applicability of images as research tools. So far, this aspect of hysteria research has been neglected in the humanities.

This chapter has the following structure. In the first part, I chart the gradual dismissal of images as investigation tools by linking it to the development of psychological theories of hysteria's aetiology in the late nineteenth and early twentieth centuries. The second part of the chapter is dedicated to discussing the subsequent division, relabelling, and the putative disappearance of hysteria in the second half of the twentieth century. In the third part, I analyse the circumstances that made the gradual reappearance of the image-based hysteria research possible. Finally, the closing part of the chapter examines how the current neuroimaging hysteria research legitimises the somatic framework that has given rise to it.

2.1 Gradual Dismissal of Images as Epistemic Tools From Hysteria Research

The demise of Charcot's image-based hysteria research at the end of the nineteenth and beginning of the twentieth centuries has been widely discussed in the humanities.[18] Across different accounts, this demise has been consistently framed in celebratory terms as a sign of scientific progress.[19] The dominant interpretation is that Freud rectified Charcot's mistakes. He achieved this by turning his "attention away from the seduction of the image" and the "empirically self-evident" external manifestations of hysteria.[20] More specifically, we are told that due to the insights gained during his four-month internship under Charcot in 1885 and 1886, Freud later challenged the epistemic validity of the visual evidence fabricated at the Salpetrière.[21] Reacting to Charcot, Freud rejected the images, whose creation had relied on the elaborate staging of the hysteria patients' bodies, and turned to the use of language. In doing so, Freud moved away "from the crudity of seeing to the subtlety of hearing."[22]

In what follows, I will suggest an alternative interpretation that does not ascribe the disappearance of image-based hysteria research to a single individual. Instead, drawing on Jäger's theory of transcriptivity, I will show that the loss of the epistemic functions of images in hysteria research was a gradual process inextricably linked to a cumulative shift in the conceptualisation of this disorder. We will see that first hypnosis and then hysteria ceased to be viewed as physiologically determined neurological conditions and became reconceptualised as subjective, highly individualised psychological phenomena. Importantly, I will claim that this shift was not induced by Freud alone. In particular,

18 See, e.g., Harrington, *Cure Within*, 59–60; Shorter, *From Paralysis to Fatigue*, 196–200; and Scull, *Hysteria*, 129–30.

19 See, e.g., Didi-Huberman, *Invention of Hysteria*, 278–9; Rose, *Field of Vision*, 38; and Showalter, *Female Malady*, 147–58.

20 Rose, *Field of Vision*, 97, 114. See also Didi-Huberman, *Invention of Hysteria*, 80; Gilman, *Seeing the Insane*, 200–4; and Showalter, *Female Malady*, 154–55.

21 See Didi-Huberman, *Invention of Hysteria*, 80, 279; Gilman, *Seeing the Insane*, 204; and Rose, *Field of Vision*, 96–7.

22 Gilman, "Image of the Hysteric," 415.

I will foreground the crucial contributions of Freud's two contemporaries, Hippolyte Bernheim and Pierre Janet. Further, I will argue that, as the new conceptual framework began to crystallise, various images, which Charcot had used as epistemic tools in his hysteria research, were successively rendered both meaningless and useless from the medical perspective. To demonstrate this claim, in the following three sections, I will trace how images as epistemic tools gradually disappeared from hysteria research. First, I will discuss how Hippolyte Bernheim challenged the Salpêtrian views on hypnosis and its links to hysteria. In the subsequent two sections, I will analyse the two competing psychological conceptions of hysteria developed by Charcot's most prominent pupils, Pierre Janet and Sigmund Freud. In my analysis, I will avoid making normative statements or taking sides with individual researchers. Rather, I will examine the broader epistemic contexts within which each of these three researchers operated.

2.1.1 Bernheim: Hypnosis as an Unvisualisable Psychological Phenomenon

The initial major challenge against Charcot's research was launched in the mid-1880s by Hippolyte Bernheim, a professor of medicine at the University of Nancy.[23] Bernheim's outright criticism primarily addressed Charcot's use of hypnosis. Nevertheless, it also inevitably affected Charcot's image-based findings on hysteria, many of which, as we have discussed previously, had been derived from the experimental application of hypnosis. The rivalry between the Salpêtrière and Nancy schools of hypnosis continued until the 1890s, attracting attention both within and beyond purely scientific circles.[24] Consequently, numerous historical and contemporary studies have analysed this famous battle of opinions from which, according to most interpretations, Bernheim had emerged as the winner.[25] The consensus is that Bernheim exposed the Salpêtrian hysteria research as "an elaborate theatre of illusions" in which the hypnotised patients merely enacted physical symptoms in line with Charcot's expectations.[26] Yet, such accounts have tended to emphasise only a single aspect of Bernheim's criticism while glossing over the irreconcilable differences between the concepts of hypnosis developed by each school.[27] In this section, I will argue that to understand Bernheim's dismissal of the Salpêtrian image-based research, we must examine the differences between the two schools' discordant conceptual frameworks.

A major point of contention between Bernheim and Charcot was how hypnosis and hysteria related to each other. Bernheim conceded that manifestations of hysteria could be produced in a hypnotised subject.[28] Nevertheless, he vehemently opposed

23 See Bernheim, *De la suggestion*, 91–95.

24 See Goetz, Bonduelle, and Gelfand, *Charcot*, 311.

25 See, e.g., Harrington, *Cure Within*, 58–60; Moll, *Hypnotism*, 94–95; Showalter, *Hystories*, 37; and Scull, *Hysteria*, 134.

26 Harrington, *Cure Within*, 59.

27 Notable exceptions are Hajek, "Fear of Simulation"; and Mayer, *Sites of Unconscious*. These two studies offer more nuanced comparative examinations of the hypnosis research at the Salpêtrière and Nancy schools.

28 Bernheim, *Suggestive Therapeutics*, viii.

Charcot's view that hypnosis was an artificial neurosis analogous to hysteria.[29] He also disagreed with Charcot's claim that only hysteria patients could be hypnotised. Bernheim contended instead that the hypnotic state could be induced in almost everyone, as it was merely an exaggeration of the normal susceptibility to suggestion, which all human beings possessed to some extent.[30] Even more to the point, Bernheim questioned Charcot's central tenet that hypnosis comprised three distinct nervous states (i.e., lethargy, catalepsy, and somnambulism), each of which was characterised by distinct physical features. As discussed in chapter 1, by visualising what he designated as the generic physical signs of lethargy and catalepsy, Charcot generated novel insights into hysteria's underlying neurological basis and diagnostically distinguished genuine patients from simulators.[31] However, Bernheim stated that after hypnotising thousands of subjects, he could neither reproduce Charcot's three hypnotic states nor their purportedly distinct physical signs, such as neuromuscular hyperexcitability.[32] This statement represented an indirect but very potent attack on the validity of Charcot's entire image-based hysteria research.

The Salpêtrière and Nancy schools derived their divergent views on the relationship between hysteria and hypnosis from their opposing understanding of hypnosis. Bernheim famously asserted that the crucial difference between the two schools' understanding of hypnosis consisted in the disparate roles they attributed to suggestion.[33] He defined suggestion as the influence that an idea, communicated by a hypnotist, exerted on the mind of a subject, who accepted this idea without verification.[34] According to Bernheim, the Salpêtrians misrecognised the central importance of suggestion in hypnosis.[35] Many historical and present-day accounts have uncritically adopted Bernheim's stance, attributing to it an almost dogmatic value.[36] But, in my opinion, this stance misrepresents the role Charcot accorded to suggestion concerning both hypnosis and hysteria.

Admittedly, Charcot insisted that during lethargy, "the mental inertia is so absolute that in general it is impossible to enter into relation with a hypnotised subject or to communicate to him any idea by any process whatever."[37] In other words, while in the state of lethargy, hypnotised subjects were insusceptible to suggestion. Nevertheless, Charcot maintained that suggestion was possible during catalepsy and somnambulism. And he used suggestion systematically in his numerous cataleptic and somnambulistic experiments, some of which were analysed in the previous chapter.[38] My analysis has shown that suggestion represented the cornerstone of Charcot's hypnotic modelling of paralysis as the exemplary symptom of traumatic hysteria. Moreover, I have argued that

29 Bernheim, viii.
30 Bernheim, 149.
31 See sections 1.2.1 and 1.2.2.
32 Bernheim, *Suggestive Therapeutics*, 87–91.
33 Bernheim, viii–ix.
34 Bernheim, x, 15. See also Bernheim, "Suggestion and Hypnosis," 1213.
35 Bernheim, *Suggestive Therapeutics*, 91.
36 See, e.g., Ellenberger, *Discovery of the Unconscious*, 89; and Moll, *Hypnotism*, 298.
37 Charcot, "Lecture 21: Brachial Monoplegia," 290.
38 For details, see sections 1.2.2 and 1.3.2.

for Charcot, autosuggestion—which he defined as a process of unconscious cerebration through which a fixed idea of motor or sensory loss induced genuine physical symptoms—represented the pathophysiological mechanism underlying hysteria. Thus, contrary to Bernheim's claim, suggestion occupied a crucial role in both schools' approaches to hypnosis and was also an essential element in Charcot's theorising of hysteria. Yet, as I hope to demonstrate in what follows, each school operated with a distinctly different understanding of what constituted suggestion and how suggestion transpired in the hypnotised subjects' minds. I will also claim that these different views, in turn, had consequences not only on how hypnosis could be related to hysteria but also on whether the hypnotically induced effects could be meaningfully measured and visualised.

To facilitate a direct comparison with Bernheim, let us summarise the central tenets of the Salpêtrian views on hypnotic suggestion. Similarly to Bernheim, the Salpêtrians also defined hypnotic suggestion as an operation that consisted "in introducing, cultivating, and confirming an idea in the mind of the subject," which then resulted in a sensation, gesture, or movement.[39] Yet, the Salpêtrians insisted that "the idea is an epi-phenomenon; taken by itself, it is only the indicative sign of a physiological process, [which is] solely capable of producing a material effect."[40] Hence, in this view, suggestion relied on purely physiological mechanisms. For example, as we saw in Charcot's somnambulistic experiments, an idea of paralysis could be communicated through a direct verbal injunction or, more indirectly, through physical intervention, such as a light blow. In each case, the suggestion had to produce "dynamic modifications" in the motor centres of the brain to give rise to an actual paralysis.[41] To induce visual hallucinations (e.g., seeing a bird or a butterfly), a verbal suggestion had to produce excitations in the brain's visual centre and thus revive the sensory impressions the subject had previously experienced. Put differently, visual hallucinations elicited through a verbal suggestion relied on the activity of the same cortical sensory centre as the perception of an actual physical object.[42] Moreover, as discussed previously in detail, Charcot argued that all neurophysiological processes that underpinned hypnotic suggestion represented a form of uncontrolled higher-order cerebral reflexes. Consequently, Charcot and his team repeatedly emphasised that all hypnotic phenomena induced through suggestion were "distinguished by their automatic," entirely involuntary character.[43]

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39 Binet and Féré, *Animal Magnetism*, 184.

40 Binet and Féré, 173.

41 Binet and Féré, 185. See also ibid., 184, 335, 348.

42 As pointed out by Binet and Féré, the only difference between a real visual sensation and a visual hallucination consisted in the process through which the excitation of the cerebral centre of vision was initiated: "When a real sensation of colour is experienced, the sensation results from an excitement of the retina, and it reaches the centre of visual sensation by the paths of vision, by the optic nerve, the chiasma, the optic tracts, etc. The sensation of colour suggested by words, that is, the hallucinatory image, results from the excitement of the organ of hearing, and it is reflected in the centre of auditory sensation before it reaches the centre of vision." Binet and Féré, 251–52.

43 Charcot, "Lecture 21: Brachial Monoplegia," 290.

It was this purely somatic framework that Bernheim opposed through his redefinition of suggestion. Bernheim insisted that the transformation of an externally suggested idea into a resulting sensation or movement was not executed through the excitation of the anatomically localised cerebral centres but instead through the working of the imagination. According to Bernheim, in hypnotic suggestion, it was "the subject's imagination alone which is rendered active and which causes all the phenomena."[44] Somewhat vaguely, Bernheim defined imagination as a peculiar "aptitude for mentally creating an image of the suggestions induced by speech, vision, or touch."[45] This image, in turn, was "as vivid as if it had an objective cause"—i.e., an external physical stimulus—so that the hypnotised subject accepted it as reality. [46] Bernheim further claimed that in the waking condition, the activity of the imagination was restrained by the higher faculties of the brain, which included "reason, attention and judgment."[47] However, a mere distraction of attention, such as closing one's eyes or falling asleep, sufficed to free the imagination from the control of reason and let it reign free.[48] Thus, Bernheim contended that the hypnotic condition was best described as an artificially modified psychological state in which the imagination was given free play to transform ideas suggested into various mental images, such as dreams and hallucinations. The brain then accepted these mental images without further verification and carried them out in the form of actions, sensations, or movements.[49] There was nothing pathological about this condition, as it did not create any extraordinary phenomena but merely exaggerated the normal susceptibility to suggestion by intensifying the activity of the imagination.[50]

Crucially, Bernheim argued that the activity of the imagination did "not rest upon any known anatomical or physiological fact."[51] Instead, he viewed imagination as a curiously dematerialised, purely psychological capacity that varied considerably across subjects depending on their personalities and individual temperaments.[52] In Bernheim's view, how each hypnotised subject translated the idea suggested by the hypnotist into an action depended exclusively on the vividness of their imagination. For Bernheim, the subject was not a merely passive receiver of the idea that the doctor had impressed into his mind, but someone who carries out "a suggestion as he conceives it, as he interprets it."[53] Contrary to Charcot, Bernheim asserted that the subject remained conscious during all phases of hypnosis.[54] In another opposition to Charcot, Bernheim also contended that in responding to the doctor's suggestions, the hypnotised subject

44 Binet and Féré, *Animal Magnetism*, 205.
45 Bernheim, *Suggestive Therapeutics*, 132–33.
46 Bernheim, 133.
47 Bernheim, x.
48 Bernheim, 130–42, 147.
49 Bernheim, x. See also Bernheim, "Suggestion and Hypnotism," 1214.
50 Bernheim, *Suggestive Therapeutics*, 149.
51 Bernheim, 151.
52 Bernheim, 9, 17, 90.
53 Bernheim, 28.
54 Bernheim, 92.

"carries on active intellectual work."[55] For this reason, the same hypnotic suggestion manifested "itself in different subjects in different ways," depending on how each of them elucidated the idea they received.[56]

Hence, we can say that, by placing the imagination centre stage, Bernheim not only rejected Charcot's physiological determinism but also vehemently opposed the view that hypnosis could turn subjects into "pure and simple automatons."[57] Whereas the Salpêtrians regarded the susceptibility to suggestion as a sign of the subject's morbidly weakened will,[58] Bernheim disagreed. He argued that the hypnotised subject's cooperation was a necessary precondition for the success of any hypnotic suggestion since "no one could be hypnotised against his will."[59] Bernheim thus foregrounded the hypnotised subjects' individuality. And even more radically, he attributed to experimental subjects an active role in the hypnotic process since their interpretation of the suggested idea decidedly influenced the outcome. In effect, Bernheim reconceptualised hypnosis as a relational phenomenon based on the dynamic interaction between the doctor and a hypnotised subject.

Seeking empirical validation for his views on hypnosis, Bernheim challenged the findings of a series of Salpêtrian experiments on hypnotically induced visual hallucinations. These experiments had been performed by Alfred Binet and Charles Féré, two of Charcot's pupils, who spearheaded the hypnosis research at the Salpêtrière from the mid-1880s.[60] Reflecting Charcot's views, Binet and Féré argued that hallucinatory images elicited in a hypnotised subject by a verbal suggestion had the same seat in the brain as the perception of actually existing external objects.[61] Paul Richer delivered the initial empirical support for this claim. Specifically, Richer had shown that patients with hysterical colour-blindness (i.e., achromatopsia) could not be induced to hallucinate the colours, which they were unable to perceive in their waking state.[62] The Salpêtrians attributed this parallel loss of the abilities to perceive as well as to hallucinate a particular colour to the same underlying functional lesion of the cerebral cortex. Furthermore, they argued that this lesion consisted in the dynamic inhibition of the cortical centre of vision.[63]

In the next step, Binet and Féré systematically expanded Richer's initial finding through a battery of experiments. Their experiments were meant to demonstrate that a visual hallucination could produce a sensation of a complementary colour, be doubled by a prism, enlarged by a magnifying glass, reflected in a mirror, or concealed by an opaque body. Some of the simpler experiments involved the so-called phenomenon of chromatic contrast. "If, for instance, a piece of paper divided by a line is presented to a hypnotized subject, and it is suggested to her that one half is red, the sensation

55 Bernheim, 144.
56 Bernheim, 15.
57 Bernheim, 210.
58 See sections 1.2.2 and 13.2.
59 Bernheim, *Suggestive Therapeutics*, viii.
60 See Binet and Féré, *Animal Magnetism*, 211–76.
61 Binet and Féré, 249.
62 Binet and Féré, 248–49.
63 Binet and Féré, 249.

of the complementary colour, green, occurs on the other half. If, after awaking, the sensation of red remains, so also does the sensation of green."[64] Other experiments were more elaborate. For example, a "portrait of a given person may be made to appear on a square of white paper, and a series of experiments may be performed on this imaginary portrait… If a magnifying glass is placed before the imaginary portrait, the subject declares that it is enlarged, and if the lens is sloped, the portrait is distorted. If the sheet is placed at a distance equal to twice the focal length of the lens, the portrait appears to be inverted."[65] Furthermore, it "may be suggested to the subject that an object is placed on a given point of the table, and if a mirror is placed behind that point the patient immediately sees two objects… [I]f the mirror is advanced, withdrawn, or inclined, so that it could no longer reflect the supposed object, the double vision ceases."[66]

The shared aim of all these experiments was to prove that hypnotically induced visual hallucinations followed the same optical laws as the perception of actually existing objects and, therefore, had to have the same material basis. However, in the course of their experiments, Binet and Féré were forced to admit that they were not always able to obtain entirely consistent results. Sometimes the visual hallucinations appeared to behave according to the optical laws. At other times they did not.[67] Nevertheless, Binet and Féré did not view this lack of consistency as an epistemic problem. Instead, they somewhat vaguely justified the empirical inconsistencies with the following statement: "Just as experiments in physics sometimes miss fire, so it is with experiments in cerebral physiology."[68] Moreover, they argued that "if under favourable conditions" their experiments were successful even in a single instance, these exemplary positive results offered sufficient empirical proof that hallucinatory images had a physiological basis.[69] These 'favourable conditions' included formulating the verbal suggestion in a way that left no room for ambiguity and choosing patients in whom the hypnotic susceptibility was particularly pronounced.[70]

Bernheim reproduced some of Binet's and Féré's experiments that either relied on the induction of chromatic contrasts or made use of prisms to elicit optical transformations of hallucinatory images.[71] For this purpose, he hypnotised not only hysteria patients with unilateral blindness but also "non-hysterical women of medium intelligence and good judgment."[72] Significantly, Bernheim's choice of the experimental subjects, which established a relation of analogy between hysteria patients and healthy individuals, already represented a direct challenge to the Salpêtrians. Like Binet and Féré, Bernheim also obtained inconsistent results—the hallucinatory images

64 Binet and Féré, 250. Ibid., 230.

65 Binet and Féré, 230.

66 Binet and Féré, 232–33. For additional experiments, see ibid., 226–76.

67 See, e.g., Binet and Féré, 230, 234, 241.

68 Binet and Féré, 241.

69 Binet and Féré, 230.

70 See Binet and Féré, 254, 336.

71 For a detailed description of these experiments, see Bernheim, *Suggestive Therapeutics*, 47–50, 95–104.

72 Bernheim, 96.

sometimes conformed to the optical laws and sometimes did not.[73] But despite similar experimental results, Bernheim and his Salpêtrian rivals offered two entirely diverging interpretations. As I am about to show, each interpretation was grounded in a distinctly different set of intermedial references.[74] Moreover, we will see that much of the discussion concerning the potential meaning of the experimental results focused on elucidating the nature and potential location of the patients' internal mental images.

To explain the positive results of their optical experiments, Féré and Binet conjectured that the hallucinatory image produced in the hypnotised subject through verbal suggestion did "not remain in his brain in a vague and floating state."[75] Instead, the hallucinatory image was projected onto the outside world and associated with some distinctive visual feature of an actual physical object in the hypnotised subject's environment. A particular visual feature of the external object thus became the reference point ("point de repère") for the exteriorised hallucinatory image.[76] As a result of this association, in the sensory centre of the subject's brain, the hallucinatory image merged with the visual sensations arising from the external object that served as its reference point in the physical world.[77] Because of such merging, any modification that optical instruments produced on the external reference point also necessarily affected the associated hallucinatory image.[78] Féré and Binet considered that in positing this explanation, they succeeded in providing sufficient proof for the purely physiological nature of hypnotically induced hallucinations. However, Bernheim disagreed.

According to Bernheim, the hallucinatory image "has no objective reality, follows no optical laws, but obeys solely the caprices of the imagination."[79] If the hallucinatory image sometimes did behave like an image of a real physical object, it was only because the hypnotised subject was eager to please the physicians and acted accordingly. She either deduced the optical laws from previous experience, overheard the experimenters discuss the desired results, or in some other way guessed their expectations and then imagined the optically correct visual effects.[80] In other words, Bernheim insisted that what the hypnotised subjects 'saw' was a fictitious image, which existed in their

73 Bernheim, 96–104.

74 Jäger, "Transcriptivity Matters," 49.

75 Binet and Féré, *Animal Magnetism*, 225.

76 Binet, "L'hallucination," 492. It appears that Binet and Féré considered such reference points to be entirely arbitrary.

77 For more details, see Binet and Féré, *Animal Magnetism*, 220–24, 242. Notably, Binet and Féré argued that an equivalent mechanism underpinned normal perception, which also consisted of "a synthesis of external sensations with internal images," which, in turn, were constructed by the mind and projected onto the external environment. Ibid., 244. However, in normal perception, internal images had a secondary role and served to complete the sensations induced by the external object. In hypnotic hallucinations, the internal images became dominant. Binet and Féré declared that hypnotic hallucination "must, therefore, be a disease of external perception." Ibid. In other words, they viewed hypnotic hallucinations as a pathological form of sensory perception in which the mental images induced through verbal suggestion disproportionally modified the visual sensations elicited by actual external objects.

78 Binet, "L'hallucination," 492–93.

79 Bernheim, *Suggestive Therapeutics*, 103–4.

80 See Bernheim, 95–104.

imagination only and had no physiological basis whatsoever. Bernheim conceded that impressions from the outside world still traversed the subjects' retina and created a sensorial image in their cerebral visual centre. Yet, he insisted that the subject's imagination effaced the resulting physical image, displacing it with a purely fictitious mental image.[81]

By analogy, Bernheim further posited that neither hypnotically induced nor actual hysterical blindness had anything to do with functional lesions of the cerebral sensory centres. He conjectured instead that both genuine hysterical and artificially produced hypnotic blindness were merely a particular form of negative hallucinations.[82] He argued that, in both cases, the subject could not see because his imagination obliterated all his visual sensations. In the case of hypnotically induced blindness, the imagination was activated by the hypnotist's suggestion. In the case of hysterical blindness, the inability to see arose from the patient's "diseased imagination."[83]

In effect, Bernheim claimed that to produce hallucinations, imagination had to override normal physiological processes. In his view, the laws of physiology applied neither to hysterical blindness nor to hypnotically induced hallucinations. He forcefully stated that "hysterical and suggestive amaurosis [i.e., blindness] have no anatomical localization. Their seat is not in the retina, nor in the optic nerve, nor in the cortical centre for vision. They are real, but exist only in the patient's imagination."[84] This conjecture makes evident that Bernheim and the Salpêtrians operated with two mutually discordant frames of reference when interpreting not just the findings of their hypnotic experiments on visual hallucinations but also hysterical blindness. For the Salpêtrians, the distinctive feature of hypnotic visual hallucinations and hysterical blindness was their hypothesised physiological nature. For Bernheim, the distinctive feature of hypnotic visual hallucinations and hysterical blindness was the hypothesised lack of any localisable physiological basis. These two views were mutually irreconcilable.

Next, Bernheim expanded his explanation to all hypnotically induced effects and to all types of hysterical symptoms.[85] He asserted that all physical manifestations of hypnosis were purely psychological phenomena in which the subject's imagination could produce arbitrary changes in their organic functions.[86] Hence, according to Bernheim, neither hypnotic phenomena nor hysterical symptoms had any "objective characteristics, but only subjective ones."[87] Whereas much of the dispute between Bernheim and the Salpêtrians discussed so far centred on patients' internal mental images, the importance of this particular statement is that it had direct consequences on the applicability of empirical images as research tools. Specifically, the direct implication of this statement was that visualising physiological aspects of either hypnotic manifestations or hysterical symptoms missed the very essence of these

81 Bernheim, *Hypnotisme, suggestion, psychothérapie*, 124, 136.
82 Bernheim, *Suggestive Therapeutics*, 46–48.
83 Bernheim, 49.
84 Bernheim, 50.
85 Bernheim, 50.
86 Bernheim, 48.
87 Bernheim, 104.

phenomena. Bernheim, therefore, refused to ascribe any epistemic significance to the apparent regularity of either hypnotically induced or actual hysterical symptoms whose systematic visualisation stood at the centre of the Salpêtrian research. Instead, he conjectured that his Salpêtrian rivals "imperfectly grasped the nature and the signification" of the phenomena they studied.[88]

Additionally, Bernheim suggested that the Salpêtrians possibly tainted their experimental setup by unintentionally inducing hysteria patients to produce particular kinds of physical manifestations, which accorded with their implicit expectations.[89] Misguided by their conception of hypnosis as a purely physiological phenomenon, the Salpêtrians made the "fundamental error" of thinking that their patients were mere automatons.[90] Yet, despite appearing inert, the hypnotised patients perceived and actively interpreted not just the explicitly formulated verbal instructions but also the unspoken expectations the physicians unwittingly communicated through their gestures and demeanour.

Consequently, Bernheim also dismissed Charcot's use of visualisations to diagnostically differentiate between hypnosis and hysteria, on the one hand, and simulation, on the other hand.[91] Put differently, Bernheim refused to accept that a particular visual pattern of the subjects' breathing curves or their artificially induced neuro-muscular reactions could be relied upon to disambiguate between real and intentionally simulated hypnotic manifestations. He declared such visualisations useless because the difference between the genuine and simulated phenomena did not transpire at the physiological but only at the psychological level. "[T]he patient deaf by suggestion hears, as the patient who is blind by suggestion sees, but each instant he neutralizes the impression perceived by his imagination, and makes himself believe that he has not heard."[92] In Bernheim's view, it was the subject's belief in the reality of the imagined phenomenon that differentiated a genuine hypnotic condition from a simulation. The same applied to hysterical symptoms.

According to Bernheim, although wilful simulation was not empirically measurable, it could nevertheless be detected. To do so, however, the doctor had to rely on his subjective judgment of the patient's behaviour. Drawing on his long-term experience of working with particular patients, Bernheim evaluated "their expression, their behavior, intonation of voice and manner of relating a story" to determine if these expressed "conviction and sincerity."[93] Bernheim thus regarded as meaningful precisely those idiosyncratic, subjective characteristics of the patients' behaviour, which Charcot considered noise in his experimental setup and attempted to filter out.[94] To determine if they were simulating or not, Bernheim did not measure his patients' isolated bodily

88 Bernheim, 45.

89 Bernheim, 90–92.

90 Bernheim, 91.

91 Bernheim, 13, 88–89. For a discussion of Charcot's use of respiratory curves, see section 1.2.2.

92 Bernheim, *Suggestive Therapeutics*, 50.

93 Bernheim, 176.

94 For a detailed analysis of Charcot's approach to experimentally framing his hypnotised patients' facial expressions and gestures, see section 1.2.2.

reactions. Instead, he listened to them and observed their idiosyncratic reactions, assessing their behaviour on the whole.

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To conclude, my discussion in this section has aimed to show that Bernheim decidedly shifted hypnosis into the realm of psychology, where "the cause and essence of phenomena escape" straightforward explanations.[95] In doing so, he embraced a high level of physiological indeterminacy in the experimental effects he was inducing in his hypnotised subjects. Unlike Charcot, Bernheim foregrounded the hypnotised subject's individuality and reconceptualised hypnosis as an artificially modified state of consciousness in which the imagination dominated over reason. By analogy, he declared hysterical symptoms to be the product of the patients' diseased imagination. Thus redefined, the essence of hypnosis and hysteria became their entirely psychological nature and their variability across individuals. As a result of such transcription,[96] hypnosis was no longer usable for producing generalisable insights into hysteria. Moreover, as we have seen, measuring and visualising experimentally isolated physical aspects of various hypnotic effects became devoid of any epistemic function in this particular framework. Whereas Charcot and his team viewed the hypnotic symptoms' apparent regularity as an indication of their underlying physiological nature, Bernheim considered it meaningless. As a result, Bernheim rejected the Salpêtrian images-based research on both hypnosis and hysteria.

Yet notably, Bernheim argued that, instead of being an experimental analogue of hysteria, hypnosis was a highly effective therapeutic tool.[97] In its most basic form, Bernheim's treatment consisted in hypnotising hysteria patients and then affirming in a loud voice that their symptoms would disappear. Importantly, Bernheim insisted that the "*mode of suggestion* should also be varied and adapted to the special suggestibility of the subject."[98] As he further explained, it was "sometimes necessary to reason, to prove, to convince; in some cases, to affirm decidedly; in others, to insinuate gently; for in the condition of sleep just as in the waking condition the moral individuality of each subject persists according to his character, his inclinations, his special impressionability."[99] In effect, it can be said that Bernheim used targeted verbal suggestion to treat heterogeneous hysterical symptoms by restraining the patients' purportedly diseased imagination. Having dismissed images, Bernheim reverted to words.

2.1.2 Janet: Images as Tools for Visualising Hysteria Patients' Mental States

Whereas the rivalry between the Salpêtrière and Nancy schools focused primarily on hypnosis, a more direct challenge against Charcot's neurophysiological conception of hysteria was mounted by his former pupil Pierre Janet. Significantly, although Janet

95 Bernheim, *Suggestive Therapeutics*, 139.
96 Jäger, "Transcriptivity Matters," 49.
97 See Bernheim, *Suggestive Therapeutics*, 202–7.
98 Bernheim, 210 (emphasis in original).
99 Bernheim, 210.

resolutely and repeatedly criticised Charcot's physiological determinism,[100] he never repudiated his mentor's image-based hysteria research on the whole. As I will argue in what follows, by drawing on Charcot's findings and subtly transcribing them into a different theoretical context, Janet developed a new conception of hysteria as a distinct psychological disorder.[101] Additionally, I intend to show that Janet's reconceptualisation of hysteria directly affected how he used images as investigation tools.

To begin with, Janet adopted Charcot's classification of hysterical symptoms into, on the one hand, permanent (i.e., stigmata) and, on the other hand, transitory (i.e., accidents).[102] However, the crucial difference was that in Janet's classification, permanent symptoms were no longer limited to physical manifestations of hysteria, such as anaesthesia, contractures, and paralysis. Instead, they also included amnesia, the weakness of the will, suggestibility, and permanent modifications of hysteria patients' intelligence and character.[103] Similarly, in addition to hysterical attacks, the accidents comprised somnambulism, deliria, and double personalities.[104] Even a superficial glance at this list makes it apparent that Janet placed a distinct focus on hysteria patients' various mental characteristics, which he thus elevated into individual symptoms. This focus already marked a clear departure from Charcot's predominantly somatic framework.

Even more radically, Janet conjectured that both somatic and mental symptoms of hysteria had a common cause consisting in an underlying psychological disturbance. This psychological disturbance was evident in some symptoms, such as deliria and hysterical attacks, yet masked in others, such as contractures and anaesthesia.[105] To designate this disturbance, Janet introduced the concept of dissociation. He defined dissociation as a pathological fragmentation of the otherwise integrated mental functions and contents.[106] He then deployed dissociation to explain the formation of various hysterical symptoms. With this aim in mind, he first turned to the analysis of anaesthesia, which he declared to be one of the simplest hysterical symptoms.[107]

According to Janet, to be able to say 'I feel, I see,' an individual must synthesise a massive and continual influx of isolated sensorial data (i.e., elementary sensations) with "an enormous mass of thoughts already constituted into a system" that forms

100 See Jäger, "Transcriptivity Matters," 49–50.

101 See Janet, *Mental State*, xviii.

102 Janet, xvi.

103 In Janet's classification, the 'weakness of the will' or abulia was a hysterical symptom in its own right. The characteristics of this symptom were laziness, hesitation, indecision, mental inertness, and inattentiveness. Janet considered it one of the key symptoms of hysteria. Janet, 117. For Janet's in-depth analysis of various permanent mental symptoms of hysteria, see Janet, *Major Symptoms*, 270–316.

104 See Janet, *Mental State*, 366–483. In Janet's use, the term somnambulism acquired a different meaning from the one Charcot attributed to it. Janet defined somnambulism as an abnormal sleep-like state that developed spontaneously in hysteria patients and of which they had no memory after returning to the normal state. Ibid., 413–53.

105 Janet, xvii.

106 Janet, *Major Symptoms*, 331–32.

107 Janet, 275–76.

the subject's notion of her personality (i.e., the ego).[108] Janet used the term personal perception to refer to this operation of synthesis. Moreover, he introduced the term 'the extent of the field of consciousness' to designate the maximum number of elementary sensations that an individual could assimilate within a personal perception.[109] He claimed that, in individuals with a hereditary predisposition, an experience of a traumatic event could trigger the development of a thus far latent psychological insufficiency.[110] Once this insufficiency was developed, the subject became incapable of forming a personal perception of more than only a few elementary sensations, while neglecting the rest. This, in turn, led to what Janet termed 'the narrowing of the field of consciousness.'[111] Consequently, the subject ceased to perceive the external sensations that she could not connect to her personality. At first, such retraction of consciousness represented only a "bad psychological habit,"[112] a form of temporary absent-mindedness. Notably, Janet equated this absent-mindedness with the pathological 'feebleness of attention.'[113] Yet, the crucial point was that, in hysteria patients, this absent-mindedness gradually became chronic, thus developing into full-blown anaesthesia. In Janet's view, in hysterical anaesthesia, the sensations did not disappear but merely became unconscious. They were "no longer at the disposal of the will or the consciousness of the subject."[114]

Already at this point, both Janet's indebtedness to Charcot and his extensive reworking of his former mentor's views are apparent. First, the notion of the latent hereditary predisposition triggered by a traumatic event is familiar to us from Charcot's lectures on the formation of hystero-traumatic paralysis.[115] However, contrary to Charcot, in Janet's reinterpretation, both the hereditary predisposition and the triggering effect of the trauma came to be defined in exclusively psychological terms.[116] Second, Charcot viewed the clouding of the consciousness and the "dissociation of the

108 Janet, *Mental State*, 35. For a similar definition of the ego, see Charcot, "Lecture 21: Brachial Monoplegia," 290.

109 Janet, *Mental State*, 38. "The word 'consciousness,' which we use continually in studies on the mental state of our patients, is an extremely vague word, which means many different things. When we use it in particular to designate the knowledge the subject has of himself, of his sensations and acts, it means a rather complicated psychological operation, and not an elementary and irreducible operation, as is generally believed." Janet, *Major Symptoms*, 303.

110 "Pathological heredity plays in hysteria, as in all other mental maladies, a role absolutely preponderant. A very great number of circumstances play the part of 'provocative agents,' and manifest by accidents this latent predisposition; they are hemorrhages, wasting and chronic diseases, infectious diseases, typhoid fever in particular, and, in certain cases the autointoxications, the organic diseases of the nervous system, various intoxications, physical or moral shock, overwork, either physical or moral, painful emotions, and especially a succession of that sort of emotions the effects of which are cumulative." Janet, *Mental States*, 526.

111 Janet, 40.

112 Janet, 40.

113 "The attention is painfully slow in fixing itself, is accompanied with accidents of all sorts, is quickly exhausted, and gives but a minimum of results; it forms but vague, doubtful, surprising, and unintelligible ideas." Janet, 399.

114 Janet, *Major Symptoms*, 319.

115 See section 1.3.2 for a detailed analysis.

116 See Janet, *Mental State*, 336.

ego" as temporary cerebral effects that could either be produced artificially through hypnosis or occurred spontaneously in the condition of a trauma-induced nervous shock.[117] By contrast, Janet considered the dissociation of consciousness to be a permanent psychological state that underpinned not just the formation but also the continued existence of hysterical symptoms.[118] Third, Charcot attributed hysterical anaesthesia to a functional disturbance of the cerebral sensory centres that presided over the formation of sensations.[119] Janet instead attributed hysterical anaesthesia to a purely psychological disturbance he designated as a chronic absent-mindedness. In other words, Charcot claimed that anaesthetic patients had a problem with forming sensations at the neurophysiological level. Unlike Charcot, Janet contended that the sensations were there but that the patients lost the ability to pay attention to them and could, therefore, no longer perceive them consciously.

In the next step, Janet used the concept of dissociation to explain the formation of hysterical attacks by drawing in part on Charcot's four-stage model of the *grande attaque*. Admittedly, Janet stated that Charcot's schematic model of the hysterical attack was too artificial to be applicable in clinical practice.[120] Yet, he also suggested that the model had nevertheless been epistemically useful because it disclosed the underlying regularity of the hysterical attack.[121] Moreover, unlike Bernheim, Janet argued that Charcot neither misrecognised nor fabricated the hysterical attack's underlying regularity. Instead, Charcot simply made the mistake of attributing the hysterical attack's underlying regularity to purely physiological causes.[122] Janet contended that to understand the hysterical attack and all the other symptoms of hysteria, it was necessary "to retain something of the precise method of Charcot" but apply it to the study of psychological phenomena.[123]

In Janet's view, the critical insight provided by Charcot's visual model was the discovery that the temporal course of the attack was not arbitrary but followed a

117 Charcot, "Appendix 1: Hystero-Traumatic Paralysis," 383. As discussed previously, in Charcot's view, the effects of a nervous shock occasioned by an accident typically lasted for several days or weeks, during which time the formation of the fixed idea of paralysis took place.

118 See Janet, *Mental State*, 40.

119 See section 1.3.1.

120 Janet, *Major Symptoms* 21–22. "[N]obody nowadays any longer describes the attack of hysteria as Charcot did." Ibid., 21.

121 Janet, *Mental State*, 399.

122 Janet, *Major Symptoms*, 17. In his early work, Janet claimed that the complete hysterical attack, as described by Charcot and Richer, actually existed in its 'natural form' but was a rare phenomenon. Janet, *Mental States*, 386–89. Later, he suggested that by experimentally inducing hysterical attacks through hypnosis, the doctors at the Salpêtrière might have unwittingly modified their patients' attacks according to this pattern. He conjectured that potential modifications arose from the doctors' lack of understanding of unintentional psychological effects their experimental interventions produced. By thinking they were experimentally manipulating purely physiological phenomena, his colleagues failed to realise that they were introducing their ideas into the hypnotised subjects' somnambulistic dreams and thus potentially reshaping the original phenomena they aimed to study. Janet, *Major Symptoms*, 113–14.

123 Janet, *Major Symptoms*, 18.

regular order.[124] Drawing on Charcot, Janet stated that the epileptoid period tended to precede the stage of large movements, whereas the phenomena of delirium only took place at the end of the attack.[125] In effect, at the formal level, Janet largely adopted Charcot's model but introduced one change. He conflated the period of passionate attitudes and the delirium into a single category, thus reverting to a tripartite model of the attack. Even more importantly, unlike Charcot, Janet associated each period of the attack with a particular psychological state. Specifically, he equated the first period with exaggerated emotions (e.g., anger, fear), the second with tics and convulsions (e.g., weeping, choking, dancing), and the third with hallucinations and dreams.[126] Put simply, whereas Charcot differentiated between emotionally expressive and inexpressive periods of the attack,[127] Janet regarded all aspects of the attack to be emotionally expressive. Janet thus redefined the hysterical attack as a symptom that comprised an entire "ensemble of emotional manifestations," which were expressed through the patient's attitudes, physiognomy, movements, dreams, and words.[128] Janet posited that such emotional manifestations were the very essence of the hysterical attack since they reproduced the patient's subconscious fixed ideas.[129] In Janet's definition, subconscious fixed ideas comprised a group of thoughts, mental images, and emotions that had arisen in response to some forgotten traumatic event from the patient's past.[130]

Janet contended that the formation of such fixed ideas hinged on the same hereditary psychological insufficiency, which he had deployed to explain the nature of hysterical anaesthesia. As discussed previously, in Janet's view, the formation of hysterical anaesthesia entailed a disassociation of single sensations from the patient's consciousness. To give rise to fixed ideas, the narrowing of consciousness had to produce slightly different effects. In this case, an entire system of mutually coordinated mental images that had developed in the subject's mind during a traumatic event became disassociated from the subject's voluntary control.[131] These mental images became fully isolated from the subject's personal perception and, therefore, unconscious. Thus detached, the mental images remained not only coherently grouped among themselves but also associated with previously related thoughts and emotions.[132] That is, despite the same psychological mechanism underlying their

124 Janet, *Mental State*, 399.

125 Janet, 399–400.

126 Janet, 396. For Janet's detailed description, see ibid., 366–400.

127 See section 1.1.3 for a detailed discussion.

128 Janet, *Major Symptoms*, 102. See also ibid., 104.

129 Janet, *Mental State*, 280, 393.

130 See Janet, 282–85, 288–90, 381.

131 Janet, 259–61, 513.

132 Janet, 245–46. "Any idea, well understood, quite clear, forms in reality in our mind a whole, a system of different images, each having special properties diversely co-ordinated… The thought of a bouquet of roses or the thought of a cat contains alike numerous elements grouped around each other in a very close dependency. We have but to point out in these ideas the notion of the colour of the flowers, the colour and form of the cat, then numerous images of smell, touch, hearing, etc.,—in a word, as we were saying, these ideas are veritable systems of images." Ibid., 244.

formation, what differed between anaesthesia and the hysterical attack was the mental content that became dissociated from the patient's consciousness.

Janet further insisted that although called forth by an experience of either psychological or physical trauma, fixed ideas could only develop in predisposed subjects due to their inherent suggestibility.[133] Similarly to Charcot, Janet designated as suggestion those "subconscious acts" that led to the exaggerated development of fixed ideas in an entirely automatic manner.[134] Thus this process occurred outside the subject's will, conscious perception, and memory. But unlike Charcot, who understood suggestion to be a distinctly physiological process, Janet argued that suggestion was primarily a psychological mechanism. Its primary characteristic was the dissociation of consciousness, or in other words, the splitting of mental contents from the patient's awareness.[135]

Moreover, Janet additionally expanded the meaning of suggestion. In Janet's definition, suggestion did not only refer to the psychological mechanism underpinning the formation of fixed ideas. Instead, suggestion also designated the abnormal way in which the fixed ideas subsequently acted on the patient's body to both produce and maintain hysterical attacks. Specifically, it was through suggestion that once they had developed, the fixed ideas tended to automatically and compulsively repeat themselves with mechanical regularity.[136] Once activated in the form of hysterical accidents, the fixed ideas completely overtook the subject's mind. They then triggered an association of images, which reproduced themselves in a fixed order that had been established through a previous mental synthesis during the traumatic experience.[137] For example, "X. has a crisis of convulsions and utters shrieks of pain when she thinks of her husband, and an ecstatic attack full of delicious dreams when she thinks of her lover… Is., in consequence of a rape and a clandestine confinement, presents at first an anorexia (fixed idea of subconscious suicide), then anger and violence (subconscious idea of homicide to avenge herself)."[138] Hysteria patients remained entirely unaware that they were incessantly repeating a fixed succession of past thoughts, emotions, and images through their hysterical attacks.

While under the powerful influence of their fixed ideas, the subjects were closed off to the outside world. They found themselves in an abnormal state of dissociated consciousness that Janet designated as somnambulism.[139] According to Janet, this dissociated state was equivalent to hypnosis. The only difference between hypnosis and somnambulism was that the latter phenomenon developed spontaneously in hysteria patients under the influence of their fixed ideas, whereas hypnosis was

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This quote shows that, like Charcot, Janet also drew on the theory of associationism we discussed previously.

133 Janet, 526.

134 Janet, 251. See also ibid., 278, 409; and Janet, *Major Symptoms*, 318.

135 Janet, *Mental State*, 249, 251. For a discussion of Charcot's views on suggestion, see sections 1.2.2 and 1.3.2.

136 Janet, *Mental State*, 246.

137 Janet, 249.

138 Janet, 404.

139 Janet, *Major Symptoms*, 289.

artificially induced under controlled conditions.[140] Hence, Janet aligned himself with Charcot and against Bernheim by claiming that both hypnosis and susceptibility to suggestion were mutually analogous pathological phenomena specific to hysteria. Contrary to Bernheim's notion of the free play of the imagination, Janet thus redefined suggestion as an unconscious compulsion to repeat fixed ideas. Furthermore, Janet argued that this unconscious compulsion did not only lead to the production of hysterical attacks. The same unconscious compulsion also underpinned the formation of amnesias, contractures, hallucinations, paralysis, and a host of other symptoms.[141] Janet thus instituted suggestion into a highly distinct yet also intrinsically pathological psychological mechanism that was constitutive of hysteria on the whole. To underscore this point, Janet referred to hysteria as "a disease due to suggestion."[142]

By his own admission, in developing his new conception of hysteria, Janet drew extensively on Charcot.[143] However, my analysis has underscored that Janet substantially reinterpreted the concepts and notions he had adopted from his former mentor. We have discussed previously that Charcot used the notion of the fixed idea to explain the formation of hysterical paralysis of traumatic origin. According to Charcot, the fixed idea of motor weakness, which originated in the transitory disturbances of sensibility induced by the local shock, gave rise to physical paralysis through the mechanism of a cerebral reflex.[144] By displacing the cerebral reflex with a psychological automatism, Janet proposed a more complex mechanism. As detailed above, in Janet's interpretation, the fixed idea was no longer derived from simple sensations but instead comprised an entire system of mutually coordinated thoughts, mental images, and emotions.

Moreover, as I have shown in the previous chapter, Charcot implicitly envisioned the formation of hysterical symptoms as a relatively straightforward neurophysiological chain of cause and effect that led to the production of an anatomically localisable functional brain lesion. It was to the existence of this hypothesised brain lesion that Charcot ascribed the regularity of the resulting hysterical symptoms. By contrast, the psychological automatism that Janet posited functioned as a dynamic "pathological vicious circle."[145] Janet contended that fixed ideas developed only in patients who already exhibited the weakness of the will, absent-mindedness, and the retraction of the field of consciousness as permanent symptoms of hysteria. Put simply, Janet emphasised that the formation of fixed ideas did not take place in early but only in more advanced stages of hysteria.[146] Once formed, the fixed ideas, in turn, caused further

140 Janet, 114.

141 See Janet, *Mental State*, 325, 356–57. "There are such [fixed] ideas in systematic [hysterical] contractures, for instance, when a patient seems to hold her feet stretched because she thinks herself on the cross." Janet, *Major Symptoms*, 324. "And do not forget that those pretended hysterogenic points are merely spots in which certain peculiar sensations easily arise, associated with the remembrance of an affecting event." Ibid., 100.

142 Janet, *Major Symptoms*, 330.

143 Janet, 324.

144 See Charcot, "Appendix 1: Hystero-Traumatic Paralysis," 384–86.

145 Janet, *Mental State*, 410.

146 Janet, *Major Symptoms*, 320.

dissociation of consciousness and weakening of the will, thus both giving rise to new and aggravating the already existing symptoms.[147] Therefore, for Janet, the hysteria patient's mind operated as a self-perpetuating psychological feedback loop. Within this loop, each disturbance produced multiple, far-reaching effects, all of which then mutually reinforced one another.

In Janet's view, however, none of the dynamic psychological processes that underpinned various hysterical manifestations was unambiguously localisable to distinct brain regions.[148] Notably, Janet did not entirely dismiss the possibility that hysteria had some unknown physiological basis, which was impossible to identify at the time.[149] According to Janet, "the fact that a system is psychological should not cause us to conclude that it is not at the same time anatomical."[150] Yet, he remained highly sceptical about the existence of a functional brain lesion as the underlying cause of a particular hysterical symptom.[151] Unlike Charcot, Janet conjectured that even if hysteria depended on some unknown functional alterations of the brain, "it is not likely that these alterations, whatever be their cause, are absolutely isolated in an entirely healthy organism. The actions and reactions of the various parts of the nervous system and even of all the organs, one upon the other, are so numerous that insufficiency in the working of the cerebral apparatus is accompanied by many other troubles."[152]

Unsurprisingly, in Janet's model, the underlying mechanical regularity of hysterical symptoms had nothing to do with physiology. Thus, Janet disagreed with Charcot that each hysterical symptom was characterised by a universal pattern of regularity (i.e., a type) shared across patients.[153] Instead, Janet argued that hysterical symptoms varied from patient to patient but that the regularity of the symptoms was manifested at the individual level. In short, the symptoms remained "always the same for the same patient."[154] This regularity, as Janet asserted, was determined by the idiosyncratic content of a particular patient's fixed ideas.[155] Specifically, he claimed that a single patient's mind was repeatedly invaded by always the same set of mutually interconnected fixed ideas. These ideas manifested themselves through a particular

147 Janet, *Mental State*, 364.

148 "You will understand, once for all, that the word 'mind' represents the highest functions of the brain and probably the functions of the cortex. It is out of respect for the scientific method that we employ the word 'mind' and that we do not permit ourselves metaphysical speculations on the unknown alterations of the cerebral cells." Janet, 52. See also ibid., 514–15.

149 "Someday, perhaps, these physiological modifications, which accompany cerebral insufficiencies, will be determined in a manner precise enough to enable us to show a fundamental physiological phenomenon, to which all the details of the delirium of persecution may be related, and another by which all the phenomena of hysteria may be explained with precision. We shall then have a physiological definition of hysteria. We think that at the present day such a definition would be extremely vague and would not clearly embrace the characteristic phenomena of the disease." Janet, 514.

150 Janet, *Major Symptoms*, 179.

151 Janet, 322–23; Janet, *Mental State*, 515–16.

152 Janet, *Mental State*, 514.

153 Janet, 403–4. See also Janet, *Major Symptoms*, 129–30.

154 Janet, *Mental State*, 403.

155 Janet, 205.

combination of symptoms specific to each patient.[156] As a result, the patient always had "the same attacks, the same attitudes, the same stigmata," remaining "indefinitely the same, under the same emotion, without adapting herself to the indefinitely changeable circumstances around."[157] To understand the unique dynamics of the underlying pathological loop in an individual clinical case, the physician had to analyse each patient's mental states. Only in this way could the physician uncover the specific fixed ideas and mental images that a particular patient kept reliving through their symptoms. Put differently, the psychological mechanisms of dissociation provided a useful conceptual framework for understanding hysteria in general. However, what mattered in the clinical practice was the "search for an interpretation proper to each subject."[158]

Importantly, Janet's shift towards the purely psychological causation of hysteria substantially impacted his stance on the potential utility of images as epistemic tools. Working at the Salpêtrière, first as Charcot's pupil and later as the director of the psychological laboratory, Janet continued the tradition of measuring and visualising hysteria patients' various physiological functions and physical symptoms. He thus produced photographs of patients' contractures and pathological postures, tables of their fluctuating temperature and urinary excretions, body maps of their anaesthesia, graphs of their reaction times, curves of their tremors and breathing function, as well as perimetric maps of their various visual disturbances.[159] Yet, even when he included the resulting images in his publications, Janet repeatedly emphasised the fundamentally ambiguous nature of these images.[160]

For Janet, empirical images of hysteria patients' bodies were potentially revelatory only in as much as they could provide insights into the individual's mental states and thus uncover the psychological causation of each hysterical symptom.[161] But Janet warned that psychology "is not yet advanced enough to admit of many precise measures."[162] He argued that without sufficient prior knowledge about how exactly hysteria's underlying psychological mechanisms translated into actual physical symptoms, there were two key challenges. First, it was difficult to determine which specific bodily function to measure in the first place. Second, it was far from clear how to interpret the resulting images. Moreover, Janet cautioned that by experimentally isolating and measuring only a single physiological aspect of a particular hysterical symptom, the physician might unintentionally disturb the underlying mental state he wished to study.[163] Janet, therefore, declared it useless and misleading to deploy images

156 According to Janet, when several fixed ideas co-existed in the mind of the same patient, these ideas were mutually dependent and organised in layers. Janet, *Mental State*, 404–5.

157 Janet, 407.

158 Janet, *Major Symptoms*, 333.

159 See in particular Janet, *Idées fixes*.

160 See Janet, 106–8, 347. See also Janet, *Major Symptoms*, 129–30.

161 See, e.g., Janet, *Mental State*, 67–74, 449. See also Janet, *Major Symptoms*, 69–77.

162 Janet, *Mental State*, xiv.

163 Janet, xiv.

with the goal of engaging "in rough anatomy."[164] Such practice, as he warned, would merely result "in not knowing what we look at."[165]

However, I want to emphasise that Janet's criticism was not aimed at the wholesale rejection of empirical images. Instead, I suggest that Janet's criticism specifically targeted those research approaches in which the patient was treated as a representative of a general type. Due to his reconceptualisation of hysteria as a primarily psychological disorder and his insistence on the specificity of every single patient,[166] Janet had to develop a different approach to using images as epistemic tools than Charcot. Janet thus insisted that images of hysteria patients' bodies had to be interpreted in conjunction with additional information, which provided complementary insights into the individual subject's psychology. He asserted that "we should, before all, know well our subject in his life, his education, his disposition, his ideas, and that we should be convinced that we can never know him enough. We must then place this person in simple and well-determined circumstances and note exactly and on the spur of the moment what he will do and say."[167] Contextualised in such a way, visualisations of individual patients' bodily functions could be used to study the patients' changing mental states. This meant that even when he used the same kinds of images as Charcot had, Janet interpreted the images differently.

A pertinent example of Janet's different approach to images as epistemic tools was provided by his use of the perimetric maps, which visualised the contraction of hysteria patients' visual fields. In the previous chapter, we have discussed how Charcot used such images to establish specific patterns common to all hysteria patients, which he then instituted into diagnostic tools. Janet continued to use the same measurement procedures as Charcot to produce perimetric maps. Yet, Janet attributed a different meaning to the resulting images. First, Janet argued that the visual field "contracted in the same manner as the field of consciousness."[168] In other words, unlike Charcot, who ascribed the hysteria patients' concentric contraction of the visual field to a functional lesion of the cerebral sensory centres, Janet claimed that the underlying cause was purely psychological.[169] Second, Janet declared that the most interesting aspect of the visual field was not its particular shape but the extreme variability of its size in a single patient over time. As he stated, the visual field "seems, in its widening and contraction, to follow all the modifications which the mind of the patient undergoes; it is, as it were, the barometer of hysteria for certain patients."[170]

Drawing on this insight, Janet started to systematically examine hysteria patients' visual fields in both spontaneously developed and artificially induced psychological states. He established that depending on whether the patients were tired, emotional, engaged in an intellectual effort, hypnotised or allowed to get drunk, their visual field

164 Janet, xiv.
165 Janet, xiv.
166 Janet, 404.
167 Janet, xiv.
168 Janet, 68.
169 Janet, 68.
170 Janet, 69.

extended and contracted in a highly individual way. Specifically, "[p]reoccupations, emotions, and, above all, fixed ideas in the subject's mind" contracted the visual field.[171] This led Janet to conclude that perimetric maps could be used as indicators of hysteria patients' disturbances of attention. In other words, the more preoccupied the patients were with their fixed ideas, the less attention they could pay to external stimuli. Hence, by systematically producing and analysing perimetric maps, Janet could follow the fluctuating intensity with which fixed ideas invaded a particular patient's consciousness. In Janet's use, these images no longer signified a neurophysiological but instead a psychological dysfunction. It can thus be argued that Janet submitted these images to an intermedial transcription through which they acquired a new function in the clinical context.[172]

Janet also semantically transcribed the visual disturbance Charcot designated as the transposition of the red circle. As discussed in section 1.3.1, Charcot regarded this specific disturbance of colour vision as specific to hysteria and declared it to be one of the disorder's most important diagnostic signs due to its presumed neurological basis. Janet disagreed. He states that the "loss of colours has been examined with exaggerated accuracy; a visual field of colours has been drawn, and efforts have been made to prove that in hysteria this visual field is modified in a regular manner, the visual field of blue, for instance, becoming in this disease smaller than that of red. It may be so, but I advise you to be cautious in this study."[173] According to Janet, what mattered in such cases was "the influence that the association of idea" played in the perception of colours of each individual.[174] To emphasise this point, Janet provided a highly idiosyncratic psychological explanation for one of his patients who exhibited this baffling symptom. "A young woman saw red flowers put on her father's coffin. It made her very angry, because these flowers constituted a political emblem; she now holds red in abhorrence, and has on that account a very fine perception of red and a visual field for red more extended than for white."[175]

Similarly, Janet systematically generated graphic inscriptions of hysteria patients' various respiratory disturbances. Unsurprisingly, all of the resulting inscriptions were characterised by "an absence of regularity and harmony."[176] But far from merely classifying the visual patterns of various pathological modifications of the breathing rhythm, Janet focused on exploring their underlying psychological nature. By comparing multiple graphic inscriptions that were repeatedly obtained for each patient, Janet concluded that a disturbed respiratory pattern persisted as long as that patient "was in a state of absent-mindedness and revery."[177] As soon as the patient's attention was "attracted through any process," the respiratory disturbance vanished, and the

171 Janet, 70.

172 Jäger, "Transcriptivity Matters," 49–50.

173 Janet, *Major Symptoms*, 204.

174 Janet, 205.

175 Janet, 205.

176 Janet, 251. For details on Janet's study of various respiratory disturbances, including respiratory paralyses and hiccoughs, see ibid., 245–64.

177 Janet, 254.

breathing pattern "became again nearly normal."[178] It was under the influence of fixed ideas, which were dominant during the state of absent-mindedness and reduced attention, that various respiratory disturbances came to the fore. By contrast, both the dominance of such fixed ideas and the resulting respiratory problems receded "when the subject was more awake and more active."[179] As the examples concerning both respiratory curves and perimetric maps demonstrate, Janet used empirical images as tools that allowed him to gauge his patients' mental states and thus gain insights into the person-specific dynamics of their fixed ideas.

Yet, even more radically, Janet did not rely exclusively on visualisations of hysteria patients' various physiological disturbances to make inferences about their mental states. He also devised a diagram that allowed him to directly visualise one particular psychological symptom—hysterical amnesia. In this case, his goal was to develop a graphic scheme that displayed "various disturbances of memory in a very simple manner and makes their different varieties clearly perceptible to the eye."[180] The result was a line graph that consisted of two intersecting coordinate axes. The horizontal axis designated "different periods of the [patient's] course of life in their order of appearance."[181] The vertical axis referred to the same period but as a remembrance. Within the thus established temporal coordinate system, 'normal memory' was visualised by a triangle formed between the horizontal axis and the diagonal line drawn from the graphs' zero point. Within this triangle, any deficits in the patient's memory were marked by black areas of different sizes, shapes, and orientations. Simply put, the black areas denoted those visually represented periods from the past that the patient could no longer remember. This simple visualisation enabled Janet to translate various temporal patterns of memory loss into distinct, visually recognisable spatial patterns. At a more general level, Janet used the resulting diagrams to map and classify different types of amnesia.[182] Just as importantly, such diagrams enabled him to gain insights into each patient's idiosyncratic memory loss and to causally relate this loss to particular life events that had possibly triggered it.

Despite such sophisticated ways in which he used different visualisations to gauge and monitor hysteria patients' fluctuating mental states, to be able to cure them, Janet had to go a step further. Hence, he carried out what he referred to as 'psychological research.'[183] This research aimed to uncover the particular content of each patient's persistent fixed ideas by reconstructing the memories of the traumatic events that had initially triggered the formation of the fixed ideas. The process did not just entail measuring and visualising the patients' mental and physiological functions. Janet also closely observed the patients' physiognomy and attitudes, listened to their stories,

178 Janet, 254.

179 Janet, 254.

180 Janet, 70.

181 Janet, 70.

182 For different diagrammatic visualisations of what Janet categorised as continuous amnesia (loss of all memories of events occurring after the onset of amnesia), retrograde amnesia (loss of all memories of events preceding the onset of amnesia), and reciprocal somnambulism (alternating periods of memory loss), see Janet, 69–77; and Janet, *Idées fixes*, 109–55.

183 Janet, *Mental State*, 284.

hypnotised them, and repeatedly engaged them in the act of automatic writing.[184] In short, Janet's 'psychological research' comprised a combined use of both image-based and language-based methods that could be flexibly adapted to each patient's individual character and circumstances.

Yet, Janet insisted that once the content of the symptom-causing fixed ideas was successfully uncovered through his elaborate method, the problem was by far not solved. The toxic fixed ideas did not disappear on their own.[185] Instead, the doctor had to obliterate the mental images that comprised the patient's fixed ideas by displacing them with a set of sufficiently similar but emotionally less negatively charged mental images. To achieve this, Janet used targeted verbal suggestions to introduce a modified mental image into the hypnotised patient's subconscious and thus bring the vicious psychological circle to a halt. For example, after protracted psychological research, Janet determined that in a patient named Marie, "crises of terror were the repetition of an emotion she had experienced in seeing, when she was sixteen, an old woman killed by falling down a stairway."[186] Using suggestion, Janet changed the original image into one in which "the old woman had simply stumbled and was not killed."[187] After that, Marie's crises stopped.

But according to Janet, even if, in response to the treatment, a patient stopped having hysterical symptoms, her cure might have been merely apparent. He argued "that a mind that has been obsessed by a fixed idea remains for some time, even after the disappearance of the fixed idea, in a state of very particular weakness, very open to suggestions and quite in a condition to receive a number of new fixed ideas."[188] For the cure to be complete, the patient's mind had to return "to its state of primitive integrity."[189] In such a case, the patient ceased to be susceptible to suggestion and was, therefore, no longer hypnotisable. Hence, in Janet's psychologically oriented approach to hysteria, suggestion played multiple roles. On the one hand, suggestion is understood as a pathological process underpinning the formation and perpetuation of hysterical symptoms. On the other hand, targeted hypnotic suggestion could be deployed in the clinical context as a potential cure for hysterical symptoms and an indicator of the patient's full recovery.

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184 See Janet, 280–81. To induce automatic writing in his patients, Janet first distracted their minds by engaging them in some conscious activity, such as asking them to read aloud. He then placed a pencil in their anaesthetic hand and, while their mind was absent, suggested that they write a few words. Janet claimed that the patients executed this injunction in an entirely unconscious manner. He also argued that "the automatic writing thus obtained will allow us to verify those sensations, remembrances, and reflections whose existence we had heretofore merely supposed." Ibid., 256. Additionally, he contended that the automatic writing "will reply to our questions and reveal to us a thousand innermost thoughts which the subject would not confide to us or of which even she was completely ignorant." Ibid., 256. For an insightful analysis of the experimental use of automatic writing in psychology, see Koutstaal, "Skirting the Abyss."

185 Janet, *Mental State*, 412.

186 Janet, 284.

187 Janet, 285. For Janet's full account of curing Marie, see ibid., 282–85.

188 Janet, 405.

189 Janet, 405.

In effect, Janet redefined both the treatment of hysteria and the assessment of the patient's recovery in purely psychological terms. As discussed in chapter 1, Charcot's treatment centred on the use of physical interventions, such as massage, hydrotherapy, electrical stimulation, and most of all, exercises that entailed systematic retraining of voluntary movements. Such physical interventions aimed to induce targeted changes in the patients' brain dynamics, thus causing the disappearance of the functional lesions that occupied the cerebral motor and sensory centres.[190] Hence, the effectiveness of the therapy was assessed in strictly physiological terms, as the re-establishment of both normal motor and sensory functions, which was measured and visualised in the form of diagrams.[191] By contrast, Janet relied on hypnosis combined with verbal intervention to manipulate each patient's mental content selectively. His explicit aim was to rid his patients of disturbing fixed ideas, which he defined as "veritable systems of images."[192] Moreover, the potential success of this psychological intervention was determined in decidedly immaterial terms, without any reliance on physiological measurements or any use of empirical visualisations. If Janet's treatment worked, the patient became resistant to the very psychological intervention that had brought on the recovery.

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In sum, my analysis in this section has shown that Janet never explicitly denied the possibility of hysteria having some still undiscovered neurophysiological basis. Yet, in developing his dynamic concept of hysteria as 'a disease due to suggestion,' Janet first and foremost aimed to provide psychological explanations for his patients' heterogeneous symptoms. Such psychological reframing of hysteria allowed him to shift the emphasis away from the search for underlying general types and universal physiological laws, which had characterised Charcot's approach. Rather, Janet placed the focus of his hysteria research on "analysing, in each particular case, the mental state of the patient," whom he understood as a singular individual.[193] With this purpose in mind, in addition to listening to his patients' words—which provided him with information about their life experiences and allowed him to access their mental images—Janet also measured and visualised their physical symptoms. Hence, Janet's investigation of hysteria as a 'mental malady' productively combined immaterial, verbally conjured images, on the one hand, and empirical measurement-based

190 Admittedly, Charcot also sometimes used hypnosis combined with verbal suggestions to treat hysterical symptoms. In Charcot's interpretation, hypnosis produced more or less analogous neurophysiological effects as the physical treatment. Charcot, "Lecture 22: Brachial Monoplegia," 308. Nevertheless, Charcot regarded the methodical physical exercise as "more prudent and often more efficacious." Ibid., 309n. Conversely, he argued that, from the therapeutic point of view, hypnotic suggestion "has not so far given all the results that we were justified in expecting from it. Its scope of action is limited," and its curative effects on hysteria "restricted." Charcot and Tourette, "Hypnotism in the Hysterical," 609. Furthermore, Charcot claimed that hypnosis was less suited to therapeutic purposes as its effects were often difficult to control. Its induction could often lead to the unwitting production of new hysterical symptoms in the patient instead of the cure intended.

191 See section 1.3.2 for details.

192 See Janet, *Mental State*, 244.

193 Janet, *Major Symptoms*, 337.

visualisations, on the other. Yet, in direct opposition to Charcot, Janet did not interpret the empirical images as indicators of the symptoms' underlying physiological basis. Instead, as we have seen, he used them as tools for uncovering the repetitive patterns of the patients' fluctuating mental states, which, in turn, he viewed as manifestations of their pathological fixed ideas. Through such intermedial transcription,[194] Janet radically reshaped empirical images into tools of psychological research.

2.1.3 Freud: Using Language to Uncover the Symbolic Nature of Hysterical Symptoms

Pierre Janet was neither the only nor the most prominent Charcot's pupil who challenged his former mentor's neurophysiological conception of hysteria. In the eulogy he delivered at Charcot's funeral in August 1893, Freud commended his former mentor for having restored dignity to hysteria. Charcot, so Freud, had led to significant advances in the medical understanding of this "most enigmatic of all nervous diseases."[195] However, in the eulogy's closing words, Freud also stated that further advances in the scientific knowledge of hysteria would inevitably "lessen the value of a number of things that Charcot [had] taught us."[196] At that point, Freud was already developing his own theories of hysteria as a purely psychological disorder. As I will argue in this section, it was a direct consequence of his semantic refashioning of hysteria that Freud dismissed empirical images as research tools and shifted to the use of spoken language.[197]

One of Freud's earliest published works on hysteria was an unsigned contribution to Villaret's encyclopaedia from 1888.[198] In this article, Freud largely adhered to Charcot's views. Hence, he attributed hysteria's aetiology exclusively to heredity. Following Charcot, he also stated that the role of all other factors—such as trauma, intoxication, emotional excitement, and organic illnesses—was merely secondary and "as a rule overrated in practice."[199] In another parallel to Charcot, Freud defined hysteria as based "wholly and entirely on physiological modifications" of the "the conditions of excitability in the different parts of the nervous system."[200] Nevertheless, already at this point, Freud also emphasised that the presumed anomaly of the nervous system underpinning hysteria was unrelated to anatomy. Instead, somewhat vaguely, he conjectured that hysteria arose from "the influence of psychical processes on physical processes in the

194 Jäger, "Transcriptivity Matters," 49–50.

195 Freud, "Charcot," 19.

196 Freud, 23.

197 Freud's theorising of hysteria went through several intricate, convoluted and, at times, even mutually contradictory developmental stages. Both the details of this development and the relation of Freud's views on hysteria to his general theories of the human psyche are beyond the scope of this enquiry. For a lucid overview of the historical development of Freud's ideas, see, e.g., Ellenberger, *Discovery of the Unconscious*, 418–570.

198 See Freud, "Hysteria," 39.

199 Freud, 50.

200 Freud, 41.

organism."[201] He further explained that the interplay of multiple unconscious mental processes, such as "changes in the passage and the association of ideas, inhibition of the activity of the will, magnification and suppression of feelings," gave rise to hysteria.[202] But similarly to Charcot, Freud declared that what mattered in these processes was not a particular mental content of conscious and unconscious ideas. Crucial was that these processes induced "a different distribution of excitations" in the nervous system.[203] Thus, although this early article indicated Freud's interest in the role of psychological factors in hysteria, at this point, his approach remained firmly rooted in Charcot's neurological framework.

A more substantial departure from Charcot's views became evident in Freud's comparative study of organic and hysterical paralyses.[204] Interestingly, it was none other than Charcot who suggested to Freud the topic of this study as early as 1886.[205] However, although he had written the first draft in 1888, it was only in 1893 that Freud published the finished article.[206] During this period, marked by his collaboration with the Viennese doctor Joseph Breuer, Freud's views on hysteria began to shift. As a result, in this article, Freud substantially redefined Charcot's key concept of the functional brain lesion as the underlying cause of hysteria. As discussed in chapter 1, Charcot claimed that in hysterical paralysis, a transitory functional lesion causing the symptom was located in the motor centres of the cerebral cortex. Moreover, I have shown that, according to Charcot, such a lesion consisted in the functional inhibition of this centre.[207] In his study, however, Freud posited a different explanation. He claimed that Charcot had erroneously equated the functional lesion underpinning hysteria with a transitory organic disturbance of the brain, "such as an oedema, an anaemia or an active hyperaemia."[208] Freud provided no proof to substantiate his claim. Additionally, he vehemently rejected Charcot's notion that the lesion was anatomically localisable. Contrary to Charcot, Freud contended that if the brain lesion causing hysterical paralysis was indeed a purely functional alteration, it had to be entirely independent of the brain anatomy.[209] He further asserted that to understand the nature of this lesion, it was necessary to abandon the neurophysiological framework and move instead "on to the psychological ground."[210]

In Freud's reinterpretation, a functional lesion underlying hysterical arm paralysis consisted in the inaccessibility of the idea of the arm to the "association with the other

201 Freud, 49.

202 Freud, 49.

203 Freud, 57. For Freud's views on the relationship between psychical (i.e., psychological) and physiological phenomena from this period, see Freud, "Preface to Bernheim," 82–85.

204 See Freud, "Organic and Hysterical Paralyses."

205 Freud, 160.

206 See Freud, 158–59.

207 For a detailed discussion, see section 1.3.2.

208 Freud, "Organic and Hysterical Paralyses," 168. The disturbances listed by Freud refer either to a swelling or to anomalies in the blood flow. I have found no mention of such disturbances in Charcot's lectures on hysteria.

209 Freud, 169.

210 Freud, 170.

ideas constituting the ego."[211] Yet, at this point, Freud no longer referred to the idea in a physiological sense—as a somatic innervation. Unlike Charcot, Freud referred to the idea in a purely psychological sense—as a particular mental content. As he explained, in this case, the idea of the arm was a "popular conception" of this organ, which was derived from "our tactile and above all our visual perceptions."[212] This idea, which in Freud's view represented a precondition for the execution of a voluntary movement, remained in itself unimpaired. Nevertheless, the ego could no longer access it. As Freud somewhat cryptically stated, the idea of the arm became inaccessible because it had been fixated in a subconscious association with a large amount of affect stemming from a memory of a trauma, which had caused the paralysis.[213]

Next, Freud went on to unpack his cryptic claim by explaining that all external stimuli and events generated a surplus of affect or, in other words, an emotional charge.[214] To stay healthy, the ego had to release such a surplus of affect either through some motor reaction or through associative thought activity.[215] If such elimination of the affect was suppressed for whatever reason, the memory of the event attained "the importance of a trauma."[216] In such cases, the undischarged affect remained in the subject's subconscious and became "the cause of permanent hysterical symptoms."[217] The proof for the validity of this explanation, Freud argued, was the fact that once the suppressed affect had been "wiped out," the idea of the arm was "liberated" from the subconscious association, and the hysterical paralysis was thus cured.[218]

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211 Freud, 170.

212 Freud, 170. It is interesting to note that Freud tacitly borrowed this formulation from Pierre Janet. Janet was the first to suggest that "the singular limitation of paralyses and anaesthesias is far more connected with popular ideas than with anatomical boundaries." Janet, *Mental State*, 338. See also Janet, *Major Symptoms*, 154–58. As discussed in chapter 1, unlike Janet and Freud, Charcot interpreted the geometric shapes of hysterical paralyses and anaesthesias as a clear sign of their cortical origin, ascribing them to a functional disturbance of the brain's motor and sensory centres that controlled particular muscle groups or parts of the limb.

213 Freud, "Organic and Hysterical Paralyses," 171–72.

214 Freud's conception of affect has undergone many changes across his different writings and is considered one of the most obscure aspects of psychoanalysis. See, e.g., Solms and Nersessian, "Freud's Theory of Affect," 5. Solms and Nersessian have argued that "the most fundamental of Freud's ideas about affect is the notion that felt emotions are a conscious *perception* of something which is, in itself, unconscious. According to Freud, affects are perceived in a distinctive modality of consciousness that is irreducible to the other perceptual modalities. The qualities of this modality are calibrated in degrees of *pleasure* and *unpleasure*… Affect is further distinguished from the modalities of vision, hearing, somatic sensation, etc., by the fact that its adequate stimuli arise from within the subject, not from the outside world." Ibid., 5–6 (emphasis in original). For an in-depth analysis of Freud's evolving conception of affect, see also Stein, *Psychoanalytic Theories of Affect*, 1–34.

215 Freud, "Organic and Hysterical Paralyses," 171–72.

216 Freud, 172. At this point, Freud did not offer any further explanation for this cryptic formulation. As we will see shortly, in the context of his analysis of the hysterical attack, Freud offered a more precise formulation of his views on traumas.

217 Freud, 172.

218 Freud, 171.

Drawing on my analysis so far, I suggest that the crucial difference between Charcot's and Freud's conceptions of hysteria's underlying functional brain lesion did not primarily consist in the dichotomy between the organic and ideational processes, as implied by Freud.[219] In my view, the crucial difference consisted in the distinct roles that Charcot and Freud ascribed to emotions. In Charcot's approach, the emotional commotion accompanying a physical trauma activated the hereditary and, until then, only latent 'weakness' of the ego, thus allowing the fixed idea of motor paralysis to inhibit the functioning of the cerebral motor centres.[220] Hence, a transitory emotion played merely a precipitating role by invoking a state of consciousness (i.e., a nervous shock) that was conducive to the formation of paralysis. However, in Freud's reinterpretation, it was no longer a pathological idea of paralysis that directly caused the symptom. Instead, the undischarged emotional content that became associated with the unimpaired conception of the affected body part led to the formation of hysterical paralysis. Moreover, the disturbance arising from the undischarged emotional content was no longer localisable to the motor centres of the brain cortex. Freud thus effectively decoupled the functional lesion from cerebral anatomy and placed the affect centre stage in the psychological processes that gave rise to hysterical paralysis.

Having reconceptualised hysterical paralysis, Freud then turned to analysing the hysterical attack. His views on the hysterical attack were summarised in his draft of the "Preliminary Communications," the paper he co-wrote with Breuer and published in January 1893.[221] This draft is significant for our discussion because, as I intend to show, it contained a subtly veiled yet pointed criticism aimed at Charcot's use of images in hysteria research. As the point of departure for his analysis, Freud used Charcot's four-stage model of the major hysterical attack. With his synoptic scheme, so Freud, Charcot succeeded in providing a description of the general type of the hysterical attack, which was inclusive enough to account for a large variety of individual cases.[222] Thus, unlike Bernheim, Freud did not imply that Charcot's visual model was either artificially fabricated or false. Instead, Freud criticised Charcot's approach to studying the hysterical attack for remaining merely descriptive.

According to Freud, the problem with Charcot's visual description was that it failed to provide insights into the attacks' underlying mechanism. It shed "no light at all on any connection there may be between the different phases, on the significance of attacks in the general picture of hysteria, or on the way in which attacks are modified in individual patients."[223] By contrast, Freud declared that he was able to gain deeper insight into the nature of hysterical attacks not by watching or visualising his patients' gestures and facial expressions, but "by questioning them under hypnosis."[224] Talking to his patients

219 Freud, 168–70.

220 For details, see section 1.3.2.

221 See Freud, "Hysterical Attacks," 151–54. Although presumably written in 1892, this draft was first published in 1940. See Freud, *Standard Edition*, 1:146. The final paper was included as the introduction to the famous *Studies on Hysteria*. See Breuer and Freud, "Preliminary Communications," 1–18.

222 Freud, "Hysterical Attacks," 151.

223 Freud, 151.

224 Freud, 151.

allowed him to investigate their changing mental states during the attack and thus penetrate behind the mere surface of the phenomena Charcot had described. Although not explicitly stated, Freud's implication was clear—words appeared better suited than images for uncovering the psychological nature of the hysterical attack. Hence, using spoken language as his research tool, Freud explicitly set out to develop a "theory of the hysterical attack."[225]

Similarly to Janet, Freud asserted that the attacks always entailed the same mental content in each patient.[226] However, unlike Janet, Freud claimed that "the essential portion of a hysterical attack is comprised in Charcot's phase of *attitudes passionnelles*."[227] Freud further asserted that the essence of this particular phase of the attack was a hallucinatory reproduction of the patient's unconscious traumatic memories, which had initially given rise to the symptom. In itself, this statement appeared merely to confirm the views that the Salpêtrians had already espoused.[228] But the novelty of Freud's approach consisted in the explanation he offered about how this pathological "mnemic content" came to exist.[229]

In Freud's view, traumatic memories were produced by a specific psychological defence mechanism. This mechanism facilitated the suppression into the subconscious of all those experiences, ideas, and intentions that evoked unbearable emotions, either because their content was incompatible with the patient's ego or because they clashed with the social restrictions.[230] As a result, the individuals could not free themselves from the "affective states," which thus remained attached to the repressed memory and entered the subconscious.[231] Here, the suppressed affects continued to produce effects in the form of hysterical attacks and other symptoms. Moreover, various additional psychological impressions that either temporally coincided with the repressed memory, or were similar to it, were also suppressed into the subconscious.[232] In the process, these additional mental contents also became a constitutive part of the patient's trauma. In effect, at this point, Freud redefined trauma as a psychological concept whose content was highly subjective. In his vocabulary, trauma no longer referred to a physical injury. Instead, it was constituted by any impression or a set of impressions, even apparently trivial ones, whose accompanying distressing emotional content the individual failed to discharge.[233]

In their jointly authored *Studies on Hysteria*, published in 1895, Freud and Breuer went further in challenging Charcot's views on hysteria. Here, they explicitly repudiated

225 Freud, 151.

226 Freud, 152.

227 Freud, 152. For Janet's reworking of Charcot's four-stage model of the attack, see the previous section.

228 For details, see sections 1.1.2 and 1.1.3.

229 Freud, "Hysterical Attacks," 152.

230 Freud, 153–54. Later, Freud foregrounded the role of ideas, thoughts, and memories of sexual nature as the primary cause of hysteria. See, e.g., "Case of Hysteria," 113–15.

231 Freud, "Hysterical Attacks," 153.

232 Freud, 153.

233 Freud, 154. As is evident here, similarly to Charcot and Janet, Freud also drew on the theory of associationism.

Charcot's fundamental tenet that a hereditary neurophysiological defect was the aetiological cause of hysteria.[234] They asserted that not the heredity but "external events determine the pathology of hysteria."[235] In their view, emotionally charged memories of the patient's past were not acting indirectly, as mere incidental provocative agents, but were, in fact, the direct cause of hysteria. Freud and Breuer succinctly formulated this standpoint by famously declaring that "[h]ysterics suffer mainly from reminiscences."[236] They thus effectively transformed hysteria from an inherited neurological illness—as Charcot saw it—into a disorder of purely psychological aetiology "with affective processes in the front rank."[237]

In a separate paper published in 1894, Freud also introduced a new category of 'neuro-psychoses of defence' or 'psychoneuroses' in which he grouped hysteria, obsessions, and phobias, declaring them all to be mental diseases.[238] According to Freud, the symptoms of all disorders in this group arose through the same psychological defence mechanism, which entailed repressing unbearable ideas into the unconscious.[239] As discussed previously, in Charcot's use of the term, neuroses merely designated neurological disorders that lacked an identifiable organic brain lesion. Freud thus redefined neuroses as purely psychological disorders.

Additionally, to explain how the repressed pathogenic memories acted on the body of hysteria patients, Freud introduced a novel theoretical concept of conversion. In Freud's model, conversion became the fundamental pathological characteristic of hysteria.[240] Freud somewhat vaguely defined conversion as a hypothetical psychological process through which the repressed emotional content was transformed into a chronic somatic symptom.[241] Owing to conversion, the traumatic memory, to which the patient had no conscious access, became substituted by a physical symptom that served as the symbol of this memory. The symbolisation rendered the suppressed memory innocuous while at the same time burdening the patient with a symptom. The symptom, which Freud designated as "a mnemic symbol," lodged itself in the consciousness "like a sort of parasite."[242] Importantly, the distinctive characteristic of the hysterical symbol was

234 See Breuer and Freud, *Studies on Hysteria*.

235 Breuer and Freud, "Preliminary Communication," 4.

236 Breuer and Freud, 7.

237 Freud, "Five Lectures," 18.

238 See Freud, "Neuro-Psychoses of Defence," 43–45.

239 See Freud, 58.

240 Freud, "Five Lectures," 18.

241 See Freud, "Neuro-Psychoses of Defence," 49. Freud did not provide any clear-cut explanation of how exactly the emotional charge (i.e., affect) was "transformed into something somatic." Ibid. He cryptically stated that the conversion "proceeds along the line of the motor and sensory innervation which is related—whether intimately or loosely—to the traumatic experience." Ibid. For a similarly cryptic definition of conversion, see also Breuer and Freud, "Case Histories," 86.

242 Freud, "Neuro-Psychoses of Defence," 49. It is interesting to note that whereas Freud designated the hysterical symptom as a parasite, Janet used the term parasite to refer to hysteria patients' unconscious fixed ideas. See Janet, *Mental State*, 267, 270, 466. In doing so, Janet explicitly drew on Charcot, who used the term parasite to designate any idea that a physician introduced into the mind of a hypnotised subject during hypnosis utilising suggestion. See Charcot, *Oeuvres complètes*, 3:335–36.

that the patient remained unaware of the association between the symptom and the repressed trauma.

The introduction of the concept of conversion had one significant advantage—it allowed Freud to do something that neither Charcot nor Janet had been able to do. Using the concept of conversion, Freud could explain why different patients developed particular hysterical symptoms. Having declared each hysterical symptom to be a symbol of a particular psychological trauma, Freud claimed that each symptom was unambiguously determined by the nature of the patient's personal traumatic experience.[243] Freud differentiated between two types of conversion—conversion by simultaneity and conversion by symbolisation in the narrower sense.[244] In the first case, the memory of the traumatic event was converted into a physical sensation that the patient experienced simultaneously with a trauma. For example, facial neuralgia could develop due to an emotionally painful experience that coincided with a slight toothache. In the second case, the patient developed a symptom as "a somatic expression for an emotionally-coloured idea." [245] In other words, facial neuralgia could also arise in response to a verbal insult that symbolically felt like a slap in the face.[246] The symbolisation was thus the result of the associative linking of ideas that occurred beyond the patient's conscious control. Additionally, Freud argued that the symbolisation was less dependent on personal than on cultural factors since it had the same source as figures of speech, such as metaphors.[247] In Freud's interpretation, the hysterical symptom became a physical expression of personal distress. But, at the same time, Freud regarded such expressions as culturally encoded. His view was thus in direct opposition to Charcot's tenet that hysterical symptoms were "always the same, in all countries, all times, all races, in short universally."[248]

Based on my analysis so far, it can be said that by redefining somatic symptoms as symbols of repressed traumatic experiences and emotions, Freud, in effect, dematerialised hysteria. As a result of his redefinition of hysteria, Freud largely circumvented the physiology, which stood at the very centre of Charcot's research. This also meant that, for Freud, somatic symptoms of hysteria were no longer of interest in themselves. Hence, he took a decidedly different approach to analysing them than Charcot. As discussed in chapter 1, Charcot systematically used various types of visualisations to prove that somatic symptoms of hysteria had a distinct neurophysiological basis. By contrast, Freud used somatic symptoms merely as entry points into the psyche. Owing to such intermedial transcription,[249] the apparent

243 Freud, "Psychical Mechanism," 31. Freud thus directly contradicted Charcot's view (see section 1.3.2) that triggering events and external circumstances in no way determined either the type or the characteristics of the resulting hysterical symptoms.

244 Breuer and Freud, "Case Histories," 178–79.

245 Breuer and Freud, 180.

246 Breuer and Freud, 180.

247 Breuer and Freud, 181. As discussed in chapter 1, Carpenter and Charcot believed that the associative linking of ideas was influenced by the subject's personal habits but primarily determined by the organic nexuses established among the different cerebral centres.

248 Charcot, "Lecture 1: Introductory," 13.

249 Jäger, "Transcriptivity Matters," 49–50.

physiological regularity of hysterical symptoms—as displayed by Charcot's multiple visualisations—no longer retained any epistemic salience. As mentioned above, Freud did not explicitly reject Charcot's visualisations as fabrications. Yet, he regarded them as epistemically irrelevant since they merely described surface manifestations of hysteria and thus failed to disclose the actual nature of this disorder.

Moreover, as I have pointed out previously, the use of empirical images allowed Charcot to bypass his patients' subjective experiences and personal histories, which he treated as noise that needed to be filtered out to obtain 'objective' medical facts. Unlike Charcot, Freud was explicitly interested in his patients' subjective traumatic experiences, repressed ideas, emotional conflicts, idiosyncratic behaviours, and personal statements.[250] Therefore, I argue that Freud did not dismiss images out of reaction to Charcot.[251] Instead, he dismissed images because they could not penetrate the patients' mental states and uncover their highly individual psychological experiences. Put simply, empirical images stemming from measurements of patients' physiological functions were ill-suited to the epistemic requirements of Freud's psychological reorientation that aetiologically decoupled hysteria from the body.

The only images that appeared to fit seamlessly into Freud's hysteria research were those of fleeting and highly subjective nature, such as mental images, dreams, metaphors, and figures of speech. Such images were purposefully elusive and ambiguous.[252] They could, therefore, not be adequately translated into visual representations without destroying their essence. Freud could access such fluid, subjective mental images in all their polysemantic symbolic richness only through language. Hence, I suggest that Freud's use of mental imagery and Charcot's handling of visualisations concerning hysteria occupied two opposite ends of the spectrum. First, all of Charcot's empirical images we analysed in the previous chapter were inscriptions, or to use Latour's expression, immutable mobiles.[253] That is, Charcot produced images that were immutable, mobile, flat, scalable, reproducible, superimposable, and optically consistent.[254] By contrast, the mental imagery Freud dealt with was both immaterial and fundamentally unobservable.[255] Second, at the epistemic level, the aim of Charcot's visualisations was to produce insights generalisable to all cases of hysteria. In direct

250 See, e.g., Breuer and Freud, "Case Histories."

251 See Gilman, "Image of the Hysteric," 415.

252 What I mean here is not that the images generated by Charcot were unambiguous, but merely that—as epistemic tools—they were produced to serve a specific purpose and thus ascribed a fixed meaning. Their potential ambiguity was unintended and interfered with their epistemic function. By contrast, Freud's immaterial images were purposefully ambiguous. See, e.g., Breuer and Freud, "Case Histories," 173–81.

253 Latour, "Visualization and Cognition," 7.

254 Latour, 20–22.

255 Freud did, however, create various graphic visualisations to illustrate different aspects of the psychical apparatus according to his theories. As demonstrated by the medical historian Cornelius Borck, Freud's usage of illustrations was primarily aimed at underscoring the essentially unvisualisable nature of psychological mechanisms. Such images were thereby thoroughly subordinated to the theory and denied any active knowledge-producing role. See Borck, "Freud's Illustrations," 85.

opposition to this, the symbolic meaning of the mental imagery discussed by Freud was interpretable only in relation to each patient's personal experience.

Freud's refocusing of attention from physiology to psychology, from empirical data to subjective accounts, and from visualisable hysterical symptoms to repressed traumatic memories, necessitated the introduction of a new, more adequate research tool. For this purpose, Freud developed the 'analytic method of psychotherapy'—i.e., psychoanalysis—whose cornerstone became the technique of free association.[256] The crux of this technique was to encourage patients to report whatever came to their minds, thus enabling the physician to uncover each individual's suppressed traumatic memories. Significantly, Freud did not use speech only as an epistemic tool with which he generated new insights into the psychological mechanisms underpinning the formation of a particular hysterical symptom. He also used speech as a therapeutic instrument. He claimed that once the repressed memories were made conscious and the accompanying affect released by putting it into words, the hysterical symptoms would disappear.[257] Thus, as a therapeutic instrument, talking fulfilled a twofold purpose. First, it facilitated the process of conversion in the opposite direction. It did so by uncovering the repressed memory that the physical symptom symbolised. Second, by serving as "a substitute for action," [258] the spoken language produced a cathartic effect—it allowed the patient to discharge the strangulated affect that had given rise to the symptom. It can, therefore, be argued that the speech operated both as a precondition for the cure and as the cure itself.

Interestingly, the shift from visual representation to verbal language had one subsidiary effect that fitted smoothly into Freud's framework. In chapter 1, I have shown that Charcot's image-based research effectively compartmentalised the hysterical body into multiple symptoms—each symptom had to be visualised separately using a different type of image or a specifically tailored combination of images. By contrast, Freud was able to integrate all of the patient's heterogeneous symptoms into a single unifying narrative—a case history.[259] The purpose of each case history was to verbally reconstruct the highly individual traces of the concealed memories considered to possess the required traumatic force and the symbolic suitability to cause the patient's symptoms.[260] However, such a narrative reconstruction was by no means a straightforward process. The difficulty was not only due to the patient's subconscious resistance to evoking the repressed memories,[261] but also because the narrative consisted of multiple interrelated layers.

Specifically, Freud contended that a single traumatic event rarely caused hysteria. Instead, in most cases, the disorder arose from what Freud referred to as the summation of partial traumas.[262] New traumatic experiences revived old repressed memories and

256 See Freud, "Psychotherapy of Hysteria," 255–305; and Freud, "Five Lectures," 29–39.

257 Breuer and Freud, "Preliminary Communication," 17. See also Freud, "Psychical Mechanism," 35. As discussed previously, Janet held a different view. See section 2.1.2.

258 Breuer and Freud, "Preliminary Communication," 8.

259 See Breuer and Freud, "Case Studies."

260 Freud, "Aetiology of Hysteria," 191–93.

261 Freud, "Five Lectures," 23–24.

262 Breuer and Freud, "Case Studies," 173–74; and Freud, "Psychotherapy of Hysteria," 287–88.

formed associative links with them. This led to the creation of an elaborate web of symbolic relations among the repressed mental contents, which, in turn, gave rise to mutually interconnected hysterical symptoms. As a result, each hysterical symptom could acquire more than one meaning and thus serve "to represent several unconscious processes simultaneously."[263] Moreover, Freud emphasised that, due to the dynamic interactions among the repressed partial traumas, "a symptom can change its meaning or its chief meaning."[264] Importantly, to cure a patient, it was necessary to discover all partial traumas and their polysemantic relations to one another.[265] Freud thus viewed various symptoms as intrinsic parts of a highly ambiguous and symbolically encoded narrative, whose multiple hidden meanings he could only decipher through the systematic use of language. Instead of measuring and visualising hysterical symptoms in search of their underlying physiological patterns, Freud submitted the symptoms to symbolic interpretations.

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To summarise, my analysis in this and the previous two sections showed that the parallel development of several competing psychogenic conceptions of hysteria at the end of the nineteenth century jointly led to the gradual dismantling of Charcot's neurological understanding of this disorder. Throughout my analysis, I have highlighted how the semantic transcription of hysteria from a brain disease into a mental disorder resulted in a dismissal of images as research tools.[266] However, whereas both Bernheim's and Janet's views were initially highly influential, both researchers fell into oblivion by the early twentieth century.[267] In contrast, Freud's theoretical refashioning of hysteria had far-reaching historical consequences. Owing to the widespread acceptance that Freud's more general psychological theories achieved in the first decades of the twentieth century, hysteria migrated from the domain of neurology to psychiatry.[268] Like the rest of psychiatry, hysteria entered a period during which psychogenic theories of psychiatric illnesses replaced the previously more dominant organic ones.[269]

Within this new theoretical framework, speech became and remained the dominant tool for diagnosing, investigating, and treating hysteria for most of the twentieth century.[270] It thus became the responsibility of a psychiatrist to diagnose hysteria by interviewing patients in order to establish the underlying psychological causes of their symptoms and, subsequently, to treat them through various forms of speech therapy.[271] Furthermore, due to the prevalence of the Freudian psychological model, physiological

263 Freud, "Case of Hysteria," 47.
264 Freud, 53.
265 See Freud, "Psychotherapy of Hysteria," 288–95.
266 Jäger, "Transcriptivity Matters," 49.
267 Ellenberger, *Discovery of the Unconscious*, 89, 406–9.
268 See, e.g., Micale, *Approaching Hysteria*, 28.
269 Shorter, *History of Psychiatry*, 145.
270 See, e.g., Nichols, Stone, and Kanaan, "Problematic Diagnosis," 1267–70; and Stone et al., "Disappearance," 13–16.
271 Stone et al., "Disappearance," 13, 16.

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.