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Understanding conversion disorder: How contemporary brain imaging is shedding light on an early Freudian concept

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ABSTRACT

Conversion disorder, also called Functional Neurological Symptom Disorder is poorly understood by many in the medical profession and is associated with considerable health care costs. Sigmund Freud, in an early, pre-psychoanalytic period paper, suggested that *hysterical motor paralyses* arose from a "functional or dynamic lesion" which was no different from an organic one, but rather an altered expression of it. He linked this functional brain disturbance to an excess of affect, a faulty conceptualization on the part of the symptomatic individual of how the affected organ works, and elements of dissociation and dual consciousness. One hundred and thirty years later converging functional imaging studies provide support for the excess affect component of his hypothesis. A small but growing fMRI literature has revealed bottom-up hyperactive neural activity in limbic regions and a potential failure of top-down regulation from prefrontal regions. Aberrant functional connectivity of limbic-motor regions now provides a mechanistic model that sheds light on an early Freudian theory explaining, in part, how symptoms of Conversion Disorder arise.

Conversion disorder, also called Functional Neurological Symptom Disorder and until 1980, Hysterical Neurosis, is common in adults and pediatric populations, and accounts for an estimated $1.2 billion in annual emergency department and inpatient costs in the USA. (Stephen et al., 2021) Recent advances in functional brain imaging have led to new mechanistic understandings of how symptoms arise. Concurrent with these advances has been a move away from once dominant psychoanalytic and Freudian concepts of the disorder with the emphasis on constructs like primary and secondary gain and the putative etiological role of early-life traumas, typically sexual (Stone and Edwards, 2011). The waning Freudian influence is best captured by successive iterations of the Diagnostic and Statistical Manual of Mental Disorders (DSM). This conceptual shift is reflected in the DSM5, where reference to "conflicts or other stressors" preceding symptom onset is no longer considered an essential diagnostic criterion for the disorder (American Psychological Association (APA), 2013).

What is informative in this historical re-evaluation of Freudian thinking is an early paper by Freud written when he was a still a practicing, conventional neurologist (Freud, 1893a). It can also be found in volume 1 of The Standard Edition of the Complete Psychological Works of Sigmund Freud: Pre-Psycho-Analytic Publications and Unpublished Drafts (Freud, 2001). In light of what contemporary brain imaging tells us about the neural origins of conversion symptoms, revisiting Freud's careful neurological observations and theorizing that predate his psychoanalytic period is helpful in rounding out a more complete mechanistic understanding of how conversion symptoms arise.

In 1885 the 29-year old Sigmund Freud was given a travel grant by the Faculty of Medicine in Vienna. Firmly rooted in neurology and with research interests in histology and neuroanatomy, Freud set off for the Salpetriere Hospital in Paris. On his return at the end of March 1886, he wrote a note of thanks to his sponsors informing them that he had hoped to study "secondary atrophies and degenerations that follow on affections of the brain in children." (Freud, 1893b) While the pathological material was available to him, the laboratory facilities were not and instead he had turned his attention to the rich and diverse clinical material offered by the Salpetriere's 5000 residents. Encouraged by Jean-Martin Charcot, who held the Chair in Neuropathology at the Salpetriere, he narrowed his inquiry to understanding the perplexing neurological basis to hysteria.

Freud came away from Paris in thrall his teacher. "Charcot, who is both one of the greatest of physicians and a man whose common sense is the order of genius, simply demolishes my views and aims" he wrote to

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Martha Bernays, his future wife. "Many a time after a lecture … I have no more wish to work at my own simple things. My brain is sated as after an evening at the theatre. Whether the seed will ever bring forth fruit I do not know; but what I certainly know is that no other human being has ever affected me in such a way." (Freud, 1953) Passages like these suggest that it was Charcot's influence which led Freud to move away from neurology into psychopathology (Jones, 1953). The admiration did not stop there. Three years after leaving Paris, Freud named is first son after the great neurologist. But first, there were two publications related to Charcot that required Freud's attention. On his return from Vienna, Freud quickly translated the third volume of Charcot's Salpetriere lectures into German and had them published in German before they came out in French (Charcot, 1886).

The subject of the second publication was either directly suggested by Charcot, or else owes much to Charcot's influence (Strachey, 2001; Koehler, 2003). It was likely first written in French and entitled "Quelques considérations pour une étude comparative des paralysies motrices organiques et hysteriques." (Freud, 1893a) The history of this paper and its delayed publication, occupying a watershed period in Freud's career, is particularly informative in elucidating the shift in his conceptualization of hysteria and his nascent attempt at finding a rational, cerebral cause for the disorder. Freud initially made reference to this paper in his Easter 1886 report to his sponsors (Freud, 1893b) but it took seven years before the paper was published. In the intervening period he made sporadic reference to it in his correspondence, but never divulged the reason for the delay (Strachey, 2001). The content and structure of the paper, however, contains clues that could explain the lengthy hiatus.

The paper is divided into four sections. In the first three Freud focuses on the clinical signs that delineate an organic motor paralysis from a hysterical one. He precisely dissects that anatomical and clinical inconsistencies that define hysterical paralyses contrasting them with the well-defined signs and symptoms of an organic one. Towards the end of the third section he quotes Charcot who maintained that hysterical paralyses arose from cortical lesions that were purely functional or dynamic, hence their absence at post-mortem. Freud agrees and cites edema and active hyperemia as examples of cerebral processes that can give rise to dynamic lesions. However, he goes on to note that paralyses caused by edema, for example, would still have to display the characteristics of an organic paralysis and could not produce the "dissociation and intensity of hysterical paralyses." Clearly, a more complete explanation was needed to reconcile these inconsistencies. In section four of his long-delayed publication, Freud provides it, and in it we see a shift in his thinking from the empiricism that until then had defined his career as a neurologist to the theorizing that would come to distinguish his subsequent career in psychiatry. The process was gradual, unfolding over a number of years and was influenced during this period by his collaboration with Breuer (Breuer and Freud, 1957) and, in particular, the theories of Pierre Janet (1920)

"I will attempt to indicate, finally, what the lesion that is the cause of hysterical paralyses might be like" writes Freud in his concluding section. He goes on to invoke the concept of a "functional or dynamic lesion" suggesting that it was no different from an organic one, but rather an altered expression of it. With reference to Janet, Freud agrees that what is amiss in hysterical symptoms is the patient's conception of how the body works, one that is not based on a deep knowledge of neuroanatomy. The lesion in a hysterical paralysis of an arm, for example, is therefore an alteration of the conception, or the idea, of the arm. This last point is of crucial importance. It has been suggested that Freud could not have completed his paper until he had developed an original theoretical framework to explain hysterical symptoms (Macmillan, 1990). He found it in Janet's theory. Janet had recently suggested that the anatomically atypical nature of hysterical symptoms could be explained by the faulty idea a person had about the organ affected. With reference to hysterical blindness for example, he observed that the "… anesthesia bears not only on the retina, but on the conjunctiva and even on the eyelids: the amaurotic hysterical has spectacles of anesthesia on the face" before going on to conclude "… the ideas we conceive of our organs play an important role and determine [symptom] distribution." (Janet, 1892) This concept in turn appeared in Freud's paper, published one year after Janet's paper. "There is no doubt" Freud opined, "that if the material conditions corresponding to the conception of the arm are profoundly altered, the conception will also be lost." And to Freud, the disruptive condition he refers to is a disturbance in affect.

A number of examples, one comical, are given to illustrate the point: the loyal subject who refuses to wash his hand because his sovereign has touched it; the breaking of the glass from which we have drunk the health of a young married couple; the burning of a dead chief's horse, weapons, even his wives along with his dead body. To Freud, "the force of all these actions is clear. The quota of affect, which we attribute to the first association of an object has a repugnance to letting it enter into a new association with another object and consequently makes the idea of the (first) object inaccessible to association." Having arrived at the crux of his argument, Freud goes on to reiterate it, expand on it and touch on the how the symptoms can be successfully treated with "hypnotic psychotherapy." In doing so, the word "subconscious" enters the Freudian lexicon for the first time thereby signifying the symptoms are involuntary. Using italics to emphasize key points in his argument Freud notes that "… in every case of hysterical paralysis, we find that *the paralyzed organ or the lost function is involved in a subconscious association which is provided with a large quota of affect and it can be shown that the arm is liberated as soon as this quota is wiped out*." Should the individual be unsuccessful in processing this surplus of affect, the retained subconscious memory of this failure is likened to 'psychical trauma.' Freud's originality here was to take Janet's concept of a faulty concept and link it to affect, dissociation and a dual consciousness (Macmillan, 1990).

Nineteenth-century scientists used drawings to record what they observed through a microscope and formulate hypotheses for things they could not see (Gamwell, 2006). Freud was no exception and from his drawings we see his career transformation from the objective certainty afforded by his initial histological and subsequent anatomical observations prior to his Charcot period (see Fig. 1A) to his theoretical sketches on his return from Paris, in which he attempted to explain how the relationship between the body and cortex was not topological, but functional (Fig. 1B). This break with empiricism was not comfortable for Freud, but necessary to explain what he saw as the neural origins of hysteria (Solms, 2006). Affect could be observed clinically, but like all mental phenomena could not be seen down a microscope or at post-mortem. Having outlined his theory as to the origins of hysterical symptoms, he concluded that "… the lesion in hysterical paralysis must be entirely independent of the anatomy of the nervous system."

Deconstructing Freudian theories of Conversion Disorder in contemporary neuroscience allows us to approach questions with scientific rigour and systematically test hypotheses. Several critical elements are relevant. Freudian constructs applicable to Conversion Disorder that have been studied is this manner include the idea of aberrant expectation, namely that the neurological symptom represents the patient's abnormal expectation or conceptualization of how a symptom should present. This sets the atypical presentation apart from typical neurological symptoms that match the underling neuroanatomy (Baek et al., 2017; Voon et al., 2016; Pareés et al., 2012). In addition, a core principle of Conversion Disorder, namely that symptoms are involuntary or unconscious rather than deliberately produced has also been explored (Voon et al., 2010a). Here we focus on the Freudian concept elucidated in our introduction above, that an excess of affect may underlie functional symptom production.

The most commonly studied forms of Conversion Disorder are functional movements such as tremor, weakness, gait disorders or dystonia, and psychogenic non-epileptic seizures characterized by intermittent episodes. Both presentations are marked by high comorbidity with depression and anxiety, disorders characterized by aberrant emotional processing. A meta-analysis of studies with in-depth interview methods highlighted a history of childhood maltreatment,

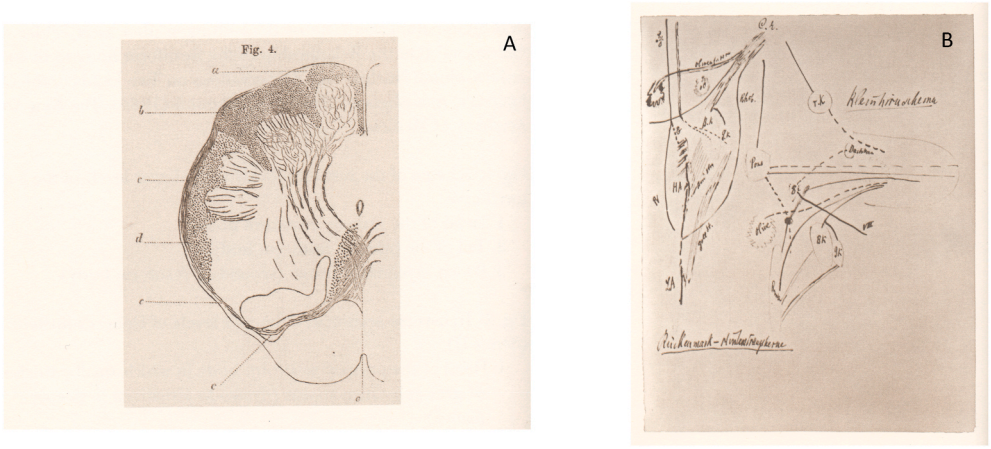

Fig. 1. Two drawings by Freud showing his academic shift from precise anatomical depictions (A) to theoretical attempts at explaining the functional relationship between the body and cortex (B).

particularly emotional neglect relative to physical or sexual abuse, along with greater stressors prior to the onset of the disorder (Ludwig et al., 2018). Conversion Disorder is associated with multiple pathophysiological mechanisms suggesting that several pathways might lead towards the expression of the disorder (Voon et al., 2016; Espay et al., 2018a). Although not all patients report a history of trauma, preceding stressors or have comorbid psychiatric disorders, brain imaging and electrophysiological examination can provide neural insights into aberrant emotional processing and potential mechanistic links with functional symptom expression.

Early functional MRI studies in Conversion Disorder on sensorimotor processing in motor and sensory symptoms highlighted a potential role for aberrant emotional processing and limbic motor integration. These studies using vibrotactile stimulation demonstrated abnormal activity in regions potentially implicated in integration of limbic and motor processing such as the caudate (Vuilleumier et al., 2001) or in paralimbic regions, including the anterior cingulate and anterior insula (Ghaffar et al., 2006; Burke et al., 2014).

Several key imaging studies were then designed to target emotional processing. The first fMRI study to focus on emotional processing assessed facial expressions known to activate limbic or emotional processing regions. When people with conversion disorder were compared to healthy individuals, the former showed greater activity in the amygdala, a phylogenetically old region involved in processing emotion and arousal and assigning importance to stimuli. Unlike the control group, they also failed to habituate over time to the stimuli (Voon et al., 2010b). Crucially, there was also greater functional connectivity between the amygdala and the supplementary motor area (SMA), a region implicated in motor preparation, thus highlighting abnormal interactions between limbic and motor regions. This could in theory explain how pathological affect altered motor symptom production. Indeed, in primate studies and in a human tractography study, direct white matter connections are observed between the amgydala on the one hand and the primary motor cortex, motor cingulate and post-central gyrus on the other (Grezes et al., 2014). These amygdala and SMA hyperactivity findings, including aberrant functional connectivity in response to emotional stimuli, have been replicated (Aybek et al., 2015; Hassa et al., 2017). Aberrant activity and connectivity between paralimbic regions implicated in a saliency network (periacqueductal grey, insula), cognitive control regions (cingulate, lateral prefrontal cortex) and motor regions (precentral gyrus, putamen and cerebellum) both in response to emotional stimuli (Aybek et al., 2015; Espay et al., 2018b, 2018c; Szaflarski et al., 2018), aversive losses (Morris et al., 2017), to a stress task (Allendorfer et al., 2019) and at rest (Diez et al., 2019) have been shown. Beyond functional differences, structural abnormalities in Conversion Disorder likewise highlight similar neural regions. For example, cortical thickness abnormalities have been shown in the anterior cingulate (Perez et al., 2018) while correlations between anterior insula volumes and physical health severity ratings on the one hand and between amygdala volumes and mental health and anxiety symptoms on the other, have been noted (Perez et al., 2017). A history of early life physical abuse further correlates with aberrant resting connectivity between the amygdala, insula and motor cortices, and also between limbic and cognitive control regions (Diez et al., 2020).

Several studies have attempted to explore this specific emotion-motor link. Aberrant amygdala-supplementary motor area activity and connectivity has been observed during motor preparation (Voon et al., 2011) and more specifically in a task condition of emotional stimuli paired with passive movement of a functional hemiparetic limb (Hassa et al., 2017) thus linking negative emotion with a conversion-motor symptom. Negative emotional imagery paired with force grip as a measure of motor control, was abnormally sustained in patients as compared to the decay observed in healthy individuals (Blakemore et al., 2016). Attempting to downregulate emotions in an MEG task similarly was associated with lower frontal alpha activity and hyperactvity of sensorimotor regions relative to healthy controls (Fiess et al., 2015). Abnormal physiological responses such as a startle response to affective imagery further emphasize the link between emotional and motor brain regions (Seignourel et al., 2007).

An imaging study directly focusing on abnormal affect and 'repressed memory' compared stressful 'escape' events with other threatening control events (Aybek et al., 2014). People with conversion disorder were asked to review their personal stressful life events. Researchers then determined whether the life event was linked to functional symptoms based on the degree of threat posed and the possible role of the symptoms as a form of 'escape'. During an MRI, those individuals with conversion disorder were asked to recall these 'escape' events and their brain activity compared to that elicited by other equally threatening control events. The imaging findings showed that the 'escape' event was associated with increased left dorsolateral prefrontal cortical activity and decreased left hippocampal activity compared to that seen in healthy individuals, suggesting a 'direct suppression' of the traumatic memory. Similarly, conversion patients showed amygdala hyperactivity and greater amygdala-supplementary motor area functional connectivity to negative stimuli, again highlighting aberrant limbic-motor connectivity.

Consistent themes unite the functional brain imaging studies reviewed above. Converging data underline the hyperactivity of limbic regions and aberrant connectivity between these anatomical foci and motor and cognitive control regions. People with conversion disorder show aberrant bottom-up neural reactivity in limbic and paralimbic regions possibly related to childhood experiences along with abnormal top-down regulation from the cingulate and lateral prefrontal cortex. By

highlighting a critical abnormality in limbic-motor processing, we provide empirical support for the core Freudian conceptualization of pathological affect influencing symptom expression.

The origins of conversion symptoms have long challenged the medical system. The list of luminaries who have turned their attention to this perplexing question includes Hippocrates, Galen, Paracelsus, Burton, Harvey, Willis, Sydenham, Cullen, Briquet and Pinel amongst others (Veith, 1965). Some, like Laycock (Reynolds, 2020) showed a prescient insight into possible unconscious underpinnings of quasi-neurological symptoms fifty years before Freud's paper discussed here. Others, like Reynolds presaged Freud and Janet by two decades when he noted "that some symptoms of the most serious disorders of the nervous system, such as paralysis, spasm, pain, and otherwise altered sensations, may depend upon a morbid condition of emotion, or idea and emotion or of idea alone." (Reynolds, 1869) What contemporary neuroimaging has given us are the tools to explore these insights and affix a more precise, but as yet imperfect, neural explanation, to them. We have concluded that fMRI studies provide cautious support for Freud's view that excess affect is integral to symptom development. Freud of course went on to expound numerous other theories, which are beyond the scope of this paper and which continue to draw criticism (Dufresne, 2007).

Functional imaging has the potential to shed similar light on many other theories purporting to explain the association between brain function and conversion symptoms, be they the conceptual distortions that characterise Janet's hypothesis (Janet, 1920), or a failure of willed volition, as expounded by Brodie (Hawkins, 1865), to give but two examples. A burgeoning fMRI literature on the topic has been summarized recently and provides an neuroimaging road map for the way ahead (Perez et al., 2021). One of the recommendations cited is the need for longitudinal studies and it is here that the concept of excess affect can further be put to the test. To begin with, there is a need for replication studies with adequate sample sizes across multiple imaging centres. The pathways toward Conversion Disorder are likely multifactorial. Given that a specific mechanism may only be relevant in a subgroup of patients, identifying this underlying subgroup will in theory be important when it comes to tailored therapies, be they psychotherapy or antidepressant medications, to give but two examples. In addition, carefully designed cognitive studies investigating the interaction between emotional processes and the specific functional motor, sensory or somatoform symptoms may be required. We conclude by hypothesizing that successfully treating a functional paresis by alleviating emotional distress (an excess of affect) will be matched by pre-and post-treatment fMRI findings of reduced extraneous limbic, and concomitantly increased motor cortical activation.

Author statement

Anthony Feinstein and Valerie Voon were equally responsible for conceptualizing the work, writing the original draft and editing, reviewing and revising the manuscript.

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