

research into hysteria largely died out.²⁷² Drawing all these aspects together, I suggest that the twentieth century can be fittingly characterised as a visual hiatus in hysteria research. Yet, this hiatus was not without consequences. In what follows, I will argue that the visual hiatus contributed to the increasing invisibility of hysteria in the medical context, finally culminating in the apparent disappearance of this age-old disorder by the end of the twentieth century.

2.2 The Putative Disappearance of Somatic Manifestations of Hysteria

After centuries of a convoluted and turbulent history,²⁷³ during which the medical interest in this disorder periodically intensified and waned, hysteria appeared to have reached the highest point of its scientific visibility in the works of first Charcot and then Freud. However, at some undefined turning point in the second half of the twentieth century, this disorder mysteriously disappeared.²⁷⁴ Although the putative disappearance of hysteria seems to be a generally accepted fact, there is little agreement as to why and to what extent the heterogeneous symptoms that once comprised this disorder ceased to exist. Multiple authors, who understand hysteria in Freudian terms as a symbolic expression of personal discontent, converge on the view that all hysterical symptoms have vanished because they became redundant.²⁷⁵ Some of these authors have contended that hysterical symptoms have disappeared because Freud had successfully disclosed their true nature. As a result, hysterical symptoms became subjectively unrewarding, and patients stopped manifesting them.²⁷⁶ Others have claimed that the symptoms became obsolete due to the socio-cultural changes that had brought an end to female social oppression and sexual repression.²⁷⁷

Conversely, several medical historians have suggested alternative explanations for hysteria's purported disappearance.²⁷⁸ The point in common across such different accounts is that hysteria has not disappeared entirely as a pathological entity. Instead, it underwent changes and thus adapted to the new era. For instance, Mark S. Micale has argued that from 1895 to 1910, due to advances in medical knowledge, hysteria was "broken down into its constituent symptomatological parts."²⁷⁹ The resulting parts were then redistributed to either organic neurological diseases or newly defined psychiatric disorders. Only a fraction of the historical disorder was conveyed to the present, forming "enormously reduced usages of the hysteria concept in current-day psychiatric medicine."²⁸⁰ By contrast, Elaine Showalter and Edward Shorter have contended that

272 Stone et al., 13. I will discuss this point in more detail in the following sections.

273 For a succinct overview, see Micale, *Approaching Hysteria*, 19–29.

274 See, e.g., Kinetz, "Is Hysteria Real," n.p.

275 For a detailed overview of studies whose authors have espoused this view, see Micale, "Disappearance," 499n7, 500n8.

276 Veith, *Hysteria*, 273–74.

277 For an overview, see Micale, "Disappearance," 500n9.

278 See Micale, "Disappearance"; Shorter, *From Paralysis to Fatigue*; and Showalter, *Hystories*.

279 Micale, "Disappearance," 525.

280 Micale, 525.

hysteria has not so much vanished as mutated into new forms of “culturally permissible expressions of distress.”²⁸¹ Yet, while Micale, Showalter, and Shorter deny the complete disappearance of hysteria, they nevertheless insist that the “gross and florid” motor and sensory symptoms from Charcot’s and Freud’s famous case studies are no longer among us.²⁸²

Paradoxically, precisely these supposedly no longer existing symptoms—such as paralyses, convulsive seizures, anaesthesia, and blindness—happen to be at the focus of functional brain imaging studies of hysteria, which have started appearing in the closing years of the twentieth century.²⁸³ A possible conclusion could be that such studies utilise a relatively novel set of imaging technologies in an attempt to breathe new life into hysteria and thus artificially revive a long-discarded medical entity. Alternately, it can be contended, as I will in the following three sections, that the ‘classic’ somatic symptoms of hysteria have never actually disappeared. They merely became invisible due to the medical community’s waning interest in them. Moreover, I will argue that this waning interest arose in response to major conceptual shifts that psychiatry underwent in the second half of the twentieth century.²⁸⁴

Specifically, I intend to show that the conceptual shifts, whose details I will analyse shortly, resulted in three distinct yet mutually interrelated developments. First, hysteria turned into a loosely grouped set of medically unexplainable somatic symptoms. Second, these somatic symptoms came to be viewed in the medical context as essentially undiagnosable. And third, all somatic manifestations of hysteria became summarily equated with intentional simulation. In other words, we will see that in the second half of the twentieth century, hysteria once again attained a similarly contested status as it had had before Charcot launched his systematic image-based research into this enigmatic disorder. In the following, my analysis will primarily deal with the somatic

281 Showalter, *Hystories*, 15. Shorter refers to the culturally accepted manifestations of hysteria as “the symptom pool” and claims that, at present, it comprises elusive complaints, such as highly subjective sensations of psychosomatic pain and fatigue. Shorter, *From Paralysis to Fatigue*, 1–10, 267. Showalter suggests a different classification by listing not only chronic fatigue but also multiple personality disorder, recovered memories of sexual abuse, the Gulf War syndrome, satanic ritual abuse, and alien abduction as contemporary manifestations of hysteria. Showalter, *Hystories*, 12.

282 Micale, “Disappearance,” 498. See also Shorter, *From Paralysis to Fatigue*, 196–200, 267–73; and Showalter, *Hystories*, 15.

283 Tiisonen et al., “Hysterical Paraesthesia”; Yazici and Kostakoglu, “Cerebral Blood Flow”; and Marshall et al., “Hysterical Paralysis.”

284 A group of contemporary neurologists have similarly argued that the lack of medical interest has caused the apparent disappearance of hysteria. However, they have ascribed this loss of interest to the professional division between psychiatry and neurology, which took place at the beginning of the twentieth century. In their words, this division left hysteria in “a no-man’s land between these two specialities.” See Stone et al., “Disappearance,” 12. In what follows, I will posit a different explanation for the waning of medical interest in hysteria in the second half of the twentieth century.

symptoms of hysteria that once stood at the centre of Charcot's research and are now the focus of functional neuroimaging studies.²⁸⁵

2.2.1 The Transformation of Hysteria into a Medically Unexplained Disorder

Since the introduction of standardised classifications of mental diseases in the second half of the twentieth century, hysteria as a medical entity in all its taxonomic incarnations has been determined by the definitions, diagnostic criteria, and labels that the prevailing nosological systems ascribed to it. The *Diagnostic and Statistical Manual of Mental Disorders (DSM)* of the American Psychiatric Association (APA) and the *International Classification of Diseases (ICD)* of the World Health Organisation (WHO) have established themselves as the two dominant classification systems in contemporary psychiatry.²⁸⁶ Importantly, periodical updates of these classification systems have done much more than passively reflect the ongoing conceptual shifts in the understanding of psychiatric disorders in general and hysteria in particular. Apart from providing the basis for the diagnosis and treatment of patients, the classification updates have also acted as generators of new conceptual shifts that have decisively informed subsequent medical research. As explicitly stated by the authors of the *DSM*, they have aimed to provide “the field with a summary of the state of the science relevant to psychiatric diagnosis and letting it know where gaps existed in the current research, with hopes that more emphasis would be placed on research within those areas.”²⁸⁷ Hence, as my analysis will show, each classification update has had significant consequences for diagnosing and researching hysteria.

From the 1950s until today, hysteria has undergone multiple dramatic and far-reaching changes with each successive update of the *ICD* and *DSM*.²⁸⁸ These changes have included repeated fragmentation and relabelling of hysteria, as well as multiple revisions of its diagnostic criteria. Micale has designated this process as “the clinical and terminological dismemberment” of hysteria.²⁸⁹ However, in what follows, I will argue that even more than the dismemberment itself, what decisively contributed to the increasing invisibility of hysteria in the medical context was how its nosological successors came to be redefined across different updates. More specifically, I will claim that the most significant aspect of this process was the gradual reconceptualisation of hysteria into a set of medically unexplained somatic symptoms. To prove this point, in this section, I will trace the taxonomic transformations hysteria underwent across

285 See, e.g., Burke et al., “Ancillary Activation”; de Lange, Roelofs, and Toni, “Self-Monitoring”; van der Kruijs et al., “Emotion and Executive Control”; and Voon et al., “Involuntary Nature.”

286 A section on mental diseases was included for the first time in the 6th edition of the *ICD*, which was published in 1948. See WHO, “History of *ICD*.” The first edition of *DSM* followed four years later. See APA, *DSM-I*. See also APA, “*DSM* History.”

287 APA, “*DSM* History,” n.p.

288 See, e.g., APA, *DSM-II*, 39–40; APA, *DSM-III*, 241–60; and APA, *DSM-5*, 291–327.

289 Micale, *Approaching Hysteria*, 292.

the first three successive editions of the *DSM*.²⁹⁰ Later in this chapter, I will show that the shifts in how hysteria's contemporary nosological successors were encoded in the *DSM-IV* made the reappearance of image-based research into this disorder possible at the end of the twentieth century.

The initial step in the nosological transformation of hysteria occurred in 1952, with the publication of the first edition of the *DSM*. In *DSM-I*, hysteria was split up into dissociation and conversion reactions, both of which were included within the category of psychoneurotic disorders.²⁹¹ The decisive influence of the two major psychogenic concepts of dissociation and conversion, which had been developed by Janet and Freud respectively, was evident not just in the new taxonomy but also in the manual's explicit emphasis on the causative role of psychological factors. Dissociation and conversion were defined as two distinct psychological mechanisms with which the patient subconsciously reacted to subjectively perceived danger.²⁹² In line with Janet's research, the *DSM-I* specified dissociative reaction as "a type of gross personality disorganisation," whose symptoms comprised an array of disturbances in identity and memory.²⁹³ These included amnesia, dream states, stupor, somnambulism, and dissociated personalities. Conversely, as typical manifestations of conversion reactions, the *DSM-I* listed various pseudoneurological somatic deficits, such as anaesthesia, paralysis, and movement disturbances.²⁹⁴ Echoing Freud, the latter symptoms were designated as symbolic somatic expressions of an underlying mental conflict.

Rather undemonstratively, the *DSM-I* replaced the historical term 'hysteria' with new diagnostic labels. However, in my opinion, what was particularly remarkable about the *DSM-I*'s relabelling of hysteria was the resulting separation of the psychological and somatic manifestations of this disorder. No explanation was offered for this division. This is all the more surprising since such a division stood in stark contrast to the most prominent nineteenth-century conceptions of hysteria in which highly diverse symptoms had been consistently regarded as manifestations of a single disorder. The *DSM-I*'s approach thus directly contradicted Charcot's neurological and Janet's and Freud's psychogenic theories of hysteria, all three of which had posited a unifying mechanism for both physical and psychological symptoms.

With the publication of the revised *DSM-II* in the late 1960s, the term hysteria was temporarily reinstated into the official medical nomenclature, albeit only in its adjectival form, as a hysterical neurosis.²⁹⁵ Yet also in this updated version, it was explicitly stated that the "distinction between conversion and dissociative

290 There are considerable differences in how hysteria has been coded in the *DSM* and *ICD*. My analysis is restricted to the *DSM*, as it is considered more dominant in the research context, which represents the focal point of my enquiry. See Trimble, *Biological Psychiatry*, xiv.

291 See APA, *DSM-I*, 32–33. Other psychoneurotic disorders included anxiety and depressive reactions. *Ibid.* For Freud's initial introduction of the category of psychoneurosis, see Freud, "Neuro-Psychoses of Defence."

292 APA, *DSM-I*, 31–32.

293 APA, 32.

294 APA, 31–33.

295 See APA, *DSM-II*, 39–40.

reactions should be preserved.”²⁹⁶ Hence, the *DSM-II* retained the bipartite division of hysteria into somatic and psychological symptoms, which the previous edition had introduced. The categorisation of individual symptoms remained unchanged, as did the conceptualisation of both types of hysterical neuroses as purely psychogenic disorders.²⁹⁷

The most substantial taxonomic and conceptual transformation of hysteria took place in 1980, with the publication of the *DSM-III*. This much-discussed and often criticised edition marked a paradigm shift in psychiatric nosology.²⁹⁸ The previous two editions operated with short, glossary definitions of mental disorders, emphasising their presumed psychological aetiologies. By contrast, the *DSM-III* introduced explicit diagnostic criteria and checklists of salient symptoms, thus mirroring diagnostic models from general medicine.²⁹⁹ This descriptive, symptom-based focus was derived from a purportedly “atheoretical” approach to the aetiology and pathophysiology of psychiatric disorders.³⁰⁰ But, in effect, it targeted the deletion of the psychoanalytically informed aetiologies, which had been dominant in the psychiatric context until that point.³⁰¹ As a result of this general reorientation, the category of neuroses came to be viewed as an outdated and highly contested Freudian concept and thus abolished from psychiatric nosology.³⁰² The disorders that had previously been designated as neuroses were renamed and relegated to other sections of the manual. In the process, the *DSM-III* permanently deleted the term hysteria from the official medical nomenclature. However, as I am about to show, far more significant than the expunging of its name was the conceptual refashioning to which hysteria was submitted in the *DSM-III*.

We have seen that in the previous editions of the *DSM*, the mental and somatic symptoms of hysteria had already been separated into two distinct diagnostic labels, yet nevertheless remained classified within the same category of neuroses. But the *DSM-III* went a step further. In the new edition, the mental and somatic symptoms of hysteria were split asunder into two completely separate diagnostic categories. Different disturbances of consciousness, identity, and memory, which in the previous *DSM* editions had been listed as symptoms of the dissociative type of hysterical neurosis, were now accorded the status of individual disorders.³⁰³ These were then grouped into a newly established umbrella category of dissociative disorders. An even more substantial change consisted of introducing a separate new umbrella category of somatoform disorders.³⁰⁴ Within this new category, various somatic symptoms

296 APA, 39.

297 APA, 40.

298 See, e.g., Scull, *Hysteria*, 182–86.

299 First, “Development of *DSM-III*,” 127.

300 APA, *DSM-III*, 7.

301 First, “Development of *DSM-III*,” 132–33.

302 APA, *DSM-III*, 9–10.

303 For details, see APA, 253–60.

304 APA, 241–51.

that had previously comprised hysteria became redistributed in two novel diagnostic subcategories—conversion and somatisation disorders.³⁰⁵

The newly introduced diagnosis of conversion disorder displaced the conversion type of hysterical neurosis used in the *DSM-II*. It retained the focus on ‘classic’ pseudoneurological symptoms that entailed various forms of sensory and motor disturbances.³⁰⁶ Significantly, the straightforward psychogenic causation from the previous editions was displaced by a more ambiguous definition. According to the new definition, the physical symptoms were “apparently an expression of a psychological conflict or need.”³⁰⁷ Through this subtle shift in the formulation, the symptoms were, in effect, left without any clear aetiology. For the lack of a better explanation,³⁰⁸ the symptoms continued to be linked to psychological factors, but more loosely than in the previous editions of the *DSM*. Concerning conversion disorder, the *DSM-III* still allowed for a somatic symptom to be interpreted as a symbolic resolution of an underlying psychological problem.³⁰⁹ However, to do so, a physician had to prove that “there is a temporal relationship between an environmental stimulus that is apparently related to a psychological conflict or need and the initiation or exacerbation of the symptom.”³¹⁰ In fact, in this reformulation, psychological stressors no longer had the role of direct causative factors, as Freud had defined them. Instead, once again, the environmental stressors became reduced to mere precipitating factors, as Charcot had viewed them.³¹¹ The retained symbolic value of symptoms appeared to sit somewhat uneasily with this reformulation.

Moreover, under the label of somatisation disorder, the *DSM-III* inaugurated a prototypical somatoform disorder, emphasising—somewhat surprisingly—that this novel diagnostic category had been historically referred to as hysteria.³¹² Just as

305 The umbrella category of somatoform disorders included additional subcategories such as psychogenic pain disorder, hypochondriasis, and atypical somatoform disorder. See APA, 247–52. Since these disorders were not directly linked to Charcot’s concept of hysteria, I will disregard them in my analysis.

306 The symptoms included “paralysis, aphonia, seizures, coordination disturbance, akinesia, dyskinesia, blindness, tunnel vision, anosmia, anesthesia, and paresthesia.” APA, 244.

307 APA, 244.

308 APA, 241.

309 APA, 244.

310 APA, 244.

311 However, whereas Charcot, as discussed previously, posited the hereditary ‘weakness’ of the nervous system as the underlying cause of hysteria, the *DSM-III* did not. Thus it remained unclear why environmental stressors triggered hysterical symptoms in some individuals but not in others.

312 APA, *DSM-III*, 241. In fact, somatisation disorder was an artificially constructed hybrid. In terms of content, this novel diagnostic entity was derived from the seminal work by Michael Perley and Samuel Guze. Starting from the 1960s, these two American psychiatrists tried to establish a set of quantifiable and clinically testable diagnostic criteria for a polysymptomatic form of hysteria which they referred to as Briquet’s syndrome. They insisted that hysteria, i.e., Briquet’s syndrome, started early in life and was characterised by a multitude of dramatic, recurring symptoms that affected many different organ systems and were not reducible to conversion disorder. They also argued that hysteria was a distinct disease entity that could be validly diagnosed. For details, see, e.g., Guze, “Diagnosis of Hysteria”; Guze, “Validity and Significance”; Guze and Perley, “History of Hysteria”; and Perley and Guze, “Clinical Criteria.” At the formal level, the term ‘somatisation’

surprisingly, *DSM-III* stated that whereas conversion disorder was rare in clinical practice, somatisation was common.³¹³ As defined in the *DSM-III*, somatisation disorder entailed “multiple and recurring somatic complaints of several years’ duration.”³¹⁴ In addition to the pseudoneurological symptoms already listed under conversion disorder, somatisation also included somatic complaints that affected many other organ systems.³¹⁵ In other words, the two diagnostic entities partly overlapped. But somatisation was defined as more chronic and encompassing more diverse symptoms than conversion disorder. The *DSM-III* listed thirty-seven different symptoms.³¹⁶ These included paralysis, seizures, dizziness, psychosexual dysfunction, menstrual irregularity, palpitation, and gastrointestinal disturbances. To qualify for this quintessentially polysymptomatic diagnosis, a female patient had to exhibit at least fourteen and a male at least twelve symptoms.³¹⁷ The *DSM-III* remained pointedly tacit about the somatisation disorder’s potential aetiology or its relation to psychological factors, thus placing the diagnostic focus exclusively on symptom counting.

Hence, it can be said that the *DSM-III* not only upheld but also considerably amplified the division of hysteria into mental and somatic manifestations, which the previous editions had instituted. Yet, as my analysis has aimed to show, the *DSM-III* appeared to struggle in particular with reconciling the somatic manifestations of hysteria with their presumable psychogenic causation. Within the previously dominant psychoanalytic framework, the Freudian notion of conversion with its implicit mind-body dualism had enjoyed an almost axiomatic character. Psychoanalysis thus avoided posing the question as to how exactly psychological factors could traverse the chasm between the mind and the body to give rise to physical symptoms.³¹⁸ Yet, as mentioned previously, with the *DSM-III*, psychiatric disorders started to be increasingly modelled in reference to physical diseases.

In this new, biologically informed frame of reference, the presumed psychogenic causation of hysteria’s psychological symptoms did not appear to present a problem. Consequently, we have seen that the psychological symptoms of hysteria, all of which were classified within the group of dissociative disorders, have remained relatively stable nosological constructs across various *DSM* updates. But this was not the case with the physical symptoms of hysteria. Without any empirical proof to support the

stemmed from a different context. The *DSM-III* adopted it from psychosomatic medicine, where, by the late 1960s, it was already regarded as a “semantic muddle.” Lipowski, “Consultation Psychiatry,” 413. In an attempt to curtail its semantic ambiguity, the psychiatrist Lipowski defined somatisation as “the tendency to experience, conceptualize, and/or communicate psychological states or contents as bodily sensations, functional changes, or somatic metaphors.” Lipowski, 413 (emphasis in original). Lipowski insisted that the term somatisation should be used only on “a descriptive basis until psychological and physiological mechanisms can be worked out” for its symptoms. Lipowski, 413. It is such a descriptive approach that the *DSM-III* adopted by merging Briquet’s syndrome and somatisation into a newly fashioned diagnostic entity of somatisation disorder.

313 APA, *DSM-III*, 241.

314 APA, 241.

315 APA, 241.

316 APA, 243–44.

317 APA, 243.

318 As discussed in section 2.1.3, Freud remained vague on this point.

Freudian concept of conversion or a consistent theory to explain how it came about, the existence of a speculative psychological mechanism through which emotionally charged experiences were transformed into somatic phenomena became contested.³¹⁹ As discussed above, the *DSM-III* approached this problem by downplaying the role of psychogenic factors in conversion disorder and by introducing a newly constructed diagnostic entity of somatisation disorder.

As a result of the *DSM-III*'s conceptual reframing, somatic expressions of hysteria, which Freud had already decoupled from both anatomy and physiology, now also became partially detached from the psyche. However, the application of the symptom-based approach to hysteria proved to be a double-edged sword since physical manifestations of this disorder appeared to be unexplainable without recourse to psychological constructs. The attenuation of the putative psychological causation placed once more centre stage the symptoms' paradoxical physical characteristics that had baffled physicians for centuries. The renewed focus on physical symptoms made it clear that the existing state of medical knowledge could not offer an alternative explanatory model for hysteria's vague, multiple, and confusing manifestations. As explicitly stated in the *DSM-III*, "[a]lthough the symptoms of Somatoform Disorders are 'physical,' the specific pathophysiological processes involved are not demonstrable or understandable... For that reason, these disorders are not classified as 'physical disorders.'"³²⁰ Hence, the "essential feature" of somatoform disorders in the *DSM-III* became the presence of "physical symptoms suggesting physical disorder," but for which "no demonstrable organic findings or known physiological mechanisms" could be found.³²¹ The somatic symptoms previously attributed to hysteria were thus explicitly declared to be medically unexplainable phenomena.

To sum up, despite the deletion of the term 'hysteria' from the official medical nosology, the *DSM-III* never proclaimed hysterical symptoms non-existent. Yet, we have seen that the manual's purportedly atheoretical framework failed to accommodate somatic symptoms of hysteria. In the new framework, these symptoms appeared to defy not only sound logic but also the entire medical knowledge. Unable to account for them, the *DSM-III* loosely and somewhat randomly grouped these symptoms into newly defined disorders, which not only partly overlapped but also lacked any diagnostic specificity. As a result, the defining characteristics of conversion and somatisation disorders became the fundamentally paradoxical nature of their clinical manifestations. The highly heterogeneous symptoms of these disorders were no longer regarded as entirely attributable to psychological factors. But rather inconveniently, they turned out to be even less explainable either in relation to clearly delineated medical conditions or in terms of any known physiological mechanisms. It is thus no exaggeration to say that

319 See, e.g., Lipowski, "Consultation Psychiatry," 401–2, 412–13. See also Guze and Perley, "History of Hysteria," 960.

320 APA, *DSM-III*, 241.

321 APA, 241.

the intermedial transcription of hysteria undertaken by the *DSM-III* had no positive consequences for the medical understanding of this disorder.³²²

2.2.2 Diagnostic Elusiveness of Somatic Symptoms of Hysteria

As we will discuss in detail in this section, the uncertainty about how to define the nature of various somatic symptoms of hysteria has been accompanied and considerably compounded by the growing insecurity about how to diagnose them reliably. In fact, I intend to show that these two processes were mutually and dynamically related. I will argue that the reconceptualisation of hysteria analysed above has led to the increasing uncertainty about the epistemic adequacy of the diagnostic tools that had thus far been used and the growing fear of potential misdiagnosis. We will see that, due to this development, hysterical symptoms came to be regarded not only as medically unexplainable but also as essentially undiagnosable.

In the closing decades of the twentieth century, parallel to the waning influence of Freud's theoretical views on hysteria, his methodological approach to diagnosing this disorder was also submitted to increasingly fierce criticism.³²³ As discussed previously, Freud used language to access and narratively reconstruct a chain of the repressed traumatic memories, which, as he argued, caused the development of each patient's idiosyncratic hysterical symptoms. However, a rising number of critics started to contend that instead of listening to his patients, Freud had coerced them into fabricating narratives compatible with his theories of hysteria.³²⁴ Freud came to be characterised as "a bullying interrogator," who forced "reminiscences on his patients, eliciting confabulations rather than actual memories."³²⁵ As a consequence of this re-evaluation, Freud's claim that hysterical symptoms represented a symbolic resolution of repressed traumatic memories started to lose credibility. This, in turn, led to further marginalisation of the diagnostic relevance of the patients' prior life events in clinical practice, which *DSM-III* had already set in motion.³²⁶

Apart from the criticism pointed at Freud, various authors also started to raise more general questions about the adequacy of language for diagnosing hysteria. These concerns arose from the changing notions of what counted as a valid psychiatric diagnosis, which, since the 1970s, became increasingly grounded in the use of quantitative empirical methods. For instance, as early as 1972, Feigner et al. influentially emphasised the diagnostic importance of laboratory findings, which they declared to be "generally more reliable, precise, and reproducible than are clinical descriptions."³²⁷ In this new context, the patients' recounting of their past life events came to be viewed as

322 I am using the term intermedial transcription in Jäger's sense. Jäger, "Transcriptivity Matters," 53.

323 For a particularly scathing criticism of Freud, see Webster, *Why Freud Was Wrong*. See also Borch-Jacobsen, *Making Minds and Madness*, 9–13, 37–63, 141–82; and Szasz, *Myth of Mental Illness*, 70–79.

324 See, e.g., Borch-Jacobsen, *Making Minds and Madness*, 12–13. For a succinct overview of such views, see Showalter, *Hystories*, 40–43.

325 Showalter, *Hystories*, 42.

326 See APA, *DSM-IV*, 453–54, 457. I will return to this point later in the chapter.

327 Feigner et al., "Diagnostic Criteria," 57. According to Feigner et al., included "among laboratory studies are chemical, physiological, radiological, and anatomical (biopsy and autopsy) findings.

potentially biased, unverifiable, and, in effect, unreliable.³²⁸ This shift in attitude was stated in no uncertain terms in the fourth edition of the *DSM*. The *DSM-IV* explicitly warned the physician faced with a potential diagnosis of conversion disorder to avoid “undue reliance on [patients’] subjective complaints.”³²⁹ Instead, the physician was advised to supplement and cross-reference each patient’s potentially unreliable self-report of stressful events with “additional sources of information (from associates or records).”³³⁰

Moreover, this growing distrust of patients’ subjective accounts of their illness was combined with the doctors’ growing unwillingness to engage in an interpretation of the potential relevance that stressful events might have had in triggering the onset of hysterical symptoms. A frequently raised objection was that psychological factors were common in many psychiatric conditions and thus not specific to hysteria. Therefore, even if established, a temporal association between a particular traumatic event and the onset of the hysterical symptom could be purely coincidental and, as such, meaningless.³³¹ I suggest that due to the increasing dismissal of the Freudian interpretational framework, which had endowed them with a symbolic value, the patients’ life events suddenly appeared too variable and idiosyncratic to be unambiguously related to the symptoms.

The already difficult situation was further complicated because many patients, believing that they were suffering from an organic illness, avoided psychiatrists and insistently sought advice from general practitioners or non-psychiatric specialists.³³² However, non-psychiatrists felt even less equipped to deal with the potential role of psychological factors in the development of hysteria’s puzzling symptoms.³³³ In fact, both in the psychiatric and non-psychiatric contexts, the reliance on language as a diagnostic tool for discovering specific psychological stressors that were possibly aetiologically related to the symptom came to be regarded as a hindrance to a reliable diagnosis. In a curious parallel to Charcot, doctors once again became reluctant to diagnose their patients by listening to them and instead turned to observing and measuring their bodies.

This renewed focus on the hysteria patients’ bodies was additionally bolstered through crucial changes in the official diagnostic criteria of hysteria’s nosological successors. Starting with the *DSM-II*, the diagnosis of hysteria’s somatic manifestations required their clear-cut clinical differentiation from similar physical symptoms caused by a detectable neurological lesion.³³⁴ In effect, through the introduction of this

Certain psychological tests, when shown to be reliable and reproducible, may also be considered laboratory studies in this context.” *Ibid.* See also *ibid.*, 57–61.

328 See, e.g., Craig, “Life Events,” 89.

329 APA, *DSM-IV*, 448.

330 APA, 454.

331 See, e.g., Hallett, “Crisis for Neurology,” 269.

332 See, e.g., Wileman, May, and Chew-Graham, “Medically Unexplained Symptoms,” 181–82.

333 Wileman, May, and Chew-Graham, 182.

334 APA, *DSM-II*, 40.

criterion, hysteria once again became a differential diagnosis of exclusion.³³⁵ Yet, proving that the symptoms were not caused by an organic lesion of the nervous system necessitated a thorough neurological assessment. This, in turn, meant that psychiatrists could no longer diagnose hysteria on their own. In other words, the diagnosis of exclusion had to be performed by a neurologist. Furthermore, by the time the *DSM-IV* was published in 1994, the requisite diagnostic evaluation was additionally expanded to include a “careful review of the current [symptom] presentation, the overall medical history, neurological and general physical examinations, and appropriate laboratory studies.”³³⁶ But paradoxically, such an elaborate medical assessment aimed to prove that the patient was actually physically healthy. Specifically, two key aspects that served to support the diagnosis of hysteria’s contemporary successors were, first, the absence of positive findings on laboratory tests and, second, a confirmation that the somatic symptoms were incongruent with known anatomical pathways.³³⁷ Both aspects were regarded to confirm that hysterical symptoms lacked any organic basis.

However, these seemingly simple diagnostic requirements turned out to be difficult to fulfil in actual clinical practice. As medically unexplained phenomena in the strong sense of this term, hysteria’s nosological successors were defined entirely in negative terms—their diagnostic descriptions focused not on what they were but only on what they were not.³³⁸ As a result, there was no specific laboratory measurement or a viable technology on which a doctor could rely to diagnose hysteria unambiguously. Instead, the doctor was required to perform a diagnosis using “appropriate investigation” to provide sufficient evidence that the symptoms could not be attributed to any other neurological disease or a general medical condition.³³⁹ It can thus be argued that the purpose of such investigation was to impart the impression of medical validity to the diagnosis of hysteria by grounding the somatic symptoms’ apparent lack of organic basis in “objective findings” delivered by laboratory tests.³⁴⁰ But the major problem was that what comprised ‘appropriate investigation’ remained an open question since the *DSM* never defined a cut-off point or provided any official guidelines. Decisions such as what to measure, with which technology, and when to stop were left to the discretion of the diagnosing physician. Consequently, these decisions varied considerably in the actual clinical practice, depending on the doctor’s level of training and experience, the type of medical speciality, and even the country of residence.³⁴¹ Therefore, I suggest that far from offering an eagerly sought-after solution to curbing hysteria’s elusiveness,

335 For Charcot’s initial reliance on the differential diagnosis of exclusion, see, e.g., Charcot: “Lecture 12: Hysterical Contracture”; and Charcot, Lecture 20: Brachial Monoplegia.” See also section 1.3.1.

336 APA, *DSM-IV*, 456.

337 APA, 455.

338 See APA, *DSM-III*, 241–47; and APA, *DSM-IV*, 448, 452–54.

339 APA, *DSM-IV*, 457.

340 APA, 448. Notably, the situation I am describing here was reminiscent of the problems with which nineteenth-century physicians grappled before Charcot introduced the visual diagnostic tools discussed in section 1.3.1. As I have argued in that section, by using images, Charcot was able to redefine the diagnosis of hysteria in positive terms. However, we have also seen that Freud discarded such use of images through his psychogenic reconceptualisation of hysteria.

341 See, e.g., Espay et al., “Opinions and Clinical Practices,” 1366.

laboratory tests introduced an additional diagnostic variable that proved challenging to control.

To make matters even more complicated, in 1994, the *DSM-IV* introduced yet another diagnostic criterion. Contrary to the previous editions, which insisted on a straightforward exclusion of physical diseases, the *DSM-IV* explicitly acknowledged that somatoform disorders could often co-occur with other neurological and general medical conditions.³⁴² This meant that even if the clinical examination or laboratory tests did reveal the presence of an organic illness, such findings did not necessarily preclude the additional diagnosis of hysteria's nosological successors. In such cases, the diagnosis of hysteria was still warranted if the doctor concluded that the somatic symptom in question was too excessive to be entirely attributed to the organic illness or explained by the laboratory findings.³⁴³ In fact, this 'new' criterion only reaffirmed historical accounts according to which hysterical symptoms were often accompanied by other mental and physical disorders.³⁴⁴ Yet, the introduction of this criterion further contributed to the growing impression that hysteria's nosological successors were veritable "diagnostic puzzles," which in actual clinical practice were almost impossible to solve.³⁴⁵

The diagnostic uncertainty was additionally aggravated by the perennial fear of misdiagnosis. In particular, this fear has kept haunting all hysteria's nosological incarnations ever since Eliot Slater's influential study "Diagnosis of 'Hysteria'" was published in 1965.³⁴⁶ In this study, Slater severely criticised hysteria's diagnosis of exclusion, arguing that it was impossible "to build up a picture of an illness out of elements which are severally the evidence of absence of illness."³⁴⁷ Slater argued that by diagnosing their patients with hysteria, the physicians effectively left them undiagnosed. To prove his point, Slater summarised the results of a follow-up study he and a colleague performed in 1962 by re-examining eighty-five patients who had initially been diagnosed with hysteria at the National Hospital in London in 1951, 1953, and 1955.³⁴⁸ Based on the analysis of the follow-up data, Slater concluded that in about a third of the patients in his sample, the physical symptoms had been mistakenly attributed to hysteria, thus leaving serious organic diseases unrecognised.³⁴⁹ Due to

342 APA, *DSM-IV*, 450, 453.

343 APA, 453, 455.

344 See the previous chapter.

345 Mayou, "Medically Unexplained Physical Symptoms," 534.

346 Slater, "Diagnosis of 'Hysteria.'"

347 Slater, 1396.

348 Slater, 1397–98.

349 Slater's narrative regarding both the actual frequency of misdiagnosis and the presence of demonstrable organic illness at the follow-up is difficult to follow and, at times, confusing. His study ends with a statement that only about 40% of altogether eighty-five patients who had initially received the diagnosis of hysteria remained without any diagnosable organic disease at the follow-up. Slater, 1397–98. Some of Slater's readers have erroneously taken this statement to mean that the remaining 60% of the patients had been mistakenly diagnosed with hysteria. As a result, Slater is often misquoted in the medical literature as having proven a misdiagnosis rate of hysteria that is considerably above 50%. See, e.g., Crimlisk et al., "Slater Revisited," 582; Allin, Streeruwitz, and Curtis, "Understanding Conversion Disorder," 207. However, through a close

this high misdiagnosis rate, several patients had died by 1962 from untreated organic illnesses. In the forcefully formulated conclusion, Slater called hysteria a dangerous myth, “a disguise for ignorance and a fertile source of clinical error.”³⁵⁰ Moreover, he declared hysteria to be “not only a delusion but also a snare.”³⁵¹

Over the following decades, multiple follow-up studies have attempted to attenuate the damage Slater had inflicted on the credibility of hysteria as a diagnosis. By analysing new data, various authors have strived to demonstrate that the rate with which organic diseases were either overlooked or misdiagnosed as hysteria was significantly lower than suggested initially.³⁵² According to such systematic reviews, the misdiagnosis of hysteria’s nosological successors since the 1970s has been at a consistent level of 4% on average, which is comparable to other neurological and psychiatric disorders.³⁵³ Nevertheless, the doubt apparently lingered. Perhaps the most telling indication of the lingering doubt is that in 1994, the authors of the *DSM-IV* still felt the need to explicitly refute the claims of high misdiagnosis rates of hysteria, which Slater had made almost thirty years earlier.³⁵⁴

To conclude my analysis in this section, I argue that even if misdiagnosis ceased to be an issue by the early 1990s, a more substantial problem regarding the diagnosis of hysteria prevailed. We have seen that having been defined only through the absence of known diseases, hysteria’s nosological successors lacked even a single diagnostic criterion of inclusion. Defined in such terms, the somatic symptoms of hysteria were not only medically unexplained but also essentially unmeasurable and thus only indirectly diagnosable. Hysteria was effectively reduced to a puzzling leftover that remained after all other medically diagnosable disorders were excluded. Yet, the process of exclusion in itself proved problematic because, in each clinical case, the physician had to reach an essentially arbitrary decision when to stop looking for other possible organic

reading of Slater’s study, I have counted twenty-eight misdiagnosed patients out of eighty-five. This amounts to a misdiagnosis rate of approximately 33%. The rest of the patients received a combined diagnosis of both hysteria and an additional organic disorder. The discrepancy arose because most of these patients no longer suffered from hysteria at the follow-up, whereas their organic disorders persisted. See Slater, “Diagnosis of ‘Hysteria,’” 1398–99.

350 Slater, “Diagnosis of ‘Hysteria,’” 1399.

351 Slater, 1399. Slater’s claim echoed the criticism that had been repeatedly levelled at hysteria throughout its long history. For example, shortly before Charcot launched his image-based research aimed at proving that hysteria was a genuine illness, his older colleague Charles Lasegue famously disagreed. Lasegue contended that hysteria was a wastebasket diagnosis for otherwise unexplained symptoms. See Goldstein, *Console and Classify*, 324. For even older instances of such criticism, see Showalter, *Hystories*, 15–16.

352 For a succinct overview of follow-up studies of hysteria since 1965, see Stone et al., “Review of Misdiagnosis.” Stone et al. have suggested that Slater had, in fact, overestimated the rate of hysteria’s misdiagnosis during the 1950s due to “the poor methods.” *Ibid.*, 5, article 989. See also Guze et al., “Follow-Up.”

353 See Stone et al., “Review of Misdiagnosis,” 1, article 989.

354 Without explicitly mentioning Slater, the *DSM-IV* referred to, by that point, the almost mythical 50% misdiagnosis rate of hysteria. APA, *DSM-IV*, 453.

disorders. Furthermore, even after excluding potential organic causes, the remaining symptoms were still not unambiguously categorisable. The additional problem was that no laboratory tests could reliably differentiate between actual hysterical symptoms and a host of other vaguely understood and medically unexplained phenomena.³⁵⁵ In short, in the last quarter of the twentieth century, hysteria became so fuzzy and elusive as to appear increasingly unreal. As we are about to see in the following section, the growing doubt in the physical reality of its somatic manifestations made hysteria an exceedingly unpopular medical diagnosis in all its nosological updates.

2.2.3 Increasing Medical Invisibility of the 'Problematic Patient'

So far, we have discussed the substantial transformations that hysteria as a medical entity underwent in the second half of the twentieth century and the formal diagnostic challenges that arose as a consequence. In this section, we will examine how the refocusing of medical attention on somatic manifestations of hysteria while at the same time defining them in purely negative terms shaped the diagnostic encounter between doctors and patients. On the one hand, I will analyse how the diagnostic transformations discussed above have led to a revival of the doctors' perennial suspicion that hysteria patients were merely simulating their symptoms instead of suffering from a genuine disorder. On the other hand, I will also argue that the late-twentieth-century patients' reluctance to accept what they perceived as an offensive diagnosis additionally contributed to turning hysteria into an increasingly invisible disorder in the medical context.

As long as the understanding of hysteria remained framed within Freudian psychoanalytic terms, the possibility that patients were simulating their symptoms was not accorded any clinical significance.³⁵⁶ Having placed the symbolic meaning of hysterical symptoms centre stage, Freud had skilfully circumvented the uncomfortable question of their potential physical reality. What mattered was not the somatic nature of the symptoms but the psychological content for which they stood. However, as we have seen, with the waning influence of Freud's symbolic interpretation, the diagnostic focus of hysteria shifted back towards the symptom-based clinical picture. In this new context, the question of hysterical symptoms' physical 'reality' resurfaced once more

355 In the late 1990s, it became a matter of heated debate if hysteria's nosological successors were conceptually and diagnostically distinguishable from a range of possibly related clinical conditions that were equally characterised by the lack of any demonstrable physical abnormality. Jointly referred to as functional somatic syndromes, these conditions include multiple chemical sensitivity, sick building syndrome, chronic fatigue syndrome, fibromyalgia, irritable bowel syndrome, chronic whiplash, chronic Lyme disease, the Gulf War syndrome, food allergies, hypoglycaemia. To this date, the delineation between present-day forms of hysteria and other functional somatic syndromes remains unresolved. For discussions of the relation of these syndromes to contemporary manifestations of hysteria, see Barsky and Borus, "Functional Somatic Syndromes"; Fink, Rosendal, and Olesen, "Classification of Somatization"; Fink et al., "Syndromes of Bodily Distress"; Kroenke, Sharpe, and Sykes, "Classification of Somatoform Disorders"; and Wessely, Nimnuan, and Sharpe, "Functional Somatic Syndromes."

356 See, e.g., APA, *DSM-I*, 31–33.

as a major epistemic concern.³⁵⁷ Consequently, it was already in 1968 that the *DSM-II* introduced as one of the diagnostic requirements the need to differentiate between ‘genuine’ and feigned somatic symptoms of hysteria.³⁵⁸ By the time the *DSM-IV* was published almost thirty years later, this requirement had advanced into one of the key diagnostic criteria.³⁵⁹

But, in actual practice, meeting this requirement proved to be particularly problematic, thus adding yet another obstacle to an already challenging diagnosis. The major hurdle turned out to be the diagnostic features of ‘genuine’ hysterical symptoms that lacked specificity and rested entirely on the exclusion of known organic diseases. As a result, no physical measurements or laboratory tests existed that a physician could deploy to distinguish between a ‘real’ and a ‘simulated’ hysterical symptom.³⁶⁰ In other words, not only were there no designated tests for ‘objectively’ establishing the presence of ‘genuine’ hysterical symptoms. There were also no tests that could be used to exclude feigning. As explained by one doctor, in the context of general medicine, to simulate an organic illness, an individual has to deploy a physical method that typically leaves “an evidence trail ([for example,] the culturing of faecal bacteria from a wound that will not heal).”³⁶¹ However, to simulate hysterical symptoms, “all the patient needs is a flair for the theatrical—and consequently the means of its detection is limited.”³⁶²

Hence, somewhat paradoxically, to prove the ‘reality’ of the hysterical symptom, the physician was expected to demonstrate the patient’s “lack of conscious intent” in producing it.³⁶³ This, in turn, meant that, unless they were able to either elicit an outright confession or catch a patient in the act of feigning, physicians had to make subjective inferences about their patients’ putative intentions. Whether they decided that a particular patient was genuinely sick or merely pretending to be sick, physicians could not provide any ‘objective’ evidence for their assessment.

What complicated the situation even further was that the *DSM-III* introduced and the *DSM-IV* retained an additional diagnostic distinction by splitting feigning into two separate categories.³⁶⁴ The two new categories were malingering and factitious disorder. In both cases, the symptoms were judged to be intentionally produced. But malingering was understood to be motivated by external “goals such as financial compensation, avoidance of duty, evasion of criminal prosecution, or obtaining drugs.”³⁶⁵ Strictly speaking, malingering was declared a form of deception consciously performed by an essentially healthy individual. By contrast, the factitious disorder was

357 In chapter 1, I discussed how the question of simulation represented one of the major clinical and epistemic concerns in the Salpêtrian hysteria research, which Charcot attempted to solve through the targeted use of images as diagnostic tools. See, in particular, section 1.2.1.

358 APA, *DSM-II*, 40. Interestingly, this requirement did not apply to psychological symptoms of hysteria. See *ibid.*

359 APA, *DSM-IV*, 450, 452.

360 Kanaan, “Functional or Feigned,” 15–16.

361 Kanaan, 15.

362 Kanaan, 15.

363 APA, *DSM-IV*, 455.

364 APA, *DSM-III*, 246; and APA, *DSM-IV*, 457.

365 APA, *DSM-IV*, 457.

defined as a psychiatric condition that arose entirely from a pathological psychological need to assume the sick role and, therefore, lacked any discernible external motives.³⁶⁶ According to the *DSM-IV*, to diagnose hysteria's nosological successors, doctors had to exclude both malingering and factitious disorder.³⁶⁷ Thus, apart from having to infer if the patients were simulating their symptoms, doctors were now also required to make judgments about the patients' underlying motives, "especially relative to potential external rewards or the assumption of the sick role."³⁶⁸

Inadvertently, these additional diagnostic specifications put the diagnosis of hysteria on even shakier grounds since many doctors had difficulties fulfilling them in the clinical setting.³⁶⁹ Unable to unambiguously and reliably delineate 'genuine' medically unexplained somatic manifestations of hysteria from those that were purportedly intentionally feigned, doctors became increasingly distrustful of patients who exhibited these puzzling symptoms. As a result, many doctors came to believe that although hysteria patients were not necessarily intentionally simulating their illness, they suffered from purely imaginary symptoms, which were physically "impossible."³⁷⁰ Put differently, the unspoken implication was that hysteria patients unintentionally deceived both themselves and their doctors by genuinely believing to have symptoms that they could not possibly have. By contrast, other medical professionals went so far as to deny the existence of hysteria as a medical condition and attributed all of its physical manifestations to patients' wilful deception.³⁷¹

Moreover, it appears to me that the doctors' distrust of their patients was further reinforced by how the *DSM-IV* described individuals who merited the diagnosis of hysteria's nosological successors. Reflecting further shifts in the conceptualisation of hysteria, the *DSM-IV* emphasised the diagnostic significance of the patients' purported 'abnormal illness behaviour.'³⁷² In a somewhat derogatory tone, the *DSM-IV* stated that individuals with hysterical symptoms usually expressed "their complaints in colorful, exaggerated terms," and led lives that were "as chaotic and complicated as their medical histories."³⁷³ Additionally, the *DSM-IV* declared that "antisocial behavior, suicide threats and attempts, and marital discord" were not uncommon in such

366 "Whereas an act of malingering may, under certain circumstances, be considered adaptive, by definition a diagnosis of a Factitious Disorder always implies psychopathology, most often a severe personality disturbance." APA, *DSM-III*, 285.

367 APA, *DSM-IV*, 457.

368 APA, 454.

369 Kannan et al., "In the Psychiatrist's Chair," 2893.

370 Kannan et al., 2894.

371 Kannan et al., 2893; Kannan, Armstrong, and Wessely, "Limits to Truth-Telling," 299; and Stone, Carson, and Sharpe, "Assessment and Diagnosis," i3.

372 In 1969, psychiatrist Issy Pilowsky introduced the term 'abnormal illness behaviour' to designate those patients who complain of physical symptoms and "remain uninfluenced by the doctor's explanation" that due to the absence of a detectable "objective pathology," they were not entitled to be placed in the type of sick role as they had expected. Pilowsky, "Abnormal Illness Behaviour," 349. Pilowsky expressly developed this concept in an attempt to solve "the controversy over the use of terms such as hysteria, hypochondriasis and neurasthenia." *Ibid.*, 350.

373 APA, *DSM-IV*, 446.

individuals.³⁷⁴ The patients were further characterised as impulsive, overemotional, suggestible, tending towards dependency and the adoption of a sick role, and behaving in a dramatic and histrionic fashion.³⁷⁵ This description was uncannily reminiscent of the nineteenth-century views of hysteria patients as untrustworthy, deceitful, troublesome, and attention-seeking.³⁷⁶ Thus, hysteria patients once again came to be perceived not only as challenging to diagnose due to their ambiguous symptoms but also as “more difficult to treat” because of their supposedly manipulative character traits and “abnormal behaviour.”³⁷⁷ As a result, physicians were increasingly reluctant to deal with such purportedly problematic patients and reacted to them “through referral or avoidance.”³⁷⁸

On the other end of the spectrum, protracted and ambiguous diagnostic encounters proved even more frustrating for patients than for doctors. However, as opposed to their nineteenth-century counterparts, late-twentieth-century patients no longer accepted the position of passive recipients of medical diagnoses.³⁷⁹ Many patients felt offended by the diagnosis of hysteria, even when the physicians used seemingly more neutral nosological variations—such as conversion, somatisation, and somatoform disorders—or described the symptoms less specifically as psychogenic or medically unexplained.³⁸⁰ Hence, it seems to me that the actual problem was more profound than the choice of particular terminology. Instead, most patients were under the impression that, regardless of what particular label the doctors used, their chronic and often debilitating somatic symptoms were implicitly regarded as ‘unreal,’ ‘all in the head,’ and ‘imaginary.’³⁸¹ Put simply, patients felt doubted and denied the reality of their medical problems. And even if their medical problems were acknowledged, patients were often blamed for the symptoms, which were dismissively attributed to their purportedly ‘abnormal illness behaviour.’³⁸²

Most patients were additionally troubled by the lack of clear-cut medical explanations for their symptoms, and even more so by the absence of treatment options apart from psychotherapy.³⁸³ Many were also unwilling to comply with a diagnosis that categorised them as having a psychiatric disorder, which they perceived as socially stigmatising.³⁸⁴ Convinced that they were suffering from ‘real’ physical symptoms,

374 APA, 446.

375 APA, 454.

376 For Freud’s uncannily similar description of the nineteenth-century doctors’ distrustful attitudes towards hysteria patients, see Freud, “Five Lectures,” 10–12.

377 Kanaan et al., “In the Psychiatrist’s Chair,” 2891–92. The literature on this topic abounds. See, e.g., Deighton, “Problem Patients”; Groves, “Hateful Patient”; Hahn et al., “Difficult Doctor-Patient Relation”; and Lin et al., “Frustrating Patients.”

378 Epstein, Quill, and McWhinney, “Somatization Reconsidered,” 218–19.

379 Mayou et al., “Somatoform Disorders,” 848.

380 Stone et al., “What Should We Say to Patients,” 1449–50.

381 Stone et al., 1449–50; and Richardson and Engel, “Evaluation and Management,” 21, 23.

382 See, e.g., Salmon, Peters, and Stanley, “Patients’ Perceptions,” 373–74.

383 Hallett, “Crisis for Neurology,” 270.

384 Richardson and Engel, “Evaluation and Management,” 28. For a more general account of mental illness stigma, see Byrne, “Psychiatric Stigma”; and Byrne, “Stigma of Mental Illness.” As even doctors admitted, it “is hard to escape the strongly prevalent public attitudes that psychological

these individuals either went from one specialist to another in search of a more satisfying medical explanation or remained undiagnosed due to a breakdown in the relationship with their doctors.³⁸⁵ Feeling even more challenged by such ‘problematic’ patients, doctors came to regard the diagnosis of contemporary forms of hysteria almost as “difficult to communicate as a terminal illness.”³⁸⁶ As a result, they became even more avoidant in making it.

In summary, my analysis in this and the previous two sections has shown that hysteria once again became a medically unexplainable disorder in the last quarter of the twentieth century. Detached from any clear psychological causation and defined by an array of its puzzling somatic symptoms that lacked an apparent physical basis, hysteria appeared ‘unreal’ and ‘impossible’ to doctors. As a result, both doctors and patients started to shun this diagnosis in all its official nosological transformations and alternative unofficial designations. Regardless of whether they were referred to as hysterical, somatoform, conversion, functional, psychosomatic, psychogenic, non-organic, stress-related, or medically unexplained, the symptoms became essentially invisible in the medical context.

But despite the lack of medical interest in them, it seems that the baffling hysterical symptoms have never disappeared. Instead, multiple epidemiological studies from the last few decades have gathered empirical data on the prevalence of hysterical symptoms in present-day clinics. According to such studies, somatic symptoms of hysteria have remained just as frequent in contemporary medical practices as they had been during Charcot’s time.³⁸⁷ Specifically, several studies conducted in Europe and North America have reported that the incidence of different hysterical symptoms in new neurological patients ranges from 5% to 42%.³⁸⁸ Additional studies have shown that hysterical symptoms are not limited to neurological clinics but represent “a common problem across general medicine.”³⁸⁹ The same studies have also suggested that the apparent invisibility of hysteria within the medical contexts was at least to some extent perpetuated by the fact that patients were often dismissed without being given a

difficulties are something minor or ‘not real’ and usually signify a distinct lack of moral fibre.” Edwards, Stone, and Lang, “Change the Name,” 850.

385 “If there is any reason for doctor-patient mistrust, the relationship can quickly become outwardly adversarial and result in mutual rejection.” Richardson and Engel, “Evaluation and Management,” 18.

386 Kannan, Armstrong, and Wessely, “Limits to Truth-Telling,” 300.

387 Stone et al., “Disappearance,” 12–13.

388 See, e.g., Agaki and House, “Epidemiology”; Carson et al., “Outcome”; Carson et al., “Symptoms Matter”; Factor, Podskalny, and Molho, “Psychogenic Movement Disorders”; Fink, Hansen, and Søndergaard, “First-Time Referrals”; and Lempert et al., “Frequency.” Considerable discrepancies in the estimated incidence of hysterical symptoms between various epidemiological studies reflect the problem of definition regarding these symptoms. Whereas some authors have focused only on cases that fulfilled the diagnostic criteria of conversion disorder in line with the current version of the DSM, others have operated with a much broader category of medically unexplained symptoms.

389 Nimnuan, Hotopf, and Wessely, “Epidemiological Study,” 361. See also Lazare, “Conversion Symptoms.”

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.