definite diagnosis.[390] This, in turn, has posed additional difficulties for estimating with sufficient accuracy the actual incidence of hysterical symptoms in the current clinical settings. Nevertheless, even according to the lowest estimates in contemporary epidemiological studies, present-day manifestations of hysteria seem to be no less frequent than schizophrenia.[391] Unlike schizophrenia, until very recently, not only did hysteria merit hardly any clinical interest, but it also ceased to be the topic of any systematic scientific research.[392]

However, in the remainder of this chapter, we will see that this situation gradually began to change by the beginning of the twenty-first century. Furthermore, I will show that, in a remarkable parallel to Charcot's image-based research, the present-day resurgence of scientific interest in hysteria turned out to be closely related to the implementation of cutting-edge imaging technologies. And as will become apparent by the end of my enquiry, these new imaging technologies deliver images that are very different from the ones with which Charcot worked in the framework of his hysteria research.

2.3 The Reappearance of Image-Based Hysteria Research

Somewhat paradoxically, precisely when multiple humanities scholars emphatically declared hysteria to be a no longer existing medical phenomenon,[393] three contemporary scientific studies of this elusive disorder appeared. The studies by Tiihonen et al., Yazici and Kostakoglu, and Marshall et al. were all published in the closing decade of the twentieth century.[394] They had several features in common. First, they all investigated medically unexplained somatic symptoms. For the most part, all three studies focused on limb paralysis, which, in line with the *DSM* criteria that were valid at the time, was diagnostically attributed to conversion disorder.[395] Second, in addition to the official *DSM* label, the authors of all three studies explicitly

390 See, e.g., Agaki and House, "Epidemiology," 84; and Nimnuan, Hotopf, and Wessely, "Epidemiological Study," 366.

391 Agaki and House, "Epidemiology," 83. Schizophrenia is a neurodegenerative disorder that belongs to the psychotic spectrum. Patients suffer from hallucinations, delusions, flat affects, disorganised behaviour, and cognitive impairments, thus often having problems recognising what is real. APA, *DSM-IV*, 273–78.

392 Stone et al., "Disappearance," 13.

393 Bronfen, *Knotted Subject*, xi; Micale, *Approaching Hysteria*, 29; Micale, "Disappearance," 498; Shorter, *From Paralysis to Fatigue*, 196–200, 267–73; and Showalter, *Hystories*, 15.

394 See Tiihonen et al., "Hysterical Paraesthesia"; Yazici and Kostakoglu, "Cerebral Blood Flow"; and Marshall et al., "Hysterical Paralysis."

395 In the Tiihonen et al. study, a single patient had one-sided paralysis accompanied by anaesthesia. The Yazici and Kostakoglu study was conducted on five patients whose diverse somatic symptoms included paralysis, speech loss, and gait disturbances. For details, see Yazici and Kostakoglu, "Cerebral Blood Flow," 164–66. The single patient in the Marshall et al. study manifested a chronic one-sided paralysis that had lasted for two and a half years.

designated the paralysis as 'hysterical' in the main text of their articles.[396] Moreover, two of these studies also used the term 'hysterical' in their respective titles.[397] Finally, and most significantly, these three studies were the first to use functional brain imaging technologies to study a hysterical symptom of interest. Essentially, these three studies pioneered the application of functional brain imaging in the medical investigation of hysteria.

In short, at the very height of hysteria's medical invisibility, several neurologists and psychologists suddenly declared hysterical paralysis a topic worthy of scientific enquiry and chose to use cutting-edge neuroimaging tools to investigate it. However, apart from their undeniable landmark character, in what follows, I will argue that what was no less remarkable about these three studies is how much they lagged behind comparable functional neuroimaging research into other mental disorders. Specifically, I will contend that although the availability of the new imaging modalities was a necessary precondition for hysteria to become once again an object of image-based medical research, it was in itself not sufficient. Instead, I will show that a prior shift in the conceptualisation of hysteria was indispensable to make the functional imaging technologies applicable to studying this medically unexplained disorder. Having shown this, I will then trace the trajectory through which what at first might have seemed like a random compilation of sporadic functional neuroimaging studies gradually coalesced into a distinct area of contemporary hysteria research. But before we turn to addressing the conceptual shifts that, as I will claim, enabled the appearance of contemporary image-based hysteria research, it is necessary to make a short detour. We first need to discuss in more general terms the epistemic possibilities and ramifications that the advent of new neuroimaging technologies in the last third of the twentieth century has brought.

2.3.1 The Advent of New Brain-Based Investigation Tools

Starting in the 1970s, the gradual advent of neuroimaging technologies has enabled new ways of measuring and visualising various static (i.e., anatomical) and dynamic (i.e., functional) features of the living brain. At first, these technologies included computed tomography (CT), magnetic resonance imaging (MRI), single-photon emission tomography (SPECT), and positron emission tomography (PET).[398] Additionally, by the early 1990s, functional magnetic resonance imaging (fMRI) was developed.[399] Both CT and MRI provide detailed spatial information about brain anatomy.[400] Conversely,

396 Tiihonen et al., "Hysterical Paraesthesia," 134; Yazici and Kostakoglu, "Cerebral Blood Flow," 163, 165, 166; and Marshall et al., "Hysterical Paralysis," B1, B2, B6.

397 Tiihonen et al., "Hysterical Paraesthesia"; and Marshall et al., "Hysterical Paralysis."

398 For a detailed overview of these imaging technologies and their early application in psychiatry, see, e.g., Andreasen, *Brain Imaging*.

399 For a short history of fMRI, see, e.g., Huettel, Song, and McCarthy, *Imaging*, 15–24.

400 Andreasen, *Brain Imaging*, x.

PET, SPECT, and fMRI generate indirect measurements of neural activity, thus allowing researchers to make inferences about how the human brain works.[401]

Importantly, the common feature of all these technologies is that they produce digital data in the form of two-dimensional (2D) slices from which a three-dimensional (3D) visualisation of the brain can be rendered. Since these technologies provide information about the brain's structure and function in distinctly spatial terms, their advent has given rise to scientific studies that focus on functional localisation.[402] The underlying premise of functional localisation is that the activity of distinct parts of the cerebral cortex supports particular mental processes.[403] This premise informs cognitive neuroscience, a research field that, since the 1970s, investigates "how the human brain creates the human mind."[404] Similarly, it is with the aim of relating symptoms of mental illnesses to anatomically localisable disturbances of normal brain functions that neuroimaging has found application within psychiatry.[405]

Functional localisation, however, is not a new idea. In the previous chapter, we discussed how, more than a century before the arrival of neuroimaging technologies, Charcot performed brain lesion studies that were already informed by a comparable principle.[406] We saw that within the framework of his anatomo-clinical method, he aimed to correlate distinct clinical signs of a neurological disorder, which he had observed during a patient's lifetime, with localised damage to the brain tissue discovered through autopsy. Moreover, I have argued that both Charcot's postmortem studies of patients suffering from various organic diseases and his image-based hysteria research were informed by the nineteenth-century paradigm of cerebral localisation.[407] The formal birth of this paradigm was linked to the famous discovery made by Charcot's contemporary, the French surgeon Paul Broca.[408]

In 1861, by performing a brain autopsy of a patient who had lost the ability to speak, Broca detected a circumscribed structural lesion in the left frontal lobe.[409] Drawing on this empirical finding, Broca deduced that this particular brain region was involved in speech production. In subsequent years, Broca repeated this procedure with additional patients who had suffered from speech loss. Through repeated autopsy results that overlapped with his initial finding, he thus corroborated the claim that speech production was localised in a specific brain area, which now carries Broca's

401 See, e.g., Bear, Connors, and Paradiso, *Exploring the Brain*, 173–75; and Mayberg, "Neuroimaging and Psychiatry," S31–32.

402 Raichle, "Historical and Physiological Perspective," 4.

403 See, e.g., Huettel, Song, and McCarthy, *Imaging*, 1.

404 Gazzaniga, Doron, and Funk, "Perspectives on the Human Brain," 1247.

405 Andreasen, *Brain Imaging*, ix–x.

406 Goetz, Bonduelle, and Gelfand, *Charcot*, 75–78.

407 As discussed in detail in chapter 1, in his image-based hysteria research, Charcot indirectly made inferences about the underlying functional disturbances of his patients' brains by systematically measuring and visualising derangements of their various physiological functions.

408 Finger, *Minds Behind the Brain*, 143. For a short overview of how Charcot's localisationist studies intersected with Broca's research, see Goetz, Bonduelle, and Gelfand, *Charcot*, 127–34.

409 Finger, *Minds Behind the Brain*, 137–44.

name.[410] However, despite the initial successes of this method, it soon became apparent that lesions studies were too coarse to allow mapping of more complex cognitive functions and mental disorders to brain systems.[411] Among others, the inherent limitations of postmortem lesion studies include "artifactual effects of the death process, the necessity to study predominantly elderly individuals, and a scarcity of informative samples of brain tissue." [412]

By surpassing many limitations inherent to the nineteenth-century lesion studies, neuroimaging technologies have opened up new possibilities of functional localisation.[413] For instance, one of the key advantages of structural neuroimaging technologies is that they enable neurologists to detect not only permanent lesions but also more transitory tissue abnormalities without any need for a physical intrusion into the brain.[414] In other words, although they facilitate the establishment of putative links between changes in the static neural architecture and mental deficits in a manner similar to the nineteenth-century localisation paradigm, the crucial difference is that the new imaging technologies allow the examinations of living patients.[415]

Additionally, unlike lesion studies, neither structural nor functional neuroimaging is limited to investigating pathological cases. For example, one particularly widely publicised MRI-based study established a connection between the superior spatial navigation abilities of London taxi drivers and the increase in the size of a specific brain structure called the hippocampus.[416] Thus, for the first time in history, the advent of neuroimaging has made possible localisation studies of cerebral functions in healthy human brains.[417] In doing so, these imaging technologies have provided researchers

410 Finger, 144–45.

411 See Price and Friston, "Neuropsychologically Impaired Patients," 380–81.

412 Andreasen, *Brain Imaging*, ix.

413 Less flatteringly, neuroimaging has also been compared to the pseudoscientific practice of phrenology, which was developed in the late eighteenth century by Franz Joseph Gall and became popular in the early nineteenth century. Gall contended that the size and the shape of a person's skull matched the size and the shape of the person's brain and that various areas of the brain were specialised for performing particular mental functions. He further contended that the larger a particular brain area was, the more developed was the mental function this area controlled. He thus argued that based on the bumps and indentations of an individual's skull, it was possible to make inferences about that person's mental faculties. By the 1820s, Gall's views had been discredited and shunned as pseudoscience. For details on phrenology, see Finger, *Minds Behind the Brain*, 119–36. For accounts that have compared neuroimaging to phrenology, see, e.g., Uttal, *New Phrenology*; Hagner, "Das Hirnbild als Marke"; and Hagner, "Das Genie und sein Gehirn," 204–7. In fact, Michael Hagner has introduced the term 'cyber-phrenology' to designate the localisationist orientation of neuroimaging. See Hagner, "Das Hirnbild als Marke," 45; and Hagner, "Das Genie und sein Gehirn," 206.

414 Mayberg, "Neuroimaging and Psychiatry," S31.

415 See, e.g., Walterfang et al., "White Matter Volume Changes," 210–15.

416 See Maguire et al., "Hippocampi of Taxi Drivers," 4398–403.

417 Strictly speaking, non-invasive investigation of brain function was already feasible in the late 1920s, owing to the invention of the method called electroencephalography (EEG). EEG measures the electrical activity of neurons using electrodes placed on the surface of the subject's head. Yet, unlike PET and fMRI, EEG has a very low spatial resolution, which does not allow precise localisation of the measured neural activity to a specific brain region. Therefore, it cannot be used

with an incomparably more flexible approach to investigating functional anatomy than lesion studies. As a result, present-day researchers no longer have to focus on ascribing function to a particular area that had been damaged by disease or injury but can choose which brain regions to investigate. Moreover, the functional neuroimaging technologies have opened up the until that point unthinkable possibility of studying abnormal brain function even in the absence of any detectable anatomical brain damage. This possibility, as we will see later, has proved crucial for the resurgence of image-based hysteria research.

Another particularly significant advantage of functional neuroimaging is that it offers considerably more fine-grained insights into the workings of the living brain than the methods Charcot had at his disposal. Specifically, functional neuroimaging is not limited to linking a specific function to a single brain region. Instead, it enables researchers to relate a particular cognitive process to a complex, spatially distributed pattern of neural activity.[418] Called functional networks, such distributed patterns of neural activity are understood to result from dynamic interactions and functional relations among different, spatially distinct parts of the brain.[419] This integrative approach to investigating brain function has gained increasing significance since the mid-1990s with the introduction of new analytical methods of functional connectivity. These methods permit scientists to explore "the way in which brain regions communicate with one another and [how] the information is passed from one brain area to the next."[420]

Hence, it can be said that instead of merely enforcing a simplified and reductive one-to-one mapping of mental function to strictly dedicated anatomical regions, functional neuroimaging research creates a far more complex picture of the human brain as a highly interconnected and dynamic system. According to the emerging insights, on the one hand, multiple brain regions can be active simultaneously to jointly support a particular cognitive process.[421] On the other hand, each anatomical structure can participate in different cognitive functions. The complexities of such mapping will become apparent in the subsequent chapters when we move to an in-depth analysis of individual functional neuroimaging studies in the context of present-day hysteria research.

However, it should also be emphasised that in neuroimaging, the activity of a particular brain region during the performance of a particular cognitive function is defined in purely biological terms. Specifically, the underlying brain activity is understood to comprise a potentially detectable and quantifiable set of mutually related physical changes in neural chemistry, physiology, and metabolism.[422] In fact, different functional neuroimaging technologies measure various aspects of brain

for unambiguously associating a particular brain structure with a function. See Baars and Gage, *Cognition, Brain and Consciousness*, 101–6.

418 See, e.g., Poldrack, Mumford, and Nichols, *Handbook*, 130.

419 Huettel, Song, and McCarthy, *Imaging*, 4.

420 Bijsterbosch, Smith, and Beckmann, *Resting State*, 2.

421 Huettel, Song, and McCarthy, *Imaging*, 4.

422 Huettel, Song, and McCarthy, 113–15.

metabolism and neurophysiology as a proxy for neural activity.[423] In turn, the cognitive processes associated with such indirectly measured brain activity are also framed in distinctly neurobiological terms. Simply put, although functional imaging technologies are used for investigating the human mind, there "is no getting away from the fact that these are brain-based tools."[424] This also means that the extent to which different neuroimaging technologies can provide potential insights into normal cognitive functions—and cognitive dysfunctions entailed in various psychiatric disorders—is necessarily constrained by the precision and accuracy with which they can measure and visualise the underlying neurophysiological processes. Hence, to be able to make informed judgments about the findings generated through neuroimaging, it is necessary to understand what a particular technology measures, how, and with which constraints. For this reason, my analysis in the subsequent chapters will pay particular attention to these aspects.

Methodologically, another crucial aspect is that functional neuroimaging can only establish a correlation—and not an actual causal relation—between the localised neurophysiological changes measured and a particular cognitive event.[425] This has significant epistemic consequences for the interpretation of visual findings obtained in the context of functional neuroimaging. First, the mere co-occurrence of the indirectly measured spatially distributed neural activity and the specific cognitive process does not prove that each brain region designated as active is necessary for executing that particular cognitive process.[426] Instead, multiple anatomical areas may be coactive without serving the same function. Second, it cannot be claimed that the local pattern of neural activity identified through neuroimaging is sufficient for performing the cognitive function of interest. This is because some regions that participate in that cognitive function may nevertheless have remained unregistered by the imaging technology at hand.[427]

In short, based on a functional imaging study alone, a specific pattern of neural activity cannot be unambiguously associated with a cognitive function or dysfunction under investigation.[428] Hence, to acquire an evidentiary status, any inference about the neural underpinning of a specific cognitive process derived from functional neuroimaging must be semantically contextualised. This is typically achieved by embedding the neuroimaging findings into a broader theoretical framework or by combining them with converging experimental results obtained through other technologies and alternative research methods.[429] In other words, the interpretation

423 For details, see, e.g., Raichle, "Historical and Physiological Perspective," 7, 11.

424 Savoy, "History and Future Directions," 35.

425 Welshon, *Philosophy, Neuroscience and Consciousness*, 197. Correlation is a statistically based measurement of dependence between two variables. If two variables are correlated, they co-vary. Importantly, however, a high correlation between two variables does not suffice to establish a causal relation between them, as any co-variation may be purely coincidental. Ibid, 221–22.

426 Huettel, Song, and McCarthy, *Imaging*, 366.

427 Welshon, *Philosophy, Neuroscience and Consciousness*, 197–204.

428 Welshon, 196. For a detailed discussion of these issues, see Kurthen, "Pushing Brains," 5–22.

429 Bechtel and Stufflebeam, "Procuring Evidence," 72.

of functional neuroimaging results is challenging and far from straightforward, and all insights thus obtained are highly mediated.[430]

As a result, the mapping of cognitive processes onto distinct anatomical areas of the brain by means of functional neuroimaging has historically progressed in a series of consecutive stages. In the early days, each imaging technology was first used to reproduce the functional localisations that had already been established through lesion and animal studies.[431] After such a preliminary period of methodological validation, the investigation of functional neuroanatomy in healthy subjects followed.[432] The research into normal cognitive processes, in turn, provided the necessary semantic basis for subsequent neuroimaging studies of pathophysiology in patients with different organic deficits.[433] Finally, it was only in the next stage that functional neuroimaging started to be applied to the search for the potential neurobiological basis of various psychiatric disorders.[434] However, for reasons we will discuss in the following section, hysteria's nosological successors at first remained excluded from this process.

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So far, I have sketched the general epistemic ramifications that arose from the advent of functional neuroimaging. In particular, I have foregrounded the entirely new empirical approaches to investigating the human mind that the novel neuroimaging technologies have opened up. But I have also indicated some of the technologies' limitations and emphasised the purely brain-based, neurophysiological framing of mental and cognitive processes that neuroimaging entails. Drawing on these insights, we can now turn to analysing the gradual process through which, as I will argue, the neuroimaging technologies first indirectly enabled the reappearance of image-based hysteria research, whose integral part they then became.

2.3.2 A Winding Road Towards the First Functional Neuroimaging Study of Hysteria

By the early twenty-first century, functional neuroimaging would be celebrated for delivering crucial new insights into an array of psychiatric disorders.[435] However, in the 1970s and the early 1980s, the applicability of neuroimaging technologies in this area of research was not yet a given. At that time, psychiatry was still dominated by psychogenic models of mental illnesses.[436] As my analysis in this section will show, the potential epistemic utility of the neuroimaging technologies, as brain-based research tools that generate only inferential knowledge about psychological states, first had to

430 In chapter 3, we will see that this has consequences both on how neuroimaging experiments are conceived and on how the detected patterns of brain activity are interpreted.

431 Farah, "Brain Images, Babies, and Bathwater," S22.

432 Price and Friston, "Neuropsychological Patients," 345.

433 Price and Friston, 345.

434 See, e.g., Ingvar and Franzén, "Abnormalities of Cerebral Blood Flow."

435 See, e.g., Andreasen, "Linking Mind and Brain."

436 See, e.g., APA, *DSM-II*.

be established. Moreover, the use of functional neuroimaging was not just expensive and time-consuming, but in the case of SPECT and PET, it also entailed the patients' exposure to radiation.[437] Thus, as we are about to see, neuroimaging technologies were at first applied only selectively to those psychiatric disorders for which sufficient assumptions existed about their potential neurobiological basis. I will argue that this was why the pioneering functional neuroimaging study of hysteria lagged decades behind comparable studies of other psychiatric disorders.

The gradual revival of biological psychiatry was initiated in the 1950s with the development of the first antipsychotic and antidepressant drugs that focused on treating mental illnesses by causing changes in brain chemistry.[438] This development received further impetus from growing molecular biologic research into the genetic underpinnings of mental disorders since the 1970s.[439] Yet, during the 1960s and 1970s, the increasing re-biologisation of psychiatry was challenged by the antipsychiatry movement. Representatives of this movement claimed that mental disorders lacked any biological basis and should instead be viewed as purely socially constructed and even in part invented categories.[440]

A particularly vocal representative of this movement was the Hungarian-American psychiatrist Thomas Szasz. Szasz famously declared that, unlike a 'genuine' disease, which was characterised by "a physicochemical state of the bodily disorder," mental illness was merely a metaphor used for labelling human suffering.[441] To make his point, Szasz focused in particular on deconstructing hysteria, which he considered the paradigmatic example of an invented illness. In his influential book *The Myth of Mental Illness*, he redefined hysteria as a type of "pantomime," a form of non-discursive communication that deployed body signs.[442] He further argued that because hysteria was a sign-using behaviour, or "an idiom rather than an illness, it was senseless to inquire into its 'causes.'"[443] In short, according to Szasz, hysteria had no biological basis whatsoever. Szasz's criticism of hysteria fell on fertile ground, reinforcing at the time already influential views on this disorder's non-existence.[444]

Contrary to hysteria, somatic approaches to other psychiatric illnesses—particularly schizophrenia—continued to gain growing acceptance. Admittedly, in the early 1970s, there was still no empirical proof of any underlying anatomical or biochemical abnormalities in the brains of patients diagnosed with schizophrenia.[445] Nevertheless, multiple studies that clearly demonstrated the efficacy of antipsychotic drugs in treating schizophrenia, in turn, indicated that this disorder could have a potential

437 Price and Friston, "Neuropsychological Patients," 351.

438 For a detailed description of the birth of psychopharmacology and its influence on the re-biologisation of psychiatry, see Shorter, *History of Psychiatry*, 246–62.

439 Shorter, 240–46.

440 Shorter, 273–77.

441 See Szasz, *Myth of Mental Illness*, 40–41.

442 Szasz, 229. For details, see ibid., 107–47.

443 Szasz, 146.

444 See section 2.2.2 for a discussion of Eliot Slater's dismissal of hysteria as a mere myth.

445 Ingvar and Franzén, "Abnormalities of Cerebral Blood Flow," 426.

neurobiological basis that was worth investigating.[446] Accordingly, the first functional neuroimaging study involving schizophrenia patients was conducted as early as 1974.[447] In this pioneering study, Ingvar and Franzén used a precursor to SPECT to investigate potential changes in the brain function in twenty chronic schizophrenia patients who showed advanced cognitive deterioration.[448] The resulting images disclosed an abnormal reduction of the regional blood flow in the patients' frontal brain areas.[449] Ingvar and Franzén attributed this aberrant blood flow pattern to a pathological reduction of the associated brain activity in these areas. Moreover, they suggested that the patients' abnormally low level of activity in the frontal lobe might constitute the "functional disturbance underlying schizophrenia."[450] Two years later, a study by Johnstone et al. used CT scans to examine potential anatomical abnormalities in chronic schizophrenia patients.[451] This study reported a significant enlargement of patients' lateral brain cavities (i.e., ventricles), thus delivering the first image-based finding of macroscopic structural cerebral changes in schizophrenia.[452]

Due to the success of these initial studies and the rising popularity of SPECT and PET as research tools, both functional and structural neuroimaging of schizophrenia intensified in the following decades.[453] This trend was additionally amplified by the subsequent advent of fMRI in the early 1990s.[454] As a result, image-based findings of multiple structural and functional brain abnormalities associated with schizophrenia accumulated over the subsequent years. And although a clear-cut neurological basis of schizophrenia has so far remained elusive, the intensity of the neuroimaging research into this disorder has never abated.[455] Furthermore, during the 1980s, almost all psychiatric disorders underwent a process of re-biologisation similar to schizophrenia and, in turn, became objects of sustained neuroimaging research.[456] Hysteria, however, was not among them.

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446 For an overview of studies conducted in the 1960s on the efficacy of antipsychotics in treating schizophrenia, see Lopez-Munos et al., "Clinical Introduction of Chlorpromazine," 128–29.

447 Ingvar and Franzén, "Abnormalities of Cerebral Blood Flow." The study measured regional cerebral blood flow by using a radiotracer Xe-133. For details on this technology, see Devous, "Imaging Brain Function," 195.

448 Ingvar and Franzén, "Abnormalities of Cerebral Blood Flow," 425.

449 Ingvar and Franzén, 425.

450 Ingvar and Franzén, "Distribution of Cerebral Activity," 1485.

451 Johnstone et al., "Cerebral Ventricular Size."

452 Johnstone et al., 924.

453 For an overview of these studies, see, e.g., Blakemore, "Schizophrenia and Brain Imaging," 650–59; Coffman, "Computer Tomography," 17–45; Devous, "Imaging Brain Function," 195–204; Gur and Gur, "Imaging in Schizophrenia"; Holcomb et al., "Positron Emission Tomography," 321–30, 339–42.

454 Gur and Gur, "Imaging in Schizophrenia," 333–34.

455 For details, see, e.g., Birur et al., "Brain Structure, Function and Neurochemistry"; and Blakemore, "Schizophrenia and Brain Imaging."

456 These disorders included depression, autism, Alzheimer's disease, obsessive-compulsive disorders and anxiety. For details, see Holcomb et al., "Positron Emission Tomography," 330–38. For a lucid sociological study of how, despite decades of intensive neuroimaging research, straightforward biological causes of autism still remain out of reach, see Fitzgerald, *Tracing Autism*.

Importantly, the initial neurobiological redefinition of schizophrenia and other psychiatric disorders was facilitated not only through early pharmacological and genetic research but also through systematic neurophysiological and biochemical studies.[457] By contrast, hysteria remained excluded from all aspects of this process. As discussed previously, due to the influence of Freud's legacy, hysteria was initially regarded as the quintessential psychogenic disorder and hence remained embedded in the psychoanalytic framework longer than other mental illnesses.[458] Unsurprisingly, as long as hysteria was regarded as a direct product of idiosyncratic life experiences, it made little sense to search for its potential biological basis. And even as Freud's influence started to wane in the second half of the twentieth century, no other generally accepted interpretational model of hysteria emerged.[459]

In the period between the 1950s and 1980s, only a few sporadic neuropsychological and EEG-based neurophysiological studies of hysterical symptoms were conducted.[460] At first, some promise appeared to emerge from studies of so-called somatosensory evoked potentials that implemented scalp electrodes to register the brain's electrical activity in response to sensory stimulation of the skin.[461] A couple of early studies reported abnormal potentials in patients with hysterical anaesthesia, thus suggesting possible underlying neuropathology.[462] But the initial findings were soon contradicted by several subsequent studies, all of which registered normal evoked potentials from different neural domains in hysteria patients.[463] The latter findings were interpreted as evidence of intact early motor and sensory cerebral processing. This interpretation, in turn, further reinforced the already prevalent view that hysteria lacked a neurological basis. Such measurements of normal potentials were even accorded diagnostic value concerning hysteria, with some neurologists using them to "rule out any structural abnormality."[464] Characterised by the absence of detectable physiological or anatomical neuropathology,[465] and still somewhat vaguely linked to psychological factors, hysteria thus appeared to be doubly detached from the body. In such a context, it seems hardly surprising that the implementation of functional imaging, as a set of at the time still novel and, therefore, not universally applicable brain-based tools, was not deemed feasible for investigating hysteria.

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457 See Blakemore, "Schizophrenia and Brain Imaging," 649; and Devous, "Imaging Brain Function," 190.

458 See section 2.2.1.

459 See APA, *DSM-III*, 241.

460 For summaries of sparse neurological research from this period, see Sierra and Berrios, "Hysteria," 193–94; Trimble, *Biological Psychiatry*, 195; and Yazici and Kostakoglu, "Cerebral Blood Flow," 166–67.

461 "Somatosensory evoked potentials are a simple, noninvasive means by which the physician may evaluate the integrity of the central sensory pathways from the peripheral nerve through to the cerebral cortex." Kaplan, Friedman, and Gravenstein, "Somatosensory Evoked Potentials," 504–5.

462 For the initial study, see Hernandez-Peón, Chávez-Ibarra, and Aguilar-Figueroa, "Case of Hysterical Anaesthesia." For an overview of subsequent studies, see Sierra and Berrios, "Hysteria," 192.

463 Hallett, "Neurophysiologic Studies," 63; and Sierra and Berrios, "Hysteria," 192–93.

464 Kaplan, Friedman, and Gravenstein, "Somatosensory Evoked Potentials," 502. See also Yazici and Kostakoglu, "Cerebral Blood Flow," 167.

465 See APA, *DSM-III*, 241.

However, by the 1990s, the organicist approaches to mental functions and dysfunctions became part of the mainstream scientific practice.[466] Twenty years of converging research appeared to lend increasing support to the stance that all mental processes were associated with potentially measurable brain activity.[467] This, in turn, led to an all-embracing implementation of functional neuroimaging, at the forefront of which was the novel fMRI technology.[468] Through the intensifying neuroscientific research, the majority of higher mental functions thus came to be interpreted in terms of underlying neurophysiological correlates of either structural or functional kind.[469] These functions included attention, sensory processing, inhibition, executive control, and volition, to name a few. Moreover, in this context, mental disorders came to be regarded as "distortions of normal brain functions or loss of such functions."[470] The *DSM-IV*, published in 1994, announced its adherence to the organicist approach to mental disorders in no uncertain terms. Its authors stated that "the term mental disorder unfortunately implies a distinction between 'mental' disorders and 'physical' disorders that is a reductionist anachronism of mind/body dualism. A compelling literature documents that there is much 'physical' in 'mental' disorders and much 'mental' in 'physical' disorders."[471]

This new viewpoint, so I suggest, had direct implications on how the *DSM-IV* redefined the nosological successors of hysteria. Admittedly, the manual, by and large, retained the general subdivision and terminology the previous edition had introduced.[472] Yet, the *DSM-IV* substantially refashioned the diagnostic criteria of somatoform disorders. First, the *DSM-IV* additionally attenuated the role of psychological factors in somatoform disorders by reducing it to a mere unspecified temporal association between a stressor and the initiation or exacerbation of the symptom.[473] Second, the *DSM-IV* explicitly banished the fundamental Freudian tenet that somatic symptoms were symbolic expressions of underlying psychological conflicts.[474] In effect, the individual patients' idiosyncratic traumatic life events were no longer deemed to determine the symptom semantically, as Freud had claimed. Thus, the loosely retained temporal link between a stressful life event and the initiation of illness appeared to have a purely incidental character and could no longer be used to explain why a patient developed a particular symptom.

466 See Goldstein, "Decade of the Brain," 239.

467 Goldstein, 239. For a more popular review of relevant studies, see, e.g., Damasio, "How the Brain Created the Mind."

468 See Cabeza and Nyberg, "Imaging Cognition 2," 1–47. See Huettel, Song, and McCarthy, *Imaging*, 419.

469 See Posner and Rothbart, "Neuronal Theories of Mind."

470 Andreasen, *Brain Imaging*, ix.

471 APA, *DSM-IV*, xxi. The current biological psychiatry, although prevalent, is by no means uncontested. For a critical analysis, see, e.g., Kirmayer and Gold, "Re-Socializing Psychiatry," 307–30.

472 Compare APA, *DSM-III*, 241–47; and APA, *DSM-IV*, 445–57.

473 APA, *DSM-IV*, 457.

474 APA, 454.

But even more importantly, the *DSM-IV* halted the thus far continual dematerialisation of hysteria's somatic symptoms we discussed in the previous sections. As already pointed out, the *DSM-III* explicitly required that hysterical symptoms could not "be explained by a known physical disorder or pathophysiological mechanism."[475] By contrast, the *DSM-IV* reformulated this diagnostic criterion, stating that somatic symptoms could not "after appropriate investigation, be fully explained by a known general medical condition, or by the direct effects of a substance, or as a culturally sanctioned behaviour or experience."[476] Thus, although still characterised in diagnostic terms by the absence of measurable organic damage, somatic manifestations of hysteria ceased to be defined through an explicit exclusion of potential physiological mechanisms.[477] This change in the formulation did not affect how hysteria's somatic symptoms were diagnosed. As already analysed in detail, doctors continued to struggle with diagnostic challenges in clinical practice. However, I contend that this subtle diagnostic redefinition of hysteria indicated a change of perspective from which this disorder was viewed in the research community.

We have seen that during the 1970s and 1980s, the lack of any detectable neurological anomaly was interpreted as 'objective' proof of what appeared to be hysteria's non-organic and non-physiological character. But by the mid-1990s, due to the broader shifts in the conceptualisation of mental diseases, a different interpretation became viable. In the new context, the lack of detectable anatomical neuropathology could now be taken to imply the presence of a potentially measurable disturbance of brain activity as a tenable cause of the puzzling somatic manifestations of hysteria. I argue that this semantic transcription was an essential prerequisite for the applicability of functional neuroimaging technologies as epistemic tools in the scientific investigation of hysteria.[478] Consequently, only in 1995 did the first functional neuroimaging study of a hysterical symptom appear.[479] In this pioneering study, Tiihonen et al.

475 APA, *DSM-III*, 247.

476 APA, *DSM-IV*, 457.

477 Admittedly, the *DSM-IV* also stated that conversion symptoms "typically do not conform to known anatomical pathways and physiological mechanisms, but instead follow the individual's conceptualisation of a condition." See APA, 453. Yet, this was a phenomenological description of the symptoms' clinical features and not a diagnostic criterion.

478 I am using the term transcription in Jäger's sense. See Jäger, "Transcriptivity Matters," 49.

479 Tiihonen et al., "Cerebral Blood Flow," 134–35. As of 1992, multiple SPECT studies appeared that focused on hysterical attacks, which in the current medical terminology are referred to as non-epileptic seizures. See, e.g. Price et al., "Non-Epileptic Seizure Disorder." My analysis will disregard these studies since they did not use SPECT to discover the possible neurobiological basis of this hysterical symptom. Instead, their explicit aim was to determine the potential diagnostic utility of SPECT in differentiating between non-epileptic and epileptic seizures. The starting premise of these studies was that a SPECT scan taken during a non-epileptic seizure should show a lack of any pathological brain activity, unlike a scan obtained during a genuine epileptic attack. The hysterical symptom was thus defined in purely negative terms—as the absence of a discernible abnormal pattern of cerebral blood flow associated with epilepsy. See, e.g., Varma et al., "SPECT in Non-Epileptic Seizures," 89–91. In other words, unlike Tiihonen et al., these studies did not operate under the assumption that hysterical symptoms were attributable to a detectable disturbance of brain activity. For an overview of these studies, see Neiman et al., "Utility of Ictal SPECT," 211–12.

set out to identify potential neurophysiological underpinnings of hysterical paralysis accompanied by anaesthesia in a female patient whose neurological "examination including computed tomography (CT) and electroencephalogram (EEG) was normal."[480]

Tiihonen et al. used SPECT to measure the regional cerebral blood flow in the patient while her paralysed hand was exposed to electrical sensory stimulation.[481] They then repeated the same measurement procedure six weeks later. By that point, the patient's symptoms had spontaneously disappeared. The comparison of these two measurements showed that, before her recovery, the patient had decreased neural activation in the somatosensory areas and increased activation in the frontal parts of her brain.[482] The abnormal pattern of neural activation was demonstrated by SPECT scans that visualised distinctly altered blood flow in these two areas of the patient's brain before but not after her recovery. Hence, with these images, the Tiihonen et al. study delivered the initial tangible indication that somatic symptoms of hysteria might be related to identifiable neurophysiological alterations in the brain.[483]

How exceptional even this tentative linking of hysterical symptoms to the body appeared at that point is perhaps best demonstrated by the way in which Tiihonen et al. interpretatively framed their empirical findings. They conjectured that the "simultaneous activation of frontal inhibitory areas and inhibition of the somatosensory cortex" could have arisen in response to "distressing psychological events," which in the case of their patient included "extreme stress due to her current marital and domestic situation."[484] This interpretation was highly speculative since the study did not explicitly test the potential role of a particular stressor in triggering the patient's symptoms. Apparently, with this interpretation, Tiihonen et al. attempted to reconcile the radically new neurobiological nature of their findings with, at the time, still apparently more acceptable psychogenic accounts. That is, rather than suggesting a clear-cut break with the previous psychogenic conceptual framework, Tiihonen et al. tried to embed their new findings into it. As we will see later, with the increasing number of functional neuroimaging studies, this situation would change, and a more clearly delineated neurophysiological interpretation of hysteria as a brain-based disorder would gradually emerge. Yet, despite the somewhat hesitant conclusion that they drew from their imaging findings, Tiihonen et al. made the first crucial step in this direction.

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In summary, even before it became directly implicated in specific studies of hysterical symptoms, the successful application of functional neuroimaging within the broader research into various cognitive functions and dysfunctions began to reinforce a general stance that mental and physical disorders were not mutually irreconcilable concepts. Although this general conceptual shift towards a biological framework at first only

480 Tiihonen et al., "Cerebral Blood Flow," 134.
481 Tiihonen et al., 134.
482 Tiihonen et al., 134.
483 See Tiihonen et al., 134, fig. 1.
484 Tiihonen et al., 134.

indirectly and tentatively affected hysteria, it sufficed to usher in a new era of functional neuroimaging investigation of this disorder. Since their inception, neuroimaging technologies have thus become powerful research tools whose application in psychiatry was not only made possible by the newly won primacy of the organicist perspective but had also additionally fortified this perspective.

2.3.3 Gradual Emergence of fMRI-Based Hysteria Research as a Sustained Scientific Practice

Following the publication of the first neuroimaging study of hysterical paralysis, at first, nothing happened. Then, in 1997 and 1998, two more functional neuroimaging studies of somatic symptoms of hysteria appeared.[485] In one study, SPECT was used to investigate five patients with heterogeneous symptoms. In the other, a woman with hysterical paralysis underwent a PET scanning. The introductory parts of these two studies contained clues as to why the first SPECT-based finding of the regional cerebral blood flow abnormalities in hysterical paralysis was initially met with silence. The authors of the 1998 study designated the Tiihonen et al. findings as "provocative."[486] Along similar lines, Marshall et al. suggested that conversion disorder/hysteria was in itself a controversial research topic because the very existence of this disorder was still doubted by many.[487] However, the appearance of two additional studies furnished further empirical indications that somatic symptoms of hysteria might indeed have neurophysiological underpinnings. Despite the lack of overlap in their imaging findings, the cumulative effect of the three initial studies proved intriguing enough to spark further interest in using functional brain imaging to investigate hysteria. In what follows, I will trace how this at first sporadic interest gradually coalesced into a persistent and clearly defined image-based research that soon became united around a single functional neuroimaging technology—the fMRI.

After a considerably delayed and hesitant start, functional neuroimaging enquiry into hysteria's puzzling somatic manifestations finally began to gain momentum in 2000. The authors of the two PET studies published that year were far less timid than their predecessors in interpreting their image-based results. "We postulate that positron emission tomography (PET) will provide objective evidence of hysterical pathophysiology," declared Spence et al. confidently.[488] "Since the psychological processes responsible for hysterical paralysis occur via physiological brain activity, functional imaging might reveal some of the neuropsychological mechanisms," claimed Halligan et al.[489] In other words, the authors of both studies explicitly stated their conviction that hysteria had a potentially detectable biological basis. Just as importantly, they forcefully expressed their confidence that functional brain imaging was the pertinent tool for investigating hysteria's hypothesised biological basis. Hence, it

485 Marshall et al., "Hysterical Paralysis"; and Yazici and Kostakoglu, "Cerebral Blood Flow."
486 Yazici and Kostakoglu, "Cerebral Blood Flow," 163.
487 Marshall et al., "Hysterical Paralysis," B1.
488 Spence et al., "Disorder of Movement," 1243.
489 Halligan et al., "Hypnotic Paralysis," 986.

appears that by the beginning of the twenty-first century, functional neuroimaging studies of hysteria have ceased to be viewed as either provocative or controversial. Instead, they finally joined the ranks of the broader neuroimaging research into psychiatric disorders.

Such growing acceptance of using functional brain imaging to investigate hysteria has been reflected in the continually rising number of published studies. Based on my search of the medical literature, twenty-two functional neuroimaging studies of various somatic symptoms of hysteria appeared in the first decade of the twenty-first century.[490] In the second decade of the twenty-first century, eighty-three additional studies followed.[491] Significantly, my account here rests on the inclusion of only

490 Burgmer et al., "Movement Observation"; Cojan et al., "Self-Control"; Cojan et al., "Inhibition"; de Lange, Roelofs, and Toni, "Motor Imagery"; de Lange, Roelofs, and Toni, "Self-Monitoring"; Egloff et al., "Somatosensory Deficits"; Garcia-Campayo et al., "Somatization"; Ghaffar, Staines, and Feinstein, "Sensory Conversion Disorder"; Gündel et al., "Somatoform Pain"; Hakala et al., "Severe Somatization"; Halligan et al., "Hypnotic Paralysis"; Kanaan et al., "Repressed Memories"; Mailis-Gagnon et al., "'Hysterical' Anesthesia"; Okuyama et al., "Psychogenic Visual Disturbance"; Saj, Arzy, and Vuilleumier, "Spatial Neglect"; Spence et al., "Disorder of Movement"; Stoeter et al., "Somatoform Pain"; Stone et al., "Simulated Weakness"; Tanaka et al., "Pseudohysterical Hemiparesis"; Vuilleumier et al., "Sensorimotor Loss"; Ward et al., "Differential Brain Activations"; and Werring et al., "Visual Loss." My cutoff point for the studies that appeared in the first decade of the twenty-first century is December 31, 2009.

491 Allendorfer et al., "Psychological Stress"; Arthuis et al., "Cortical PET"; Aybek et al., "Life Events"; Aybek et al., "Emotion-Motion Interactions"; Baek et al., "Motor Intention"; Becker et al., "Conversion Blindness"; Bègue et al., "Metacognition"; Blakemore et al., "Aversive Stimuli"; Bryant and Das, "Neural Circuitry"; Burgmer et al., "Mirror Neuron System"; Burke et al., "Ancillary Activation"; Conejero et al, "Altered Brain Metabolism"; Czarnecki et al., "SPECT Perfusion"; de Greck et al., "Emotional Empathy"; de Greck et al., "Reward"; de Lange, Toni, and Roelofs, "Altered Connectivity"; Dienstag et al., "Motor Control"; Diez et al., "Fast-Tracking"; Ding et al., "Connectivity Density"; Ding et al., "Connectivity Networks"; Dogonowski et al., "Recovery"; Espay et al., "Neural Responses"; Espay et al., "Functional Dystonia"; Espay et al., "Functional Tremor"; Guo et al., "Anatomical Distance"; Hassa et al., "Motor Control"; Hassa et al. "Motor Inhibition"; Hedera, "Metabolic Hyperactivity"; Huang et al. "Spontaneous Activity"; Karibe et al., "Somatoform Pain"; Kim et al., "Functional Connectivity"; Koh et al., "Shared Neural Activity"; Kryshtopava et al., "Phonation in Women"; LaFaver et al., "Before and After"; Lemche et al., "Somatization Severity"; Li et al., "Causal Connectivity"; Li et al., "Insular Subregions"; Li et al., "Regional Activity"; Li et al., "Regional Brain Function"; Liu et al., "Functional Hubs"; Luauté et al., "Simulation, conversion, ou majoration?"; Luo et al., "Pain Processing"; Matt et al., "Cortex Deactivation"; Maurer et al., "Impaired Self-Agency"; Monsa, Peer, and Arzy, "Self-Reference"; Morris et al., "Avoidance"; Nahab et al., "Sense of Agency"; Noll-Hussong et al., "Affective Meaning Construction"; Noll-Hussong et al., "Sexual Abuse"; Otti et al., "Chronic Pain"; Otti et al., "Somatoform Pain"; Ou et al., "Nucleus Accumbens"; Ou et al., "Regional Homogeneity"; Pan et al., "Functional Connectivity"; Rota et al., "Vision Loss"; Roy et al., "Dysphonia"; Saj et al., "Mental Imagery"; Schoenfeld et al., "Hysterical Blindness"; Schrag et al., "Dystonia"; Shimada et al., "Cerebellar Activation"; Sojka et al., "Processing of Emotions"; Song et al., "Regional Homogeneity"; Spengler et al., "Voice Loss"; Stankewitz et al., "Fronto-Insular Connectivity"; Su et al., "Interhemispheric Connectivity"; Su et al., "Regional Activity"; Su et al., "Connectivity Strength"; Szaflarski et al., "Facial Emotion Processing"; van Beilen et al., "Conversion Paresis"; van der Kruijs et al., "Executive Control"; van der Kruijs et al., "Dissociation in Patients"; van der Kruijs et al., "Resting-State Networks"; Voon et al., "Emotional Stimuli"; Voon et al., "Involuntary Nature"; Voon et al., "Limbic Activity"; Wang et al., "Clinical

those studies that investigated somatic symptoms explicitly attributed to conversion disorder or somatisation, as well as their diagnostic successors in the *DSM-5*.[492] I have disregarded neuroimaging studies that dealt with a range of other medically unexplained diagnoses whose relation to hysteria remains a matter of debate among experts.[493] This exclusion has two reasons. First, it aims to safeguard the term hysteria, as I use it here, from becoming too fuzzy. Second, it enables me to focus on examining the epistemic function of images in the contemporary neuroscientific studies of those somatic symptoms that had been at the centre of Charcot's image-based research on hysteria. For this reason, in the remainder of this enquiry, my primary focus will remain limited to neuroimaging studies of symptoms such as paralysis, contractures, anaesthesia, tremor, blindness, pain, mutism, and pseudo-epileptic seizures.

Additionally, this strict delineation is also necessary because, since 2000, there have been considerable terminological inconsistencies across neuroimaging studies of hysterical symptoms. Although most researchers still expressly relate these symptoms to the historical diagnosis of hysteria,[494] they have stopped explicitly using the term 'hysterical' in their studies.[495] Instead, they have deployed different labels, such as conversion disorder, somatoform, somatic, somatisation, non-organic, psychogenic and, more recently, functional.[496] To sidestep the terminological confusion that dominated the neuroimaging literature in the first two decades of the twenty-first century, I will continue to use the term hysteria when referring to all contemporary neuroimaging studies.

Compared to several thousand functional neuroimaging studies on psychiatric disorders such as schizophrenia or depression published by 2020, the contemporary image-based investigation of somatic hysteria, which comprises about one hundred

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Significance"; Wegrzyk et al., "Functional Connectivity"; Wei et al., "Default-Mode Network"; Yoshino et al., "Neural Responses to Pain"; Yoshino et al., "Regional Neural Responses"; Yoshino et al., "Therapy"; and Zhao et al., "Functional Connectivity." My cutoff point for the studies that appeared in the 2010s is December 31, 2019. Since my focus is on the hysteria research from the first two decades of the twenty-first century, functional neuroimaging studies published since January 1, 2020 will not be discussed in this book.

492 In the *DSM-5*, the umbrella category somatoform disorders was renamed somatic symptoms and related disorders. Its central subcategory, previously referred to as somatisation, was relabelled somatic symptom disorder. See APA, *DSM-5*, 309. We will discuss these changes in section 2.4.2.

493 I have disregarded neuroimaging studies that investigated a range of monosymptomatic functional syndromes, such as chronic fatigue disorder or fibromyalgia, as well as other medically unexplained symptoms whose relation to hysteria remains unclear. See, e.g., Wessely, Nimnuan, and Sharpe, "Functional Somatic Syndromes." Due to my strict focus on the somatic expressions of hysteria, all dissociative disorders (i.e., dissociative identity disorder, psychogenic amnesia and depersonalisation) have also been left out of my account.

494 See, e.g., Aybek et al., "Life Events," 52; Bègue et al., "Metacognition," 251–52; Cojan et al., "Inhibition," 1026; and Kanaan et al., "Repressed Memories," 202.

495 One notable exception is the 2011 study in which the patient's medically unexplained visual loss is explicitly designated as hysterical blindness. See Schoenfeld et al., "Hysterical Blindness."

496 See, e.g., Espay et al., "Functional Dystonia"; Lemche et al., "Somatization Severity"; Otti et al., "Somatoform Pain"; and van Beilen et al., "Conversion Paresis."

research papers for the same period, may appear negligible in size.[497] However, I argue that despite its small size, it nevertheless merits serious attention, as it has consolidated into a distinct, coordinated, and sustained research effort, which has once again rendered visible a once highly contentious disorder. A pertinent indication of this development is that multiple individual researchers and research teams have, over the years, repeatedly used brain imaging to systematically investigate hysterical symptoms from multiple perspectives by building on their own and their colleagues' previous work.

For instance, between 2007 and 2010, the Dutch researchers de Lange, Roelofs, and Toni published three consecutive studies of hysterical/conversion paralysis.[498] In their consecutive studies, two of which I will analyse in the following chapter, de Lange, Roelofs, and Toni applied varying experimental conditions and used different, mutually complementary approaches to analysing their neuroimaging data. Similar examples abound of researchers who have systematically examined hysterical symptoms across several fMRI studies over the last fifteen years.[499] Furthermore, in 2010, Roloefs also co-authored with her British and American colleagues a neuroimaging study that investigated the potential role of emotions in hysterical tremor.[500] Hence, connections among researchers are not limited to mutual cross citations of published findings but also include direct collaborations across different teams and institutions.

An additional sign of the growing maturity of neuroimaging hysteria research is the extent to which both its thematic and geographic scope widened within the first decade of the twenty-first century. Whereas the early research mainly focused on hysterical paralysis, subsequent studies have diversified to encompass a range of somatic symptoms such as tremor, non-epileptic seizures, contractures, blindness, anaesthesia, and pain.[501] Moreover, although it already started as an international endeavour with the initial studies conducted across Europe, neuroimaging of hysteria has soon spread around the globe. Based on the publication output, it can be said that

497 My search of MEDLINE, the National Library of Medicine's (NLM) extensive online database (www.ncbi.nlm.nih.gov/pubmed), for functional neuroimaging studies of schizophrenia returned more than 7800 published articles, whereas for depression, more than 9700. The search was performed on January 7, 2020.

498 See de Lange, Roelofs, and Toni, "Self-Monitoring"; de Lange, Roelofs, and Toni, "Motor Imagery"; and de Lange, Toni, and Roelofs, "Altered Connectivity."

499 For additional examples of researchers who have systematically examined hysterical symptoms across several fMRI studies, see Espay et al., "Neural Responses"; Espay et al., "Functional Dystonia"; Espay et al., "Functional Tremor." Another pertinent example is Valerie Voon. See Voon et al., "Emotional Stimuli"; Voon et al., "Involuntary Nature"; Voon et al., "Limbic Activity"; Baek et al., "Motor Intention"; and Morris et al., "Avoidance." For multiple studies co-authored by Selma Aybek, see Aybek et al., "Life Events"; Aybek et al., "Emotion-Motion Interactions"; Bègue et al., "Metacognition"; Blakemore et al., "Aversive Stimuli"; and Wegrzyk et al., "Functional Connectivity." See also footnote 505 below.

500 See Voon et al., "Emotional Stimuli."

501 See, e.g., Ghaffar, Staines, and Feinstein, "Sensory Conversion Disorder"; Gündel et al., "Somatoform Pain"; Schoenfeld et al., "Hysterical Blindness"; van der Kruijs et al., "Emotion and Executive Control"; and Voon et al., "Involuntary Nature."

the most active research teams are currently situated in the UK, Switzerland, Germany, the Netherlands, the USA, Canada, Israel, Australia, China, and Japan.[502]

Even more significantly, the expansion and diversification of research interests started to be accompanied by efforts at systematising the newly won insights into the neural basis of hysteria. Thus, as of 2004, a gradually increasing number of literature reviews of neuroimaging hysteria research have begun to appear in specialised neurological and neuropsychiatric journals.[503] Typically, such meta-studies have synthesised the individual imaging findings by bringing them in relation to one another to draw more general conclusions about the nature of hysterical symptoms.[504] Additionally, multiple meta-studies have also evaluated individual imaging studies from the methodological point of view, analysed their strengths and weaknesses, and suggested potential directions for future research. In many cases, the authors of the literature reviews have been particularly prolific participants in the functional neuroimaging investigation of hysteria.[505]

Finally, I suggest that the consolidation of contemporary hysteria research has been closely linked to the choice of a particular functional neuroimaging technology as the primary investigation tool. During its initial phase in the late 1990s and early 2000s, the emerging hysteria research appears to have been rather conservative in its use of neuroimaging tools. Until 2003, all studies of hysterical symptoms employed PET and SPECT, although fMRI was already used as an investigation tool in other areas of psychiatric research.[506] Functional MRI (fMRI) was developed in the early 1990s out of the older structural MRI technology.[507] Within only several years after its first applications in human subjects in 1992, fMRI advanced to the most widely used functional imaging technology across the neurosciences.[508] The veritable boom

502 For an overview of these studies, see footnotes 490 and 491 above.

503 See, e.g., Bell et al., "Hysteria and Hypnosis"; Black et al., "Conversion Hysteria"; Boeckle et al., "Meta-Analysis"; Broom, "Neuroscience of Hysteria"; Browning, Fletcher, and Sharpe, "Critical Review"; Carson et al., "Since the Millennium"; Conejero et al., "Neuroanatomy"; Ejareh dar and Kanaan, "Etiology"; Harvey, Stanton, and David, "Neurobiological Understanding"; Lang and Voon, "Future Directions"; Scott and Anson, "Neural Correlates"; Voon, "Functional Neurological Disorders: Imaging"; Voon et al., "Functional Neuroanatomy"; Vuilleumier, "Brain Circuits"; and Vuilleumier, "Neurophysiology of Self-Awareness." See also Hallett, "Crisis for Neurology"; and my analysis of how Hallett's declaration of crisis additionally fueled the early neuroimaging research on hysteria in Muhr, "Recent Trajectory."

504 See, e.g., Browning, Fletcher, and Sharpe, "Critical Review"; Carson et al., "Since the Millennium"; Voon et al., "Functional Neuroanatomy"; Vuilleumier, "Brain Circuits"; and Vuilleumier, "Neurophysiology of Self-Awareness."

505 For example, Patrik Vuilleumier has co-authored numerous functional neuroimaging studies on hysteria. See Vuilleumier et al., "Sensorimotor Loss"; Bègue et al., "Metacognition"; Blakemore et al., "Aversive Stimuli"; Cojan et al., "Inhibition"; Cojan et al., "Self-Control"; Luauté et al., "Simulation, conversion, ou majoration?"; and Saj, Arzy, and Vuilleumier, "Spatial Neglect." For the list of fMRI studies co-authored by Valerie Voon, see footnote 499 above.

506 See, e.g., Blakemore, "Schizophrenia and Brain Imaging," 652–55.

507 See Huettel, Song, and McCarthy, *Imaging*, 193–208.

508 Huettel, Song, and McCarthy, 3–4.

in general neuroscientific research since the end of the twentieth century is often attributed to the introduction of this particular neuroimaging technology.[509]

A shared feature of PET, SPECT, and fMRI is that they all generate visualisations of the living brain, which contain only indirect information about the neural activity. This is because all these technologies make use of the fact that neural activity is correlated with local changes in cerebral metabolism and blood flow.[510] However, each technology measures a different aspect of the physiological response to neural activity.[511] PET and SPECT rely on the injection of small amounts of radioactive substances called radiotracers into the subject's bloodstream to register changes either in the cerebral blood flow or brain metabolism.[512] By contrast, most fMRI methods utilise a combination of external magnetic fields to measure the effects of a naturally occurring neurophysiological phenomenon as a proxy for neural activity.[513] This neurophysiological phenomenon comprises an experimentally established linkage between local changes in the blood flow and oxygen consumption in active areas of the brain.[514] For this reason, the resulting images are referred to as blood-oxygenation-level dependent (BOLD) fMRI. Moreover, each of these three neuroimaging technologies uses a distinct type of scanner, whose operations are underpinned by different physical theories. Consequently, the processes of data acquisition and analysis, as well as the specific type of information encoded in the resulting brain images diverge significantly across all three technologies.[515]

Hence, to use the term introduced by the philosopher of science Ronald Giere, SPECT, PET, and fMRI offer markedly different instrumental perspectives on the brain activity of interest.[516] Significantly, this does not mean that these technologies produce quintessentially different kinds of knowledge or mutually irreconcilable results. On the contrary, PET, SPECT, and fMRI can all be used to probe the presumed neurophysiological basis of hysteria.[517] Such overlapping use of different instrumental perspectives only reinforces the apparent "objectivity" of the findings, ensuring that converging measurements—although obtained through different technologies—can

509 Raichle, "Brain Mapping," 122.

510 See, e.g., Devous, "Imaging Brain Function," 147–50; and Raichle, "Historical and Physiological Perspective," 4–20.

511 See Raichle, "Historical and Physiological Perspective," 3–21.

512 Cabeza and Nyberg, "Imaging Cognition II," 2.

513 The term technology, as I deploy it here, refers to the use of a particular kind of scanner. Some scanners can be employed to measure highly diverse aspects of the brain. Different measurement foci of the same technology are here referred to as methods. Functional MRI includes different methods, each of which provides information about different functional aspects of the brain. For a detailed overview of these methods, see Giesel et al., "MR-basierte Methoden." See also Huettel, Song, and McCarthy, *Imaging*, 122–46.

514 For details, see Ogawa et al., "Oxygenation-Sensitive"; and Ogawa et al., "Blood Oxygenation."

515 See, e.g., Huettel, Song, and McCarthy, *Imaging*, 4–9, 197–98.

516 Giere has offered a succinct description of several neuroimaging technologies as part of the analysis from which his concept of scientific perspectivism was derived. See Giere, *Scientific Perspectivism*, 56–59.

517 Compare, e.g., Vuilleumier et al., "Sensorimotor Loss"; and Ghaffar, Staines, and Feinstein, "Sensory Conversion Disorder."

indeed be ascribed the status of scientific evidence.[518] However, as we are about to see, what differs across these technologies is the flexibility with which research questions can be asked and the degree of precision with which these questions can be answered.

In this respect, fMRI has several advantages over PET and SPECT. Since it does not rely on the injection of radioactive substances, subjects can undergo repeated fMRI scanning without any risk to their health.[519] Additionally, fMRI provides a considerably better spatial resolution than PET or SPECT, thus allowing a more precise anatomical localisation of neural activity.[520] And although more detailed, fMRI images are also acquired more quickly. Hence with fMRI, one image is acquired every 1–3 seconds instead of over several minutes, as is the case with PET and SPECT.[521] This means that fMRI provides a larger quantity of data with a considerably better temporal resolution, which is of crucial importance because what is being measured are dynamic neurophysiological processes. Finally, what is particularly significant is that, compared to SPECT and PET, fMRI allows researchers to deploy much more complex and fine-grained sets of experimental conditions under which the subjects' neural responses are measured.[522] This, in turn, enables researchers to pose more nuanced questions about the neural underpinnings of the mental phenomena of interest.[523]

I suggest that it is due to all these advantages taken together that, after only a handful of PET and SPECT studies, fMRI came to the forefront of hysteria research and, as of 2004, largely displaced the use of the other two functional neuroimaging technologies.[524] From this point onwards, functional neuroimaging studies of hysterical symptoms started to grow in number, as discussed above. Moreover, both the proliferation and the thematic diversification of hysteria research can be traced back to the adoption of fMRI as a more powerful and flexible functional neuroimaging technology.[525] Therefore, it can be argued that through the shift to fMRI as the primary epistemic tool, contemporary neuroimaging investigation of hysteria came of age and crystallised into a systematic and sustained image-based research endeavour that is here to stay. Due to the crucial epistemic role of this technology in the current image-based hysteria research, the rest of my inquiry will focus exclusively on fMRI, thus disregarding the few studies of hysterical symptoms that were conducted using other technologies.

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518 Giere, *Scientific Perspectivism*, 57–58.

519 Conversely, due to the strict limitations of radiation exposure, only a few PET/SPECT scans of a single subject can be made. Moreover, SPECT/PET scanning is costly and time-consuming because the radioactive tracer has to be created in a particle accelerator directly before the imaging. Huettel, Song, and McCarthy, *Imaging*, 197–98.

520 Huettel, Song, and McCarthy, 198.

521 Huettel, Song, and McCarthy, 197–98.

522 Huettel, Song, and McCarthy, 198.

523 We will discuss this in the following chapter.

524 Since 2004, only a few neuroimaging studies of hysterical symptoms were conducted using PET or SPECT. See, e.g., Arthuis et al., "Cortical PET"; Rota et al., "Vision Loss"; Tanaka et al., "Pseudohysterical Hemiparesis"; Schrag et al., "Dystonia"; and Ward et al., "Differential Brain Activations."

525 Compare studies listed in footnotes 490 and 491 above.

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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.