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Visualising the Hypnotised Brain:
Hysteria Research from Charcot to Functional Brain Scans

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Abstract

Contrary to the widely held belief in the humanities that hysteria no longer exists, this article shows that the advent of new brain imaging technologies has reignited scientific research into this age-old disorder, once again linking it to hypnosis. Even though humanities scholarship to date has paid no attention to it, image-based research of hysteria via hypnosis has been hailed in specialist circles for holding the potential to finally unravel the mystery of this elusive disorder. Following a succinct overview of how hypnosis was used in the nineteenth century hysteria research, the article details how the relationship between hysteria and hypnosis is currently renegotiated in the context of brain imaging studies. It shows that the current research has so far failed to deliver on its promise of uncovering the link between hysteria and hypnosis. It further argues that despite huge technological advances in imaging technologies, contemporary researchers grapple with conceptual problems comparable to those that plagued their nineteenth century predecessors.

Keywords: Hysteria, conversion disorder, hypnosis, Charcot, functional brain images, PET, fMRI, comparison, similarity.

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Introduction

Hysteria is an age-old disorder that has continued to puzzle medical authorities throughout its history. Whereas theories of its origin, approaches to its diagnosis and attempts at its treatments have varied considerably over the centuries (Micale 1995: 19-29), one aspect of this disorder has remained constant. No undisputed organic cause has ever been established for the morass of its heterogeneous and constantly changing symptoms that include, but are not limited to, paralyses, pseudoepileptic seizures, blindness, contractures, tremors, pain, loss of speech, and anaesthesia. This confusing diversity of symptoms has led many medical authorities throughout hysteria's centuries-long history to doubt the reality of this disorder.

During the last quarter of the nineteenth century, French neurologist Jean-Martin Charcot conducted his (in)famous research aiming to prove that hysteria was a genuine neurological disease caused by an invisible functional brain lesion. Charcot and his collaborators relied heavily on both various visualising technologies—including photography—and the experimental use of hypnotism (Bourneville & Regnard 1877, 1878, 1879-80, Charcot 1889). Yet Charcot's neurophysiological understanding of hysteria fell into disrepute shortly after his death. As a result, both Charcot's image-based approach to investigating hysteria and his use of hypnotism as a research tool were abandoned.

In this article, I will argue that more than a century after the demise of Charcot's model of hysteria some of his long abandoned concepts are currently seeing a revival within the field of imaging neuroscience. As I will show, the use of relatively novel functional neuroimaging technologies that allow scientists to non-invasively visualise local brain activities in living individuals has given rise to new hysteria research. Once again, hysteria research deploys images to conceptualise this disorder as functional brain pathology. Moreover, in a striking parallel to Charcot, several of the contemporary image-based studies have made experimental use of hypnosis to investigate present-day forms of hysterical symptoms (Halligan et al. 2000, Ward et al. 2003, Cojan et al. 2009a, Cojan et al. 2009b, Burgmer et al. 2013). These studies are the principal focus of this article.

My above statements may seem surprising, since in the humanities-bound literature hysteria is commonly viewed as a medical disorder that no longer exists (Micale, 1995, Bronfen 1998). Admittedly, the dominant nosological systems, such as DSM and ICD, officially stopped using the term "hysteria" by the end of the 20th century. They replaced it with new, constantly shifting diagnostic labels, such as conversion disorder, somatisation, hypochondriasis, pain disorder and many more. As opposed to hysteria's highly problematic etymological connection to the uterus, all of these new labels explicitly avoid defining the disorder as a purely female condition. There is currently no clear consensus in the medical

community as to which and how many, or if any of the newly defined nosological entities correspond to the nineteenth-century hysteria. Nevertheless, a number of contemporary neurologists converge on the view that conversion disorder represents hysteria's legitimate contemporary successor with symptoms analogous to those exhibited by Charcot's patients (Feinstein 2001). Some of these authors use the terms hysteria and conversion disorders interchangeably (see for example Halligan et al. 2000, Cojan et al. 2009a), presumably to emphasise their belief in the continuity between the conditions to which these two diagnostic labels refer. It is this approach that my article will be informed by.

There is an extensive literature in the humanities that critically discusses how Charcot implemented both images and hypnosis in his hysteria research (see for example Showalter 1985, Harrington 1987, Micale 1995, Bronfen 1998, Showalter 1998, Didi-Huberman 2003). The consensus in this literature is that Charcot unscientifically used both images and hypnosis in order to fabricate his representation of hysteria. As opposed to the wealth of studies on Charcot, humanities scholarship to date has paid no attention to the epistemic effects of the current neuroimaging investigations of hypnotically modelled hysterical symptoms. The aim of this article is to take the first step towards filling this gap by looking at how neuroscientists currently attempt to instrumentalise hypnosis within the framework of image-based hysteria research, as well as how these attempts relate to Charcot's approach. Thus, the main focus of this article is on the use of hypnosis in the current brain imaging research of hysteria. But before turning to the discussion of the current research, I will provide a succinct overview of Charcot's views on the interrelatedness of hysteria and hypnosis. My analysis will circumvent the prevalent overtly critical approaches that frame Charcot's research as an intentional fabrication (Didi-Huberman 2003), and argue instead that it was a scientific endeavour in its own right that was nevertheless plagued by conceptual problems.

It should be noted that the brain imaging studies I analyse in this article are conducted as basic research. Even though they actively contribute to hysteria's new visibility within the current brain imaging research by promising to untangle this age-old disorder's mystery (Ward et al. 2003: 295), their findings remain without any foreseeable clinical applications. To this date, the number of brain imaging studies of hysteria modelled through hypnosis is still small and, as we will see, their conclusions are partly conflicting. Not only are the tentative results of this research distant from everyday medical practice, they are also rarely mentioned in popular press and then only in vague and general terms (see for example Bell 2010). As shown by Joseph Dumit (2004), present brain imaging research into disorders such as schizophrenia and depression has had an impact on wider cultural discourses on mental health, illness and normality. In contrast,

the current neuroscientific revival of hysteria and its ongoing refashioning into a neurological disorder has so far been largely confined to specialist circles and has remained removed from the general public. It nevertheless deserves to be closely scrutinised as it partakes in the more general neuroscientific project of focusing on the brain with a view to managing the mind, which has been widely discussed and criticised in the humanities (see for example Beaulieu 2000, Dumit 2004, Vidal 2009, Pickersgill 2013, Rose & Abi-Rached, 2013).

This article contributes to this criticism of neuroscience's apparent ability to once and for all resolve the mysteries of the mind as it questions the promissory discourse of the brain imaging studies that use hypnosis with the hope of providing decisive new insights into the presumed neural underpinnings of hysteria. What kind of knowledge about hysteria do the seemingly straightforward images of the hypnotised brain produce? How do these finding relate to Charcot's concepts of the relationship between hysteria and hypnosis? Do the brain imaging technologies really hold the key to uncovering the relationship between these puzzling phenomena? These are the questions this article will address in detail. It will show that the present conflation of image-based investigation of hysteria with the use of hypnosis not only revives Charcot's long discarded concepts, but also their underlying problems and constraints.

Charcot: Hypnosis as Hysteria's Analogue

The underlying hypothesis that informed Charcot's entire research endeavour was that hysteria is a neurological disorder. In the initial stage of his research he applied to hysteria the same approach he successfully used in relation to other neurological disorders by attempting to establish a specific structural brain lesion as the potential cause of the disorder (Charcot 1877: 294-5). When the dissection of the deceased hysterical patients' brains failed to produce any detectable organic damage, Charcot came to the conclusion that the disorder must be caused by what he termed as the dynamic or functional lesion. He understood this functional lesion as an invisible yet nevertheless physiological disturbance of the brain (Charcot 1889: 14).

Charcot based his hypothesis of functional lesion on the fact that symptoms of hysteria closely resembled those of organic diseases caused by a circumscribed anatomical lesion. By drawing on neuromimesis, i.e. the visual similarity between the symptoms of hysteria and those of corresponding organic disorders, Charcot reasoned that lesions of admittedly different types must cause both categories of symptoms, yet with similar neuroanatomical locations (Ibid). In one case the lesion was proven to be structural, and in the other presumed to be of functional nature.

A similar comparative approach formed the foundation for Charcot's subsequent use of hypnosis as an experimental tool in his hysteria research. Charcot viewed the hypnotic condition as an abnormal sleep-like state that could only be induced to the full extent in hysterical subjects (Charcot 1890: 299). He never developed a consistent theoretical explanation of hypnosis, but adopted instead a phenomenological approach to studying it. Charcot and his collaborators experimented with various ways of artificially producing and manipulating the hypnotic state in their patients. They then induced in the hypnotised subjects transient hysterical symptoms and compared them to their 'genuine' counterparts. Moreover, they systematically measured and registered the physical effects of their experimental interventions (Charcot 1890).

Based on these experiments, Charcot concluded that hypnosis was a purely physiological phenomenon made up of three distinct phases: lethargy, catalepsy and somnambulism. Moreover, he claimed that in each of these phases the hypnotised patients manifested distinct, highly characteristic and measurable physical conditions, which resembled various hysterical symptoms (Charcot 1889: 290-295). By visualising and comparing both spontaneously developed and hypnotically induced hysterical symptoms through photography and the graphical tracing of respiratory curves, Charcot declared these differently produced symptoms to be identical. Drawing on the visual similarity between their physical expressions, Charcot thus contended that hysteria and hypnosis relied on the same neurophysiological mechanisms. In other words, he postulated that a functional brain lesion caused both of them, and he used both photography and graphical tracings to visually substantiate his claim.

Consequently, Charcot termed hypnosis an "artificial neurosis" (Charcot 1890: 298) and started using it experimentally to produce as well as terminate different hysterical symptoms at his own will. In a carefully constructed experimental setup, he plunged his subjects in various stages of hypnotic trance and then instigated and terminated hysterical contractures, paralyses, anaesthesias, mutism and blindness. Hypnosis thus became hysteria's experimental analogue in his research. The usefulness of this approach seemed almost self-evident. Charcot was no longer dependent on his patients to spontaneously develop a specific symptom of interest. Instead, through hypnosis he could replicate any hysterical symptom and thereby fully control its type, anatomical distribution, severity, duration and temporal development. He relied on such use of hypnosis not only for the sake of scientific investigations of the symptoms, but also for demonstrations during both his medical and public lectures.

Yet even though the close entanglement of hysteria and hypnosis endowed Charcot's research project with experimental flexibility, in the end it also turned out to be its major weak spot. Charcot's carefully constructed experimental edifice

crumbled when a rival doctor, Hyppolite Bernheim, contested his contention that hypnosis was a purely neurophysiological phenomenon, and a pathological one at that. Bernheim's counterclaim was that hypnosis could only be properly understood as a normal and natural psychological condition during which the subject exhibits a pronounced susceptibility to suggestion (Bernheim 1889: 149). According to Bernheim, a hypnotised subject did not manifest characteristic physical symptoms as Charcot claimed, but merely acted in response to the hypnotist's either implicit expectations or explicitly formulated instructions (Ibid).

In essence, Bernheim's critique fully negated the validity of Charcot's experimental use of hypnosis in hysteria research. It effectively reinterpreted Charcot's experiments as mere role-playing between the hypnotist and his subjects. Within this new context, the visual similarity between hypnotic and hysterical phenomena—as evidenced by a plethora of photographs and graphical tracings—became meaningless. Instead of implying the existence of a common brain lesion, these images could just as easily be viewed as visual documentation of either conscious or unconscious simulation. Soon, Charcot's own pupils followed suit, not only by abandoning the use of hypnosis as an experimental tool, but also by questioning Charcot's very understanding of hysteria as a neurological disorder. Pierre Janet, Joseph Babinski and Sigmund Freud developed their own theories of hysteria all of which departed from their teacher's neurological model. A common point was that they all reframed hysteria as a form of a psychological disorder, an 'all-in-the-mind' illness without a clear-cut physiological origin.

Twenty-first Century Revival of the Link Between Hysteria and Hypnosis

Throughout most of the 20th century, Freud's model of hysteria as a mental illness caused by the supressed memories of past traumatic events provided the dominant framework for diagnosing and treating this disorder. Within the medical terminology, new labels displaced the term hysteria and its lingering, long out-dated etymological link to the female uterus as the erroneously presumed origin of the disease. The new diagnostic categories—including conversion disorder, somatoform disorders and dissociation disorders—placed the emphasis on the causative role of psychological factors in the development of present-day manifestations of hysteria. Yet these new labels failed to make the baffling symptoms more acceptable either to patients or to doctors. Patients who kept appearing in clinics with the symptoms comparable to those that had previously been categorised as hysterical have often remained undiagnosed (Stone et al. 2008). Decreasing diagnostic frequency coupled with waning research interest made hysteria invisible.

This situation began to gradually change in the late 1990s with the publication of several functional neuroimaging studies investigating hysterical symptoms (Tiihonen et al. 1995, Marshall et al. 1997). First using PET (positron emission tomography) and then almost exclusively relying on fMRI (functional magnetic resonance imaging), a number of neurologists started to claim that hysterical symptoms are related to visualisable dysfunctions of the brain (see for example Spence et al. 2000, Vuilleumier et al. 2001, Burgmer et al. 2006, Stone et al. 2007). Functional brain scans generated by these studies seem to suggest that—despite the lack of any detectable anatomical brain damage—the hysterical patients' patterns of neural activities differ distinctly from those of comparable healthy subjects.

The number of neuroimaging studies of hysteria published to date remains very small. Moreover, the insights provided by this research remain tentative, since the individual studies diverge in their results. Yet, despite the current inability of the contemporary image-based research to provide a consensus as to which concrete patterns of neural activity could underlie various hysterical symptoms, this strand of research has nevertheless been successful in reviving the idea of hysteria as a brain disorder. In fact, it can be said that the new research focuses on visualising Charcot's hypothesised functional brain lesion.

Interestingly, the current research has revived an additional aspect of Charcot's approach to investigating hysteria. In the context of functional neuroimaging studies, hypnosis is once again gaining currency as a potentially useful research tool that allows scientists to controllably replicate hysterical symptoms of interest under experimental conditions (Oakley & Halligan 2009). In other words, hypnosis is being used anew as an experimental analogue of hysteria. As I will show in the subsequent sections, even though the present-day implementation of hypnosis to study hysteria is driven by the use of novel brain imaging technologies, it nevertheless manifests several significant parallels to Charcot's use of hypnosis more than a hundred years earlier.

Hypnosis as a Poorly Understood Tool for Studying Hysteria

Although hypnosis has been repeatedly used in neuroscientific research to model hysterical symptoms within the last twenty years (Halligan et al. 2000, Cojan et al. 2009b, Burgmer et al. 2013, Deeley et al. 2013), its nature remains scarcely understood. One of the major issues is that the current state of research has not yet been able to resolve the long-standing controversy initially ignited by the conflict between Charcot and Bernheim. There is still no decisive proof as to whether hypnosis corresponds to a distinct altered state of mind with underlying neurophysiological changes as presumed by Charcot, or to a hypnotised subject's

compliance with the hypnotist's suggestion as claimed by Bernheim (Oakley 2008). Even though both of these divergent stances have supporters, the neuroimaging community tends to associate hypnosis with a distinct neurophysiological state and focuses on generating data that supports such a view. Neuroimaging studies of hypnosis thus actively contribute to the constitution of what Anne Beaulieu termed the "mind-in-the-brain" by searching for visual proof that the hypnotic condition—understood as a distinct mental state—correlates with a set of identifiable brain processes (Beaulieu 2000:7).

So far the findings of basic research into hypnosis are inconclusive. Several neuroimaging studies have implicated the controlled induction of a hypnotic condition with distinct changes in the pattern of neural activity (Oakley & Halligan 2009: 264-5, McGeowan et al. 2009). Overall, however, the results are mutually inconsistent and no unequivocal neural basis of hypnotic condition has yet been identified (Oakley 2008). Nevertheless, such provisional findings of the intrinsic research into hypnosis provide the conceptual basis for the neuroimaging studies of hypnotically induced hysterical symptoms (see for example Cojan et al. 2009: 862-3).

Whereas neuroimaging studies appear to support Charcot's view of hypnosis as an altered mental state, his other views seem to have fared worse with contemporary researchers. They have explicitly discarded both Charcot's claim that hypnosis represents a primarily pathological condition and his division of it into three distinct stages (Laurence et al. 2008: 230). To investigate both hypnosis in its own right and hypnotically induced hysterical symptoms, today's researchers no longer use patients. Instead, they recruit healthy individuals previously tested to be free of any psychiatric disorders. Moreover, Charcot's three consecutive stages of hypnosis with their measurable physical signs have been displaced by new categories of hypnotic depth and hypnotisability. Despite these apparent changes, in what follows I will argue that contemporary research has not fully shaken off the legacy of Charcot's concepts.

Hypnotic depth refers to the perceived intensity of the hypnotic experience. Since variations of hypnotic depth have been associated with measurable changes in the neural activity (Oakley 2008: 20-21), maintaining it at a considerable and stable level throughout the experiment represents an important precondition for a neuroimaging study involving hypnotically-induced hysterical symptoms. Unable to objectively measure it, researchers instead train their experimental subjects to assess their hypnotic depth through self-reporting while lying inside the scanner. Thus, whereas Charcot judged the efficacy of the hypnotic induction based on the physical conditions his patients exhibited, neuroimaging studies rely instead on the subjects' self-evaluation. Based on their experience of the hypnotic condition, gained prior to the imaging experiment, the subjects are asked to grade their level

of hypnotic depth in relation to a chosen numerical scale (Oakley et al. 2007). Despite this attempt at quantification, such estimation of hypnotic depth remains a highly subjective measure that is difficult to reliably compare across different individuals.

Another descriptive measure used in contemporary hypnosis research is called hypnotisability or hypnotic suggestibility. It refers to the individual's tendency to respond to hypnosis and hypnotic suggestions that modify his/her sensory experience and behaviour. To determine a subject's hypnotisability, s/he is first induced into a hypnotic trance and then exposed to a sequence of suggestions that systematically alter his/her perception, motor behaviour and memory. A variety of standardised scales are then used to quantify the individual's hypnotisability depending on the degree of their responsiveness to these suggestions (Woody et al. 2005). Based on their score, the individual's hypnosability is categorised as high, medium or low. Despite the use of standardised scales to quantify its variations among individuals, the potential reasons behind this variability remain unclear (Laurence et al. 2008). Further, whether these different levels of hypnotisability represent the individual's innate trait or if they can be modified through training remains a matter of debate (Ibid: 232). The dividing line in this debate corresponds to the different views that researchers hold on hypnosis in general. Those who view hypnosis as a mere compliance with the hypnotist's suggestions claim that hypnotisability is a learned ability (Ibid). In the neuroimaging community, however, hypnotisability is generally regarded as an unmodifiable trait with a genetic component (Bell 2010).

Significantly, all neuroimaging studies of hypnotically-induced symptoms of hysteria to date have been performed on healthy volunteers who had been previously assessed as highly hypnotically suggestible. Thus, the participants of these studies have already been preselected for their increased responsiveness to developing hypnotic phenomena that closely resemble hysterical symptoms. In his research, Charcot regarded such increased susceptibility to suggestion as an indicator of latent hysteria, a pathological state of the nervous system that has not been triggered yet to produce visible symptoms. Within current research, the high hypnotisability is merely registered as a phenomenological fact that allows for easy modelling of hysterical symptoms (Oakley et al. 2007). On the surface, the selected participants' increased responsiveness to hypnotic suggestion appears to have a purely instrumental role in current hysteria research. Explicitly, it is not ascribed any meaning in itself, either as a potential sign of pathology or of normalcy. What remains unmentioned in these studies, however, is that on average only one in ten adults receives high scores on the standardised scales, which makes high hypnotisability a relatively rare trait (Bell 2010). Against the historical backdrop of Charcot's research, we should thus not overlook the possibility that this particular

choice of experimental subjects has epistemic consequences. It can be argued that by selecting experimental subjects based on such a rare trait, the current research at least implicitly revives and even reinforces the presumably pathological link between increased suggestibility and hysteria.

In fact, several behavioural studies have taken one step further in this direction and directly tested Charcot's assumed pathological association between hysteria and hypnosis. Two studies reported a higher level of hypnotic susceptibility in hysterical patients relative either to patients suffering from other conditions or to healthy individuals (Kuyk et al. 1999, Roelofs et al. 2002). However, their results were contradicted by other studies that failed to establish any statistically significant evidence of increased hypnotisability in patients exhibiting hysterical symptoms (Goldstein et al. 2000, Litwin et al. 2001, Moene et al. 2001). Within the current research context, the presumed pathological correlation between hysteria and hypnosis remains an unresolved issue at the empirical level. Nevertheless, it can be said that the lingering effects of Charcot's initial claims seem to at least implicitly influence the current research. This is reflected in the specific selection of experimental subjects based on their high responsiveness to hypnotic suggestion, but also in the recurring although so far inconclusive attempts to find evidence for a presumed correlation between hypnotic susceptibility and the presence of hysterical symptoms in diagnosed patients.

Imaging the Brain: Hypnosis as a(n) (Un)Reliable Model of Hysteria

As discussed previously, Charcot viewed the phenomenological similarity between the spontaneously developed hysterical symptoms and their hypnotically-induced counterparts as the definitive proof that comparable neural processes underlie both hysteria and hypnosis. Charcot was limited to visualising the external physical manifestations of hypnotic suggestion and then using these images to make inferences about their possible neurological causes. Today's researchers instead rely on the state-of-the-art technologies to visualise distinct patterns of brain activities attributable to the investigated hypnotic phenomena. The arrival of new brain imaging technologies has thus shifted the focus from the surface of the human body to the "space inside the scull" (Beaulieu 2000).

Although these imaging technologies have often been hailed, especially in the popular press, for enabling neuroscientists to observe the human brain at work (see for example Zimmer 2014), they do not facilitate any direct access to the brain activities of interest. In fact, to even arrive at a visualisation of brain activity, scientists use complex machinery to first produce data which then undergo several

stages of data processing. The resulting brain scans are thus highly constructed images based on which scientists make inferences about brain activity.

Even more importantly, these images do not allow any direct comparison of such complex and diffuse phenomena such as hysteria and hypnosis. To be able to use brain imaging technologies for establishing if hysteria and hypnosis share a neural basis, researchers rely on experimental set-ups. As we will see in the following analysis, it is through experimental set-ups that researchers artificially isolate chosen aspects of both hysteria and hypnosis and translate them into seemingly clear-cut brain scans which they can then compare. Thus to answer the question of how researchers use brain images to investigate the relation between hysteria and hypnosis, we have to take a closer look at the decisions they make when designing their experiments.

A pioneering neuroimaging study by Halligan et al. (2000) used PET to visualise the brain activity of a 25-year-old healthy, highly hypnotisable male. Following a standard hypnotic induction, the depth of which was monitored throughout the experiment, a left leg paralysis was produced through hypnotic suggestion. The paralysis was modelled to closely resemble the hysterical leg paralysis previously studied with PET by Marshall et al. (1997). The subject was then placed inside a PET scanner and instructed to prepare to move or try to move either his normal or his hypnotically paralysed leg on cue. However, since both legs were tightly restrained, no actual movement took place. This experimental design was identical to the one performed by the hysterical subject in the Marshall et al. (1997) study.

While the subject performed these tasks, PET images of his brain were collected by the scanner. In the subsequent process of data analysis, Halligan et al. compared the subject's neural responses during the attempt to move the hypnotically paralysed leg to the neural responses during the attempt to move the normal leg. The result was a brain map with a distinct pattern of neural activations that showed a significant overlap with the brain map previously published by Marshall et al. Based on the visual similarity of the brain activations between a hysterical patient and the hypnotised subject, Halligan et al. argued that their imaging results supported the view that "hysterical and hypnotic paralysis share common neural systems" (Halligan et al. 2000: 987). Their statement not only echoed Charcot's initial claim that hypnosis and hysteria rely on overlapping neurological mechanisms, but also appeared to finally provide it with a visual proof in the form of brain scans. In another parallel to Charcot, Halligan et al. concluded that owing to their shared neural mechanisms, "hypnotic phenomena provide a versatile and testable model for understanding and treating conversion hysteria symptoms" (Ibid).

However, the optimism of the Halligan et al. study was somewhat overstated, since the findings of such a single-case comparison cannot be generalised beyond the individual patient. Attempting to circumvent this problem, subsequent neuroimaging studies of hypnotically induced paralysis shifted to recruiting groups of highly hypnotisable patients for the experiments. This was the case with the Ward et al. study (2003) that also used PET, but this time to visualise the differential neural activations associated with the attempted movement during hypnotically induced left leg paralysis relative to rest in a hypnotised state. As opposed to Halligan et al, the Ward et al. experiment was performed on twelve male volunteers whose legs were unrestrained during the measurement.

After statistically averaging their results across all participants, Ward et al. arrived at a pattern of neural activations that failed to fully replicate the results of the Halligan et al. study. Although there was a partial overlap between the brain activations detected by these two studies, there were also considerable differences. Some of the brain areas that, according to Halligan et al., played an important role in hypnotic paralysis remained inactive in Ward et al. study (2003: 310). Conversely, the new study generated brain maps that showed a more diverse pattern of neural activations, implicating a possible contribution of additional brain areas (Ibid: 302). Ward et al. thus opened the possibility that different brain mechanisms underlie hypnotic paralysis than those suggested by the previous study. Moreover, the authors suggested that although hypnosis may be useful for modelling hysterical symptoms in a controlled fashion, "paralysis produced by suggestion following hypnotic induction may not readily transfer to patients", since the real-life hysterical symptoms appear to be more complex than those modelled through hypnosis (Ibid: 311).

More recent studies into the putative link between hysteria and hypnosis used fMRI, a neuroimaging technology with a higher spatial and temporal resolution than PET. Moreover, researchers have started to implement more complex experimental designs instead of merely instructing subjects to attempt to move while lying inside a scanner. For instance, in the studies by Cojan et al. (2009a, 2009b), subjects were instructed by means of a visual cue first to prepare a hand movement, and then either to execute it by pressing a button, or to abort it. In their first study, Cojan et al. (2009a) used this task to investigate the neural activation underpinning a left arm paralysis in a single female patient. They then repeated the task with the group of twelve volunteers, who either performed it in a normal state of wakefulness or during hypnosis combined with a suggestion of left hand paralysis (Cojan et al. 2009b). For each of these studies, the researchers computed respective activation patterns for different aspects of the selected task. Their images showed "some similarities but also clear differences" between neural activations associated with hypnotically-induced and hysterical paralysis (Cojan et

al. 2009a: 1035). Thus, they concluded that despite some shared underlying neural activations, both hypnosis and hysteria also entail some specific and mutually distinct neural processes (Ibid: 1036). In this case, brain maps appeared to suggest that the phenomenological similarity of hypnotic and hysterical symptoms does not translate into a shared neural basis.

Burgmer et al. (2006/2013) came to a similar conclusion through a different experimental design. In two parallel studies, they asked their subjects to perform an identical task—first to observe a video of a moving hand and then to try to imitate the represented movement. In the first study (Burgmer et al. 2006), they investigated the neural activity of four patients with hysterical hand paralysis while performing the task. In the subsequent study, they scanned nineteen healthy volunteers, both in a normal state and under hypnosis accompanied by a suggestion of a hand paralysis (Burgmer et al. 2013). Even though their studies implicated partly different brain regions than the Cojan et al, they also established partial discrepancies in the neurological underpinnings between hysterical and hypnotically-induced paralysis (Ibid: 443). They tentatively ascribed these differences to the transitory nature of hypnotic paralysis as opposed to its hysterical counterpart, while also admitting that the relationship between hypnosis and hysteria "requires further consideration" and investigation (Ibid).

The above overview makes one aspect of the current research stand out: Researchers draw their conclusions about the potential similarity between neural underpinnings of hypnotic and hysterical paralysis by visually comparing images of the brain activation patterns derived from parallel experiments. A separate experiment is first conducted with patients exhibiting a hysterical paralysis limited to a particular limb. The same experiment is then repeated with highly hypnotisable healthy individuals in whom an equivalent paralysis has been hypnotically induced. Each of these experiments produces respective images showing patterns of neural activation attributed, on the one hand, to hysterical and, on the other hand, to hypnotic paralysis. These images are then compared to each other to establish to what extent and at which anatomical locations in the brain the patterns of neural activation either overlap or show clear differences.

However, the caveat behind this apparently clear-cut approach is that there is no direct way of using functional neuroimaging technologies to determine the distinct neural activity underlying such complex and poorly understood phenomena as either hysteria or hypnosis. Firstly, brain images in general grant only an indirect access to brain activity and this access is mediated through the given technology. Secondly, a set of brain images resulting from a concrete neuroimaging study is produced through the chosen experimental procedures and conditions, which are built into these images. As we have seen in the examples above, different researchers used different imaging technologies, implemented

different tasks and contrasted different experimental conditions. Some studies had a single experimental subjects, while others recruited a group of participants. Some studies compared the attempted movement during hypnotically-induced paralysis to a resting state in hypnotic condition, whereas other contrasted it with an attempted movement in the state of normal wakefulness. Some researchers restrained their subjects to make their movement impossible, whereas others did not. Some opted for a very simple, others for a more complex task. All these choices were informed by the researchers' theoretical assumptions and hypotheses about the neural underpinning of both hysteria and hypnosis (see for example Cojan et al. 2009b: 863, Burgmer et al. 2013: 438).

All of the above decisions had an epistemic effect on the results of the respective studies, thus contributing to the mutual discrepancies and even contradictions among their findings. However, without a reliable theoretical understanding of what hypnosis is to start with, none of these competing experimental approaches and their respective findings can be evaluated as more valid than their alternatives. In effect, the search for common neural mechanism behind hysteria and hypnosis by means of brain imaging has so far come up with inconclusive results, leaving the relationship between these two phenomena unresolved.

Conclusion

The advent of functional neuroimaging led not only to the resurgence of scientific interest into hysteria, but also revived Charcot's long-abandoned concept of the functional brain lesion as the potential cause of hysteria and once again brought into focus the use of hypnosis as an experimental tool. Initially, this new image-based hysteria research was celebrated in specialist circles for its potential to finally unravel the age-old mystery of this elusive disorder (Tallabs 2005, Oakley 2006). Yet, as I have aimed to show in this article, the brain imaging studies so far have been much less successful in providing new and transformative knowledge of hysteria than in reopening many of the conceptual problems that haunted Charcot's original research endeavour.

In a parallel to Charcot's approach, contemporary researchers use one scarcely understood phenomenon to model another that is equally poorly understood. On the surface, the use of hypnosis as an experimental tool seems to offer a much greater control in studying the hysterical symptoms that are of interest. It appears to allow researchers to induce, modify and stop "behaviourally indistinguishable versions" of the same symptoms as and when they deem appropriate, or to artificially isolate a particular aspect of the symptom (Ward et al. 2003: 310). However, since the very nature of these induced phenomena as well as their relation to spontaneously developed hysterical symptoms remains opaque, the apparent control offered by

hypnosis turns out to be deceptive. In fact, as we have seen in the examples above, by displacing hysterical symptoms with their hypnotically modelled counterparts, researchers introduce into their experimental setup additional ambiguities for which they are unable to fully account.

In another parallel to Charcot, contemporary researchers rely on the comparisons of images derived from matching experimental setups to establish if hysteria and hypnosis share common neural underpinnings. As opposed to Charcot, who relied on the images that depicted the outside of the patients' body, contemporary researchers use technologies that allow them to visualise the neural activity from inside the experimental subject's head. Yet despite their technological superiority, these images are not able to provide insights into hysteria without a clear theoretical framework that would guide both their production and their interpretation. Charcot's images became meaningless when Bernheim questioned the validity of his theoretical framework. Similarly, the epistemic validity of these only seemingly straightforward images of the hypnotised brain remains problematic within current hysteria research as long as there is no clear theoretical understanding of either hysteria or hypnosis within which their production and interpretation could be anchored. Thus, when using hypnosis to investigate hysteria, present-day researchers grapple with similar conceptual problems as Charcot once did.

Notwithstanding the enormous technological advances in the imaging technologies between Charcot's time and today, the major challenge that any image-based research of hysteria via hypnosis faces seems to be situated at the conceptual and methodological level. The main question is not what we can see in the image—whether they show us the surface of the body or allow access to the space inside the brain—but how their meaning is constituted within the scientific context. Without a plausible theoretical framework to guide their production and interpretation, such images remain epistemically ambiguous and unable to produce new insights into either hysteria or hypnosis. The coupling of the new visualising technologies with the experimental use of hypnosis has thus so far failed to deliver on the promise of solving the hysteria's mystery. In fact, what these seemingly straightforward multi-coloured images of the brain have made visible so far is that hysteria remains just as elusive a phenomenon as it has been for centuries.

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