosed with hysteria and reported that half of his patients developed clear neurological or psychiatric conditions, indicating a high rate of misdiagnosis. However, a more recent study (Crimlisk et al 1998) reviewed 73 consecutive patients with neurologically unexplained symptoms and found, in contrast to Slater, a low incidence of neurological conditions that might have explained their initial symptoms (3 out of 69 patients). Seventy-five percent of their sample had a psychiatric diagnosis (predominantly affective, anxiety, or somatization disorders) at presentation, and 45% were diagnosed with a personality disorder. In a follow-up study by Binzer and Kullgren (1998), none of the 30 patients with conversion disorder was subsequently reclassified as suffering from a neurological disease. Similar findings were reported by Carson et al (2003) who followed up a cohort of neurology clinic attendees whose symptoms were rated as "not at all" or only "somewhat" related to an organic disease. None of the 66 people followed up had acquired an organic diagnosis at eight months. Fourteen percent of the participants rated their condition as "worse", 40% rated it "the same", and 46% had subjectively improved. These results may be biased by the relatively small

sample size (90 patients) and drop out of cases, such that 73% of the cohort was reassessed at eight months. Stone et al (2003) in a 12-year follow up reported that 83% of patients had no change in diagnosis, but that 29% had retired early on medical grounds. To our knowledge the only prospective study was undertaken by Binzer and Kullgren (1998) who reviewed 30 individuals with motor conversion disorder over a period of 2–5 years. At follow-up, 19 patients had completely recovered, with only 3 being unchanged or worse. It is possible that this apparent "better outcome" in later studies is related to both improvement in the diagnosis of neurological conditions (particularly using neuroimaging techniques) and the reclassification of disorders such as the dystonias as neurological conditions. An additional confound for these studies is a small sample size, which leads to difficulties in ascertaining the true incidence and prevalence and natural history of the condition.

Acknowledging the limitations of sample size of these studies there are factors associated with good prognosis that include male gender, acute onset, a short duration of symptoms, an acute precipitating event, change in marital status (either marriage or divorce), premorbid psychiatric diagnosis, good premorbid health, and the absence of a co-existing medical condition. In contrast, poor prognosis is associated with subclinical personality pathology, co-existing medical illness, poor perception of their own wellbeing, and a high score on the Beck Hopelessness Scale and pending litigation. In children, conversion symptoms may remit spontaneously. Pehlivanturk and Unal (2002) found that 85% of children with conversion disorders recovered completely at 4 years, and another 5% had shown some improvement. Indicators of a good prognosis included early diagnosis and good premorbid adjustment. There may be an additional effect related to the clinical nature of conversion disorder. Patients who had sensory symptoms at presentation tended to have a better outcome than those who had presented with weakness (Crimlisk et al 1998), while up to one third of globus hystericus cases may become chronic (Finkenbine and Miele 2004). At 10-year follow-up, 30/56 patients assessed by Mace and Trimble (1996) were still troubled by their presenting symptoms.

Clinical approaches to the treatment of conversion disorder

Hypnosis

There are limited case reports of clinical improvement following hypnosis (see Singh and Lee 1997). On balance, however, the authors do not feel that these provide an evidence base from which to support the use of hypnosis.

Psychotherapy

The cornerstone of treatment of conversion disorders is psychotherapy aimed at elucidating the emotional bases of the symptoms. A multidisciplinary approach to rehabilitation may be beneficial (Moene et al 2002; Wald et al 2004). While the evidence base for these therapies is limited, success has been claimed with psychoanalysis, cognitive behavior therapy (CBT), behavior modification, and family therapy. The successful use of CBT in other medically unexplained conditions such as chronic fatigue syndrome (Whiting et al 2001) make this is a potential area for further research.

Medication

There is little evidence to guide pharmacotherapy in conversion disorder, and in the UK there are no NICE (National Institute for Clinical Excellence) guidelines available at present. The clinical evidence for pharmacotherapy in conversion disorder is extremely limited and consists of case reports. Thus, therapeutic success has been reported with haloperidol (Masuda et al 2003), tricyclic antidepressants (Cybulska 1997), and ECT (Cybulska 1997; Yazici et al 2004). These studies serve to emphasize the importance of screening for comorbid psychiatric conditions.

Conclusions

Despite recent advances in our understanding of the epidemiology, etiology, and treatment of common psychiatric disorders, our understanding of conversion disorder remains limited. The literature reveals there is a limited knowledge regarding the incidence and prevalence of this condition, but studies in cohorts of patients from specialist services suggest that psychiatrists are improving their ability to identify cases of conversion disorder. While not supporting the notion of a "diagnosis of exclusion" regarding physical illness, it is important to identify and treat any comorbid psychiatric illness that may affect the ultimate outcome. The neuroimaging findings of altered prefrontal functioning suggest that this condition may be sensitive to drug treatments, which can modify neural activation in these areas. A combination of treatment with antidepressant medication and appropriate psychotherapy and multidisciplinary rehabilitation focusing on improving the patient's level of functioning and reducing their

subjective distress may be the most effective treatment at present. There is a clear need for further systematic research in this area.

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