Tracing Hysteria's Recent Trajectory

From a Crisis for Neurology to a New Scientific Object in Neuroimaging Research

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Throughout its several millennia-long medical history that dates back to antiquity, hysteria has consistently been considered a mystery.[1] There are two major reasons for hysteria's mysteriousness. On the one hand, this disorder comprises an array of heterogeneous and mutually seemingly incompatible symptoms that often coexist in the same patient. These include partial or complete limb paralysis, permanent muscular contractions, involuntary tics and tremors, convulsive fits, loss of sensitivity to touch and pain, chronic pain, as well as various disturbances of vision, hearing and speech, to name just a few.[2] On the other hand, no undisputed organic cause has ever been found for these diverse somatic symptoms.[3] The earliest theories, which gave hysteria its name, attributed its symptoms to the wondering womb (i.e., hystera in Greek means uterus). By contrast, in the Middle Ages, hysteria was equated with demonic possessions.[4] In the ensuing centuries, doctors returned to defining hysteria's aetiology in naturalistic terms by variously attributing it to disturbances of the mind, brain, humoral fluids, passions, 'hysterical temperament' and, once again, the female reproductive system.[5] Following this meandering trajectory, hysteria reached the apex of its medical and cultural visibility in the late nineteenth and early twentieth centuries. This was primarily owing to the research conducted first by the French neurologist Jean-Martin Charcot and then by Charcot's former pupil and founder of psychoanalysis, Sigmund Freud.

1 For an overview of hysteria's history, see Micale, *Approaching Hysteria*, 19–29; and Trimble and Reynolds, "Brief History."

2 See, e.g., Charcot, *Diseases of the Nervous System*, 69–83; and Popkirov, *Funktionelle neurologische Störungen*, 35–181.

3 See Trimble and Reynolds, "Brief History," 3–10.

4 See Micale, *Approaching Hysteria*, 19–20.

5 See Micale, 20–24; and Trimble and Reynolds, "Brief History," 7–9.

Charcot considered hysteria a neurophysiological illness caused by a potentially reversible disturbance of brain function.[6] Conversely, Freud claimed that hysteria lacked any physiological basis and was caused by suppressed memories of some traumatic events from the patient's past.[7] Freud's psychogenic framing of hysteria dominated medicine in the twentieth century. However, in the second half of the twentieth century, as Freud's views started to be questioned by the medical community, hysteria became increasingly unpopular, both as a diagnosis and an object of sustained scientific enquiry.[8] In fact, the medical community soon dropped the very term 'hysteria' as a nosological designation and replaced it with a plethora of new diagnostic labels that have kept changing ever since.[9] In the process, hysteria seemed to disappear as a medical condition. Against this background, it is, perhaps, unsurprising that the currently dominant view in the humanities is that hysteria ceased to exist.[10]

Contrary to this view, since the turn of the twenty-first century, a growing number of epidemiological studies have shown that hysterical symptoms are prevalent in present-day neurological clinics, comprising approximately a fifth of all cases.[11] Such high prevalence means that regardless of "profession, setting or specialisation, anyone working in neurology will frequently (if not daily) encounter" patients with such symptoms.[12] Moreover, multiple contemporary medical studies have delivered empirical evidence that the physical characteristics of the present-day manifestations of hysteria overlap with the symptoms exhibited by Charcot's and Freud's patients.[13] Drawing on these studies, this chapter is informed by the view that, despite the fluctuating terminology used in the medical context to designate them, hysterical symptoms have neither disappeared nor have their clinical features changed over the centuries.

But, initially, apart from establishing their continued presence and the constancy of their clinical features, the medical community remained at a loss about how to deal with these baffling somatic symptoms that lack any apparent organic cause. In fact, for the major part of the first two decades of the twenty-first century,

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6 See Charcot, *Diseases of the Nervous System*, 13–14, 278. For more details on Charcot's neurophysiological research on hysteria in which various visualisation techniques, including photography, played major epistemic functions, see Muhr, *From Photography to fMRI*, chapter 1.

7 Freud, *Standard Edition*, 147–54, 169–72.

8 Nicholson, Stone, and Kanaan, "Problematic Diagnosis," 1267–68.

9 For a detailed analysis of this process, see Muhr, *From Photography to fMRI*, chapter 2.

10 See, e.g., Bronfen, *Knotted Subject*, xi; and Micale, *Approaching Hysteria*, 29.

11 For an overview of these studies, see Carson and Lehn, "Epidemiology," 51.

12 Popkirov, *Funktionelle neurologische Störungen*, 6 (my translation).

13 See, e.g., Stone et al., "'Disappearance' of Hysteria," 14; Stone and Aybek, "Functional Limb Weakness," 213–28; and Stone and Vermeulen, "Functional Sensory Symptoms," 271–81.

the medical community even struggled with how to label the symptoms. Some medical experts continued, for a while, to refer to these symptoms as 'hysterical,' whereas others chose to use a host of other official and unofficial designations.[14] These included such diverse terms as conversion disorder, somatisation, somatoform disorder, medically unexplained, non-organic, pseudoneurological and psychogenic symptoms. It is only recently that neurologists have mainly settled on the current designation of functional neurological disorder.[15]

This chaotic situation prompted the American neurologist Mark Hallett to declare hysterical symptoms under all their various official and unofficial designations "a crisis for neurology" in an article published in 2006.[16] So far, humanities scholars have remained oblivious both to the high prevalence of hysterical symptoms in contemporary clinics and to Hallett's declaration of crisis. But as I will show in this chapter, although Hallett's declaration of crisis failed to reach a non-specialist audience, it proved to be highly productive in the medical context. This declaration, I will argue, played a role in the gradual transformation of hysteria into a new scientific object in the current neurological research, or more specifically, imaging neuroscience.

It should be noted that, in what follows, I will continue to use the terms 'hysteria' and 'hysterical' to summarily designate the heterogeneous somatic symptoms discussed in this chapter. In doing so, I do not mean to imply the existence of hysteria as a unitary transhistorical disease entity. Instead, in continuing to use the term hysteria, I primarily want to achieve two things. First, I aim to avoid the terminological confusion that has characterised the medical research into these symptoms during the period I am examining in this chapter. Second, and even more importantly, I want to emphasise that, as detailed above, present-day neurologists insist that, despite the changing terminology, the symptoms they are investigating are the same as those that stood at the centre of Charcot's and Freud's research more than a century earlier.

My analysis in this chapter is informed by the approach set forth by Lorraine Daston in her introduction to the edited volume *Biographies of Scientific Objects*.[17] This approach consists in examining "how a heretofore unknown, ignored, or dispersed set of phenomena is transformed into a scientific object that can be observed and manipulated, that is capable of theoretical ramification and empirical surprises, and that coheres, at least for a time, as an ontological entity."[18] As Daston perti-

14 See, e.g., Trimble and Reynolds, "Brief History," 3.

15 See Trimble and Reynolds, 3; and Hallett, Stone, and Carson, *Functional Neurologic Disorders*, ix–x.

16 Hallett, "Crisis for Neurology," 269.

17 See Daston, "Scientific Objects."

18 Daston, 5.

nently pointed out, although they "may not be invented," scientific objects "grow more richly real as they become entangled in webs of cultural significance, material practices, and theoretical derivations. In contrast to quotidian objects, scientific objects broaden and deepen."[19] Hence, this approach foregrounds "the distinctively generative, processual sense of the reality of scientific objects, as opposed to the quotidian objects that simply are."[20]

Following Daston, my aim in this chapter is to trace the transformation of hysteria from a set of somatic symptoms that the medical community had ignored for decades into a distinct scientific object, i.e., a clearly defined target of sustained and systematic neuroimaging research. I will thereby argue that Hallett's declaration of crisis provided an important impulse for transforming hysteria into a scientific object as it attributed medical significance to these heretofore overlooked symptoms. Finally, I will demonstrate that by generating new empirical insights into the neurological basis of hysteria, neuroimaging studies of this disorder have gradually stabilised hysteria as a scientific object in its own right by the end of the 2010s. Since it investigates the 'thickening and deepening' of hysteria as a scientific object of neuroimaging research, this chapter is conceived as an interdisciplinary enquiry situated at the intersection of science and technology studies (STS), historical epistemology and the history of medicine. But before we can turn to discussing the stabilisation of hysteria into a scientific object of neuroimaging studies, we must first examine what, according to Hallett, constituted the crisis at the beginning of the twenty-first century.

Declaration of Crisis and Its Effects on the Medical Community

As a neurologist specialising in disturbances of movement, Hallett's initial declaration of crisis for neurology—and much of his subsequent analysis—centred primarily on a particular set of hysterical symptoms, which, at the time, were known as psychogenic movement disorders.[21] These symptoms comprise different types of excessive involuntary movements, such as tremors, tics, permanent muscular contractions, gait disturbances and parkinsonism.[22] Yet, already in the second paragraph of his article, Hallett made it clear that his declaration of crisis encompassed a much larger set of highly heterogeneous somatic symptoms such

19 Daston, 13.

20 Daston, 13.

21 Hallett, "Crisis for Neurology," 269. The current designation for these symptoms is functional movement disorders. See Hallett, "Crisis Resolved," 971.

22 Hallett, "Crisis for Neurology," 270.

as "blindness, weakness, paralysis, sensory loss, aphonia, and, most frequently, psychogenic nonepileptic seizures (pseudoseizures)."[23]

Previously grouped under the medical label of hysteria, when Hallett wrote his article, these symptoms were jointly referred to by medical experts as "medically unexplained."[24] The common defining characteristic of these diverse symptoms—from blindness to gait disturbances—was that, despite looking like symptoms of organic diseases, they lacked an apparent organic cause. To be more exact, although these symptoms resulted in "severe impairment in social, occupational, or other important areas of functioning," when patients exhibiting them were submitted to systematic medical examination, doctors could not find any measurable abnormalities.[25] Thus, apart from a vague and, by that time, already contested assumption that psychological factors might somehow contribute to the symptoms' formation, there was no medical knowledge about the physiological basis of these symptoms.[26] If anything, these symptoms appeared physiologically impossible and unreal, which, at the time, served as a sufficient justification to interchangeably designate them as 'hysterical,' 'medically unexplained' or 'psychogenic.'

Hallett's formulation of the crisis that these symptoms came to represent in the first decade of the twenty-first century is worth quoting to its full extent as it effectively sums up the problems the medical community was facing. "The nature of the crisis is that there are many patients, we don't understand the pathophysiology, we often don't know how to make the diagnosis, we don't know how to treat the patients, the patients don't want to hear that they have a psychiatric disorder and they go from doctor to doctor, psychiatrists don't seem interested anyway, and the prognosis is terrible."[27]

If we unpack this statement, it becomes apparent that, according to Hallett, hysterical symptoms represented a twofold crisis from the medical point of view. First, they represented what could be termed a crisis of knowledge, which consisted in the medical community's lack of understanding of the symptoms' nature. Importantly, as Hallett further explained, this lack of understanding encompassed both the symptoms' potential causes and the underlying mechanism that determined the

23 Hallett, 269.

24 Hallett, 269. The official medical labels at the time included somatoform disorders, somatisation disorder and conversion disorder. See APA, *DSM-IV*, 445–57. For a detailed discussion of how the medical category of hysteria was transformed into these nosological labels, which by the mid-2010s then gave way to other nosological designations, see Muhr, *From Photography to fMRI*, chapter 2.

25 APA, *DSM-IV*, 452.

26 "Because psychological factors are so ubiquitously present in relation to general medical conditions, it can be difficult to establish whether a specific psychological factor is etiologically related" to these symptoms. APA, 453.

27 Hallett, "Crisis for Neurology," 269.

symptoms' clinical evolution.[28] Second, and as the direct consequence of the crisis of knowledge, hysterical symptoms also represented what could be called a crisis of action for the medical community. Simply put, doctors were at a loss about how to deal with the patients either in terms of diagnosis or medical treatment. Not knowing what course of action to take when faced with these puzzling symptoms, "[n]eurologists generally sent the patients away without any plan."[29]

The situation that Hallett described was not new. In fact, accounts of undiagnosable and, therefore, essentially untreatable patients who exhibited unexplainable somatic symptoms without any detectable organic basis abound in the medical literature in the late twentieth century.[30] However, in such accounts, the patients were declared to be problematic or frustrating and summarily dismissed as attention-seeking simulators who did not suffer from any genuine illness.

What is perhaps easy to overlook at a superficial glance is that by designating the medical community's inability to deal with hysterical symptoms as a crisis, Hallett introduced a major semantic revision. He effectively shifted the blame away from the patients. In Hallett's reinterpretation, the patients were no longer problematic. Instead, it was the doctors who were unable to help the patients (i.e., the crisis of action) because they lacked an understanding of the nature of the baffling hysterical symptoms (i.e., the crisis of knowledge). But importantly, Hallett's assertion of the crisis was not a mere passive acknowledgement of medicine's shortcomings. I argue that this assertion actively signalled—and thus further reinforced—a gradually emerging change of medical attitudes towards hysteria. My argument is supported by two aspects of Hallett's assertion of crisis, which foreground such changing attitudes towards present-day manifestations of hysteria.

The first aspect is that Hallett directly challenged the widespread view in the medical community I mentioned earlier, according to which hysteria patients were simulators who voluntarily produced fake symptoms to deceive their doctors. While challenging this view, Hallett nevertheless admitted that diagnostically differentiating between hysteria and malingering could be difficult in clinical practice due to multiple puzzling clinical features of hysterical symptoms. These features included the symptoms' "abrupt onset; spontaneous remissions;" considerable variations in "severity, and body distribution; decrease or disappearance with distraction," as well as "response to placebo, suggestion, or psychotherapy."[31] Yet despite these puzzling

28 "Although psychiatric etiology is very likely relevant in most cases, we don't really know this for sure, and the pathophysiology is unknown." Hallett, 269.

29 Hallett, "Crisis Resolved," 971.

30 See, e.g., Hahn et al., "Doctor-Patient Relation." For a more detailed analysis of this situation, see Muhr, *From Photography to fMRI*, section 2.2.3.

31 Hallett, "Crisis for Neurology," 270.

features, Hallett forcefully insisted that hysterical symptoms "are not 'not real.' They are very real and a major problem for the patient."[32]

This statement is highly significant because by acknowledging the reality of hysterical symptoms, Hallett effectively foregrounded their medical relevance. The implication was that because they suffer from what Hallett designated as 'very real' symptoms, these patients, of whom there are many, could no longer be simply sent away as malingerers. Instead, their complaints had to be taken seriously. This, in turn, meant that these patients warranted clinical intervention to alleviate their suffering. But since there were no known clinical interventions that helped these patients,[33] Hallett's statement also meant that the seemingly unexplainable hysterical symptoms had to be paid scientific attention to uncover their hidden nature. In other words, to potentially solve the crisis of action, the medical community first had to address the underlying crisis of knowledge. And as Hallett explicitly pointed out in his article, already at that point, some researchers were "becoming interested" in investigating hysteria, but there was "a long way to go."[34] In 2006, when Hallett published his article, there were many open questions. For example, how to provide reliable empirical evidence for the physical reality of hysterical symptoms despite their apparent lack of organic basis? How to investigate the symptoms' specificity despite their heterogeneity and the tendency to fluctuate when submitted to examination? Moreover, how to reliably differentiate hysterical symptoms from similar symptoms of other diseases?

This brings us to the second aspect in which Hallett's declaration of crisis expressed changing attitudes to hysteria. Apart from foregrounding the physical reality of hysterical symptoms, Hallett also explicitly rejected Freud's purely psychogenic theory of hysteria, which had dominated medicine throughout the twentieth century. Admittedly, Hallett conceded that a psychological factor might have aetiological relevance in some patients. Yet, he insisted that it was "not clear how this [psychological factor] generates" the hysterical symptom in question.[35] And since Freud's approach failed to account for the development of symptoms, Hallett forcefully concluded that "[w]e now need a better theory."[36]

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32 Hallett, 270.

33 Hallett, 269.

34 Hallett, 271. In fact, Hallett himself was part of such a group of researchers, who in October 2003 "gathered together to discuss the state of the art regarding psychogenic movement disorders" in the framework of the inaugural international meeting dealing with this subset of hysterical symptoms. Hallett et al., *Psychogenic Movement Disorders*, vii. The result of this meeting was a book published in 2006, the same year in which Hallett declared hysteria to be a crisis for neurology. See Hallett et al.

35 Hallett, "Crisis for Neurology," 269.

36 Hallett, 269.

Moreover, in opposition to Freud's claim that hysterical symptoms defy physiology, Hallett posited that such symptoms are determined by a distinct, even if at the time still unknown, pathophysiology.[37] Hence, the implication entailed in Hallett's call for a new theory was that one possible way out of the crisis was to develop an understanding of the hysterical symptoms' unknown pathophysiology through medical research. However, the challenge, so it would seem, lay in how to determine what might constitute this unknown neurological pathophysiology. After all, the only accepted medical fact about hysteria at that point was that patients showed no detectable local nerve damage or structural brain pathology.[38]

But notably, by the time Hallett declared hysterical symptoms a crisis for neurology, a handful of studies that used different functional neuroimaging technologies to examine the potential pathophysiology of these symptoms had already appeared. To be more exact, ten functional neuroimaging studies of somatic hysterical symptoms were published from 1995 to 2006.[39] Three of the early studies used SPECT (single-photon emission computed tomography), whereas four used PET (positron emission tomography).[40] Three subsequent studies used fMRI (functional magnetic resonance imaging), a newer technology that by the mid-2010s became dominant in this research.[41] Each of these different functional neuroimaging technologies allowed researchers to indirectly measure and visualise hysteria patients' neural activity and thus make inferences about how their brains function. However, each technology entailed the use of a different measurement procedure and generated data about the neural activity that had different spatial and temporal resolutions.[42] In addition to using different neuroimaging technologies, the ten initial studies also investigated different hysterical symptoms. One study focused on patients with hysterical blindness, another two on hysterical sensory disturbances, whereas the rest investigated hysterical paralysis.

37 Hallett, 269.

38 Hallett, 270.

39 Burgmer et al., "Movement Observation"; Halligan et al., "Hypnotic Paralysis"; Mailis-Gagnon et al., "'Hysterical' Anesthesia"; Marshall et al., "Hysterical Paralysis"; Spence et al., "Disorder of Movement"; Tiihonen et al., "Cerebral Blood Flow"; Vuilleumier et al., "Sensorimotor Loss"; Ward et al., "Differential Brain Activations"; Werring et al., "Visual Loss"; and Yazici and Kostakoglu, "Cerebral Blood Flow."

40 Tiihonen et al., "Cerebral Blood Flow"; Vuilleumier et al., "Sensorimotor Loss"; Yazici and Kostakoglu, "Cerebral Blood Flow"; Halligan et al., "Hypnotic Paralysis"; Marshall et al., "Hysterical Paralysis"; Spence et al., "Disorder of Movement"; and Ward et al., "Differential Brain Activations."

41 Burgmer et al., "Movement Observation"; Mailis-Gagnon et al., "'Hysterical' Anesthesia"; and Werring et al., "Visual Loss."

42 For a succinct overview of these technologies, the mutual comparison concerning their respective epistemic possibilities and limitation, and their gradual introduction into hysteria research, see Muhr, *From Photography to fMRI*, sections 2.3.1–2.3.3.

Yet, regardless of their differences, all ten studies had in common that they implemented some type of functional neuroimaging technology with the explicit aim of linking hysterical symptoms to an abnormal pattern of brain activity. Put differently, in the context of these neuroimaging studies, the search for the unknown pathophysiology underlying hysterical symptoms was defined in terms of a potentially detectable disturbance of function that affected one or more brain regions. And although Hallett did not explicitly mention any of these studies in his declaration of crisis, their scientific goals seemed to perfectly align with his call to action that foregrounded the need for scientific research into hysteria's underlying pathophysiology.[43]

Until 2006, neuroimaging studies of hysterical symptoms were still few in number, although they already clearly indicated the emergence of a new research approach.[44] After Hallett declared the crisis, such studies have continually grown in number.[45] Importantly, my intention in this chapter is not to posit a causal relationship between Hallett's declaration of crisis and the growing number of neuroimaging studies of hysterical symptoms from 2007 onwards, as this would be a gross and unwarranted oversimplification.[46] However, it appears to me that by declaring a medical crisis, Hallett lent urgency to any research approach that could productively address the baffling hysterical symptoms and thus provide at least provisional insights into these symptoms' unknown pathophysiology. Prior to Hallett's declaration, neuroimaging studies of hysteria could have been written off as seemingly useless and wastefully expensive investigations into exceedingly rare symptoms that lacked broader clinical or scientific significance. After Hallett's declaration, such studies could be claimed to offer critical epistemic insights into vaguely understood and, until that point, under-researched symptoms, which affected many patients.

In short, what I am suggesting is that Hallett's designation of hysterical symptoms as a crisis for neurology served as an impetus for, at the time, already emerg-

43 It is safe to assume that Hallett was well acquainted with the early neuroimaging findings since the book he co-edited in 2006 contained an article that provided a systematic overview of functional neuroimaging studies of hysteria from 1995 to 2004. See Fink, Halligan, and Marshall, "Neuroimaging of Hysteria."

44 See my analysis about the initial emergence of fMRI-based hysteria research as a sustained scientific practice in the mid-2010s in Muhr, *From Photography to fMRI*, section 2.3.3.

45 See, e.g., Balachandran et al., "Stress"; Blakemore et al., "Aversive Stimuli"; Hassa et al. "Motor Inhibition"; Stone et al., "Simulated Weakness"; and van Beilen et al., "Conversion Paresis."

46 Elsewhere, I have examined how the gradual re-biologisation of psychiatry, the intensifying of broader neuroimaging investigation of the neural underpinning of various cognitive functions in healthy individuals and in clinical populations, as well as the adoption of fMRI as the dominant functional neuroimaging technology have all played roles in establishing neuroimaging research on hysteria as a sustained scientific practice. See Muhr, *From Photography to fMRI*, sections 2.3.1–2.3.3.

ing neuroimaging research into these symptoms by justifying the wider medical salience of this research in its early stages.[47] Two aspects support this assumption. First, several neuroimaging studies explicitly quoted Hallett's declaration of crisis to emphasise the medical relevance of their investigation.[48] Second, following his declaration of crisis, Hallett took an active part in the neuroimaging research into hysteria. Since 2006, he has co-authored multiple neuroimaging studies of hysterical symptoms and several major systematic reviews that summarised the interim results of the ongoing neuroimaging research into hysteria.[49] At a more general level, Hallett has also significantly contributed to catalysing broader medical research into hysteria. He has co-edited a seminal textbook on hysterical/functional neurological symptoms published in 2016 and recently co-founded an international professional society that gathers health professionals and scientists to "advance scientific research pertaining to functional neurological disorders."[50]

So far, we have examined Hallett's declaration of crisis and its effects on mobilising neuroimaging research into the neurophysiological basis of hysterical symptoms. But although in his 2006 article Hallett foregrounded the medical relevance of hysterical symptoms, in his description, these symptoms were still vaguely defined as "medically unexplained."[51] As I will argue in the remainder of this chapter, it is owing to the preliminary insights that the neuroimaging research has generated over the following decade and a half that hysterical symptoms have "attain[ed] their heightened ontological status" as a scientific object.[52] This new status, so I suggest, is also reflected in the fact that, especially in the research context, these diverse symptoms are now jointly referred to as manifestations of functional neurological disorder.[53]

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47 My analysis is informed by Lorraine Daston's pertinent insight that to be transformed into scientific objects, "phenomena that exist on the fringes or beneath the surface of the scientific collective consciousness" have to be charged with cultural and epistemic significance so as to "rivet scientific attention" and thus become not just visible but also perceived as amenable to sustained research activity. Daston, "Scientific Objects," 6.

48 See, e.g., Schrag et al., "Dystonia," 771.

49 See, e.g., Hallett, "Crisis Resolved"; Perez et al., "Research Agenda"; and Voon et al., "Involuntary Nature."

50 "About Us," Functional Neurological Disorder Society, accessed May 15, 2022, https://www.fndsociety.org/about-us/. See also Hallett, Stone, and Carson, *Functional Neurologic Disorders*. For an analysis of how the broader medical research into hysteria has both been influenced by and fed into the neuroimaging research into this disorder, see Muhr, *From Photography to fMRI*, section 2.4.2.

51 Hallett, "Crisis for Neurology," 270.

52 Daston, "Scientific Objects," 10.

53 See, e.g., Hassa et al., "Amygdala Activity," 1, 613156.

Transforming Hysteria into an Increasingly Coherent Scientific Object

Elsewhere, I have analysed the neuroimaging research into hysteria within the first two decades of the twenty-first century by systematically examining preliminary theoretical assumptions and empirical notions that have heuristically shaped this research.[54] Here, however, I want to approach this research from a different perspective. This section will summarise four initial insights into hysteria's underlying neuropathology that neuroimaging research has delivered so far. In doing so, I intend to show that, as a result of these insights, hysterical symptoms have been gradually transformed from a loose set of seemingly mysterious, medically unexplained manifestations into a more clearly defined and "coherent category of investigation."[55] The neuroimaging insights include that, first, hysteria is distinct from simulations; second, hysterical symptoms differ from similar symptoms of organic diseases; third, hysterical symptoms intensify in stressful situations; and fourth, hysterical symptoms might be underpinned by microscopic neuroanatomical anomalies.

Hysteria Is Distinct from Intentional Simulation

Since 2006, multiple fMRI studies have explicitly compared hysterical symptoms to intentional simulation.[56] Their shared aim was to determine how the genuine hysterical symptoms differ from the feigned counterparts at the neural level. In most studies, patients with acute hysterical limb paralysis that lasted only a few days or patients with chronic hysterical paralysis that lasted several months to several years were compared to healthy individuals asked to feign limb paralysis.[57] Only one study, authored by Voon et al., departed from this approach and focused on the symptom of hysterical tremor. Moreover, unlike the other studies, Voon et al. did not compare the brain activity patterns across different groups of subjects—i.e., hysteria patients and healthy individuals. They opted instead for another type of experimental design called within-subjects analysis, which entails comparing the brain activity in the same group of subjects across different experimental conditions.[58] Specifically, Voon et al. compared the brain activity of hysteria patients experiencing involuntary intermittent hysterical tremor in their affected arm to the same hysteria patients' brain activity while they were voluntarily imitating the tremor with the same arm.

54 See Muhr, *From Photography to fMRI*, chapter 4.

55 Daston, "Scientific Objects," 8.

56 See Cojan et al., "Inhibition"; Hassa et al., "Motor Inhibition"; Stone et al., "Simulated Weakness"; van Beilen et al., "Conversion Paresis"; and Voon et al., "Involuntary Nature."

57 See, e.g., Cojan et al., "Inhibition," 1027–28; and Stone et al., "Simulated Weakness," 963.

58 Voon et al., "Involuntary Nature," 226.

Importantly, the differences among the fMRI studies that contrasted hysterical symptoms with intentional simulation were not limited to the type and chronicity of the symptom they investigated or to decisions on how many groups of subjects to recruit. What also differed across the studies was the kind of task that participants performed while the scanner measured their brain activity. In some studies, the experimental subjects were instructed to actively move a designated limb on cue in a prescribed way (e.g., flexing their foot or wrist).[59] Conversely, in another study, the subjects were asked to sometimes perform and sometimes withhold the prepared movement.[60] In yet another study, the subjects were told to simply relax and not oppose the motion that one of the experimenters passively imposed onto their limbs.[61] Moreover, even the instructions to healthy subjects on how to simulate hysterical paralysis varied across studies. For instance, in one study, the healthy participants were merely told to pretend that their limb "was too weak and heavy to move."[62] By contrast, in a more recent study, healthy subjects underwent a six-day systematic training on how to simulate hysterical paralysis convincingly.[63]

This concise analysis has foregrounded that fMRI and comparable neuroimaging technologies do not provide any straightforward, unmediated access to hysteria patients' underlying brain dysfunction. Instead, to provide potential insights into hysteria's pathophysiology, fMRI has to be embedded into carefully constructed experimental settings. This means that researchers have to make multiple interpretational decisions while designing and conducting their fMRI-based experiments. As the examples above have shown, such decisions include—but are not limited to—choosing which particular hysterical symptom to study, how many participants to recruit, whether or not to include a group of healthy control subjects and which experimental task to use.[64] Importantly, all such decisions have far-reaching epistemic consequences as they inform the findings of neuroimaging studies.

Taking this into account, we should not be surprised that all fMRI studies into the neurophysiological differences between hysterical symptoms and intentional simulation discussed above obtained mutually disparate experimental results.[65]

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59 Stone et al., "Simulated Weakness," 962–63; and van Beilen et al., "Conversion Paresis," 5, e25918.

60 Cojan et al., "Inhibition," 1028.

61 Hassa et al., "Motor Inhibition," 720.

62 Stone et al., "Simulated Weakness," 963.

63 Hassa et al., "Motor Inhibition," 720.

64 Other important decisions, whose analysis is beyond the scope of this chapter, are various technical parameters of fMRI data acquisition and details of multistage statistical analysis to which the resulting data have to be submitted in order to yield interpretable results. For a detailed analysis of these aspects of fMRI imaging, see Muhr, *From Photography to fMRI*, chapter 3.

65 Compare, e.g., Cojan et al., "Inhibition," 1026; and Hassa et al., "Motor Inhibition," 719.

What needs to be emphasised, however, is that, despite mutual inconsistencies, all studies converged on one point. Without exception, the imaging results disclosed that distinctly different brain activation patterns underpinned genuine hysterical symptoms as opposed to those that were intentionally simulated.[66] But from study to study, these differential activations patterns occupied diverse anatomical brain regions that only partly overlapped across studies.[67]

Thus, on the whole, the fMRI studies that compared hysterical symptoms to intentional simulation have failed to identify a pattern of brain activation which could be used reliably to diagnostically differentiate between genuine hysteria patients and simulators. Yet despite this shortcoming, these fMRI studies managed to deliver cumulative evidence that genuine hysterical symptoms were underpinned by different neural mechanisms than malingering. In doing so, these studies provided empirical support to Hallett's initial claim that hysterical symptoms were distinct from intentional simulation and, therefore, 'real' in the physiological sense of the word. In effect, by empirically demonstrating that hysteria was distinct from simulation, these fMRI studies significantly heightened the status of hysteria as a scientific object that deserves to be the focus of sustained neuroimaging research. Simply put, it is owing to these fMRI studies that hysterical symptoms have grown "more richly real" in the medical context.[68]

Hysterical Symptoms Are Distinct from Similar Symptoms of Other Organic Diseases

More recently, several functional neuroimaging studies have directly addressed the well-known fact that many hysterical symptoms resemble symptoms of other diseases that are caused by detectable organic disturbances.[69] For centuries, such similarities have baffled medical practitioners and represented a considerable diagnostic challenge.[70] Due to the intensified systematic work on establishing and validating diagnostic criteria within the last two decades, the clinical differentiation between hysterical symptoms and their organic counterparts has become more reliable.[71] Yet, hardly anything is known about how such seemingly similar symptoms differ at the

66 See Cojan et al., "Inhibition," 1036; Hassa et al., "Motor Inhibition," 720; Stone et al., "Simulated Weakness," 968; van Beilen et al., "Conversion Paresis," 17, e25918; and Voon et al., "Involuntary Nature," 226.

67 For a detailed analysis of the individual findings of these four fMRI studies, see Muhr, *From Photography to fMRI*, section 4.1.1.

68 Daston, "Scientific Objects," 13.

69 Espay et al., "Functional Dystonia"; Espay et al., "Functional Tremor"; Schrag et al., "Dystonia"; and Szaflarski et al., "Facial Emotion Processing."

70 See, e.g., Charcot, *Diseases of the Nervous System*, 14.

71 See Gasca-Sala and Lang, "Diagnostic Criteria."

neural level. It is this particular knowledge gap that the authors of one PET and three fMRI studies chose to address.

In a PET study by Schrag et al. and an fMRI study by Espay et al., patients with hysterical contractures—i.e., permanent muscular contractions that are now called functional dystonia—were compared to patients with organic dystonia.[72] Another fMRI study by Espay et al. compared patients with hysterical tremor to patients with the organic counterpart of this symptom, called essential tremor.[73] Finally, an fMRI study by Szaflarski et al. compared patients suffering from intermittent hysterical attacks—now called functional non-epileptic seizures—to patients diagnosed with epilepsy.[74] In addition to hysteria patients and patients with comparable organic symptoms, each study also included a third group of healthy control subjects.

Similarly to the examples discussed in the previous subsection, these four studies investigated diverse symptoms and deployed very different experimental designs. In the PET study by Schrag et al., patients with hysterical and organic contractures as well as healthy subjects were merely asked to perform simple limb movements on cue while their brains were scanned.[75] By contrast, the three fMRI studies used a more complex experimental design that comprised multiple tasks. Specifically, in the fMRI studies of hysterical contractures and tremor, participants were engaged in a simple motor task of finger-tapping, as well as two additional experimental tasks. During the latter two tasks, the participants were exposed to two different types of emotional stimuli.[76] In one of these tasks, the subjects viewed images of standardised facial expressions of emotions such as sadness, happiness and fear. During another task, the subjects were shown "a series of offensive or disgusting images" from the International Affective Picture System (IAPS), a database of photographs that are widely used in current psychological research as standardised visual stimuli for inducing affective responses.[77]

The Szaflarski et al. study into hysterical seizures also used a task during which the participants were presented with standardised photographs of faces with different emotional expressions.[78] But unlike the purely task-based studies discussed so far, Szaflarski et al. additionally deployed a more novel fMRI-based approach referred to as the resting-state connectivity. In this approach, instead of engaging the

72 Schrag et al., "Dystonia," 770; and Espay et al., "Functional Dystonia," 136.

73 Espay et al., "Functional Tremor," 179.

74 Szaflarski et al., "Facial Emotion Processing," 193.

75 Schrag et al., "Dystonia," 772.

76 Espay et al., "Functional Dystonia," 138; and Espay et al., "Functional Tremor," 180–81.

77 Espay et al., "Functional Tremor," 180. For a detailed discussion of methodological and interpretative challenges entailed in using both the standardised emotional facial stimuli and the IAPS images in neuroimaging research into hysteria, see Muhr, *From Photography to fMRI*, section 4.3.2.

78 Szaflarski et al., "Facial Emotion Processing," 195.

subjects in an experimental task, the neuroimaging data is acquired while they are simply resting in the scanner and doing nothing.[79] The study's authors then applied different types of statistical analyses to the resting-state fMRI data to identify how the so-called intrinsic brain connectivity patterns—i.e., spontaneous interactions among various cerebral regions while the subjects are not engaged in any external task—differ across the two groups of seizure patients and healthy subjects.[80]

Unsurprisingly, since they focused on different hysterical symptoms, used different neuroimaging technologies (PET and fMRI), deployed different experimental paradigms (task-based and resting-state) and combined diverse affective and motor tasks, the four neuroimaging studies yielded very different results. But on the whole, each study found significant differences between hysteria patients and patients with similar organic symptoms at the neural level, either in terms of brain activation patterns or resting-state connectivity patterns.[81] Hence, taken together, these studies delivered preliminary empirical evidence that hysterical symptoms have a "distinctive cortical and subcortical pathophysiology" that distinguishes them from similar symptoms of other organic diseases.[82] Moreover, despite their mutually disparate results, in all four studies, hysteria's putative "pathophysiological signature" incorporated brain regions known to play a role in the neural processing of emotions.[83]

In short, these findings indicated that, unlike their organic counterparts, hysterical symptoms are associated with disturbances of emotion regulation. The authors of all four studies pointed out that they could not distinguish whether the disturbances they identified were causally related to hysterical symptoms or represented "compensatory changes" induced by the symptoms.[84] But despite their limitations, the four studies analysed in this subsection provided new empirical insights into the neurophysiological specificity of hysteria/functional neurological disorder. Hence, these studies further heightened this disorder's status as an increasingly distinct scientific object in neuroimaging research.

Hysterical Symptoms Are Reinforced through Negative Emotional Experiences

Another significant recent development in the neuroimaging research into hysteria is the emergence of studies that explicitly examine how stressful experiences and

79 Szaflarski et al., 193.

80 Szaflarski et al., 196.

81 Espay et al., "Functional Dystonia," 142–44; Espay et al., "Functional Tremor," 185; Schrag et al., "Dystonia," 780; and Szaflarski et al., "Facial Emotion Processing," 203.

82 Schrag et al., "Dystonia," 771.

83 Schrag et al., 780.

84 Schrag et al., 779.

negative emotional situations modulate various hysterical symptoms.[85] Also in this context, different studies have adopted a variety of experimental approaches to testing if and how acute stressful experiences worsen hysterical symptoms. To gain an overview of these versatile approaches and the initial insights they have generated so far, we will take a look at four exemplary fMRI studies published between 2016 and 2021.

In two such studies, researchers used standardised affectively charged images from the aforementioned IAPS database. But in each study, the visual stimuli were embedded into a very different type of task. For example, in their 2016 study, Blakemore et al. focused on hysteria patients with mixed motor symptoms, including paralysis, dystonia and tremor.[86] While lying inside the scanner, both patients and healthy control subjects were asked to exert consistent pressure on a force-measuring device that was placed in their hands. While performing this action, the participants were exposed to either pleasant or unpleasant images from the IAPS.[87] The participants' brain activity and the force output they produced were synchronously measured.

The comparison of the thus obtained results revealed that in healthy subjects, regardless of the affective content of images, the force output decayed gradually over time. In patients, however, the expected decay of force was absent when they viewed aversive images.[88] Even more interestingly, the neuroimaging data revealed that while viewing aversive images, patients had heightened activity across multiple brain regions involved in the processing of emotional salience.[89] Drawing these findings together, Blakemore et al. concluded that hysteria patients exhibited "heightened processing of emotional salience" when exposed to stressors.[90] Blakemore et al. further conjectured that this dysfunctional processing of negative affects elicited non-volitional "defensive behaviour" and modulated the patients' motor responses, thus resulting in a temporary worsening of their hysterical symptoms in stressful situations.[91]

Similarly to Blakemore et al., Hassa et al. in their 2021 study also focused on hysteria patients with mixed symptoms, which, in addition to motor disturbances and paralysis, included diverse sensory problems such as numbness, pain and hearing loss.[92] In their fMRI experiment, Hassa et al. exposed hysteria patients and

85 See, e.g., Allendorfer et al., "Psychological Stress"; Balachandran et al., "Stress"; and Blakemore et al., "Aversive Stimuli."

86 Blakemore et al., "Aversive Stimuli," 230.

87 For a detailed description of the task, see Blakemore et al., 230–32.

88 Blakemore et al., 233.

89 Blakemore et al., 234–38.

90 Blakemore et al., 229.

91 Blakemore et al., 229.

92 Hassa et al., "Amygdala Activity," 3, 613156.

healthy participants to unpleasant and neutral IAPS images, asking both groups of subjects to engage in an emotion regulation strategy called cognitive reappraisal. Specifically, "subjects were instructed to directly find an explanation that could reduce the emotional impact of the negative image; for example, when seeing images presenting war or violence they suggested themselves that the scenes were not real but made-up movie scenes, that blood was not real but theater blood, etc."[93] The fMRI findings revealed that, despite the cognitive reappraisal, when compared to healthy subjects, hysteria patients responded to unpleasant stimuli with abnormally increased activation of the amygdala, a subcortical structure known to be involved in the processing of negative emotions. Based on these findings, Hassa et al. suggested that explicit emotion regulation strategies seem ineffective in hysteria patients since these strategies "primarily operate on higher neural processing levels that are different from the amygdala."[94] In short, according to the authors of this study, aversive stimuli automatically trigger exaggerated affective responses in hysteria patients, which these patients are unable to suppress through conscious cognitive effort.

Two other mutually related fMRI studies focused on a single hysterical symptom—non-epileptic seizures.[95] In fact, both the 2019 study by Allendorfer et al. and the 2020 study by Balachandran et al. were conducted by the same research group and, instead of visual affective stimuli, used the same mathematical task to induce an acute stress response in experimental subjects. The task consisted of a control condition and a stressful condition. In the control condition, the subjects performed a simple subtraction while receiving positive feedback. In the stressful condition, the subjects were not only confronted with a more demanding mathematical problem but also, irrespective of their actual performance, received pre-recorded negative feedback (e.g., "You are not responding quickly enough for your answers to be counted.").[96] Thus, the stress-inducing task was not only cognitively more difficult but also included an additional "component of social evaluative threat."[97]

Apart from fMRI data, Allendorfer et al. also measured physiological stress responses, such as heart rate changes and the level of cortisol and alpha-amylase in saliva, whereas Balachandran et al. assessed the subjects' accompanying anxiety and mood disturbances. Based on the analyses of their respective fMRI datasets, both Allendorfer et al. and Balachandran et al. concluded that patients with hysterical

93 Hassa et al., 3, 613156.

94 Hassa et al., 8, 613156.

95 Allendorfer et al., "Psychological Stress"; and Balachandran et al., "Stress."

96 Allendorfer et al., "Psychological Stress," 3, 101967.

97 Allendorfer et al., 3, 101967. In specialist terms, this particular task is called the Montreal Imaging Stress Task.

seizures exhibit decreased neural responses to stress, especially in the so-called limbic brain regions, such as the amygdala and the hippocampus.[98] Allendorfer et al. also found that the decreased neural responses in these emotion-processing cerebral regions correlated with patients' dampened heart rate responses to perceived stress.[99] Moreover, Balachandran et al. established that the reduced reactivity of the hippocampus was associated with the severity of the patients' accompanying anxiety and mood disturbances.[100]

It is worth noting that the findings of hysteria patients' dampened neural responses to stress in the latter two studies contradicted the two studies analysed previously, which found abnormally enhanced neural responses to aversive stimuli in hysteria patients' emotion-processing brain regions. Some of the discrepancies are probably due to the different symptoms these studies investigated and the different types of stimuli and tasks they deployed. However, despite the unresolved discrepancies in their respective findings, all four studies had one salient point in common. They all suggested that hysteria patients have dysfunctional neural processing of stressful experiences, which, in turn, modulates the intensity of their symptoms and interfere with their cognitive abilities. These preliminary insights have provided a new empirical indication of hysteria's potential neurophysiological specificity and thus introduced a fruitful direction for future research. In doing so, these studies have "yield[ed] ever more layers of hidden structure" of hysteria, thus further stabilising and enriching its status as a scientific object in neuroimaging.[101]

Hysteria May Be Associated with Microscopic Anatomical Brain Changes

Finally, I would like to draw attention to a new research direction that has started to emerge within the neuroimaging investigation of hysteria in the 2010s. This research direction is driven by a slowly but continually growing number of studies that utilise novel statistically-based quantitative methods of imaging brain anatomy.[102] Such studies have begun to challenge the long-held view that hysteria patients entirely lack any underlying neuroanatomical damage. Admittedly, the absence of any visually identifiable anatomical anomaly in standard structural MRI scans is still used to support the diagnosis of hysteria in clinical contexts.[103] Hence, it is not hysteria patients' preservation of normal brain structure at the macro level that is currently

98 Allendorfer et al., 8, 101967; and Balachandran et al., "Stress," 107.

99 Allendorfer et al., "Psychological Stress," 8, 101967.

100 Balachandran et al., "Stress," 117.

101 Daston, "Scientific Objects," 13.

102 For a succinct overview of such studies, see Bègue et al., "Structural Alterations." See also Perez et al., "Research Agenda," 7–8, 102623.

103 Bègue et al., "Structural Alterations," 2, 101798.

coming into question. Instead, studies using new structural imaging methods suggest that hysteria patients might exhibit microscopic structural anomalies that remain invisible in standard MRI scans.

Several structural neuroimaging methods have so far been deployed in studies that focused either on patients with mixed symptoms or patients with a single hysterical symptom, such as seizures or paralysis.[104] Some of these methods allow researchers to examine various changes in the grey matter architecture of the patients' brains. For example, a method called voxel-based morphometry (VBM) enables an automated voxel-wise comparison of standard structural brain scans from hysteria patients and healthy subjects. Such a comparison serves to identify differences in the local grey tissue density across various anatomical structures between these groups of subjects.[105] Using this method, several studies have reported that, relative to healthy subjects, hysteria patients have either decreased or increased volume of different cerebral structures, such as the amygdala, insula and parts of the motor cortex.[106] Other studies that used different surface-based morphometric analyses detected changes in the cortical thickness, surface areas and curvatures of multiple subcortical and cortical brain structures.[107] More recently, researchers have also started to deploy state-of-the-art computational methods such as diffusion tensor imagining (DTI) to study possible changes in the patients' structural connectivity by visualising white matter tracts that physically connect various brain regions.[108]

The preliminary findings of such structural neuroimaging studies are mutually inconsistent, lack replication and are challenging to interpret.[109] To begin with, the actual medical meaning of the reported microscopic structural brain changes is currently far from evident. What is also an open question is how such structural abnormalities relate to functional disturbances that fMRI studies of hysterical symptoms have identified. Moreover, it remains unclear if the microscopic structural abnormalities reported so far might be causally related to the development of hysterical symptoms or if, conversely, they arise as a consequence of the illness.[110]

Overall, it can thus be said that the emerging structural neuroimaging research into hysteria has, until now, created more questions than answers. Yet, crucially, this research strand has opened up the possibility that hysteria's underlying pathophysiology may not be limited to a purely functional brain disturbance but could also entail associated microstructural anomalies. In doing so, the preliminary structural

104 See Bègue et al., "Structural Alterations," 4–10, 101798.
105 Perez et al., "Research Agenda," 8, 102623.
106 Bègue et al., "Structural Alterations," 3, 11–12, 101798.
107 Bègue et al., 12, 101798.
108 Bègue et al., 12–13, 101798.
109 Bègue et al., 14–15, 101798.
110 Bègue et al., 1, 101798.

neuroimaging findings have further strengthened the solidity of hysteria's status as a brain-based disorder. At the moment, it seems likely that the line of enquiry, which combines fMRI with structural imaging method to jointly search for hysteria's underlying structural and functional brain anomalies, will gain momentum in the coming years.

Thus, drawing things together, I argue that the various strands of neuroimaging research into hysteria discussed in this section testify to the increasing complexity and the continual intensification of the scientific endeavour to delineate the neurophysiological reality of the elusive hysterical symptoms. The novel neuroimaging technologies have enabled the exploration of connections between the patients' externally observable symptoms and their essentially invisible functional and structural brain pathologies, which had hitherto evaded scientists. Using these imaging technologies, present-day researchers have been able to generate "results, implications, surprises, connections manipulations, [and] explanations" that have opened up new epistemic perspectives of hysteria, thus heightening its "ontological status" as a genuine neurophysiological disorder and not a medically unexplainable phenomenon.[111]

Conclusion

To conclude, I have shown in this chapter that, a decade and a half after the American neurologist Mark Hallett designated it as a crisis for neurology, hysteria, now referred to as functional neurological disorder, has stabilised into an increasingly clearly defined scientific object of sustained neuroimaging research. As my analysis has demonstrated, this process has been facilitated by the insights into hysteria's underlying pathophysiology generated by multiple neuroimaging studies. These studies have revealed that hysteria is distinct from intentional simulation and organic disorders with similar symptomatology, that the clinically observable intensification of hysterical symptoms in stressful situations has a neural basis, and that, in addition to functional brain disturbance, the symptoms may be underpinned by subtle anatomical brain anomalies. Currently, these insights are preliminary, highly fragmentary and far removed from any clinical application. Yet, they have transformed hysteria from a collection of elusive symptoms loosely defined as medically unexplained into what is now perceived as a genuine brain-based disorder. Moreover, I have argued that in the process, these heretofore ignored symptoms have gradually coalesced into a scientific object in its own right, which is now "solid, capacious, ordered, intricate and deep enough to sustain research and theoretical explication."[112]

111 Daston, "Scientific Objects," 10.

112 Daston, 7.

However, as Mark Hallett admitted in the follow-up article published in 2019, the crisis he had initially declared in 2006 has not yet been resolved. Much remains unknown about hysteria's functional and structural neuropathophysiology since, to quote Hallett, "the brain is complicated."[113] Moreover, as my analysis has pointed out, none of the neuroimaging methods used in the research provides a transparent window into the patients' brains. Instead, using these methods is associated with a host of methodological challenges, which, as we have seen, often lead to mutual discrepancies across the studies. As detailed in this chapter, reconciling such discrepancies is far from straightforward. But gradually, researchers are becoming increasingly better at dealing with and, in part, even resolving some of the methodological challenges entailed in the neuroimaging research on hysteria. This might be why Hallett has predicted that, in another ten years, "the crisis will be over."[114]

I am not a medical expert. But to me, Hallett's prediction seems somewhat overoptimistic. Despite the intensifying neuroimaging research into hysteria, what has emerged so far is a series of more or less isolated and, to some extent, mutually inconsistent insights that have failed to be synthesised into an overarching, unifying framework. For example, what currently eludes researchers is how and under which conditions various functional and structural brain anomalies they have discovered so far mutually interact to give rise to a particular type or a particular combination of co-occurring hysterical symptoms. Moreover, it is unclear whether and to what extent adverse life experiences might contribute to the development or exacerbation of hysterical symptoms and if such environmental factors dynamically interact with other contributing factors, such as genetic predisposition and personal traits.

In line with Hallett, I too think that the medical insights into hysteria will "thicken and quicken with [the continued] inquiry."[115] However, as centuries of its winding medical history have shown, hysteria is a difficult nut to crack due to its highly heterogeneous manifestations, which might not even all have the same underlying neurophysiological mechanisms. Moreover, as even Charcot argued more than a century earlier, it is conceivable that various individual differences across patients, such as their psychological and physical makeup, personal habits and sociocultural conditions, additionally shape hysterical symptoms thus contributing to their variability.[116] And none of these accompanying factors can be readily studied through neuroimaging alone. Therefore, it appears to me that solving hysteria's mystery by identifying its presumed underlying neuropathophysiology, should it occur, will take longer than another decade. And even if the crisis of knowledge is solved one day, this would not immediately alleviate the crisis of action. Translating

113 Hallett, "Crisis Resolved," 973.

114 Hallett, 973.

115 Daston, "Scientific Objects," 13.

116 Charcot, *Diseases of the Nervous System*, 400.

the insights won through medical research into actual clinical practice by developing novel diagnostic and treatment approaches that, in turn, can effectively resolve the lingering crisis of action would surely take much longer.

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