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Hysterical Conversion *The Reverse of Anosognosia?*

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INTRODUCTION

Hysteria has been the subject of controversy for many years, with theorists arguing about whether it is best explained by a hidden organic cause or by malingering and deception. However, it has been shown that hysterical paralysis cannot be explained in any of these terms. With the recent development of cognitive psychiatry, one may understand psychiatric and organic delusions within the same conceptual framework. Here I contrast hysterical conversion with anosognosia. They are indeed remarkably similar, though the content of their respective delusions is the opposite. In hysterical paralysis, patients are not aware of their preserved ability, whereas in anosognosia for hemiplegia, patients are not aware of their disability. Four main explanations have been provided to account for anosognosia: metacognitive, attentional, motor, and motivational views. I will apply each of these accounts to hysterical paralysis and show that, at each level, hysterical conversion is the reverse of anosognosia. I will suggest that hysterical paralysis results from the interaction between attentional somatosensory amplification and affective inhibition of action.

Effingham felt paralyzed. He could not, as Gerald receded along the lighted corridor, have lifted a finger or uttered a sound....He was paralyzed, like a creature bitten by an insect or a snake, and lying there living, breathing, and waiting to be eaten. (Murdoch, 1963, p. 185)

Murdoch's *The Unicorn* is a story of paralyzed stillness, of people who should act and do nothing, although they want to. Intense fear sometimes makes us freeze

as if we were paralyzed, like Effingham. We know that we have no motor deficit, but we feel that we cannot move an inch. Luckily, this temporary paralysis does not last long. We get over it and we do not conclude that there is something wrong in us just because we feel a rigid coldness in our limbs. Patients with hysterical conversion may feel like Effingham. As much as they might wish to, they cannot move. However, their functional paralysis perseveres and hysterical patients are convinced that it is due to an organic cause despite evidence to the contrary. They are unaware of their preserved ability to move.

When asked to raise your left arm, you can do it; even before you perform the required movement, you know that you can do it. You do indeed know that you are not paralyzed and that you are free to move without constraint. Hysterical patients lack such metacognitive awareness of their abilities. Another type of patient also displays a disruption of the ability awareness: patients with anosognosia. They present the reverse pattern of hysterical patients. Although they are organically paralyzed, they feel that they can move. They are not aware of their disability. But is the basis of the awareness of one's disability the same as the basis of the awareness of one's ability? If so, one would expect the same type of explanation to account for both hysterical conversion and anosognosia. Drawing a parallel with anosognosia, which has been extensively studied, I will shed a new light on hysterical conversion and suggest that Effingham is not so far from being hysterical.

HYSTERICAL PARALYSIS: ORGANIC, FEIGNED, OR DELUSIONAL?

Hysteria has been the subject of controversy for many years. It has even been suggested that it does not exist (Miller, 1999); however, it is not because one finds adequate organic causes that they do not exist. Some patients first diagnosed as hysterical had indeed been later found to suffer with an organic disease (Slater & Glithero, 1965). However, it has been evaluated that only 1–5% of patients diagnosed as being hysterical may present with an underlying occult organic cause (Crimlisk et al., 1998; Stone, Sharpe, Rothwell, & Warlow, 2003). In addition, brain imaging techniques have recently provided an objective assessment of the reality of hysterical symptoms (Spence, Crimlisk, Cope, Ron, & Grasby, 2000). A better understanding of hysteria is thus required—even more so because of the frequency of hysteria in clinical practice (from 1 to 4% of all diagnoses in hospitals in Western countries). Hysteria exists, raising a number of fascinating questions about its origins and mechanism.

A variety of explanations have been provided during the nineteenth century (for review, see Mace, 2001). First conceived as resulting from an increased excitability of the nervous system, hysteria was soon considered to be a functional rather than a structural disorder (Charcot, 1889). Psychological accounts emphasized the triggering role played by emotions (Carter, 1853) or by the unconscious residuum of a “fixed idea” (Janet, 1907). Freud (1894) provided a synthesis of these different dimensions. In hysteria, a psychological trauma, probably of sexual origin, is rendered innocuous by converting it into somatic disorders.

Nowadays, hysteria is referred to as “conversion disorder” in the fourth edition of the American Psychiatric Association’s *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV) and classified under the somatoform disorders. It is characterized by three key features:

1. The patient has symptoms similar to those encountered in physical illness (e.g., paralysis or anesthesia) that cannot be explained by organic neurological lesions or medical diseases.¹
2. The patient experiences these functional disorders as real symptoms over which he or she has no voluntary control. Patients are not malingering.
3. Because the symptoms have no organic cause, they must have psychological causes. They may result from the conversion of psychological trauma.

The difficulties raised by the diagnosis of hysteria are compounded by the fact that symptoms vary from one patient to the other. Here I will limit myself to hysterical paralysis. I will leave open the question of other kinds of hysteria.

Patients with hysterical paralysis cannot produce voluntary movements. They feel paralyzed. However, hysterical paralysis differs from organic paralysis at the neural level and at the behavioral level. At the neural level, motor pathways are intact. When asked to move, contractions of agonistic and antagonistic muscles are found (Merskey, 1995). Hysterical patients also show normal and symmetric motor-evoked potentials (MEPs) when transcranial magnetic stimulation is applied over the motor cortex (Magistris, Rosler, Truffert, Landis, & Hess, 1999; Meyer et al., 1992).

At the behavioral level, hysterical patients do not display the same motor deficits as neurological patients. According to Freud, the symptoms in hysterical paralysis are limited to restricted parts of the body and they are characterized by excessive intensity. Furthermore, hysterical motor behaviors suffer from a lack of consistency. For example, patients with hysterical aphonia may be unable to whisper, but able to cough (Spence, 2001). The degree of disability varies depending on the social context and the patient’s emotional state. One can even pharmacologically induce temporary remission: A patient with hysterical quadriplegia showed restored movements under diazepam (Ellis & Young, 1990). Patients may also produce normal movements when asleep. In addition, when applying Hoover’s test, the “paralyzed limb” moves when the contralateral (“healthy”) limb is flexed and the patient is distracted (Sonoo, 2004). It has thus been suggested that the dysfunction is limited to voluntary movements and does not affect automatic reflex movements (Athwal, Halligan, Fink, Marshall, & Frackowiak, 2001; Spence, 1999).

One may therefore wonder whether patients are not feigning their paralysis because of the secondary gain that they would obtain for being ill. Individuals who feign paralysis may indeed display the same inconsistency of performance. However, it has been shown that hysterical paralysis does not activate the brain areas that are activated by feigning motor weakness (Spence et al., 2000). Three patients with hysterical weakness were compared with four healthy control subjects who were instructed to feign motor weakness. All participants had to perform movements with 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; feigners showed decreased activity in right prefrontal cortex relative to conversion patients.

Hysterical paralysis cannot be confused with organic paralysis or with feigned paralysis. Rather, one may interpret it as a case of *delusional paralysis*. Hysterical patients would have the delusion that they are paralyzed—in the same way that anosognosic patients have the delusion that they are not paralyzed (Davies, Aimola Davies, & Coltheart, 2005). What exactly would be the content of their delusion? Hysterical patients believe that they cannot make any movement due to an organic cause. Hysteria differs from anosognosia in that part of the hysterical patients' beliefs is true. It is true that they cannot make voluntary movements. However, it is false that they cannot make *any* movement and it is false that it is due to an organic cause. This ambivalence explains why it is difficult to classify hysterical conversion as a pure case of delusion. Delusions are defined as false beliefs firmly sustained despite obvious proof or evidence to the contrary (DSM-IV). They are associated with a strong feeling of conviction and are often characterized by a lack of appropriate affect and a lack of influence on practical reasoning.

Just based on these two latter criteria, one can see that hysterical paralysis displays heterogeneous features. On the one hand, it is true that hysteria is often associated with a lack of emotional concern relative to the symptoms (i.e., “la belle indifférence”). On the other hand, patients act in the ways in which they ought to act if they believed their delusion. They go to hospital to be diagnosed and cured. However, they do not believe physicians when they find no organic cause. They maintain their belief against what almost everyone else believes. Their experience of their inability to move overwhelms all the scientific medical evidence that can be provided. They are convinced that there must be a physical cause that explains their paralysis. Therefore, they have a false belief, and as such they can be considered as delusional. However, their delusion is partly—but not fully—justified. They have good reasons to believe that they are physically paralyzed because de facto they cannot move. Their paralysis is, however, limited to voluntary movements and it cannot fully justify their belief in a complete organic paralysis. I will come back later to the justification of their belief.

HYSTERIA AND ANOSOGNOSIA

The inconsistency of motor behavior and the differences in brain activity have shown that hysterical paralysis cannot be explained by a hidden organic cause or by a tentative of malingering and deception from the patient. It needs its own account. For a long time, it has been the privilege of psychoanalytic theory. However, with the recent development of cognitive psychiatry (David & Halligan, 1996), one may understand psychiatric and organic delusions within the same conceptual framework (Davies et al., 2005; Frith, 1992).

Here I will contrast hysterical conversion with anosognosia.² They are indeed remarkably similar, though the content of their respective delusions is the opposite. In both cases, patients do not move. However, in hysterical conversion, patients are not aware of their preserved ability, whereas in anosognosia, patients are not

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aware of their disability. The dissociation between ability and awareness concerns the same domains in both syndromes: anesthesia, amnesia, aphasia, blindness, and paralysis. It is also interesting to note that there is evidence that both syndromes affect the left limbs more than the right limbs (Gagliese, Schiff, & Taylor, 1995; Galin, Diamond, & Braff, 1977; Pascuzzi, 1994; Stern, 1983), although a recent meta-analysis has questioned the existence of such an asymmetry in hysteria (Stone et al., 2002). Finally, both types of patients display an attitude of “*belle indifférence*.” Their emotional attitude toward their handicap is not appropriate. In light of these considerations, it seems reasonable to consider anosognosia as a model of understanding of hysterical conversion. As with hysteria, I will focus on anosognosia for hemiplegia.

These last 20 years, there has been an extended literature on anosognosia. Patients who suffer severe deficits following brain damage remain unaware of their handicap and deny it. It was first described by Babinski (1914) to denote the loss of recognition of a hemiplegia. Nowadays, it is encountered in at least 20–30% of hemiplegics after an acute stroke (Stone, Halligan, & Greenwood, 1993). Although the underlying mechanisms of anosognosia are not yet fully understood, several accounts have been provided to explain how one can remain unaware of one’s handicap. Four main explanations have been provided to account for this disorder:

- Metacognitive account: Anosognosia results from the failure to inferentially discover that one is hemiplegic (Levine, Calvanio, & Rinn, 1991; Ramachandran, 1995).
- Attentional account: Anosognosia results from the lack of attention towards one’s own body (Cutting, 1978).
- Motor account: Anosognosia results from the disruption of action monitoring (Heilman, 1991).
- Motivational account: Anosognosia results from a psychologically motivated denial—an unconscious defense mechanism that attenuates the potential distress of hemiplegia (Weinstein & Kahn, 1955).

These explanations are not mutually exclusive. Given the variety of cases in anosognosia, one can also expect a variety of explanations, and each case can involve several dysfunctions (Davies et al., 2005). Here I do not intend to review them in detail or to take a side in the debate about the necessary and sufficient conditions for anosognosia. Instead, I will analyze each account as it might apply to hysterical paralysis.

Beforehand, it is important to notice one main difference between anosognosia and hysteria. One can define anosognosia uniquely in terms of deficit (i.e., a deficit of awareness of hemiplegia). In contrast, hysteria is characterized not only by negative symptoms like hemiplegia, but also by positive symptoms like gait disturbance and tremor. Most cognitive models focus on negative symptoms, and I will follow their lead here. It is less clear, however, how positive symptoms can be accommodated by this neuropsychological approach. One should keep this in mind, even if one has no satisfactory account of positive symptoms.

THE METACOGNITIVE LEVEL

According to Levine et al. (1991), sensorimotor deficits are not phenomenologically salient and need to be discovered. In other words, one assumes that one is healthy, unless one is provided with evidence to the contrary. The default hypothesis is that one is not paralyzed. One needs to monitor one's performance reflectively to detect anomalies. The presence of an anomaly provides no immediate awareness that one cannot move, and one will persevere in believing that one can move until one discovers by self-observation that this is not the case. Ramachandran (1995) compares this discovery to a change of paradigm.

According to the metacognitive view, patients suffer from anosognosia because they have difficulties in discovering their deficit. This discovery is all the more difficult for them in the case of anosognosia for hemiplegia because hemiplegia is often associated with neglect, as we will see, and they can draw incorrect inferences about the affected side on the basis of the healthy side. Consequently, anosognosic patients do not switch to a new paradigm, although they have all the reasons to do so. What about hysterical paralysis? The reverse happens. Hysterical patients switch to a new paradigm, although they have no reason to do so. In hysterical conversion, the default rule is true. There is nothing to discover. Yet, patients do not believe the default rule.

There are at least two possible interpretations of this surprising change of paradigm. First, patients cannot move. They do have a reason to give up the default rule. It makes sense for them to believe that they are paralyzed. This first interpretation does not explain why hysterical patients cannot move, but only why they believe that they cannot move. In contrast, the second interpretation is less modest and may help to account for the paralysis *per se*. One possibility is that self-monitoring is disturbed. Patients draw incorrect inferences based on what they observe. They can move, but they do not realize that they can move. Because they believe that they cannot move, they do not intend to move and they do not move. The change of paradigm is then self-justifying. Although not justified at the beginning, it is justified afterwards. Put another way, the change of paradigm is either the consequence of a motor deficit or its cause. To settle the debate between these alternatives, one needs first to review the different possible explanations of hysterical paralysis at the attentional, motor, and motivational levels.

THE ATTENTIONAL LEVEL

One reason that could explain why both anosognosic patients and hysterical patients do not discover their respective state is that they do not get much information from their body due to sensory loss and/or attentional deficit. One consequence of the metacognitive view is indeed that the anomaly does not pop up in awareness. It is not always salient enough to attract attention, particularly if the sensory signal is weak. Therefore, one needs to observe one's performance, and if one does not, one will not form the correct beliefs about abilities and disabilities.

In support of the attentional view, it has been shown that anosognosia often co-occurs with sensory loss and unilateral neglect. Interestingly, both neglect and

anosognosia are momentarily attenuated during vestibular stimulation (Cappa, Sterzi, Vallar, & Bisiach, 1987). In personal neglect, patients do not pay attention to the contralateral side of their body. For example, they may comb their hair only on one side. They may also not notice that half of their body is not moving. One may thus suggest that neglect contributes to the lack of awareness of paralysis in anosognosia. As for hysterical paralysis, it has often been compared with motor neglect (Vuilleumier et al., 2001). In motor neglect, patients “forget” to use the contralateral side of their body. For example, they may try to perform bimanual tasks by using their right hands only. Consequently, the left limbs never move because they are neglected. One may thus suggest that neglect contributes to the lack of awareness of the preserved ability to move in hysteria.

According to this view, hysterical conversion is due to an attentional dysfunction resulting from an increase in inhibition of afferent stimulation, preventing the ongoing sensory or motor activity from entering awareness (Ludwig, 1972, Oakley, 1999; Sierra & Berrios, 2001). Hysterical patients have indeed been shown to be impaired in a series of attentional tasks (Bendefeldt, Miller, & Ludwig, 1976). More recently, a disruption of the evoked potential P300 associated with attentional processing has been discovered in hysterical patients (Lorenz, Kunze, & Bromm, 1998). Subjects were repeatedly stimulated on the left hand and occasionally received a “deviant” stimulus either on the right hand or on another finger on the left hand. The deviant stimulus elicited a P300 response for both hands in control subjects. However, a patient with hysterical anesthesia on the right hand showed no P300 response when the stimulus was applied on the right affected hand. The patient was not able to direct his attention to the novel stimulus when it was applied on the hysterical side.

However, the role of attention is controversial for both syndromes. On the one hand, Bisiach, Vallar, Perani, Papagno, and Berti (1986) showed double dissociation between neglect and anosognosia. Neglect is not a necessary condition for the unawareness of hemiplegia. On the other hand, in hysterical conversion, several results show that there is no deprivation of information related to the body, in fact, quite the reverse. First, far from a somatosensory deficit, a preserved activity has been found in somatosensory primary areas SI and SII, which is even slightly increased in comparison with the unaffected side (Hochstetter, Meinck, Henningsen, Scherg, & Rupp, 2002). As for the attentional deficit, clinical reports reject it, emphasizing, on the contrary, the therapeutic importance of distracting the patient’s attention from his affected limb. As previously said, patients move their paralyzed limb when sedated or distracted (Spence, 1999). The principle underlying Hoover’s test is to direct the patient’s attention away from the affected limb: “The only necessary technique is to urge the patient to concentrate on the abducted [‘healthy’] leg and pay no attention to the unabdacted [‘paralyzed’] leg” (Sonoo, 2004). This technique is consistent with studies showing that patients display an attentional bias toward their body.

In normal subjects, responses in somatosensory evoked potential and in skin conductance decrease over time after repeated identical stimulations. But in hysterical patients, there is no habituation effect (Horvath, Friedman, & Meares, 1980; Moldofsky & England, 1975). They pay as much attention at the end of the

sequence as at the beginning. In addition, patients with somatoform disorders, such as hysteria, have a biased attention towards the tactile modality immediately following exposure to threatening body-relevant information (Brown, Poliakoff, & Kirkman, 2007). More controversially, it has also been found that patients with somatoform dissociations have higher scores on the SomatoSensory Amplification Scale (SSAS), which includes statements like the following (Barsky, Wyshak, & Klerman, 1990):

1. Sudden, loud noises really disturb me.
2. I am very uncomfortable when I am in a place that is too hot or too cold.
3. I cannot stand pain as well as most people can.
4. I find I am often aware of various things happening in my body.
5. I am quick to sense the hunger contractions in my stomach.

To sum up, hysterical paralysis may result from an attentional disturbance, but the disturbance appears to be one of overattention, rather than underattention, to the body. In this proposal, patients would spend disproportionate amounts of time focusing on their bodies and scanning for evidence of disease, increasing the likelihood of finding evidence of bodily dysfunction (Brown, 2007).³ Whereas anosognosic patients might be compared to lazy scientists who do not discover what is under their noses, hysterical patients are neurotic scientists who constantly look for evidence that is not there.

THE MOTOR LEVEL

We have seen that attentional deficit cannot fully account for anosognosia. As an alternative explanation, it has been suggested that the origin of anosognosia for hemiplegia is a disruption of the motor system (Adair et al., 1997; Berti, *Spinazzola, Pia, & Rabuffetti*, 2007; Gold, Adair, Jacobs, & Heilman, 1994, Heilman, 1991). According to Heilman (1991), the main proponent of this view, anosognosic patients have a deficit of intention. Similarly, since the nineteenth century, it has been defended to understand hysterical paralysis in terms of a “disorder of the will” (Spence, 2001): “It is not the muscles which refuse to obey the will, but the will itself which has ceased to work” (Brodie, 1837); according to Paget (1873), “They say, ‘I cannot; it looks like ‘I will not’; but it is ‘I cannot will.’”

Both anosognosia and hysteria may be due to a lack of intention. But both types of patients report that they consciously intend to move. Are they lying or mistaken? Not necessarily. It is important here to distinguish between two kinds of intention (Pacherie, 2000; Searle, 1983). *Prior intentions* represent the goal of the action as a global unit (e.g., “I intend to drink”). They are too rough grained to specify the movements that have to be performed. The motor system needs to anchor the prior intention in a specific context and to determine the means that will be required to reach the goal—that is, the *intention in action*. Intentions in action represent the action as a dynamic sequence of specific movements (e.g., “I intend to reach the tap and to turn it clockwise with my right hand”). It initiates the action, guides it, and monitors its effects. According to the motor view, it is

only the intention in action that would be impaired, although this claim is rarely made explicit (Spence, 2001).

Several experimental results argue in favor of a deficit of intention in action for both anosognosia and hysteria. In anosognosia, it was found that a patient showed no activation of the pectoral muscles when asked to squeeze his left paralyzed hand, whereas he showed normal bilateral muscle activation when squeezing his right hand (Gold et al., 1994). Interestingly, the authors also tested hemiplegic patients *without* anosognosia, who showed normal bilateral activation for both hands. This was taken as evidence that the anosognosic patient was not even trying to squeeze his hand. In hysterical paralysis, patients showed a hypoactivation of the dorsolateral prefrontal cortex when asked to move, which prompted some to postulate a dysfunction of internal movement initiation (Roelofs, van Galen, Keijsers, & Hoogduin, 2002; Roelofs, de Bruijn, & van Galen, 2006; Spence, 1999; Spence et al., 2000). However, these results have not been replicable, and it is possible to question their interpretation.

For anosognosia, Berti et al. (2007) could not find abnormal muscle activation in her patients. As for hysteria, Marshall, Halligan, Fink, Wade, and Frackowiak (1997) and Vuilleumier et al. (2001) found similar activation during action preparation and initiation in hysterical patients and healthy controls. They did not find hypoactivation of the dorsolateral prefrontal cortex in patients. Furthermore, Vuilleumier (2005) noted that this hypoactivation might be related to depression, which is often associated with hysteria.

Here I do not intend to settle this debate, but rather to understand how an intentional deficit could lead either to anosognosia or to hysterical paralysis. What is surprising is that the very same deficit might be thought to induce delusions with opposite contents (i.e., “I can move” vs. “I cannot move”). Let us see first how it might work for anosognosia. The theoretical framework of the motor system posits two internal models (Wolpert, Ghahramani, & Jordan, 1995). The inverse model simulates the execution of action, and the forward model anticipates the sensory feedback. When performing a movement, the expected state is congruent with the sensory feedback, indicating that I am moving as intended. If there is no intention, as in anosognosia, the motor system does not expect any sensory feedback indicating a movement and therefore does not signal that there is a discrepancy between the intention and the outcome. Consequently, the lack of movement does not challenge the default hypothesis (i.e., “I can move”).

What about hysterical paralysis? How can it be explained by a comparable lack of intention and a comparable lack of incongruence? The default hypothesis is the same: I can move. The outcome of the comparators does not convey any odd signal; the absence of movement is consistent with the lack of intention to move. Yet, the patient concludes that he cannot move. It is difficult to understand how one can reach such a conclusion on this basis. The content of hysterical delusions cannot be explained by an intentional deficit.

It is interesting to note here an asymmetry between ability awareness and disability awareness. I can conclude that I can move from the fact that I am moving. However, the reverse is not true: I cannot conclude that I am moving from the fact that I can move. Similarly, I can conclude that I am not moving from the fact that

I cannot move, but I cannot conclude that I cannot move from the fact that I am not moving. The logical link between action awareness and ability awareness is not bidirectional. The direction of the arrow changes for abilities and disabilities. The lack of action awareness does not suffice to conclude that I am paralyzed. I also need to be aware that I intend to move. It is only the discrepancy between my intention to move and the absence of movement that entitles me to conclude that I am paralyzed (or that there are external constraints preventing me from moving). A deficit of intention, as assumed by Spence (2001), prevents such justification for hysterical delusions without providing any other. It makes sense for anosognosia, but not for hysteria.

The problem may be, however, more downstream in the motor system, when the motor command is dispatched to the body. When comparing hysterical patients and healthy controls, different brain activations during action execution were found (Marshall et al., 1997; Vuilleumier et al., 2001). There was a hypoactivation of executive motor areas in conjunction with increased activation of frontal and cingulate areas in hysterical patients. Hysterical paralysis may result from an inhibition of movement execution, rather than from a dysfunction of movement initiation. According to this explanation, patients with hysterical paralysis have the intention to move. The motor system predicts the sensory outcome of the movement, which is compared with the sensory feedback indicating the absence of any movement. There is a discrepancy ("I am not moving although I intended to move") that challenges the default hypothesis. I am justified to conclude that I cannot move. The motor disruption is upstream in anosognosia with a deficit of intention, and it is more downstream in hysteria with a deficit of execution.

To sum up, whereas anosognosia can be understood in terms of a deficit of intention, this cannot account for hysterical paralysis. But perhaps hysterical paralysis can be understood in terms of inhibition of intention execution. The disruption of execution explains why patients feel paralyzed, but one still needs to explain why execution is inhibited. One needs to go a step backward and understand the origin of the inhibition to give a full account of hysterical paralysis.

THE MOTIVATIONAL LEVEL

There are at least two classes of explanation of delusion: motivational theories, coming from the psychodynamic tradition, and deficit theories, coming from cognitive neuropsychiatry (McKay, Langdon, & Coltheart, in press). Motivational theories understand delusions as a mechanism of defense to relieve pain, tension, and anxiety. Deficit theories understand delusions as the result of abnormal perceptual experiences and cognitive deficits. The former provides a positive conception of delusions (by the emotional benefits they confer), whereas the latter provides a negative conception (as the side effect of the combination of deficits).

This debate is illustrated in anosognosia: As we have seen, anosognosia can be understood in terms of intentional and/or attentional deficits, but it has also been suggested that it results from the drive to be well and the preservation of self-esteem (Weinstein & Kahn, 1950; for review, see Aimola Davies et al., this volume). This latter hypothesis has been criticized by Bisiach and Gemiani (1991),

who showed that several characteristics of anosognosia could not be explained by motivational factors. Hysterical conversion has also been explained by motivational factors like secondary gain (e.g., avoiding military service). However, Freud himself, who is at the origin of the notion of secondary gain, did not believe that it could fully account for hysterical paralysis. Here I would like to provide an alternative explanation of hysterical conversion that takes into account affective factors, without falling back to the secondary gain theory.

Interestingly, the debate between motivational and deficit theories has been until very recently a question of all or nothing. It was either all motivational or all dysfunctional. There was no middle ground. However, they are not incompatible. Both can play a role in explaining the emergence and the maintenance of delusions (Aimola Davies et al., this volume; McKay et al., in press). Motives can explain the credibility and saliency of delusional beliefs. They can also explain why patients prefer keeping their false beliefs, although they are rationally untenable. The fact that they play a role, however, does not imply that they are a sufficient condition. According to this new theoretical trend, motives and deficits work hand in hand, but they do not interact with each other. Anosognosia can be understood as the summation of deficits of attention, intention, and memory plus voluntary denial to defend oneself against depressive overwhelm. However, there is no interaction between both types of factors. The treatment of evidence may be motivationally biased, but deficits are not motivationally triggered. In contrast, I would like to suggest here that motivational and affective factors are at the origin of motor deficits.

Inhibition can be understood via a dual process of regulatory control—that is, executive inhibition and motivational inhibition (Gray, 1982; Shallice & Burgess, 1993). Executive inhibition refers to deliberate suppression of motor behavior because of the context or competing goals. This inhibition is accompanied with relatively low anxiety activation. It involves the same frontal–striatal–thalamic neural loops as executive function. Motivational inhibition refers to anxiety-provoked interruption of behavior in the context of emotionally salient incentive cues. It invokes the activity of the limbic system and anterior cingulate cortex. This system detects and responds to immediate contextual cues for punishment, unexpected “mismatch,” or social unfamiliarity (Nigg, 2003).

We said that hysterical paralysis results from inhibition of execution, but what type of inhibition—executive or motivational? Brain imaging studies might help us to decide. Hysterical patients display hyperactivity of the limbic system and of the anterior cingulate cortex (Roelofs et al., 2006; Vuilleumier et al., 2001). The authors conclude that during action initiation, some signals are generated in the limbic and cingulate cortex due to affective and motivational factors that actively inhibit the motor cortex, preventing the execution of normal movements. Consequently, to explain the motor deficit in hysterical paralysis, one needs to take into account motivational factors. However, this does not mean that we are back to the secondary gain theory of hysteria. The motivations inducing the inhibition are of a completely different kind. Instead, it has been suggested to compare hysterical paralysis with stereotyped modes of reactivity to environmental events that are perceived as stressful and hostile (Kretschmer, 1948; Vuilleumier, 2005; Whitlock, 1967).

In recent years, ethologists working with nonhuman primates have established four distinct fear responses that proceed sequentially in reaction to increasing threat: freeze, flight, fight, and fright (Bracha, 2004; Gray, 1982). The initial freeze response is the “stop, look, and listen” response associated with fear. Hofer (1970) exposed rodents to a variety of predator-related stimuli in an open space with no means of escape. All rodents entered a deep phase of freeze, persisting for up to 30 minutes. The freeze response can constitute a survival advantage in some cases. Prey that remains “frozen” during a threat is more likely to avoid detection because the visual cortex and the retina of mammalian carnivores primarily detect moving objects rather than color. The next response is to flee; if that does not work, the following response is to fight. The last step in the sequence of fear-circuitry responses after fighting is tonic immobility, or “playing dead” (also referred to as fright). This response occurs during direct physical contact with the carnivore. Tonic immobility may enhance survival when a predator temporarily loosens its grip on captured prey under the assumption that it is indeed dead.

From the outside, hysterical paralysis looks like freeze response and tonic immobility, both characterized by motor arrest and protective immobility, like Effingham in *The Unicorn*. A basic function of the motor system of all animals is indeed to protect the body from attack and collision (Cooke & Graziano, 2003; Dosey & Meisels 1969; Schiff, Caviness, & Gibson, 1962). Animals can thus sometimes adopt awkward fixed postures while waiting for termination of the unfavorable situation (Klemm, 2001). It is interesting to note that the reactive response does not stop as soon as the threat stops. Animals exposed to significant shock stimuli in an escape-proof environment freeze with subsequent shock exposure. Subsequent introduction of routes of escape in these animals does not elicit escape behavior. The animals remain frozen and continue to exhibit helplessness (Seligman, 1975).

In addition, there does not need to be repetitive shock stimuli to establish the conditioned freeze response. In an event of great arousal and threat, only one trial may be enough (Scaer, 2001). In traumatized patients, it has been shown that any stimulus occurring in the peripersonal space where the person first experienced the threat (e.g., the approaching car) is conceived as threatening. As a result, passing a hand around the periphery of the patient’s visual field at the distance of 3–4 feet will often produce an arousal response in the region of perception of prior threat, perpetuating the kindled trauma reflex (Scaer, 2001).

On the basis of these findings, it has been suggested that hysterical paralysis takes root in the primary stereotypical response to danger—that is, the freeze response (Vuilleumier, 2005).⁴ The lack of recovery from the freezing, also encountered in animals, is explained by long-term conditioning, which can be elicited by a single shock of great intensity. It is not surprising, therefore, that ambient stress often enhances the symptoms of hysterical conversion.⁵

HOW TO BECOME HYSTERICAL

Two things need to be explained in delusions: (1) how the false belief is accepted as a serious and credible proposition, and (2) how the false belief is maintained despite evidence to the contrary. Two-factor theories of delusions have suggested

that these two things demand independent explanations: The first factor triggers an initial implausible thought (and thus contributes towards explaining the thematic content of a particular delusion), and the second factor explains the uncritical adoption and maintenance of an implausible thought as a delusional belief (Davies, Coltheart, Langdon, & Breen, 2001; Langdon & Coltheart, 2000). To provide a full account of hysterical paralysis, one needs therefore to specify both first and second factors. We have examined different disruptions that explain the etiology of the hysterical delusion. Let me recapitulate and articulate them. This would help us to understand the first factors.

Your attention is focused on your own body. You feel external stimuli as constant threatening intrusions in your personal space. This elicits a high level of anxiety and automatic defensive reflexes to protect you from these external stimuli. This in turn inhibits your motor system. You cannot move anymore, although you intend to move (and you are aware that you intend to move). Based on the discrepancy between your intentions and the absence of movements, you conclude that you are paralyzed. When you are distracted from your own body, you do not experience anxiety due to the danger threatening your body and, as a result, you are able to move.

In this model, can we still consider hysterical patients as delusional? Disturbances of the attentional system and the motor system, due partly to affective factors, induce the inability to move. To conclude that one cannot move is therefore not a rationally untenable belief. It is credible and one does not need to appeal to further reasoning biases to explain it. The evidence is there: Patients cannot make some movements. However, not only do they believe that they are unable to move now, but they also believe that they have a long-lasting paralysis due to an organic cause and that they cannot make *any* movement. In contrast, when we freeze in front of a danger, like Effingham, we do not believe that we have an enduring clinical condition.

Hysterical patients therefore make two kinds of errors: first, about the extent of their paralysis—they overgeneralize based on restricted evidence and, second, about the source of their paralysis—they provide a fully unjustified account of the origin of their paralysis that goes against all the clinical evidence. Both components of their delusional beliefs arise as normal responses to the unusual experience of their inability to move (Maher, 1999). But why do patients maintain their belief that they suffer from a complete organic paralysis?

In the two-factor model, the answer to this question is provided by second factors. In anosognosia, the nature of the second factor differs from the nature of the first factor (Davies et al., 2005). It corresponds to a deficit in the cognitive mechanisms responsible for belief evaluation and revision, linked to working memory deficit. What about hysterical delusion? Hysterical patients do not reject their paralysis belief when clinical exams and diagnosis reveal the absence of any organic damage. Furthermore, their awareness of their ability to make automatic reflex movements should undermine their conclusion that they cannot move, but it does not; the delusion remains intact. Can this be explained on the sole basis of the first factors (i.e., somatosensory amplification combined with affective inhibition

of action)? Or is there a need for a second factor of a different kind? What are the conditions of maintenance of the hysterical delusion?

The main evidence available to the patients that they are not paralyzed is that they can sometimes move. How do patients fail to realize that they are not paralyzed when they do see their limb stretching, as in Hoover's test? It has been shown recently that patients have abnormal brain activations also when observing movements (Burgmer et al., 2006). This deficit is not surprising according to the mirror neuron hypothesis (Rizzolatti, Fadiga, Gallese, & Fogassi, 1995). If action execution and action observation activate the same brain areas, the deficit of the former must have consequences for the latter.

Given the observation deficit, one may wonder what happens when the patient sees herself moving. One possibility is that she does not perceive the observed movement qua movement of her own body. The movement might be represented in the same way as the movement of a robot or the movement of the clouds in the sky. It would not be considered as a valid input for the motor system. The patient would not be aware of moving and therefore would not conclude that she can move. This is highly speculative, but it may explain why patients' delusions are not disturbed by their automatic movements. Alternatively, we have seen that patients can move when they are distracted. They may therefore not notice their own movements because of their lack of attention.

A further difference between Effingham and a hysterical patient is that Effingham recovers very quickly and starts moving again. In contrast, the affective inhibition of action is constantly reactivated in the patient. Brown (2002, 2006) provides a model that explains the maintenance of medically unexplained symptoms by appealing only to primary factors. Because of somatosensory amplification, patients constantly check whether the symptom is still present. In addition, they engage in a range of behaviors directed at bringing about symptom relief or reducing negative affect.

These illness behaviors and the anxiety induced by the symptoms increase attention to the body. The high level of body-focused attention in turn increases the activation of the representation of the symptom stored in memory and the likelihood of subsequent reselection, setting up a vicious cycle. In the case of hysterical paralysis, the anxiety brought about by somatosensory amplification induces action inhibition. Hysterical patients are not like Effingham; they keep feeling threatened and therefore stay frozen. There is no need for a further explanation of why they maintain the belief that they are paralyzed. If, indeed, one remains unable to move for a long time, one is entitled to conclude that one is paralyzed. The maintenance of the inhibition suffices to account for the maintenance of the delusion. There is no need for a second, more cognitive factor, in contrast with anosognosia.

CONCLUSION

Hysterical paralysis is the reverse of anosognosia. In the latter, patients do not feel paralyzed, although they are physically unable to move; in the former, they feel paralyzed although they are physically able to move. At the metacognitive level,

anosognosia arises from a lack of change of paradigm, whereas hysteria arises from an unexpected change of paradigm. At the attentional level, anosognosia results from a deficit of attention toward the body; hysteria results from too much attention toward the body. At the motor level, anosognosia is caused by an upstream deficit of intention, whereas hysteria is caused by a more downstream deficit of execution inhibition. At the motivational level, anosognosia is due to the high-level drive to be well enhancing the credibility and the saliency of the delusional belief, but hysteria is due to low-level, anxiety-driven defensive responses inhibiting the motor system. Finally, anosognosic delusions require second factors to account for their maintenance, whereas hysterical delusions are justified beliefs and do not need such additional factors.

This account can explain hysterical paralysis, but does not pretend to explain other kinds of hysterical conversion, such as hysterical blindness. However, one may be able to shed a new light on this latter phenomenon by drawing the same kind of parallel, with Anton's syndrome this time (i.e., visual anosognosia).

ACKNOWLEDGMENT

I would like to thank Tim Bayne for his very helpful comments.

NOTES

1. Hysterical conversion may sometimes coexist with a real organic brain disease, but the lesion cannot explain the specific symptoms that are displayed. For instance, in a rehabilitation department, one third of the neurological patients exhibited at least one "hysteria-like" behavior in addition of their neurological symptoms (Eames, 1992).
2. Alternatively, one may compare hysterical conversion with hypnosis (McConkey, 2001; Oakley, 1999).
3. Somatosensory amplification may account for the dissociation between voluntary and automatic movements. It would be only when patients consciously intend to move that they would pay too much attention to their body. The somatosensory amplification would then result in the inability to move. During automatic movements, in contrast, one scarcely pays attention to one's body.
4. According to this view, inhibition of execution is merely an automatic reflex. In what sense then is it influenced by motivational factors? The underlying motive of the freeze response is to protect one's own body. The freeze response has been selected by evolution for this purpose. The brain circuitry of action inhibition involved in hysterical paralysis is part of the motivational loop. But the inhibition is not under the patient's voluntary control. One could claim that the evolutionary basis of hysterical paralysis is motivational, but not hysterical paralysis per se. Alternatively, one could emphasize that hysterical symptoms depend on the level of anxiety and thus that they directly result from the emotional state.
5. Interestingly, this explanation can account not only for negative symptoms of hysterical paralysis (e.g., inhibition of execution), but also for its positive symptoms (e.g., tremor).

REFERENCES

- Adair, J. C., Schwartz, R. L., Na, D. L., Fennell, E., Gilmore, R. L., & Heilman, K. M. (1997). Anosognosia: Examining the disconnection hypothesis. *Journal of Neurology, Neurosurgery, Psychiatry*, 63, 798–800.
- Aimola Davies, A. M., Davies, M., Ogden, J. A., Smithson, M., White, R. C. (this volume).
- Athwal, B. S., Halligan, P. W., Fink, G. R., Marshall, J. C., & Frackowiak, R. S. J. (2001). Imaging hysterical paralysis. In P. W. Halligan, C. Bass, & A. S. David (Eds.), *Contemporary approaches to the study of hysteria* (pp. 216–234). New York: Oxford University Press.
- Babinski, J. (1914). Contribution à l'étude des troubles mentaux dans l'hémiplégie organique (anosognosie). *Revue Neurologique*, 27, 845–848.
- Barsky, A. J., Wyshak, G., & Klerman, G. L. (1990). The Somatosensory Amplification Scale and its relationship to hypochondriasis. *Journal of Psychiatric Research*, 24, 323–334.
- Bayne, T., & Pacherie, E. (2005). In defense of the doxastic conception of delusions. *Mind and Language*, 20(2), 163–188. AU: not cited in text; OK to delete?
- Bendefeldt, F., Miller, L. L., & Ludwig, A. M. (1976). Cognitive performance in conversion hysteria. *Archives of General Psychiatry*, 33(10), 1250–1254.
- Berti, A., Spinazzola, L., Pia, L., & Rabuffetti, M. (2007). Motor awareness and motor intention in anosognosia for hemiplegia. In P. Haggard, Y. Rossetti, & M. Kawato (Eds.), *Sensorimotor foundations of higher cognition*. Oxford: Oxford University Press.
- Bisiach, E., & Geminiani, G. (1991). Anosognosia related to hemiplegia and hemianopia. In G. P. Prigatano & D. L. Schacter (Eds.), *Awareness of deficit after brain injury: Clinical and theoretical issues* (pp. 17–39). Oxford: Oxford University Press.
- Bisiach, E., Vallar, G., Perani, D., Papagno, C., & Berti, A. (1986). Unawareness of disease following lesions of the right hemisphere: Anosognosia for hemiplegia and anosognosia for hemianopia. *Neuropsychologia*, 24(4), 471–482.
- Bracha, H. S. (2004). Freeze, flight, fight, fright, faint: Adaptationist perspectives on the acute stress response spectrum. *CNS Spectrums*, 9(9), 679–685.
- Brodie, B. C. (1837). *Lectures illustrative of certain nervous affections*. London: Longman.
- Brown, R. J. (2002). The cognitive psychology of dissociative states. *Cognitive Neuropsychiatry*, 7(3), 221–235.
- Brown, R. J. (2006). Medically unexplained symptoms: A new model. *Psychiatry*, 5(2), 43–47.
- Brown, R. J., Poliakoff, E., & Kirkman, M. A. (2007). Somatoform dissociation and somatosensory amplification are differentially associated with attention to the tactile modality following exposure to body-related stimuli. *Journal of Psychosomatic Research*, 62(2007) 159–165
- Burgmer, M., Konrad, C., Jansen, A., Kugel, H., Sommer, J., Heindel, W., et al. (2006). Abnormal brain activation during movement observation in patients with conversion paralysis. *Neuroimage*, 29, 1336–1343.
- Cappa, S., Sterzi, R., Vallar, G., & Bisiach, E. (1987). Remission of hemineglect and anosognosia during vestibular stimulation. *Neuropsychologia*, 25, 775–782.
- Carter, R. B. (1853). *On the pathology and treatment of hysteria*. London: Churchill.
- Charcot, J. M. (1889). *Clinical lectures on the diseases of the nervous system* (Vol. III). London: New Sydenham Society.
- Cooke, D. F., & Graziano, M. S. A. (2003). Defensive movements evoked by air puff in monkeys. *Journal of Neurophysiology*, 90, 3317–3329.
- Crimlisk, H. L., Bhatia, K., Cope, H., David, A., Marsden, C. D., & Ron, M. A. (1998). Slater revisited: 6 year follow up study of patients with medically unexplained motor symptoms. *British Medical Journal*, 316, 582–586.

- Cutting, J. (1978). Study of anosognosia. *Journal of Neurology, Neurosurgery and Psychiatry*, 41, 548–555.
- David, A. S., & Halligan, P. W. (1996). Editorial. *Cognitive Neuropsychiatry*, 1, 1–3.
- Davies, M., Aimola Davies, A., & Coltheart, M. (2005). Anosognosia and the two-factor theory of delusions. *Mind and Language*, 20(2), 209–236.
- Davies, M., Coltheart, M., Langdon, R., & Breen, N. (2001). Monothematic delusions: Towards a two-factor account. *Philosophy, Psychiatry & Psychology*, 8, 133–158.
- Dosey, M. A., & Meisels, M. (1969). Personal space and self-protection. *Journal of Personality and Social Psychology*, 11, 93–97.
- Eames, P. (1992). Hysteria following brain injury. *Journal of Neurology, Neurosurgery, Psychiatry*, 55, 1046–1053.
- Ellis, H. D., & Young, A. W. (1990). Accounting for delusional misidentifications. *British Journal of Psychiatry*, 157, 239–248.
- Freud, S. (1894). The neuropsychoses of defense. In *The complete psychological works* (Vol. III, pp. 45–61). London: Hogarth.
- Frith, C. D. (1992). *The cognitive neuropsychology of schizophrenia*. Hove, East Sussex: Lawrence Erlbaum Associates.
- Gagliese, L., Schiff, B., & Taylor, A. (1995). Differential consequences of left- and right-sided chronic pain. *Clinical Journal of Pain*, 11, 201–207.
- Galín, D., Diamond, R., & Braff, D. (1977). Lateralization of conversion symptoms: More frequent on the left. *American Journal of Psychiatry*, 134, 578–580.
- Gold, M., Adair, J. C., Jacobs, D. H., & Heilman, K. M. (1994). Anosognosia for hemiplegia: An electrophysiologic investigation of the feed-forward hypothesis. *Neurology*, 44(10), 1804–1808.
- Gray, J. A. (1982). *The neuropsychology of anxiety: An enquiry into the functions of the septo-hippocampal system*. New York: Oxford University Press.
- Heilman, K. M. (1991). Anosognosia: Possible neuropsychological mechanisms. In G. P. Prigatano & D. L. Schacter (Eds.), *Awareness of deficit after brain injury. Clinical and theoretical issues*. New York: Oxford University Press.
- Hochstetter, K., Meinck, H. M., Hemmingsen, P., Scherg, M., & Rupp, A. (2002). Psychogenic sensory loss: Magnetic source imaging reveals normal tactile evoked activity of the human primary and secondary somatosensory cortex. *Neuroscience Letters*, 323, 137–140.
- Hofer, M. (1970). Cardiac and respiratory function during sudden prolonged immobility in wild rodents. *Psychosomatic Medicine*, 32, 633–647.
- Horvath, T., Friedman, J., & Meares, R. (1980). Attention in hysteria: A study of Janet's hypothesis by means of habituation and arousal measures. *American Journal of Psychiatry*, 137, 217–220.
- Janet, P. (1907). *The major symptoms of hysteria*. New York: MacMillan.
- Klemm, W. R. (2001). Behavioral arrest: In search of the neural control system. *Progress in Neurobiology*, 65, 453–471.
- Kretschmer, E. (1948). *Hysteria: Reflex and instinct*. London: Peter Owen.
- Langdon, R., & Coltheart, M. (2000). The cognitive neuropsychology of delusions. *Mind and Language*, 15, 183–216.
- Levine, D. N., Calvanio, R., & Rinn, W. E. (1991). The pathogenesis of anosognosia for hemiplegia. *Neurology*, 41(11), 1770–1781.
- Lorenz, J., Kunze, K., & Bromm, B. (1998). Differentiation of conversive sensory loss and malingering by P300 in a modified oddball task. *Neuroreport*, 9, 187–191.
- Ludwig, A. M. (1972). Hysteria: A neurobiological theory. *Archives of General Psychiatry*, 27, 771–777.

- Mace, C. (2001). All in the mind? The history of hysterical conversion as a clinical concept. In P. W. Halligan, C. Bass, & A. S. David (Eds.), *Contemporary approaches to the study of hysteria* (pp. 1–11). New York: Oxford University Press.
- Magistris, M. R., Rosler, K. M., Truffert, A., Landis, T., & Hess, C. W. (1999). A clinical study of motor evoked potentials using a triple stimulation technique. *Brain*, *122*(Pt2), 265–279.
- Maher, B. A. (1999). Anomalous experience in everyday life: Its significance for psychopathology. *Monist*, *82*, 547–570.
- Marshall, J. C., Halligan, P. W., Fink, G. R., Wade, D. T., & Frackowiak, R. S. J. (1997). The functional anatomy of hysterical paralysis. *Cognition*, *64*, B1–B8.
- McConkey, K. M. (2001). Hysteria and hypnosis: Cognitive and social influences. In P. W. Halligan, C. Bass, & A. S. David (Eds.), *Contemporary approaches to the study of hysteria* (pp. 203–214). New York: Oxford University Press.
- McKay, R., Langdon, R., & Coltheart, M. (In press). Models of misbelief: Integrating motivational and deficit theories of delusions. *Consciousness and Cognition*. AU: pls provide update
- Merskey, H. (1995). *The analysis of hysteria: Understanding conversion and dissociation*. London: Gaskell.
- Meyer, B. U., Britton, T. C., Benecke, R., Bischoff, C., Machetanz, J., & Conrad, B. (1992). Motor responses evoked by magnetic brain stimulation in psychogenic limb weakness: Diagnostic value and limitations. *Journal of Neurology*, *239*, 251–255.
- Miller, E. (1999). Conversion hysteria: Is it a viable concept? In P. W. Halligan & A. S. David (Eds.), *Conversion hysteria: Towards a cognitive neuropsychological account* (pp. 181–192). Hove, East Sussex: Psychology Press.
- Moldofsky, H., & England, R. S. (1975). Facilitation of somatosensory average-evoked potentials in hysterical anesthesia and pain. *Archives of General Psychiatry*, *32*, 193–197.
- Murdoch, I. (1963). *The unicorn*. London: Vintage, 2000.
- Nigg, J. T. (2003). Response inhibition and disruptive behaviors: Toward a multiprocess conception of etiological heterogeneity for ADHD combined type and conduct disorder early-onset type. *Annals of the New York Academy of Sciences*, *1008*, 170–182.
- Oakley, D. A. (1999). Hypnosis and conversion hysteria: A unifying model. *Cognitive Neuropsychiatry*, *4*, 243–265.
- Pacherie, E. (2000). The content of intentions. *Mind and Language*, *15*(4), 400–432.
- Paget, J. (1873). The clinical methods on the nervous mimicry of organic diseases. *Lancet*, *727–729*.
- Pascuzzi, R. M. (1994). Nonphysiological (functional) unilateral motor and sensory syndromes involve the left more often than the right body. *Journal of Nervous and Mental Disorders*, *182*, 118–120.
- Ramachandran, V. S. (1995). Anosognosia in parietal lobe syndrome. *Consciousness and Cognition*, *4*, 22–51.
- Rizzolatti, G., Fadiga, L., Gallese, V., & Fogassi, L. (1995). Premotor cortex and the recognition of motor actions. *Cognitive Brain Research*, *3*, 131–141.
- Roelofs, K., de Buijn, E., & van Galen, G. P. (2006). Hyperactive action monitoring during motor-initiation in conversion paralysis: An event-related potential study. *Biological Psychology*, *71*(2006), 316–325.
- Roelofs, K., van Galen, G. P., Keijsers, G. P., & Hoogduin, C. A. (2002). Motor initiation and execution in patients with conversion paralysis. *Acta Psychologica (Amsterdam)*, *110*, 21–34.
- Scaer, R. (2001). *The body bears the burden: Trauma, dissociation and disease*. Binghampton, UK: Haworth Press.
- Schiff, W., Caviness, J. A., & Gibson, J. J. (1962). Persistent fear responses in rhesus monkeys to the optical stimulus of “looming.” *Science*, *136*, 982–983.

- Searle, J. (1983). *Intentionality*. Cambridge: Cambridge University Press.
- Seligman, M. (1975). *Helplessness: On depression, development and death*. San Francisco: Freeman.
- Shallice, T., & Burgess, P. W. (1993). Supervisory control of action and thought selection. In A. Baddeley & L. Weiskrantz (Eds.), *Attention: Selection, awareness and control: A tribute to Donald Broadbent* (pp. 171–187). Oxford: Clarendon Press.
- Sierra, M., & Berrios, G. E. (2001). Conversion hysteria: The relevance of attentional awareness. In P. W. Halligan, C. Bass, & A. S. David (Eds.), *Contemporary approaches to the study of hysteria* (pp. 192–202). New York: Oxford University Press.
- Slater, E., & Glithero, E. (1965). A follow-up of patients diagnosed as suffering from “hysteria.” *Journal of Psychosomatic Research*, 9, 9–11.
- Sonoo, M. (2004). Abductor sign: A reliable new sign to detect unilateral nonorganic paresis of the lower limb. *Journal of Neurology, Neurosurgery, Psychiatry*, 75, 121–125.
- Spence, S. A. (1999). Hysterical paralyses as disorders of action. In P. W. Halligan & A. S. David (Eds.), *Conversion hysteria: Towards a cognitive neuropsychological account* (pp. 203–226). Hove, East Sussex: Psychology Press.
- Spence, S. A. (2001). Disorders of willed action. In P. W. Halligan, C. Bass, & A. S. David (Eds.), *Contemporary approaches to the study of hysteria* (pp. 235–250). New York: Oxford University Press.
- Spence, S. A., Crimlisk, H. L., Cope, H., Ron, M. R., & Grasby, P. M. (2000). Discrete neurophysiological correlates in prefrontal cortex during hysterical and feigned disorder of movement. *Lancet*, 355, 1243–1244.
- Stern, D. B. (1983). Psychogenic somatic symptoms on the left side: Review and interpretation. In M. S. Myslobodsky (Ed.), *Hemisyndromes: Psychobiology, neurology, psychiatry* (pp. 415–445). New York: Academic Press.
- Stone, J., Sharpe, M., Carson, A., Lewis, S. C., Thomas, B., Goldbeck, R., et al. (2002). Are functional motor and sensory symptoms really more frequent on the left? A systematic review. *Journal of Neurology and Neurosurgery*, 73, 578–581.
- Stone, J., Sharpe, M., Rothwell, P. M., & Warlow, C. P. (2003). The 12 year prognosis of unilateral functional weakness and sensory disturbance. *Journal of Neurology and Neurosurgery*, 74, 591–596.
- Stone, S. P., Halligan, P. W., & Greenwood, R. J. (1993). The incidence of neglect phenomena and related disorders in patients with an acute right or left hemisphere stroke. *Age and Ageing*, 22, 46–452.
- Tiihonen, J., Kuikka, J., Viinamaki, H., et al. (1995). Altered cerebral blood flow during hysterical paraesthesia. *Biological Psychiatry*, 37, 134–135.
- Vuilleumier, P. (2000). Anosognosia. In J. Bogousslavsky & J. L. Cummings (Eds.), *Behavior and mood disorders in focal brain lesions* (pp. 465–519). Cambridge: Cambridge University Press.
- Vuilleumier, P. (2005). Hysterical conversion and brain function. *Progress in Brain Research*, 150, 309–329.
- Vuilleumier, P., Chicherio, C., Assal, F., Schwartz, S., Slosman, D., & Landis, T. (2001). Functional neuronanatomical correlates of hysterical sensorimotor loss. *Brain*, 124, 1077–1090.
- Weinstein, E. A., & Kahn, R. L. (1955). *Denial of illness: Symbolic and physiological aspects*. Springfield, IL: Charles C Thomas.
- Whitlock, F. A. (1967). The etiology of hysteria. *Acta Psychiatrica Scandinavia*, 43, 144–162.
- Wolpert, D. M., Ghahramani, Z., & Jordan, M. I. (1995). An internal model for sensorimotor integration. *Science*, 269(5232), 1880–1882.

