Conversion disorder: towards a neurobiological understanding

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Abstract: Conversion disorders are a common cause of neurological disability, but the diagnosis remains controversial and the mechanism by which psychological stress can result in physical symptoms "unconsciously" is poorly understood. This review summarises research examining conversion disorder from a neurobiological perspective. Early observations suggesting a role for hemispheric specialization have not been replicated consistently. Patients with sensory conversion symptoms have normal evoked responses in primary and secondary somatosensory cortex but a reduction in the P300 potential, which is thought to reflect a lack of conscious processing of sensory stimuli. The emergence of functional imaging has provided the greatest opportunity for understanding the neural basis of conversion symptoms. Studies have been limited by small patient numbers and failure to control for confounding variables. The evidence available would suggest a broad hypothesis that frontal cortical and limbic activation associated with emotional stress may act via inhibitory basal ganglia–thalamocortical circuits to produce a deficit of conscious sensory or motor processing. The conceptual difficulties that have limited progress in this area are discussed. A better neuropsychiatric understanding of the mechanisms of conversion symptoms may improve our understanding of normal attention and volition and reduce the controversy surrounding this diagnosis.

Keywords: conversion disorder, hysteria, neurophysiology, functional imaging, SPECT, fMRI

Introduction

The concept of "hysteria" or conversion disorder has always been controversial (Halligan and David 1999), but there is no doubt that medically unexplained neurological symptoms are common and cause considerable disability. About one third of patients in neurology outpatient clinics have symptoms thought to be "non-organic" in nature (Carson et al 2003; Snijders et al 2004) and these symptoms have a poor prognosis for recovery (Carson et al 2003; Stone et al 2003). Despite early concerns that many patients labeled with hysteria or conversion disorder are later found to have an organic explanation for their symptoms (Slater 1965), more recent studies suggest that the incidence of misdiagnosis is now low and that the stability of a diagnosis of conversion disorder in well-investigated patients is high (Couprie et al 1995; Crimlisk et al 1998).

Conversion disorder is defined by DSM-IV as a deficit of sensory or motor function that cannot be explained by a medical condition and where psychological factors are judged to be associated with the deficit because symptoms are preceded by conflicts or other stressors (APA 1994). It is differentiated from factitious disorder by the fact that the symptom or deficit is not intentionally produced or feigned. In ICD-10, conversion symptoms are classified as dissociative disorders (eg, dissociative motor disorder), with similar diagnostic criteria (WHO 1992).

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At present, we have very limited understanding of the mechanism by which psychological stress can "convert" into physical symptoms. The concept of "konversion" was first introduced by Freud and Breuer. Breuer speculated on possible neurological mechanisms of conversion symptoms when he discussed the "abnormal excitability" of the nervous system and "weakness of resistances" in particular paths of conduction (Freud and Breuer 1978). However, it is Freud's psychodynamic conceptualization of conversion disorder that remains inextricably linked with the diagnosis in current classifications. The lack of understanding of the neural mechanism by which psychological stressors can unconsciously result in physical symptoms is an important reason for the ongoing controversy and stigma surrounding the diagnosis.

Empirical research in this area has tended to lag behind theoretical speculation (Halligan, Bass, et al 2000). However, advances in functional imaging and neuropsychological testing offer the opportunity to narrow the gap between psychoanalytical theory and neurobiological explanations of conversion disorder. One difficulty facing research in this field is the complexity of the conceptual issues and variable ways in which terminology has been used. To try to address this issue, we have attempted to define the way in which important terms have been used in this paper as precisely as possible (Table 1).

Table 1 Definitions and explanations of important terminology

Consciousness and awareness "conscious" "unconscious" "awareness" "explicit/implicit"	The term "consciousness" can be used in several ways (Zeman 2001), but in this paper we will refer to consciousness as the qualitative, subjective dimension of experience. Thus, mental processes of which we are *aware* (eg, perceiving an object, forming an intention to move) would be said to be "conscious" or "explicit", whereas mental processes of which we are not aware (eg, a pupillary light reflex, "blindsight", implicit memory) would be said to be "unconscious" or "implicit". In this sense, consciousness is inextricably linked with attention and volition.
Attention	William James provided a seminal definition of attention as "the taking possession by the mind, in clear and vivid form, of one out of what seem several simultaneously possible objects or trains of thought. Focalization, concentration, of consciousness are of its essence" (James 1890). Current neuropsychological models see attention as a set of processes that serve to enhance sensory, motor, and cognitive processing (Gitelman 2003). Deficits in attention may be relevant to sensory conversion symptoms.
Volition, willed action, and intentions	Volition or "will" is the faculty of consciously making a choice or selecting an action (James 1890). "Willed actions" (eg, walking on a slippery surface) can be differentiated from stereotyped, routine actions (eg, walking normally) by the fact that we consciously attend to them and select them (Jahanshahi and Firth 1998). To perform a willed action we first form an "intention" and then execute the action. Evidence from functional imaging and neurophysiology suggests that these stages have a distinct neurological basis (Lau et al 2004; Waszak et al 2004). Deficits in volition, or translating intentions into actions, may be relevant to motor conversion symptoms.
Hierarchical processing "higher" or "downstream" processing	Hierarchical processing refers to perception occurring via a stepwise process in which representations of sensory information are initially simple and gradually become more abstract, holistic, and multimodal (Grill-Spector and Malach 2004). Thus we may refer to "early" processing (eg, the primary visual cortex) and "higher" or "downstream" processing (eg, response to faces in the fusiform gyrus). Several functionally specialized parallel "streams" of hierarchical processing may occur (eg, the dorsal and ventral streams in the visual system for spatial localization and object recognition respectively). It is not known at what stage in this process "awareness" or "consciousness" arises.
Inhibition, excitation, modulation	Terms such as "inhibition", "excitation", and "modulation" are often used rather imprecisely in the literature to refer to the effect that one brain region or pathway may have on another. In fact, cortical and subcortical regions have rich, reciprocal connections to other structures and function within complex networks and circuits that are not yet fully understood. However, simplified models of the important ways in which specific regions may interact are useful to help us form testable hypotheses about mechanisms of conversion symptoms.
P300 potential	The P300 potential is a late positive deflection in an event-related response, which occurs when subjects detect a "target" stimulus, but not when they ignore it or fail to detect it. It is therefore thought to reflect conscious processing of the stimulus (Picton 1992).
Blindsight	Blindsight occurs uncommonly in patients with damage to the primary visual cortex. Despite having no conscious awareness of visual perception, these patients can perform simple visual discrimination tasks in forced choice (Weiskrantz 1996). This phenomenon is thought to reflect implicit visual processing in extrastriatal pathways.
Neglect	Neglect occurs in patients with nondominant inferior parietal lobe damage. It is characterized by reduced awareness of stimuli in the hemispace contralateral to the lesion. This reduced awareness is more marked when stimuli are present in the unaffected hemispace. The neglect can sometimes be improved by drawing patients' attention to the stimulus (Driver and Mattingley 1998).

This review will describe early observational studies of conversion symptoms, neurophysiological studies of conversion disorder, and recent findings from the use of functional neuroimaging to investigate both conversion disorder and feigning. While this is not a systematic review of all research in this area we have attempted to be as comprehensive as possible. We conducted multiple searches in Medline, EMBASE, and PsycINFO databases using the keywords "conversion", "hysteria", "dissociative", "medically unexplained", or "non-organic illness". We obtained additional articles through our knowledge of research in this area and from cross-referencing between papers. Although related disorders including nonepileptic seizures and psychogenic movement disorders may share a similar etiology, the vast majority of research in this area has focused on motor and sensory conversion symptoms. We are confident that our discussion on functional imaging includes all published studies of conversion disorders.

The study of conversion disorder from a neuropsychiatric perspective should provide not only a greater understanding of the etiology and management of this disorder, but also valuable insights into normal cognitive processes, including volition and attention.

Early observational findings

Ever since the late 19th century observers have commented that hysterical hemianesthesia and hemiplegia tend to be seen more commonly on the left than the right (Briquet 1859; Jones 1908; Purves-Stewart 1924). This observation appeared to be confirmed in 1977 when Stern found a significantly higher proportion of left-sided conversion symptoms in both left-handed and right-handed patients (Stern 1977). Three hypotheses have been proposed to explain this apparent laterality of conversion symptoms (Axelrod et al 1980). Psychodynamic theorists suggested that neurotic symptoms tend to be seen on the left because of a psychological association between the right and left sides and good and evil, respectively (Axelrod et al 1980). There has, however, been little empirical support for this so-called "evaluative hypothesis" (Roelofs et al 2000). The "convenience hypothesis" suggested that patients would tend to develop symptoms on the side that caused them the least inconvenience; however, the predominance of left-sided symptoms in left-handed patients argues against this (Stern 1977). The "hemispheric specialization" theory proposed a neurobiological explanation for this apparent lateralization (Roelofs et al 2000). It was suggested that the right hemisphere has a particular role in emotional processing, making it more likely to mediate affectively determined symptoms (Stern 1977). However, recent neuroimaging studies do not support the simplistic notion of right-sided dominance for emotional processing (Wager et al 2003).

Recent studies have questioned whether conversion symptoms are in fact lateralized at all (Roelofs et al 2000). In response to this ongoing debate, Stone and colleagues recently undertook a systematic review looking at more than 100 studies that addressed this issue (Stone et al 2002). They found a small but significant increase in the incidence of symptoms on the left side of the body (58% of patients) but felt that the possibility of reporting bias made this finding inconclusive (Stone et al 2002).

Another early approach to understanding the neural mechanisms of conversion disorder was the study of patients with conversion disorder associated with organic lesions. Patients with nonepileptic seizures are more likely to have right hemisphere pathology than those with epileptic seizures, supporting possible right hemisphere involvement in conversion symptoms (Devinsky et al 2001). In contrast there have been case reports of conversion symptoms occurring after injury or infarction of the left cerebral hemisphere (Drake 1993). Other case studies have found left-sided symptoms of probable hysterical origin to be ameliorated by right anterior thalamotomy (Andy 1973).

A study of patients referred to a unit for treating behavioral disorders after brain injury found that more than 30% of patients had symptoms suggestive of a hysterical cause (Eames 1992). Diffuse insults such as anoxia or hypoglycemia were much more common in those with hysterical symptoms and indeed were almost invariably present in these cases (Eames 1992). The authors suggested that these findings may point to a role for the basal ganglia and diencephalon (structures particularly vulnerable to such insults) in the emergence of conversion symptoms. However, patients with diffuse and localized brain injury may differ in other neurological and psychological respects, suggesting alternative explanations for this finding.

In summary, clinical studies have implicated various brain regions in conversion disorders but many of these findings are based only on case reports and have not been consistently replicated. There is no strong evidence to support the hypothesis of predominant right hemisphere involvement. More recently, research has turned to other techniques to investigate the neural basis of conversion symptoms.

Neurophysiological studies

In 1972, Ludwig proposed a model of conversion disorders in which symptoms result from a dysfunction of attention due to increased corticofugal inhibition of afferent stimulation (Ludwig 1972). Such a theory was supported by early findings of reduced somatosensory-evoked responses from the affected compared with the normal leg in a patient with hysterical hemianesthesia (Hernandez-Peon et al 1963). However, this finding was not reliably reproduced and normal evoked potentials are now thought to be a hallmark of conversion disorder (Sierra and Berrios 1999). Research using magnetoencephalography (MEG) has found that patients with psychogenic sensory loss also have normal evoked activity in the secondary somatosensory cortex (Hoechstetter et al 2002). Current understanding of how somatosensory processing mediates conscious sensory experience is limited. Both the primary and secondary somatosensory cortices have been suggested to be of particular importance (Johansen-Berg et al 2000; Schwartz et al 2005), but these findings suggest that any altered processing in patients with conversion symptoms must occur further downstream.

More recently studies have focused on the P300 component of event-related response (Picton 1992). Lorenz and colleagues designed a paradigm in which patients were asked to verbally report the awareness of electrical stimuli being applied to their hands during EEG recording (Lorenz et al 1998). When healthy patients were asked to feign lack of awareness of the stimulus on one side, a P300 component of the event-related potential was seen. This was thought to represent the processing of the stimulus and the active withholding of a response. When the same paradigm was used in a patient with sensory loss due to conversion disorder no P300 response to stimulation of the affected limb was seen. This provides the first evidence that conversion disorder is neurophysiologically distinct from feigning, but the evidence awaits replication. Interestingly, a lengthening of latency and a decrease in amplitude of P300 waves has also been described in patients with visual neglect due to parietal lobe lesions (Lhermitte et al 1985). This lends some support to the analogy between symptoms seen in right parietal lesions and those in conversion disorder.

Functional neuroimaging

The emergence of functional neuroimaging has provided new methods with which to test hypotheses about the neural circuits underlying conversion symptoms (see Table 2). However, the heterogeneous nature of symptoms seen in conversion disorder and the varying methodologies used makes comparison between studies difficult.

In 1995 the first reported functional imaging study was conducted on a female nurse who had developed left-sided hysterical paralysis and paresthesia (Tiihonen et al 1995). Single-photon emission computed tomography (SPECT) studies were conducted while her left median nerve was stimulated, both while she was symptomatic and when she had recovered (6 weeks later). There was increased perfusion in the right frontal lobe, and hypoperfusion in the right parietal region when her symptoms were present (Tiihonen et al 1995). Similar results were obtained when Marshall and colleagues performed positron emission tomography (PET) on a female patient with a left-sided paralysis that met the criteria for conversion disorder (Marshall et al 1997). Regional cerebral blood flow was compared between episodes of rest, preparing to move, and attempting to move both her unaffected and affected leg. When preparing to move her affected left leg, there was activation of the left lateral premotor cortex and both cerebeller hemispheres relative to the resting condition, suggesting a readiness to move. However, when this patient attempted to move her affected leg, the right premotor and primary sensori-motor cortex failed to activate normally, but there was increased activation in the right anterior cingulate and right orbitofrontal cortex. It was hypothesized that inhibitory pathways involving the orbitofrontal cortex and anterior cingulate may "disconnect" the premotor areas from the primary motor cortex, preventing the patient's conscious intention from being translated into action. The authors suggest that the activation seen when this patient was preparing to move her affected limb provides evidence against feigning. However, they do not address the issue of whether preparing to move can itself be feigned.

More recently, attempts have been made to replicate these case study findings in larger groups of patients. Yazici and Kostakoglu used SPECT to measure resting cerebral blood flow in five patients with astasia–abasia. They found that four of these five patients had decreased perfusion in their left temporal areas and one patient had decreased perfusion in their left parietal lobe compared with the right side (Yazici and Kostakoglu 1998). However, significant methodological issues question the validity of these findings. Most importantly, the contralateral hemisphere does not provide an adequate control condition, particularly as patients' symptoms were bilateral. In addition, the patients studied had heterogeneous symptoms and many had

previous or current psychiatric conditions requiring medication or ECT.

Some of these methodological problems were addressed in a study by Vuilleumier and colleagues in 2001 (Vuilleumier et al 2001). SPECT studies were performed on seven right-handed patients with strictly unilateral loss of motor function (with or without associated sensory loss) who had no other psychiatric or medical conditions. Scans were repeated in a subset of patients, once their symptoms had resolved allowing them to act as their own controls (Vuilleumier et al 2001). This study was also unique in using controlled stimulation with passive vibration rather than actual movements to activate the sensorimotor cortex. Vibration is known to activate motor and sensory cortical areas via proprioceptive pathways (Seitz and Roland 1992). The authors argue that this may reduce confounding due to ambiguity of instructions, strategy, degree of effort, and "conflict reaction" seen when patients are asked to perform voluntary movements. The data obtained were analysed using both statistical parametric mapping and region-of-interest segmentation, comparing activation in 20 pre-defined anatomical regions. Both methods of analysis showed reduced blood flow in the contralateral thalamus, caudate, and putamen during passive vibration when patients were symptomatic compared with when their symptoms had resolved (Vuilleumier et al 2001). They also found that lower activation in the contralateral caudate was a predictor of poor recovery. The authors comment that the basal ganglia and thalamus are strategically placed in neuronal circuits to modulate sensory and motor signals and thus may affect conscious sensory processing or willed action. This theory is supported by the observational studies of brain-injured patients with conversion disorder described above (Eames 1992), and is consistent with the proposed role of cortical–subcortical circuits in volition (Spence and Frith 1999). The caudate nucleus has direct limbic inputs from the amygdala and orbitofrontal cortex, suggesting a possible link with previous reports of increased orbitofrontal activation in conversion symptoms (Vuilleumier et al 2001). The authors hypothesize that emotional stressors act via limbic inputs from the orbitofrontal cortex and amygdala to modulate basal ganglia–thalamocortical circuits, leading to a selective deficit of willed action (Vuilleumier et al 2001).

More recently, functional magnetic resonance imaging (fMRI) has been used to study the Blood Oxygenation Level Dependent (BOLD) response to sensory stimulation in four patients with nondermatomal somatosensory deficits (Mailis-Gagnon et al 2003). These patients met diagnostic criteria for conversion disorder but also had chronic pain in the affected limbs. Unperceived stimuli applied to the affected limb were associated with a different pattern of BOLD response to both innocuous and noxious stimuli,

Table 2 Functional imaging studies of conversion disorder

Authors	Imaging modality	Patients' symptoms	N	Control condition	Paradigm	Perfusion changes associated with conversion symptoms
Tiihonen et al 1995	SPECT	L hemiparesis and hemisensory loss	1	Same patient when recovered	Stimulation of L median nerve	↑ R frontal cortex ↓ R parietal cortex
Marshall et al 1997	PET	L hemiparesis	1	Unaffected side	Attempting to move	↓ R premotor and primary sensorimotor cortex ↑ R anterior cingulate cortex and R orbitofrontal cortex
Yazici and Kostakoglu 1998	SPECT	Astasia–abasia	5	Contralateral hemisphere	Resting	↓ L temporal and parietal cortex
Vuilleumier et al 2001	SPECT	Hemiparesis +/− sensory loss	7	Same patients when recovered	Passive vibration stimulation	↓ contralateral thalamus and striatum
Mailis-Gagnon et al 2003	fMRI	Nondermatomal somatosensory deficits	4	Unaffected limb	Innocuous and noxious stimulation	↑ rostral anterior cingulate cortex ↓ thalamus, anterior cingulated (posterior region), ventrolateral prefrontal cortex, anterior insula
Werring et al 2004	fMRI	Visual loss	5	Healthy controls	Visual stimulation	↓ visual cortex ↑ L inferior frontal, L insula, L striatum, thalami, midbrain, L posterior cingulate
Spence et al 2000	PET	L arm weakness	2	Healthy controls	Movement of joystick	↓ L DLPFC

Abbreviations: DLPFC, dorsolateral prefrontal cortex; fMRI, functional magnetic resonance imaging; L, left; PET, positron emission tomography; SPECT, single-photon emission computed tomography; R, right.

compared with perceived stimuli applied to the unaffected limb. Unperceived stimuli failed to activate the anterior insula, thalamus, anterior cingulate cortex, and ventrolateral prefrontal cortex, but caused increased activation in the rostral anterior cingulate cortex. Unperceived stimuli also caused deactivation in primary and secondary somatosensory areas, posterior parietal, and prefrontal cortex. However, the generalizability of these findings is limited by the prominence of chronic pain in these patients, and the interpretation of deactivation seen on fMRI remains controversial (Hutchinson et al 1999).

Another fMRI study compared five patients with medically unexplained visual loss meeting criteria for conversion disorder with normal controls (Werring et al 2004). During visual stimulation, these patients had reduced activation in their visual cortex and increased activation in their left inferior frontal cortex, left insula, left corpus striatum, bilateral thalami, limbic structures, midbrain, and the left posterior cingulate cortex. The authors note that the networks activated in these patients are similar to those that are activated in blindsight and suggest this may imply a shift towards implicit visual processing in hysterical blindness (Werring et al 2004). Alternatively, they speculate that this activation may represent complex visual processing resulting in inhibition of primary visual areas. It is, however, difficult to interpret this apparent left-sided activation given that patients with both left-sided and right-sided visual deficits were included in this study.

The findings of all of these functional imaging studies are summarized in Table 2.

Conversion disorder versus feigning

Functional imaging has also been used to compare those with conversion disorder with others who have been instructed to feign their symptoms. Spence et al used PET to compare three patients with weakness due to a conversion syndrome with both normal controls and controls instructed to feign weakness (Spence et al 2000). During a simple motor task, those with conversion syndrome had hypoactivation of their left dorsolateral prefrontal cortex (LDLPFC) compared with both normal controls and feigners (Spence et al 2000). This would be consistent with the known role of the dorsolateral prefrontal cortex in volition and willed action (Frith et al 1991; Jahanshahi et al 1995). However, while the patients in this study were euthymic, they all had a past history of depression, which has been suggested as an alternative explanation for the left frontal hypo-activity seen (Vuilleumier et al 2001).

An alternative, novel approach in this field has been the use of hypnosis as a model for conversion disorders. Hypnosis can produce a compelling experience of subjective paralysis and thus may have some conceptual links with conversion disorder. In order to establish hypnotically induced paralysis as a model for conversion disorder, Halligan and colleagues replicated the methods used in an earlier imaging study of a patient with conversion disorder and demonstrated that a subject with hypnotic paralysis activated similar brain areas when they tried to move the affected limb as those seen in conversion disorder (Halligan, Athwal, et al 2000). Based on these results Ward et al (2003) conducted a study using 12 healthy volunteers who each had 2 different paralysis conditions created under hypnosis: a subjectively experienced paralysis, and a condition where subjects were aware there was no paralysis but were instructed to feign weakness. PET scanning measured regional cerebral blood flow at rest and on attempting to move the affected left leg. Using Statistical Parametric Mapping analysis there was no significant difference in activation during attempts to move between the two paralysis conditions (Ward et al 2003). However, a small volume correction analysis based around a region of interest from a previous study (Spence et al 2000) did find increased activation in the left ventrolateral prefrontal cortex (LVLPFC) during feigning compared with subjectively experienced paralysis (Ward et al 2003). The authors argue that this is broadly consistent with the finding of Spence et al (2000) of activation of the LDLFC during feigning, not only because of the proximity of these regions, but also because Spence et al reported a separate peak of significant activation in the LVLPFC. The LVLPFC may therefore have a role in the conscious volitional inhibition seen in feigning (Ward et al 2003). This would be consistent with functional imaging studies of deception, which have found lying to be associated with increased activity in the VLPFC (Spence et al 2001). It should, however, be noted that the effect seen in the Ward et al study was small and only significant in one of the two methods of analysis used. While the hypnosis paradigm avoids many of the confounders and co-morbidities that have limited other studies, it remains uncertain how far these results can be generalized. The neurobiological processes involved in such short-lived, experimental conditions may be very different to those involved in the more chronic and complex cases seen in clinical practice (Ward et al 2003).

Conclusions

Conversion disorders are common and cause considerable morbidity but, despite attracting ongoing controversy and theoretical interest, there has been relatively little empirical research in this field.

Early neuropsychiatric studies of conversion symptoms focusing on the apparent importance of laterality and patients with brain lesions failed to produce consistent findings. Neurophysiological studies have suggested that conversion disorder results from changes in higher-order cortical processing. The development of functional neuroimaging has provided a new paradigm with which to study the neural basis of conversion, but the few studies to date have often been limited by small sample sizes and significant methodological issues, and there have been some inconsistencies in their findings.

At present the evidence available suggests a broad hypothesis that frontal cortical and limbic activation associated with emotional stress may act via inhibitory basal ganglia–thalamocortical circuits to produce a deficit of conscious sensory or motor processing. Functional imaging and neurophysiological evidence to support the distinction between conversion and factitious disorders is limited.

Difficulties with classification and terminology continue to hamper research in this area. The place of conversion disorder in psychiatric classification depends on its presumed unconscious psychological mechanism and so necessarily relies on clinicians' judgments which may be subjective and difficult to validate (Broome 2004). This has led some authors to question the usefulness of a distinction between conversion and factitious disorders (Austen and Lynch 2004; Shapiro and Teasell 2004). However, we would argue that there is an important difference in phenomenology between these two disorders. Current theories of consciousness suggest a continuum of awareness of intentions or perceptions that may fluctuate over time (Zeman 2001). We would suggest that the functional anatomical basis of symptoms is likely to differ between patients with different levels of awareness and that this is an important area to study.

To use functional imaging more effectively to understand the psychopathology of conversion disorder, we first need a clear cognitive model of the information processing deficits that underlie these symptoms. At present, our understanding of the "neural correlate of consciousness" is limited, making it harder to propose cognitive models of "unconscious" processes. Studies of the visual system have so far provided the best understanding of the neural basis of awareness. Blindsight and visual neglect, like hysterical blindness, are both characterized by reduced awareness of visual stimuli with preservation of some aspects of visual processing. Indeed, the one functional imaging study of hysterical blindness found activations in very similar areas to those previously reported in blindsight (Werring et al 2004). Comparisons between these syndromes may provide useful insights into relevant cognitive processes and suggest hypotheses about brain regions which may be important in conversion symptoms.

Studies with larger numbers of patients and better attempts to control for confounders are needed to further test these hypotheses. Despite increasingly sophisticated study designs and imaging techniques, conceptual problems may continue to limit progress in this area. However, by beginning to understand the neurobiology of conversion disorder we may gain valuable insights into the cognitive processes involved in attention and volition and reduce some of the controversy and stigma associated with this common condition.

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