The concept of hysteria or conversion disorder, firmly anchored in psychoanalytic theory, postulates the emergence of physical symptoms as an unconscious attempt to resolve a painful psychic conflict. The advantages accruing from the adoption of the sick role contribute to perpetuate the disability. Two major problems with this concept have detracted from its clinical value. The first is the difficulty in establishing the occurrence, relevance and timing of a ‘sufficient’ psychological stressor. The second is to distinguish ‘unconscious’ motivation from feigning. Faced with these difficulties and threatened by the fear of missing treatable neurological conditions, clinicians have opted for a diagnosis by exclusion, invoking conversion disorder only after detailed investigations have failed to find an alternative explanation for the symptoms. The advent of telemetry and non-invasive imaging and the better characterization of some neurological disorders (e.g. the dystonias) have made it possible to diagnose as neurological hitherto ‘unexplained’ symptoms. Future advances will, no doubt, help to explain others, but there is strong evidence to suggest that for a core of symptoms the diagnosis of hysteria or conversion disorder will remain valid. The strongest evidence comes from follow-up studies of carefully investigated patients (Binzer and Kullgren, 1998; Crimlisk et al., 1998) for whom conversion disorder is a stable diagnosis and not the harbinger of unsuspected neurological disease. To understand the brain mechanisms that predispose to and help to maintain these symptoms remains our greatest challenge, but perhaps one we are now able to meet with the help of functional imaging.

In this issue, Vuilleumier and colleagues report the functional neuroanatomical correlates of hysterical sensorimotor loss in seven patients using SPECT (single photon emission computerized tomography) (Vuilleumier et al., 2001). Patients were included in the study if the unilateral sensorimotor symptoms had been present for <2 months and detailed investigations had excluded organic pathology. Stressful events were reported by all in connection with the onset of symptoms. When simultaneous passive vibration to both hands, a stimulus known to elicit widespread activity in sensory and motor areas, was compared with a resting condition, bilateral and symmetrical rCBF increases were observed in parietosomatosensoy, frontal premotor and anterior prefrontal cortical areas. By contrast, rCBF was reduced in the thalamus, putamen and caudate contralateral to the side of the symptoms and low caudate rCBF was associated with poor prognosis. Interestingly, these asymmetries disappeared in the four patients scanned after recovery. The authors suggest that decreased activity in circuits involving basal ganglia and thalamus could impair motor readiness resulting in abnormal voluntary movements and that emotional stressors could inhibit these circuits through the input from the amygdala and orbitofrontal cortex.

This study follows a few case reports that have investigated the neural networks involved in hysterical paralysis using different functional imaging modalities and exploring different aspects of motor behaviour. The methodological differences of these studies have yielded somewhat conflicting results. A point of disagreement is whether the inability to perform voluntary movements results from abnormalities in readiness to move, or whether the problem occurs later due to the interruption of a normally generated motor programme. The study of Vuilleumier and colleagues suggests that the abnormality may lie in the generation of motor programmes and readiness to move, while other studies have suggested that normally generated motor programmes may be interrupted at a later stage. Thus Marshall and colleagues reported normal brain activation using PET when the patient was asked to prepare to move the paralysed leg (Marshall et al., 1997). This was interpreted as evidence against feigned paralysis. Failure to move was explained as resulting from inhibition of prefrontal regions involved in willed action by the right orbitofrontal and anterior cingulate areas that were activated when the patient attempted to move the paralysed leg. A similar finding was reported by Spence and colleagues, who examined patients with hemiparesis using PET employing a paradigm that required the subjects to move a joystick in freely chosen sequences (Spence et al., 2000). Deactivation of the left dorsolateral prefrontal cortex (DLPFC) was detected in all patients, regardless of the side of the symptoms. As the DLPFC is specifically activated by willed action, these findings suggest that hysterical motor symptoms involve the higher components of volition, although the specific inhibitory circuits were not identified in this study. Taking advantage of the similarities between imagined and actual motor performance, Maruff and Velakoulis also reported that ability to generate motor plans was intact in a patient with
hysterical paralysis when compared with normal controls (Maruff and Velakoulis, 2000), suggesting once again that inhibition of normal motor programmes occurs at a later stage. The neural mechanisms involved in other hysterical symptoms (e.g. blindness) remain to be determined, but it seems likely that inhibition of normal neural networks will be common to all.

The distinction between conscious (feigning) and unconscious motivation has been considered one of the great weaknesses of the psychoanalytic concept of hysteria and it is often forgotten that patient’s awareness changes and what starts as unconscious may become conscious and vice versa (Kendell, 1982). This would suggest that different neural networks could be implicated in explaining the same symptom in a given patient depending on chronicity, therapeutic interventions or environmental changes. Tardif and colleagues, using event-related potentials to explore feigned impairment of recognition memory, have suggested that feigning requires more complex or additional cognitive activity than that exhibited by normally performing subjects (Tardif et al., 2000). However, this observation also seems to apply to patients in whom conscious motivation is not suspected, as evidenced by functional imaging studies mentioned here, and a search for more specific patterns of activation may be more fruitful. The study of Spence and colleagues is so far the only one that has directly compared hysterical and feigned weakness (Spence et al., 2000). Using a joystick to generate freely chosen sequences paced by an auditory tone to ensure similar speed of movement, feigners exhibited hypofunction of the right anterior prefrontal cortex compared with controls. This pattern of activation was different from that of patients with hysterical weakness who exhibited hypoactivity of the left DLPFC. It remains to be determined whether these differences reflect conscious versus unconscious motivation or whether they simply result from differences in chronicity between the ‘acute’ feigned symptoms and the more chronic hysterical weakness.

The findings of these few studies are intriguing rather than conclusive, but other studies are certain to follow using less invasive functional imaging techniques and including bigger samples and repeated testing. Unexplained symptoms are common enough and costly enough to deserve this much attention.

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References