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.

underpinnings of the baffling clinical features of hysterical symptoms.[195] As discussed previously, much of this research has initially dealt with the symptom of hysterical paralysis. In this context, different research teams have deployed various experimental tasks endeavouring to elucidate which neural mechanism gives rise to hysteria patients' perplexing, externally observable loss of voluntary movement.[196] We have seen that the central and still unresolved question within this strand of research is: At which point of its production (i.e., planning, initiation, or execution) is the voluntary movement in hysterical paralysis impaired? Yet, as my analysis in the following two sections will show, the authors of more recent studies have gradually expanded this somewhat narrow initial focus. In doing so, researchers have begun to investigate a variety of other sensorimotor manifestations of hysteria and use fMRI to pose increasingly more nuanced questions about the nature of hysterical symptoms.

First, fMRI studies of hysterical sensory disturbances have started to appear.[197] Moreover, since 2010, fMRI research into the so-called positive motor symptoms has steadily gained pace.[198] These symptoms include various forms of aberrant or excessive movement, such as tremors, tics, contractures, and gait abnormalities. In addition to paying attention to previously neglected hysterical symptoms, the authors of more recent fMRI studies have also introduced another important shift. They have begun to address the discrepancy between the patients' self-reported sense of impaired control over their sensory and motor functions, on the one hand, and the apparently 'objective' negative results of the clinical tests, on the other hand. Consequently, the major questions these studies deal with are: Which neural mechanisms could be responsible for the patients' subjective sense of limb paralysis—i.e., genuinely wanting to and making an effort to move but not being able to—despite the lack of any detectable neurological damage?[199] Why do both sensory and motor symptoms worsen when the patients pay close attention to them yet diminish with distraction?[200] Why do patients, according to their self-reports, perceive their hysterical tremor as not being self-generated, although clinical tests show that this symptom has all the features of intentionally produced movement?[201]

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195 For a discussion of the salient clinical characteristics of various hysterical symptoms, see section 2.4.2.

196 See section 3.1.1.

197 For studies of hysterical sensory disturbances, see, e.g., Becker et al., "Conversion Blindness"; Mailis-Gagnon et al., "'Hysterical' Anesthesia"; Saj et al., "Mental Imagery"; and Werring et al., "Visual Loss."

198 For studies of positive motor symptoms, see, e.g., Espay et al., "Functional Dystonia"; Espay et al., "Functional Tremor"; Voon et al., "Involuntary Nature"; and Voon et al., "Limbic Activity."

199 Bègue et al., "Metacognition," 261.

200 Spence, "Cognitive Executive," 227.

201 These features include "variable or non-stereotyped movements, distractibility, entrainment (e.g. where movement characteristics such as tremor frequency or dystonic posturing cannot be maintained during contralateral and competing movements), or the presence of a Bereitschafts-potential." Nahab et al., "Sense of Agency," 2, e0172502. Confusingly, all these features are regarded to be defining characteristics of voluntary movements and are typically absent in tremors of organic origin. Ibid.

The overview of these research questions makes it clear that, in addition to the continued search for the potential neural mechanisms that would explain how various hysterical sensory and motor disturbances arise, one other concern has advanced to the forefront of the fMRI-based investigation of hysteria. To put it plainly, present-day researchers have become increasingly interested in using fMRI to delineate the neurocognitive processes that underpin the patients' subjective experiences of their symptoms. Importantly, the underlying axiomatic assumption that informs such studies is that the patients' hysterical symptoms are real and not a product of malingering. Hence, it can be said that this new research strand directly builds upon the findings of the early fMRI studies.

In the following two sections, I will demonstrate that fMRI research into the neurophysiological basis of hysteria patients' perceived lack of control over their bodies has been informed by several action-guiding concepts, which have been borrowed from cognitive neuroscience. These concepts include the sense of self-agency, motor intention, and attention. In each section, we will examine how these concepts have been implemented in fMRI experiments to generate new neurophysiological insights into the subjective aspects of both sensory and motor manifestations of hysteria. I will argue that although still tentative and fragmentary, these new image-based findings have nevertheless succeeded in endowing the patients' subjective experience of their hysterical symptoms with newly won credibility in the medical context.

4.2.1 Searching for the Neural Basis of the Perceived Involuntariness of Hysterical Symptoms

Whereas patients with hysterical paralysis report that their subjectively perceived intention to move results in an inexplicable lack of action, those with tremors and related positive motor symptoms claim that their excessive movements are entirely involuntary. Paradoxically, however, behavioural measurements suggest that the production of positive motor symptoms relies on the same neural pathways as voluntary movements.[202] As discussed earlier, because of such apparently inexplicable incongruities between the symptoms' measurable features and the patients' reported experience of having no control over their symptoms, the medical community equated hysteria with malingering throughout most of the twentieth century. In fact, it is only since the second decade of the twenty-first century that fMRI hysteria research has begun to offer a potential way out of this impasse. From this point onwards, fMRI research has started to facilitate a neurophysiological reframing of the patients' subjective experience "of not being able to will their bodies to do what they want."[203] Just as importantly, this new research strand has also focused on trying to develop a plausible neurophysiological explanation for why the hysteria patient's "body is making movements that they do not want."[204]

202 See, e.g., Voon et al., "Involuntary Nature," 223.

203 Kranick and Hallett, "Neurology of Volition," 313.

204 Kranick and Hallett, 313.

The current reframing of hysteria patients' subjective experiences has drawn on the concept of the 'sense of agency.' This concept has been used in cognitive neuroscience since the late 1990s to explain how the feeling of ownership over our self-generated actions comes about.[205] In cognitive neuroscience, the concept of self-agency "implies a control mechanism that causally relates actions to their effects."[206] Referred to as the 'comparator model,' this control mechanism operates by continually "matching predicted and actually experienced consequences of movement."[207] According to this model, if the comparison between the motor intention and its outcome results in a close match, the subject experiences a strong sense of agency, and the movement feels voluntary. By contrast, a mismatch between the predicted sensory consequences of the intended action, on the one hand, and the feedback from the actually executed movement, on the other, results in a reduced sense of self-agency. In such a case, the subject no longer has the experience of being the cause of one's actions.[208] Instead, the subject perceives the movement as involuntary. Two particular aspects of the comparator model are significant for our discussion. First, in this model, the experience of self-agency is "inferred *retrospectively*, after an action has been performed and its consequences are known."[209] Second, the sense of agency is closely tied to motor intention and is, therefore, also referred to as a "post-intention" process.[210] As will become apparent in the course of this section, this interrelatedness of the concepts of intention and self-agency has had an important role in fMRI hysteria research.

Deploying such a broadly defined concept of self-agency, several studies have used fMRI to search for aberrant patterns of neural activity that could underpin hysteria patients' subjective experience of the symptoms' involuntary nature.[211] The initial assumption of these exploratory studies was that the perceived involuntariness of hysterical symptoms reflected the patients' disturbed sense of agency, which was expected to arise from a break somewhere "along the intention-action-effect chain."[212] However, my analysis will show that since both the location and the exact nature of this putative break were unknown, the precise role of fMRI maps has been to identify such potential breaks. In what follows, I will trace the trajectory through which four exemplary studies have addressed this epistemic challenge with increasing success. These four studies, I will argue, have generated fMRI maps supporting the conjecture that the patients' perceived lack of control over hysterical symptoms might indeed have a potentially identifiable neurophysiological basis.[213]

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205 See Chambron, Sidarus, and Haggard, "Sense of Agency," 1, article 320.

206 Chambron, Sidarus, and Haggard, 1, article 320.

207 Chambron, Sidarus, and Haggard, 1, article 320.

208 Chambron, Sidarus, and Haggard, 2, article 320.

209 Chambron, Sidarus, and Haggard, 1, article 320 (emphasis in original).

210 Roelofs, Teodoro, and Edwards, "Neuroimaging," 3, article 12.

211 See, e.g., Hassa et al. "Inhibition"; Maurer et al., "Impaired Self-Agency"; and Voon et al., "Involuntary Nature."

212 Chambron, Sidarus, and Haggard, "Sense of Agency," 1, article 320.

213 Baek et al., "Motor Intention"; Nahab et al., "Sense of Agency"; Voon et al., "Involuntary Nature"; and Voon et al., "Limbic Activity."

The first study that deployed fMRI to explore why hysteria patients who exhibit aberrant movements perceive them as involuntary was published in 2010.[214] Voon et al. recruited eight hysteria patients with a rare type of so-called intermittent positional hand tremor. The specificity of this type of tremor was that it was absent at rest and that the patients could perform various intentional hand movements without triggering its onset.[215] An additional significant selection criterion in the Voon et al. study was the exclusion of all patients whose tremor entailed head movements.[216] Admittedly, by choosing such a strictly delineated and rare symptom, Voon et al. struggled with recruiting a sufficient number of patients and potentially limited the generalisability of their findings to other types of hysterical tremor.[217] Yet, this symptom was specifically chosen "to permit comparative analysis of voluntary vs. involuntary movement" using an elegant and straightforward task that entailed two conditions.[218] In one task condition, patients were instructed to place the affected arm in a position that triggered their involuntary tremor. In the other task condition, they were asked to use the same hand, while in the asymptomatic state, to intentionally mimic the tremor of the identical frequency and amplitude as their involuntary tremor.

The researchers obtained two significant findings by computing the fMRI activation map that contrasted the brain activities during the involuntary and voluntarily mimicked tremor. First, the fMRI map displayed the absence of differential activation in the primary motor cortex across the compared conditions. The map thus provided empirical support for the aforementioned hypothesis that involuntary and voluntary tremor utilise the same neural pathways.[219] Second, the same fMRI map also revealed reduced activation in the brain region called the right temporoparietal junction (TPJ) during hysterical relative to intentionally mimicked tremor.[220] Significantly, previous neuroimaging studies in healthy individuals suggested that the TPJ plays a crucial role in generating the sense of agency. More specifically, the authors of multiple studies have argued that the comparison between the predicted sensory consequences of the intended movement (i.e., the feed-forward signal) and the actual action (i.e., the sensory feedback) takes place in this region.[221] Yet, contrary to the findings obtained by Voon

214 Voon et al., "Involuntary Nature."

215 Voon et al., 224. "Positional tremors arise when a patient's tremor is brought on during specific positioning of the involved body part. They can be distinguished from postural tremor, wherein a patient's tremor is elicited in any posture, and from task-specific tremor, wherein a patient's tremor occurs only during a certain task." Schaefer et al., "Positional Tremor," 768.

216 Voon et al., "Involuntary Nature," 224. This criterion is typical for all fMRI studies recruiting hysteria patients with positive motor symptoms. Since, as discussed previously, even minimal head movements can render the fMRI data unusable, all patients whose tremor affects their upper body are disqualified from participating in such studies. See, e.g., Baek et al., "Motor Intention," 1625.

217 The symptom's clinical rarity is best illustrated by the fact that to recruit eight subjects who participated in their study, the authors had to screen 156 patients with positive motor symptoms over five years. Voon et al., "Involuntary Nature," 224.

218 Voon et al., 224.

219 Voon et al., 226.

220 Voon et al., 226.

221 Voon et al., 226.

et al., in healthy subjects, a discrepancy between intention and effect that resulted in the perceived loss of agency was associated with the increased activity in the TPJ.

To explore why their patients showed the opposite and thus unexpected effect of reduced activation in this region, Voon et al. used their data to compute an additional task-related connectivity map for the TPJ.[222] The resulting map showed reduced functional connectivity between the TPJ and the brain areas involved in the sensory feedback in hysterical relative to mimicked tremor.[223] In their interpretation of this aberrant connectivity pattern, Voon et al. drew on the fact that the neural pattern in the patients' activation fMRI map did not indicate any disturbance in the sensory feedback. Hence, Voon et al. suggested that the problem might lie in the other component entailed in the comparison—i.e., the feed-forward signal. More precisely, they conjectured that the decreased connectivity could indicate that in hysterical tremor, the "movement arises without conscious intention and there may not be a feed-forward signal."[224] They further hypothesised that with a sensory prediction signal lacking, no actual comparison could occur in the TPJ. Crucially, this conjecture could explain why the patients had decreased activity in the TPJ, as indicated by the fMRI activation map and, at a more general level, why they experienced their tremor as not being self-generated.

As foregrounded by my analysis, Voon et al. succeeded in deploying fMRI maps to generate at least tentative empirical support for the patients' subjective accounts of the involuntary nature of their symptoms. Just as importantly, based on their combined interpretation of the fMRI activation and task-based connectivity maps, Voon et al. managed to provide a more precise formulation for the provisional assumption that hysteria patients had an impaired sense of agency. As we have seen, they attributed the perceived involuntariness of tremor to a possible disturbance in the intentional processes, which, in turn, resulted in the abnormal generation of the movement's sensory predictions. In short, Voon et al. suggested that the patients' impaired sense of agency arose from a break situated in the early stages of the intention-action-effect chain. However, their study was unable to answer why the patients' motor intention was disturbed and how.

In 2011, the same research team published another fMRI study. The new study built directly upon the initial findings and was explicitly designed to address precisely those aspects that had eluded the researchers in their previous study. Hence, this time, Voon et al. focused on delineating the potential impairment of motor intention in hysteria patients with multiple positive motor symptoms.[225] Moreover, in the new study, Voon et al. additionally chose to tackle the broader questions of how and why the patients' aberrant unintentional movements were initiated at the neural level.[226] To address these questions through fMRI, the researchers designed a considerably more elaborate experimental setup than in their previous study. Apart from eleven patients with different positive motor symptoms (tremor affecting different body parts,

222 Voon et al., 226.

223 To calculate the connectivity map, Voon et al. deployed the PPI analysis discussed in section 3.4.4.

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

225 Voon et al., "Limbic Activity, " 2396.

226 Voon et al., 2397.

contractures, and gait disturbance), this study also included age- and gender-matched healthy control subjects.

During the fMRI acquisition, both subject groups carried out a so-called action-selection task. In doing so, the subjects were required to perform "both internally and externally generated movement."[227] The task consisted of a preparation and execution phase, both of which were introduced by visual cues. The subjects were given a response box and instructed to use their right hand to press either the left or the right button, depending on the type of visual cue they saw. During the preparation phase, the subjects either saw a directional cue (arrows pointing left or right) or a neutral one (arrows pointing upward). The directional cues were designed to induce externally determined actions. By contrast, during the neutral cue, the subjects could freely choose which button to press.[228] When a red cross appeared on the screen, the subjects executed the planned action by pressing one of the buttons. The design of this task was derived from the researchers' hypothesis that "the process of voluntarily initiating an internally generated as compared to an externally generated response might engage similar motor preparatory systems utilized during the internal generation of involuntary conversion movements."[229] To put it more plainly, the task was meant to isolate the patterns of neural activity induced by the contrast between freely chosen and externally directed movements in patients relative to healthy subjects. The researchers conjectured that identifying this particular pattern of differential neural activity would allow them to explain why patients, "rather than their intended movement of reaching for a cup, for instance, may experience an involuntary action such as tremor."[230]

Having calculated the activation maps, Voon et al. identified decreased activity in the supplementary motor area (SMA) in patients relative to healthy subjects during the movement preparation phase for both freely chosen and externally directed actions. According to the neuroimaging literature, the SMA is implicated in "the subjective urge and the intention to move," as well as in the sense of being in control of one's actions.[231] Drawing on this literature, Voon et al. suggested that the SMA was "a potential nodal point of motor impairment" in hysteria patients with abnormal movements.[232] This meant that their newly calculated fMRI maps provided empirical support for the hypothesis Voon et al. had put forth in their previous study concerning the impaired intention in patients with positive motor symptoms. In fact, owing to the new maps, Voon et al. were now able to explicitly link the previously hypothesised cognitive disturbance (i.e., impaired intention) to a decreased activity of a specific brain region, the SMA.

Moreover, the current study generated two additional findings. First, the same activation maps that showed decreased activity in the SMA during the movement preparation in patients relative to controls displayed additional patterns of aberrant

227 Voon et al., 2396.
228 Voon et al., 2398.
229 Voon et al., 2397.
230 Voon et al., 2402.
231 Voon, "Functional Neurological Disorders: Imaging", 340.
232 Voon et al., "Limbic Activity," 2401.

activations. These included the increased activity in the limbic brain regions that comprised the amygdala, the anterior insula, and the posterior cingulate cortex.[233] As Voon et al. suggested, this abnormal pattern of hyperactivity meant that patients were assigning undue emotional salience to "external or internal stimuli, states or memories," which, in turn, additionally interfered with the initiation of their intended movements.[234] Second, the task-based connectivity map that contrasted internally with externally generated actions in patients relative to healthy control subjects displayed a decreased neural coupling between the SMA and the dorsolateral prefrontal cortex (dlPFC). The author attributed this aberrant connectivity pattern to "a potential impairment in top-down regulation from regions associated with higher motor control" during movement preparation.[235] In short, the voluntary action selection system appeared to be functionally disconnected from the higher-order control.[236] Importantly, these additional findings provided empirical support for the researchers' initial conjecture that patients had problems translating the intended into actual movements.

At this point, Voon et al. attempted a synthesis of the image-based findings generated by both of their fMRI studies. In doing so, they postulated a potential mechanism to explain how aberrant and excessive hysterical movements arise at the neural level and why patients perceive the resulting movements as involuntary. According to this mechanism, when the patient is under stress, "previously mapped conversion motor representation may hijack the voluntary action selection system."[237] More specifically, due to the decreased activity of the region critical to the motor initiation (i.e., the SMA) and its disconnectedness from the prefrontal brain areas responsible for the top-down regulation of action selection (i.e., the dlPFC), the preparation for the execution of the intended movement is disturbed. At the same time, the abnormally hyperactive limbic regions that are associated with assigning emotional salience may indirectly facilitate the initiation of some previously learnt aberrant movement patterns—i.e., motor representations.[238] Once initiated, such aberrant movement patterns "hijack the voluntary action selection system," thus triggering the manifestation of positive motor symptoms such as tremor.[239]

Next, Voon et al. slightly modified their initial explanation of how the patients' lack of the sense of self-agency arose. By taking into account their more recent findings, this time, they postulated that the "aberrant conversion motor prediction may conflict with intended motor prediction, resulting in a mismatch between prediction and outcome and hence the sense of involuntariness."[240] In other words, not the complete lack of feed-forward signal, as previously hypothesised, but its abnormal generation led to the patient's perception that the resulting action was involuntary. In effect, this new

233 Voon et al., 2400.
234 Voon et al., 2402.
235 Voon et al., 2402.
236 Voon et al., 2396.
237 Voon et al., 2402.
238 Voon et al., 2402.
239 Voon et al., 2396.
240 Voon et al., 2402.

explanation for hysteria patients' loss of self-agency was considerably more precise than the one Voon et al. had previously posited in their initial study.

As we have seen, Voon et al. developed the mechanism detailed above to account for the generation of hysterical tremor and other positive motor symptoms that entail excessive movements. Yet, remarkably, this mechanism shows some surprising parallels to the explanation of the formation of hysterical paralysis (i.e., loss of movement) that Charcot had postulated more than a century earlier. As discussed earlier, Charcot conjectured that in a state of emotional commotion, during which the control of the higher-order cerebral regions was attenuated, a sensory idea (i.e., a mental representation) of limb weakness, which stemmed from the experience of light physical injury, could hijack the brain. Charcot further argued that after a necessary period of unconscious mental 'incubation,' this idea could become dominant enough to inhibit the motor centres of the brain and thus result in paralysis.[241]

Significantly, both the mechanism suggested by Charcot and the one proposed by Voon et al. implicate the role of impaired top-down regulation. Even more importantly, both mechanisms posit that the voluntary motor initiation is hijacked by the involuntary activation of an aberrant, previously mapped mental representation.[242] Nevertheless, there are also some important differences. First of all, the aberrant mental representation in Charcot's mechanism is a sensory idea of limb weakness. By contrast, in the mechanism proposed by Voon et al., the aberrant mental representation consists in a movement programme that was acquired "through implicit learning process."[243] But the crucial differences between these two mechanisms lie elsewhere. The mechanism put forth by Voon et al. is conceptually far more detailed than Charcot's. Moreover, owing to the utilisation of fMRI, each of the purported cognitive components in this mechanism is associated with clearly delineated sets of mutually interacting brain regions, such as the SMA, TPJ, amygdala, insula, and dlPFC. Finally, and this is by no means unimportant, Voon et al. explicitly focused on providing a neurophysiological explanation for why hysteria patients subjectively experience having no control over their movements. Charcot did not, or maybe, due to the limitations of the imaging methods he was using, simply could not explicitly address this particular question.

So far, we have analysed two fMRI studies that utilised the mutually related concepts of self-agency and motor intention to probe how the brain produces positive motor symptoms and why hysteria patients perceive the resulting movements as not being self-initiated. However, Voon et al. only indirectly addressed the hysteria patients' perceived involuntariness of their symptoms. To be sure, Voon et al. used specifically devised cognitive tasks that were meant to isolate the involuntary aspects of hysterical symptoms. Yet, they did so without asking the study participants to assess and report

241 For details, see section 1.3.2.

242 Interestingly, despite such apparent parallels, Voon et al. did not refer to Charcot's conjectures about the underlying mechanism of hysterical symptoms. However, in their initial paper, they made a somewhat laconic comment that "[s]tudies of conversion disorder date back to the work of Charcot." Voon et al. "Involuntary Nature," 223. This comment indicates that they must have been familiar with Charcot's theories.

243 Voon et al., "Limbic Activity," 2397.

on their actually perceived sense of agency. By contrast, two fMRI studies published in 2017 explicitly shifted the focus to examining the patients' metacognitive abilities to accurately judge their own sense of self-agency and the onset of their motor intentions.[244]

In the first of these studies, Nahab et al. deployed a virtual reality task to compare the neural responses induced by externally modulated loss of control over movement between hysteria patients with positive motor symptoms and healthy control subjects. Inside the scanner, the subjects performed sequential finger tapping at their own pace with their right hand. They did so while wearing a data glove that recorded their voluntary, internally generated movements.[245] While performing the finger tapping, the subjects observed a simulated hand on the computer screen that either entirely (100%), not at all (0%), or partially (75%, 50%, and 25%) mimicked their movement in near real-time. The subjects were deliberately not informed about the experiment's goal, which was to assess "how the brain responds" to the perceived loss of self-agency.[246] Instead, the participants were merely told to continue moving their fingers according to their own pace, even if the projected hand did not always do what they intended. Before the fMRI data acquisition, the simulated hand was calibrated to each subject's individual hand movements. Additionally, the subjects practised controlling the projected hand in the 100% condition to develop "a sense of ownership" over it.[247]

The subsequent analysis of the fMRI data showed that in healthy subjects, a network of brain areas, which previous neuroimaging studies have linked to the sense of agency, was differentially activated across the changing task conditions.[248] To be more exact, in healthy subjects, the synchronous activity of multiple brain regions responded in a graded way to the externally manipulated, gradually increasing loss of control over the simulated hand on the screen. By contrast, in patients, some of the same brain areas—particularly the pre-supplementary motor area (pre-SMA) and the dorsolateral prefrontal cortex (dlPFC)—reacted differently. Specifically, both the pre-SMA and the dlPFC failed to be differentially activated by the increasing discrepancy between the voluntary finger movements these individuals were performing and the observed virtual hand motion that they were supposedly thereby controlling.[249]

As discussed above, Voon et al. attributed the aberrant activity of these two particular brain regions to the disturbance of motor intention and its translation into action. Nahab et al., however, extended the finding of their colleagues. Based on the interpretation of the fMRI maps generated by their study, Nahab et al. suggested that the pre-SMA and dlPFC did not only play key roles in motor intention by participating in "the generation of the motor program."[250] The researchers conjectured instead that these brain regions were also "critical components for accurately judging volition."[251]

244 Baek et al., "Motor Intention"; and Nahab et al., "Sense of Agency."
245 Nahab et al., "Sense of Agency," 3–4, e0172502.
246 Nahab et al., 5, e0172502.
247 Nahab et al., 4, e0172502.
248 Nahab et al., 9, e0172502.
249 Nahab et al., 9, e0172502.
250 Nahab et al., 10, e0172502.
251 Nahab et al., 10, e0172502.

In effect, Nahab et al. thus argued that the hysteria patients' impaired sense of agency was not limited to potential disturbances in the generation of motor intention but also entailed a selective dysfunction of the pre-SMA and dlPFC. As Nahab et al. explained, due to this selective dysfunction, hysteria patients were also unable to accurately assess their actual control over the self-generated movements.[252] In short, Nahab et al. postulated that the neural disturbances underlying hysteria patients' loss of self-agency were far more dynamic and complex than conjectured by the authors of the previous studies.

Significantly, the above interpretation of their fMRI maps was further reinforced by the behavioural data that Nahab et al. additionally collected. To this end, after the fMRI data acquisition, the subjects in their study performed the same virtual reality task outside the scanner. This time, however, the subjects were asked to explicitly judge and report their perceived level of agency over the movement of the simulated hand. The analysis of the behavioural data showed that, contrary to healthy subjects, "the patients claimed significant control when they had none and felt less than full control when control was complete."[253] The patients also exhibited "much greater variability in their perceived level of control" than healthy subjects.[254] Crucially, the discrepancies between the actual and subjectively perceived levels of control over the virtual hand obtained through self-reports correlated with the abnormal patterns of brain activity in the patients' fMRI maps. Nahab et al. thus concluded that the impaired haemodynamic responsiveness of the pre-SMA and dlPFC to the changing loss of movement control represented "the strongest evidence to date" that hysteria patients' perceived involuntariness of hysterical symptoms had a physiological basis.[255]

Finally, by explicitly building upon the studies analysed above, Baek et al. came up with yet another way to explore hysteria patients' impaired sense of agency through the use of fMRI. Baek et al. hypothesised that in addition to faulty intentional processes, as suggested by Voon et al., hysteria patients might also have a disturbed ability to experience their own motor intentions consciously.[256] Hence, Baek et al. set out to explore hysteria patients' potentially impaired "awareness of voluntary motor intention" and to identify the neural underpinnings of any such impairment.[257] With this aim in mind, Baek et al. asked the study participants to assess the subjective timing of their consciously perceived intentions and actions during the process of fMRI data acquisition.

Contrary to the studies discussed above, Baek et al. recruited twenty-six patients with mixed motor symptoms. In addition to various types of excessive movements ("non-epileptic seizures, tremor, chorea, tics, gait abnormalities, dystonia, myoclonus"), the symptoms in their sample also included both full and partial paralysis.[258] Owing

252 Nahab et al., 10, e0172502.

253 Nahab et al., 5, e0172502.

254 Nahab et al., 7, e0172502.

255 Nahab et al., 10, e0172502.

256 Baek et al., "Motor Intention," 1625.

257 Baek et al., 1625.

258 Baek et al., 1625. For a detailed discussion of the dominant approach to patient selection in task-based fMRI studies of hysterical symptoms, see section 3.1.3.

to this atypical sampling strategy, Baek et al. were able to directly compare the neural correlates of agency between these different manifestations of hysteria. As a control group, Baek et al. also recruited twenty-five healthy volunteers.

During the fMRI scanning, both the patients and healthy control subjects performed a variation of the famous Libet's task.[259] Specifically, the subjects were required to watch a red ball rapidly revolving around an unnumbered clock face and press the button whenever they wanted. To ensure that their actions were freely chosen, the participants "were asked to act as spontaneously as possible and in particular to avoid preselecting a position of the ball to trigger the button press."[260] The task consisted of two sets of trials. In one set, the subjects had to attend to the position of the ball when they perceived the intention to press the button. In the other set, they were asked to focus on the position of the ball at the moment when they actually pressed the button.

Having collected both the behavioural and fMRI data for all study participants, Baek et al. turned to their analysis. To begin with, Baek et al. compared the behavioural data between patients and healthy controls. In this comparison, they used the differences between the timings of the subjects' respective judgments of intention and action "as an implicit measure of conscious awareness of volitional intention."[261] The comparison revealed that in patients, as opposed to healthy controls, the interval between the two

259 In 1983, Benjamin Libet developed an oscilloscope 'clock' with a quickly rotating red dot to experimentally answer the question: "when does the conscious wish or intention (to perform the act) appear?" Libet, "Free Will," 49. In a seminal study, Libet et al. used EEG to measure the brain activity of healthy subjects who were asked to pay attention to the position of the dot when they felt a conscious urge to move. See Libet et al., "Conscious Intention." With this study, Libet et al. generated findings that appeared to "put constraints on views of how free will may operate." Libet, "Free Will," 47. The measurements they obtained of the so-called Bereitschaftspotential showed that the "onset of cerebral activity clearly preceded by at least several hundred milliseconds the reported time of conscious intention to act." Libet et al., "Conscious Intention," 623. Based on these measurements, Libet et al. concluded that voluntary movements were initiated by unconscious neural processes. However, Libet et al. also emphasised that their findings did not entirely deny the existence of free will. Instead, they argued that "the final decision to act could still be consciously controlled during the 150 ms or so remaining after the specific conscious intention appears. Subjects can in fact 'veto' motor performance during a 100–200 ms period before a prearranged time to act." Libet et al., 623. Libet's claim that voluntary movements are initiated unconsciously has ignited an ongoing debate. Multiple subsequent studies have since been published that have both supported and challenged his findings. For instance, the authors of one recent study have suggested that "intention consciousness does not appear instantaneously," as assumed by Libet, but instead "builds up progressively." Guggisberg and Mottaz, "Timing and Awareness," 1, article 385. Guggisberg and Mottaz have thus argued that "the timing of conscious intention reported by the participants [using the Libet's clock] might therefore be only the culmination of preceding conscious deliberations." Guggisberg and Mottaz, 8, article 385. It is important to emphasise that Baek et al. provided an overview of the criticism that has questioned the validity of using Libet's clock to assess the onset of conscious intention in absolute terms. See Baek et al., "Motor Intention," 1634. Moreover, they circumvented this problem because the aim of their study was not to determine the onset of conscious intention in absolute terms. Rather, their aim was to test the hypothesis that hysteria patients "would have delayed motor intention awareness" relative to healthy control subjects. Baek et al., 1625.

260 Baek et al., "Motor Intention," 1626.

261 Baek et al., 1628.

types of judgments was significantly shorter. This shortening was due to the patients' abnormally delayed awareness of the intention to move relative to the movement itself.[262] Another important discovery was that this delay was more pronounced in patients with positive motor symptoms, such as tremor, than in those with paralysis.

Based on these behavioural findings, Baek et al. drew two key conclusions. First, hysteria patients with mixed motor symptoms appeared to exhibit impaired awareness of their movement intentions, which, in turn, contributed to their disturbed sense of agency and the subjective experience of their symptoms as involuntary. Second, Baek et al. argued that the interval between the two types of judgments (i.e., the timing of intention and the timing of action) had been "postulated to be used by the subject to monitor [and assess] the desirability and effect of the action" selected.[263] Hence, Baek et al. suggested that a significantly reduced duration of this "veto period" in patients with positive motor symptoms "would have a higher likelihood of resulting" in maladaptive movements such as tremors.[264] In other words, Baek et al. posited that patients with tremor had shorter time available and thus less chance to consciously inhibit undesirable movements whose initiation had been triggered without their awareness.

To delineate the potential neural correlates of the cognitive disturbances they identified by analysing the behavioural data, Baek et al. calculated an fMRI map for the contrast between the judgments of intention versus movement. The resulting map revealed decreased brain activity in the inferior parietal cortex (IPC) in patients relative to controls.[265] Previous neurocognitive research has suggested that "the intentional feelings evoked in the IPC may lie upstream" of the SMA.[266] To be more exact, the IPC has been associated with a highly unspecific "subjective feeling of 'wanting to move,'" whereas the activity of the SMA with "an uncontrollable 'urge' to produce a specific," already planned movement.[267] Explicitly drawing on this research, Baek et al. argued that the hysteria patients' disturbance in generating motor intention took place at a considerably earlier stage of neural processing than initially suggested by Voon et al., who had associated it with the SMA. In short, Baek et al. attributed hysteria patients' perceived lack of agency not just to "core deficiencies" in intentional processes but also to the patients' considerably delayed awareness of the motor intention once it was formed.[268]

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To summarise, the studies analysed above have deployed the mutually interrelated concepts of motor intention and the sense of agency to fruitfully direct their exploratory fMRI-based investigation of the potential neural underpinning of hysteria patients' subjective lack of control over their symptoms. We have seen that with each new

262 Baek et al., 1629.
263 Baek et al., 1634.
264 Baek et al., 1634.
265 Baek et al., 1629–30.
266 Baek et al., 1633.
267 Baek et al., 1633.
268 Baek et al., 1624.

study, its authors used these action-guiding concepts to formulate increasingly more clearly defined research questions and developed specifically tailored fMRI-based experimental setups to address these questions. Produced in such a context, the resulting fMRI brain maps could be used productively to open up new perspectives on hysteria. Admittedly, as discussed in chapter 1, Charcot devised several experimental setups meant to demonstrate hysterical symptoms' involuntary nature. Yet, beyond ascribing this involuntariness to what he referred to as the reflex action of the brain, Charcot was unable to provide a more precise explanation for it. Hence, only the recent fMRI research has made it possible not just to demonstrate that hysterical symptoms are involuntary but also to explore how this happens at the neurocognitive level.

It should be noted that the image-based findings discussed above are still preliminary and fragmentary. Nevertheless, my analysis has underscored that the neurocognitive mechanisms posited by the four studies at the centre of our discussion were not mutually conflicting. Instead, these studies complemented one another, thus producing increasingly more refined insights. What started as a broad search for a hypothesised break somewhere along the intention-action-effect chain in experimentally modelled voluntary movements gradually progressed to more complex and fine-grained studies, which focused on elucidating hysteria patients' abilities to assess their own sense of agency. Crucially, the most recent findings suggest that hysteria patients' sense of impaired agency may not be attributable to a single disruption along the intention-action-effect chain. Contrary to initial assumptions, the patients' loss of perceived control over their actions appears to be caused by several mutually interacting functional disturbances that affect multiple brain regions. In my view, the four studies analysed above provide pertinent examples of how fMRI can be implemented in non-reductive ways to explore hysteria patients' subjective experience of their symptoms by framing it as a complex and dynamic neurocognitive phenomenon with a distinct although still unknown physiological basis.

Admittedly, all four fMRI studies discussed in this section placed the patients' subjective experience of the involuntary nature of their symptoms into a decidedly somatic framework. Moreover, each study entailed an erasure of the idiosyncratic differences across individuals through statistical averaging. These limitations, however, represent necessary preconditions for the potential epistemic productivity of the fMRI maps that aim to provide a neurophysiological explanation for the patients' lack of control over their symptoms. Accepting such limitations seems to be a reasonable compromise if we consider that before the appearance of this research, the baffling hysterical symptoms had been dismissed as malingering and the patients' accounts of the involuntary nature of these symptoms regarded as fictional. The studies analysed above have delivered empirical evidence supporting the veracity of patients' self-reports. And even more importantly, these studies have also begun to unravel the potential neurophysiological reasons underpinning the patients' subjective experiences of their symptoms.

4.2.2 Exploring How to Experimentally Frame Hysteria Patients' Attentional Dysfunctions

We have seen in the previous section that the strand of fMRI research aimed at elucidating the neural basis of the patients' self-reported involuntariness of hysterical symptoms has focused primarily on excessive movement and, to a lesser extent, on paralysis. Sensory manifestations of hysteria have thereby been entirely disregarded. This selective focus was by no means accidental. It was due to the fact that the concepts of self-agency and motor intention are not readily applicable to the investigation of hysterical sensory symptoms.[269] As we will see in this section, another aspect of the patients' subjective experience of their symptoms has enabled researchers to expand the focus by addressing both sensory and motor manifestations of hysteria using similarly conceived fMRI experiments. Such studies have aimed to uncover why the patients' self-reported perception of their motor and sensory symptoms fluctuates with changing circumstances.[270] Specifically, not only the patients' awareness of having a symptom but also the perceived severity of the symptom appear to wax and wane depending on how distracted each patient is. For example, many patients appear to be unaware of their sensory impairments before undergoing a targeted clinical examination.[271] Others start with an apparently mild sensory or motor disturbance, which gradually intensifies in the course of the examination or on repeated testing. By contrast, it has been shown that a mere act of distracting the patient can lead to a temporary remission of sensory and motor symptoms. For instance, under the influence of sedatives, sensory abnormalities are "transiently but substantially reduced."[272]

In what follows, I will examine how, in an attempt to provide a neurobiological explanation for such puzzling inconsistencies in the patients' experience of their symptoms, several fMRI studies have productively deployed the action-guiding concept of attention they borrowed from cognitive neuroscience.[273] In the neuroscientific context, attention is defined as a set of cognitive processes whose purpose is to select relevant information for focused neural processing while ignoring the rest of incoming stimuli.[274] Defined in such terms, attention does not rely on a single mechanism. Instead, attention is understood to involve three distinct yet mutually interacting cognitive processes of alerting, orienting, and executive control. Alerting "is defined as achieving and maintaining a state of high sensitivity to incoming stimuli; orienting is the selection of information from sensory input; and executive attention involves

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

270 See, e.g., Ghaffar, Staines, and Feinstein, "Sensory Conversion Disorder"; Mailis-Gagnon et al., "'Hysterical' Anesthesia"; and Saj, Arzy, and Vuilleumier, "Spatial Neglect."

271 Mailis-Gagnon and Nicholson, "Somatosensory Deficits," 594.

272 Mailis-Gagnon et al., "'Hysterical' Anesthesia," 1502. Moreover, patients who report one-sided hysterical blindness can read a stereoscopic text, which requires good vision in both eyes. Stone et al., "Potential Solutions," 372. Similarly, patients suffering from voice loss cannot speak but can sing. Bryant and Das, "Neural Circuitry," 290.

273 Bègue et al., "Metacognition"; Burke et al., "Ancillary Activation"; Ghaffar, Staines, and Feinstein, "Sensory Conversion Disorder"; and Mailis-Gagnon et al., "'Hysterical' Anesthesia."

274 Baars and Gage, *Cognition, Brain and Consciousness*, 276.

mechanisms for monitoring and resolving conflict among thoughts, feelings, and responses."[275]

Based on converging neuroimaging findings, each of these three cognitive processes is thought to be associated with the activity of a discrete system of brain regions that are jointly referred to as attentional networks. The alerting attentional network "has been associated with thalamic as well as frontal and parietal regions of the cortex," whereas the orienting network appears to involve "posterior brain areas, including the superior parietal lobe and temporal parietal junction."[276] Finally, the "executive attention network relies on the anterior cingulate and lateral areas of the prefrontal cortex."[277] As my analysis will show, by positing an unknown functional disturbance somewhere among these widely distributed brain regions, fMRI-based hysteria research has found a way of experimentally addressing the patients' vacillating inability to accurately perceive the presence and severity of their symptoms.

Published in 2003, the Mailis-Gagnon study was the first fMRI experiment that explicitly posed the question of how the "attentional state can modulate sensory-evoked responses" in patients suffering from hysterical anaesthesia.[278] The researchers recruited four patients with sensory deficits that arose "in the absence of substantial pathology" and exhibited different anatomical distributions across the patients.[279] The patients had lost sensibility to touch, pinpricks, and cold in the anaesthetic areas. In each case, the sensory deficits were accompanied by chronic pain that affected approximately the same anatomical areas as the anaesthesia. All patients experienced a similar level of pain. On the day of the fMRI imaging, they subjectively rated the pain intensity as seven to eight on a scale from zero to ten.[280]

While lying inside the scanner, the patients were exposed to blocks of two different types of passive tactile stimulations that alternated with periods of rest. One set of blocks comprised painful mechanical and the other non-painful brushing stimulations. Each type of stimulation was separately applied either to the patients' anaesthetic or sensate side of the body.[281] The critical aspect of the experimental design was that the patients were instructed to keep their eyes closed throughout the scanning. This injunction was meant to prevent the patients from paying explicit attention to whether the painful or non-painful stimulation was applied to their bodies.[282]

Despite the tiny sample size that made the statistical validity of their results problematic, Mailis-Gagnon et al. submitted the fMRI data to group analysis and calculated four activation maps.[283] Each group-level map displayed the brain activations induced by either painful or non-painful stimulation relative to rest for

275 Posner and Rothbart, "Attention Network," 7.

276 Posner and Rothbart, 7.

277 Raz, "Attentional Networks," 29.

278 Mailis-Gagnon et al., "'Hysterical' Anesthesia," 1501.

279 Mailis-Gagnon et al., 1501.

280 Mailis-Gagnon et al., 1502.

281 Mailis-Gagnon et al., 1502.

282 Mailis-Gagnon et al., 1506.

283 For a discussion of the adverse effect of small sample sizes on the epistemic validity of fMRI maps, see section 3.4.3.

the affected and the unaffected sides separately. The patients reported that they could perceive all painful and non-painful stimuli on their healthy side but none of them on their anaesthetic side. However, the visual comparison of the group maps calculated separately for the anaesthetic and the sensate body sides delivered surprising results. This comparison showed that both painful and non-painful stimuli the patients had reported as unperceived nevertheless induced a complex pattern of activations across their brains.[284] Yet, this was not the only insight.

As expected, the fMRI maps computed for the perceived stimuli (those delivered to the sensate side of the patients' bodies) showed differential patterns of activation for pain and touch comparable to those found in healthy subjects.[285] By contrast, the patterns of activation induced by the unperceived stimuli displayed multiple abnormalities. Some of these abnormalities included the lack of expected activation in several brain areas, such as the insula, thalamus, and inferior frontal cortices. Further abnormalities included unexpected deactivation relative to baseline (i.e., rest) in the prefrontal regions, the somatosensory, and the postparietal cortex.[286] Additionally, parts of the anterior cingulate cortex (ACC) were activated only by the unperceived but not by the perceived stimuli. Just as interestingly, the maps also clearly showed that the patients' brains responded differently to the painful as opposed to the non-painful stimuli even when these were not consciously perceived.[287] Put differently, although the patients were entirely unaware that their affected side had been exposed to two different types of stimuli, their brains appeared to register the difference. Finally, the patients' somatosensory cortex showed a decreased response to the stimuli administered to their insensate side, thus providing neurophysiological support for the patients' reported lack of sensation in the affected body parts.[288]

Notably, the study's key finding was not the unsurprisingly reduced activation of the somatosensory cortex during the unperceived stimuli. Rather, the crucial discovery was the accompanying aberrantly suppressed activity in the prefrontal and posterior parietal regions together with the hyperactivation of the ACC. The author conjectured that these accompanying anomalous activations indicated disturbed emotional regulation and abnormal "attention cortical processing during the unperceived stimuli."[289] Interestingly, this anomalous pattern encompassed all three attentional networks with their respective alerting, orienting, and executive control functions.[290] Although Mailis-Gagnon et al. did not explicitly mention this fact, it was nevertheless reflected in their interpretation.

Specifically, Mailis-Gagnon et al. hypothesised that the "dynamic aberrations of brain function" during the unperceived stimuli could be the result of an unsuccessful attempt of the central nervous system to shut down "all peripheral inputs originating

284 Mailis-Gagnon et al., "'Hysterical' Anesthesia," 1503–6.
285 Mailis-Gagnon et al., 1503.
286 Mailis-Gagnon et al., 1503–4.
287 Mailis-Gagnon et al., 1503–4.
288 Mailis-Gagnon et al., 1504–5.
289 Mailis-Gagnon et al., 1506.
290 Compare Raz, "Attentional Networks," 26–32.

in or associated with the painful limb in an effort to control pain."[291] Further, they conjectured that these 'dynamic aberrations' might have initially developed in an emotionally charged situation either due to a minor physical injury or without any discernible external cause. Mailis-Gagnon et al. thus suggested that, following an unpleasant physical or emotional sensation, in predisposed individuals, the brain selectively withdrew attention from all sensory information coming from the affected body part to minimise the experience of pain.[292] Unfortunately, this maladaptive mechanism failed to control pain. Instead, it gave rise to the "suppression of the cutaneous and often deep sensation," thus resulting in sensory and often also in "variable motor deficits."[293]

Although considerably more detailed in neurocognitive terms, the basic tenets of this mechanism show a striking similarity to Janet's views on the role of attentional disturbances in the formation of hysterical sensory loss. But unlike Mailis-Gagnon et al., Janet viewed attention as a mental faculty and did not associate it with any localised brain activity.[294] Moreover, although the details of the proposed mechanisms underpinning hysterical sensorimotor loss differed considerably between Mailis-Gagnon et al. and Charcot,[295] they did have one thing in common. Both mechanisms posited that the symptoms were caused by anatomically localisable dynamic aberrations of brain function. Despite the apparent parallels, Mailis-Gagnon et al. did not explicitly refer to either Janet's or Charcot's work, with which they may or may not have been acquainted.

Importantly, Mailis-Gagnon et al. also admitted that, instead of the interpretation delineated above, their imaging findings could alternatively be attributed to a different cognitive mechanism. As they explained, the same pattern of fMRI activations could also be taken to suggest that hysteria patients directed too much attention to their ongoing pain. Such an aberrant attentional focus would, in turn, interfere with their brain's normal processing of incoming sensory information.[296] Yet, the researchers argued that the latter explanation seemed less likely because the patients in their study were instructed to keep their eyes closed. Nevertheless, Mailis-Gagnon et al. conceded that, due to the lack of explicit behavioural data, the possibility that the patients' attentional focus had fluctuated during the measurement could not be entirely ruled out.[297] In sum, Mailis-Gagnon et al. succeeded in tentatively linking

291 Mailis-Gagnon et al., "'Hysterical' Anesthesia," 1506.

292 Significantly, "attention in the sense of orienting to [or away from] sensory objects can actually be involuntary and can occur unconsciously." Raz, "Attentional Networks," 21. In my view, Mailis-Gagnon et al. had such an involuntary withdrawal of attention in mind as the underlying mechanism of hysterical sensory loss because they explicitly argued that the brain and not the subject shuts down the sensory inputs. Such a formulation implies that this process is carried out unconsciously.

293 Mailis-Gagnon et al., "'Hysterical' Anesthesia," 1506.

294 See Janet, *Mental State*, 40, 399. For a detailed account of Janet's conception of hysterical anaesthesia, see section 2.1.2.

295 For details regarding Charcot's conjectures, see section 1.3.2.

296 Mailis-Gagnon et al., "'Hysterical' Anesthesia," 1506.

297 Mailis-Gagnon et al., 1506.

hysteria patients' subjective experience of sensory loss to an anatomically localisable dysfunction that affected multiple attentional networks. However, the researchers could not unambiguously attribute this disturbance to a unique cognitive mechanism. Yet, in my opinion, the most important aspect of this study were not its tentative imaging findings but that it opened up new questions, which other researchers subsequently took up.

Two years later, another fMRI study approached the question of the potential role of attentional processes in hysterical sensory loss from a different perspective.[298] Its authors, Ghaffar, Staines, and Feinstein, recruited an equally tiny sample of only three female patients with chronic left-sided sensory loss in either the hand or the foot. Inside the scanner, each patient was exposed to blocks of vibrotactile stimulation that alternated with rest.[299] But compared to the Mailis-Gagnon et al. study, there was one critical difference in the experimental design Ghaffar, Staines, and Feinstein chose to deploy. In this case, the experimental manipulation was not limited to unilateral limb stimulation applied to either the anaesthetic or the sensate side of the body separately. Instead, Ghaffar, Staines, and Feinstein included a third experimental condition. During this condition, both the patients' healthy and affected limbs were exposed simultaneously to bilateral vibrotactile stimulation.[300] In developing this experimental design, the researchers aimed to test if these disparate modes of stimulation (i.e., unilateral versus bilateral) would differently engage the patients' attention by either focusing it on or withdrawing it from the symptom.

Taking into account their tiny sample size and the differences in the symptom manifestations among the patients, the researchers refrained from calculating group-level brain maps. Instead, they computed separate maps for each subject and for each of the three experimental conditions.[301] The principal finding derived from the visual comparison of the nine resulting fMRI maps was that unilateral and bilateral stimulations produced markedly different neural responses in each patient. Unilateral stimulation of the unaffected limb relative to rest activated the primary somatosensory cortex (S1 region) on the opposite side of the body in all three patients.[302] This particular activation pattern was comparable to the one the same research team had obtained in a previous study in which they exposed healthy subjects to the same unilateral vibrotactile stimulation.[303] Based on this fact, Ghaffar, Staines, and Feinstein concluded that the patients retained normal neural responsiveness on their healthy side. By contrast, when the stimulation was limited to the affected limb, it failed to activate the appropriate S1 region. Importantly, the latter result was in line with the patients' self-reported absence of conscious tactile sensations on the affected side of the body.[304]

298 Ghaffar, Staines, and Feinstein, "Sensory Conversion Disorder."

299 Ghaffar, Staines, and Feinstein, 2036.

300 Ghaffar, Staines, and Feinstein, 2036.

301 Ghaffar, Staines, and Feinstein, 2037. The three conditions included bilateral stimulation, unilateral stimulation of the healthy side, and unilateral stimulation of the affected side.

302 Ghaffar, Staines, and Feinstein, 2036.

303 Ghaffar, Staines, and Feinstein, 2038.

304 Ghaffar, Staines, and Feinstein, 2036.

But the surprising finding was that the simultaneous stimulation of the affected and the unaffected limb elicited a bilateral activation of the S1 regions similar to the one seen in healthy subjects under the same condition.[305] Simply put, whereas the designated S1 region remained inactive during the unilateral stimulation of the affected limb, the bilateral stimulation managed to activate this region. Furthermore, the comparison of all nine maps revealed additional, either abnormally increased or decreased activations in multiple brain areas outside the primary somatosensory cortex. The researchers drew two significant conclusions from the maps. First, they argued that, by focusing the patients' attention on the affected limb, the unilateral stimulation suppressed the activity in the designated S1 region.[306] By contrast, the bilateral stimulation acted as a distraction that shifted the patient's attention away from the affected limb, thus temporarily lifting the symptom-specific suppression of the activity in the somatosensory cortex.

Second, the researchers suggested that the suppressed activity of the S1 region was most likely caused by the interactions among the "multiple sites of additional activation/deactivation," including the orbitofrontal cortex (OFC), ACC, thalamus, and striatum.[307] Ghaffar, Staines, and Feinstein tentatively linked these multiple aberrant activations to a disturbance in attentional processes. The problem was, however, that these patterns of activation varied considerably across the maps calculated separately for each experimental subject.[308] Hence, the authors concluded that an unambiguous interpretation of these additional activations was not possible due to the small sample size and the differences among their participants. Despite this limitation, the essential contribution of this study was showing that distractions not only changed the patients' subjective self-reported experience of the symptoms but also induced measurable alterations in their brain activity. In effect, Ghaffar, Staines, and Feinstein delivered empirical findings to support the conjecture that when hysteria patients are distracted, their brains process incoming stimuli differently.

In 2014, the same research group published a new fMRI study. The researchers' explicit aim was to explore the role of the additional activation patterns they had discovered in 2006 but could not fully account for at the time.[309] In addition to reusing the fMRI data from their previous study, the researchers recruited seven more subjects with unilateral anaesthesia.[310] Since Burke et al. were interested in identifying the anatomical distribution of the brain regions that, according to the hypothesis derived from their previous study, suppressed the activity of the somatosensory cortex, this time, they only deployed unilateral stimulation. Hence, during the fMRI scanning, unilateral stimulation was applied separately to the patient's numb or sensate limb. Importantly, due to a larger participant sample, in this case, the researchers were

305 Ghaffar, Staines, and Feinstein, 2036, 2038.

306 Ghaffar, Staines, and Feinstein, 2037–38.

307 Ghaffar, Staines, and Feinstein, 2038. See also Ibid., 2026.

308 Ghaffar, Staines, and Feinstein, 2036–37.

309 In addition to three researchers who authored the previous study, the group now included Matthew Burke and Jonathan Downar. See Burke et al., "Ancillary Activation," 333.

310 Burke et al., 334.

able to compute a direct statistical comparison between the neural effects induced by the stimulation of the symptomatic and the asymptomatic limb. This allowed them to filter out the individual differences in the task-induced brain activities across the subjects and generate insights that were potentially generalisable beyond their patient sample.[311]

The resulting group-level fMRI map revealed the expected suppression of the activity in the primary somatosensory cortex contralateral to the anaesthetic side but also disclosed significantly increased activations in ten additional cortical and subcortical brain areas.[312] By referencing multiple neuroimaging studies, the authors argued that some of these regions—e.g., the insula—were associated with the processing of emotion. Yet, the majority of aberrantly activated regions—the right temporoparietal junction (TPJ), ACC, striatum, and thalamus—represented parts of all three attentional networks.[313] Drawing on their imaging findings, Burke et al. concluded that hysterical anaesthesia was not related to any disturbance in the initial neural processing of sensory stimuli, since this remained intact. Instead, the symptom appeared to arise from the "failed sensory integration," which took place later in the processing chain, and was associated with the abnormal functioning of the higher-order brain regions, such as the "parietal cortex, ACC, striatum and thalamus."[314] Put simply, hysteria patients exhibited normal initial cortical responses to external stimuli. However, it was because of the widespread disturbances of attentional mechanisms that these initial cortical responses were unable to enter higher stages of sensory processing and, as a result, became selectively disconnected from conscious awareness. The patients' brains thus failed to organise the incoming stimuli into a coherent perception.[315]

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311 For a discussion on the relation between the type of statistical analysis used and the generalisability of the resulting maps, see section 3.4.2.

312 Specifically, the areas of the increased ancillary activation "included the right paralimbic cortices (anterior cingulate and insula), right temporoparietal junction (TPJ) (angular gyrus and inferior parietal lobe), bilateral dorsolateral prefrontal cortex (middle frontal gyri), right orbital frontal cortex (superior frontal gyrus), right caudate, right ventral-anterior thalamus and left angular gyrus." Burke et al., "Ancillary Activation," 335.

313 Burke et al., 335, 337–38.

314 Burke et al., 337–38.

315 Interestingly, a similar conclusion was drawn by the authors of a simple and elegant single-case fMRI study performed on a hysteria patient with an unusual sensory symptom called left spatial neglect. This symptom is characterised by the impaired ability to respond to either sensory or visual stimuli on one side of the body. See Saj, Arzy, and Vuilleumier, "Spatial Neglect," 2552. While lying inside the scanner, the patient performed a so-called line-bisection test. This test consisted of a set of intersecting lines, half of which were correctly centred, whereas the other half had deviations either to the left or to the right. The patient was asked to judge if the bisection mark was placed at the centre or not. Behavioural data demonstrated that the patients made significantly more errors when judging leftward and centred than rightward bisecting lines. By contrast, the fMRI map showed that all stimuli induced normal initial processing. Ibid., 2553. Nevertheless, the patient was unable to correctly perform the line bisection judgments. The authors attributed her failed performance to the abnormally increased task-induced activity of the ACC region that was clearly indicated in the fMRI map. Although they were unable to specify the exact mechanism, Saj, Arzy, and Vuilleumier conjectured that the aberrant activation of the ACC might suggest

In short, Burke et al. decisively linked hysteria patients' "functional unawareness" of incoming sensory stimuli—i.e., anaesthesia—to a circumscribed "dysfunction of attentional centres."[316] Although Burke et al. postulated a considerably more complex neurocognitive mechanism, I suggest that they, in effect, provided empirical support for Janet's initial conjecture. As discussed previously, Janet claimed that patients with hysterical anaesthesia did not stop having sensations but instead became unable to consciously perceive them due to a pathological 'feebleness of attention.'[317] Using fMRI, Burke et al. semantically transcribed Janet's hypothesised psychological mechanism into a decidedly neurological one.[318] However, Burke et al. were unable to identify the specific role of each abnormally activated attentional centre. As they pointed out, several of the activated brain regions in their study, although "most classically associated with sensory integration and attention," are also "thought to be implicated in multiple high-level cognitive functions," such as 'theory of mind' and self-agency.[319] Therefore, disentangling how exactly each of these regions contributed to the formation of hysterical anaesthesia proved challenging. Another interpretational challenge Burke et al. could not resolve was how the different brain regions identified by their study mutually interacted to give rise to hysterical anaesthesia. So far, both questions remain open, awaiting further research.

In the meantime, a study published by Bègue et al. in 2018 has generated an fMRI finding that supported yet another of Janet's conjectures. According to this conjecture, a comparable mechanism of attentional dysfunction might be implicated not only in hysterical anaesthesia but also in motor symptoms.[320] In designing their study, Bègue et al. explicitly drew on the previous fMRI findings that pointed to hysteria-related "disturbances in self-awareness and self-monitoring functions."[321] Hence, Bègue et al. chose to examine the neurocognitive mechanisms underlying hysteria patients' potentially diminished ability to monitor, assess, and adjust their actions while performing motor tasks. To address this question, Bègue et al. developed an attention-demanding motor task that required the participants of their study to closely and continually monitor the visual effects of their performance.

While lying inside the scanner, ten patients with highly heterogeneous motor symptoms and ten healthy control subjects performed a visually guided hand movement task that consisted of 110 trials.[322] In each trial, using a joystick, subjects had to move a

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"impaired access to conscious control." Ibid., 2554. In other words, the problem appeared to lie in the disturbance of top-down attentional processes due to which normal initial cortical responses became selectively disconnected from conscious awareness. Hence, the two fMRI studies that used different experimental tasks to investigate two different types of sensory symptoms came to very similar conclusions regarding the neurocognitive mechanism that potentially causes the loss of sensations in hysteria.

316 Burke et al., "Ancillary Activation," 337–38.

317 For more details on Janet's views on hysterical anaesthesia, see section 2.1.2.

318 I am using the term transcription here in Jäger's sense. See Jäger, "Transcriptivity Matters," 50.

319 Burke et al., "Ancillary Activation," 337.

320 Bègue et al., "Metacognition."

321 Bègue et al., 252.

322 The symptoms included paralysis, tremor, gait disturbances, and contractures. Bègue et al., 253.

cursor in a straight line from the starting position at the bottom to the target position at the top of the screen. So far, the task may seem trivially simple. However, in 79% of the trials, the computer introduced deviations into the cursor's trajectory.[323] To reach the target position with a straight line, the participants had to compensate for the externally induced deviations. The participants were informed that deviations would occur in some trials. Yet, they neither knew when nor how often.[324] Hence, the unexpected deviations forced participants to pay close visual attention to the changing position of the cursor. The task thus explicitly diverted the participants' attention away from their actual hand movements, which they were unable to observe directly. Instead, the task fixed the participants' attention to the abstract visualisation of the consequences of their movements, which appeared on the screen.

After each trial, the participants reported if they had detected any deviation and rated the confidence of their responses. The fMRI data were collected during the movement trials and during the subjects' confidence ratings. Moreover, the computer tracked the exact trajectory each participant had drawn on each trial. To gain insights into the participants' task performance, Bègue et al. first analysed the behavioural data. These showed that the patients tended to make "a more curved trajectory" than the healthy control subjects.[325] As explained by the researchers, this finding indicated that the patients required larger deviations to notice them in the first place, whereas smaller deviations eluded them. However, the behavioural data also showed that both the patients and healthy subjects detected the deviations with the same level of accuracy.[326] Similarly, the two groups exhibited a comparable level of confidence in the ability to assess their motor actions. This was significant for two reasons. On the one hand, this meant that the subsequent comparison of the underlying neural activations across the groups was not confounded by potential differences in the respective task performances between patients and control subjects.[327] On the other hand, it also provided the researchers with empirical proof that, because the subjects "monitored their task performance adequately," their attention did not fluctuate during the experiment.[328]

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323 Bègue et al., 254.

324 The subjects were merely "told that such deviations did not occur all the time, and that when they did, they never occurred in the beginning or the end of the trajectory, but always at some point around the middle of the movement." Bègue et al., 254.

325 Bègue et al., 255.

326 Bègue et al., 255. The level of accuracy in both groups amounted to approximately two-thirds of the trials. Notably, this "balanced proportion of detected and undetected deviations" was not an accident but an intended aspect of the task. Ibid. 254. To ensure it, the researchers determined the magnitude of the deviation for each subject individually "by starting with a deviation angle of 30 degrees, and then adjusting the angle online through a staircase procedure, so as to obtain a balanced proportion of detected and undetected deviations overall. The staircase procedure made the task more difficult after two consecutive correct responses by increasing the next deviation by 2.64 degrees, but made it easier after an incorrect response by reducing the next deviation by 1°." Ibid.

327 Bègue et al., 258.

328 Bègue et al., 256. As discussed above, in the Mailis-Gagnon et al. study, the lack of evidence that the patients' attention did not fluctuate throughout the task made their findings difficult to interpret.

Next, Bègue et al. calculated multiple functional maps for different aspects of the task. First, they computed fMRI activation maps for the entire phase of the movement execution, from the moment the subject started to push the joystick until reaching the target position. These maps displayed "globally similar" patterns of activations between the patients and healthy controls.[329] Based on this overlap, the authors concluded that the patients' "elementary motor functions" were intact.[330] Additionally, the researchers calculated separate fMRI maps for what they termed the conscious and the unconscious monitoring of movements. These maps revealed significant differences in the underlying brain activities between patients and controls. In this context, conscious monitoring was defined as the contrast between the neurocognitive processes induced by consciously detected and corrected deviations as opposed to those that remained undetected.[331] Conversely, unconscious monitoring was isolated by comparing the brain activities elicited by, on the one hand, undetected yet nevertheless corrected deviation and, on the other hand, the absence of deviations.

The maps computed for the conscious monitoring of movements displayed significantly higher activations "in motor, visual and cerebellar regions" in healthy subjects.[332] The maps also disclosed that the patients "activated very few areas in this contrast."[333] According to Bègue et al., these activation patterns suggested that, during the conscious motor control, healthy subjects but not patients relied on "sensory-motor integration and vision."[334] Notably, two maps computed for the unconscious monitoring of motor action delivered the most insightful findings. The map for healthy subjects was empty, indicating that the unconscious monitoring did not elicit any statistically significant brain activation in this group.[335] By contrast, the patients' map revealed increased activations in "several areas in motor and attentional networks," such as the left precentral gyrus, left pre-supplementary area (pre-SMA), ACC, right IFG, and right precuneus.[336] These activations indicated that the patients' brains, unlike those of the healthy subjects, "responded mainly to unconsciously detected/adjusted deviations."[337] Bègue et al. attributed this aberrant activation pattern to hysteria patients' disturbances of the higher-level attentional processes. In effect, the fMRI maps disclosed that the patients were mostly unaware of the exact corrective movements they performed to compensate for the externally induced deviations of the cursor's trajectory.

Drawing their imaging results together, Bègue et al. conjectured that hysteria patients and healthy subjects used disparate "mechanisms and sources of information"

329 Bègue et al., 259.
330 Bègue et al., 259.
331 Bègue et al., 257.
332 Bègue et al., 260.
333 Bègue et al., 260.
334 Bègue et al., 251.
335 Bègue et al., 257. Since it was devoid of any statistically significant activation for this contrast in healthy subjects, this map was not visualised in the published study.
336 Bègue et al., 257.
337 Bègue et al., 257.

while monitoring and assessing their motor actions.[338] The healthy subjects' corrective movements were derived from their conscious assessment of the altered visual feedback. Conversely, this explicit system of error-monitoring and motor control was impaired in the patients. The patients' preserved ability to correct externally induced deviations in the cursor's trajectory indicated that their automatic processing of movement remained intact.[339] However, these automatic processes failed to be integrated into conscious awareness due to the disturbances in the patients' attentional networks. As a result, patients monitored and adjusted their ongoing motor performance "without direct conscious access to the underlying sensorimotor parameters."[340]

Finally, Bègue et al. computed an additional set of fMRI maps, which showed that the patients and healthy subjects engaged different brain areas when rating the confidence of their ability to detect deviations. To perform this metacognitive judgment, healthy subjects relied primarily on sensorimotor information. This was indicated by the activation in their precuneus and the middle temporal region.[341] Patients, by contrast, engaged the hippocampus and the amygdala. This activation pattern suggested that the hysteria patients' evaluation of visuomotor decisions might be "abnormally tagged with affective valence" or "at least partly influenced by memory associations rather than by sensorimotor signals only."[342] Hence, similarly to Mailis-Gagnon et al. and Burke et al., Bègue et al. also concluded that multiple disturbances in attentional and emotion processing mutually influenced one another. Taken together, the findings of these studies suggest that hysteria patients' subjective experience of their symptoms arises from a complex interplay of functional deficits that affect multiple brain subsystems. But how exactly such interactions occur could not be unambiguously identified in the resulting fMRI maps.

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To conclude my analysis in this section, I argue that the fMRI studies which relied on the action-guiding concept of attention have succeeded in producing new empirical insights into why hysteria patients' awareness of their puzzling symptoms fluctuates depending on the level of their distractedness. Admittedly, the number of fMRI studies that have so far deployed this action-guiding concept remains relatively scant. And almost all of them have been performed on small sample sizes, which means that their findings are far from conclusive and of potentially limited generalisability. Despite these limitations, my analysis has shown that this strand of research has grown in complexity by developing increasingly fine-grained ways of experimentally manipulating the hysterical subject's attention to make its neural correlates measurable by fMRI. As we have seen, these interventions have ranged from merely asking the subjects to close their eyes, to exposing them to alternating unilateral and bilateral

338 Bègue et al., 251.
339 Bègue et al., 261–62.
340 Bègue et al., 252.
341 Bègue et al., 260–61.
342 Bègue et al., 261.

sensory stimulation, and, finally, to devising a complex motor task that distracted the patients from the movements they were induced to perform.

Most significantly, the particular strength of these studies is that, due to the gradual experimental revision of the action-guiding concept of attention we discussed above, they have managed to generate sufficiently converging empirical results. The overall insight emerging from these studies is that hysteria patients' diminished subjective awareness of their perceptual and motor abilities are associated with multiple functional deficits across the attentional networks. As we have seen, the current findings suggest that each of these potential deficits can differently affect various aspects of the higher-order sensory integration or conscious movement control, thus resulting in different hysterical symptoms. Moreover, according to the studies analysed in this section, such attentional deficits are further aggravated by possible dysfunctions in the patients' emotion processing. Interestingly, this unknown role of emotion processing in the formation and maintenance of hysterical symptoms has taken centre stage in multiple fMRI studies to whose discussion we will now turn.

4.3 Imaging Hysteria Patients' Aberrant Neural Processing of Experimentally Induced Emotional States

Throughout this book, we have kept returning to the fact that hysteria has been repeatedly linked to emotional dysfunction and stressful life events during its long history. As discussed earlier, hysteria was regarded as an essentially psychogenic disorder for most of the twentieth century. Yet, such linking has much deeper historical roots. Across different historical periods and changing medical contexts, emotionally charged experiences had been variously ascribed the role of either causative, triggering, or contributing factors in the development of this puzzling disorder.[343] As we have seen in chapter 1, even Charcot, who had framed hysteria in decidedly neurological terms, nevertheless emphasised the role of emotional events in triggering the onset of its physical symptoms. If we consider such continuing historical entanglement between hysteria and emotions, it may come as a surprise that functional neuroimaging research avoided directly addressing this topic for more than a decade.

Indeed, not before 2007 did the first fMRI study appear that explicitly focused on investigating the neural correlates of emotional processing in a single female patient.[344] By that point, the authors of an increasing number of fMRI studies, some of which we analysed in the previous sections, generated imaging findings that indirectly indicated a potential role of emotions in the formation of various hysterical symptoms.[345] Specifically, fMRI maps that the authors of these studies had computed to isolate the brain dysfunctions underlying either motor or sensory manifestations of hysteria displayed additional abnormal activations. These were located in the brain regions not

343 For a succinct overview of the vacillating medical understanding of the nature of hysteria throughout this disorder's long history, see Micale, *Approaching Hysteria*, 19–29.

344 Kanaan et al., "Repressed Memories."

345 See, e.g., Bègue et al., "Metacognition"; and Burke et al., "Ancillary Activation."