

# Neural Correlates of Motor Conversion Disorder

Rebekah L. Scott and J. Gregory Anson

Conversion Disorder affects voluntary motor and sensory function and involves unexplained neurological symptoms without an organic cause. Many researchers have attempted to explain how these symptoms arise but the neural correlates associated with Conversion Disorder remain largely unknown to clinicians and neuroscientists alike. This review focuses on investigations of Conversion Disorder (with motor symptoms) when deficits in voluntary movement occur. No single consistent hypothesis has emerged regarding the underlying cortical mechanisms associated with motor Conversion Disorder. However, findings from electrophysiology, neuroimaging, and behavioral research implicate the involvement of prefrontal networks. With further research using measurement techniques precise in spatial as well as temporal resolution, the conflict associated with two views of the neural correlates of motor Conversion Disorder may be resolved. This will provide a better understanding of the impairment associated with the preparation, generation, and execution of intentional movement in Conversion Disorder.

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Conversion Disorder “involves unexplained symptoms or deficits affecting voluntary motor or sensory function” (American Psychiatric Association, 1994) that cannot be attributed to an organic neurological cause. The symptoms of Conversion Disorder are thought to be generated unconsciously, arising from psychological stress, trauma, or conflict (Vuilleumier, 2005). It has been reported in 1–3% of outpatient referrals in mental health clinics, and in the general population, rates range from 0.01% to 0.3% (American Psychiatric Association, 1994).

Conversion Disorder originated as hysteria. However, dissatisfaction with the term *hysteria* has led to attempts to modify the label, which in turn, has led to an abundance of diagnostic nomenclatures in the literature (for example, somatoform disorders [Ron, 1994], psychogenic pain or paralysis [Pillai, Markind, Streletz, Field, & Herbison, 1992], dissociative disorders [Kihlstrom, 1992b], conversion or functional symptoms [Reuber, Mitchell, Howlett, Crimlisk, & Grunewald, 2005], and hysterical neurosis [Timsit-Berthier, Delaunoy, Koninckx, & Rousseau, 1973]), which has made diagnosis and consistent description difficult. Even

attempts by internationally recognized standard references have not achieved consensus and remain confusing. In the current International Classification of Diseases (ICD-10; World Health Organization, 1992), hysteria (Conversion Disorder) is classified as a "Dissociative Disorder," whereas the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV; American Psychiatric Association, 1994) defines the same phenomena as a "Somatoform Disorder." This has created an ongoing and complicated dilemma for clinicians who are faced with reliably assessing and distinguishing patients who present with organic disease from those with symptoms in the absence of organic lesions and from those who are feigning a disorder (consciously generating a symptom). Given this lack of consensus, discrete categorization of the deficits associated with Conversion Disorder should be regarded cautiously. In this review, the classification of Conversion Disorder is consistent with that described in the DSM-IV (American Psychiatric Association, 1994). This classification scheme includes extensive information about diagnostic features and a diagnostic criterion and divides Conversion Disorder into four subtypes based on the nature of the conversion symptoms. The four subtypes include: Conversion Disorder with motor symptom or deficit, with sensory symptom or deficit, with seizures or convulsions, or with mixed presentation.

Specifically, the aim of this review is to focus on the research that has examined Conversion Disorder with unexplained motor symptoms. In particular, research attempting to associate motor conversion symptoms (where there is cessation of movement) with an underlying neurophysiological correlate will be reviewed. Much of this research has investigated unexplained neurological symptoms associated with Conversion Disorder in the context of motor control. Research examining sensory or seizure symptoms associated with Conversion Disorder is not included (for a review of these subtypes, see Trimble, 2001; Vuilleumier, 2005).

Within Conversion Disorder with motor symptoms, the phrase *unexplained symptoms* is often used synonymously with *psychogenic symptoms* and implies that characteristics of Conversion Disorder "mimic the entire spectrum of neurological disease" (Stone & Zeman, 2001). Unexplained motor symptoms are inconsistent, impeding everyday motor function because of the disruption in the production of "normal" voluntary movement. Symptom presentations range from negative symptoms (loss of function) to positive symptoms (exaggeration or disorganization of existing function). The most frequent include paralysis, give-way weakness, dystonia, tremor, jerks, and gait disturbances such as dragging of the affected limb, all without an organic neurological cause. It has been reported that weakness or partial paralysis generally involves whole movements affecting the upper and lower limbs (Stone & Zeman, 2001). Patients frequently display a lack of movement (and no change in the electromyogram) despite subjective self-reports of effort (Burgmer et al., 2006; Marshall, Halligan, Fink, Wade, & Frackowiak, 1997) and significantly less force in the affected compared with the unaffected limb (de Lange, Roelofs, & Toni, 2007; Roelofs, de Bruijn, & van Galen, 2006).

From a theoretical perspective, several explanations have been offered that attempt to provide answers to why these symptoms occur and the neuropsychological processes and neural mechanisms that underlie them. Many of the theories attempting to link conversion symptoms with mechanisms in the central nervous

system are speculative, and until recently, few of the neuropsychological or neurophysiological hypotheses proposed have been tested experimentally (Vuilleumier, 2005). Recent research has extended this notion to investigate how these symptoms might be represented in the brain and the way in which the voluntary motor system is affected by Conversion Disorder (see Halligan, Bass, & Marshall, 2001; Merskey, 1995; Vuilleumier, 2005, for helpful reviews).

This review contains the following sections: A Brief History of Hysteria, Electrophysiological Studies of Conversion Disorder, Imaging Conversion Disorder With Motor Symptoms, Motor Imagery and Conversion Disorder, and Neural Mechanisms.

## A Brief History of Hysteria

The concept of hysteria dates back to hieroglyphic representations in ancient Egyptian medical papyri (Veith, 1965), where symptoms were thought to arise from a displacement of the uterus. Such “hysterical” phenomena were frequently observed as headaches, hysterical suffocation, spasms, and fits (Merskey, 1995) and consequently, hysteria was considered to be a physical illness. Toward the 18th century, the traditional Descartes dualism relationship between the mind and body (that the mind controls the body but the body can also influence the mind) was commonly recognized, and hysterical symptoms began to be viewed as distinct from organic illness. Because of limited anatomical and physiological knowledge, questions arose about the mind and how it might influence the body to cause physical symptoms. Emerging theories in the 19th century acknowledged the involvement of the central nervous system associated with hysterical symptoms (see for example, Breuer & Freud, 1955; Brodie, 1837; Charcot, 1889; Janet, 1907). In particular, Freud’s psychodynamic theory contributed significantly to the study of hysteria because his account of symptom generation, in which unconscious conflict and affective motive are transformed into bodily complaints, paved the way for the idea of conversion symptoms.

The term *hysteria* first appeared in the sixth edition of the ICD (ICD-6; World Health Organization, 1948), which was the first ICD revision to include a separate section on mental disorders. Four years later, the first edition of the DSM (DSM-I; American Psychiatric Association, 1952) was published as an alternative to the ICD, with a focus on clinical utility. The DSM-I referred to hysteria as “conversion reaction” and suggested this term is synonymous with “conversion hysteria.” The DSM-I emphasized that anxiety was an important feature of hysteria. It was thought that one could control anxiety through the defense mechanism of “conversion,” whereby the underlying mental conflict is “converted into functional symptoms in organs or other parts of the body, usually those that are mainly under voluntary control” (American Psychiatric Association, 1952).

Following several revisions of each classification system (which included the reintroduction of the term *hysteria* in the DSM-II and the change of the term *reaction* to *neurosis* in the ICD-8), the American Psychiatric Association finally abolished *hysteria* from psychiatry because of the increased public stigma associated with the label when referring to woman and because of the fact the label already encompassed multiple meanings. Instead, the disorder fell under the category of somatoform disorders and was termed *Conversion Disorder* in the DSM-III

(American Psychiatric Association, 1980), while the ICD removed the label *neurosis* and eventually categorized hysteria under dissociative disorders (World Health Organization, 1992).

Despite numerous alterations to the label, the concept of hysteria has endured for over a century (Veith, 1965). The DSM endeavored to select suitable diagnostic terms that would “reduce confusion and ambiguity to a minimum” (American Psychiatric Association, 1968), however, as pointed out by Brown and colleagues (Brown, Cardena, Nijenhuis, Sar, & van der Hart, 2007), the separation between somatoform and dissociative disorders has led researchers and clinicians to mistake these two groups of diagnoses as unrelated, thus causing significant confusion. Given the high correlation of symptoms and the similar nature of the underlying processes between somatoform disorders and dissociative disorders, the appropriate classification system continues to be a major controversy (Brown et al., 2007).

## Electrophysiological Studies of Conversion Disorder

Although there are few experiments using the electrophysiological techniques of transcranial magnetic stimulation (TMS) and electroencephalography to investigate motor Conversion Disorder, these methodologies provide objective temporal measures of brain function. These techniques enable a dissociation of neurological and conversion symptoms and provide an insight into the disruption of the mechanisms of motor preparation and execution.

The technique of TMS has been used to provide a measure of integrity of the central and peripheral nervous system. TMS involves a coil of copper wire that produces a large current flow through the discharge of an electrical capacitance. The current flow produces a magnetic field perpendicular to the coil (Rothwell, 1997). When applied over the surface of the skull, electrical eddy currents are induced in the brain, which depolarize axons in the cerebral cortex and subcortical tissue. Magnetic stimulation of the motor cortex evokes a muscle response (motor evoked potential; MEP) that can be recorded using electromyography (EMG). The MEP represents the outcome of direct stimulation of the corticospinal axon as well as transsynaptic (indirect) activation of corticospinal neurons (Terao & Ugawa, 2002).

Evidence of dysfunction can be identified through analysis of the latency and amplitude of the MEP (Barker, Jalinous, & Freeston, 1985; Merton & Morton, 1980; Meyer et al., 1992). For example, paralysis results from a dysfunction in the central motor pathways (Cantello, Boccagni, Comi, Civardi, & Monaco, 2001). Investigations of organic paralysis using TMS have revealed abnormal central motor conduction as evidenced by reduced amplitudes and prolonged latencies and even absent MEPs (Meyer et al., 1992). TMS can, therefore, be used as a tool to distinguish between neurological symptoms and symptoms of psychogenic origin (Halligan et al., 2001), as it would be expected that MEPs of motor Conversion Disorder in which there is no organic lesion would present normal amplitude and time characteristics.

Schriefer, Mills, Murray, and Hess (1987) examined four patients who had functional arm weakness. They reported normal MEP amplitudes and latencies in

each patient, indicating that the weakness was not due to impaired transmission. Further studies have confirmed and extended these results. A variety of unexplained motor symptoms have been examined (e.g., apparent quadriplegia, bilateral lower limb numbness and paralysis [Pillai et al., 1992]; functional paraplegia [Jellinek, Bradford, Bailey, & Symon, 1992]; functional quadriplegia [Morota, Deletis, Kiprovski, Epstein, & Abbott, 1994]; psychogenic weakness—either monoparesis, hemiparesis, or paraparesis [Meyer et al., 1992]; nonneurological disorders [Magistris, Rösler, Truffert, Landis, & Hess, 1999]; psychogenic paralysis in patients diagnosed with Conversion Disorder, Factitious Disorder, or Malingering [Cantello et al., 2001]). In all of these studies, TMS assessment was used, and normal MEP amplitudes and latencies were reported. These results confirmed that motor pathways in these patients were intact and reinforce the use of MEPs to aid in differentiating between organic and psychogenic paralysis. Foong, Ridding, Cope, Marsden, and Ron (1997) administered TMS to two patients before and after the recovery from left hemiparetic symptoms and also reported normal MEPs in both patients. Foong et al. also obtained response thresholds for evoking an EMG response and response-stimulus intensity curves by stimulating the optimal scalp location and recording the EMG response with increasing stimulus intensity. Normally, increased TMS intensity applied to the motor cortex results in increased MEP amplitude. However, Foong and colleagues observed that at moderate intensity, a cerebral asymmetry occurred in which the right hemisphere (left hemiparesis) was less excitable than the left hemisphere.

TMS has been used as a therapeutic tool in treating patients with Conversion Disorder. Schönfeldt-Lecuona, Connemann, Viviani, Spitzer, and Herwig (2006) investigated the effect of repetitive TMS (rTMS) in four patients with nonorganic limb paralysis. Over 5–12 weeks, the patients received rTMS on working days for two hours over the motor cortex contralateral to the affected limb. Over the course of the treatment, motor function was completely restored in one patient and was markedly improved in two patients (while no effect was observed with one patient who was diagnosed with Malingering). Schönfeldt-Lecuona and colleagues concluded this may be attributed to an enhanced input to the motor cortex facilitating the reacquisition of volitional movement. No control stimulation condition was included, thus, the possibility of a placebo effect cannot be ruled out.

Electroencephalography (EEG) is another electrophysiological technique that has been used in the diagnosis of Conversion Disorder. EEG provides precise temporal information about changes in brain electrical activity as it enables cortical electrical potentials to be recorded from the surface of the scalp in real time. Abrupt changes in the normal human EEG pattern characterized by changes in frequency and amplitude can be indicative of abnormality in brain function. For example, paroxysmal abnormalities in the EEG evident during epileptic seizures (Gazzaniga, Ivry, & Mangun, 2002). In assessing patients thought to have Conversion Disorder, EEG has frequently been used to avoid misdiagnosis of epilepsy in patients who have nonepileptic seizures. However, few attempts have been made using EEG to link identification of functional symptoms of motor Conversion Disorder with underlying neural mechanisms.

Although EEG provides excellent temporal resolution, it has relatively poor spatial resolution. This is because the electrical activity reflects the summated activity of excitatory synaptic potentials from a large population of neurons; thus,

changes in EEG can be contributed to by volume conduction making localization of change difficult. One way to control for the effects of volume conduction is through task manipulation within an experimental design. Different specific external events or precued parameters prior to stimulus presentation elicit brain activity that when aligned on the event and averaged, permits measurement of an event related potential (ERP) that may be associated with the specific nature of the task. Changes in latency and amplitude measures of ERPs enable an insight into various human cognitive functions (Coles, 1989). In addition to using EEG as a monitoring tool in epilepsy, EEG can be used to verify “normal” evoked potentials in patients presenting with unexplained motor and sensory symptoms (Halligan et al., 2001; Howard & Dorfman, 1986).

The measurement of ERPs to investigate brain function in individuals with Conversion Disorder has focused primarily on evoked potentials associated with hysterical sensory symptoms. Although beyond the scope of this review, it is noted that these studies aimed to demonstrate electrophysiological responses that were either abnormal or intact in the face of functional symptoms associated with hysterical anesthesia, blindness, and deafness. Reviews of research on sensory symptoms have been conducted by Whitlock (1967) and more recently by Vuilleumier (2005). Generally, they report normal amplitudes and latencies in somatosensory evoked potentials, visual evoked potentials, and brainstem auditory evoked potentials indicating that sensory pathways were functionally intact despite the presence of hysterical sensory symptoms.

Despite a number of research studies suggesting Conversion Disorder disrupts the voluntary motor system by affecting volitional movement (Brodie, 1837; Halligan et al., 2001; Marshall et al., 1997; Spence, 1999), investigations of Conversion Disorder by measuring and evaluating motor related ERPs do not appear to have been conducted (e.g., motor ERPs the *Bereitschaftspotential* [BP] and contingent negative variation [CNV] associated with preparation for self-paced and externally cued movements, respectively).

The readiness potential, or BP, an ERP related to volitional movement, was first described by Kornhuber and Deecke (1965). The BP is characterized by a ramp-like negative shift in the EEG preceding self-initiated, self-paced movement by as much as 1,000 ms (Coles & Rugg, 1995). The BP has useful clinical application in psychogenic disorders such as Conversion Disorder. Examining the BP provides important insights into how voluntary movement is generated and executed (Colebatch, 2007). Two studies have examined the BP in a psychogenic disorder. Toro and Torres (1986) investigated one patient presenting with weakness in the right upper and lower extremities, followed by a sudden onset of abnormal twitches in his left foot and both hands. EEG was recorded during these episodes. A slow negative wave, time-locked to the foot twitches, was observed over the midline with a centroparietal distribution, suggesting the brain mechanisms associated with his movements were similar to those in normal volitional movement. The second investigation by Terada et al. (1995) suggested a BP would be observed if the movements were a product of a voluntary mechanism but would be absent before an involuntary movement. Six patients with psychogenic myoclonus were investigated by Terada and colleagues (1995) who reported the presence of a BP preceding myoclonic jerks in five of the six patients and described these as similar to the slow EEG shift seen before mimicked jerks. Terada et al.

(1995) and Toro and Torres (1986) have suggested the recording of the EEG and measuring the BP to be clinically useful tools for investigating motor Conversion Disorder (for a detailed review of the BP and clinical applications, see Colebatch, 2007; Shibasaki & Hallett, 2006). Marsden (1986) and Ron (1994) also reported that abnormalities in the BP may be seen in patients with hysterical paralyses. However, an association between changes in BP and Conversion Disorder during attempted voluntary movement is yet to be resolved. BPs are limited to assessing intention to move rather than motor preparation per se because movement initiation is not contingent on an imperative stimulus.

The CNV is an ERP related to movement preparation linked to a specific stimulus event. Walter, Cooper, Aldridge, McCallum, and Winter (1964) first described the CNV as slow surface negativity that developed during the foreperiod providing it preceded a motor response in a cued reaction time task. The use of CNV in psychiatry and neurology is well-established, but investigations comparing CNV amplitude and morphology in a clinical population contrasted with healthy controls has provided varied results (Tecce & Cattanaach, 1987). It is evident from several studies that CNV development is disrupted and CNV amplitude is lower in depressed patients (Giedke & Bolz, 1980; Hansenne & Anseau, 2001; Timsit-Berthier et al., 1973) and in individuals with high levels of anxiety or stress (Knott & Irwin, 1973; Low & Swift, 1971; McCallum & Walter, 1968). Although these symptoms of neurosis may coexist with Conversion Disorder, there appears to be no empirical research that has recorded and measured the CNV in patients clinically diagnosed with Conversion Disorder with motor symptoms. Timsit-Berthier and colleagues (1973) examined CNV amplitude, morphology, and duration in psychotic and neurotic patients and in a control group. The neurotic group consisted of 135 patients with symptoms of depression, phobias, obsessions, and “mechanisms of conversion” ( $n = 90$ ), although whether any of these patients were diagnosed with Conversion Disorder is uncertain. A warning stimulus (click; S1) followed by a series of flashes (imperative stimulus; S2) was presented, after which the participant pressed a button that extinguished the flashes. Results showed that hysterical neurotic patients generated small amplitude (typically less than  $5 \mu\text{V}$ ) Type A “field dependency” CNVs, where the maximum negativity is quickly reached and is larger after S1 than S2. Vuilleumier (2005) has suggested there is “anecdotal evidence that conversion patients may show an abnormal CNV.” In an earlier paper, Vuilleumier and colleagues (2001) stated that studies using evoked potentials (e.g., TMS to record MEPs) have “shown normal motor responses and early sensory components, but non-specific alterations in later components such as P300 or CNV” (p. 1086).

If the motor symptoms of Conversion Disorder are truly psychological in nature and recordings of motor and sensory evoked potentials are normal indicating intact corticospinal pathways, why, then, do Conversion Disorder patients exhibit cessation of voluntary movement? It might be inferred that the neurological mechanisms associated with Conversion Disorder are likely to be premotor in nature. In the following section, investigations of Conversion Disorder using neuroimaging will be reviewed. Imaging techniques, such as positron emission tomography (PET) and functional magnetic resonance imaging (fMRI) for example, offer greater precision in the spatial domain, enabling neuroscientists to localize cortical regions that are more (or less) active during specific cognitive

processes. In psychiatric disorders, neuroimaging has complemented electrophysiological studies by extending existing knowledge of the biological substrate underpinning the disorders. Research using neuroimaging to examine Conversion Disorder has contributed to the development of two hypotheses about why patients exhibit a cessation of voluntary movement: One hypothesis is that the mechanism generating motor programs is faulty and leads to impaired motor preparation. A second hypothesis is that motor programs are generated normally but are not able to be “released” or “ignited,” leading to delays and disruption before the execution phase of movement. The term *motor program* itself is sometimes viewed as controversial and in this review refers to the perspective held by Rosenbaum that motor programs are “a functional state that allows particular movements, or classes of movements, to occur” (Rosenbaum, 1991). Although the motor program concept is primarily a behavioral phenomenon, there have been recent attempts to link the behavior to neurobiological mechanisms concerned with motor preparation, initiation, and execution (Anson, Hyland, Kötter, & Wickens, 2000; Braitenberg, 1978; Palm, 1990; Wickens, Hyland, & Anson, 1994). The conflicting preparation versus execution hypotheses have been deduced from the results of various imaging experiments reviewed in the following, but it is difficult, if not impossible, to separate preparation and execution effects from these data because of the slow time constants of the cerebrovascular responses (e.g., in fMRI). Because motor programming effects are almost always inferred from precise temporal measurements, EEG and ERP measures offer a better opportunity to examine potential motor preparation deficits in individuals with Conversion Disorder.

## Imaging Conversion Disorder With Motor Symptoms

The use of brain imaging has escalated in popularity and accessibility. The growing trend to measure structural and functional changes in the brain has contributed significantly to improving understanding of the neural mechanisms underpinning disorders of action and psychiatric conditions such as depression, schizophrenia, hallucinations, and other disorders with medically unexplained symptoms (e.g., Abou-Saleh, 2006; Frith & Dolan, 1998; Fusar-Poli & Broome, 2006). Neuroimaging techniques, for example single-photon emission computed tomography (SPECT), PET, and fMRI, that index regional cerebral blood flow (rCBF) changes indirectly to neuronal synaptic activity are the most frequently used and are well-suited to investigations of Conversion Disorder with motor symptoms (Vuilleumier, 2005). Despite conflicting and inconsistent results because of factors such as small sample sizes and comorbidity and heterogeneity of clinical deficits, the advantage of localizing anatomical cortical regions of interest has enabled an advance in the psychiatric and psychological literature through integration of knowledge from neurology and psychiatry to attempt to understand the pathophysiology underlying motor Conversion Disorder.

Although many of the imaging studies have not employed a movement task per se and often lack the inclusion of a healthy control group for comparison, they merit inclusion because they have revealed insights into cortical areas activated during symptom presentation and potential neurophysiological mechanisms



underlying motor conversion symptoms that ultimately affect the preparation and control of voluntary movement. Two possible mechanisms that might underpin Conversion Disorder have been described. Evidence has emerged for a frontocortical mechanism involving the anterior cingulate and orbitofrontal cortices, structures thought to inhibit the motor cortex (Marshall et al., 1997; Tiihonen, Kuikka, Viinamäki, Lehtonen, & Partanen, 1995). Alternatively it has been suggested that the involvement of striathalamocortical circuits and dorsolateral prefrontal cortex results in impaired volition and dysfunctional motor programs (Spence, Crimlisk, Cope, Ron, & Grasby, 2000; Vuilleumier et al., 2001; see Table 1 in Montoya, Price, & Lepage, 2006; and in Stone et al., 2007, for a brief summary of the findings of these neuroimaging studies).

One of the first neuroimaging studies (Tiihonen et al., 1995) assessed a woman with hysterical paralysis and paresthesia on her left side. SPECT was used to measure changes in CBF during electrical stimulation of the left median nerve (at rest) while the patient was symptomatic and again following recovery six weeks later. During the symptomatic stage, Tiihonen and colleagues found an unexpected decreased perfusion of the right parietal cortex associated with increased perfusion in the right frontal lobe during nerve stimulation. After recovery, increased activity in the right parietal cortex was observed leading the authors to suggest that psychogenic paresthesia may be associated with “simultaneous activation of frontal inhibitory areas and inhibition of the somatosensory cortex.” It is difficult to determine whether the observed increase in blood flow to the frontal region was present throughout the duration of the patient’s symptoms because only two scans were taken for this study, both of which were during sensory stimulation. Whether the effects observed could be solely attributed to her hysterical symptoms and not other associated changes in mental state could not be concluded. Furthermore, a case study does not allow for the results to be generalized across patients because hysteria is a protean disorder.

These caveats aside, Tiihonen and colleagues demonstrated a possible neurophysiological correlate of hysterical phenomena, indicating that adverse psychological events can produce changes in brain physiology associated with an observed change in sensorimotor function. Similar findings were reported by Yazici and Kostakoglu (1998) who investigated five patients with astasia-abasia (an inability to walk or stand normally) who had bilateral conversion symptoms. From SPECT measures they reported hypoperfusion of the left parietal and left temporal lobes, which may have indicated regional-specific cortical inhibition.

The finding that “higher” frontal brain regions inhibit “lower” brain regions (e.g., the motor cortices), a mechanism put forth by Tiihonen et al. (1995), was supported by Marshall et al. (1997) who also carried out a case study of a woman with chronic left-sided paralysis brought about by “psychological stress and trauma.” In the experiment, the patient was instructed to either “prepare to move” or to “execute a movement” with her affected (left) and then unaffected (right) leg. The right leg response was included as a control. PET scans were taken throughout the four experimental conditions and during rest (baseline control condition). The patient was instructed to try as hard as possible to lift each leg on a metronome beat; however, in all conditions, both legs were strapped down to restrict movement and to control for confounding effects of sensory and proprioceptive feedback. The results of this study should, therefore, be interpreted in terms of the

ability to command contraction of the leg muscles rather than as instruction that would result in movement (Athwal, Halligan, Fink, Marshall, & Frackowiak, 2001). Results of the PET imaging (relative to baseline) indicated normal brain activation in the premotor areas, cerebellum, and dorsolateral prefrontal cortex (DLPFC) during motor preparation of both limbs (evidence against feigning) and in the left primary motor cortex upon moving the unaffected (right) leg. In contrast, when asked to attempt to move her paralyzed leg, movement was absent and there was no activation of right premotor areas or primary motor cortex. This observation was accompanied by no change in muscle excitation on a recording of continuous surface EMG. An increase in activation of the right anterior cingulate cortex (ACC) and right orbitofrontal cortex (OFC) was observed, areas that have not been previously reported as showing increased activity in studies investigating normal movement generation or attempted movement (Athwal et al., 2001).

The ACC (Brodmann Areas [BA] 24/32) and OFC (BA 10) are part of the rostral limbic system and have been implicated in mediating emotional and motivational processes (Devinsky, Morrell, & Vogt, 1995; Roelofs et al., 2006), playing a role in inhibiting spontaneous movements (Aron, Robbins, & Poldrack, 2004; Marshall et al., 1997; Paus, Petrides, Evans, & Meyer, 1993), involved in action-monitoring functions such as detecting errors and behavioral conflict (Ridderinkhof, Ullsperger, Crone, & Nieuwenhuis, 2004; Roelofs et al., 2006), and modulating freezing reactions in response to changes in behavioral stress (Lacroix, Broersen, Weiner, & Feldon, 1998; Lacroix, Spinelli, Heidbreder, & Feldon, 2000). Lesions in these areas have been reported in disorders affecting cognition such as schizophrenia and depression (Carter, Mintun, Nichols, & Cohen, 1997) and disorders affecting voluntary movement (after a stroke), including motor neglect and impairments of motor initiation (Athwal et al., 2001). The ACC and OFC are closely connected to nearby prefrontal and premotor areas via anatomical projections from large layer V pyramidal neurons and are central to the executive control of cognition (Carter et al., 1998; Devinsky et al., 1995). With reference to their experiment, Marshall and colleagues speculated that movement of the left leg was inhibited by increased activation of the ACC and OFC. They concluded that the hemiparalysis was triggered by the patient's "will to move," where *will* refers to the ability to choose one's own actions. Although there was a mismatch between the participant's intention to move and the actual movement outcome, intention or "will" was thought to be intact. The presence of ACC activation (Paus, 2001) and DLPFC activation (Spence & Frith, 1999) during the attempted movement of both the affected and unaffected leg supports this conclusion because it indicates the patient was choosing to perform an action (see reference to Spence et al., 2000, below). These findings support the view that in Conversion Disorder, volition is intact and the capability to generate motor programs appears to be intact, although may be interrupted later during execution (Athwal et al., 2001; Broome, 2004; Ron, 2001). In a different approach, Halligan, Athwal, Oakley, and Frackowiak (2000) used hypnosis and reported similar findings. With a single-subject design, Halligan and colleagues employed the same experimental protocol as in the preceding study. In addition, a healthy participant was tested after hypnotic induction in which left-leg paralysis was suggested. The results for the patient and the control participant were the same, thus, Halligan and colleagues

concluded that conversion and hypnotic symptoms shared common neural mechanisms involving prefrontal circuits.

Increased activation in prefrontal circuits (specifically ACC) was found in an ERP study examining the temporal characteristics of changes in ACC activity during preresponse action monitoring in six patients with unilateral conversion paresis who were asked to generate a button press movement with their affected limb (Roelofs et al., 2006). No control group was included in this study. The ACC has also been shown to be activated during motor tasks that involve conflict during response selection due to simultaneously prepared responses, as seen for example in the “flankers task” (Eriksen & Eriksen, 1974) as employed in Roelofs and colleagues’ experiment. This conflict is seen in the N2 component (a negative deflection in the EEG, 200–400 ms following stimulus onset) of the ERP and is thought to reflect preresponse conflict evoked by incongruent stimuli leading to concurrently activated competing responses (Ursu, Stenger, Shear, Jones, & Carter, 2003; van Veen & Carter, 2002; van Veen, Cohen, Botvinick, Stenger, & Carter, 2001). An increase in the N2 amplitude was observed in the affected limb compared with the unaffected limb and thought to be representative of hyperactive action monitoring. It appears that increased prefrontal activity, particularly in the ACC, is linked to two processes: action monitoring and, potentially, response conflict.

In sum, research points toward an inhibitory neural mechanism to explain motor Conversion Disorder. Tiihonen et al. (1995) and Marshall et al. (1997) have proposed that volition is intact in Conversion Disorder and explained the deficit in generating movement as an excessive inhibitory effect of prefrontal structures (ACC and OFC) on the motor cortex. Motor programs appear to be generated normally, but their processes are later disrupted during the execution phase. Alternative views indicate significant disagreement with a “disrupted execution” hypothesis. Vuilleumier et al. (2001) and Spence et al. (2000) support the notion that the inability to initiate and perform voluntary movement may be due to abnormalities in the genesis of motor programs and the intent or readiness to move. Therefore, a second and alternative neural mechanism to explain motor Conversion Disorder has been proposed in which it is argued that patients have a deficit in volition that involves the left DLPFC and striatohalamocortical circuits. This alternative explanation implicates different structures and functions in seeking to explain the cause(s) of Conversion Disorder. It is also possible, given the complexity of brain structure and function, that both hypotheses are correct and that Conversion Disorder is associated with deficits in both planning and execution (premotor stage).

Spence and colleagues have been particularly interested in the role of volition in Conversion Disorder. They view hysteria as a disorder of the “will” and have hypothesized dysfunction in the cortical regions involved in volition (Spence, 1996, 1999; Spence & Frith, 1999), especially DLPFC, BA 9/46 (see also Libet, Freeman, & Sutherland, 1999), as these regions are important for spontaneous response generation. In particular, activation of the left DLPFC in normal subjects has been associated with intentionally choosing a response, evident in tasks involving finger movements or speaking a word (Frith, Friston, Liddle, & Frackowiak, 1991), in a movement sequence involving a joystick (Spence et al., 1997), in normal subjects making mouth movements (Spence, Hirsch, Brooks, & Grasby, 1998), in a complex motor task requiring either hand (Haaland, Elsinger, Mayer,

Durgerian, & Rao, 2004), in a rhythmic tapping task requiring “fully conscious motor control” (Stephan et al., 2002), and in simultaneous bimanual movements (Anson, Scott, & Hyland, 2007). In contrast, psychiatric patients that exhibit a deficit in action have shown prefrontal dysfunction associated with a reduction in left DLPFC activation. This has also been observed at rest in participants with schizophrenia (Liddle et al., 1992; Spence et al., 1998) and depression (Dolan et al., 1993; Elliott et al., 1997).

Spence et al. (2000) sought a neurobiological distinction between hysterical motor symptoms and feigned paralysis. They compared two patients with left arm weakness to eight control subjects, two of whom were instructed to feign difficulty in moving their left upper limbs. Participants moved a joystick in a paced, self-selected sequence to the left or to the right with either the left or right hand. PET results showed hypoactivation in left DLPFC in patients compared with feigners and controls. Feigners exhibited hypofunction of the right anterior prefrontal cortex, an area reported to be involved in volitional inhibition (for a review, see Aron, 2007; Aron et al., 2004). No repeated measurements were taken after recovery. Hypoactivity of left prefrontal areas has been associated with depression (Dolan et al., 1993), and although neither patient was receiving medication, effects due to depression could not be ruled out.

Because hysterical patients “specifically ‘deactivate’ the left DLPFC during action” (Spence, 2001), Spence and colleagues concluded that a neurophysiological difference existed between hysterical and feigned symptoms. Consistent with the hypothesis that hysteria is a disorder of willed action, it was proposed that dysfunction of the left DLPFC, a region “specifically activated by the internal generation (‘choice’) of action” or volitional movement, was associated with hysteria. From a holistic perspective, these findings support those of Marshall et al. (1997) that Conversion Disorder is associated with prefrontal cortex dysfunction.

However, two important issues need to be considered. First, Marshall and others argued that bilateral DLPFC activation was evidence against faking, but as pointed out by Spence (2001), “intentional feigning is also an ‘act.’ . . . how can we know that a patient is ‘trying’?” (p. 246). The imaging studies are, thus, limited in providing an objective measure of a patients’ conscious intention to move. The results from Spence et al. (2000) provide some support that feigned paralysis can be functionally parsed from hysterical dysfunction. Second, it appears that the findings of Marshall and colleagues of right prefrontal cortex activation are consistent with those of feigners but are inconsistent with those from hysterical patients (who show abnormality in the left prefrontal cortex; Terao & Collinson, 2000). This conjecture was sharply rebutted by Halligan, Oakley, Athwal, and Frackowiak (2000), who maintained the patient imaged by Marshall et al. was appropriately diagnosed with Conversion Disorder according to the criteria outlined in the DSM-IV (American Psychiatric Association, 1994). It is an oversimplification of the imaging data given the differences in the task, the cognitive functions of each discrete cortical region, and the nature of their activations to assume that the underactivation of the right prefrontal cortex in feigners (Spence et al., 2000) is equivalent to an overactivation of the right prefrontal cortex in hysterical patients (Marshall et al., 1997).

Vuilleumier and colleagues (Vuilleumier et al., 2001) used SPECT in a carefully controlled experimental design in which they measured the changes in rCBF

at rest and while employing passive vibration to both the affected and unaffected limbs simultaneously before and after symptom recovery in seven patients with acute hysterical unilateral motor deficits. The patients were not required to make a movement. Instead, passive vibration was used to evade the potential problems (ambiguity of instructions, motivational differences) associated with engaging in active motor tasks (such as those used by Marshall et al., 1997, and Spence et al., 2000). Patients demonstrated an increase bilaterally in rCBF in the frontal and parietal regions during stimulation (prerecovery) compared with rest. This finding is consistent with intact neurological function and normal evoked potentials in electrophysiological studies (e.g., Cantello et al., 2001; Jellinek et al., 1992; Meyer et al., 1992; Morota et al., 1994; Pillai et al., 1992; Schriefer et al., 1987). Comparison of activation while asymptomatic and stimulation before recovery revealed a consistent reduction in rCBF in the thalamus and basal ganglia (caudate and putamen) contralateral to the deficit. Activity returned to normal after the symptoms had resolved, leading the authors to suggest that “hysterical conversion deficits may entail a functional disorder in striatothalamocortical circuits,” circuits that are vital for voluntary movement. Through the interactions between basal ganglia thalamocortical circuits, the prefrontal circuit (dependent on connection loops with DLPFC and lateral OFC), and the limbic circuit (dependent on connection loops with ACC and medial OFC), there are many direct and indirect pathways through which emotional, affective, and motivational processes could influence and modulate volition (Alexander, Crutcher, & DeLong, 1990; Brown & Pluck, 2000; Vuilleumier, 2005). In the case of Conversion Disorder, failure of these processes could potentially impair motor preparation and initiation and produce abnormal movements (Vuilleumier, 2005).

In a recent experiment, Burgmer et al. (2006) sought to avoid the limitation present with tasks requiring an overt movement by incorporating a movement observation task. Four patients with hemiparesis were compared with seven healthy control participants. Using fMRI, brain activation patterns were investigated while participants were presented with images of a left and right hand at rest and of the hands opening and closing at a frequency of 1 Hz (for 15 s) separately in a movement observation task and imitative execution task. The execution task required participants to observe the moving left and right hand and copy the same movement. Patients were instructed to continue trying even if they were unable to perform the movement. Contrary to previous imaging studies (Marshall et al., 1997; Spence et al., 2000), fMRI results of the motor execution task revealed no difference in cortical activation patterns between Conversion Disorder patients and control participants. In patients, no difference was found between the affected and unaffected hands. No visible movement was present in the patients' affected hand during the attempted movement execution task, although this was only investigated qualitatively. A difference in activation patterns was found in the movement observation task: A deficit in contralateral motor cortex activation was observed in the conversion patients for the affected hand, indicating, perhaps, a disruption of movement initiation, possibly from simultaneous agonist and antagonist muscle contraction or disruption of internal movement representation (Burgmer et al., 2006).

Stone and colleagues (Stone et al., 2007) also used fMRI and examined four patients with Conversion Disorder (DSM-IV diagnoses) with unilateral motor symptoms affecting the leg. Results of the Conversion Disorder patients compared

with four healthy controls simulating unilateral weakness during a repetitive ankle plantarflexion task of alternating ankles revealed both similarities and differences between patients and controls feigning weakness. The degree of similarity of movement and effort between patients and controls was self-reported by the participants. During the task, two of the Conversion Disorder patients showed minimal ankle movement, while the other two patients had difficulty moving their ankles. Both patients and controls showed a greater reduction in activation of the motor cortex contralateral to their “weak” limb compared with their unaffected leg, a finding supporting Marshall et al. (1997). However, patients also showed greater activation bilaterally in the basal ganglia and lingual gyri, and left insula and inferior frontal cortex when moving the weak limb but not the unaffected limb. Because these areas have been implicated in motor preparation, the authors suggested this may reflect “genuine motor preparation” in the patient group, perhaps requiring greater mental effort. In addition, patients exhibited hypoactivation of OFC, in contrast with the findings of Marshall et al. (1997), who found an increase in the right ACC and OFC. Control participants simulating weakness showed activation of the contralateral supplementary motor area, whereas patients did not, suggesting an excess of movement planning in the feigned weak limb of controls relative to their unaffected limb and impairment of voluntary movement planning in those with Conversion Disorder.

## Motor Imagery and Conversion Disorder

It is also of interest to examine whether mental motor representations are disrupted in Conversion Disorder. Roelofs and colleagues reported several investigations examining motor dysfunction in Conversion Disorder with a focus on motor imagery initially using behavioral (e.g., reaction time) paradigms (Roelofs et al., 2001; Roelofs, van Galen, Keijsers, & Hoogduin, 2002) and later incorporating fMRI (de Lange et al., 2007). Motor imagery can be used to study movement generation because it has been shown to have a number of common characteristics with motor execution, for example, in performance duration and cortical networks (Jeannerod, 1997; Jeannerod & Frak, 1999) while controlling for processes such as sensory feedback (de Lange et al., 2007).

Roelofs et al. (2001) explored mental motor representations in motor Conversion Disorder. Participants were either implicitly cued or explicitly instructed to simulate a movement during two mental motor rotation tasks in addition to two control reaction time (RT) tasks. The implicit task required mental rotation of hands and feet images presented on a screen. In the explicit task, participants were instructed to mentally rotate their own hands and feet to the position of the images on the screen without actually moving. All tasks were measured using verbal responses, commonly used in RT paradigms, although a less precise measure of RT than manual responses, resulting in longer reaction times and larger standard deviations (e.g., Feyerisen, 1997). Although both verbal and manual RT measuring instruments can ensure an accuracy of within 1 ms, verbal responses involve various complex processing stages for the production of speech, which result in the formation of articulatory scores whereby articulation can be initiated through the activation of the laryngeal and supralaryngeal apparatus (Levelt, 2001).

Roelofs and colleagues found, in line with Kihlstrom’s cognitive theory of memory and dissociation (1992a; 1992b), that explicit (intentional or willed)

motor imagery was greatly disturbed (a general slowing of reaction time) compared with the implicit mental rotation task. In the explicitly cued task, patients reported an inability to imagine rotating their affected hand and foot on 9% and 51% of the trials, respectively. Significantly slower RTs were found on all RT tasks for patients compared with control participants; slower RTs were observed for the patients' affected arm compared with their unaffected arm in the explicitly instructed task. Mental rotation was slower in the implicit mental task. The finding that explicit motor functioning is disrupted is in agreement with Marshall et al. (1997) and Tiihonen et al. (1995), who suggested higher cognitive processes, for example intentionality was impaired in Conversion Disorder and implicated frontal inhibitory structures. These authors argued that mental motor representations are important for movement planning and preparation and concluded that the results of this motor imagery research suggest that intentionally generated movement is impaired and may "already manifest itself in movement preparation" (p. 23). While the explanation of an inhibitory frontocortical mechanism may hold true here, the research of Marshall et al. (1997) and Tiihonen et al. (1995), as reviewed in the previous section and in contradiction with Roelofs et al. (2001), proposed that in Conversion Disorder, the disruption of movement is due to impairment during the motor execution phase and that motor programs are generated normally and movement preparation is intact.

Subsequently, Roelofs et al. (2002) examined the notion that Conversion Disorder involves a dissociation between higher and lower level motor control that is not only symptom specific but can be symptom independent. Kihlstrom's dissociation theory refers to higher level processes as those that are explicit or intentional, whereas lower level information processes are those that are implicit or automatic (Kihlstrom, 1992a, 1992b). Based on their previous findings and according to dissociation theory, Roelofs and colleagues (2002) hypothesized that a specific slowing of motor initiation (described as involving motor planning and measured using reaction time to indicate the duration of motor preparation) and not a general slowing involving motor execution (measured using speech duration) would be observed in patients with Conversion Disorder. Verbal responses were recorded in all four conditions: an implicit mental rotation task, an explicit mental rotation task, and two control RT tasks. Patients had significantly slower RTs in all tasks (see also Roelofs et al., 2001) and were slower with their affected arm compared with their unaffected arm in the explicitly cued task. No significant effects were found for response duration.

Taken together, the results of Roelofs and colleagues are partially supportive of Marshall et al. (1997) and Tiihonen et al. (1995) in that Conversion Disorder is associated with dissociation between higher and lower level motor processes, with a specific impairment in explicit motor initiation. This occurred whether the response required a spatial or motor component that was either symptom specific or symptom independent. However, the finding by Roelofs and colleagues that motor preparation but not execution is affected in Conversion Disorder is inconsistent with Marshall et al. (1997) and Maruff and Velakoulis (2000), who indicated that the ability to generate motor plans in Conversion Disorder was intact but that initiation was disrupted during the motor execution phase.

Maruff and Velakoulis (2000) investigated the ability to volitionally control (explicitly instructed) real and imagined motor performance by measuring movement duration to 10 different target widths using the Visually Guided Pointing

Task (VGPT). Ten healthy participants were compared with a group of 10 healthy individuals instructed to feign a unilateral arm impairment, one participant diagnosed with Conversion Disorder exhibiting paralysis in his left arm and hand, and one participant with a left arm injury following a football collision. Results indicated that on both the real and imagined motor task, movement time for the healthy participants and the participant with a left arm injury conformed to Fitts' Law (Fitts, 1954). Similar movement times were observed for the real and the imagined task. In contrast, the group feigning an impairment showed a slowing of performance on the imagined task compared with the real task, but only the imagined task showed the pattern of results consistent with Fitts' Law. Similar results were reported for the patient with Conversion Disorder when completing the task with his affected arm.

These results indicate that although patients with Conversion Disorder and healthy subjects instructed to feign injury can intentionally slow imagined performance, they have little control over the speed and accuracy requirements (constraints) of the task. Based on Jeannerod (Jeannerod, 1994, 1997), who proposed that imagined movements are the stored internal representation of motor plans, Maruff and Velakoulis (consistent with Marshall et al., 1997) concluded that motor imagery and consequently motor planning remained intact in these individuals. Individuals who appear to have a motor deficit cannot anticipate correctly the effects of a structural limb impairment. Implementing a task that requires an objective assessment (e.g., whether the movement can be described by Fitts' Law) may prove useful as a diagnostic instrument to dissociate conversion symptoms from neurological motor symptoms.

de Lange et al. (2007) combined a mental rotation task with fMRI to investigate the difference in imagined actions between the affected and unaffected limb of eight conversion paralysis patients. Similar to previous findings (Roelofs et al., 2001), motor imagery evoked during the hand-laterality judgment task revealed no difference between hands in performance of RT or error rate. During motor imagery of the affected and unaffected hand, dorsal parietal and premotor cortex activity increased as a function of rotation angle. This indicated that the conversion patients could imagine both hands moving with equivalent cerebral activity in the motor systems of each hemisphere, contradicting Marshall et al. (1997), who found a decrease in preparatory activity as a result of excessive inhibition from activity in ACC and OFC.

Motor imagery of the affected hand also recruited activity in additional cortical areas independent of biomechanical complexity. This was observed in the superior temporal cortex, ventromedial (vmPFC) and dorsomedial prefrontal cortex, regions that normally decrease during cognitive tasks but "failed to deactivate" during motor imagery in the participants in this study. Rather than associating vmPFC activity with an inhibitory mechanism acting on the motor system (Marshall et al., 1997), de Lange and colleagues suggested this reflected heightened self-monitoring processes during imagery of the affected limb (Roelofs et al., 2006).



## Neural Mechanisms

The neural correlates of Conversion Disorder remain equivocal despite technological advances in brain imaging and sophisticated electrophysiological measurement techniques. In general, the data offer evidence that conversion symptoms are represented in the brain and that cortical dysfunction can be localized. However, the heterogeneous nature of the experimental designs employed and the patient samples that exhibit a diverse range of symptoms with coexisting disorders and (in many cases) the limited power in drawing general conclusions from case studies limit the interpretations (Vuilleumier, 2005).

Within these limitations, it appears that patients with motor Conversion Disorder exhibit different cortical activation patterns compared with healthy controls and different patterns in contrast to healthy participants feigning a deficit in voluntary movement. Two conflicting hypotheses have emerged (see also Broome, 2004; Stone et al., 2007): One is that a neural mechanism underlying Conversion Disorder is responsible for excessive inhibition of voluntary movement. That is, motor programs may be generated normally and later disrupted during the execution phase (Marshall et al., 1997); Tiihonen et al. (1995) suggested “higher” frontal brain regions inhibit “lower” brain regions. A second hypothesis proposes that a deficit in normal movement activation is due to abnormal motor preparation (Spence et al., 2000; Vuilleumier et al., 2001) and proposes that the inability to initiate and perform voluntary movement results from abnormalities in the genesis (rather than the execution) of motor programs and motor preparation. This hypothesis is supported by hypoactivation in the left DLPFC and a deficit in striatothalamocortical circuits (areas responsible for volitional movement), which impairs motor readiness resulting in slowed movement initiation.

A common idea associated with both hypotheses is that motor conversion paralysis is accompanied by a disruption between brain regions controlling intention and execution of movement (Krem, 2004). Both hypotheses implicate a network of areas in the prefrontal cortex, which function in a manner of “executive control” when “‘top-down’ processing is needed; that is, when behaviour must be guided by internal states or intentions” (Aron et al., 2004; Miller & Cohen, 2001). Given Conversion Disorder is considered a protean disorder, this begs the question whether there is a unique neural mechanism underlying the wide variety of conversion symptoms (motor or sensory) or whether functional and structural alterations in a number of neural networks can give rise to a distorted representation of body function as experienced in Conversion Disorder. It is plausible, perhaps likely, that the neural mechanism is itself heterogeneous. Broome (2004) has proposed a model that integrates the neurobiological underpinnings of these studies and engenders support for both hypotheses. Broome speculates the ACC (an area associated with motivation, intention, and effort) depends on activation of the OFC, which precedes decision making and motor initiation. ACC activation may, therefore, suppress activation of the DLPFC (an area important for action goal formation and the subjective awareness of volition), suppress hippocampi (which

may repress the memory of movement intention), influence motor function through their inputs into basal ganglia-thalamocortical circuits (Alexander et al., 1990), and therefore, inhibit the motor cortices, ultimately leading to the cessation of movement as evident in motor paralysis (Broome, 2004). The connections between the prefrontal cortex and basal ganglia play a cohesive role in modulating the functions of the frontal lobe and through the projections back to the motor cortex influence volitional goal-directed behavior. Perturbations in these circuits, for example, increased psychological stress inherent in Conversion Disorder, could, therefore, lead to “circuit-specific behavioural alterations” (Alexander et al., 1990). The parsimony of the combined model proposed by Broome (2004) awaits further research. Alexander and colleagues concluded, “It can be easily appreciated moreover, how lesions affecting different stations within a given circuit could result in a disruption of the same behavioural functions,” a statement very pertinent to the conflicting findings related to the neural mechanisms underlying Conversion Disorder.

Individuals with Conversion Disorder (with motor symptoms) have difficulties producing voluntary movement but it is unclear whether this is due to disruption during the motor preparation phase or the motor execution phase of movement initiation. Furthermore, the literature is inconsistent with respect to the neural mechanisms and neural processes that might underpin the motor symptoms of Conversion Disorder. One way to examine whether preparation, or execution, or both aspects of generating voluntary movement are affected is to use a behavioral design that can independently manipulate the nature (anatomical segment) and amount (number of decisions) of information provided to the participant before and during the “getting ready to move” period. The parameter precuing technique developed by Rosenbaum (1980; see also Leonard, 1953) provides a useful framework and can be used to investigate the effects of different movement parameters (such as the direction or extent of a movement) on motor preparation, initiation, and execution. This paradigm enables more detailed inferences about the construction of motor preparation before the onset of a specific movement response (Rosenbaum, 1980, 1983). Electrophysiological techniques (e.g., EEG and EMG) can be used to accurately measure and infer the temporal characteristics of brain and muscle activity contributing to motor preparation and performance. ERPs such as the CNV or BP can be derived from the EEG and are related to preparatory process in the motor and premotor cortical areas prior to action. These measures would permit the examination of the time course, amount of activity, and nature of motor preparation in individuals with Conversion Disorder. To our knowledge such investigations have not been conducted and offer potential for seeking greater understanding of the neurobehavioral nature of Conversion Disorder.

## Conclusion

Attempts to connect conversion symptoms with specific neural correlates or mechanisms date back to the 19th century, but a comprehensive model of how Conversion Disorder symptoms are represented in the brain is yet to emerge. From a neuromotor control perspective, the findings from behavioral, electrophysiological, and neuroimaging studies are not in agreement. Currently, two disparate

mechanisms have been invoked to explain motor Conversion Disorder: The inability to execute normal voluntary movement could be due to impairment during motor preparation *or* disruption during the phase of motor execution itself or from a complex interaction of both mechanisms.

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