that some of these action-guiding concepts have become increasingly refined across multiple studies, whereas others proved difficult to operationalise experimentally. I will also foreground that a few of these concepts have followed a wayward trajectory of fluctuating epistemic efficacy concerning hysteria. Let us now examine the details of this process and the epistemic effects it has generated in the first two decades of the twenty-first century.

4.1 Examining Hysteria's Relationship to Malingering and Hypnosis

Throughout this enquiry, we have seen that at various points of its history, in clinical and research contexts, hysteria has been repeatedly compared to both feigning and hypnosis. This ongoing comparative investigation has been rooted in the fact that, based on observation alone, hysterical symptoms are "behaviourally indistinguishable" from both their intentionally simulated and hypnotically induced counterparts.[33] We have discussed how this inability to reliably distinguish hysteria from intentional simulation has been perennially framed in negative terms as a hindrance to an accurate diagnosis. Also, we have analysed how Charcot explicitly attempted to tackle this problem by using visualisations of breathing curves as a clinical tool for differentiating between genuine patients and simulators.[34] Just as importantly, I have shown that Charcot drew on the visual similarities between hysterical and hypnotically induced physical symptoms in favourable terms as an epistemic justification for his use of hypnosis to experimentally model hysteria. We are also familiar with the fact that Charcot's use of hypnosis was, at the time, severely criticised by Bernheim but defended by Janet.[35]

If we take into account this long history of their mutual comparison, it is unsurprising that, from the very start, both malingering (i.e., intentional feigning) and hypnosis have played important roles in informing the functional neuroimaging investigation of hysteria.[36] What is equally unsurprising is that this research strand has focused on the symptom of hysterical limb paralysis. This is because, as already demonstrated by Charcot, the behavioural similarities between genuine hysterical and either hypnotically induced or intentionally feigned limb paralysis are particularly easy

33 Ward et al., "Differential Brain Activations," 310.

34 See section 1.2.2.

35 For Charcot's use of hypnosis to model hysterical symptoms, see sections 1.2, 1.2.1, 1.2.2, and 1.3.2. For Bernheim's criticism and Janet's defence of Charcot's approach to hypnosis, see sections 2.1.1 and 2.1.2, respectively.

36 See Ward et al., "Differential Brain Activations"; Halligan et al., "Hypnotic Paralysis"; Spence et al., "Disorder of Movement"; and Stone et al., "Simulated Weakness." As discussed in section 2.2.3, the *DSM-III* introduced a distinction between two types of feigning, which has been retained ever since. Malingering was defined as the intentional feigning performed by an essentially healthy subject. By contrast, factitious disorder was designated as a psychiatric condition arising from an unconscious psychological need to assume the sick role through feigning. See APA, *DSM-III*, 285. The former type of feigning—i.e., malingering—plays a role in fMRI hysteria research. In line with the current neuroimaging literature, in the remainder of this chapter, I will use the terms malingering and feigning interchangeably to refer exclusively to the intentional fabrication of hysterical symptoms by healthy subjects.

to monitor and evaluate visually.[37] Yet, the significant difference to previous approaches is that in the fMRI research, the search for the potential reasons that underpin these apparent similarities takes place at a different physiological level.

Specifically, the starting point of fMRI studies is the externally observable overlap between physical manifestations of hysterical and either intentionally simulated and/or hypnotically induced paralysis.[38] But the explicit aim of such studies is to discover if and to what extent these phenomenologically similar and thus possibly related physical manifestations have a shared neural basis. As discussed in chapter 1, Charcot tried to answer precisely the same question more than a century earlier. However, his means of comparison were limited to photographing his subjects' bodily gestures and facial expressions, measuring their muscular tremors, tracing their breathing pattern, and mapping their anaesthesia. Owing to fMRI, present-day researchers can investigate hysteria's relationship to malingering and hypnosis by comparing the patterns of brain activity associated with each of these conditions, respectively. Importantly, the tacit but so far unproven assumption that informs this comparison is that not just hysteria but also malingering and hypnosis are characterised by distinct and highly specific cognitive processes whose neural correlates can be unambiguously measured by means of fMRI.

As I will show in the following two sections, both the shift in the level at which the comparison is performed and the assumptions that inform it have had significant consequences on how malingering and hypnosis are currently being reframed in fMRI-based hysteria research. I will argue that, on the one hand, this shift has opened up possibilities of providing new insights into the nature of hysterical paralysis. On the other hand, it has also given rise to new methodological challenges that researchers are only gradually learning to address. We will see that although malingering and hypnosis can be designated as vague concepts due to their lack of clear-cut definitions, they have nevertheless been epistemically useful in their action-guiding roles within fMRI-based hysteria research.

4.1.1 Testing Various Conditions of Comparison between Hysteria Patients and Malingering Subjects

At its outset, the application of functional neuroimaging to the study of hysteria appeared to hold the promise of providing "objective evidence of hysterical pathophysiology, distinct from feigning."[39] Such findings, in turn, were expected to

37 For now, it suffices to say that while lying in the scanner and trying to move the paralysed limb on cue—with the paralysis being genuine, simulated, or hypnotically induced—subjects are visually monitored so that researchers can evaluate the quality of their task performance. See, e.g., Stone et al., "Simulated Weakness," 963. In the case of hysterical blindness or anaesthesia, for instance, such external comparison in the quality of the task performance between actual patients and healthy controls simulating these symptoms would not be possible. Researchers would instead have to rely exclusively on the experimental subject's potentially unreliable self-reports to establish the behavioural similarity between the two groups. Why this is important will become apparent in the course of my analysis.

38 See, e.g., Burgmer, "Mirror Neuron System," 438; Van Beilen et al., "Conversion Paresis," 5, e25918.

39 Spence et al., "Disorder of Movement," 1243.

affect how this disorder would be diagnosed in the foreseeable future. However, with the gradually growing number of studies that generated mutually inconsistent results, it soon became apparent that the distinction between genuine and intentionally simulated hysterical symptoms at the neural level was far more elusive than initially hoped.[40] Hence, so far, it has been impossible to unambiguously delineate hysterical symptoms from malingering in terms of distinct underlying neural mechanisms. Yet, in what follows, I will argue that the epistemically productive aspect of this particular research strand was that the authors of fMRI studies have successively learnt to deploy the comparison with deliberate feigning to ask increasingly more complex questions about hysteria. To trace the trajectory of this development, we will now turn to the analysis of three exemplary fMRI studies. In each of the three studies, researchers used intentional feigning to examine different aspects of hysterical limb paralysis.[41]

Published in 2007, the Stone et al. paper on conversion paralysis was the first fMRI study to investigate this hysterical symptom by explicitly comparing it to malingering.[42] The study's objective was very broadly defined by the following two questions: "Does conversion disorder have consistent neural correlates? How do these differ from the neural correlates of deliberately feigned or simulated weakness?"[43] To address these questions, Stone et al. recruited four patients with partial or full one-sided functional leg paralysis that lasted longer than nine months. The researchers also recruited four healthy volunteers of matching age and gender.

Aiming to isolate the neural correlates of hysterical paralysis through fMRI, Stone et al. instructed their experimental subjects to attempt to perform a cued movement. This movement involved first stretching one and then the other ankle by pointing the toes downwards towards the sole. As stated by the researchers, they specifically chose this task because the inability to perform such a movement was "unusual in neurological diseases but common in functional weakness."[44] In other words, Stone et al. decided to use a task that their patients could not carry out and would, therefore, result in "zero or minimal ankle movement" in their affected leg.[45] Unlike patients, healthy controls were asked to perform a slightly different task. It entailed attempting to bend the left or right foot on cue while simultaneously simulating paralysis in one ankle. The initial, somewhat unspecific instruction that the healthy subjects had received was to pretend that one of their ankles was "too weak and heavy to move."[46]

Before data acquisition, both groups of subjects spent thirty minutes on an MRI simulator to train their respective tasks. When asked to describe their experience, the patients reported having "a sense of mental effort" in trying to tense the weak leg.[47] Moreover, this sense of effort was accompanied by "a feeling that the 'message was

40 See, e.g., Stone et al., "Simulated Weakness," 967.

41 In the order in which I will analyse them in this section, these studies are Stone et al., "Simulated Weakness"; van Beilen et al., "Conversion Paresis"; and Hassa et al., "Inhibition."

42 Stone et al., "Simulated Weakness."

43 Stone et al., 961.

44 Stone et al., 963.

45 Stone et al., 963.

46 Stone et al., 962.

47 Stone et al., 963.

not getting through."[48] Based on this description, Stone et al. additionally specified the instruction to healthy subjects on how to simulate hysterical paralysis. They thus directed the 'malingerers' to "reproduce this combination of mental and physical effort when trying to move the feigned weak ankle but not to actually make a movement."[49] To ensure that they complied with the instructions, all subjects were closely monitored during the fMRI measurement. The fact that there were only negligible visually observed differences in the degrees of movements of the 'affected' leg across patients and feigners served to validate that the healthy subjects simulated paralysis with sufficient accuracy.

Following the image preprocessing, Stone et al. first performed single-subject analyses to isolate the neural activation patterns induced by the contrast between the attempted movements in the 'weak' and the 'normal' leg. Subsequent group analyses were performed separately on the fMRI data stemming from four patients with paralysis, on the one hand, and from healthy simulators, on the other hand. These analyses resulted in two fMRI maps, one for each group of subjects.[50] By visually comparing the activation patterns across the two maps, Stone et al. concluded that limb "weakness in established conversion disorder is associated with a distinctive pattern of activation, which overlaps with but is different from the activation pattern associated with simulated weakness."[51] As expected, the shared lack of movement across both groups was reflected in the reduced and more diffuse activation of the motor cortex for the weak relative to the healthy leg in both subject groups. But more significantly, the major difference revealed by the images was that patients but not feigners additionally activated a complex pattern of subcortical brain regions and deactivated parts of the prefrontal cortex.[52] At this point, it may seem as if Stone et al. have succeeded in delineating the neural correlates of hysterical paralysis that were distinct from intentional feigning. However, their apparently straightforward fMRI findings had several caveats.

First, their claim of qualitatively different patterns of brain activity between patients and feigning subjects had limited epistemic validity because it was not derived from a direct statistical comparison of the task-induced effects between the two groups. Instead, as we have seen, Stone et al. based their claim of distinct activation patterns between patients and feigners on the visual comparison of two independently calculated fMRI maps. Additionally, these maps were produced by separately comparing two different tasks with the same baseline condition that entailed a normal movement of the healthy ankle.[53] In neuroimaging literature, inferences drawn from visual comparisons

48 Stone et al., 963.

49 Stone et al., 963.

50 Stone et al., 963.

51 Stone et al., 961.

52 Specifically, patients "activated a network of areas including the putamen and lingual gyri bilaterally, left inferior frontal gyrus, left insula, and deactivated right middle frontal and orbitofrontal cortices." Stone et al., 961. Controls, but not patients, "activated the contralateral supplementary motor area." Ibid.

53 As discussed above, one task consisted in the attempted movement of the actually paralysed ankle, whereas the other in the attempted movement of the ankle while deliberately feigning its paralysis.

of separately calculated fMRI maps are referred to as the "imager's fallacy."[54] Such inferences are considered to lack empirical validity and to have limited epistemic value. The underlying problem entailed in the imager's fallacy is that the "presence versus absence of a significant effect across two comparisons (e.g., groups) does not demonstrate a significant difference between the two."[55] Put simply, to establish if there is an actual significant difference between the two experimental groups, their respective activation patterns have to be compared directly through statistical analysis.

The second, empirically just as problematic caveat in the Stone et al. study was that many clusters of the active voxels, especially in the patients' group-averaged map, dwindled to the point of disappearance after the researchers performed the multiple comparisons correction.[56] Thus, this final step of data processing effectively erased most of the differences in the activation patterns between patients and feigners. Conceivably, the reason for this unwanted outcome was the tiny sample size that consisted of only four subjects per group. Stone et al. tried to circumvent the problem by publishing their findings both as uncorrected and corrected fMRI maps.[57] Yet, they used only the uncorrected maps to delineate and interpret the difference in the patterns of brain activity between patients and feigners.[58] This made their interpretation unreliable since, as discussed in the previous chapter, uncorrected fMRI maps contain multiple false-positive clusters. Hence, it is highly likely that at least some of the areas of activation that Stone et al. discussed in their interpretation as actual findings were mere artefacts of the statistical analysis and thus meaningless.

The third and final caveat concerned the experimental design of the study. Stone et al. deployed a potentially confounded comparison of the failed movement between patients and feigners.[59] Moreover, the instructions they gave to healthy participants of their study on how to simulate hysterical paralysis were decidedly vague. Interestingly, the authors explicitly admitted that their relatively unspecific directions on how to feign paralysis probably induced a mixture of different neurocognitive processes across their experimental subjects. As suggested by Stone et al., some participants might have imagined not being able to move their muscles. Others, instead, possibly imagined that they were faced with an insuperable, imaginary resistance. However, each of these different strategies was "likely to give rise to different patterns of activity" and, consequently, lead to ambiguous results.[60]

Undoubtedly, Stone et al. were well aware of the empirical tentativeness of their fMRI maps. They, therefore, used the maps to make only very hesitant hypotheses about the potential underlying neural mechanism of hysterical paralysis. By interpreting the patients' comparatively more complex patterns of brain activity in the uncorrected

54 Poldrack et al., "Guidelines for Reporting," 410.

55 Poldrack et al., 410.

56 As discussed in section 3.4.3, this procedure is required to minimise the amount of false-positive results, i.e., inactive voxels that during statistical analysis were falsely declared active.

57 See Stone et al., "Simulated Weakness," 964, fig. 1; and ibid., 965, fig. 2.

58 Stone et al., 963.

59 In section 3.1.1, I discussed why using an experimental task that patients cannot perform is considered epistemically inadequate in the current fMRI research.

60 Stone et al., "Simulated Weakness," 968.

maps, the authors conjectured that the individuals with hysterical paralysis attempted "to move with greater resulting mental effort" than feigners.[61] Furthermore, Stone et al. contended that the patterns of activation in the patients' fMRI map probably suggested a "disorganization in the executive control in the movement."[62]

However, the first part of their interpretation appears somewhat circular since it merely reflected the patients' self-reported sense of increased effort. In fact, Stone et al. not only failed to define clearly the notion of the 'mental effort,' but they also used it inconsistently. On the one hand, they invoked mental effort to account for the patients' "more diffuse" pattern of activation on the whole.[63] On the other hand, they also explicitly attributed mental effort to the patients' increased activity in the parts of the parietal and prefrontal cortex that tend to be activated in "tasks demanding attention."[64] Yet, more problematically, the latter part of the researchers' interpretation became even more speculative. Specifically, Stone et al. based their conjecture about hysteria patients' disorganised executive motor control on the activation patterns that largely disappeared after the maps were corrected for false positives. Despite the unresolved methodological challenges they had faced and the resulting difficulties in interpreting the thus obtained imaging findings, Stone et al. nevertheless concluded that intentional simulation appeared to differ from hysteria at the neural level. At the same time, they were forced to admit that their study could not determine this difference unambiguously and that further research was required.

As our following example will show, by explicitly addressing the limitations of the Stone et al. study, other researchers subsequently developed more sophisticated approaches to experimentally operationalising the comparison between hysteria and malingering. The van Beilen et al. study, published in 2011, demonstrates that this new approach entailed a distinctly different way of embedding the concept of intentional feigning into the fMRI-based experimental framework.[65] To begin with, van Beilen et al. drew on the hypothesis that, unlike deliberate feigning, partial hysterical paralysis (i.e., paresis) developed "unintentionally in reaction to psychological and environmental factors."[66] But, as van Beilen et al. stated, precisely this presumed unintentional aspect of hysteria patients' inability to perform normal movements was challenging to study with fMRI. To be more exact, the problem was that fMRI "as a method in general does not discriminate between abnormal task-evoked cerebral activity which causes a symptom, and abnormal activity which is a result of a symptom."[67] As van Beilen et al. pointed out, when "they are moving unnaturally, healthy subjects all show

61 Stone et al., 968.

62 Stone et al., 968.

63 Stone et al., 966.

64 Stone et al., 966. Interestingly, as discussed previously, a similar notion of 'voluntary effort' played a crucial role in Charcot's experiment that relied on the use of graphic inscriptions to differentiate between genuine hypnotic catalepsy (and hysteria) on the one hand, and intentional feigning, on the other. But, contrary to Stone et al., Charcot argued that the feigning subject had to invest voluntary effort to maintain the simulation. For a detailed discussion, see section 1.2.2.

65 Van Beilen et al., "Conversion Paresis."

66 Van Beilen et al., 1, e25918.

67 Van Beilen et al., 2, e25918.

seemingly abnormal cerebral activity."[68] In what can be interpreted as a thinly veiled criticism of the Stone et al. study, van Beilen et al. declared that a simple contrasting of unintentionally developed hysterical and intentionally feigned paralysis in an fMRI study was uninformative and could not be used to isolate their respective neural underpinnings.

To circumvent this problem, van Beilen et al. employed a more complex experimental setup. The underlying idea of their approach was to break down the intended comparison between hysterical and feigned paralysis into several mutually related components. Thus, in addition to comparing nine patients to thirteen healthy individuals who were instructed to feign a partial hand paralysis, van Beilen et al. also included an additional group of controls subjects. This third group comprised twenty-one healthy subjects whose role was to perform the motor tasks normally, without feigning any movement disability. Such tripartite structuring allowed the researchers to compare "the cerebral correlates of conversion paresis (unintentional) abnormal movement to both feigned (intentional) abnormal movement and normal movement."[69]

Just as significantly, to isolate the neural correlates of abnormal movement in both actual and intentionally feigned paralysis, van Beilen et al. chose to use a more complex, multipart motor task. The task entailed not only components of active movement execution but also the so-called explicit motor imagery.[70] In one set of conditions, the experimental subjects were instructed to flex and extend either their left or right wrist at the pace indicated by a flickering dot. This flickering dot appeared on the screen that the patients viewed while they were lying inside the scanner. In another set of experimental conditions, the subjects were asked only to imagine flexing and extending their left or right wrist on cue without performing any movement.[71] All four conditions (i.e., active and imagined movement, left and right hand) were interspersed randomly throughout the experiment. Before the data acquisition, all subjects spent two minutes outside the scanner practising the wrist movements as shown to them by an instructor.

Apart from learning how to perform the tasks, the subjects in the malingering group additionally received the following instruction: "[W]hile you are in the MR scanner you have to simulate a paresis of your right/left hand as you would do if you had to convince a medical examiner that your hand is partly paralyzed, feels heavy and is difficult to move."[72] Stone et al. merely instructed their healthy participants to simulate paralysis during attempted movements but paid no attention to the pauses between these conditions.[73] By contrast, van Beilen et al. explicitly asked their subjects

68 Van Beilen et al., 2, e25918.

69 Van Beilen et al., 3, e25918.

70 Van Beilen et al., 4–5, e25918.

71 This type of task is called explicit motor imagery. In implicit motor imagery tasks, such as the one used in the case study analysed in the previous chapter, participants are covertly induced to imagine performing a particular movement without being aware of it. See section 3.1.1. By contrast, in explicit motor imagery tasks, participants are directly instructed to imagine carrying out a particular movement without actually performing it.

72 Van Beilen et al., "Conversion Paresis," 5, e25918.

73 In the Stone et al. study, each time they were given the cue to try to move the 'affected' ankle, the healthy subjects were also explicitly reminded that this was their weak side. See Stone et al.,

to maintain feigning throughout the experiment. Furthermore, in the van Beilen et al. study, healthy individuals not only had to pretend to have paralysis but also to specifically focus on convincing a medical expert of it. The apparent aim behind these additional specifications was to ensure a continually high quality of simulation throughout the experiment. Finally, to avoid any observable differences in the degree of paralysis between feigners and patients, van Beilen et al. videotaped all subjects during the scanning. Based on these recordings, two independent neurologists quantified the severity of each subject's either actual or feigned paralysis on a 1–5 points rating scale.[74] Hence, unlike Stone et al., van Beilen et al. deployed a quantitative evaluation method. They did so to ensure that the comparison between the neural correlates of actual and feigned paralysis was not confounded by potential differences in the degrees of wrist weakness between patients and control subjects.

In the next step, van Beilen et al. submitted the acquired fMRI data to multiple statistical within- and between-group analyses. First, they generated group-level maps that contrasted the affected to the unaffected side in patients and feigners separately. They duly reported these results yet refrained from committing the imager's fallacy. Instead, to delineate the differences in the neural correlates between hysterical and feigned paralysis, van Beilen et al. computed additional fMRI maps based on a direct statistical comparison between groups. At the level of between-group analyses, the researchers examined the changes in the patterns of brain activity between the subjects' affected and unaffected hands depending on whether the task involved movement execution or imagery. They chose to test multiple contrasts that differently combined these particular aspects of their experimental manipulation across the three experimental groups. These combinations included: a) patients versus normal controls; b) normal controls versus feigners; c) patients versus feigners; d) normal controls versus both patients and feigners; and e) patients versus both normal controls and feigners.[75] Each contrast resulted in a separate fMRI map that visualised a complex pattern of differential neural activations for a particular comparison.

The exact details of the resulting activation patterns are too complex to discuss here. However, what is of interest to our enquiry is that by integrating the findings from their multiple fMRI maps, van Beilen et al. obtained two potentially significant insights. First, by comparing the movement execution of the affected hand in patients versus feigners, van Beilen et al. identified decreased activation in the brain area called the supramarginal gyrus.[76] Additionally, a separately computed fMRI map showed that the same brain area was also underactivated during the imagined movement of the affected

"Simulated Weakness," 963. The obvious implication of this instruction is that the experimental subjects were not expected to maintain feigning throughout the experiment but only on cue.

74 Van Beilen et al., "Conversion Paresis," 5, e25918.

75 Van Beilen et al., 5, e25918. 'Normal controls' is an admittedly inelegant phrase the authors used to emphasise that this group consisted of healthy control subjects instructed to move normally, unlike the other group of healthy control subjects who were asked to feign paralysis. I have adopted this phrase here for the lack of a better, equally short alternative.

76 Van Beilen et al., "Conversion Paresis," 7–8, e25918.

hand in patients compared to both normal controls and feigners.[77] Drawing these two imaging findings together, van Beilen et al. concluded that this abnormal pattern of activity was specific to hysterical paralysis. By referencing neuroimaging literature on the functional role of this area in various neurological conditions, van Beilen et al. proposed that the abnormal activation of the supramarginal gyrus in patients with hysterical paralysis led to the "ineffective movement initiation."[78] In other words, the decreased activity of this brain region appeared to underpin the patients' "unintentional inability to translate conscious motor plans into adequate movements."[79] Second, van Beilen et al. calculated an additional fMRI map for the contrast between patients and feigners for the movement execution with the affected hand. This map disclosed that patients had decreased activations in the prefrontal brain areas and in the region within the parietal cortex called the precuneus.[80] Van Beilen et al. conjectured that this particular pattern of aberrant neural activity "may be specific for the unintentional nature" of hysterical paralysis.[81]

Taken together, the multiple maps generated by van Beilen et al. appeared to demonstrate that there were significant differences in the patterns of brain activity between patients with hysterical paralysis and healthy individuals instructed to feign the symptom deliberately. Based on these maps, the researchers concluded that hysteria patients exhibited not only aberrant "internally generated, movement initiation" but also disturbances "within the hierarchical organization of motor control."[82] Hence, the implication was that hysterical paralysis arose from multiple functional disturbances that affected various stages of volitional movement. But, it remained unclear if and how these different disturbances mutually interacted to give rise to paralysis. Despite this limitation, it can be said that the deployment of a carefully structured multilevel comparison with intentional feigning played an epistemically productive role in this fMRI study of hysterical paralysis. Importantly, I have shown that van Beilen et al. have moved beyond simple experimental contrasting of hysteria and malingering. By developing a more sophisticated experimental framing of malingering, they were able to generate novel hypotheses about the neurophysiological underpinnings of hysterical paralysis.

In a similar vein, another more recent fMRI study employed a comparison with malingering to examine if motor inhibition indeed played a role in hysterical paralysis,

77 In neuroimaging literature, the terms underactivation, hypoactivation, and hypoactivity are used interchangeably. All these terms refer to a decreased activity of a particular region for a given contrast of experimental conditions or across different groups of participants. See van Beilen et al., 8–15, e25918. Consequently, such areas are denoted as underactivated or hypoactive for the given contrast. Conversely, the terms hyper- and overactivation are used to denote an increased activity of a particular region across experimental conditions or groups compared. Ibid. I have adopted this terminology in this chapter.

78 Van Beilen et al., 11, e25918.

79 Van Beilen et al., 11, e25918.

80 Van Beilen et al., 11–12, e25918.

81 Van Beilen et al., 11, e25918.

82 Van Beilen et al., 11–12, e25918.

as suggested by some neuroimaging findings but contested by others.[83] Like van Beilen et al., Hassa et al. also deployed a comparison across three different groups of subjects. They thus contrasted hysteria patients' task-elicited brain activities with those of healthy control subjects in both feigning and non-feigning conditions. However, instead of asking their experimental subjects to either imagine or execute a hand movement, Hassa et al. chose to deploy a different experimental task. They exposed their study participants to passive motor stimulation. This meant that during the fMRI data acquisition, an investigator flexed and extended the participant's right or left wrist at a fixed pace, with periods of rest in between.[84] The subjects were explicitly instructed not to interfere with this externally imposed movement.[85] Yet, the most interesting twist that Hassa et al. introduced into fMRI hysteria research was not limited to the type of motor task they used. Even more importantly, Hassa et al. substantially redefined the empirical implementation of intentional feigning. Specifically, in this study, before scanning, medical experts systematically trained healthy subjects on how to simulate partial hysterical hand paralysis convincingly. In fact, as we will see, this is the only fMRI study in which the otherwise relatively vague action-guiding concept of intentional feigning was defined in clear-cut operational terms.

To this end, twelve healthy subjects underwent a "structured video and mental imagery training" at least thrice a day for six days.[86] Crucially, this meant that all participants were explicitly taught to feign the arm paralysis uniformly. The participants were required to record both the frequency and the exact duration of their training sessions.[87] After completing the training, the subjects were submitted to extensive testing to assess the quality of their feigning and the ability to maintain it for a prolonged period. For this purpose, the subjects were observed during eight "pre-established situations before and in preparation for the MRI."[88] In addition to

83 Hassa et al., "Inhibition." In section 3.5.1, I discussed how de Lange, Roelofs, and Toni challenged the findings of several early neuroimaging studies that had posited motor inhibition as the underlying neural mechanism of hysterical paralysis. As we will see throughout this chapter, whether or not motor inhibition plays a role in hysterical paralysis and if then what type (i.e., conscious or unconscious, externally triggered or internally driven) remains an unresolved question. Hence, we will keep encountering this question in multiple studies when discussing the interpretation of the resulting fMRI maps. For more general neurocognitive research into different types of motor inhibition, see, e.g., Ostilio and Garraux, "Unconscious Control"; and Schel et al., "Stimulus-Driven Inhibition."

84 Hassa et al. chose this particular task because it had been shown to elicit robust "activity in the sensorimotor network that is also active when the movement is voluntarily executed." Hassa et al., "Inhibition," 720. Moreover, Hassa et al. argued that this particular task allowed them to circumvent potentially confounding differences in the subjects' intentions and motivation that are associated with an active motor initiation. Ibid.

85 It is worth reminding ourselves at this point that Charcot often deployed passive movement in his hypnotic experiments with hysteria patients. For details, see section 1.2.2.

86 Hassa et al., "Inhibition," 720.

87 Hassa et al., 722. According to the reports submitted, the overall training duration ranged from 50 to 155 minutes, with half of the participants having trained for more than 100 minutes.

88 Hassa et al., 720. "In one situation the testing was explicit (positioning of the simulated paretic arm on a ball in lying position), while in seven other situations it was implicit: (e.g. lying down on the back, grasping the questionnaire). The subjects knew about the rating of the simulation but

such elaborate pre-scanning preparations, the healthy subjects also received clear-cut directions on how to behave on the day of data acquisition. They were instructed to continually maintain the feigned right-sided hand paralysis not only inside the scanner but from the moment they entered the research facility.

After collecting the fMRI data for all study participants, Hassa et al. computed functional maps based on the statistical comparison of the neural responses triggered by the passive movement of the affected hand between patients and healthy subjects who either did or did not feign paralysis. The resulting fMRI maps delivered some surprising results. The maps showed that both hysteria patients and trained feigners exhibited "neural activity in neighboring but different lateral inferior frontal regions."[89] These areas had been previously shown to be "part of the motor inhibition network."[90] Hence, Hassa et al. suggested that, on the whole, this activation pattern represented "strong evidence" for the major role of motor inhibition both in hysterical and simulated paralysis.[91] Yet, the differences between patients and feigning subjects were just as revealing. During the "passive movement of the affected right hand conversion disorder patients exhibited activations in the bilateral triangular part of the inferior frontal gyri (IFG), with a left side dominance compared to controls in non-feigning condition. Feigning controls revealed for the same condition a weak unilateral activation in the right triangular part of IFG."[92]

In short, the maps revealed that the activated areas across the groups comprised similar but "not exactly the same neural ensembles" of the IFG.[93] Based on this finding, Hassa et al. conjectured that two different types of motor inhibition were involved in hysterical and simulated paralysis. They argued that the motor inhibition was "maintained by an unconscious process" in patients but by a voluntary one in feigners.[94] Moreover, the researchers claimed that their hypothesis regarding the involvement of two distinct types of inhibition was further supported by the clear difference in the activation of the medial prefrontal cortex (mPFC) between patients and feigning subjects. Hassa et al. attributed this differential activity of the mPFC to the patients' disturbed sense of ownership over their actions.[95] In other words, the differential activity of the mPFC suggested that healthy feigners were aware of their own active resistance to the imposed passive movement in the 'affected' limb, whereas patients were not.

From the epistemic point of view, the potential differences in the nature of inhibitory processes between hysteria patients and trained malingered that Hassa et al. disclosed were highly significant. But, in my opinion, a particularly innovative aspect of this study was that it revealed the previously unknown partial resemblance between

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did not know when this would happen. The rating was performed by two trained investigators and documented on an analogue scale from 1 to 5 points for each of the eight situations." Ibid.

89 Hassa et al., 725.

90 Hassa et al., 725.

91 Hassa et al., 725.

92 Hassa et al., 719.

93 Hassa et al., 725.

94 Hassa et al., 725.

95 Hassa et al., 726.

the neural patterns in patients and feigners. As the authors surmised, this discovery probably arose from the fact that they had trained their healthy subjects how to feign paralysis convincingly and to gain the ability to maintain the simulation consistently over extended periods.[96] Hence, by considerably refining the experimental comparison between hysterical and feigned paralysis, Hassa et al. were able to generate imaging results that led to new insights into the underlying mechanism of hysterical paralysis. Importantly, the implication of their discovery was not that hysteria and malingering were identical or even indistinguishable at the neural level. Instead, their imaging results suggested that the loss of movement in hysterical paralysis was underpinned by a related neural mechanism that healthy subjects use to prevent externally imposed movement execution. The key distinction, however, was that in hysteria patients, the triggering of this mechanism happened unconsciously, without the patients' voluntary intervention. Interestingly, as discussed in chapter 1, Charcot had posited a similar conjecture more than a century earlier using imaging methods that remained limited to visualising the surface of the patients' bodies.[97] But, as opposed to Hassa et al., Charcot had tentatively localised the presumed neural disturbance in the sensory and motor centres of the brain.

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Taken together, all the findings analysed in this section are strictly preliminary, and it remains to be seen if future fMRI studies will support or refute them. For this reason, the aim of my discussion was not to evaluate their epistemic validity. Rather, I set out to show how intentional feigning developed from a vague empirical notion into a useful action-guiding concept whose operational character became increasingly more clearly defined across these three exemplary studies. Initially, malingering was framed as a somewhat uncontrolled intentional production of a fake symptom that, on the surface, resembled its hysterical counterpart. The aim was a simple contrasting of a 'genuine' and a 'fake' symptom for the sake of determining their presumably distinct neural correlates. However, as we have seen, not only was such comparison too unspecific, but it was also confounded by the fact that healthy subjects were left to their own devices concerning which mental strategy they chose to use when simulating. Unsurprisingly, the imaging results thus obtained proved ambiguous and difficult to interpret. Yet, by drawing on the limitations of the early findings, the authors of subsequent studies have developed more fine-grained and precisely defined comparisons. These entailed deploying multipart experimental tasks and comparing the patients' neural patterns not just to feigners but also to healthy subjects who 'acted normally.'

But even more importantly, I have underscored how, across the studies, the researchers have gradually introduced stricter operational definitions of intentional feigning. They did so by beginning to more clearly instruct and even explicitly train their healthy subjects how to simulate hysterical paralysis with sufficient quality, as well as how to maintain the high quality of simulation for extended periods. Especially in the Hassa et al. study, the intentional feigning was no longer limited to a mere

96 Hassa et al., 725.

97 See section 1.3.2.

production of a fake symptom that appeared similar to an actual hysterical symptom. Instead, it entailed using a clearly prescribed underlying mental strategy, thus ensuring that feigning was characterised by more uniform neural correlates across the study participants. As foregrounded by my analysis, it was owing to the increasing specificity with which intentional feigning was defined in operational terms that this action-guiding concept could be deployed productively to generate fMRI maps, which revealed surprising new insights into hysteria.

To summarise, despite the long history of relating hysteria to intentional feigning in both clinical and research settings, their mutual comparability was not a given in the context of fMRI experiments. Instead, the comparability of hysteria and malingering first had to be constructed by dividing their experimental comparison into multiple components and training healthy control subjects how to feign a hysterical symptom in a uniform and consistent way. Having thus been adapted to the procedural logic of an fMRI experiment, the action-guiding concept of malingering became epistemically productive in relation to hysteria.

4.1.2 Discovering Similarities and Differences between the Neural Patterns Associated with Hypnosis and Hysteria[98]

The previous section has outlined how the fMRI-based experimental comparison of hysteria and intentional feigning has systematically focused on searching for potential differences in their respective neural underpinnings. Conversely, functional neuroimaging investigation of the relationship between hysteria and hypnosis set out to identify their presumably shared neural basis by focusing on the symptom of limb paralysis.[99] The explicit intention has been to revive the approach Charcot had instituted more than a hundred years earlier, in which hypnosis was used to experimentally model hysterical symptoms.[100] As discussed earlier, in Charcot's deployment, this approach comprised measuring, visualising, and comparing various physical characteristics of hysterical symptoms and their hypnotically induced counterparts.[101] By contrast, we will see that in present-day fMRI studies, researchers compare hysterical to hypnotically induced symptoms by using functional brain maps to examine a potential overlap in their underlying neural patterns.

However, such a shift in the level of comparison from external to internal physiological processes has generated some unexpected results. As my analysis will show, several recent fMRI studies that compared hysterical with hypnotically induced limb paralysis using identical experimental tasks have discovered not only similarities but also significant differences at the neural level.[102] Such findings have raised the question of whether hypnosis can be used to adequately model hysteria in fMRI

98 An earlier version of this section was included in part in a published journal article. See Muhr, "Hypnotised Brain."

99 Halligan et al., "Hypnotic Paralysis," 986.

100 Halligan et al., 986.

101 For a detailed discussion, see sections 1.2.1, 1.2.2 and 1.3.2.

102 See Burgmer et al., "Mirror Neuron System"; and Cojan et al., "Self-Control."

research. Hence, this section will trace the trajectory that hypnosis as an action-guiding concept has followed in fMRI-based hysteria research—from an initially promising experimental model of hysteria to one of questionable adequacy. Throughout the section, I will highlight how functional brain maps have facilitated this revision. But before we turn to analysing the individual studies that have shaped this trajectory, we need to examine how the scientific understanding of hypnosis has changed since Charcot's time. In other words, we must first take a look at how hypnosis is operationally defined in the current fMRI research.

Despite the growing scientific research that focuses on elucidating its nature and on using it as a model for exploring a range of neurological and psychiatric disorders, including hysteria, hypnosis remains vaguely understood.[103] The current hypnosis research combines multiple methodological approaches that target behavioural, phenomenological, physiological, and neurocognitive aspects of hypnosis.[104] However, one major issue is that this research has been unable to resolve the long-standing controversy, which can be traced back to the initial conflict between Charcot and Bernheim. Is hypnosis a distinct altered state of consciousness determined by specific yet unknown underlying neurophysiological changes, as conjectured by Charcot? Or is it a subjective psychological experience shaped by the hypnotised individual's compliance with the hypnotist's suggestion, as claimed by Bernheim? To put it more directly, experts continue to disagree on whether the hypnotised subject's altered state of consciousness is a defining physiological characteristic of hypnosis or "merely one of the many subjective effects of suggestion."[105] Both the neurobiological and the sociocognitive perspective, as they are currently called, have their fervent supporters.[106]

From the neurobiological perspective, hypnosis is operationally defined as a distinct neurophysiological state characterised by "a change in baseline mental activity."[107] This neurophysiological change is, in turn, "experienced at the subjective level as an increase in absorption, focused attention, disattention to extraneous stimuli and a reduction in spontaneous thought."[108] Such an altered state of consciousness "in which normal patterns of communication between separate cognitive systems are perturbed" is called the hypnotic trance.[109] It is typically elicited through a formalised procedure of hypnotic induction. While inside the scanner, experimental subjects receive standardised verbal instructions via headphones. The purpose of the instructions is to induce hypnotic

103 For a general historical overview of hypnosis research in the twentieth century, see McConkey, "Generations and Landscapes."

104 See Jamieson and Hasegawa, "New Paradigms," 133–37.

105 Lynn et al., "Hypnosis and Neuroscience," 145.

106 For detailed accounts of different positions in this debate, see, e.g., Jamieson and Hasegawa, "New Paradigms"; Kihlstrom, "Domain of Hypnosis"; and Lynn et al., "Hypnosis and Neuroscience."

107 Oakley and Halligan, "Hypnotic Suggestion," 264.

108 Oakley and Halligan, 264. For summaries of neuroimaging research on hypnosis, see Barabasz and Barabasz, "Hypnosis and the Brain"; Kihlstrom, "Neuro-Hypnotism"; Oakley and Halligan, "Hypnotic Suggestion"; and Oakley, "Hypnosis, Trance and Suggestion."

109 Oakley and Halligan, "Hypnotic Suggestion," 265.

trance through suggestions of attentional absorption and relaxation.[110] For instance, in one fMRI study of hypnotic paralysis, the induction comprised: "(1) visual fixation on a projected central cross-hair and listening to the experimenter's voice; (2) suggestions of ocular fatigue at continued fixation, eye closure and deep physical (muscle) relaxation along with counting 1–20; and (3) instructions for relaxed and passive multimodal imagery ('Special Place' or 'Safe Place')."[111] Several neuroimaging studies have associated such a controlled induction of hypnotic trance with distinct changes in the patterns of neural activity.[112] Overall, however, the results are inconsistent and have so far failed to unambiguously prove the existence of an unequivocal neural basis of the hypnotic state.[113]

Following the induction, a variety of typical hypnotic effects can be produced. These include different "alterations in sensory experience and motor control, amnesia and the adoption of false beliefs about the self and the environment."[114] The production of each such phenomenon requires a targeted suggestion. If successful, the suggestion produced effects that hypnotised individuals subjectively experience as entirely involuntary, as if happening by themselves.[115] For example, hypnotic paralysis is produced by verbally suggesting to an experimental subject that the limb on one side of their body has become progressively heavy, stiff, and immobile.[116] If responsive to this suggestion, the hypnotised subject loses all voluntary control over that particular limb.

In the so-called intrinsic research into hypnosis, multiple neuroimaging studies have aimed to identify distinct neural correlates of various physical effects induced through targeted verbal suggestion.[117] These effects included altered pain perception, hypnotic blindness, auditory hallucinations, and involuntary movements. However, to this date, the imaging findings generated by this research remain inconclusive and

110 Initially, there were some concerns that the efficacy of hypnotic induction could be negatively affected by the unavoidable features of the fMRI scanning procedure. These included the protracted duration and noisiness, the claustrophobic atmosphere of the scanner, and the need to convey the instructions and suggestions remotely via headphones. One study tested this explicitly and concluded that the features of the fMRI environment had no measurable adverse effect on either the hypnotic condition or the subjects' responsiveness to suggestions. See Oakley, Deeley, and Halligan, "Hypnotic Depth," 54.

111 Deeley et al., "Suggested Limb Paralysis," 414.

112 Oakley and Halligan, "Hypnotic Suggestion," 264–65.

113 See Lynn et al., "Hypnosis and Neuroscience," 154–60.

114 Oakley and Halligan, "Hypnotic Suggestion," 264.

115 Halligan and Oakley, "Hypnosis and Beyond," 112.

116 See, e.g., Cojan et al., "Self-Control," 872. Interestingly, as discussed previously, Charcot induced hypnotic phenomena through explicit verbal and implicit non-verbal suggestions, such as touch and gesture. By contrast, all neuroimaging studies analysed here used only verbal suggestions. This can probably be attributed to the fact that non-verbal suggestions would be difficult or impractical to administer to a subject who has to lie motionless inside the scanner.

117 Intrinsic research focuses on exploring the nature of hypnosis in its own right. By contrast, instrumental research uses hypnosis "as a tool for exploring other psychological processes and phenomena." Oakley, "Hypnosis as a Tool," 3. For an overview of intrinsic neuroimaging research into hypnosis, see, e.g., Oakley, "Hypnosis, Trance and Suggestion," 372–78.

tenuous.[118] Nevertheless, such provisional findings, which have linked hypnosis to distinct, potentially identifiable neurocognitive mechanisms, provide the conceptual basis for functional neuroimaging studies that compare hypnotically induced to hysterical paralysis.[119] Hence, in a striking parallel to Charcot, targeted use of suggestion once again plays a role in contemporary hypnotic modelling of hysterical symptoms. Even more importantly, in another parallel to Charcot, in the current neuroimaging research, a targeted suggestion is understood to induce changes in the hypnotised subjects' perception, thoughts, and behaviour by producing still unknown modifications in their brain activity.[120]

Yet, some of Charcot's other central tenets about hypnosis have been explicitly discarded in the current neuroimaging research. For instance, although subjects can be induced through hypnotic suggestion to perform actions they perceive as involuntary, current research does not support Charcot's view that hypnotised subjects at any point act like mere automatons.[121] Current research has also dispensed with Charcot's claim that hypnosis is primarily a pathological condition.[122] Consequently, whether they investigate hypnosis in its own right or deploy it to model hysterical symptoms, present-day researchers no longer use patients. Instead, unlike Charcot, they recruit healthy volunteers, most often university students.[123] In fact, to qualify as study participants, healthy volunteers have to undergo extensive medical screenings to verify that they are free from psychiatric and neurological disorders. Moreover, current research has also rejected Charcot's division of hypnotic phenomena into three distinct stages, which, as he claimed, were defined by distinct and measurable physical signs.[124] In the present-day context, Charcot's three consecutive stages of hypnosis have been displaced by the new categories of hypnotic depth and hypnotisability. As we are about to see, these two categories serve to quantify differences in subjects' responses to both the hypnotic induction and the subsequent targeted suggestions.

Hypnotic depth is defined as the subjectively perceived intensity of the individuals' experience during hypnosis.[125] Put simply, this measure designates the level of hypnotic trance as estimated by the hypnotised individual. What matters from the perspective of fMRI research is that variations of hypnotic depth have been shown to produce measurable changes in the neural activity.[126] To avoid such unwanted confounds, researchers strive to maintain a constant level of hypnotic depth in each subject throughout the experiment. Just as importantly, researchers also aim to obtain a comparably high level of hypnotic depth across all participants in their group studies.[127]

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118 See Lynn et al., "Hypnosis and Neuroscience," 147–50.

119 See, e.g., Cojan et al., "Self-Control," 862–63.

120 By contrast, Bernheim explicitly denied that hypnotic effects produced through suggestion could be related to the activity of localised cerebral centres. See section 2.1.1.

121 Barnier and Nash, "Introduction," 1.

122 Laurence, Beaulieu-Prévost, and du Chéné, "Measuring," 230.

123 See, e.g., Cojan et al., "Self-Control," 872; and Deeley et al., "Suggested Limb Paralysis," 413.

124 For details, see section 1.2.

125 Oakley, Deeley, and Halligan, "Hypnotic Depth," 34.

126 Oakley, "Hypnosis, Trance and Suggestion," 382–83.

127 Cojan et al., "Self-Control," 873.

But to achieve this, researchers have to be able to assess the experimental subjects' hypnotic depth. This, however, has proven challenging because, by its very definition, hypnotic depth is an experiential measure that cannot be determined based on the experimental subjects' observable behaviour. Instead, to determine the hypnotic depth, functional neuroimaging studies rely on subjects' verbal self-reports.[128] Hence, before fMRI data acquisition, hypnotised subjects, who had been specifically trained for this in pre-scanning sessions, are asked to rate and report their hypnotic depth on a given numerical scale.[129] In some studies, researchers also ask their subjects to repeatedly rate the hypnotic depth during the pauses between the task conditions to ensure that the effects of the induction have not worn off.[130] Despite such comprehensive efforts at quantifying it, hypnotic depth remains a distinctly subjective measure that appears difficult to compare across individuals.

Another key descriptive measure used in contemporary research to identify variations in hypnotic effects across individuals is hypnotisability or hypnotic susceptibility. This measure denotes "the extent of a subject's behavioral response to hypnosis."[131] Different standardised scales for measuring hypnotisability were developed in the second half of the twentieth century.[132] The two most widely used are the individually administered Stanford Hypnotic Susceptibility Scale: Form C (SHSS:C)—which is referred to as the 'gold standard' in hypnosis research—and the group-administered Harvard Group Scale of Hypnotic Susceptibility: Form A (HGSHS:A).[133] To deploy these scales, researchers first have to induce their subjects into a hypnotic state and then expose them to a predetermined sequence of standardised test suggestions of increasing difficulty. The standardised test suggestions systematically

128 In general hypnosis research, alternative methods of measuring hypnotic depth that do not depend on verbal self-reporting have also been developed. For example, hypnotised subjects were given a hand-held device and asked to move its dial to indicate continual changes in their hypnotic experience. For details, see McConkey, Wende, and Barnier, "Measuring Change." But due to the spatial limitations of the scanner, the use of such a device proved impractical in fMRI studies. See Oakley, Deeley, and Halligan, "Hypnotic Depth," 34.

129 See Cojan et al., "Self-Control," 872–73. Multiple standardised self-report scales of hypnotic depth are used in hypnosis research. Yet, different scales deploy different self-evaluation criteria and non-overlapping numerical scales (e.g., 0–10, 1–10, or 1–50+). See Cox and Bryant, "Advances," 317–18. For a detailed comparison of some of these scales, see Tart, "Self-Report Scales." Interestingly, in none of the case studies I analyse in this section have the authors specified which of the standard self-report scales they had deployed. See Cojan et al., "Self-Control," 873; Burgmer et al., "Mirror Neuron System," 438; and Halligan et al., "Hypnotic Paralysis," 986.

130 Cojan et al., "Self-Control," 872–73.

131 Barnier and Nash, "Introduction," 10.

132 For details, see Woody and Barnier, "Hypnosis Scales."

133 Kihlstrom, "Hypnosis," 31. Forms A, B, or C are various versions of the same scale used for different screening purposes. Woody and Barnier, "Hypnosis Scales," 255–56. The Stanford and Harvard scales are not mutually exclusive. In fact, the "optimal screening procedure for hypnosis research is to begin with HGSHS:A, which allows subjects to familiarize themselves with hypnotic procedures, and also provides a first approximation of their hypnotizability. Then, high-scoring subjects can be invited to return for a final assessment with SHSS:C." Kihlstrom, "Hypnosis," 30. For details on these scales, see Weitzenhoffer and Hilgard, *Stanford Scale*; and Shor and Orne, *Harvard Scale*.

alter the hypnotised subjects' motor behaviour, perception, and memory.[134] Based on the pre-established scoring criteria, researchers then separately assess the subjects' observable behavioural responses to each test suggestion. The subject's level of hypnotic susceptibility is represented by a single overall score, which is obtained by summing up the individual items on the scale.[135] Depending on the overall score, the individual's hypnotisability is categorised as high, medium, or low.

In effect, this division into different levels of hypnotisability serves to determine the extent to which the standardised "hypnosis-as-procedure" succeeds in generating the intended "hypnosis-as-product" in different individuals.[136] Simply put, hypnosis-as-product is more reliably induced in subjects with high than in those with low hypnotisability. Although hypnotisability is routinely quantified in present-day hypnosis research, the reasons behind its variability across individuals remain unknown.[137] Another question that is still up for debate is whether different levels of hypnotisability represent an innate trait or if they can be modified through training.[138] Researchers who regard hypnosis as mere compliance with the hypnotist's suggestions tend to claim that hypnotisability is a learned ability.[139] The neuroimaging community, by contrast, views hypnotisability as an innate, unmodifiable trait and focuses on searching for its neural correlates.[140]

Significantly, in fMRI studies using hypnosis to model hysterical symptoms, healthy volunteers are first extensively screened with the Stanford and/or Harvard scales. Only those who score as "highly hypnotizable" are selected as study participants.[141] As discussed earlier, Charcot regarded such increased responsiveness to suggestion as an innately pathological state and an indicator of latent hysteria. By contrast, in current research, high hypnotisability is merely registered as a phenomenological fact that allows for easy modelling of hysterical symptoms. Thus, at least on the surface, the selected participants' increased responsiveness to hypnotic suggestion seems to have a purely instrumental role in neuroimaging studies of hysteria. Having said this, however, what typically remains unmentioned in fMRI studies of hypnotically modelled hysterical symptoms is that, on average, less than 10% of the general population receive high scores on the standardised scales.[142] This makes high hypnotisability a relatively rare trait.

Moreover, two recent behavioural studies have suggested that high hypnotisability might be more pronounced among hysteria patients than in healthy individuals

134 Suggestions influencing motor behaviour (such as hypnotic paralysis) are regarded as less difficult than those that induce visual and auditory hallucinations or age regression. See Woody and Barnier, "Hypnosis Scales," 256.

135 Woody and Barnier, 256. Both HGSHS:A and SHSS:C entail a dozen test suggestions, each of which a subject can either pass or fail. Hence, the maximum score that can be obtained is twelve.

136 Barnier and Nash, "Introduction," 7.

137 See Laurence, Beaulieu-Prévost, and du Chéné, "Measuring," 248; and Kihlstrom, "Hypnosis," 21–26.

138 Laurence, Beaulieu-Prévost, and du Chéné, "Measuring," 232.

139 Laurence, Beaulieu-Prévost, and du Chéné, 232.

140 Bell et al., "Hysteria and Hypnosis," 336.

141 Cojan et al., "Self-Control," 872.

142 Kihlstrom, "Patterns of Hypnotic Response," 100.

or those suffering from other psychiatric conditions.[143] These initial results were contradicted by several subsequent behavioural studies that failed to establish any statistically significant evidence of increased hypnotisability in patients with hysterical symptoms.[144] Hence, for the time being, the potential correlation between hysteria and hypnosis remains unresolved at the empirical level. But against the historical backdrop of Charcot's research, we should not overlook the possibility that the current fMRI research could perhaps inadvertently contribute to the revival of a presumably pathological link between increased hypnotic responsiveness and hysteria through its targeted selection of highly hypnotisable experimental subjects.

So far, we have analysed how hypnotic phenomena are currently defined and experimentally framed within the broader context of cognitive neuroscience. Drawing on the insights we have won through this analysis, we can now turn to examining the findings of neuroimaging studies concerning the potential neural overlap between hypnosis and hysteria. The first functional neuroimaging study to explore hypnotically suggested leg paralysis as an experimental analogue for hysterical paralysis was performed in 2000.[145] The single participant in this PET-based study was a 25-year-old man who scored "positively on those items of the Harvard group scale of hypnotic susceptibility dealing with ideomotor responses, motor rigidity, and inhibition of movement."[146] After hypnotic induction and the assessment of hypnotic depth, the researchers used targeted verbal suggestions to produce in their subject a left-sided leg paralysis. Importantly, the male subject's hypnotic paralysis was specifically modelled to match the clinical features of a longstanding hysterical leg paralysis in a female patient, who had been the subject of a PET study the same research team conducted three years earlier.[147]

In both studies, the researchers used the same neuroimaging technology and deployed the identical experimental task. In each case, they instructed the subject to either prepare to move or attempt to move their affected or unaffected leg on cue. Following the data acquisition, Halligan et al. also deployed the same statistical analysis as in the previous study. In doing so, they calculated a PET functional brain map that visualised those brain areas, which had been differentially activated by the subject's failed attempt to move the hypnotically paralysed relative to the intact leg. The resulting map displayed a lack of activation in the motor regions and selectively increased activations in the right orbitofrontal (OFC) and the anterior cingulate cortex (ACC).[148] Crucially, the anatomical location of the hypnotised subject's pattern of activation strikingly resembled the findings obtained three years earlier for the female patient with hysterical paralysis. Strictly speaking, the exact coordinates differed slightly across

143 See Kuyk, Spinhoven, and van Dyck, "Hypnotic Recall"; and Roelofs et al., "Hypnotic Susceptibility."

144 See Goldstein et al., "Dissociation, Hypnotizability"; Litwin and Cardeña, "Seizure Variables"; and Moene et al., "Hypnotizability, Dissociation and Trauma."

145 Halligan et al., "Hypnotic Paralysis."

146 Halligan et al., 986.

147 Marshall et al., "Hysterical Paralysis."

148 Halligan et al., "Hypnotic Paralysis," 987.

the two maps. Yet, the peak activations nevertheless showed "an overlapping spatial distribution located within the same cytoarchitectural regions."[149]

Based on the similar spatial distributions of the brain activations separately identified in the hysterical patient and the hypnotised subject, Halligan et al. drew several conclusions. First, echoing the claims made by Charcot more than a century earlier, Halligan et al. argued that their imaging results supported the view that "hysterical and hypnotic paralysis share common neural systems."[150] They further suggested that owing to this overlap in the underlying neural patterns, hypnotic phenomena could be used as "a versatile and testable model for understanding and treating conversion hysteria symptoms."[151] Finally, Halligan et al. conjectured that hypnotically induced paralysis was produced through top-down unconscious inhibition of voluntary movement, the same neurocognitive mechanism that they postulated to underpin hysterical paralysis in their previous study.[152] To support their interpretation, Halligan et al. quoted findings of early neuroimaging studies, as well as more speculative neurocognitive accounts, which had posited that "frontolimbic inhibitory processes" underlie a variety of hypnotic phenomena.[153] In short, Halligan et al. first tentatively established the relation of analogy between hysteria and hypnosis. Then, drawing on this analogy, they used hypnotic paralysis to explicitly reinforce their previously advanced hypothesis that motor inhibition was the neurocognitive mechanism underpinning hysterical paralysis.

However, with the shift to the fMRI technology and the accompanying refinement in the experimental design we discussed in the previous chapters, the conclusion drawn by Halligan et al. about the role of executive inhibition in both hysteria and hypnosis was challenged. Some researchers suggested that the use of PET technology, due to its limited spatial resolution, may have critically restricted the "investigation of the modulation of motor control systems by suggestive processes, given the anatomical proximity" of the relevant brain regions.[154] Moreover, subsequent functional neuroimaging investigations of hysterical paralysis delivered results that diverged from the findings of the Halligan et al. study. As exemplified by the case study analysed in chapter 3, other researchers identified additional abnormal patterns of task-induced activations in cases of hysterical paralysis. Consequently, several research teams proposed that neural mechanisms distinctly different from executive motor inhibition gave rise to hysterical paralysis.[155]

Explicitly drawing on these conflicting findings, Cojan et al. designed two parallel fMRI studies in 2009. They aimed to investigate the potential role of motor inhibition in both hysterical and hypnotic paralysis by deploying a so-called go/no-go task.[156] In both studies, subjects were first shown an initial visual cue instructing them

149 Halligan et al., 987.

150 Halligan et al., 987.

151 Halligan et al., 987.

152 Marshall et al., "Hysterical Paralysis," B6.

153 Gruzelier, "Working Model," 5. See also Oakley, "Hypnosis and Hysteria," 249–52, 259–62.

154 Deeley et al., "Suggested Limb Paralysis,"413.

155 See, e.g., de Lange, Roelofs, and Toni, "Self-Monitoring"; and Vuilleumier et al., "Sensorimotor Loss."

156 Cojan et al., "Self-Control," 863; and Cojan et al., "Inhibition," 1027.

to prepare to move their left or right hand. Next, depending on the type of the subsequent cue, the subjects were expected either to execute the planned movement by pressing a button (the go condition) or to abort the movement (the no-go condition).[157] These task conditions were designed to separately probe three different stages of movement—motor intention (preparation), execution (go cue), and voluntary inhibition (no-go cue).[158] In their first study, Cojan et al. used this go/no-go task to investigate the neural activations underpinning acute left arm paralysis of ten days' duration in a single female patient.[159] The patient's task performance was compared to a group of twenty-four healthy individuals instructed to move normally, as well as to six additional subjects who feigned left-hand paralysis.[160]

In the second study, the researchers used the same go/no-go task with a group of twelve volunteers. The volunteers performed the task in the 'normal' state of wakefulness and during a hypnotic trance combined with a suggestion of left-hand paralysis.[161] The second study also included a control group of six subjects who were not hypnotised but merely performed the go/no-go task while intentionally simulating paralysis. In both studies, the explicit purpose of including the control group of feigning subjects was to enable the researchers to isolate the neural activations specific to hysterical and hypnotic paralysis, respectively, and distinct from a voluntary simulation.[162] In both studies, the 'malingerers' were "told that they served as controls for a study of stroke patients with hemiplegia, and asked to act 'as if' they were suffering from motor weakness and unable to move their fingers." Hence, the healthy subjects were not provided with much detail on how to simulate paralysis.[163]

In each of these two parallel studies, Cojan et al. computed multiple activation maps that contrasted various aspects of the motor task across the three groups of subjects. Additionally, they also calculated fMRI connectivity maps. The latter maps identified the brain regions that were differently functionally coupled with the primary motor cortex in either hypnotic or hysterical paralysis relative to the 'normal' condition and simulation. Cojan et al. drew a series of conclusions by interpreting all the resulting

157 The visual cues were variously coloured hand images—grey for preparation, green for the go condition, and red for the no-go. See Cojan et al., "Inhibition," 1028.

158 Cojan et al., 1027.

159 Significantly, as emphasised by the study's authors, the fact that their sample included a single patient with a symptom that lasted only a few days makes it questionable if their findings on hysterical paralysis can be generalised to individuals with the chronic form of this disorder. Cojan et al., 1035.

160 Cojan et al., 1027.

161 Cojan et al., "Self-Control," 863.

162 Cojan et al., "Inhibition," 1037; and Cojan et al., "Self-Control," 863. Notably, the latter Cojan et al. study is a pertinent example of how different action-guiding concepts in hysteria research (such as hypnosis and simulation) are not mutually exclusive but can, instead, be fruitfully combined within a single experimental setup. Later in the chapter, we will encounter additional examples that have combined other action-guiding concepts.

163 Cojan et al., "Inhibition," 1028; and Cojan et al., "Self-Control," 873. Both Cojan et al. studies predate the van Beilen et al. study we analysed in the previous section and which, as discussed, marked a shift in the precision with which intentional feigning came to be operationally defined in fMRI hysteria research.

maps. First, based on the normal preparatory motor activity shown in the activation maps, the researchers suggested that motor intention was preserved both in hysterical and hypnotic paralysis. Instead, their findings indicated that, in both types of paralysis, only the subsequent execution of the planned movement was interrupted.[164]

Second, the researchers discovered that both the voluntary inhibition (modelled by the no-go trials in the 'normal' condition) and the intentional simulation of paralysis resulted in the increased activation of the right interior frontal gyrus (rIFG). By contrast, this differential activity of the rIFG was present neither in hysterical nor in hypnotic paralysis during the go condition. In accordance with previous neuroimaging literature, Cojan et al. attributed the task-induced selective hyperactivation of the rIFG to active inhibition of motor commands.[165] They thus concluded that unlike simulation, which "resulted from active suppression of motor output by right IFG," both hypnotic and hysterical paralyses differed from voluntary restraint.[166] In effect, Cojan et al. conjectured that neither hysterical nor hypnotic paralyses acted "through direct motor inhibition."[167]

Third, Cojan et al. argued that the comparison of the maps generated by their parallel studies disclosed not only similarities but also clear differences between neural activations associated with hypnotically induced and hysterical paralysis. To begin with, Cojan et al. listed the similarities between hysterical and hypnotic paralysis. Aside from the aforementioned preserved motor planning, a particularly significant similarity consisted in the hyperactivation of the posterior midline brain area called the precuneus. Additionally, the connectivity maps showed that both in hysterical and hypnotic paralysis, the precuneus also displayed enhanced interaction with the primary motor cortex.[168] Drawing on previous neuroimaging studies, Cojan et al. conjectured that these patterns reflected a recruitment of "multisensory mental imagery and

164 Cojan et al., "Self-Control," 864. This meant that both the hysteria patient and the hypnotised subjects could form covert motor plans.

165 Cojan et al., 866. The claim that hysterical paralysis does not act through active motor inhibition was subsequently challenged by the Hassa et al. study (published in 2017) we analysed in the previous section, as well as by the Dogonowski et al. study (published in 2018) we will discuss in section 4.4.2. See Dogonowski et al., "Recovery"; and Hassa et al., "Inhibition." However, both of these more recent studies attributed motor inhibition to different brain regions. As we have seen, Hassa et al. argued that hysterical paralysis arose from unconscious inhibition that was mediated through the increased activity of the left IFG. Hassa et al., "Inhibition," 725. By contrast, Dogonowski et al. claimed that the inhibition was due to "the excessive 'veto' signal generated in medial prefrontal cortex." Dogonowski et al., "Recovery," 269. Thus, the question as to whether motor inhibition plays a role in hysterical paralysis (and if then which brain regions mediate it) remains unresolved in the current research. Interestingly, both Cojan et al. and Hassa et al. came to the overlapping conclusions that malingering was underpinned by conscious motor inhibition, which, in turn, was associated with the increased activity of the right IFG. Compare Cojan et al., "Inhibition," 1031; and Hassa et al., "Inhibition," 719.

166 Cojan et al., "Self-Control," 869–70.

167 Cojan et al., 871.

168 Cojan et al., "Inhibition," 1034–35; and Cojan et al., "Self-Control," 869–70.

memory, particularly in relation to representations of the self."[169] In short, both hypnosis and hysteria involved an increase in self-monitoring processes.

Apart from this partial overlap, the maps also revealed several patterns of activations in the frontal brain areas, which were specific to hypnosis. The comparison between the hypnotic and normal states, irrespective of the motor task conditions (i.e., prepare, go, no-go), showed a global increase in the activity of the anterior cingulate cortex (ACC) and the orbitofrontal cortex (OFC). Notably, these were the very same frontal regions to which Halligan et al. had attributed the role of active motor inhibition in hypnotically induced paralysis.[170] However, contrary to Halligan et al., Cojan et al. argued that this pattern of activation, because it remained unchanged across all motor task conditions, should be understood as "an effect of 'state' that was not directly related to inhibitory processes underlying hypnotic paralysis."[171] In fact, Cojan et al. suggested that this pattern reflected "motivational factors associated with enhanced focusing and monitoring."[172]

Moreover, hypnosis relative to the normal state was characterised by hyperactivation in the right IFG and deactivation in the right inferior parietal lobule. This activation pattern was similar across the go and no-go trials for both the affected and the unaffected hand.[173] Importantly, this particular activation was absent in hysterical paralysis and, according to Cojan et al., reflected "a modulation in attentional and executive monitoring functions" specific to the hypnotic condition.[174] Drawing these findings together, Cojan et al. posited that hypnotic paralysis involved "a profound reconfiguration of activity within executive control systems mediated by anterior prefrontal and parietal areas."[175] This reconfiguration resulted in the suppression of the subject's responses to external stimuli, thus "allowing internal mental representations generated through the hypnotic suggestion to guide motor behavior."[176]

The comparison of the maps also showed that, unlike its hypnotic counterpart, hysterical paralysis was associated with a notable increase in the activation in a different frontal brain region called the ventromedial prefrontal cortex (vmPFC).[177] The increase in the activation of the vmPFC was present both during the preparation and execution of the movement with the affected hand. During these two task conditions, the vmPFC additionally exhibited a pattern of increased functional connectivity with the primary motor cortex. Quoting neuroimaging studies that had ascribed the activity of the vmPFC to the processes of emotional regulation and introspection of feelings, Cojan et

169 Cojan et al., "Self-Control," 870–71.

170 Cojan et al., 868–69.

171 Cojan et al., 869.

172 Cojan et al., 869.

173 Cojan et al., 868. It should be noted that this general increase of the rIFG activation across all motor task conditions relative to the non-hypnotic state does not contradict the finding discussed above concerning the lack of selective modulation in this region during the go trials of the hypnotically paralysed hand.

174 Cojan et al., "Inhibition," 1036.

175 Cojan et al., "Self-Control," 868.

176 Cojan et al., 872.

177 Cojan et al., "Inhibition," 1036.

al. suggested that this region was "a critical node through which affective information" could "influence voluntary motor control" and thus produce hysterical paralysis."[178]

In sum, Cojan et al. argued that although both hypnosis and hysteria were associated with the increased self-monitoring and memory processes, there were nevertheless significant differences concerning the content and nature of these processes in each condition. Enhanced attentional control and filtering of external stimuli were specific to hypnosis and absent in hysteria. By contrast, the distinctive characteristic of hysterical paralysis consisted in the key role of emotional control and affectively laden memories. It was this particular involvement of emotional processes that the hypnotically modelled symptoms appeared to lack.

Hence, according to Cojan et al., despite their phenomenological similarity, hysterical and hypnotic paralyses were produced by partly related but, in effect, markedly different neurocognitive mechanisms. As analysed above, each neurocognitive mechanism was associated with the activity of the disparate brain regions and entailed mutually distinct cognitive processes. The fMRI findings by Cojan et al. thus directly contradicted not just the conclusion drawn by Halligan et al. but also Charcot's claim that hysteria and hypnosis relied on overlapping neural mechanisms. Importantly, a clear implication of these findings was that hypnotically induced paralysis might not be an adequate experimental model for investigating hysterical paralysis. If, as Cojan et al. suggested, emotional regulation played a crucial role in generating hysterical symptoms, its absence in hypnotically induced paralysis represented a serious epistemic problem. This meant that, when used as an experimental model of hysteria, hypnosis failed to replicate one of this disorder's essential characteristics. Interestingly, Cojan et al. chose not to express this implication explicitly but left it instead to their readers to draw the obvious conclusion.

A few years later, another team of researchers discovered an additional, potentially significant difference between hysterical and hypnotic paralysis. In 2013, Burgmer et al. used hypnosis to replicate a study they had conducted seven years earlier on four patients with hysterical hand paralysis.[179] In other words, just as Cojan et al., Burgmer et al. investigated a potential neural overlap between hypnosis and hysteria by conducting two parallel studies—one with hysteria patients and another with hypnotised individuals. Consequently, in both Burgmer et al. studies, the respective participants performed an identical experimental task. In their hypnosis study, Burgmer et al. recruited nineteen healthy, highly hypnotisable subjects. They scanned these subjects in the 'normal' state and under hypnosis combined with the suggestion of left-hand paralysis.[180] As in their previous study with hysteria patients, Burgmer et al. instructed the highly hypnotisable subjects to perform a motor task consisting of three conditions. These conditions included: first, watching a still image of resting left or right hand; second, passively viewing a video of moving left or right hand; and, third, imitating the movement shown in the video. Burgmer et al. calculated fMRI activation maps by contrasting either the observation or the imitation of the movement

178 Cojan et al., 1035.

179 See Burgmer et al., "Mirror Neuron System"; and Burgmer et al., "Movement Observation."

180 Burgmer et al., "Mirror Neuron System," 438.

during hypnosis with the 'normal' waking state. In each case, the side-specific control conditions of a resting hand served as a baseline.

The fMRI maps calculated to isolate the effects specific to hypnotic paralysis during movement imitation showed decreased activation of several motor areas. The same maps also disclosed increased activations in the anterior cingulate cortex (ACC), middle frontal gyrus (MFG), and the insula.[181] It is presumably due to using another type of a motor task that this pattern of activations implicated partly different brain regions than those identified in the Cojan et al. study. These differences notwithstanding, Burgmer et al. explicitly supported the interpretation posited by Cojan et al. Hence, they also argued that hypnotic paralysis was not attributable to a direct top-down inhibition arising from the engagement of the prefrontal brain areas. This argument was further supported by an additional connectivity analysis, which showed no changes in the functional coupling between the inhibitory frontal regions and the sensorimotor cortex during hypnosis.[182] Based on their maps, Burgmer et al. suggested that hypnotic paralysis was enacted through "a modification of body and motor conceptualization," shifts in attention, increased conflict detection, and "constant self-monitoring processes."[183]

Interestingly, the maps Burgmer et al. calculated for the experimental condition of movement imitation already showed a lack of overlap in the patterns of activations between hysterical and hypnotic paralysis.[184] However, Burgmer et al. chose to ignore these differences, arguing that "[a]ctive movement is problematic to investigate in patients with conversion disorder" since they cannot perform it correctly.[185] Instead, to compare the neural correlates of hypnotic and hysterical paralysis, Burgmer et al. chose to focus on the experimental condition of passive movement observation. They referenced several previous studies of healthy individuals, which had shown that passive movement observation activated "the neuronal network that is also associated with the actual action."[186] Based on these findings, Burgmer et al. conjectured that passive observation could be used to indirectly study movement generation in both hysterical and hypnotic paralysis by elegantly eliminating the need for the potentially confounding active motor initiation.

The central finding of their initial study was that, contrary to healthy subjects, hysteria patients showed a distinct hypoactivation of the cortical motor areas while observing the movement of the affected compared to the unaffected hand. Burgmer et al. suggested that this "failure of movement observation to initiate motor action" reflected "a disturbance in the involuntary, preconscious levels of motor control."[187] Specifically, they concluded that patients with hysterical paralysis were unable to

181 Burgmer et al., 442.

182 Burgmer et al., 443.

183 Burgmer et al., 442–43.

184 Compare Burgmer et al., "Mirror Neuron System," 440–43; and Burgmer et al., "Movement Observation," 1339–41.

185 Burgmer et al., "Movement Observation," 1341.

186 Burgmer et al., 1337. These studies have found that "observation of biological movement typically leads to generation of an internal motor representation of the observed action, enabling the observer to understand and interpret the actions of others." Ibid., 1342.

187 Burgmer et al., 1342.

conceptualise movement by translating "the abstract task specifications into specific muscle commands."[188] Yet, in the study with hypnotically induced paralysis, the experimental condition of viewing the movement of the paralytic as opposed to the unaffected hand produced no differential neural activation.[189] Put differently, the latter finding suggested that, unlike hysterical paralysis, hypnotic paralysis was not associated with the decreased activation of the motor cortex during movement observation of the affected hand. Burgmer et al. attributed this unexpected discrepancy between the neurological underpinnings of hysterical and hypnotically induced paralysis to the differences in the duration of these two conditions. "While most patients with a conversion paralysis are affected by this disease for months, hypnotic paralysis is brief and confounded by the implicit knowledge of its transient nature."[190] They further conjectured that long-lasting motor deficits in hysteria could lead to a compensatory reorganisation of the functional neural architecture that transient hypnotic paralysis could not model. Burgmer et al. thus implicitly raised the question if, due to the possibly insurmountable differences in their chronicity, hypnosis was an adequate experimental model for hysteria.

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In sum, by indicating that hypnosis and hysteria might engage similar brain processes, early PET studies raised hope that hypnosis could be used as hysteria's experimental analogue, as initially practised by Charcot. The potential advantages seemed self-evident. After all, hypnosis offered researchers considerable "control over the type and spatio-temporal characteristics of the impairments produced."[191] At least apparently, it allowed researchers to induce more homogenous symptoms in much larger samples of experimental subjects, who had been preselected to exhibit increased responsiveness to hypnotic suggestion.

However, by employing more sophisticated experimental setups, subsequent fMRI research generated image-based findings that revealed previously unknown neurobiological differences between hysteria and hypnosis. The image-based findings by Cojan et al. and Burgmer et al. have led to a transcriptive re-negotiation of the relationship between hysteria and hypnosis, particularly regarding their presumably shared neurophysiological basis.[192] These studies have shown that despite being "behaviourally indistinguishable,"[193] spontaneously developed hysterical symptoms and their hypnotically modelled counterparts rely on the engagement of partly different brain regions, which are associated with mutually disparate cognitive processes. The crucial distinctions have included the involvement of emotion processing in hysterical

188 Burgmer et al., 1342. Interestingly, Burgmer et al. thus contradicted the finding of Cojan et al. that motor movement preparation is preserved in hysterical paralysis. See Cojan et al., "Inhibition," 1030.

189 Burgmer et al., "Mirror Neuron System," 443.

190 Burgmer et al., 443.

191 Oakley and Halligan, "Hypnotic Suggestion," 268.

192 I am using the term transcription in Ludwig Jäger's sense, as a medium-specific process of meaning production. See Jäger, "Transcriptivity Matters," 64–65.

193 Ward et al., "Differential Brain Activations," 310.

but not in hypnotic paralysis and considerable disparities in the duration between spontaneously developed and artificially induced symptoms. In effect, hypnotically induced paralysis that explicitly was modelled to resemble hysterical paralysis at the purely phenomenological level has been revealed to miss some of the defining features of hysterical paralysis at the neurocognitive level.

Overall, the fMRI studies discussed in this section were epistemically highly productive because they generated image-based discoveries that have challenged the previously held views concerning the presumed analogy between hysteria and hypnosis. Yet, at the same time, these findings have also made apparent the epistemic limitations of using hypnosis, which is scarcely understood in its own right, to guide the fMRI research into an enigmatic disorder such as hysteria by relying exclusively on the externally observable similarities between these two conditions as the starting point for their experimental comparison. That the current fMRI research seems to struggle with these limitations is perhaps best illustrated by the following fact. As of 2013, no new studies that explicitly use hypnosis to model hysteria's somatic symptoms were published by the end of that decade.[194]

Nevertheless, since fMRI research into both hysteria and hypnosis in their own right continues, it remains to be seen if this situation will change. With the increasing understanding of both hysteria and hypnosis, future researchers might one day develop a novel approach to modelling hysterical symptoms through hypnosis. But to avoid unwanted ambiguities, I suggest that in such a case, the use of hypnosis should not be limited to merely phenomenologically replicating hysteria's physical manifestations. Instead, a more productive approach would need to consider the underlying, currently still unknown neurocognitive features specific to hysteria and hypnosis, respectively. Should this happen, hypnosis might once again re-emerge as a potentially epistemically productive action-guiding concept in hysteria research. For the time being, however, its epistemic efficacy in the current fMRI hysteria research appears to be problematic.

4.2 Probing the Neural Mechanisms behind the Patients' Subjective Experiences of Their Symptoms

Apart from aiming to delineate hysteria from malingering and model it through the use of hypnosis, a significant portion of fMRI-based studies in the first two decades of the twenty-first century has focused on the search for the neurophysiological

194 In fact, studies using fMRI to investigate the neural underpinning of hypnotic paralysis have continued to appear. Moreover, the authors of some of such studies have claimed that their findings might have direct implications for hysterical paralysis. See, e.g., Deeley et al., "Suggested Limb Paralysis"; Ludwig et al., "Hypnotic and Simulated Paralysis"; Pyka et al., "Hypnotic Paralysis." But such claims remain questionable since, contrary to the examples analysed above, these more recent studies did not explicitly compare hysterical and hypnotic paralysis using identical fMRI-based experimental setups. Instead, they merely speculated that their hypnosis-specific findings might be extrapolated to hysteria. In this section, I have disregarded such studies. In my opinion, these studies are not part of the fMRI investigation into hysteria but instead belong to the intrinsic hypnosis research.