CHAPTER 23

Hysterical conversion and brain function

Patrik Vuilleumier*

Laboratory for Behavioral Neurology and Imaging of Cognition, Clinic of Neurology & Department of Neurosciences, University Medical Center, Faculty of Psychology & Education Sciences, University of Geneva, Geneva, Switzerland

Abstract: Hysterical conversion disorders represent “functional” or unexplained neurological deficits such as paralysis or somatosensory losses that are not explained by organic lesions in the nervous system, but arise in the context of “psychogenic” stress or emotional conflicts. After more than a century of both clinical and theoretical interest, the exact nature of such emotional disorders responsible for hysterical symptoms, and their functional consequences on neural systems in the brain, still remain largely unknown. However, several recent studies have used functional brain imaging techniques (such as EEG, fMRI, PET, or SPECT) in the attempt to identify specific neural correlates associated with hysterical conversion symptoms. This article presents a general overview of these findings and of previous neuropsychologically based accounts of hysteria. Functional neuroimaging has revealed selective decreases in the activity of frontal and subcortical circuits involved in motor control during hysterical paralysis, decreases in somatosensory cortices during hysterical anesthesia, or decreases in visual cortex during hysterical blindness. Such changes are usually not accompanied by any significant changes in elementary stages of sensory or motor processing as measured by evoked potentials, although some changes in later stages of integration (such as P300 responses) have been reported. On the other hand, several neuroimaging results have shown increased activation in limbic regions, such as cingulate or orbitofrontal cortex during conversion symptoms affecting different sensory or motor modalities. Taken together, these data generally do not support previous proposals that hysteria might involve an exclusion of sensorimotor representations from awareness through attentional processes. They rather seem to point to a modulation of such representations by primary affective or stress-related factors, perhaps involving primitive reflexive mechanisms of protection and alertness that are partly independent of conscious control, and mediated by dynamic modulatory interactions between limbic and sensorimotor networks. A better understanding of the neuropsychobiological bases of hysterical conversion disorder might therefore be obtained by future imaging studies that compare different conversion symptoms and employ functional connectivity analyses. This should not only lead to improve clinical management of these patients, but also provide new insights on the brain mechanisms of self-awareness.

Introduction

Since more than a century, hysteria has continuously fascinated both clinicians and theorists interested in altered states of self-consciousness. As a medical condition in which patients may present with various somatic symptoms without a recognized organic illness, hysteria still constitutes a poorly understood class of disorders at the border between psychiatry and neurology, with many different appearances and names. A variety of classifications and explanations have been proposed over the years, with different emphasis on psychological or neurobiological factors, but none is still

*Corresponding author. Tel: +41 (0)22 3795.381; Fax: +41 (0)22 3795.402; E-mail: patrik.vuilleumier@medecine.unige.ch

DOI: 10.1016/S0079-6123(05)50023-2
Fig. 1. Illustration of paraparesis (astasia–abasia) of hysterical origin (drawn by Jean-Martin Charcot in the 1870s).

entirely satisfactory (see Halligan et al., 2001 for a recent general overview).

In some respects, the most important change since classical descriptions of hysteria by Charcot and others at the end of the 19th century (see Figs. 1 and 2) is related to the fact that the term “hysteria” was recently eliminated from the official psychiatric terminology. Thus, hysteria is now referred to as “conversion disorder” in the DSM-IV classification (American Psychiatric Association, 1994, *Diagnostic and Statistical Manual of Mental Disorders*), where it is defined as a loss or distortion of a neurological function (e.g., paralysis or anesthesia) that is not explained by an organic neurological lesion or medical disease, arising in relation to some psychological stress or conflict, but not consciously produced or intentionally feigned. However, although the term “conversion” implies a specific mechanism whereby a primary psychological disorder is converted into bodily symptoms, the exact processes responsible for triggering this phenomenon and altering the patients’ awareness of their bodily state still remain unknown. In fact, when considering the possible neurobiological changes associated with hysterical conversion symptoms, many of the current hypotheses are not very different from those elaborated in the 19th century. Moreover, in contrast to other psychiatric conditions (such as depression, anxiety, compulsion, or phobia), surprisingly few investigations have been carried out to examine whether the “functional” symptoms experienced by hysterical patients might correspond to any
underlying “functional changes” in cerebral activity, as can be evaluated for instance using a variety of modern neuroimaging techniques.

The aim of this chapter is to present a general overview of the relationships between hysterical conversion and brain function, focusing particularly on previous attempts to determine some neurophysiological correlates of hysterical conversion disorders. This review will primarily concentrate on patients with unexplained neurological deficits in sensory and/or motor functions (i.e., hysterical paralysis or anesthesia), since such disorders are the most frequent and most easily described in terms of specific neurological pathways. Similar approaches in other domains (e.g., visual loss, deafness, or amnesia) will also be briefly mentioned, but other conversion disorders associated with more “positive” symptoms such as pseudo-seizure and abnormal movements will not be discussed here, since even less is known about their possible functional neural correlates (for review see Prigatano et al., 2002; Reuber et al., 2003). Finally, a few studies have examined the functional neuroanatomy of dissociations induced by hypnosis (Maquet et al., 1999; Faymonville et al., 2000; Halligan et al., 2000; Kupers et al., this volume), but their implications for hysterical deficits are still in part unclear and will be discussed briefly.

A better understanding of patients with hysterical conversion has important implications for several reasons, both clinical and theoretical. First, such disorders are very common in clinical practice, causing frequent problems in diagnosis and therapy for neurologists as well as psychiatrists, and resulting in important medical costs and major socio-economic burden for the patients and their relatives. Second, conversion disorders raise important theoretical questions concerning the relationships between body and mind, and the neurobiological and psychological mechanisms underlying self-awareness.

From the perspective of clinical neurology, dissociations between performance and awareness of performance are not unusual in patients with organic brain lesions, a phenomenon termed “anosognosia” whereby patients may fail to acknowledge and even explicitly deny a severe handicap resulting from their brain lesion (Vuilleumier, 2000a, 2004; Marcel et al., 2004). Anosognosia cannot be explained by psychogenic factors alone, or by general confusion, but it is often associated with a lack of emotional concern quite similar to “la belle indifference” that has typically been described in hysteria. Moreover, like anosognosia for hemiplegia, hysterical paralysis and hysterical anesthesia are often thought to affect the left more than the right limbs, without this being entirely accounted for a more frequent dominance of the right hand (Galin et al., 1977; Stern, 1983; Pascuzzi, 1994; Gagliese et al., 1995), although a recent meta-analysis has questioned the existence of such an asymmetry (Stone et al., 2002). It is also intriguing to note that the neurological functions most frequently associated with anosognosia are strikingly similar to those frequently concerned by hysteria, involving not only unilateral paralysis and anesthesia, but also blindness, amnesia, or jargon aphasia (Merskey, 1995; Vuilleumier, 2000a). Although such similarities do not indicate any clear relationships between these different disorders, these parallels between neurology and psychiatry highlight the fact that some behavioral abilities may dissociate from the subjective experience of these abilities, and that such dissociations are likely to arise from specific changes in brain function – be they due to certain types of organic damage or to certain psycho-affective states.

Incidence and evolution

Hysterical conversion disorders are thought to represent 1–4% of all diagnoses in general hospitals throughout western countries. This frequency has remained remarkably stable across the past decades despite many important changes in medicine. Thus, a retrospective survey at the National Hospital of Neurology and Neurosurgery in London, Queen Square, from 1955 to 1975 revealed a relatively constant rate of patients investigated for conversion or “functional” symptoms, ranging from 0.85 to 1.55% over decades (Trimble, 1981). A similar result was found by another study examining the diagnoses made on 7836 successive outpatient referrals at Charing Cross
Hospital between 1977 and 1987 (Perkin, 1989), which revealed a stable incidence of 3.8% of conversion disorders over the years. In Switzerland, Frei (1984) also estimated that the proportion and presentation of hysterical conversion disorders among patients seen in a large public hospital was similar in the 1920s as compared with the 1980s.

However, it is important to emphasize that the type of conversion disorders may significantly vary across different medical settings and referral sources. This might explain why the incidence of hysteria may appear to vary in some domains but not in others. It is known that a large number of patients with conversion and somatoform symptoms are never seen by either neurologists nor psychiatrists (Bridges and Goldberg, 1988; Crimlisk and Ron, 1999). Neurologists probably see many more patients with relatively acute and limited symptoms, as compared with psychiatrists who tend to see patients with more chronic and multiformal disorders (Marsden, 1986). Moreover, although there is a good consensus between neurologists as to clinical syndromes reflecting non-organic diseases (irrespective of which term is actually preferred to describe these syndromes, e.g., hysterical, functional, or psychogenic), there is usually less agreement among psychiatrists (Mace and Trimble, 1991). Furthermore, although most neurologists would agree with the DSM-IV statement that “typically, individual conversion symptoms are of short duration”, many psychiatrist have rather observed that conversion symptoms often tend to persist or reoccur in association with other psychiatric comorbidities and pathological personality traits (Ron, 1994; Binzer and Kullgren, 1998; Stone et al., 2003).

However, in most cases seen by neurologists, a rapid remission of initial symptoms is observed after appropriate behavioral treatment. The likelihood of spontaneous remission has repeatedly been found to be \( \sim 50-60\% \) after 1 or 2 years (Singh and Lee, 1997; Binzer and Kullgren, 1998; Crimlisk et al., 1998) or even greater (Folks et al., 1984). Among young patients (< 27 year-old), only 3% have symptoms for more than 1 month (Turgay, 1990). The duration of effective behavioral treatment in such cases is usually between 2 to 26 weeks (Speed, 1996). Several factors are associated with a favorable prognosis, including young age, sensory rather than motor symptoms, acuteness of presentation, onset precipitated by a stressful event, good premorbid health and good socio-economic status, as well as an absence of any other concomitant organic disease or major psychiatric symptoms — especially depression (e.g., Ford and Folks, 1985; Binzer and Kullgren, 1998; Crimlisk et al., 1998; Stone et al., 2003). Personality disorder appears as a particularly decisive prognostic factor (see Crimlisk et al. 1998). It is therefore important to identify potential risk factors during the initial evaluation of patients, in order to prevent the development of a more chronic handicap.

**Body, mind, and brain diseases**

A potential limitation to our current understanding of hysterical conversion comes from a lack of clear definition of the boundaries with some related disorders. From the perspective of psychiatry, a number of problems are still unresolved concerning the terminology and classification of functional symptoms without an organic cause. In DSM-IV, the concept of hysteria has been broken down to different disorders, including somatoform disorders on one hand and dissociative disorders on the other hand, with conversion disorders being considered as a specific category of somatoform disorders involving sensorimotor symptoms or pseudo-seizures, distinct from other somatization symptoms or psychogenic pain disorders. Furthermore, depression and chronic fatigue syndrome would fall under other diagnostic categories, although in clinical practice there is often some overlap between all such conditions. For example, many patients recovering from conversion eventually suffer from depression at a later stage (Binzer and Kullgren, 1998). Furthermore, whereas conversion disorders are defined as deficits affecting a specific neurological function such as motor strength or somatosensory perception, psychogenic memory loss is considered instead under the category of “dissociative disorders” even though memory is obviously also a specific brain function (see Markowitsch, 1999, 2003 for a
neuropsychological perspective of psychogenic amnesia). Therefore, unlike DSM-IV, the International Classification of Diseases (ICD-10) of the World Health Organization has included all sensorimotor and memory symptoms of presumed psychogenic origin under the same general category of dissociative disorders.

From a neurological perspective, it is worth noting that hysterical conversion may sometimes coexist with a real organic brain disease, although the conversion symptoms in such cases is not directly explained by this brain lesion alone. Gowers (1893) already recognized that hysteria could occasionally be a “complication” of organic brain disease, and Schilder (1935) wrote that brain dysfunction could sometimes induce “organic neurotic attitudes”. More recently, an intriguing study by Eames (1992) described a series of 167 patients who were admitted to a rehabilitation ward and reported that 32% of these patients exhibited at least one “hysteria-like” behavior during the course of their rehabilitation, as assessed by systematic rating scales used by caregivers. Such behaviors could include exaggeration, secondary gain expectancy, or non-organic patterns of the deficit. Interestingly, not all types of patients presented hysteria-like symptoms, but these behaviors were more frequent after diffuse brain lesions (e.g., closed injuries, anoxia, encephalitis) than after focal lesions (e.g., stroke), after subcortical more than cortical lesions, and in patients with extrapyramidal motor disorders more than in others (Eames, 1992). Similarly, Gould et al. (1986) reported that among a prospective series of 30 patients with an acute hemispheric stroke, “atypical signs” usually suspected to reflect non-organic “functional” origin were observed in approximately 20% of cases (e.g., changing deficit, patchy sensory loss, or “give-away” weakness). Finally, multiple sclerosis (e.g., Nicolson and Feinstein, 1994) and epilepsy (e.g., Devinsky and Gordon, 1998) constitute two other frequent neurological diseases in which not only some truly “organic” manifestations may sometimes be difficult to distinguish from non-organic disorders, but some patients may also present with a combination of apparently both “organic” and “psychogenic” manifestations at the same time. These occasional associations between hysterical conversion and neurological diseases are not only challenging for current “dichotomous” classification schemes, but in fact might potentially provide valuable clues about the neurocognitive mechanisms by which awareness of a function can be dissociated from actual abilities in a patient. However, the coexistence of these neurological diseases and conversion might also just be a coincidence, purely due to their high incidence or to any other general stress factors.

Nevertheless, despite these problems of definitions and associations, hysterical conversion is only rarely falsely diagnosed for an occult neurological condition. Although a few early studies suggested that up to 20% of patients initially diagnosed with motor conversion deficits may subsequently develop a real organic neurological disease explaining their original symptoms (Slater, 1965; Mace and Trimble, 1996), several recent studies have now clearly established that such rates were overestimated and that only 1–5% of hysterical patients may present with a underlying but occult organic cause (e.g., Crimlisk et al., 1998; Stone et al., 2003). The rarity of organic diseases detected in current follow-up studies is probably due to several factors, including a better characterization of neurological diseases and the availability of more sensitive non-invasive diagnostic procedures.

History and theories

The term of “hysteria” was forged by the ancient Greeks to indicate that physical disturbances in the uterus were the primary cause of psychic symptoms in women. By contrast, all modern theories of conversion disorders acknowledge that not only both men and women may be affected, but also a primary psychological disturbance is probably responsible for triggering subjective physical symptoms, and perhaps also for triggering some associated changes in the neurophysiological state of the central nervous system. However, the exact psychological factors to blame still remain disputed, and the possible implication of a particular
brain functions in generating an abnormal physical experience still remains unresolved.

Since the 19th century, a myriad of different theories have been put forward to explain how hysterical motor or sensory deficits might be implemented in the brain in terms of specific anatomical circuits, or in relation to putative cognitive architectures. However, most of these theories rest on purely speculative grounds, and little empirical work has been conducted to test these neuropsychological hypotheses. Although it is beyond the scope of this chapter to discuss all of these theories in great detail (for more complete reviews, see Merskey, 1995; Halligan et al., 2001), a few conjectures on the neural substrates of hysteria that have been proposed by influential neuroscientists will be briefly illustrated. Although many old theories are susceptible to misinterpretations based on our current knowledge, it is interesting to note that some of them might still appear attractive if they were rephrased using more modern concepts and terminologies from current cognitive and affective neurosciences. In fact, several recent hypotheses about the possible cerebral correlates of hysterical conversion, proposed on the basis of new functional neuroimaging results, can be traced back to strikingly similar ideas that were elaborated more than a century ago, but naturally phrased in terms of models of brain physiology from that time.

One of the first and best known hypothesis on the cerebral mechanisms of hysterical conversion was proposed by Charcot in the early 1890s (see Charcot, 1892; Widlocher, 1982; White, 1997). Charcot suggested that hysterical losses in motor or sensory functions were produced by functional alterations within the central nervous system, affecting activity of motor or sensory pathways without any permanent structural damage. Hystera was thus considered as a “neurosis” among other functional illnesses such as epilepsy or Parkinson’s disease. Charcot suggested that such functional changes in the nervous system could be induced by particular ideas, suggestions, or psychological states, as demonstrated by the effect of hypnosis on hysterical symptoms. Thus, hysterical paralysis could result from an inability to form a “mental image” of movement, or instead from an abnormal “mental image” of paralysis. In essence, these ideas are echoing the more recent proposals of a selective impairment in action “representations” for intentional motor planning (e.g., Spence, 1999), although both the terminology and concepts of brain function have considerably changed since then.

At the same time as Charcot, Janet (1894) also proposed that hysterical deficits were the result of “fixed ideas” that could take control over mental or motor functions. However, he added that such ideas could arise at an unconscious level and acted by inducing a dissociation between distinct domains of behavior (one becoming dominated by the unconscious fixed idea). Such views paved the way to the classical theory of Freud and Breuer (1895), later refined by Freud alone (Freud, 1909), who formulated a purely psychodynamic account with only minimal reference to the nervous system. In brief, according to Freud and Breuer, hysterical deficits were produced by affective motives and conflicts, which were unconsciously repressed and transformed into bodily complaints with symbolic values. For this reason, hysterical deficits did not obey anatomical constraints as observed with organic neurological lesions. This view has then naturally led to the term of “conversion disorder”, and in many respects is still prevailing today. Only later did Freud emphasize that the unconscious affective motives usually had their origin in sexual concerns from early childhood.

An attempt to integrate these different cerebral and psychological perspectives was made by the French neurologist Babinski in 1912, who also first described anosognosia after brain damage two years later (Babinski, 1914). Babinski believed that hysteria involved a form of suggestion permeating the subject’s awareness in the same way as hypnosis, but under the influence of strong emotions, and in the presence of some individual predisposition. According to Babinski, such emotions were elicited in a purely automatic and reflexive manner, and could have both physical (“organic”) and subjective (“imaginal”) manifestations. Conscious control could operate only at the level of a subsequent interpretation stage. A more sophisticated neuro-anatomical scheme was later proposed by Pavlov (1928–1941, 1933), who suggested that in predisposed individuals with a somewhat weak “resistance”, some over-excitation of subcortical
cerebral centers (possibly mediating emotional or learned conditioned responses) could lead to a re-active inhibition of cortical inputs imposed by the frontal cortex. This inhibition was responsible for hysterical paralysis or anesthesia, whose duration was proportional to the amount of resources depleted in the individual.

A number of more recent accounts based on neurophysiological speculations have similarly proposed a role for inhibitory or "filtering" mechanisms as a likely neural substrate for generating hysterical conversion deficits. Thus, in line with Babinski and Pavlov, Sackheim et al. (1979) and Stern (1983) hypothesized that affective or motivational processes might induce a selective blockage (or distortion) of sensory and/or motor inputs, resulting in their exclusion from conscious awareness. A greater involvement of the right hemisphere in emotion might account for more frequent symptoms on the left side of the body observed in some series (Stern, 1983; see Stone et al., 2002). Other researchers such as Ludwig (1972) suggested that an inhibition of sensory or motor functions could arise through gating mechanisms at the level of thalamic nuclei, under the influence of attentional factors (see also Sierra and Berrios, 1999), whereas Spiegel (1991) instead emphasized an attentional mechanism mediated by the anterior cingulate cortex. Likewise, Kihlstrom (1994) argued that conversion disorders might involve a dysfunction in the "monitoring and controlling functions of consciousness", resulting in a dissociative state that might affect the subjective experience of perception and/or voluntary action, presumably through abnormal coordination of particular neurocognitive modules. Oakley (1999) also referred to neuropsychological models of attention to suggest that during hysteria, an internal representation of the ongoing sensory or motor activity might be excluded from awareness by a central executive attentional system involving frontal and cingulate areas. Finally, Halligan and David (1999) and Marshall and colleagues (1997) as well as Spence (1999) and Spence and colleagues (2000) also speculated that representations of motor action might be inhibited in patients with hysterical paralysis, with activation in their motor cortex being actively suppressed by abnormal signals from limbic systems within orbitofrontal and cingulate cortex during volitional movements (Marshall et al., 1997). Again, such inhibition was ascribed to unconscious motivational factors.

Finally, from the mid-1970s to mid-1980s, owing to the influence of the research in split-brain patients, a number of authors interpreted hysterical neurological symptoms as a form of interhemispheric disconnection or dysregulation syndrome (Galin et al., 1977). The greater occurrence of left hemibody disorders led to the idea that the transfer of sensory or motor inputs might be impaired between the right hemisphere (involved in emotions but without language abilities) and the left hemisphere (responsible for verbal and symbolic expression), with or without some additional impairment in the right (Stern, 1983) or left hemisphere (Flor-Henry et al., 1981). However, only very indirect evidence from performance on neuropsychological tests was offered in support of these interhemispheric hypotheses (Flor-Henry et al., 1981).

In sum, most theoretical models trying to link hysterical disorders with specific neuropsychological or neurophysiological mechanisms have essentially been inspired by speculative arguments or analogies with general models of the brain and mind, rather than by the convergence of systematic empirical research. It is striking that relatively few studies over the past decades have exploited neurophysiological techniques (see below) that provide objective measures of brain functions (such as electroencephalogram (EEG) or brain imaging), allowing a better identification of neurobiological factors associated with hysterical conversion. This is all the more surprising since the well-defined neurological-like symptoms of conversion (e.g., paralysis, anesthesia, blindness, etc.) should potentially be amenable to a precise investigation with well-defined predictions about the site and type of neurophysiological dysfunction. In particular, the advent of new functional imaging techniques should now allow a refine assessment of functional correlates in brain activity potentially associated with hysterical conversion, and thus go beyond the dichotomous question of "organic" versus "non-organic" disease. A better knowledge of such functional correlates might provide
important constraints on psychodynamic theories of conversion, with greater biological and neurological plausibility, and might also improve the clinical assessment and management of patients.

Electrophysiological correlates of conversion disorders

Since EEG provided one of the first tools to measure brain activity, a number of studies from the 1960s to 1970s onward have used this technique and other related electrophysiological measures to investigate brain functions in patients with hysterical conversion. These studies can generally be considered in two broad categories: those aiming at demonstrating intact electrophysiological responses despite subjective functional losses, and those trying to determine some abnormal pattern correlating with functional symptoms.

Many reports have described that basic scalp potentials evoked during simple sensory stimulation are usually normal during hysterical sensory deficits. For instance, standard somatosensory evoked potentials (SEPs), visual evoked potentials (VEPs), or brainstem auditory evoked potentials (BAEPs) are usually found to disclose normal amplitudes and latencies in the presence of subjective anesthesia, blindness, or deafness, respectively (e.g., Howard and Dorfman, 1986; Drake, 1990). These findings clearly indicate that primary sensory pathways are both structurally and functionally intact in the patients. In the somatosensory domain, patients have also been studied using magnetoencephalography (MEG) in order to distinguish activations in primary (SI) and secondary (SII) cortical areas (Hochstetter et al., 2002), since data from healthy people typically show a differential modulation of SII but not SI activity by attention and task-related factors. However, MEG results in patients with hysterical sensory loss also showed a normal pattern of response for the characteristic components generated in both SI and SII, contralaterally and ipsilaterally to the deficit. If anything, a trend for even greater responses was observed in the patients relative to healthy subjects, in both SI and SII (Hochstetter et al., 2002). These data converge with earlier EEG results suggesting a paradoxical amplification rather than attenuation of tactile evoked responses during hysterical anesthesia (Moldofsky and England, 1975). Taken together, these findings do not appear consistent with the common hypothesis about conversion disorder, according to which sensory stimuli might be filtered out of awareness by attention-related gating mechanisms (see, e.g., Ludwig, 1972), at least at these relatively early stages of cortical processing. Moreover, these early SEPs may not necessarily correlate with subjective perceptual experience, since SI responses can still be elicited by unperceived stimuli in patients with brain tumors involving the parietal lobe but sparing SI (Preissl et al., 2001). Also, in patients in a vegetative state, devoid of any conscious perception, preserved SI activation has been shown using functional imaging and simultaneously recorded SEPs (Laureys et al., 2002). These data emphasize that SI activation does not necessarily mean conscious somatosensory perception.

On the other hand, a few other studies have reported subtle changes in paradigms that were slightly more sophisticated than just detection of simple tactile stimuli. For instance, tactile stimuli close to perceptual threshold may fail to produce normal evoked potentials in patients with sensory conversion symptoms, even when stimuli above threshold still produce normal responses (Levy and Mushin, 1973). In addition, anomalies in the rate of habituation to repeated stimulations were observed in hysterical conversion using SEPs (Moldofsky and England, 1975) as well as skin-conductance reactivity (Horvath et al., 1980). In normal subjects, responses were found to decrease over time when comparing late blocks of stimuli relative to initial blocks. Such habituation could also be observed in patients with high levels of anxiety (Horvath et al., 1980), but was not present in patients with hysterical conversion, indicating that the latter tended to process frequent and expected stimuli as if they were still novel.

Another recent single-case study reported anomalies in tactile evoked potentials, but involving a more cognitive stage of attentive processing, as indexed by the P300 component (Lorenz et al., 1998). This component is typically elicited by
novel stimuli in an “oddball” task or by infrequent targets within a stream of successive stimuli, and presumably reflects a normal orienting response to relevant stimuli. Lorenz et al. (1998) designed an elegant EEG paradigm in which they repeatedly stimulated the unaffected left hand of a man with hysterical sensory loss on the right hand, and occasionally applied a “deviant” stimuli on either the affected right hand, or on another finger of the same unaffected left hand. The patient showed a normal P300 response for deviant stimuli on the unaffected left hand, but no P300 for deviant stimuli applied to his affected hand. Standard SEPs in this patient also revealed normal activation of early cortical areas (e.g., SI) for innocuous and painful tactile stimuli, demonstrating intact sensory pathways and preserved inputs to somatosensory cortex. Furthermore, when an healthy control subject was asked to feign anesthesia on one hand, and to intentionally omit to report the infrequent stimuli on that side (i.e., like the conversion patient), a P300 was still normally evoked by these deviants on the pseudo-anesthetized hand, indicating that a reduction of P300 in the patient was not due to malingering or control by voluntary inhibition.

Interestingly, reduced P300 responses to tactile “oddball” stimuli have also been observed in patients who present with a “segmental exclusion syndrome” (Beis et al., 1998). These patients exhibit an abnormally prolonged under-use and pain of their upper limb after suffering a relatively minor injury to peripheral body tissues in one hand or one finger, and such functional exclusion of the limb cannot be explained by the severity of injury. Although this syndrome is different from conversion disorder, it has similarly been considered as a maladaptive deficit with a partly psychogenic origin, and abnormal P300 responses were interpreted as reflecting some kind of attentional inhibition or hemineglect in motor behavior (Beis et al., 1998). Accordingly, anomalies in P300 have been found for undetected stimuli not only in patients with spatial hemineglect after right parietal lobe lesions (Lhermitte et al., 1985), but also in patients with Parkinson’s disease with impairments in intentional motor planning (Kropotov and Ponomarev, 1991; Sohn et al., 1998).

Evoked potentials in other sensory modalities such as vision and audition have been less often studied beyond elementary sensory responses, perhaps because conversion disorders in such modalities are less frequent, more variable, and/or seen by physicians other than neurologists. In patients with functional blindness, a P300 was still evoked by unreported visual stimuli but with smaller amplitude (Towle et al., 1985). By contrast, a study of auditory processing in patients with hysterical deafness reported a reduced P300 response, with preservation of earlier N1 and N2 auditory responses (Fukuda et al., 1996). Some anomalies were also reported in patients with various somatization symptoms for the auditory mismatch negativity (MMN), which is normally evoked by deviant stimuli in a series of repetitive sounds (James et al., 1989). Still other studies reported more general changes in EEG spectra in patients with conversion and somatoform disorders, including abnormal ratios in frequency distribution over right and left frontal lobes (Drake et al., 1988).

Similarly, motor conversion disorders have been investigated by transcranial magnetic stimulation (TMS), typically demonstrating normal and symmetric motor evoked potentials (MEPs) when pulses were applied over the motor cortex, despite the presence of a unilateral hysterical paralysis (Meyer et al., 1992; Magistris et al., 1999). Again, such results are usually taken to indicate that motor pathways are structurally and functionally intact in these patients. Only one recent study found a decreased excitability of motor cortex in right hemisphere of two patients who had a contralateral left hysterical weakness (Foong et al., 1997b), but these cortical excitability thresholds did not change when patients recovered from their weakness. There is also anecdotal evidence that conversion patients may show an abnormal contingent negative variation (CNV) component in EEG, which is normally evoked during motor preparation in response to a cue prior to an expected to-be-judged stimulus (Drake, 1990).

Considered all together, electrophysiological data in hysterical conversion are generally consistent with the absence of organic brain pathology affecting the primary sensory or primary motor systems. Instead, any changes in brain function
associated with conversion might involve higher levels of processing or representations where sensory and/or motor signals are integrated with more complex information related to the meaning and self-relevance of stimuli and actions, (e.g., motivational significance, novelty, expectedness, etc.). This might relate to the reduced P300 response found in a few different studies using different paradigms. However, EEG and MEG investigations still remain remarkably scarce, and reported findings have too rarely been replicated to allow firm conclusions about any putative neural correlates of specific conversion symptoms.

Hemodynamic brain imaging

Over the past 10 years, functional brain imaging has literally exploded into innumerable paths of new research on the cerebral bases of various behavioral functions in humans, including not only perceptual and motor processes accessible to external objective assessment, but also much more complex mental operations related to internal affective states (Damasio et al., 2000), perceptual or motor imagery (Kosslyn et al., 1995; Ehrsson et al., 2003), and even unconscious processing or preferences (Elliott and Dolan, 1998). This functional brain mapping approach has also been successfully extended to a variety of psychiatric conditions such as depression, obsessive-compulsive disorders, phobia, post-traumatic stress disorders, schizophrenia, or hallucinations (e.g., Frith and Dolan, 1998; Parsey and Mann, 2003; Kircher, this volume). Surprisingly, however, very few neuroimaging studies have been performed in patients with conversion symptoms, despite the fact that such symptoms might often be very well suited to neuroimaging investigations.

Most neuroimaging studies of conversion used SPECT (single photon emission computerized tomography) or PET (positron emission tomography), and focused on motor rather than sensory conversion symptoms. These techniques allow only a few brain scans to be taken and provide an estimate of activity averaged over several minutes, indirectly obtained by a measure of cerebral blood flow changes during a resting state or during a task period. The first of such SPECT studies was carried out by Tiwhonen and colleagues (Tiwhonen et al., 1995) in a woman who had a long history of left hemisensory disturbances of presumed hysterical origin, and reported both decreases in right parietal activity and increases in right frontal activity when the affected hand of the patient was stimulated (as compared with a more symmetric pattern after recovery). However, this single observation was more qualitative than truly quantitative, and not statistically analyzed. Similarly, another SPECT study reported a series of five patients with heterogeneous conversion symptoms, including not only limb weakness but also vertigo and gait disturbances (Yazici and Kostakoglou, 1998), in whom brain scans at rest showed a reduction in activity for several cortical regions, predominantly in left parietal and left temporal lobes, but with a great variability across patients.

A more systematic SPECT study was conducted in our own center, in a group of seven patients who were prospectively selected based on the presence of an isolated and “focal” motor conversion disorder, with a recent onset and short duration (< 2 months) (Vuilleumier et al., 2001). Strict selection criteria were used including: unilateral weakness in upper and lower limb, with or without sensory loss in the same territory, but without any other psychogenic or neurological symptoms (such as headache, vertigo, blurred vision, etc.), without any past history of major psychiatric or neurological disease, and without any organic lesion as determined by extensive medical investigations (i.e., brain and spine MRI, SEPs, MEPs, VEPs, EMG, etc.). These patients were followed up for 6 months after the onset of their symptoms, and underwent brain SPECT scanning in three different conditions: (1) a baseline rest condition (To), with eye closed and no stimulation, when motor symptoms were present; (2) a passive activation condition (T1), with bilateral vibrotactile stimuli (50 Hz) applied to both the affected and unaffected limbs simultaneously, when motor symptoms were present; and (3) the same activation condition (T2), again with bilateral vibrotactile stimulation of the affected and unaffected limbs, after motor symptoms had recovered.
(in four patients; three others had persisting or new symptoms at 6 months follow-up). The rationale of vibrotactile stimulation was to provide an indirect activation of both sensory and motor areas in the brain (since such stimuli provide inputs not only to cutaneous but also deep tendon fibers), in a completely passive, symmetric, and reproducible manner. All voxel-based analyses in this study were done on a group basis using statistical parametric mapping (SPM) (Friston et al., 1995).

A first analysis comparing activation by bilateral vibrotactile stimulation to resting baseline (T1 > T0) revealed relatively symmetric increase in cerebral blood flow in frontal and parietal areas involved in somatosensory and motor functions, both ipsilaterally and contralaterally to the motor conversion symptoms (Vuilleumier et al., 2001). This result converges with previous electrophysiological data indicating intact sensorimotor pathways in such patients. A second, more interesting analysis compared activation by bilateral vibrotactile stimulation after recovery relative to the same stimulation during symptoms (T2 > T1), providing a direct measure of changes in brain activity specifically associated with hysterical motor weakness, irrespective of any other distinctive pattern of brain function in these patients (e.g., related to depression, anxiety, or other personality characteristics). This comparison revealed selective decreases in activity of the basal ganglia and thalamus in the hemisphere contralateral to the motor deficit, when the deficit was present as compared with recovery (see Fig. 3A).

Further, the degree of decreases in caudate nucleus and thalamus at the time of symptoms (T1) was significantly correlated with the duration of conversion, i.e., activity was lower in these two regions in patients who did not recover 6 months later, relative to those who subsequently recovered (Fig. 3B). This reduced activation in contralateral basal ganglia-thalamic circuits might therefore provide a plausible substrate for the subjective motor conversion deficits. Finally, we also performed a reverse comparison of vibrotactile stimulation during symptoms relative to recovery (T1 > T2). This showed only mild increases in somatosensory cortex contralateral to the symptoms, again converging with previous electrophysiology results suggesting that sensory processing in these early cortical stages does not seem suppressed (as previously proposed: Ludwig, 1972; Sierra and Berrios, 1999) but instead appears enhanced (Moldofsky and England, 1975; Hoechstetter et al., 2002).

These selective anomalies in subcortical brain regions during motor conversion (Vuilleumier et al., 2001) are intriguing since these regions are interconnected into functional loops forming a cortico-striato-thalamo-cortical circuit which is critical for voluntary motor action (Alexander et al., 1986) and since the striatum (especially caudate nucleus) constitutes an essential neural site within such loops where motivational signals can modulate motor preparation activity (Mogenson et al., 1980; Kawagoe et al., 1998; Haber, 2003; see Fig. 4). It is therefore conceivable that these circuits might become functionally suppressed during hysterical motor conversion under the influence of a particular affective or motivational states, resulting in an impaired “motor readiness” or impaired “motor intention” for the affected limb(s). Notably, in humans, focal lesions (e.g., stroke) affecting the basal ganglia (Watson et al., 1978; Heaton et al., 1982) and thalamus (Laplane et al., 1986; von Giesen et al., 1994) are implicated in a syndrome of unilateral motor neglect, in which patients present with impairments in motor use that cannot be explained by primary weakness but rather reflect a lack of motor intention or planning. Such a loss of motor intention has also been implicated in the failure of some brain-damaged patients to become aware of their (real) paralysis, i.e., anosognosia for hemiplegia (Gold et al., 1994; Vuilleumier, 2000b), suggesting that subjective experience of conscious motor action and volition might be linked to basal ganglia or thalamus function. Also in support of this, direct electric stimulation of lateral thalamic nuclei by depth electrodes may trigger contralateral movements with a subjective experience of voluntary action (Hécaen et al., 1949). Finally, changes in basal ganglia activity have also been implicated in immobilization behaviors exhibited by animals to protect an injured limb (De Ceballos et al., 1986).
Decreased activation during contralateral sensorimotor symptoms relative to recovery (T2 > T1)

(A) Decreased activation in the thalamus, caudate, and putamen (upper row) in the hemisphere contralateral to the limb affected by sensorimotor symptoms. Measures of regional cerebral blood flow (rCBF) were obtained by SPECT scans in seven patients (lower row) during bilateral sensorimotor stimulation by vibrotactile stimuli when their symptoms were present (T1 scan), and in four patients when their symptoms had abated 3–4 months later (T2 scan). Brain activity was decreased in all these subcortical regions in all patients in T1 as compared with the same regions in T2 or with homologous regions in the hemisphere ipsilateral to the symptoms in T1. Such decreases in the thalamus and caudate (but not in the putamen) were more severe in the initial scan (T1) in the three patients who had persisting symptoms at follow-up 4 months later, as compared with four other patients who had recovered, suggesting that the severity of decrease at the time of initial symptoms, may predict the duration of their symptoms. (From Vuilleumier et al., 2001.)

(B) Decreased activation during contralateral symptoms (scan T1) predicting clinical evolution

Symptoms after 4 months:
- Persistent (n= 3 patients)
- Improved (n= 4 patients)
Fig. 4. Schematic illustration of cortico-subcortico-cortical loops. These circuits link various areas in frontal cortex to the caudate nucleus, putamen and pallidum, thalamus, and then back to the cortex, allowing a modulation and coordination of motor commands initiated in the cortex during movement execution, but presumably also during more complex cognitive operations. Such loops provide several neural sites, particularly in the striatum/caudate, where neural signals can be modulated by affective and motivational inputs from many other brain regions (such as orbitofrontal cortex, cingulate cortex, or amygdala), constituting a cerebral system thought to be critical for the integration of volitionally guided and emotionally triggered expressions of behavior.

Other functional changes in brain areas related to voluntary motor control have been suggested by two PET studies in patients with hysterical motor deficits (Marshall et al., 1997; see also Spence, 1999; Spence et al., 2000; for review). Marshall et al. (1997) studied a single patient with a history of unilateral left leg weakness persisting more than 2 years, in a task requiring either to prepare or to execute movements with either the left (affected) or right (unaffected) limb. Whereas motor preparation (without execution) activated brain areas in a relatively symmetric manner for both limbs, motor execution compared to preparation showed a selective activation of motor cortex for the right leg movement only, but not surprisingly, there was no such activation for the subjectively paralyzed left leg. However, in the latter condition, attempts to execute movements in the left leg produced increased activation of ventromedial frontal cortex, including right anterior cingulate and right orbitofrontal cortex, which was not seen during movement execution with the unaffected right leg. It was concluded that during initiation of motor action on the affected side, some signals might be generated in the limbic ventro–medial frontal and cingulate cortex due to affective or motivational factors, and that such signals might actively inhibit the activation of motor cortex, preventing the execution of normal movements (Konishi et al., 1999; Paus, 2001). However, it is possible that this increase in frontal and cingulate activation might reflect other processes known to implicate these regions, such as difficulty, monitoring of failure (Paus et al., 1998; van Veen et al., 2004) or conflict (Dehaene et al., 2003; Badre and Wagner, 2004) due to ambiguous task demands.
(i.e., “try to move even if you cannot”). In any case, a similar pattern (reduced motor activation with increased cingulate cortex activation) was found in a follow-up study examining a single healthy subject who performed the same motor preparation and execution task as in the preceding study, but now after hypnotic suggestion of unilateral paralysis (Halligan et al., 2000). Although this result suggests some similarity between hypnotic suggestion and conversion symptoms, this classic relationship (entertained since Charcot by many others: see Bliss, 1984; Spiegel, 1991; Oakley, 1999) may still not be firmly established (see, e.g., Persinger, 1994; Foong et al., 1997a).

A subsequent PET study therefore compared three patients who had hysterical weakness (two patients in left arm and one patient in right arm), with four healthy control subjects who were instructed to feign motor weakness of the right hand (Spence et al., 2000). All participants had to perform regularly paced movements with a joystick held in their affected (or pseudo-affected) hand. As a group, patients with conversion disorder showed decreased activity in left prefrontal cortex relative to the control feigners; whereas feigners showed decreased activity in right prefrontal cortex relative to conversion patients. The authors suggested that left frontal deactivation in hysteria may reflect the role of these cortical areas in motor planning (Spence, 1999). However, left frontal hypoactivity is also frequently seen in other conditions such as depression (Mayberg, 2003). All patients in this study had a history of depression, although none needed treatment at the time of scanning. Moreover, these left frontal anomalies did not provide a direct functional substrate for the contralateral motor deficit itself, since the affected side differed across the patients. But nevertheless, some impairment in internal representations of voluntary movements is likely to be present in these patients, as also suggested by purely behavioral studies that compared mental motor imagery with the affected and unaffected hands in individuals with motor conversion (Maruff and Velakoulis, 2000; Roelofs et al., 2002).

Finally, only two recent studies used functional magnetic resonance imaging (fMRI) to examine the functional patterns of brain activity associated with hysterical conversion, concerning deficits in somatosensory processing (Mailis-Gagnon et al., 2003) and visual perception (Werring et al., 2004). Mailis-Gagnon et al. (2003) studied four patients with chronic deficits in sensation of touch and/or chronic pain affecting one or more limbs on one or both sides, while they underwent fMRI scanning during blocks of brush and mildly noxious stimulation on their affected and unaffected body parts. Results revealed different patterns for different brain areas. First, unlike stimulation on intact limbs (which were always perceived and reported), both noxious and non-noxious stimuli on the affected limb (which were not perceived or not reported) failed to activate the thalamus, insular, inferior frontal, and posterior cingulate cortices. Second, some areas activated by perceived stimuli on the intact limb were apparently deactivated during stimulation on the affected limb (relative to a resting baseline without any stimulation), including parts of contralateral SI and SII, as well as bilateral prefrontal areas. Third, anterior cingulate cortex showed a greater activation during unperceived/unreported stimulation on the affected limb than during perceived stimulation on the unaffected side. Finally, several regions within prefrontal and parietal cortex (including a superior part of SI) appeared similarly responsive to unperceived and perceived stimuli, although the commonalities of such activations was not formally tested. Moreover, it is unclear whether the deactivation reported for some areas may correspond to inhibitory effects, or be more apparent than real due to greater activation in the baseline condition. In any case, these fMRI findings suggest that abnormal sensory symptoms in these patients might have partly resulted from objective neurobiological changes in a distributed somatosensory network, which the authors tentatively attributed to attentional and emotional processes triggered by mild injuries or painful conditions, and perhaps exacerbated by a preexisting vulnerability (Mailis-Gagnon et al., 2003). The complex results from this study might partly reflect the heterogeneity of patients and the fixed-effect statistical analysis used in a small sample, but it is notable that increased activity in cingulate cortex was also found in the critical condition involving stimulation of the affected limb, as previously found during
an attempt to move the paralyzed limb in motor conversion or hypnosis (Marshall et al., 1997; Halligan et al., 2000). Likewise, a recent fMRI study of five patients with unexplained visual loss (Werring et al., 2004) found reduced activation in visual cortical areas during stimulation by wholefield color flickers, together with decreased (rather than increased) activation in anterior cingulate cortex. On the other hand, increased activity was found in several other regions including more posterior cingulate cortex, insula, temporal poles as well as the thalamus and striatum on both sides. Although different from previous findings for sensorimotor deficits, this pattern was again interpreted as generally consistent with the idea that conversion might involve inhibitory modulation of visual processing through increases in the activity of limbic areas.

Altogether, these new functional neuroimaging data provide compelling evidence that “functional” symptoms in patients with hysterical conversion may at least in part correspond to specific components in brain function, potentially underlying an abnormal awareness of perceptual and/or motor abilities. Here again, there are still too few studies, using different paradigms in different types of patients, such that no definite conclusion is possible. Nevertheless, converging results point to the existence of dynamic changes within several distinct brain areas, affecting activity in motor or sensory systems under the influence of higher order systems, for instance in relation to attention, emotion, or motivation factors. A new challenge for future research will be to provide a more precise mapping between these patterns of brain activation as revealed by neuroimaging techniques and the various psychodynamic dimensions (acute conflict or stress, depression, personality traits, etc.) that may be involved in conversion. In particular, further systematic studies are needed to better tease apart the neural correlates associated with subjective deficits themselves from other aspects potentially associated with coexisting disorders, such as changes in mood or anxiety, personality characteristics, expectations, attention, coping reactions as well as any activity conceivably related to unconscious affective motives and conflicts that were postulated by Freudian psychodynamic accounts.

**More questions and new directions**

A better understanding of functional changes in brain activity during hysterical conversion is certainly important not only because this may yield new insights into neural correlates of subjective experience and awareness, but also because this may provide new constraints on theoretical accounts of conversion, in a neurobiologically plausible framework, and therefore lead to improve the diagnosis and management of patients. There is no doubt (and perhaps no surprise) that an altered experience of bodily functions might be associated with specific modifications in the brain networks normally responsible for generating our conscious experience of such functions, as also demonstrated for even more extreme disturbances in bodily awareness (Vuilleumier et al., 1997; Blanke et al., 2004; Blanke, this volume). Demonstrating specific neural correlates of hysterical conversion may also help reassure the patients as well as their caretakers, including nurses and doctors who sometimes show negative or unsympathetic reactions when confronted with complaints unaccompanied by visible organic pathology. As already suggested by James (1896), this may eventually help convince some skeptics that hysteria is a “real disease, but a mental disease”.

Importantly, neurobiological findings should lead to refine our current psychopathological explanations of conversion, by suggesting possible mechanisms by which the mind can produce changes in the brain and body functions. Such mechanisms are likely to involve dynamic interactions between neural systems mediating specific functions (e.g., motor, somatosensory, or visual processing) and neural systems responsible for affective evaluations and reactions, based on current as well as past experiences (e.g., limbic areas in the broad sense, such as cingulate gyrus, orbitofrontal cortex, or amygdala). Such interactions between distributed brain areas might be usefully investigated by neuroimaging studies using connectivity analysis (e.g., Friston, 1994; McIntosh and Gonzalez-Lima, 1994). Various statistical tools now exist to describe how neural activity in one or several regions can either influence, or instead be contingent upon, neural activity in other regions.
New methods allowing inferences about the directionality of such influences (such as dynamic causal modeling, see Friston et al., 2003) might prove particularly valuable in this context.

Thus, in our own study of hysterical paralysis (Vuilleumier et al., 2001) demonstrating reduced activation in the contralateral basal ganglia-thalamic circuits, which became normal again after recovery from paralysis, we were also able to show that such changes co-varied with concomitant changes in other distant brain areas, where activity was not globally reduced or enhanced but rather appeared differentially coupled with the basal ganglia-thalamic circuits during paralysis. This was demonstrated using a type of principal component analysis (scaled subprofile modeling; Moller and Strother, 1991) that allowed us to identify three distinct networks of regions whose activity tended to covary together across all subjects and all sessions, irrespective of their absolute level of activity. Critically, this analysis identified a network including the caudate and thalamus, together with inferior frontal regions (areas 44/45) and orbitofrontal cortex (area 11), which was specifically coupled during paralysis in the contralateral hemisphere. Second significant network was found to include motor and sensory cortical areas (e.g., areas 1–2–3, 4–6), which were coactivated in all conditions, but less so during paralysis in the contralateral hemisphere; whereas a third significant network included anterior cingulate and temporoparietal areas (areas 39 and 40), and was mainly activated in the ipsilateral hemisphere during paralysis. Although very indirect, this network analysis suggests that hysterical paralysis did not arise in association with reduced activation of the basal ganglia-thalamic circuits alone, but this subcortical reduction was coupled with a distinct pattern of functional connectivity with inferior frontal and orbitofrontal cortex.

One hypothesis to account for this pattern is that changes in basal ganglia-thalamic circuits might implement an inhibition in motor behavior under the influence of affective or stressful signals represented in orbitofrontal or ventral frontal cortex (Mogenson et al., 1980; Kringelbach and Rolls, 2004). Such inhibitory effects might be akin to “behavioral arrest” observed in animals when subjected to threats or forced restraints (Rougeul-Buser et al., 1983; Gray, 1993; Klemm, 2001). Indeed, motor arrest and protective immobility (including behaviors such as “freezing”, “sham death”, or “alert state”) seem to constitute fundamental and stereotyped modes of reactivity to environmental events that are perceived as stressful and hostile, where animals can sometimes adopt awkward fixed postures while waiting for termination of the unfavorable situation (Klemm, 2001). It has already been proposed that such reflexive behaviors might provide a possible primitive mechanism underlying more complex hysterical and “illness” behaviors in humans (e.g., Kretschmer, 1948; Whitlock, 1967). Hysterical conversion is typically triggered by psychological stressors, and sometimes minor physical injuries, which may be experienced as potentially more harmful in the context of a subjective lack of control and helplessness. It is therefore plausible that in some individuals (perhaps due to predisposition or past history) a kind of primitive protection or avoidance mechanisms might be induced and abnormally maintained, resulting in a pathological state of alertness and attention that can modulate their sensory or motor experience, and that is reflected in neural activity within specific brain areas such as cingulate cortex and sensorimotor networks. It is also possible that similar psychobiological responses may induce behavioral changes in other modalities (e.g., blindness, deafness, dysphonia) or in more complex cognitive domains (e.g., memory) depending on some interactions between the causal events and prior experiences in a given individual. In all such cases, these changes in neural activity often found in limbic areas of cingulate or orbitofrontal cortex might indicate a primary “core” dysfunction common to different conversion disorders, inducing secondary functional changes in other brain areas connected more directly with the symptoms. However, more work will be necessary to confirm such changes in different paradigms and to compare them with other conditions such as feigning or hypnosis. Note, however, that many conversion patients present with relatively complex and multiformal symptoms, making homogenous group studies difficult, and unfortunately limiting the
generalization of the findings from one study to another.

Future studies might therefore fruitfully compare patterns of brain activity in conversion patients showing different types of deficits (e.g., motor or somatosensory loss) to determine more directly what are the changes related to specific symptoms, and convergent reactions in general (for instance, testing whether some effects in anterior cingulate and frontal cortex may arise irrespective of the type of deficit). New studies should also use different possible approaches, for instance by examining brain function more systematically during and after conversion deficits in the same individuals; by manipulating more explicitly factors related to attention, expectation or inhibition; and by investigating any differences in brain reactivity to stressful probes presented to these patients even when they have recovered from specific conversion symptoms. In addition, we need to better understand the cerebral mechanisms involved in other psychiatric disturbances characterized by dissociative symptoms, such as psychogenic amnesia or “mnestic block” (Markowitsch, 2003; Glisky et al., 2004), or depersonalization disorders affecting perception of the self and/or of the environment in stressful situations (Lanius et al., 2002; Reinders et al., 2002). New imaging data may help to clarify the possible relationships of these disorders with conversion disorders and other types of psychogenic “block” affecting conscious sensory and motor functions. Interestingly, functional neuroimaging correlates of dissociative responses have also highlighted a key role of medial prefrontal areas for integrating perceptual or motor representations with a subjective sense of conscious control (Lanius et al., 2002; Reinders et al., 2003).

Conclusions

In summary, although hysterical conversion has remained a common and fascinating disorder at the border between neurology and psychiatry, few systematic and controlled studies have yet been conducted using modern neuroimaging tools. This is surprising given the variety of currently available techniques (ERPs, MEG, PET, SPECT, fMRI) and the rich productivity of imaging research in other neuropsychiatric domains. A better understanding of functional changes in brain activity during hysterical conversion will undoubtedly provide unique insights into neural mechanisms of human consciousness, but should also improve the diagnosis, management, and assessment of prognosis in these patients. Finally, further research in this field might certainly contribute to strengthen the links between psychiatry and brain sciences.

Acknowledgments

Many thanks to F. Assal, C. Chicherio, T. Landis, and S. Schwartz for their precious collaboration, and to P. Halligan for many valuable discussions. Part of this work was supported by grants from the Swiss National Foundation.

References


