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To sum up, my analysis has shown that more than a century after the demise of Charcot's systematic use of images to frame hysteria as a brain disorder, new image-based research has appeared that has once again started to link hysterical symptoms to a still unknown brain dysfunction. Moreover, I have argued that after a slow and wavering start, this research gradually coalesced into a sustained scientific practice centred on the use of a single functional neuroimaging technology, the fMRI. Earlier, we have also discussed that the very precondition for the development of this new image-based research was the emergence of an initially tentative presumption that various somatic symptoms of hysteria might have a neurophysiological basis despite the lack of any direct empirical evidence supporting this presumption at the time. In what follows, I will analyse how fMRI-based hysteria research has started to empirically legitimate the very somatic framework that had given rise to it.

2.4 Current Neurological Reconceptualisation of Hysteria through fMRI Research

Once it had consolidated into a sustained, systematic scientific endeavour, functional neuroimaging research into hysteria started to produce tangible epistemic effects. Admittedly, so far, the findings of individual studies have been mutually too inconsistent to enable a conclusive delineation of a specific neural basis for any of the hysterical symptoms.[526] For this reason, the current fMRI-based findings concerning hysteria remain without foreseeable clinical or diagnostic applications and are instead firmly grounded in the domain of basic research. Nevertheless, in the following two sections, I will argue that despite the limited insights it has produced to this date, the continued existence of image-based research into hysteria over the past two decades has sufficed to induce a renewed reconceptualisation of this once controversial disorder. First, I will show how by generating new experimentally won insights into hysteria as a brain-based disorder, fMRI research has managed to confer a sense of reality on these elusive symptoms. Second, I will trace how this new attitude has led to the development of a more general medical interest in hysteria, thus gradually re-anchoring this disorder into a neurological context. Finally, we will see that, due to such changes, the current nosological successors of hysteria have ceased to be defined as medically unexplained or conflated with malingering.

2.4.1 Experimental Inscription of Hysteria Into the Brain

The biomedical reshaping of psychiatry in the late twentieth century we discussed so far entailed an additional relevant aspect that is of particular interest for our discussion in this section. Specifically, psychiatry has been progressively modelled along the

526 See, e.g., Baek et al., "Motor Intention," 1624; and Hassa et al., "Motor Control," 143–44. We will discuss such findings in detail in chapter 4.

parameters of natural sciences and their reliance on reproducible empirical evidence generated through quantitative measurement procedures instead of phenomenological observation.[527] In this context, particular emphasis has been placed on experimental research as the primary form of knowledge-generating practice. Hence, experimentally won data have begun to exert exceptional influence in shaping the medical and the psychiatric research practice.[528] The application of fMRI has fitted perfectly into the experimental paradigm by endowing contemporary hysteria research with the presumed epistemic validity of laboratory science.[529] As we are about to see in what follows, in the contexts of such particularly framed epistemic activity, hysteria is increasingly acquiring contours as a disorder due to functional brain pathology.

Before the advent of fMRI, researchers were trying to speculatively link either hysteria patients' observable behaviour or various clinical characteristics of their symptoms to putative biological or psychological causes.[530] By contrast, researchers nowadays deploy fMRI to produce empirical data by measuring physiological processes that correlate with the patients' neural responses to carefully designed experimental conditions. To facilitate such a measurement, researchers have to extract the patient from her everyday context and place her in a highly artificial and controlled environment. In such an experimental setup, the initial step entails positioning the patient inside a scanner located in a designated room within a hospital or research facility. Lying inside the narrow bore of the large and very loud machine, the patient is expected to remain motionless for the duration of the experiment, which can take up to an hour. During this period, she might be exposed to specifically designed stimuli, instructed to carry out a particular set of tasks, or told to rest and think of nothing in particular. Depending on the type of symptom being studied, the stimuli can include vibrotactile stimulation, pinpricking, or exposure to coloured light.[531] Alternatively, patients can be asked to respond to a succession of images or to execute a specified movement on cue.[532]

The purpose of such tasks and stimuli, or the controlled lack thereof, is to experimentally manipulate particular aspects of hysterical symptoms while the patient's brain activity is measured and visualised by the scanner.[533] The resulting imaging data must undergo a complex, multistage process of mathematical and statistical analysis

527 Pincus, "*DSM-IV*," 149–50. See also Andreasen, *Brain Imaging*, ix–x.

528 Pincus, "*DSM-IV*," 149–50.

529 I am referring here to laboratory sciences in the sense defined by Ian Hacking as "sciences [that] use apparatus in isolation to interfere with the course of that aspect of nature that is under study, the end in view being an increase in knowledge, understanding, and control of a general or generalisable sort." See Hacking, "Self-Vindication," 33.

530 Vuilleumier, "Brain Function," 314–15. See also my analysis in chapter 1 and sections 2.1.1–2.1.3.

531 See, e.g., Stoeter et al. "Somatoform Pain," 418; Werring et al., "Visual Loss," 584; and Ghaffar, Staines, and Feinstein, "Sensory Conversion Disorder," 2036.

532 See, e.g., Marshall et al., "Hysterical Paralysis," B1; and de Lange, Roelofs, and Toni, "Self-Monitoring," 2053.

533 In the following chapter, I will discuss in detail all the steps entailed in an fMRI-based experimental manipulation that I am merely sketching here in general terms.

to yield relevant information.[534] The intended outcome of such an experiment is a set of images, referred to as fMRI maps, which display the anatomical locations of the patient's brain activity of interest. The maps are commonly visualised as clearly delineated patches of bright colours that are overlaid on grey-scale brain sections or 3D brain renderings.[535] Based on such brain maps, researchers make inferences about the hysterical symptoms' neural underpinnings, which they then interpret in terms of associated cognitive functions.[536] Finally, such image-based findings of the hysteria patients' aberrant brain activity are embedded into the interpretative text of a research article and published in peer-reviewed scientific journals. Having thus acquired the status of empirically won scientific evidence for the neural underpinnings of hysterical symptoms, the image-based findings are cited in other research articles and serve as a point of reference for developing subsequent fMRI studies.[537] Hence, it is owing to fMRI maps that hysterical symptoms, which until recently were fully detached from the body, are now becoming linked to anatomically localisable brain dysfunctions.

Based on my analysis above, it can be said that a hundred years after the dismissal of Charcot's image-based search for the conjectured functional brain lesion, the hysteria patient's active brain has once again become the object of experimentally framed scientific enquiry, or to use Rheinberger's term, an "epistemic thing."[538] According to Rheinberger, within a research setting, epistemic things are inextricably linked to experimental conditions, which include "instruments, inscription devices, models organisms and the floating theorems or boundary concepts attached to them."[539] Since the hysteria patients' aberrant brain activity is accessible primarily through the mediation of functional neuroimaging, fMRI is the central experimental condition in the current empirical research into the neural basis of this disorder.[540] In fact,

534 This process will be discussed in detail in sections 3.4.1–3.4.4.

535 See, e.g., Ghaffar, Staines, and Feinstein, "Sensory Conversion Disorder," 2037.

536 See, e.g., Ghaffar, Staines, and Feinstein , 2037–38.

537 See, e.g., Cojan et al., "Inhibition," 1027.

538 Rheinberger, *History of Epistemic Things*, 28.

539 Rheinberger, 29.

540 Notably, one secondary effect of the fMRI research into hysteria was that, by pointing to a potential neural basis of this disorder, it effectively legitimised the use of different neurophysiological technologies as research tools in the study of this disorder. For example, drawing on the findings of functional neuroimaging studies, several research groups implemented a technique called transcranial magnetic stimulation (TMS) to test the excitability of neural circuits in the motor cortex of hysteria patients' brains. See, e.g., Avanzino et al., "Cortical Excitability"; Espay et al. "Cortical and Spinal Abnormalities"; Liepert et al., "Abnormal Motor Excitability"; and Quartarone et al., "Sensorimotor Plasticity." Other researchers used electroencephalography (EEG) to measure the electrical signals generated by time-locked neural responses to targeted somatosensory stimulation in patients with hysterical paralysis or sensory loss. See Blakemore et al., "Distinct Modulation"; Blakemore et al., "Disrupted Movement Preparation"; and Roelofs, de Bruijn, and Van Galen, "Hyperactive Action Monitoring." In two other studies, EEG measurements were used in conjunction with sophisticated mathematical modelling to investigate potential disturbances in the neural connectivity across different brain areas in patients with non-epileptic seizures. See Barzegaran et al., "Functional Brain Networks"; and Knyazeva et al., "Psychogenic Seizures." Finally, in three additional studies, hysterical sensorimotor disturbances were investigated with a functional neuroimaging technology called magnetoencephalography (MEG). See Fiess

considering the prior lack of a systematic empirical enquiry into this topic throughout the twentieth century,[541] it can be claimed that fMRI research was the constitutive factor in the contemporary emergence of hysteria patient's active brain as an epistemic object in the first place.

Moreover, Rheinberger has pertinently remarked that in so far as they embed the epistemic things, experimental conditions also delineate the realm of the possible access to them.[542] Drawing on Rheinberger, I suggest that the extent to which the chosen experimental condition defines the realm of the epistemically possible is particularly pronounced in the case of fMRI-based hysteria research. Specifically, I argue that due to the current absence of any uncontested theory about the underlying nature of hysterical symptoms, the entire experimental arrangement within which hysteria is, at present, being redefined as a distinct brain disorder is primarily determined by the epistemic possibilities of the fMRI technology. Since I have previously claimed that a particular conceptual shift in the understanding of hysteria was a necessary precondition for the applicability of functional neuroimaging technologies as research tools, I need to qualify my current statement that the contemporary experimental inquiry into this disorder is, in fact, not theory-driven.

To be sure, the general assumption on which the emergence of this research was predicated continues to inform it—fMRI studies of hysteria operate within a purely biological understanding of the mind.[543] Simply put, all mental processes of interest are framed in terms of underlying brain activities. However, whereas this basic neurobiological framing is a given in the current fMRI hysteria research, something else is missing. Absent in this research is what Ian Hacking has termed 'systematic theory': "theory of a general and typically high level sort about the subjects matter."[544] Specifically, ever since the demise of Freud's psychogenic model, there have been no universally accepted theories of either hysteria in general or of any of its current taxonomic successors.[545] There is also no undisputed conceptual framework that could provide a reliable explanation of the potential causes or presumed mechanisms of any of the highly heterogeneous hysterical symptoms.[546]

As a result, in the first two decades of the twenty-first century, researchers were unable to rely on a stable, well-defined theoretical framework of hysteria from which they could derive testable research hypotheses about the expected involvement of

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et al., "Emotion Regulation"; Fiess et al., "Emotionally Salient Stimuli"; and Hoechstetter et al., "Psychogenic Sensory Loss." Admittedly, these alternative neurophysiological technologies have opened up potentially valuable complementary research perspectives into the hysteria patients' active brains. However, only the few studies listed here have implemented these other technologies in the first two decades of the twenty-first century. Thus, the use of these different technologies has been sporadic and lacks the systematic quality of the current fMRI hysteria research. For this reason, we can say that for the time being, fMRI remains the dominant experimental condition in the neurobiological research into hysteria.

541 See sections 2.2.1 and 2.2.2.

542 Rheinberger, *History of Epistemic Things*, 29.

543 For an explicit expression of this view, see, e.g., Stone et al., "Change at Follow-Up" 2887.

544 Hacking, "Self-Vindication," 45.

545 See, e.g., Vuilleumier, "Brain Function," 309–10.

546 See, e.g., Hassa, "Motor Control," 143.

particular brain regions in various hysterical symptoms. Instead, they deployed fMRI as "an open reading frame for the emergence of unprecedented events."[547] In an attempt to identify and localise the hysterical symptoms' unknown neural correlates, researchers started testing various experimental setups that allowed them to generate neuroimaging data about the patients' brain activity. For example, some researchers scanned patients' brains first during the acute phase of a symptom manifestation and then after the recovery. They then attributed the differences in the neural activities between these two measurements to the hysterical symptom under scrutiny.[548] By contrast, multiple researchers aimed to pinpoint the spatially distributed differences in the brain activity induced through the experimental manipulation of the affected as opposed to the healthy side of the patient's body.[549] Alternatively, some tried to identify the neural underpinnings of hysterical symptoms by contrasting the brain activities between 'genuine' patients, on the one hand, and healthy subjects who had been instructed to pretend to have hysterical symptoms, on the other.[550] Across these various comparisons, researchers have deployed a wide range of different tasks and stimuli. Patients were exposed to heat or vibratory stimulation, asked to respond to various images or short video clips, or instructed to perform a particular kind of movement with their partly or fully paralysed limbs.[551]

Following statistical analysis of the neuroimaging data thus acquired, researchers computed and visualised functional brain maps that displayed the anatomical locations of hysteria patients' aberrant neural activities. By interpreting the resulting images, researchers then postulated which neurocognitive process could underlie a particular hysterical symptom.[552] Because they were obtained through the divergent approaches listed above, functional brain maps differed considerably across various studies. As a result, different researchers have attributed the same type of symptom to disparate cerebral dysfunctions. For instance, based on the patterns of brain activity they registered, the authors of several studies inferred that such disparate symptoms as paralysis and blindness arose from similar cognitive processes. Specifically, paralysis and blindness were suggested to involve involuntary top-down inhibition of planned movement and sensory processing, respectively.[553] However, authors of other imaging studies that obtained entirely different patterns of brain activity posited competing interpretations. Some of them ascribed hysterical paralysis and sensory loss to attentional dysregulation.[554] Others contended that these symptoms were caused by

547 Rheinberger, *History of Epistemic Things*, 31.

548 See, e.g., Dogonowski et al., "Recovery"; and Shimada et al., "Cerebellar Activation."

549 In many cases, hysterical patients exhibit symptoms only on one side of the body—a phenomenon referred to as lateralisation. See, e.g., de Lange, Roelofs, and Toni, "Self-Monitoring"; and Saj, Arzy, and Vuilleumier, "Spatial Neglect."

550 See, e.g., Stone et al., "Simulated Weakness"; and van Beilen et al., "Conversion Paresis."

551 See, e.g., de Lange, Roelofs, and Toni, "Self-Monitoring"; Ghaffar, Staines, and Feinstein, "Sensory Conversion Disorder"; Gündel et al., "Somatoform Pain"; and Spence et al., "Disorder of Movement."

552 See, e.g., Burgmer et al., "Movement Observation," 1341–42.

553 Tiihonen et al., "Cerebral Blood Flow," 134; and Marshall et al., "Hysterical Paralysis," B1–8.

554 Schoenfeld et al., "Hysterical Blindness"; Saj, Arzy, and Vuilleumier, "Spatial Neglect."

disturbances in much earlier stages of primary sensory processing and movement initiation.[555]

Despite such mutual discrepancies, the common thread across all the studies is that their authors have derived the theoretical hypotheses about the underlying neural basis of hysterical symptoms from the empirical imaging data. In other words, instead of being informed by a fixed, predefined theoretical framework, a typical fMRI enquiry into hysteria uses experimentally generated images of brain activity to create novel hypotheses and new insights into the neural underpinnings of hysterical symptoms. In effect, such studies represent pertinent examples of what the historian of science Friedrich Steinle has designated as exploratory experimentation. According to Steinle, exploratory experimentation is "driven by the elementary desire to obtain empirical regularities and to find concepts and classifications by means of which those regularities can be formulated. It typically takes place in those periods of scientific development in which—for whatever reasons—no well-formed theory or even no conceptual framework is available or regarded as reliable."[556] Most importantly, exploratory experimentation is "characterized by great openness toward new and unexpected empirical findings and a willingness to revise and reconceive regularities and their representation."[557] In short, drawing on Steinle, I argue that the use of fMRI in contemporary hysteria research has opened up the possibility of giving "unknown answers to questions that the experimenters themselves are not yet able to clearly ask."[558] And although these answers have so far remained tentative, they have produced two significant epistemic effects.

First, by building upon the experimental finding of previous neuroimaging studies, researchers are learning to formulate increasingly more complex research questions about the conjectured neurophysiological basis of hysteria. For example, in 2009, Cojan et al. decided to use fMRI to explicitly address conflicting hypotheses that previous neuroimaging studies had posited. Cojan et al. thus designed an experiment to test whether hysterical paralysis arose "from active inhibition of willed movement," or from "a functional dissociation between discrete brain networks supporting executive and sensorimotor functions."[559] This particular aspect of the exploratory character of the fMRI-based hysteria research will be discussed in detail in chapter 4. Second, there is a steadily growing number of fMRI studies, all of which have registered some cerebral dysfunction in patients with hysterical symptoms. Taken together, such studies have generated sufficient empirical findings to persuade the medical community that hysteria might indeed be a genuine brain disorder.[560]

555 Burgmer et al., "Movement Observation"; Ghaffar, Staines, and Feinstein, "Sensory Conversion Disorder"; Spence et al., "Disorder of Movement"; and Werring et al., "Visual Loss."

556 Steinle, "Entering New Fields," S70.

557 Steinle, *Exploratory Experiments*, 296.

558 Rheinberger, *History of Epistemic Things*, 28.

559 Cojan et al., "Inhibition," 1027.

560 See, e.g., Feinstein, "Advances," 917–18.

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In sum, this section has shown that the exact nature of functional brain disturbances underlying hysterical symptoms remains an open question that fMRI research continues to address through a continually growing series of exploratory experiments. However, what by now appears to be beyond question is that some as yet unknown abnormal changes in how the brain works underpin the formation of hysterical symptoms.[561] Hence, although it has so far failed to solve hysteria's puzzle, I suggest that the fMRI research has nevertheless succeeded in one thing. Through the increasingly systematic experimental inscription, this research has already managed to ground this elusive disorder in the patients' bodies, or more specifically, the patients' active brains. This semantic transcription has had far-reaching consequences on how hysteria is currently being redefined in the broader medical context. In what follows, I will now turn to discussing these consequences.

2.4.2 Transforming Medically Unexplained into 'Genuine' Somatic Symptoms

By repeatedly linking diverse somatic manifestations of hysteria to localisable brain dysfunctions, fMRI research has conferred a newly won sense of physical reality on these symptoms. Thus, fMRI research has given rise to the impression that these perplexing symptoms deserve to be paid more serious attention in the medical context than had so far been the case.[562] In this section, I will argue that this change in attitude has initiated a still-ongoing reconceptualisation of hysteria's present-day successors from controversial medically unexplained symptoms into legitimate though still vaguely understood neuropsychiatric disorders. Our ensuing discussion will focus on three mutually interrelated aspects of this process. These include, first, the broadening of the research agenda; second, a decisive shift towards a neurological framework regarding the terminology, diagnostic procedures and treatment; and third, a significant revision of hysteria's current nosological successors in the *DSM-5*. We will see that fMRI research has been involved, although at times only indirectly, in all these aspects of the current reconceptualisation of hysteria.

Despite the often mutually inconsistent findings emerging from it, the sustained fMRI-based hysteria research, on the whole, has been regarded as compelling enough to rekindle more general medical interest in this disorder that had previously been dismissed as malingering.[563] In fact, I contend that by anchoring this once contested disorder into the body, fMRI has provided epistemic justification for the gradual emergence of a much broader empirical research into present-day manifestations of hysteria within the first decade of the twenty-first century. A pertinent overview of the emerging research directions was provided by an early and highly influential compilation that gathered contributions from over twenty neurologists, neuropsychologists, and psychiatrists. Published in 2001, the monograph

561 See, e.g., Stone, "Assessment as Treatment," 12.

562 See, e.g., Hallett, "Crisis for Neurology," 269–70.

563 See, e.g., Mashall, Bass, and Halligan, "Calming Introduction," xi–xiii.

entitled *Contemporary Approaches to the Study of Hysteria: Clinical and Theoretical Perspectives* was expressly conceived as a programmatic start of a systematic "enquiry into the scientific understanding of hysteria."[564]

The monograph's editors, Peter W. Halligan, Christopher Bass, and John C. Marshall, aimed to once and for all detach hysteria from concepts such as "hysterical personality, demonic possessions, or wandering womb."[565] Instead, they placed the focus on understanding "why patients show neurological signs and symptoms seemingly without having suffered neurological trauma or disease."[566] Notably, Marshall and Halligan were among the authors of the first PET study of hysterical paralysis published in 1997 and thus belong to the pioneers of functional neuroimaging research into hysteria.[567] In this book, however, they pleaded for the establishment of a more comprehensive research agenda into hysteria, which combined neuroscientific approaches with a broader clinical perspective. Hence, in addition to the neuroimaging investigation of the disorder's underlying pathophysiology, this agenda also comprised a review of the medical history of hysteria, research into the current epidemiology, classification, and diagnosis of the clinical presentations, a systematic evaluation of a variety of potential causes, and the development of new therapeutic approaches.[568] Significantly, functional neuroimaging served both as the justification for developing such a comprehensive research agenda into hysteria and as a compelling counterargument against those who still doubted the disorder's current existence. Not only was hysteria real, the editors claimed, but what was equally beyond doubt was the existence of its specific pathophysiological mechanisms, whose empirical investigation became possible with the advent of functional neuroimaging.[569]

Over the following two decades, the proposed agenda was taken up by a continually growing number of researchers. Many of these researchers—like Marshall and Halligan—have also been active in functional neuroimaging hysteria research.[570] This resulted in the proliferation of studies focused on more systematically examining the nature of hysterical symptoms. It also led to the development of more efficient diagnostic procedures and clinical management.[571] In the initial phase, new studies were designed to address the perennially contentious topics of the apparent disappearance of hysterical symptoms from the clinical practice and the enduring

564 Mashall, Bass, and Halligan, xiv. See also Halligan, Bass, and Marshall, *Contemporary Approaches*.

565 Mashall, Bass, and Halligan, "Calming Introduction," xi.

566 Mashall, Bass, and Halligan, xi.

567 See Marshall et al., "Hysterical Paralysis."

568 Mashall, Bass, and Halligan, "Calming Introduction," v-vi.

569 Mashall, Bass, and Halligan, xiii-xiv.

570 For instance, Jon Stone was the principal author of the fMRI study Stone et al., "Simulated Weakness." Mark Hallett co-authored multiple fMRI studies, such as Maurer et al., "Impaired Self-Agency"; Nahab et al., "Impaired Sense of Agency"; and Voon et al., "Involuntary Nature."

571 See, in particular, two seminal compilations: Hallett, Stone, and Carson, *Functional Neurological Disorders*; and Hallett et al., *Psychogenic Movement Disorders*.

fear of misdiagnosis.[572] The new data have shown that hysterical symptoms are highly prevalent in medical settings. The authors of one large-scale study concluded that hysterical symptoms were the second most common reason for patients being referred to a neurologist.[573] The same study also provided evidence that hysterical symptoms can now be diagnosed with considerable accuracy. According to Stone et al., the estimated misdiagnosis rate, defined as a chance of overseeing a 'genuine' organic disease, was as low as 0.4%.[574] Next, these findings were complemented by studies whose authors compared the historical and contemporary clinical descriptions of the physical characteristics of various hysterical symptoms. The conclusion drawn from such comparisons was that physical and phenomenological features of hysterical symptoms remained consistent over the last hundred and twenty years.[575] In short, the somatic symptoms that appear in the current clinical contexts were deemed analogous to those from Charcot's, Janet's, and Freud's descriptions.

Having first delivered empirical evidence for the continued presence and current clinical significance of hysterical symptoms, in the next phase, researchers started tackling other equally contested aspects of hysteria. The new research directions thus included symptom classification, terminology, and the question of the adequacy of the official diagnostic criteria and methods.[576] Felicitously, these research directions were additionally fuelled by the concurrent preparations for the fifth edition of the *DSM*.[577] Acrimonious debates that arose in this context about how to divide and regroup individual hysterical symptoms are too complex to be dealt with here in detail.[578] But what is of interest for this enquiry is to retrace how the ongoing neurological reframing of hysteria influenced the concurrent discussions on how to rename the symptoms. Despite major disagreements among experts on multiple aspects of the prevalent terminology, the consensus emerged that a rebranding of hysteria's nosological successors was required.[579] The explicit aim of this rebranding

572 See, e.g., Fink, Steen, and Sondergaard, "First-Time Referrals"; Snijders et al., "Unexplained Neurological Symptoms"; Stone et al., "Change at Follow-Up"; Stone et al., "Myth"; and Stone et al., "3781 Patients."

573 Stone et al., "Change at Follow-Up," 2878. The authors of this study have asserted that the only more common reason for visiting a neurologist was a headache. Ibid.

574 Stone et al., 2878.

575 Stone et al., "Disappearance," 14.

576 See, e.g., Kanaan et al., "What's so Special"; Mayou et al., "Somatoform Disorders"; Nicholson et al., "Problematic Diagnosis"; Owens and Dein, "Conversion Disorder"; and Reynolds, "Classification Issues."

577 "Beginning in 2000, work groups were formed to create a research agenda for the fifth major revision of *DSM* (*DSM–5*). These work groups generated hundreds of white papers, monographs, and journal articles, providing the field with a summary of the state of the science relevant to psychiatric diagnosis and letting it know where gaps existed in the current research, with hopes that more emphasis would be placed on research within those areas. In 2007, APA formed the *DSM–5* Task Force to begin revising the manual as well as 13 work groups focusing on various disorder areas. *DSM–5* was published in 2013." APA, "*DSM* History," n.p.

578 For different positions in this debate, see, e.g., Edwards, Stone, and Lang, "Change the Name"; Reynolds, "Classification Issues"; and Starcevic, "Somatic Disorders and *DSM-V*."

579 Edwards, Stone, and Lang, "Change the Name," 850.

was to establish the terminology that would signalise two things. First, the rebranding was meant to express a change of the attitude towards patients, whose somatic complaints were now perceived as 'real.' Second, the new terminology was also meant to emphasise the adoption of the new "scientific approach to the mechanisms behind" the patients' symptoms.[580]

In the process, the use of the label 'hysteria' was given up due to its outdated etymological link to the uterus and "its connotation as a dismissive term to describe people who are overemotional and making a fuss over nothing."[581] Although popular among physicians, the term 'psychogenic' was criticised for its by then contested implication of a purely psychological aetiology and its lack of acceptance among patients, who perceived it as stigmatising.[582] The alternative labels such as 'medically unexplained,' 'non-organic,' 'conversion disorder,' 'somatisation,' and 'somatoform' were declared equally inappropriate on similar grounds.[583] Instead, a growing number of experts, particularly neurologists, have started to advocate the return to the nineteenth-century term 'functional disorder.'[584] Importantly, the adoption of this label was meant to signify the growing consensus that the somatic symptoms in question arose due to a malfunction of the structurally undamaged brain. It was argued that by avoiding the implication of psychological causation, this designation liberated both physicians and patients from "the straight-jacket of the term 'psychogenic,'" thus allowing them to focus on other factors involved in the generation and maintenance of hysterical symptoms.[585] According to its proponents, besides being regarded as inoffensive and thus acceptable to patients, another significant advantage of the label 'functional disorder' was its apparent aetiological and theoretical neutrality.[586] It was argued that the label 'functional' emphasised how symptoms arose and not why.

However, I suggest that the current use of the designation 'functional disorder' is far from atheoretical or neutral since it is directly linked to the re-embedding of hysterical symptoms into a neurological framework. Historically, Charcot deployed this term to emphasise hysteria's distinct neurophysiological nature despite the absence of a detectable anatomical lesion.[587] His use of this term was grounded in the conjecture that hysteria was caused by a functional lesion—a reversible anatomically localisable disturbance in brain function. As discussed earlier in this chapter, Freud later reinterpreted the label 'functional' in purely psychological terms to refer to pathological effects of repressed traumatic memories. Hence, the term 'functional' was used at different historical moments to designate both the hypothesised brain-based and the purportedly purely psychogenic nature of hysteria. But as my analysis above has

580 Edwards, Stone, and Lang, 850.

581 Edwards, Stone, and Lang, 850.

582 Edwards, Stone, and Lang, 850.

583 Edwards, Stone, and Lang, 850. See also Dimsdale and Creed, "Preliminary Report."

584 See, e.g., Edwards, Stone, and Lang, "Change the Name"; Hallett, "Crisis for Neurology"; and Mayou et al., "Somatoform Disorders." On the historical uses of the term, see Trimble, "Functional Diseases."

585 Edwards, Stone, and Lang, "Change the Name," 851.

586 See, e.g., Mayou et al., "Somatoform Disorders," 851.

587 See chapter 1 for details.

foregrounded, the current revival of the label 'functional' rests on the explicit semantic silencing of Freud's and the simultaneous reactivation of Charcot's interpretation of this term.[588]

It should be noted that the legitimacy of the renewed neurophysiological reconceptualisation of the term 'functional' is explicitly grounded in the empirical evidence emerging from the ongoing fMRI hysteria research.[589] Through fMRI brain maps, which visualise hysteria patients' aberrant brain activity, Charcot's concept of the functional cerebral lesion appears to be gaining its retrospective empirical validation. It can thus be said that Charcot's concept of the functional cerebral lesion has once again become semantically operative. Finally, it should not be neglected that the reactivation of the neurological context through the act of hysteria's renaming into a functional disorder was also expressly aimed at encouraging further neurobiological research into "how functional changes in the brain produce symptoms."[590] It is, therefore, hardly surprising that—although not universally accepted—'functional' has become the term of choice in the neurological literature and especially in fMRI studies since the mid-2010s.[591] In other areas, the discussions about hysteria's terminology continue unabated, as does the parallel use of multiple alternative labels.[592]

Significantly, the expansion of medical research into hysteria has led not only to the revision of terminology but also to major shifts in the diagnostic criteria and procedures. Multiple findings appeared to challenge the thus far widespread suspicion among physicians that the majority of hysteria patients intentionally feigned their symptoms. For example, neurologists started to argue that the assumption of malingering could not account for the similar ways in which different patients described their symptoms.[593] What could be even less attributed to malingering was the fact that if untreated, most hysteria patients remained symptomatic and severely disabled for many years.[594] Moreover, although their findings currently remain inapplicable in the diagnostic context, several fMRI studies have reported that distinctly different neural processes were associated with 'genuine' and intentionally feigned hysterical symptoms.[595] As a result, the consensus has emerged that since the suspicion of wilful deception appears unfounded in most cases, the explicit exclusion of malingering should no longer be attributed relevance in the clinical practice or during diagnosis.[596]

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588 I am using the term silencing here in Jäger's sense. Jäger has argued that a particular meaning can be silenced if it becomes detached from the original transcription. See Jäger, "Transcriptivity Matters," 62.

589 Stone et al., "Potential Solutions," 370.

590 Edwards, Stone, and Lang, "Change the Name," 851.

591 See, e.g., Hallett, Stone, and Carson, *Functional Neurological Disorders*. See also LaFaver et al., "Opinions and Clinical Practices," 979, 981.

592 For a criticism of this approach, see, e.g., Fahn and Olanow, "They Are What They Are"; and Reynolds, "Classification Issues."

593 Stone, "Functional Symptoms in Neurology," 186.

594 Stone, 186.

595 See, e.g., Stone et al. "Simulated Weakness"; and van Beilen et al., "Conversion Paresis." For a more detailed discussion of these studies, see section 4.1.1.

596 Stone et al., "Potential Solutions," 371.

Even more dramatically, in the process of the intensified refocusing of the clinical attention onto symptoms, a gradual reappraisal of old, long-ago discarded diagnostic approaches that rested on the so-called positive signs of hysteria took place. Most of such diagnostic signs were instituted first by Charcot and then also by several other neurologists in the late nineteenth and early twentieth centuries.[597] As discussed in chapter 1, such signs consisted in identifying symptoms' particular features or physical patterns, such as tunnel vision or a sharply demarcated, geometrically shaped distribution of anaesthesia. Charcot deemed such features not only as inconsistent with other neurological disorders but also as highly specific to hysteria.[598] But in the course of the psychogenic reinterpretation of hysteria, such physical signs had been dismissed as diagnostically unreliable and banished from neurology textbooks throughout the twentieth century.[599] Nevertheless, generations of neurologists continued to unofficially teach their younger colleagues about these signs at the patients' bedsides.[600] Yet, in stark opposition to their nineteenth-century deployment, until the 1990s, these signs were treated "as parts of neurologic lore."[601] They were regarded as "'tricks of the trade' which could be used to 'catch the patient out' and show that there was indeed nothing wrong with them."[602] Put simply, as long as hysteria remained embedded into a predominantly psychological framework, these signs, if at all used, were interpreted as an indication that hysterical symptoms lacked any physical reality.

However, since the turn of the twenty-first century, with the increasing acceptance of neurophysiological accounts that have once more linked hysterical symptoms to a potentially measurable functional disturbance of the brain, the meaning attributed to 'positive' physical signs of hysteria has shifted again. In this new semantic framework, the clinical features of hysterical symptoms have started to acquire renewed diagnostic relevance.[603] In the process, the focus has been placed on two types of physical signs. One type of sign demonstrates the 'internal inconsistency' of hysterical symptoms by showing that these symptoms are identifiable under one set of conditions but disappear when tested differently. For example, patients with hysterical leg weakness cannot flex their ankle while lying on a bed, yet they can stand or walk on tiptoes.[604]

The other type of 'positive' signs foregrounds the symptoms' incongruence with organically determined diseases. An example of such incongruence is the so-called

597 See, e.g., Gould et al., "Validity of Hysterical Signs," 593–94.

598 See section 1.3.1.

599 Gould et al., "Validity of Hysterical Signs," 596.

600 Gould et al., 596; and Stone and Edwards, "Trick or Treat," 282.

601 Stone and Edwards, "Trick or Treat," 282.

602 Stone and Edwards, 282.

603 See, e.g., Stone, "Functional Symptoms in Neurology," 182–85; and Stone, Carson, and Sharpe, "Assessment and Diagnosis," i6–11.

604 Stone et al., "Potential Solutions," 372. Another pertinent example of 'internal inconsistency' is Hoover's sign. While sitting, a patient with hysterical limb paralysis is unable to voluntarily press the heel of the affected limb against the floor and thereby extend his hip. However, when asked to flex his healthy hip against resistance by lifting the unaffected leg into the air, he involuntarily presses the affected heel into the floor. Stone, "Functional Symptoms in Neurology," 183.

tunnel vision: "A patient is found to have a field defect which has the same width at 1 m as it does at 2 m, (when it should be twice as wide according to the laws of physics)."[605] Interestingly, this particular clinical sign designates the same loss of peripheral vision Charcot systematically measured and visualised through perimetric maps.[606] Another 'incongruent' physical sign Charcot regarded as diagnostically salient and which has recently been reinstituted in the clinical context is the so-called non-anatomical sensory loss. In a striking similarity to Charcot's designation, non-anatomical sensory loss is currently described as being characterised by "sharply demarcated boundaries at the shoulder or at the groin, a shape of strictly unilateral glove or sock or involvement of only half a limb."[607]

Significantly, such 'positive' physical signs are now regarded to be specific to hysteria. Hence, in the current clinical context, neurologists are semantically reactivating the meaning Charcot had initially attributed to physical signs of hysteria. Just as Charcot once did, neurologists now use such physical signs to infer that the patient's nervous system is structurally undamaged and that an underlying functional neurological problem must be the cause of the symptom.[608] In other words, these seemingly contradictory physical features are now taken to suggest that "normal function is possible, but that the patient" simply cannot voluntarily access this normal function.[609] Importantly, this interpretation is fully aligned with the reframing of hysteria into a disorder arising from some still unknown brain dysfunction, which, as we have seen, is primarily driven by the fMRI research.

Under current medical standards, to qualify for a renewed diagnostic implementation, the clinical feature of hysterical symptoms must first undergo the process of structured empirical validation.[610] Thus, in recent years, multiple studies were carried out to test and quantify the diagnostic accuracy and reliability of hysterical symptoms' various clinical characteristics that had traditionally been used without any systematic verification.[611] As a result of this process, the number of symptoms' physical features instituted in the neurological context as sufficiently reliable

605 Stone et al., "Potential Solutions," 372.

606 For details, see section 1.3.1.

607 Daum, Hubschmid, and Aybek, "'Positive' Clinical Signs," 186. For Charcot's description of the hysteria-specific sensory loss (i.e., anaesthesia) and his use of body maps to investigate and classify its various shapes, see section 1.3.1.

608 In line with the current recommendations, this is how a neurologist should explain the diagnosis to the patient: "Your brain is having trouble sending a message to your leg to make it move, but when you are distracted the automatic movements can take place normally. This test shows me that there is a problem with the function of your nervous system, not damage to it. It's basically a problem with the function of the nervous system—a bit like a software problem instead of a hardware problem." Stone, "Assessment as Treatment," 12.

609 Edwards, Cope, and Agrawal, "Functional Neurological Disorders," 267. See also ibid., 269.

610 Daum, Hubschmid, and Aybek, "'Positive' Clinical Signs," 180.

611 The validation rests on testing the reliability of each clinical sign in samples that contain a group of patients with a hysterical symptom and a separate group of patients with a similar neurological disorder. For details, see Gasca-Salas and Lang, "Neurologic Diagnostic Criteria," 193–212. See also Daum, Hubschmid, and Aybek, "'Positive' Clinical Signs"; and Gasca-Salas and Lang, "Neurologic Diagnostic Criteria."

'positive' clinical signs of hysteria has continually risen.[612] This means that a physician, typically a neurologist, is now expected to diagnose hysteria/functional neurological disorder based on the presence of such signs instead of focusing on excluding other organic diseases.[613] Consequently, the diagnosis of hysteria is currently undergoing a transformation from exclusionary into an inclusionary examination-based procedure that rests on identifying specific physical signs.[614] It can, therefore, be argued that not only the basic research into the neural underpinning of hysteria but also its diagnosis is being framed in increasingly physical terms, thus further anchoring this puzzling disorder into the body.

Interestingly, an additional effect of this increasing anchoring of hysteria in the body is also noticeable in the shifting approaches to treating motor symptoms. On the whole, hysterical symptoms are currently regarded as "an enormous therapeutic challenge," with "most patients failing to substantially improve."[615] Until recently, the dominant treatment options have been various forms of psychotherapy and, in some cases, the use of antidepressants.[616] Yet, in the second decade of the twenty-first century, there has been a surge of clinical research into the potential effectiveness of physical therapy for treating both hysterical paralysis and different types of excessive movements (e.g., tremors, gait disturbances, and contractures).[617] This clinical research is still in the early stages, and there is currently little agreement "of what physiotherapy should actually consist of."[618] But the common denominator across different strategies currently in use is the shared focus on graded exercises that retrain normal top-down motor control through the structured repetition and reinforcement of basic movement patterns.[619] This is typically achieved by using task-oriented exercises that redirect "the patient's focus of attention toward the goal of the movement" and "away from the individual components of the movement."[620] Patients are often encouraged to rely on

612 See, e.g., Espay et al., "Current Concepts," 1132–35.

613 "For example, a patient may have multiple sclerosis but if they have a globally weak leg with a clear cut Hoover's sign, they still have 'non-organic' weakness in addition to multiple sclerosis." Stone et al., "Potential Solutions," 371.

614 There are two caveats, however. First, although highly specific to hysteria, none of these signs is infallible. This is because the signs do not rely on standardised measurement procedures but instead require neurologists to make a judgment based on their clinical training and experience. Hence, to curtail this limited diagnostic reliability, the presence of more than one 'positive' clinical sign is required to make the diagnosis of hysteria. Stone et al., 372. Second, sufficiently validated signs have so far been established only for hysterical paralysis, movement disorders, and non-epileptic seizures, whereas those for sensory symptoms are considered less reliable. The testing and the validation of additional physical signs continue to be an area of intense research. See Espay et al., "Current Concepts," 1133–35. See also Daum, Hubschmid, and Aybek, "'Positive' Clinical Signs."

615 Czarnecki et al., "Successful Treatment," 248.

616 Czarnecki et al., 248. See also Espay et al., "Current Concepts," 1138.

617 See, e.g., Czarnecki et al., "Successful Treatment"; Jacob et al., "Motor Retraining"; Nielsen et al., "Consensus Recommendation"; Nielsen et al., "Outcomes"; and Nielsen et al., "Physio4FND."

618 Nielsen et al., "Consensus Recommendation," 1113.

619 Nielsen et al., 1115–17; and Espay et al., "Current Concepts," 1138.

620 Espay et al., "Current Concepts," 1138; and Nielsen et al., "Physio4FND," 5, article 242.

visual feedback during training (such as looking at a mirror) to optimise their motor performance.[621]

What is particularly surprising is that all the key aspects of physiotherapy currently used to treat hysteria were already entailed in Charcot's dynamometric exercise discussed in chapter 1. In another clear parallel to Charcot, the current deployment of physiotherapy is explicitly based on the assumption that hysterical symptoms arise from a potentially reversible problem "with nervous system functioning."[622] Further, just as in Charcot's case, in the present-day clinical settings, targeted physical intervention is aimed at "'retraining' the nervous system" to re-establish normal brain function.[623] Hence, in the context of motor rehabilitation therapies, hysterical symptoms are framed in distinctly neurophysiological and not psychological terms. At least implicitly, this framing points to the fact that physiotherapeutic approaches to treating hysteria have been informed by the findings generated through neuroimaging research. In turn, the neurophysiological framing of hysteria continues to be reinforced through increasing empirical evidence that various forms of physiotherapy lead to measurable improvements in symptoms.[624]

Moreover, as we will discuss in detail in chapter 4, the most recent development in this direction entails the emergence of a new strand of fMRI hysteria research. Studies comprising this research strand have begun to explicitly explore how physical treatment, used alone or in combination with psychotherapy, induces a reorganisation of hysteria patients' neural activity.[625] By empirically relating therapy-induced clinical recovery to measurable and visualisable changes in brain activity, such fMRI studies are particularly effective in supporting the view that hysteria is indeed a disorder of brain function.

Finally, the research-driven refocusing of attention on the physical basis of hysterical symptoms has also had a decisive impact on the *DSM-5*, published in 2013. As a result of this impact, the *DSM-5* radically redefined nosological successors of hysteria. First, it discarded most of the terms that had been in use since the *DSM-III* and replaced them with new diagnostic labels. In this process, the umbrella term somatoform disorders became renamed "somatic symptoms and related disorders."[626] The central subcategory of somatoform disorders, previously referred to as somatisation, was now relabelled "somatic symptom disorder."[627] As a notable exception, the subcategory of conversion disorder was retained, but the alternative designation—functional

621 Espay et al., 1138. See also Nielsen et al., "Outcomes," 676.

622 Nielsen et al., "Consensus Recommendation," 1115.

623 Nielsen et al., 1115. Similarly, the authors of another contemporary study attributed the hysterical motor symptoms to "a 'disconnect' between the patient's normal brain motor program and the normal nerves/muscles used to carry out the movement; thus, the [physical] therapy would focus on eliminating that 'disconnect.'" Czarnecki et al., "Successful Treatment," 248.

624 See, e.g., Czarnecki et al., "Successful Treatment"; Jacob et al., "Motor Retraining"; Jordbru et al., "Gait Disorder"; Nielsen et al., "Outcomes"; and Nielsen, Stone, and Edwards, "Systematic Review."

625 See, e.g., Diez et al., "Fast-Tracking"; LaFaver et al., "Before and After"; and Roy et al., "Dysphonia."

626 APA, *DSM-5*, 309.

627 APA, 309.

neurological symptom disorder—was added in parenthesis.[628] In conformity with the new terminology, the refashioned diagnostic criteria placed a distinct emphasis on the presence of one or more somatic symptoms that cause significant distress and impairment in the patients' daily lives.

Further, for the first time in the history of the *DSM*, the requirement to identify even precipitating psychological factors was dropped from the official diagnostic criteria of hysteria's nosological successors. Instead, psychological traumas or—and this was new—physical traumas were merely mentioned as potential 'associated features' that could support the diagnosis of conversion disorder. Thus, according to the *DSM-5*, the onset of physical symptoms "may be associated with stress or trauma, either psychological or physical in nature. The potential etiological relevance of this stress or trauma may be suggested by a close temporal relationship. However, while assessment for stress and trauma is important, the diagnosis should not be withheld if none is found."[629] In effect, through this reformulation, the *DSM-5* explicitly banished the last remaining residues of Freudian psychogenic theories of hysteria. At the same time, the new introduction of the notion of 'physical trauma' into the manual appears to echo one of Charcot's key tenets that physical injury and organic illness can trigger the onset of hysterical symptoms. Notably, this view is currently gaining increasing acceptance, particularly among present-day neurologists.[630]

Just as significantly, the *DSM-5* ceased to define hysterical symptoms as medically unexplained or to require a definitive exclusion of malingering.[631] And even more to the point, the diagnosis of conversion disorder was redefined to incorporate the presence of the symptoms' positive clinical signs during a neurological examination.[632] The explicit aim of these radical revisions was to acknowledge that despite the limited medical knowledge about their symptoms, the "individual's suffering is authentic."[633] No longer defined in purely negative terms, hysteria's present-day successors have thus become refashioned into neuropsychiatric diagnoses in their own right. Moreover, the new diagnostic criteria have been specifically formulated in a way that makes them

628 APA, 318.

629 APA, 319–20.

630 For contemporary studies that have, akin to Charcot, explicitly linked the onset of hysterical symptoms to physical factors such as injury or organic illness, see Pareés et al., "Physical Precipitating Factors"; Stone, Warlow, and Sharpe, "Clues to Mechanism"; Stone et al., "Role of Physical Injury." Typically, such studies are based on semi-structured interviews during which patients provide information about various circumstances that had preceded the onset of their symptoms. According to one of these studies, "physical events precede the onset of functional symptoms in most" hysteria patients. Pareés et al., "Physical Precipitating Factors," 174. "Although historically neglected in favour of pure psychological explanation, they may play an important role in symptoms development by providing initial sensory data, which along with psychological factors such as panic, might drive" the formation of hysterical symptoms." Pareés et al., 174. For remarkably similar views that Charcot developed to explain the formation of what he referred to as traumatic hysteria, see section 1.3.2.

631 APA, *DSM-5*, 309.

632 APA, 319.

633 APA, 311.

"more useful for primary care and other medical (nonpsychiatric) clinicians,"[634] thus additionally shifting hysteria away from psychiatry. This shift away from psychiatry is also evident in the following statement, with which *DSM-5* characterised the clinical prevalence of hysteria's present-day manifestations. "Individuals with disorders with prominent somatic symptoms are commonly encountered in primary care and other medical settings but are less commonly encountered in psychiatric and other mental health settings."[635]

Although, on the whole, these far-reaching changes arose from the broader medical research into hysteria, in this section, I have traced the multiple ways in which functional neuroimaging has been implicated in this process, either directly or indirectly. We have seen that by providing initial tentative evidence of hysterical symptoms' neurophysiological basis, fMRI research set the whole medical field in motion and made hysteria visible again as an object of renewed clinical attention. Ever since, fMRI research has continued to provide the empirical justification for the still ongoing redefinition of hysteria into a genuine disorder, which arises from a still not understood dysfunction of the brain.

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In sum, after a meandering trajectory over the last hundred and twenty years, during which it shape-shifted from a neurological over purely psychogenic to medically unexplainable set of symptoms, hysteria has once more settled into a neurobiological conceptual framework. My analysis in this chapter has charted the double movement through which the changing theoretical frameworks within which hysteria was conceptualised and the various investigation tools used for its study have mutually influenced each other. I have shown that the use of various types of images as research tools has risen and fallen in parallel with the introduction and dismissal of somatic concepts of this disorder. Whereas they were epistemically operative within Charcot's neurophysiological framework, empirical images became ineffective in the context of psychogenic approaches to hysteria. It was only with the declining influence of the psychogenic framework that new image-based research into hysteria could gradually emerge and, in the process of its ongoing consolidation, induce a renewed anchoring of hysterical symptoms into the body.

My analysis so far has underscored how the new image-based research has been associated with a revival of scientific interest in Charcot's hypothesis of the underlying functional brain lesion. However, in the remainder of this book, I intend to show that far from merely rehashing old theories, fMRI-based hysteria research has produced and continues to produce new empirical insights into this age-old disorder. Hence, the following two chapters will examine in detail how researchers work with fMRI to investigate the neurological basis of hysteria and what kinds of insights they have generated within the first two decades of the twenty-first century.

634 APA, 309.

635 APA, 309.