Conversion disorder revisited

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Summary

Conversion disorder is defined as a psychiatric illness whose symptoms or deficits, affecting voluntary motor or sensory function, cannot be explained by a neurological or general medical condition. Proposing a strategy in the search for the neural mechanisms underlying conversion disorder is a difficult task, partly because key features of the illness inherently lie on a continuum with other psychiatric disorders, such as depression and posttraumatic stress disorder. Recent brain imaging studies have revealed neural circuits involved in complex mental processes potentially related to conversion disorder. These studies are reviewed, together with neuroimaging work in conversion disorder and brain imaging studies that have enriched the conceptualization of emotion and memory in posttraumatic stress disorder and major depression. Analysis of this information from a symptom-based rather than a disease-category perspective leads to a brain-based cognitive model of conversion disorder. This model suggests that disconnected crosstalk between the individual subdivisions of the anterior cingulate and the prefrontal cortex might provide a neuroscientific basis for the psychodynamic dissociation hypothesis, traditionally the bedrock explanation of the relationship between internal conflict and physical deficit. The model also suggests novel research approaches, as well as opportunities for potential therapeutic interventions.

\textbf{KEY WORDS:} anterior cingulate, depression, hysteria, neuroimaging, posttraumatic stress disorder, prefrontal cortex.

Introduction

Conversion disorder is characterized by the presence of symptoms or deficits affecting voluntary motor or sensory functions. Although this clinical condition suggests neurological or organic causes, it is thought to be associated with psychological stressors (DSM-IV-TR). Sigmund Freud, in 1895, emphasized the “psyche and soma” connection, proposing that conversion is the mechanism whereby an “internal” intrapsychic conflict is unconsciously, and thus unintentionally, “converted” into an “external” symptom (1). The psychodynamic concept of conversion has remained enshrined in international classifications, yet the pathophysiological mechanisms of the process remain largely obscure. Patients with medically unexplained neurological symptoms can account for up to a third of new attendees at a neurological clinic (2,3). In addition, the management of conversion disorder is complex and protracted, generating high costs to society and health funding agencies (4). The significant development of objective methods for submitting the mind-body relationship to testable experimentation has raised a crucial question with regard to conversion disorder: how can neuroscience provide a novel perspective that might help us begin to understand the biological substrates of this disorder (which in turn might offer new insights for monitoring the progress of therapeutic interventions, such as psychotherapy, body-oriented treatments and/or pharmacotherapy)?

It was again Sigmund Freud, in 1895, who first proposed linking mind and brain in the study of mental illnesses. In his work \textit{Project for a Scientific Psychology}, he suggested that the cognitive mechanisms of normal and abnormal phenomena could be explained through orderly and rigorous study of brain systems. However, in view of the state of neural sciences at the time, Freud abandoned his project as premature.

Now, more than a hundred years on, a relatively sophisticated picture of neuroscientific psychopathology is emerging thanks to advances in a variety of fields, including neuropsychology and brain imaging. In parallel, a new reading of the psychological and neuropsychological research of recent decades in domains such as memory and attention has allowed cognitive neuroscience to make remarkable progress in the unravelling of the brain structures and neural circuits involved in complex mental processes. Areas investigated include the repression of unwanted memories (5-7), the influence of emotions on behaviour (8,9), and the interaction between conflict awareness and conflict resolution (10,11).

Along with these advances, new models of illness mechanisms have been developed, giving rise to cognitive neuropsychiatry, a new research discipline, which explores the functional organization of psychiatric disorders within a framework of human cognitive neuropsychology and seeks to link this framework to the relevant brain structures and to their pathology (12). Adopting this approach, dysfunctions in specific cognitive do-
Earlier literature had already focused on the strong as-
version patients but not in the neurological patients (19).
groups, coexisting with affective symptoms in the con-
Scale than the group with established neurological dis-
significantly higher on the Hamilton Depression Rating
study, patients with motor conversion disorder scored
ria for an additional depressive disorder (18). In one
Many patients with conversion disorder also meet crite-
cannot be reversed at will (17).
functions affecting voluntary motor or sensory function.
importantly, major depression might provide a clue to
this clinical aspect, since depressed patients can also
manifest with involuntary disturbances of motor func-
tioning (24), which sometimes even escalate into ex-
treme psychomotor retardation with catatonia (25,26).
In addition, intense anxieties can be associated with cata-
tonia, a phenomenon that may be understood as an ex-
treme fear response, akin to the animal defence strate-
gy of tonic immobility (27). One case report in the litera-
ture has indeed described the association of several
episodes of catatonia with posttraumatic stress disorder
(28).
Clinical characteristics common to conversion dis-
order, depression and posttraumatic stress disorder
Patients with conversion disorder do not necessarily
manifest psychological distress over a loss of function
and, hence, may appear to be free from psychiatric dis-
turbances. This paradoxical absence of distress, despite
the presence of a physical deficit, has been described as “la belle indifférence”. Equally, the psychological fac-
tor may not be apparent at onset, only becoming evident
later on, when it emerges that there is a temporal rela-
tionship between the conflict and the onset of the symp-
tom. Although psychiatric behaviour can seemingly be
absent or masked, one key aspect, indicating that a re-
lation between conversion disorder, depression and
anxiety disorders plays a significant role, is the docu-
mented comorbidity of these illnesses. Anxiety symp-
toms common in patients with conversion disorder in-
clude panic attacks, nightmares and flashbacks (15).
Motor conversion has been reported in patients with
posttraumatic stress disorder (14,15). Although the
present review does not focus on psychological con-
cepts of dissociation, it is noteworthy that both conver-
sion disorder and posttraumatic stress disorder are trau-
ma-related disorders and as such share dissociative
phenomena, which from a psychodynamic perspective
are thought to reflect defence mechanisms serving the
function of reducing awareness of intolerable informa-
tion (16,17). In particular, dissociation involves an ap-
parent dysfunction in perception, memory or action that
cannot be reversed at will (17).
Many patients with conversion disorder also meet crite-
ria for an additional depressive disorder (18). In one
study, patients with motor conversion disorder scored
significantly higher on the Hamilton Depression Rating
Scale than the group with established neurological dis-
ease. Interestingly, denial was high in both the patient
groups, coexisting with affective symptoms in the con-
version patients but not in the neurological patients (19).
Earlier literature had already focused on the strong as-
sociation between depression and conversion disorder
(20,21). In addition, it had been argued that conversion
disorder could be more easily conceptualized as masked depression (22).
This concept is now rising to prominence once
again, given that antidepressant pharmacotherapy may
facilitate recovery from conversion disorder symptoms
(13,23), supporting the view that a mood disorder rather
than conversion might constitute the primary psychiatric
disorder (23).
Within this context, there is a further key issue to consid-
er: in important symptom domains, too, conversion dis-
order involves impairments that may present similarities
with those described in depression and posttraumatic
stress disorder. The DSM-IV-TR definition of conversion
disorder emphasizes the presence of symptoms or
deficits affecting voluntary motor or sensory function.
importantly, major depression might provide a clue to
this clinical aspect, since depressed patients can also
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ture has indeed described the association of several
episodes of catatonia with posttraumatic stress disorder
(28).
Furthermore, the DSM-IV-TR definition of conversion
disorder implies that psychological distress is associat-
ed with the onset or exacerbation of the somatic symp-
tom. Traditionally, the hypothesis of conversion sug-
gests that the symptom provides symbolic relief from an
intolerable internal conflict. In Freud’s terms, however, in
which conversion disorder is an unconscious process,
the patient is unable spontaneously to make this con-
nection. Trauma-related amnesia in posttraumatic
stress disorder may be seen, at least in part, as an un-
conscious strategy to avoid trauma-related information
disengaging attention from negative emotions asso-
ciated with the traumatic event (29). Certain features of
traumatic events have been associated with specific
memory dysfunctions. It has been suggested, for exam-
ple, that abuse by a parent or caregiver is more often ac-
companied by amnesia of the event than abuse in which
the perpetrator was unknown to the victim (30). Inter-
estingly, there is evidence that types of trauma such as
childhood abuse can lead to conversion disorder (31,32),
major depression (33,34) or posttraumatic stress disor-
der (35,36).
In addition, difficulties with mental control and inhibition
are core symptoms of conversion disorder (37), post-
traumatic stress disorder (38) and major depression
(39,40). Indeed, each of these disorders has been linked
to deficits in the ability to regulate or suppress un-
wanted thoughts. Intrusions and flashbacks, as well as
efforts to avoid thoughts and feelings associated with
the trauma, are characteristic symptom features of post-
traumatic stress disorder. Similarly, rumination about
the negative life event that may have preceded a depres-
sive episode has been implicated as a causal feature in
anxiety could serve as a stimulus for innovative re-
search in conversion disorder.
Focusing on motor conversion disorder, the present re-
view aims to address three main issues: i) How, from a
clinical point of view, might conversion disorder be relat-
ed to depression and posttraumatic stress disorder? ii)
To what extent may recent achievements in brain imag-
ing studies of normal cognitive processes and of de-
pression and anxiety contribute to the development of a
brain-based cognitive model of conversion disorder? iii)
Is there evidence that neuroimaging with its ability to vi-
sualize mental processes might support the hypothesis
that therapeutic interventions, in particular psychothera-
py, impact upon neurobiological substrates that are dys-
functional in conversion disorder?
To what extent may recent achievements in brain imag-
ing studies of normal cognitive processes and of de-
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Neural networks implicated in suppressing memories

The repression of unwanted memories is a concept central to psychoanalytic thought. Freud’s theory implied that “the essence of repression lies simply in turning something away, and keeping it at a distance from the conscious” (42). Freud provided many examples of memory repression from clinical cases, giving detailed descriptions of their behaviour patterns (43). However, the existence of memory repression has remained controversial for more than a century, partly due to the unresolved interdependence between traumatic experience and internal conflict, and the ethical and practical difficulties of studying the process in controlled experiments. What was needed was a way to investigate memory repression in the laboratory.

Two recent studies have elegantly explored the underlying inhibitory mechanisms and have begun to identify the main features of a neurobiological model of memory control that people possibly use to adapt their mental environment in response to traumatic experience (5,6). Using an event-related functional magnetic resonance imaging (fMRI) design (think/no-think task), the authors showed that when people encounter cues (one word of a learned pair) that bring to mind an unwanted memory (the associated word of the pair) and are asked to suppress the memory of that word, later recall of the rejected memory becomes more difficult. The results established a direct link between internal operations that control awareness of a memory and its later accessibility, thereby supporting a suppression mechanism that pushes unwanted memories out of consciousness, as posited by Freud. It is interesting to note that the authors’ studies were inspired by a cognitive analysis of psychogenic amnesia in abused children. As noted above, children abused by a trusted caregiver are more likely eventually to forget the abuse than those maltreated by strangers. The only way to prevent recall of the traumatic memories would thus be to adapt internally and deliberately avoid thinking of such memories – in Freud’s terms, to push them into the unconscious.

Repressing memories is a complex act that recruits a network involving increased dorsolateral prefrontal activation and reduced hippocampal activation (6). The hippocampus is important for declarative memory formation (such as the capacity to recognize recently encountered material as familiar) (44), and increased hippocampal activation is associated with increased memory formation (44). The dorsolateral prefrontal cortex is involved in critical executive functions, such as implementation of control (45), switching attention (46,47), and overcoming interference from competing representations in a range of cognitive tasks (48,49). It is worth noting that the suppressing of unwanted memories might recruit more than the dorsolateral prefrontal cortex to disengage hippocampal processing. Indeed, the authors also indicate the anterior cingulate as playing a crucial role in this network of memory suppression, signalling to the dorsolateral prefrontal cortex the need to implement strategies in response to memory intrusions (45) and/or mediating interactions between the dorsolateral prefrontal cortex and the hippocampus. These possibilities are consistent with the dense bidirectional projections of the anterior cingulate to medial temporal lobe structures, such as the hippocampus (50). Extending these investigations, Wyland et al. (7) found increased activity in the anterior cingulate when subjects were engaged in suppressing a specific, salient thought.

Functional abnormalities common to conversion disorder, major depression and posttraumatic stress disorder

On the basis of functional imaging findings, the prefrontal cortex has been proposed as a candidate region for involvement in conversion disorder, although the exact nature of this involvement is debated (51-54). In a positron emission tomography (PET) study, Spence et al. (52) reported selective deactivation of the dorsolateral prefrontal cortex, implying that conversion motor symptoms may reflect failed engagement of this cortical structure in volition control. Importantly, within the prefrontal cortex, distinct areas, particularly the orbitofrontal cortex and the anterior cingulate, might exert a powerful effect in conversion disorder. One PET study by Marshall et al. (51) of a patient with left-sided “hysterical” paralysis found that when the patient tried to move her affected leg, abnormal activity was recorded in the right orbitofrontal cortex and anterior cingulate. This abnormal activity was deemed responsible for inhibiting the patient’s volitional movements. In addition, reduced activity in the basal ganglia and thalamus observed in a single photon emission computerized tomography (SPECT) study was interpreted as a possible downstream effect of primary dysfunction of the orbitofrontal cortex and anterior cingulate in response to emotional stressors, thereby ultimately explaining the actual implementation of the motor inhibition (54).

In accordance with the proposed conceptual link between conversion disorder, mood disorders and anxiety disorders, it is worth noting that the prefrontal cortex plays a well-documented and critical role in major depression and posttraumatic stress disorder. In particular, the bulk of the literature on brain structure and function in depression has consistently shown abnormalities of the anterior cingulate (55-57) and the orbitofrontal cortex (58-60). There is also evidence that catatonic symptoms may be closely related to dysfunction in the orbitofrontal cortex (9); these findings add further support to the hypothesis that similar symptoms observed in different disease conditions share a common neural substrate.

Likewise, a growing body of evidence from neuroimaging research involving re-experiencing paradigms and traumatic reminders in posttraumatic stress disorder has found functional (56,61-63) and structural changes at the level of the anterior cingulate (64), as well as functional abnormalities of the orbitofrontal cortex (65,66).

Does the psychodynamic hypothesis reflect dissociation within a cortical conscious monitoring system?

Therefore, a common thread in these observations, spun from clinical and neurological starting points,
might be that impaired connectivity in specific prefrontal cortical networks of emotional-cognitive processing is mirrored psychologically by an inability properly to integrate negatively valenced thoughts and memories. Psychodynamic thought posits that the conversion symptom is a symbolic expression of “painful affects” related to the trauma (37). In other words, patients, instead of experiencing and describing their particular feelings, express their anxieties and conflicts through a physical symptom; this would indicate that they lack insight into its causation (4). This psychodynamic hypothesis might provide a testing ground for theories of a dissociation within a cortical conscious monitoring system in which the anterior cingulate plays a crucial role. The anterior cingulate is engaged in linking emotional information to changes in behaviour (67). More specifically, the anterior cingulate is thought to be particularly involved in the conscious self-regulation of behaviour (10), detecting conflicts between plans of action and acting as a trigger for consequent enhanced recruitment of cognitive control in the lateral prefrontal cortex (68).

However, an important problem arises when aversive emotional states (e.g., anxiety) interfere with this process. To examine this issue, it must be noted that the anterior cingulate has two major subdivisions (69), the dorsal cognitive and the rostral-ventral affective. The dorsal cognitive subdivision, which has strong reciprocal interconnections with the lateral prefrontal cortex, parietal cortex, and premotor and supplementary motor areas, is mainly implicated in cognitive processing, including complex motor control. More specifically, the dorsal cognitive subdivision has a role in cognitive mechanisms believed to be important in appropriately modifying or altering behaviour (70-72). The rostral-ventral affective subdivision, instead, maintains extensive connections with limbic structures, such as the orbitofrontal cortex and the amygdala, and is primarily involved in emotional processing. This includes regulation of emotional responses and assessment of the salience of emotional information (73), such as that coming from the orbitofrontal cortex, a crucial trigger site for emotion and particularly important when the emotional stimulus is recalled from memory (49). Importantly, another function of the affective anterior cingulate subdivision is to respond to unexpected processing conflict caused by salient emotional stimuli (74). However, when emotions are particularly strong, an emotional conflict detected by the affective subdivision may not be followed by successful cognitive control through recruitment of the lateral prefrontal cortex. This view fits in with the findings of Bishop et al. (74), who showed that heightened anxiety was associated both with lower activity in the affective anterior cingulate subdivision and with reduced recruitment of control mechanisms in the lateral prefrontal circuitry. Thus, anxiety is characterized by weaker activation of the cognitive control mechanisms required in order to maintain ongoing task processing in the presence of threat-related stimuli (74). From this perspective, it may be hypothesized that reduced recruitment and dissociation of this circuitry might also provide a possible neural basis for the psychodynamic hypothesis that conversion symptom generation is the surface manifestation of unconsciously disconnected traumatic memories. Indeed, strong activation of prefrontal areas, together with reduced hippocampal activity, is thought to account for successful conscious memory suppression in healthy individuals (6).

Furthermore, it might be argued that the affective and cognitive subdivisions of the anterior cingulate need to interact with one another in order to provide control of cognitive processing in the presence of an emotional component. Previous studies have shown decreased activation of the affective subdivision in patients with posttraumatic stress disorder (63). On the other hand, deactivation of the cognitive subdivision of the anterior cingulate has been observed during intense emotional states and in patients with depression (75,76). This is an important issue to consider, since the cognitive subdivision of the anterior cingulate is active in detecting when unfavourable circumstances call for a change of behavioural strategy (77).

Towards a brain-based cognitive model of conversion disorder

Any attempt to define a picture of neuroscientific psychopathology, especially when functional neuroimaging is the methodology used, requires a model of deficits vis-à-vis normal cognitive mechanisms (and possibly an a priori hypothesis about the neural substrates involved) that accounts for the psychopathology observed in a given illness. Proposing a strategy in the search for the neural mechanisms underlying conversion disorder is a difficult task: indeed, according to a symptom-based approach, conversion disorder can share clinical characteristics with other psychiatric disorders. However, the above considerations relating to the cortico-cortical circuitry raise the intriguing possibility that some form of disconnected crosstalk between the individual subdivisions of the anterior cingulate and the prefrontal cortex accounts for the relationship between the (overt or masked) emotional abnormalities and the motor deficit in conversion disorder. Within this context, it is also important to emphasize that extensive investigations point to the anterior cingulate as a primary structure involved in motor-related functions, such as preparation and execution of movements (78,79). Specifically, one of the unique features of the anterior cingulate is its diverse thalamic afferents and consequent ability to sample inputs from more thalamic nuclei than any other cortical region (67). This ability to sample from a wide range of thalamic inputs may be crucial in its contribution to motor response selection functions. Additionally, the anterior cingulate has projections to all major components of the striatum (67). Interestingly, behavioural deficits induced by circumscribed lesions to the primate anterior cingulate seem to interfere with the ability to initiate movement spontaneously (79). Similarly, patients with medial frontal lesions involving the cingulate cortex often show deficits in spontaneous movement and speech. Akinesia mutism, which is caused by bilateral lesions, is an extreme example of this syndrome (80,81). Furthermore, the example of catatonia may provide an interesting conceptual link to conversion disorder. According to Northoff (25), catatonia can be conceptualized as the failure of cognitive control of negative emotions due to disturbed cortico-cortical interactions (with the main contribution of the or-
bitofrontal cortex), thereby fostering less sophisticated forms of emotional processing that involve the motor system. Northoff (25) further extends his hypothesis by stating that “an analogous shift from cognitive control to motor control of emotional processing may be observed in hysterical paralysis”. This kind of replacement is psychodynamically interpreted as “sensorimotor regression”.

Experimental paradigms used in posttraumatic stress disorder and major depression may increase our understanding of conversion disorder

Our model of conversion disorder draws on the DSM-IV-TR definition that emphasizes the role of a conflict or stressor in the onset of the somatic symptom. Therefore, if future work is to further our understanding of the biological basis of conversion disorder, a central focus of research should be investigation of the trauma- or conflict-related mechanisms underlying the disorder. Because the brain mechanisms implicated in posttraumatic stress disorder are partially understood, findings in this regard could provide some leverage for exploring the nature of the brain disturbances that are involved in the aftermath of a trauma in conversion disorder. In particular, the intense surge of interest in posttraumatic stress disorder with brain imaging, over the past decade, has led to the development of a variety of experimental paradigms, involving the recollection and imagery of traumatic events (from various types of trauma) and the comparison of traumatic memory recall with other emotional states (62,82). Masked facial stimuli (for the subliminal presentation of fearful or angry faces) have been used to assess implicit (unconscious) emotional processing through the amygdala (83), whereas other tasks, such as the emotional variants of the Stroop task and cognitive interference tasks, have been employed to examine activity selectively in different regions of the anterior cingulate (8). Similarly, psychological paradigms for investigating emotional processing and disturbances in memory encoding have been developed in major depression (56,59,63,84).

However, use of the experimental paradigms that have enriched the conceptualization of memory and emotion in posttraumatic stress disorder and major depression may also shed light on the neural pathways linked to trauma and memory in conversion disorder. Indeed, this kind of approach might also help clarify whether, how, and the extent to which conversion disorder shares common neural substrates with mood and anxiety disorders. Some association of physical movement from normal conscious volition, thereby leading to unwilled motor paralysis (90). Importantly, data from single-patient case studies using brain imaging suggest that hypnosis and conversion disorder may also share common neurophysiological mechanisms involving prefrontal regions (51,91,92). Although this body of evidence is meagre, the proposed involvement of common anatomical sites suggests that hypnosis may enhance the treatment of conversion disorder symptoms. One randomized controlled clinical trial has observed improvement of motor conversion symptoms under hypnosis-based treatment (93). Unfortunately, neural measures were not assessed. Nevertheless, one PET study investigating the antinociceptive effects of hypnosis has shown that pain reduction during the hypnotic state was associated with a significant increase in functional connectivity within a network encompassing the affective and cognitive subdivisions of the anterior cingulate, the prefrontal cortex, as well as the insula, the presupplementary motor area, the striatum and the thalamus (94). On the basis of neuroimaging data, a reversal of the dysfunction in both the affective and the cognitive subdivisions of the anterior cingulate has been suggested to be a key neurobiological mechanism in the efficacious psychotherapeutic relief of distress (95). Moreover, current progress in neuromodulation suggests that behavioural treatments of movement disorders may change the functional organization of somatosensory neural networks (96). Additionally, it may be worthwhile to cite one study that employed fMRI to examine the neural circuitry implicated in reappraising highly negative scenes in unemotional terms (97). Reappraisal of highly stressful conflicts reduced subjective experience of negative affect. Interestingly, neural correlates of reappraisal were increased activation of both the anterior cingulate and the prefrontal cortex, together with decreased activation of the amygdala and orbitofrontal cortex (97). These results might specifically support the hyp-
Controversy will also surround the question of how co-impact does duration of the stressor have? What impact psychoanalytic theory) or inflicted by another human mainly internal (as it is conceptualized, for instance, in somatic pathology. Yet, researchers will need to struggle with many issues. How severe should a trauma be? Does it make a difference if the trauma or conflict is can produce a neurological symptom in the absence of somatic pathology. Yet, researchers will need to struggle with many issues. How severe should a trauma be? What types of trauma might be considered causative? Does it make a difference if the trauma or conflict is mainly internal (as it is conceptualized, for instance, in psychoanalytic theory) or inflicted by another human being, by an accident, or by a natural disaster? What impact does duration of the stressor have? What impact does premorbid psychiatric status have? Controversy will also surround the question of how co-morbidity should be dealt with. It may be hypothesized, for example, that preexisting major depression may render the two individuals more vulnerable to conversion disorder in the aftermath of a trauma. Interestingly, in the single-patient case PET study by Marshall et al. (51) a significant history for episodes of depression was reported, some of which were accompanied by mutism and lower limb weakness. Conversely, the presence of conversion disorder may increase the risk of first onset of depression. In the SPECT work by Vuilleumier et al. (54), past psychiatric illness represented an exclusion criterion, however, depressed mood was reported in five of the seven patients included in the study. In short, it might be extremely interesting to examine the extent to which each disorder increases susceptibility to the other, thereby shedding light on the possibility of a shared vulnerability to these disorders.

Indeed, data from several independent studies have shown that childhood abuse is a well-established risk factor for conversion disorder, as well as for major depression and posttraumatic stress disorder. Therefore, future studies should also aim to explore whether similar traumas may map onto a common “traumatic” neural circuit, and if/how risk factors linked, for example, to personality traits (or even personality disorders) might account for additional brain changes (perhaps in other brain sites), thereby making one individual develop conversion disorder and another posttraumatic stress disorder. Finally, research into genes and familial environment could be particularly enlightening in the specific context of conversion disorder, because it has been frequently observed that a family member with a similar history can serve as a “model” of conversion (4). Providing answers to these questions will require well-defined clinical populations. This might be one of the greatest challenges, but it should help focus research on potentially important brain circuitry and on the development of better diagnostic biomarkers, and allow patients to be treated with novel, more selective (and effective) tools at earlier stages in their illness.

Concluding remarks

In line with our brain-based cognitive model of conversion disorder, we suggest that focusing research on the conflict or stressor that precedes the onset of the somatic symptom would help establish a integrated overall understanding of how abnormal psychological states can produce a neurological symptom in the absence of somatic pathology. Yet, researchers will need to struggle with many issues. How severe should a trauma be? What types of trauma might be considered causative? Does it make a difference if the trauma or conflict is mainly internal (as it is conceptualized, for instance, in psychoanalytic theory) or inflicted by another human being, by an accident, or by a natural disaster? What impact does duration of the stressor have? What impact does premorbid psychiatric status have? Controversy will also surround the question of how co-morbidity should be dealt with. It may be hypothesized, for example, that preexisting major depression may render the two individuals more vulnerable to conversion disorder in the aftermath of a trauma. Interestingly, in the single-patient case PET study by Marshall et al. (51) a significant history for episodes of depression was reported, some of which were accompanied by mutism and lower limb weakness. Conversely, the presence of conversion disorder may increase the risk of first onset of depression. In the SPECT work by Vuilleumier et al. (54), past psychiatric illness represented an exclusion criterion, however, depressed mood was reported in five of the seven patients included in the study. In short, it might be extremely interesting to examine the extent to which each disorder increases susceptibility to the other, thereby shedding light on the possibility of a shared vulnerability to these disorders.

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