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."

directly associated with the physical symptoms under investigation. Instead, across multiple studies, the additional aberrant activations seemed to be involved in the neural processing of emotions.[346] The unavoidable implication of such incidental findings was that if they wanted to elucidate the neural mechanisms underlying hysterical symptoms, researchers would have to start exploring the potential contribution of, by that point, only indirectly conjectured "emotional dysregulation."[347]

However, as I will argue in what follows, the apparent epistemic necessity to address the hysteria patients' hypothesised emotional dysregulation raised a new question. How to subsume the patients' potentially idiosyncratic emotional reactions to the operational logic of an fMRI experiment and thus make their neural correlates both measurable and unambiguously interpretable? I will show that fMRI studies that have attempted to answer this question in the first two decades of the twenty-first century have deployed two different action-guiding concepts. One of these concepts is the memory of traumatic life events, which fMRI research has directly borrowed from Freud.[348] The second action-guiding concept is emotion processing, as it is defined in affective neuroscience.[349] In the following two sections, I will analyse how exemplary fMRI studies have deployed these two different action-guiding concepts and discuss the image-based findings these studies have generated. I intend to demonstrate that, despite their differences, both approaches have one thing in common. They are both characterised by the shared effort to control the potential messiness and epistemic ambiguity of the experimentally elicited emotional responses through a systematic curtailment of the patients' idiosyncratic subjective experiences.

4.3.1 Endeavouring to Make the Impact of the Induced Recall of Traumatic Memories Measurable through fMRI

Ever since Freud's purely psychogenic model of hysteria lost its dominance, the potential relationship between hysterical symptoms and the individual patients' stressful life events has become the topic of contentious debate in the clinical and research context.[350] We have already discussed how starting with the *DSM–III*, this highly influential diagnostic manual gradually de-emphasised the role of traumatic life experiences as the potential cause of hysteria. This development culminated in 2013 with the *DSM-5*, which eliminated antecedent psychological stressors as a diagnostic requirement.[351] Consequently, in the current clinical settings, even subjects who lack any identifiable traumatic experiences can be diagnosed with hysteria's nosological successors based purely on the characteristics of their physical symptoms. But despite their excision from the diagnostic context, traumatic life events are still regarded

346 See, e.g., Bègue et al., "Metacognition," 259; Burke et al., "Ancillary Activation," 338; Cojan et al., "Inhibition," 1035; Mailis-Gagnon et al., "'Hysterical' Anesthesia," 1505; and Stone et al., "Simulated Weakness," 966.

347 Burke et al., "Ancillary Activation," 338.

348 See, e.g., Kanaan et al., "Repressed Memories."

349 See, e.g., Voon et al., "Emotional Stimuli."

350 See, e.g., Kranick et al., "Psychopathology"; and Stone and Edwards, "Psychogenic."

351 See APA, *DSM-5*, 319–20. See also my discussion in section 2.4.2.

by many experts as significant contributing factors in the development of hysterical symptoms.[352] Admittedly, in a substantial proportion of hysteria patients, traumatic life events do not seem to be readily identifiable.[353] However, according to multiple recent studies, both early-life and proximal stressful experiences appear to be "substantially more common" in individuals who develop hysterical symptoms than either in healthy subjects or those suffering from other neurological and psychiatric disorders.[354]

Yet, apart from the statistically significant association between stressful life events and the subsequent development of hysterical symptoms, little else seems to be clear. It remains a mystery why some individuals develop hysterical symptoms even in the absence of apparent psychological stressors, some in response to minor difficulties, whereas others experience multiple adverse life events without falling ill as a result.[355] Furthermore, a distinct neuropathological mechanism through which particular adverse life events might influence the development of hysterical symptoms remains unknown. Uncovering this mechanism, however, is regarded as a crucial precondition for developing more effective treatments.[356]

Considering the urgent need for better treatments, it may seem bewildering that the fMRI research in the first two decades of the twenty-first century made hardly any effort to uncover the potential mechanism through which personal traumatic experiences might partake in the formation of hysterical symptoms.[357] As I intend to show, this neglect is not accidental. Rather, it is a direct consequence of multiple methodological challenges associated with having to empirically frame and quantify patients' highly idiosyncratic life experiences. Some of these difficulties are similar to those that characterise the diagnostic encounters between doctors and patients. As discussed previously, such difficulties are related to the perennial distrust in the veracity of patients' self-reports and the resulting problem of how to reliably identify life events relevant to the symptom formation.[358] Other methodological difficulties are specific to fMRI research. The latter type of difficulty arises from the question of how to translate individual traumatic experiences into adequate experimental stimuli. As we will see, such stimuli should enable researchers to use fMRI to unambiguously isolate the neural correlates of the patients' emotional reactions induced through the controlled recall of particular life events.

352 See, e.g., Keynejad et al., "Stress"; Ludwig et al., "Stressful Life Events."

353 See, e.g., Keynejad et al., "Stress," 813.

354 Ludwig et al., "Stressful Life Events," 307. The estimated proportion of patients who lack identifiable stressful life events varied from 14% to 77% across individual studies. Ibid., 314. See also Kranick et al., "Psychopathology"; Nicholson et al., "Life Events"; and Roelofs et al., "Impact of Early Trauma."

355 Kranick et al., "Psychopathology," 1850.

356 Kranick et al., 1850. See also Keynejad et al., "Stress," 813. It appears to me that his current stance represents an interesting parallel to Charcot's shift of focus in the mid-1880s towards developing new treatments by explicitly drawing on his insights into the neurophysiological mechanism underlying the formation of traumatic hysterical paralysis. See section 1.3.2.

357 Although hysterical symptoms are considered to be potentially reversible, due to the paucity of effective treatments, the prognosis "remains collectively poor, with disability persisting or even worsening over time." Espay et al., "Current Concepts," 1139.

358 Craig, "Life Events," 88–90.

The extent of all such methodological challenges is perhaps best illustrated by the following fact. Despite the growing fMRI research into the "influences of emotional processes on the pathophysiology" of various hysterical symptoms,[359] by the end of the 2010s, only two studies explicitly focused on examining the emotional effects of the patients' individual traumatic experiences. The first was the Kanaan et al. study, published in 2007. This study examined the emotional effects of recalling proximal traumatic life events in a single female patient with hysterical arm paralysis and recurring seizures.[360] In a related study published seven years later, Aybek et al. compared neural correlates associated with the recall of adverse life events between twelve patients with hysterical paralysis and thirteen healthy control subjects.[361] Both studies were conducted by the same research group at the King's College London.[362] And in both studies, the researchers used similar procedures to identify each participant's relevant stressful life events and then translate them into experimental stimuli. In this section, we will closely examine these procedures whose aim, as I will suggest, was to deindividualise the patients' traumatic life events in order to make the emotional impact of their recall measurable through fMRI. Additionally, I hope to demonstrate that this deindividualisation was all the more problematic since, in both studies, the authors explicitly claimed to deploy Freud's concept of trauma.

The current unpopularity of Freud's theories, particularly in the medical and neurological discourses on hysteria, was discussed in chapter 2. Thus, in a sense, the two studies I analyse in this section can be regarded as an anomaly in fMRI-based hysteria research. However, their isolated and, as I will claim, failed attempts to revive and adapt Freud's concept of trauma to the procedural logic of fMRI experiments have attracted considerable, mostly positive attention. Their findings were not only summarily quoted in other neuroimaging studies but also uncritically reported in one of the rare articles that dealt with functional neuroimaging research on hysteria in the general press.[363] Hence, although they are not typical of the fMRI-based hysteria research, on the whole, the Kanaan et al. and the Aybek et al. studies must be examined in detail to make evident their inconsistent use of images as epistemic tools.

To begin with, the authors of these two studies stated that a key challenge in identifying actual traumatic experiences lay in hysteria patients' highly specific recall bias.[364] This bias, they claimed, consisted in hysteria patients' inability to assess the emotional relevance of the past events that had triggered their symptoms. By grounding their claim in Freud's theory, the authors speculated that hysteria patients could not be aware of the actual emotional relevance of the key traumatic events precisely because

359 Blakemore et al., "Aversive Stimuli," 230. The studies that have investigated patients' emotional states through the use of standardised stimuli will be analysed in the subsequent section.

360 Kanaan et al., "Repressed Memories."

361 Aybek et al., "Life Events", 52.

362 Three of the initial study's four authors co-authored the follow-up study. Compare Kanaan et al., "Repressed Memories"; and Aybek et al., "Life Events."

363 See, e.g., Blakemore, "Aversive Stimuli"; and Hassa et al., "Motor Control." For the article in the general press, see Gale, "Freud's Hysteria."

364 Kanaan et al., "Repressed Memories," 202; and Aybek et al., "Life Events," 53.

they repressed the memory of how upsetting these experiences had been initially.[365] Since they chose to use fMRI "to elucidate the processing of the emotional events relevant" to the symptom formation, the authors concluded they first had to find a way to bypass the patients' purported recall bias.[366] Instead of relying on the patients' subjective self-reports, the researchers opted to use "more objective ratings" of what constituted the aetiologically relevant emotionally charged life events.[367] With this purpose in mind, they decided to implement a standardised method called the Life Events and Difficulties Schedule (LEDS). This method was developed in the late 1970s to enable clinicians to "quantify stressful life events" in the psychiatric population.[368]

The LEDS method entails a two-stage procedure. In the first stage, researchers conduct a two- to four-hour-long semi-structured interview with each subject. During the interview, researchers enquire about the subject's "different life domains, such as health, accommodation and employment."[369] The aim is to detect discrete experiences with potentially adverse emotional impact and to identify their exact onset. Just as importantly, researchers also collect "detailed information about the subject's plans and goals and the wider social context at the time" these specific events occurred.[370] Next, based on the information collected, the interviewer creates a narrative for each adverse life event thus identified.[371] These narratives then undergo what is referred to as the rating of the contextual meaning. At this stage, a panel of raters judge "the likely effect of the event on the average person with the plans, biography, and circumstances of the participant, but ignoring the participant's reported reaction to the event at the time."[372] Although different aspects of the events' contextual meaning can be rated, the most widely used standardised category is 'severity.'[373] Each rater quantifies the severity by estimating on a scale of 1 to 4 how threatening the likely long-term consequences of

365 Kanaan et al., "Repressed Memories," 202; and Aybek et al., "Life Events," 53. Not all present-day experts agree with this conjecture. Unlike Kanaan et al., other authors have suggested that the recall bias "can occur in both directions: patients might overly recall negative versus positive events, other patients might have experienced terrible maltreatment but deny it in interviews and questionnaires." Ludwig et al., "Stressful Life Events," 318, Panel: Issues with Methods.

366 Kanaan et al., "Repressed Memories," 202.

367 Kanaan et al., 202.

368 Kanaan et al., 202. In 1978, the psychologists George Brown and Tirril Harris developed this method to study depression. For a succinct overview, see Craig, "Life Events," 90–91. See also Brown and Harris, *Social Origins of Depression*. The LEDS method had already been used in a few studies that examined the frequency of antecedent traumatic experiences in patients with various hysterical symptoms. See, e.g., House and Andrews, "Life Events"; and Craig et al., "Somatisation Study." However, the Kanaan et al. study was the first to implement the LEDS in the context of fMRI-based hysteria research.

369 Nicholson et al., "Life Events," 2618.

370 Craig, "Life Events," 91.

371 Kanaan et al., "Repressed Memories," 203.

372 Aybek et al., "Life Events", 52. For instance, when assessing the event's severity, raters would take into account "not only of the immediate situation (say, a loss of a job) but also of the wider context (whether there are debts, whether other members of the household are in secure employment, the current level of employment opportunity in his trade and so on)." Craig, "Life Events," 91.

373 Craig, "Life Events," 91. In an acknowledgement that severity is a "crude way to describe stressful experience," there have been "continual attempts to refine" this concept by dividing it into

a particular event appear in the given circumstances.[374] The final rating is obtained through a consensus among the raters.

Applying the LEDS method, Kanaan et al. identified two "equally severe" adverse life events that had closely preceded the onset of hysterical symptoms in their female patient with right-sided paralysis.[375] The 'severe' events included her daughter's attempted suicide and her long-term partner's announcement that he was leaving her. The daughter's attempted suicide predated the symptoms' onset by a month. In contrast, the break-up announcement occurred immediately before the symptoms' onset.[376] After her partner had announced the intended break-up, the patient first lost consciousness and then developed right-sided paralysis and anaesthesia shortly afterwards. Significantly, the patient's subjective evaluation of the emotional relevance of these two events was in stark contrast to the rating panel's conclusion that they were of equal severity. Contrary to the panel's purportedly "objective ratings of her life events," the patient insisted that her daughter's suicide attempt was "a harrowing experience," whereas her partner's break-up "was not at all distressing."[377] However, it was precisely based on this significant discrepancy between the panel's rating and the personal meaning reported by the patient that Kanaan et al. categorised the break-up announcement as "an emotionally repressed event."[378] In other words, Kannan et al. implied that, although her daughter's suicide attempt was a highly stressful experience in its own right, this event was not aetiologically related to the symptoms because the patient was able to recognise its emotional impact. By contrast, the patient's inability to acknowledge the 'objective' emotional salience entailed in the break-up announcement was taken to mean that this severe event was "crucial to the genesis of her symptoms."[379]

To additionally justify their differential attribution of aetiological relevance to these two otherwise purportedly equally severe life events, Kanaan et al. went a step further. Not only did they emphasise the temporal proximity of the break-up announcement to the symptom formation, but they also took recourse to Freud's concept of secondary gain. In his later work, Freud introduced a differentiation between the primary and the secondary gain that patients could derive from hysteria.[380] According to Freud, the primary gain from falling ill consisted in the "saving of psychical effort" since the symptom formation enabled the patient to alleviate an internal psychological conflict or trauma.[381] Emphasising this point, Freud also denoted the primary gain as "a

components such as 'loss' and 'danger,' or developing new measures. Ibid., 93. See also House and Andrews, "Life Events."

374 Nicholson et al., "Life Events," 2618. On this scale, 1 refers to marked threat/severity, whereas 4 to little or no threat.

375 Kanaan et al., "Repressed Memories," 203.

376 Kanaan et al., 202.

377 Kanaan et al., 202.

378 Kanaan et al., 203.

379 Kanaan et al., 203.

380 Freud first explicitly introduced the distinction between the primary and the secondary gain in 1909 in his paper on hysterical attacks. See Freud, "Hysterical Attacks," 231–32. But it was in his Lecture 24 (1916–17) and a footnote he added in 1923 to Dora's case history that Freud elaborated on this distinction. See Freud, "Common Neurotic State," 381–85; and Freud, "Case of Hysteria," 43n.

381 Freud, "Case of Hysteria," 43n. See also section 2.1.3.

flight into illness."[382] Moreover, he stated that, in some circumstances, an additional secondary gain might arise from falling ill. Such a secondary gain consisted in attaining some "external or accidental" advantage from the illness, which thus "becomes a weapon" that can be used for defence or revenge.[383] In an implicit reference to Freud, Kanaan et al. speculated that their patient could have accrued secondary gain only concerning the announced break-up. In this case, what she could have gained by falling ill was "preventing, or at least delaying, her partner's leaving."[384] This additional aspect was meant to provide decisive proof for the conjecture that the break-up announcement—and not the daughter's suicide attempt—was "the key event" causing the symptom formation.[385]

Yet to use fMRI to test if the patient's cued recall of the two severe life events would indeed induce different emotional processing at the neural level, Kanaan et al. had to complete two more preparatory steps. First, they needed to choose one 'non-stressful' life event from the same period, which on the LEDS scale was rated as lacking any threat potential. The purpose of the non-threatening event (the patient's visit to her sister) was to serve as a baseline condition. It was in relation to the non-threatening event that the potentially negative emotional impacts of both the repressed and the equally severe events were meant to be isolated in the fMRI experiment.[386] Second, Kanaan et al. had to design a task that would induce the patient to emotionally re-experience all three life events in a controlled manner. For this purpose, the researchers created twenty-four length-matched statements for each event. A quarter of these statements were deliberately changed to contradict the facts reported by the patient.[387] The statements were divided into blocks of eight for each event and presented to the subject inside the scanner as a set of auditory recordings. Kanaan et al. took care to sequence the statements in a way that "minimize[d] the overlap of affective response between events."[388] With the aim of eliciting a vivid recall of her traumatic memories, the patient was asked to determine whether the statements referring to her life events were true or false. In addition to acquiring fMRI data, the researchers also measured the patient's reaction times and the accuracy of her responses.

Subsequent analysis of the behavioural data disclosed no statistically significant differences in the patient's reaction times or the accuracy of her responses across the events. But the fMRI map that visualised the patient's neural responses to the recall of the break-up relative to the daughter's suicide attempt revealed increased activation in the brain areas typically involved in the emotion processing. The overactive areas

382 Freud, 43n.

383 Freud, "Common Neurotic State," 383. As an example of secondary gain, Freud mentioned a woman whose illness provided a symbolic escape from her domineering husband. Ibid.

384 Kanaan et al., "Repressed Memories," 203.

385 Kanaan et al., 203.

386 Kanaan et al., 203.

387 For example, the patient "recalled having to break into her daughter's room during the overdose." Her statement 'it was easy to kick the door down' was changed into 'it was hard to kick the door down.' Kanaan et al., 203.

388 Kanaan et al., 203.

included the right amygdala and the right inferior frontal cortex.[389] Additionally, the same map also displayed a decreased activation in the area of the primary motor cortex associated with the paralysed limb. Kanaan et al. concluded that their fMRI map contradicted the patient's subjective evaluation of her traumatic experiences by showing that "the 'break-up' event was *more* emotionally salient than" her daughter's attempted suicide.[390] Moreover, the researchers argued that by linking the recall of this event to the symptom's underlying dysfunction of the motor system, their study provided "neuroimaging evidence" that the 'break-up' event appeared to produce the patient's paralysis.[391]

However, there were several major caveats to the apparently straightforward conclusion of the Kanaan et al. study. First, the resulting pattern of activations failed to support the researchers' a priori conjecture that the break-up event underwent the process of memory suppression. Based on their conjecture, Kanaan et al. had expected to identify decreased activation in both the hippocampus and amygdala, which would have reflected "inhibition of memories and emotional salience."[392] Instead, their fMRI map displayed increased activity of the amygdala and no differential activation of the hippocampus.[393] Kanaan et al. merely glossed over this significant inconsistency and, despite the lack of empirical confirmation, continued to refer to the break-up as "an emotionally repressed event."[394] Second, Kannan et al. could not provide a clear-cut explanation as to why the emotional arousal that the recall of the break-up appeared to induce at the physiological level—as indicated by the amygdala activation—remained cut off from the patient's conscious awareness. In fact, while discussing their imaging findings, Kanaan et al. admitted that the neural activations isolated by the contrast between the recall of the break-up and the "equally severe event" of the daughter's attempted suicide were not unambiguously interpretable in cognitive terms.[395]

Third, the suggested causal linking of paralysis to the particular emotional event was highly uncertain and speculative. Since the task the researchers deployed did not entail any explicit or implicit movement, the deactivation of the motor cortex in their map may have been purely incidental. Fourth, because it was a single-subject study, the imaging findings were neither empirically reliable nor generalisable to other hysteria patients.[396] Hence, on the whole, it appears to me that the Kanaan et al. study raised more questions than it answers.

In 2014, the same research team published a new study. In it, the researchers returned to the question of "the neural correlates of recall of life events judged to

389 Kanaan et al., 203.

390 Kanaan et al., 203 (emphasis in original).

391 Kanaan et al., 202. See also ibid., 204.

392 Kanaan et al., 204.

393 Kanaan et al., 203.

394 Kanaan et al., 203.

395 Kanaan et al., 203.

396 As pointed out by Kanaan et al., because it was a single-case study, the risk of both false positive and false negative voxels in their fMRI map was "considerable." Kanaan et al., 203. Even more problematically, the researchers did not perform any multiple comparisons corrections, which means they failed to control the amount of false-positive activations.

be of causal significance" in the development of hysterical symptoms.[397] Also in this case, Aybek et al. deployed the LEDS-based approach to identify the relevant life events. However, in an apparent need to avoid the ambiguities that had hampered the previous study, Aybek et al. introduced several significant methodological changes. To avoid the pitfalls of a single-case study, Aybek et al. recruited twelve patients and thirteen healthy control subjects, thus increasing the potential generalisability of their results.[398] Interestingly, although they aimed to examine the possibly causal significance of personal traumatic life events, Aybek et al. did not recruit patients based on the compatibility of their stressful experiences or the similarity of their biographies. Instead, the twelve patients, all with either unilateral or bilateral paralysis, were selected to obtain "relative symptom homogeneity" which the authors explicitly foregrounded as the study's particular strength.[399] Put simply, their sampling strategy was framed in decidedly somatic terms. In a curious parallel to Charcot's approach, Aybek et al. placed the focus not on the particular content of the adverse life events but on the type of physical effects that these events elicited in the patients.

Moreover, compared to the Kanaan et al. study, Aybek et al. started with considerably stronger assumptions about how hysteria patients cognitively processed emotionally adverse events. Referencing Freud, Aybek et al. hypothesised that patients "wilfully ignored (or repressed)" the stressful events that were causally linked to their symptoms and that "subsequent illness invariably led to some benefit or 'secondary gain.'"[400] Two aspects are of significance here. First, we have seen that, in the previous study, the authors loosely indicated the potential aetiological significance of the secondary gain without further specifying it. Here, they went a step further. They declared the purported secondary gain to be the key reason why hysteria patients repressed memories of traumatic events, thus causing a symptom to appear. However, Aybek et al. not only failed to provide any empirical evidence for this assumption but also erroneously attributed it to Freud.[401] According to Freud, although the secondary gain may contribute a motive for developing a symptom, it is "not present at the beginning of the illness" but only "appears secondarily to it" and "strengthens its stability."[402] Hence, for Freud, the primary gain, which is of psychological nature, provides the motive for falling ill, whereas a potential secondary gain merely plays a role in maintaining the illness.

Second, Freud explicitly and repeatedly characterised repression as an unconscious psychological defence mechanism.[403] Contrary to Freud, Aybek et al. defined repression

397 Aybek et al., "Life Events," 52.

398 Aybek et al., 52.

399 Aybek et al., 54, 59.

400 Aybek et al., 52.

401 Aybek et al. referenced several studies that showed the purported "presence of secondary gain" in hysteria. Aybek et al., 52. However, the researchers failed to mention that none of the studies they referred to provided evidence for the causal significance of the secondary gain in the formation of hysterical symptoms. See ibid.

402 Freud, "Case of Hysteria," 42; and Freud, "Common Neurotic State," 384.

403 Admittedly, in his early work, Freud talked about "things which the patient wished to forget, and therefore intentionally repressed from his conscious thought." Freud and Breuer, "Preliminary

as a conscious cognitive process akin to voluntary forgetting of unwanted memories.[404] They further suggested that several recent fMRI studies provided an adequate model of Freudian repression by using a so-called think/no-think paradigm.[405] Specifically, the authors of these studies have examined neural activations in healthy subjects who were asked to either think or avoid thinking about stimuli to which they had been exposed previously and none of which were related to their personal life events.[406] As we will see shortly, the assumption that repression was a conscious cognitive process played a central role in how Aybek et al. chose to analyse and interpret their fMRI data. But first, let us analyse how their assumptions about the key aetiological role of the secondary gain informed the process through which Aybek et al. identified the relevant stressful life events in their subjects and then translated them into experimental stimuli.

Based on their assumption that the secondary gain played a crucial role in the formation of hysterical symptoms, Aybek et al. decided to quantify it by applying the LEDS method. Yet to be able to measure this aspect of traumatic life events, Aybek et al. had to develop a novel LEDS category they labelled 'escape.' They defined the life event's escape potential "as the extent to which a subsequent illness might reduce the effect or consequences of the stressor, affording a socially sanctioned means to avoid a difficult situation."[407] They then introduced a scale for rating the escape potential that ranged from 0 (none) to 3 (marked).[408] On the surface, and particularly in its name, the new category of escape may have appeared to revive Freud's concept of a 'flight into illness.' Instead, I venture to say that Aybek et al. substantially distorted Freud's concept. Not only did they reduce the flight into illness to the external secondary gain. They also entirely neglected the role of the primary gain, which, as discussed above, was of central importance to Freud. This conceptual distortion is evident in the example that Aybek et al. provided on how to rate the escape potential of a stressful event. According to Aybek et al., "a spouse's sudden death would offer minimal escape potential because the individual's subsequently becoming ill would do little to alleviate the stressor; however, a partner threatening to break off a relationship would have substantial escape potential because the individual's becoming ill might prevent the partner's feeling able to abandon the individual when he or she was unwell."[409]

After introducing their new rating category of escape, in the next step, Aybek et al. identified relevant stressful events for each study participant. Following individual

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Communication," 10. But as emphasised by Freud's editor James Strachey, "the word 'intentionally' merely indicates the existence of a motive and carries no implication of conscious intention." Ibid., 10n. For Freud's explicit statements about the unconscious nature of repression, see, e.g., Freud, "Resistance and Repression," 294–98; and Freud, "Common Neurotic State," 385.

404 Aybek et al., "Life Events," 52, 56.

405 Aybek et al., 53.

406 See Anderson and Green, "Suppressing"; Anderson et al., "Unwanted Memories"; and Depue et al., "Emotional Memories." The stimuli were unrelated word pairs (e.g., ordeal and roach) or paired pictures. See Anderson and Green, "Suppressing," 366; and Depue et al., "Emotional Memories," 215.

407 Aybek et al., "Life Events," 53.

408 Nicholson et al., "Life Events," 2619.

409 Aybek et al., "Life Events," 53.

interviews with both patients and healthy subjects, the panel of raters first assessed the likely threat of each stressful life event using the standard category of severity. As in the previous study, the raters judged how an 'average' person in comparable life circumstances might react to the event in question. In doing so, the raters disregarded the individuals' reports on the subjectively perceived emotional relevance of these experiences. Those events that, due to their "matched objective threat," were classified as severe, underwent a subsequent evaluation of their escape potential.[410] The panel assessed the escape potential of severe events for both patients and healthy subjects as if the individual in question had developed hysterical paralysis. Finally, for each study participant, Aybek et al. chose one severe escape event (referred to as 'escape'), one severe non-escape event (termed 'severe'), and one 'neutral' event from the same period. Interestingly, unlike Kanaan et al., Aybek et al. provided no information about their subjects' life events. As a result, the reader is kept entirely in the dark concerning the particular contents of the life events labelled by the researchers as severe, escape and neutral.

After selecting and categorising the participants' life experiences, Aybek et al. then translated these events into experimental stimuli. They deployed the same approach as in the Kanaan et al. study. Yet, in this case, they had to create twenty-four length-matched statements for each event type and each of the twenty-five participants separately.[411] As in the Kanaan et al. study, a quarter of the statements were rendered incorrect. Also in this case, the subjects were asked to judge the statements as true or false while lying in the scanner. Contrary to the Kanaan et al. study, the statements were not presented as audio recordings but instead appeared as text on the screen. Another methodological novelty was that, after each block of eight statements about a single event, the subjects had to rate on a scale of 1 to 10 how upsetting they found the particular statements.[412] In addition to the fMRI data, the subjects' reaction times and false responses were also recorded.

As in the Kanaan et al. study, Aybek et al. began the fMRI data analysis by deploying each subject's neutral event as a baseline condition. Hence, in relation to this baseline, Aybek et al. computed the differential neural responses induced by severe and escape events separately. They then used these intermediary results of single-subject analyses as input for the group analysis. At the group-level analysis, the researchers assessed the differences in the effects of severe and escape events between the patients and healthy control subjects. As I intend to show, at this point, Aybek et al. started to inappropriately combine different analytical approaches to generate fMRI maps supporting their a priori hypothesis that hysteria patients processed escape events in a manner analogous to voluntary suppression of unwanted memory. Specifically, two previous fMRI studies have linked voluntary memory suppression in healthy subjects to the increased activation in the dorsolateral prefrontal cortex (dlPFC) and the right inferior frontal gyrus (rIFG), as well as the reduced activation in the hippocampus.[413]

410 Aybek et al., 56.

411 Aybek et al., 53.

412 Aybek et al., 53.

413 See Anderson et al., "Unwanted Memories"; and Depue et al., "Emotional Memories."

In what follows, I argue that, instead of performing a rigorous data analysis, Aybek et al. made biased choices aimed at seeking out precisely those activations the previous studies of voluntary memory suppression had identified.

At the group level, Aybek et al. first conducted a conventional whole-brain analysis by computing statistically significant differential responses to severe versus escape events between patients and healthy controls. Following the statistical thresholding and the correction of the multiple comparisons problem, Aybek et al. obtained fMRI maps. The resulting maps displayed increased activation in the supplementary motor area (SMA) and the temporoparietal junction (TPJ). Additionally, the maps disclosed decreased activation in the hippocampus during the recall of escape relative to the severe event in patients compared to healthy controls.[414] The resulting whole-brain maps thus showed the expected hypoactivation of the hippocampus. However, they failed to reveal any differential activation in either the dlPFC or the rIFG. Undeterred by these partially negative results, Aybek et al. conducted two additional selective data analyses. In each of these analyses, the search for statistically significant effects of the experimental manipulation was constrained anatomically to either the dlPFC or the rIFG as the predefined regions of interest (ROI).

By switching from the whole-brain to the ROI analysis, Aybek et al. were able to "increase the sensitivity of searches for regionally specific effects in the main experiment (by reducing the problem of multiple statistical comparisons)."[415] Moreover, due to the reduced problem of multiple comparisons in this type of analysis, Aybek et al. chose to apply a less stringent correction method than in the previous whole-brain analysis.[416] As a result, the activation maps computed through the selective ROI analyses had a more liberal statistical threshold. In the neuroimaging community, such "use of inconsistent and erratic statistical threshold in the same study" is considered biased.[417] Hence, strictly speaking, the approach deployed by Aybek et al. is known to inflate the rate of false-positive results and is, therefore, contrary to the standards of good scientific practice. Yet, such selective use of different statistical thresholds across different analyses enabled Aybek et al. to turn their initially negative into positive results. Unlike the whole-brain maps, those subsequently computed through the ROI analyses succeeded in detecting purportedly statistically significant activations both in the dlPFC and the rIFG, respectively. In one of the new maps, the rIFG appeared to show "significantly less activation" in patients than healthy subjects across both types of events.[418] Additionally, the dlPFC displayed greater activation in both patients and healthy subjects for the contrast between the escape and severe events. However, Aybek

414 Aybek et al., "Life Events," 55. For a discussion on thresholding, see section 3.4.3.

415 Friston et al., "Critique of Functional Localizers," 6.

416 In the whole-brain analysis, Aybek et al. applied a familywise error rate correction called the random field theory. In the ROI analysis, however, they used a less stringent small-volume correction (SVC). Aybek et al., "Life Events," 54. For details on SVC, see Poldrack, Mumford, and Nichols, *Handbook*, 183.

417 David et al., "Potential Reporting Bias," 7, e70104. "The use of Small Volume Correction (SVC) techniques in addition to standard whole-brain analyses may be used to alter the statistical threshold in selected ROIs, thus impacting on the number of foci reported." Ibid., 8, e70104.

418 Aybek et al., "Life Events," 55.

et al. had hypothesised that the dlPFC was differentially activated across the two groups during the recall of the escape event. To support this hypothesis, they computed yet another map, which finally appeared to 'confirm' the researchers' assumption that the dlPFC's activity "was driven by the patients."[419]

Having used different types of analyses and inconsistent statistical thresholds to make their data yield the effects for which they were looking, Aybek et al. proceeded to an equally biased interpretation of the resulting fMRI maps. First, they argued that the "increased left DLPFC activity during the escape condition relative to the severe condition in patients vs controls, together with decreased hippocampal and parahippocampal activity" provided evidence that hysteria patients processed escape events "through the mechanism of 'direct suppression.'"[420] In this type of voluntary suppression, which previous fMRI studies had experimentally modelled by asking healthy subjects to avoid thinking about cued stimuli, "the conscious recollection of an unwanted memory (mediated by the hippocampus) is disrupted by top-down regulation (mediated by the DLPFC)."[421] Notably, this apparently straightforward interpretation glossed over one significant fact. As discussed above, the dlPFC hyperactivation and the hippocampal hypoactivation were separate outcomes of two categorically different types of statistical analyses.[422] Hence, as I see it, by implying that these activations were part of the same pattern and thus jointly constituted a single neurocognitive mechanism, Aybek et al. misrepresented their imaging results.

Furthermore, to support their a priori assumption that the "activation pattern of memory suppression" displayed by their maps was analogous to Freudian repression, Aybek et al. turned to the behavioural data.[423] These showed that the escape events "were perceived as less upsetting than severe events, although both types of events were of matched objective threat," at least according to the panel's LEDS ratings.[424] Aybek et al. suggested that these findings were "compatible with Freud's concept of repression."[425] In their interpretation, the behavioural data purportedly demonstrated that "the painful aspects of the emotional stimuli presented during the escape condition" were made less upsetting through the hypothesised mechanism of voluntary

419 Aybek et al., 55.

420 Aybek et al., 55–56.

421 Aybek et al., 56.

422 In fact, what made such conflation of these separate findings problematic was not limited to the use of different statistical thresholds. The additional problem was that the whole-brain and ROI analyses pose two categorically different questions about the fMRI data. As discussed previously, a whole-brain analysis aims to localise the voxels that exhibit statistically significant responses to the experimental manipulation. By contrast, in a selective ROI analysis, the question is not where the responses are since the location is already defined by selecting the region of interest. Instead, in an ROI analysis, "the nature of the response variable is changed quantitatively, from a collection of regional responses at each voxel to a summary of their collective responses, that is, average." Friston et al., "Functional Localizers," 8. In the latter case, "the fMRI signal is characterized within a defined region and analysed as an aggregate rather than voxel by voxel." Poldrack, Mumford, and Nichols, *Handbook*, 183.

423 Aybek et al., "Life Events," 57.

424 Aybek et al., 56.

425 Aybek et al., 56.

suppression.[426] Conveniently, Aybek et al. failed to emphasise one crucial point. Both the patients and the healthy participants assessed the escape events as significantly less upsetting than those that, according to the panel's "[o]bjective ratings," had been categorised as equally severe non-escape events.[427] In short, there were no differences at the group level between the patients and healthy subjects in how they subjectively rated the emotional salience of either the severe or the escape events. In my opinion, Aybek et al. remained silent about the lack of group differences between the patients and healthy subjects because it posed a fundamental empirical challenge to the validity of their newly introduced category of escape events.

In principle, the fact that both groups equally failed to perceive the purportedly 'objective' level of threat entailed in the escape events could be taken to mean that both patients and healthy subjects repressed the emotional contents of these particular events. In such a case, however, it would make little sense to claim, as Aybek et al. did, that repression of this particular type of event played a causal role in developing hysterical symptoms. After all, the healthy participants of the study were, without exception, asymptomatic. Alternatively, the lack of behavioural differences between patients and healthy subjects could also be interpreted as an indication that the artificially constructed category of the escape events was not an adequate measure of the extent to which hysteria patients purportedly repressed their memories of traumatic life experiences. This second interpretation seems far more plausible to me. Yet Aybek et al. pointedly avoided both of these alternative explanations. Instead, they chose to ignore the behavioural data on the healthy subjects and selectively focused only on those from the patients. Such distortion of focus allowed them to use the behavioural data to erroneously support their a priori assumption that solely the stressors with a secondary gain potential (i.e., escape events) had causal significance in hysteria as the only type of events whose emotional content patients supposedly repressed.[428]

To further substantiate their claim that the way hysteria patients processed escape events was highly specific and causally related to their physical symptoms, Aybek et al. turned to the interpretation of additional activations patterns displayed by their fMRI maps. First, they conjectured that, when exposed to stress triggered by the recall of any type of adverse events, hysteria patients exhibited an impairment of early-stage emotional regulation.[429] This impairment, in turn, made hysteria patients more prone to increased emotional arousal. Aybek et al. based this conjecture on an ROI-based fMRI map that showed decreased activation in the rIFG in patients compared to healthy controls across both severe and escape events. Second, the researchers claimed that only the exposure to "a specific stressor (recall of an escape event)" triggered a highly particular neural response in the patients.[430] Significantly, this purportedly specific response was not limited to the "activation pattern of memory suppression" discussed

426 Aybek et al., 56.
427 Aybek et al., 55.
428 Aybek et al., 56–57.
429 Aybek et al., 58–59.
430 Aybek et al., 59.

above.[431] Rather, it was also "associated with abnormal activity in the TPJ and SMA, which may represent neural correlates of a patient's physical symptoms."[432] Even more speculatively, Aybek et al. suggested that the patients' increased SMA activity during the recall of escape events "may reflect an impaired ability to select the correct automatic motor plan at an unconscious level."[433] This finding seemed to align, so they claimed, with Freud's concept of conversion, since it could be taken to imply a transformation of the repressed event's emotional content into a physical symptom.

At a superficial glance, it might appear that, in the end, Aybek et al. succeeded in piecing together all their findings into a coherent narrative. It might also appear that this narrative endorsed the researchers' initial hypothesis concerning the causal significance of the newly defined category of escape events and that it provided the empirical validation for Freud's psychogenic theories of hysteria. However, my detailed analysis has shown that the seemingly clear-cut conclusions drawn by Aybek et al. were grounded in a biased combination of analytical approaches specifically tailored to find the exact patterns of activation the researchers had expected. Moreover, I have also foregrounded the researchers' often selective and thus highly problematic interpretation of the behavioural and fMRI findings that were cherry-picked to fit their a priori hypotheses. My intention here is not to imply that Aybek et al. acted in bad faith. It is more likely that their biased data analysis and interpretation were motivated by a possibly overzealous desire to curtail the ambiguity of their initial whole-brain findings. After all, as discussed previously, the same team of researchers had already faced similar methodological and interpretational challenges in their previous single-case study published in 2007. It thus seems to me that the interpretational ambiguity of the whole-brain imaging findings in both studies was an unavoidable consequence of how these findings were produced. In both the Kanaan et al. and the Aybek et al. studies, the fMRI maps were produced through what, in my view, was an arbitrary contrasting of the experimental subjects' highly idiosyncratic and essentially incomparable personal life events.

On the whole, I find problematic the researchers' attempt to subsume the complex life experiences of individual subjects to the procedural logic of an fMRI experiment by reducing these experiences to the abstract and purportedly quantifiable categories of severe escape (i.e., 'escape') and severe non-escape (i.e., 'severe') events. Admittedly, the intended purpose of such categorisation was to construct the mutual comparability of heterogeneous personal experiences and, in turn, the measurability of the emotional reactions that their cued recall induced. Yet, this categorisation rested on several

431 Aybek et al., 57.

432 Aybek et al., 59.

433 Aybek et al., 57. Significantly, Aybek et al. omitted to mention that a previous fMRI study of voluntary memory suppression in healthy patients also found increased activity in the SMA during the cued inhibition of recall. See Anderson et al., "Unwanted Memory," 233. In their interpretation, Anderson et al. emphasised that, apart from its role in movement execution, the SMA is also "activated by visual selective attention" and by "purely cognitive tasks that demand updating in memory and require no motor output." Anderson et al., 235. Since the Aybek et al. study did not entail any motor tasks, it remains unclear which of these different cognitive processes could have been associated with the patient's increased activity of the SMA.

questionable assumptions. First, the construction of the events' comparability hinged on their detachment from the felt experiences of individual subjects. This, as we have seen, was achieved by ignoring the participants' subjective assessment of their life events. What mattered instead was the rating panel's evaluation of the potential impact that a particular adverse event would have had on a hypothetical 'average' person as a common point of reference. In other words, imagined reactions of a fictive 'average' person to the participants' actual adverse experiences were declared to provide an 'objective' measure of the events' emotional impact. But as we have seen, this fictive 'average' person was neither a fixed nor even an explicitly defined concept. Rather, it was a construct that emerged through a consensus among the members of the rating panel.

Hence, the supposedly 'objective' classification and quantification of life events hinged on the raters' possibly normative assumptions about how one should emotionally react to a given traumatic situation. Despite the claims of high inter-rater reliability[434]—i.e., an agreement among different researchers—the resulting concept of the 'average' person appears to me very vague, arbitrary and problematic. Furthermore, I think it is safe to assume that, at least implicitly, the raters' judgments could have been influenced by their socio-cultural backgrounds, which may have differed from those of their study participants. Such potential socio-cultural differences necessarily inform one's explicit and implicit views on what counts as 'average' behaviour in the given circumstances and may have unintentionally biased the raters' assessment of the impact the adverse life events had on the individuals who had actually experienced them.

Equally debatable is another central assumption of the Aybek et al. study. Specifically, the fMRI data analysis was based on the implicit assumption that the only difference in the cognitive effects induced by the recall of a severe non-escape instead of a purportedly equally severe escape event consisted in the repressed emotional content of the latter type of event. In what appears to be a particularly disputable move, the researchers argued that the patients' inability to acknowledge the purportedly 'objective' severity of the escape events demonstrated that they had repressed the emotional content of these specific stressors. The researchers thereby selectively disregarded a highly significant fact that, as discussed above, the healthy subjects also disagreed with the panel's 'objective' ratings and consistently assessed the escape event as less upsetting than those categorised as severe. Just as interestingly, Aybek et al. also failed to mention that, if Freudian repression of the escape events had actually taken place, the patients would not only be unable to readily recall the emotional content of these memories. According to Freud, repressed traumatic memories were entirely inaccessible to the patients' conscious recollection.[435] Hence, the patients would not even be able to report having such memories during a LEDS interview.

434 Aybek et al., "Life Events," 53.

435 As Freud insisted, the repressed memories play no part in the patient's "thinking—do not enter into his consciousness—and thus remain unknown to him." Freud, "Psycho-Analysis," 108. In Freud's view, this "hidden psychical material" could only be uncovered through laborious long-term psychoanalysis. Ibid.

Furthermore, although never explicitly stated, the study's underlying premise was that once the diverse personal experiences had been classified into escape and severe events, they could be unproblematically translated into experimental stimuli. The resulting stimuli could, in turn, induce discrete and uniform emotional reactions in the subjects. However, we have discussed earlier that in fMRI research, even a seemingly simple task, such as trying to move a paralysed limb, is considered highly ambiguous because it elicits confounding activations related to the cognitive consequences of the failed movement. By comparison, it is even more conceivable that, in each patient, the experimentally induced recall of personal traumas gave rise to confounding idiosyncratic cognitive effects, which were impossible to either fully predict or to control. After all, the stimuli were rated in relation to a hypothetical 'average' person but used on real people whose actual reactions did not necessarily conform to the researchers' expectations.

In my opinion, the researchers also failed to consider other possible emotional aspects of the traumatic events that did not fit into their predefined categories of severity or escape. But the fact that they were not categorised did not mean that these additional emotional aspects did not affect the subjects during the recall. For example, it seems to me that, depending on their particular content—which the researchers chose not to disclose—different events could have induced a range of diverse emotional reactions, such as shame, fear, disgust, remorse or anger. Consequently, I suggest that any subtraction of the neural responses between two supposedly equally severe events with disparate 'escape potentials' or their averaging across multiple individuals with different life experiences was necessarily confounded by unaccountable cognitive effects elicited through the recall of complex personal memories. To be sure, fMRI maps computed through such comparisons displayed anatomically localisable activations. However, I argue that these images were not unambiguously interpretable and that, in turn, any attempt to impose a seemingly clear-cut meaning onto them was necessarily deceptive.

Finally, I would like to problematise the claim put forward by Aybek et al. that their imaging findings provided empirical evidence for Freud's psychogenic theories of hysteria. Throughout this section, I have delineated how Aybek et al. have distorted both Freud's concept of repression and the secondary gain to make them fit their assumptions about the possible causal role of traumatic life events in hysteria. Even more importantly, in my opinion, the quantitative handling of the patients' life events in the Aybek et al. study contradicts Freud's basic tenets about hysteria. As discussed in chapter 2, Freud argued that the traumatic impact of a particular life event could only be understood by deciphering the symbolic value and the personal meaning the individual patient attached to it. He also insisted that a single traumatic memory could have multiple simultaneous meanings. According to Freud, such concurrent meanings were not necessarily compatible with one another and could even change with time.[436] Furthermore, he claimed that the formation of hysterical symptoms was not reducible to a single traumatic event but was caused by a chain of mutually interacting memories

436 See, e.g., Freud, "Case of Hysteria," 41, 53.

of multiple adverse experiences.[437] Thus, I venture to say that to Freud, it would make little sense to detach traumatic memories from the patient's subjective assessment of their meaning and evaluate them instead in relation to a hypothetical 'average' person. And it would probably make even less sense to Freud to quantify or subtract the purported emotional impact of different memories, as Aybek et al. did.

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To conclude, my analysis has shown that far from empirically substantiating Freud's views, Aybek et al. simplified his concepts to the point of distortion. In my opinion, Aybek et al. also failed to produce any significant new insights into the possible causal relationship between the patients' personal traumatic memories and the formation of hysterical symptoms. In all due fairness, however, it appears to me that Freud's treatment of personal traumatic memories in their polysemantic richness is not readily translatable into a physiological context within which fMRI studies operate. More specifically, Freud's central tenet that the causal role of traumatic memories in hysteria is determined by the personal meanings these memories have for each patient does not seem to be empirically testable through fMRI. Hence, how to experimentally operationalise the potential aetiological role of personal traumatic memories in the formation of hysterical symptoms is still an open question in the current fMRI hysteria research. It remains to be seen if future studies will manage to find a way of making the emotional impact of personal traumatic memories measurable by fMRI in non-reductive ways. But to achieve this goal, I think that researchers will have to reconcile the medium-specific focus on producing generalisable neurophysiological findings with the need to do justice to the patients' inherently complex and unavoidably idiosyncratic subjective experiences of their traumatic life events.[438]

4.3.2 Using Standardised Visual Stimuli to Investigate Hysteria Patients' Aberrant Emotion Processing

Contrary to the scarcity of fMRI research into the effects of autobiographical traumatic events, since 2010, a continually rising number of imaging studies have used a different

437 In this chain, "the traumatic scenes do not form a simple row, like a string of pearls, but ramify and are interconnect like genealogical trees." Freud, "Aetiology of Hysteria," 196–97.

438 It is interesting to note that, at the beginning of the third decade of the twenty-first century, two fMRI studies have pioneered a new approach to investigating the potentially aetiological role of early-life adverse events in developing hysterical symptoms. This new approach, however, entirely circumvents the emotional aspects of patients' recall of adverse events and thus no longer operates with Freud's concept of traumatic memories. Instead, in this approach, self-reported early-life physical abuse is linked to patients' aberrant neural activity and connectivity patterns and then correlated with patients' expressions of genes known to play a role in "neuronal development, neurogenesis, and memory functions." Diez et al., "Endophenotypes," 3824. See also Spagnolo et al., "Gene Variation." This emerging approach examines how epigenetic modifications modulate patients' exposure to adverse early-life events, thus leading to the subsequent development of hysterical symptoms. However, such studies are beyond the scope of this book since my analysis here focuses on fMRI research into hysteria within the first two decades of the twenty-first century.

approach to investigating hysteria patients' emotional states.[439] The latter studies rely on the action-guiding concept of emotion processing they adopted from affective neuroscience.[440] In affective neuroscience, emotional states are understood "as products of distinct but interacting psychological processes" that are "implemented in the human brain" through the activation of designated neuroanatomical structures.[441] Simply put, the assumption informing the concept of emotion processing is that comparable emotional states across different individuals are underpinned by shared neural mechanisms. This assumption has enabled hysteria researchers to circumvent patients' idiosyncratic traumatic experiences and pose a more general question. How does the brain of a hysteria patient differently process emotionally salient stimuli compared to the brain of a healthy subject?

Significantly, this shift in focus has allowed hysteria researchers to use the existing sets of standardised visual stimuli that have been systematically developed and deployed in affective neuroscience to study emotion processing.[442] Such reliance on standardised pictorial material, which I will analyse shortly, has freed hysteria researchers from having to grapple with designing their stimuli, a process that, as discussed in the previous section, is fraught with difficulties. Moreover, there is an additional benefit to using the standardised visual stimuli. The same pictorial material can be applied to study a variety of hysterical symptoms, such as paralysis, tremors, pain, non-epileptic seizures, and contractures. Therefore, the use of standardised experimental stimuli facilitates "the comparison of results across different studies" and across heterogeneous hysterical symptoms.[443]

But as I will show in what follows, this new line of research has so far not managed to deliver any straightforward insights into the hysteria patients' presumably aberrant neural processing of emotions. Instead, the deployment of standardised emotional stimuli has brought multiple methodological challenges and resulted in fMRI studies

439 Aybek et al., "Emotion-Motion Interactions"; Blakemore et al., "Aversive Stimuli"; de Greck et al., "Emotional Empathy"; Espay et al., "Functional Dystonia"; Espay et al., "Functional Tremor"; Hassa et al., "Motor Control"; Lemche et al., "Somatization Severity"; Luo et al., "Pain Processing"; Morris et al., "Avoidance"; Noll-Hussong et al., "Affective Meaning Construction"; Noll-Hussong et al., "Sexual Abuse"; Sojka et al., "Processing of Emotions"; Stoeter et al., "Somatoform Pain"; Szaflarski et al., "Emotion Processing"; Voon et al., "Emotional Stimuli"; and Yoshino et al., "Neural Responses to Pain." Additional studies that have used fMRI to "determine the extent to which neuronal circuits associated with emotion processing change in response" to spontaneous recovery or targeted therapy will be analysed in section 4.4.2. Espay et al., "Neural Responses," e1788.

440 Affective neuroscience is an area of research that, since its emergence in the 1990s, has used "the concepts and methods of cognitive neuroscience" to study the neural basis of emotions. Sander, "Models of Emotions," 6.

441 Barrett and Wager, "Structure of Emotion," 83.

442 Emotion research, in general, has deployed various stimulus modalities, such as schematic drawings, photographs, movie clips, sounds and words. For an overview of these different types of standardised stimuli, see Okon-Singer, Lichtenstein-Vidne, and Cohen, "Dynamic Modulation," 482–83; and Goeleven et al., "Emotional Faces," 1094–95. Except for a single study (see Morris et al., "Avoidance"), fMRI research on hysteria has so far only made use of standardised photographic stimuli.

443 Lang, Bradley, and Cuthbert, *IAPS*, Introduction.

that have yielded mutually inconsistent imaging results. Many of these challenges, I will argue, are closely linked to often inconsistent assumptions about the nature of emotions that are implicitly built into the visual stimuli through their standardisation. Problematically, the epistemic import of such implicit assumptions on hysteria research has, until this point, been neglected. Hence, before proceeding to the analysis of individual fMRI studies that deployed the concept of emotion processing to investigate hysteria, we must first unpack the implications entailed in the standardised visual stimuli these studies have used.

Various standardised laboratory stimuli were developed in affective neuroscience to facilitate a targeted induction of purportedly unambiguous and reproducible emotional states of interest in experimental subjects.[444] Yet the caveat is that the standardisation of diverse stimulus sets was grounded in vastly different assumptions about the nature of emotions. The reason for such discrepancies is the lack of consensus among experts on what constitutes "the basic building blocks of emotional life that a science of emotions should focus on."[445] Thus, affective neuroscience operates with diverse, often mutually incompatible models of emotions.[446] Notwithstanding the individual differences, these models can be broadly divided into the basic emotion, dimensional, and appraisal approaches.[447] Since the first two of these approaches have substantially influenced fMRI hysteria research, in what follows, we will take a closer look at them.

Proponents of the basic emotion approach, whose foremost representative is the American psychologist Paul Ekman, have postulated the existence of a set of discrete emotional categories. Such discrete categories are viewed as "more elementary" than other emotions.[448] This model's six basic emotions include sadness, fear, happiness, anger, disgust, and surprise.[449] According to Ekman, each of these six separate categories constitutes a distinct, innate, and reflex-like emotional response. Ekman has also argued that each of these reflex-like emotional responses has been shaped by evolution and must, therefore, be associated with a unique pattern of brain activity.[450] Ekman's views have proven highly influential in affective neuroscience, initiating an intense search "for discrete dedicated brain systems underlying each and every basic emotion."[451] So far, however, this search has failed to deliver unambiguous results.[452] Even more influentially, Ekman has postulated that each basic emotion is associated with a distinctive and prototypical facial expression, which is universally recognisable across cultures. Ekman has explicitly acknowledged his conceptual debt to Darwin's

444 Lang, Bradley, and Cuthbert, Introduction.

445 Barrett and Wager, "Structure of Emotion," 79.

446 For a pertinent overview, see Sander, "Models of Emotions."

447 Sander, 16.

448 Sander, 9.

449 For alternative basic emotion models, see Izard, *Emotions*; Panksepp, *Affective Neuroscience*; and Plutchik, *Emotion*.

450 Ekman, "Argument for Basic Emotions," 170, 182–83.

451 Sander, "Models of Emotions," 10.

452 Barrett and Wager, "Structure of Emotion," 81. Over the years, Ekman's approach has been severely criticised. For an incisive criticism delivered from the humanities-based perspective, see Leys, *Ascent of Affect*.

research into the universality of emotional expressions in humans and animals and to Duchenne's photographic studies into the mechanism of facial movements that display emotions.[453]

Unsurprisingly, standardised visual stimuli derived from the basic emotion approach comprise photographs of facial expressions. In addition to photographs of facial expressions of the six elementary emotional categories, the standardised sets also include a baseline non-emotional condition referred to as the 'neutral face.' "The inclusion of the neutral expression is important since neutral is often a comparison condition, particularly in neuroimaging studies."[454] Moreover, in a striking parallel to Duchenne's approach, the facial stimuli used in current emotion research are not photographs of spontaneous emotional expressions. Instead, either professional or amateur actors of both genders and, in more recent sets, from diverse ethnic backgrounds were instructed to emulate the facial expressions of different basic emotions.[455] In each set, all subjects were photographed under identical conditions. These included a uniform diffuse light, a neutral background, the same distance to the camera and close cropping of the face.

The resulting images then underwent the process of validation, during which either experts or untrained volunteers rated the recognisability of the emotional facial expressions.[456] Thus, the axiomatic assumption that automatically recognisable facial expressions are intrinsically linked to distinct categories of emotions is implicitly encoded in all standardised photographic sets of emotional faces, both during their production and validation. Based on this assumption, such stimuli are widely used in affective neuroscience—and also in fMRI hysteria research—to study "the neuropsychological mechanisms of emotional facial expression perception."[457]

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453 See Ekman, "Argument for Basic Emotions," 176–79; and Ekman, "Duchenne and Facial Expression." The extent of Duchenne's influence is perhaps best illustrated by the fact that in 1978, Ekman collaborated with Friesen to develop the so-called Facial Actions Coding System (FACS). In this manual, Ekman and Friesen codified all anatomically possible facial expressions based on different combinations of contractions and relaxations of individual muscles. See Ekman and Friesen, *Facial Action Coding System*. Similarly to Duchenne, Ekman and Friesen also extensively relied on photography. For a discussion of Duchenne's photographic studies of facial expressions of emotions, see section 1.2.1.

454 Tottenham et al., "NimStim," 243.

455 "The most important and frequently used facial picture sets were developed by Ekman and colleagues. The set produced by Ekman and Friesen (1976) includes 60 black and white pictures of faces" of ten Caucasian subjects. Goeleven et al., "Emotional Faces," 1095. But the need for a larger number of stimuli and the emergence of studies showing that "the race or ethnicity of a model impacts face processing" have led to the development of alternative standardised sets. Tottenham et al., "NimStim," 242. These sets comprise colour photographs "of models from various backgrounds" and include the JACFEE [Japanese and Caucasian Facial Expressions of Emotion] by Ekman and Matsumoto, the Montreal Set of Facial Displays of Emotion, and the NimStim. Tottenham et al., 243. The sets so far used in fMRI research on hysteria are the Ekman's and Friesen's (see Aybek et al., "Emotion-Motion Interactions"), the JACFEE (see de Greck et al., "Emotional Empathy"), the Karolinska Directed Emotional Faces (see Voon et al., "Emotional Stimuli"), and the NimStim (see Espay et al., "Functional Tremor").

456 See, e.g., Tottenham et al., "NimStim," 243; and Goeleven et al., "Emotional Faces."

457 George, "Facial Expressions," 174.

However, when using such stimuli for research purposes, it is routinely disregarded that "although bearing some universality, the facial expressions of (even basic) emotions show both interindividual variability and context dependency, and their display is contingent on cultural codes."[458]

In contrast to the supporters of the basic emotion models, the proponents of the dimensional approach dispute the existence of distinct categories of hardwired emotions. Instead, drawing on Wilhelm Wundt's theories of affect, they argue that, far from being automatic responses, different emotions, such as anger or sadness, are complex constructs the brain builds up "from more fundamental, biological properties."[459] These mutually independent fundamental properties are referred to as dimensions. They include valence (i.e., the degree of pleasantness) and arousal (i.e., the degree of activation).

In a prominent dimensional model developed by James Russell, a dynamic, continually changing combination of valence and arousal is called the core affect.[460] Defined as a neurophysiological state "that sums up the individual's relationship to the environment at a given point in time,"[461] such core affect is subjectively experienced "as simply feeling good or bad, energized or enervated."[462] Thus, in this model, affective feelings are conscious subjective experiences of the core affect's dimensions, "an assessment of one's current condition." [463] Moreover, Russell has introduced an operational distinction between emotions and affects. He has designated emotions as affective experiences of limited duration directed at an intentional object—i.e., a specific event that elicited them.[464] Conversely, a core affect "can be experienced in relation to no known stimulus—in a free-floating form."[465] As pertinently summarised by psychologist and neuroscientist Lisa Feldmann Barrett, "[a]ffective feelings of pleasure and displeasure, with some level of arousal, are ever present and always changing. Only sometimes are these changes perceived as being causally related to surrounding events, and when this happens, an emotion is constructed."[466]

In effect, the defining characteristic of emotions in the dimensional approach is the individuals' subjective experiences of how the current situation impacts them. Hence, according to this approach, discrete emotional events—and their related facial expressions—are not reducible to a small number of innate categories. Rather,

458 George, 173.

459 Barrett and Wager, "Structure of Emotion," 79. For a succinct overview of Wundt's theory of affect, see Wundt, *Grundzüge*, 2: 327–45.

460 "At any point in time, core affect is a blend of pleasure and activation. The two components combine in an integral fashion, so that, subjectively, a person has one feeling rather than, for example, unpleasant and, separately, deactivated." Rusell and Barrett, "Core Affect," 809. By contrast, other dimensional models of emotions have postulated the existence of additional dimensions such as potency, dominance, approach, and withdrawal. Compare, e.g., Lang, Bradley, and Cuthbert, "Emotion, Attention"; and Rusell and Barrett, "Core Affect," 812.

461 Duncan and Barrett, "Affect," 1186.

462 Russell, "Core Affect," 145.

463 Russell, 148.

464 Russell, 147.

465 Russell, 148.

466 Barrett, "Three Principles," 383.

emotions vary continuously along the mutually independent dimensions (i.e., affective feelings) of valence and arousal, which can be combined in countless possible ways.[467] Consequently, in reference to the dimensional approach, another type of standardised visual stimuli for emotion elicitation was developed. Instead of focusing exclusively on facial expressions, such sets of so-called affective pictures comprise hundreds of colour photographs covering a broad range of topics. The topics vary from pleasant over mundane to highly threatening or upsetting real-life objects and scenes. These include smiling children, snakes, landscapes, mutilated bodies, cars, natural disasters, baby animals, acts of violence, food, and illness.[468] Due to their highly heterogeneous contents, the standardised affective pictures, unlike the facial emotional stimuli, lack uniformity at the level of formal visual features, such as composition, colour or contrast. The first and most widely used set of this kind is the International Affective Picture System (IAPS), developed in 1997 and regularly updated ever since.[469]

In the IAPS, all images underwent the process of standardisation, during which volunteers evaluated their emotional impact along the dimensions of valence and arousal.[470] After that, each image was classified according to the average ratings thus obtained. Hence, the dimensional view of emotions has been explicitly encoded into the affective pictures during their standardisation and decidedly informs their use as experimental stimuli.[471] Based on the accompanying normative numerical values, in particular concerning the valence, researchers decide which images from the IAPS to use for their study. In fact, the focus on the rated valence—i.e., the level of pleasantness or unpleasantness—is so pronounced that, when choosing the IAPS stimuli, researchers often disregard the particular visual content of individual affective pictures.[472] This means that, unlike the facial expression stimuli, the IAPS pictures are not meant to induce any categorical emotions. Instead, they have been codified to elicit more general affective responses that range from displeasing over neutral to pleasing. Admittedly, in Russell's and Barrett's view, when exposed to such pictures, experimental subjects nevertheless subjectively experience the affective feelings thus induced as particular

467 See George, "Facial Expressions," 174; and Sander, "Models of Emotions," 32–34.

468 See Lang, Bradley, and Cuthbert, *IAPS*; Kursi, Lozano, and Banaji, "OASIS," 457–58; Dan-Glauser and Scherer, "GAPED," 471–72; Marchewka et al., "Nencki"; and Wessa et al., "EmoPics."

469 See Lang, Bradley, and Cuthbert, *IAPS*.

470 A third dimension called 'dominance' or 'control' was also measured, although it proved to account for "relatively little unique variance in picture perception." Lang, Bradley, and Cuthbert, Introduction. To quantify valence, arousal, and dominance, the authors of the IAPS developed a rating instrument called SAM (The Self-Assessment Manikin). SAM consists of "a graphic figure depicting values along each of the 3 dimensions on a continuously varying scale." Ibid. For instance, "SAM ranges from a smiling, happy figure to a frowning, unhappy figure when representing the valence dimension." Ibid.

471 Significantly, the IAPS has been developed as a stimulus set for international use. Nevertheless, its cross-cultural validity remains an open question because its normative ratings were standardised on the sample of American college students. Studies that have tested how the IAPS images are rated in diverse cultural contexts have found significant similarities but also multiple cross-cultural differences. For an overview, see Mačiukaitė, Kuzinas, and Rukšėnas, "Universality," 113. See also Okon-Singer et al., "Violence."

472 See, e.g., Blakemore et al., "Aversive Stimuli," 231.

emotions. Yet, in an experimental setup, what matters is not a particular emotion a subject may experience while viewing an IAPS image. What matters is only the more general positive or negative affective valence that underpins the resulting emotional state.

Both the standardised photographs of the facial expressions of basic emotions and the IAPS affective picture stimuli have been deployed in fMRI hysteria research. But the disparate assumptions about the nature of emotions that, as analysed above, had shaped the standardisation of these different types of pictorial material were not explicitly addressed in individual studies of hysterical symptoms. Moreover, hysteria researchers offered either none or only a very vague explanation as to why they chose to use one or the other type of stimuli in their experiment.[473] Such choices, however, are not epistemically neutral. Preliminary research on healthy subjects has found that these two types of emotional stimuli elicit different patterns of brain activity, whose meaning at the cognitive level remains far from clear.[474] Most of the fMRI studies of hysteria published in the 2010s relied on the standardised images of the facial expressions, whereas the IAPS stimuli were only implemented in a few more recent studies.[475] Thus during this period, the search for hysteria patients' potential deficits in emotion processing has focused primarily on isolating aberrant neural patterns associated with discrete categorical emotional responses that were, at least in principle, meant to be reliably induced using the facial expressions stimuli. Yet, as I will show in what follows, fMRI studies of hysteria that relied on the categorical approach to emotions have yielded mutually conflicting imaging results. To unpack the potential reasons behind such inconsistencies, we will now turn to analysing these studies.

What is particularly interesting about the segment of fMRI hysteria research informed by the basic emotion approach is that multiple studies used the same experimental task, known as the implicit emotional task. In this task, study participants were shown photographs of different individuals with standardised expressions of various basic emotions and asked to identify the gender of each face they saw as quickly as possible.[476] Two aspects of the implicit emotional task are significant. First, researchers refrained from explicitly mentioning the emotional content of the stimuli

473 See, e.g., Aybek et al., "Emotion-Motion Interactions," 2, e0123273.

474 See Britton et al., "Common and Differential Networks."

475 For studies that used the IAPS stimuli, see Blakemore et al., "Aversive Stimuli"; Luo et al., "Pain Processing"; Morris et al., "Avoidance"; and Sojka et al., "Processing of Emotions." Two studies deployed both the facial expressions and IAPS stimuli. See Espay et al., "Functional Dystonia"; and Espay et al., "Functional Tremor." But in the studies by Espay et al., the two types of stimuli were used in parallel tasks and delivered mutually disjunctive results, which the authors failed to bring in relation to one other.

476 This task is not specific to hysteria research and "has been extensively investigated in healthy volunteers and patients with psychiatric disorders." Voon et al., "Emotional Stimuli," 1528. For fMRI studies of hysteria that used this task, see Aybek et al., "Emotion-Motion Interactions"; Espay et al., "Functional Dystonia"; Espay et al., "Functional Tremor"; Hassa et al., "Motor Control"; Lemche et al., "Somatization Severity"; Szaflarski et al., "Emotion Processing"; and Voon et al., "Emotional Stimuli." One exception was a study in which the facial expressions of basic emotions were embedded in a task that required the subjects to recognise and then to try to experience "the emotional state of the shown person." See de Greck et al., "Emotional Empathy," 2669.

in the task's instructions. Second, the task itself was specifically designed to focus the experimental subjects' attention on a non-emotional feature of the faces depicted, such as gender. The aim was to use the task-irrelevant emotional features of the stimuli—i.e., the standardised facial expressions of basic emotions—to induce a response called implicit emotion processing in the viewing subject. The task's underlying assumption is that the viewing subject registers and processes the task-irrelevant emotional content of the presented facial expressions in an automatic, involuntary manner. Put simply, the emotional responses to the facial expressions shown in the stimuli are thought to occur independently of the subject's intentions and without being tied to conscious processing.[477] Whether or not this assumption is actually valid is still a matter of heated debate in affective neuroscience.[478]

The fact that the authors of the studies using the implicit emotional task were interested only in the subjects' non-conscious, purportedly automatic reactions to the facial expressions of basic emotions was underscored by the kind of behavioural data they collected. In most such studies, the researchers measured the subjects' reaction times and their accuracy in identifying the gender of the faces. These measurements served as indirect indicators of implicit emotion processing.[479] Only one study carried out a post-scan assessment to test if the experimental subjects were actually able to correctly identify the standardised facial expressions of emotions when explicitly asked to do so.[480] Yet, in none of the studies were the subjects at any point asked to provide a subjective assessment of their emotional responses to the facial stimuli. Interestingly, this focus on studying emotions from the perspective of purportedly automatic neural processing while entirely circumventing the patients' subjective experience of the artificially induced emotional states is curiously reminiscent of Charcot's hypnotic experiments.[481] However, whereas Charcot physically imprinted various emotional expressions onto the patients' facial muscles, contemporary researchers merely expose their subjects to standardised images of such expressions. Nevertheless, both interventions aimed to induce categorical and purportedly automatic emotional reactions over which the hysteria patient is thought to have no voluntary control. Moreover, both interventions have their roots, either directly (in Charcot's case) or

477 Pessoa, Oliveira, and Pereira, "Top-Down Attention," 357.

478 For discussions about the automaticity of emotion processing, see, e.g., Okon-Singer, Tzelgov, and Henik, "Automaticity and Attention"; Okon-Singer, Lichtenstein-Vidne, and Cohen, "Dynamic Modulation"; Pessoa, Oliveira, and Pereira, "Top-Down Attention"; and Pessoa et al., "Neural Processing."

479 See, e.g., Voon et al., "Emotional Stimuli," 1530. Moreover, in one study, the researchers also measured relative skin conductance level as a physiological indicator of automatic emotional arousal. See Lemche et al., "Somatization Severity," 1, article 1032.

480 Szaflarski et al., "Emotion Processing," 195.

481 As discussed earlier, by referencing Duchenne's photographic studies of facial and gestural expressions of emotions, Charcot used electricity to artificially imprint chosen expressions onto the faces of his hypnotised hysteria patients. Their bodies then spontaneously reacted by producing related emotional gestures. Charcot argued that the thus induced gestures were involuntary and unconscious. He viewed them as decisive proof that the hypnotised patients' emotional responses were produced through the automatic action of the brain. For details, see section 1.2.2.

indirectly (in the present-day studies via Ekman), in Darwin's theories and Duchenne's photographic studies of the facial expressions of emotions.

But despite their use of the standardised visual stimuli and the shared focus on implicit emotion processing in hysteria patients, multiple fMRI studies obtained highly divergent brain activation patterns. In the earliest of these studies, Voon et al. compared the neural responses induced by the gender-identification task in sixteen patients with mixed positive motor symptoms (tremors, contractures, and gait abnormalities) and sixteen healthy subjects.[482] While lying inside the MRI scanner, the subjects were shown standardised images of fearful, happy, and neutral faces from the Karolinska Directed Emotional Faces set. After collecting the fMRI data, Voon et al. chose to focus their analysis on the amygdala, a bilateral set of subcortical nuclei that have "attracted a great deal of attention in the field of emotion study in general and of emotional face perception in particular."[483] The initial emotion research suggested that the amygdala's role was limited to processing negative emotional responses, particularly fear.[484] But subsequent studies have instituted "the view that the amygdala is involved with computing the affective significance of a stimulus" or, in other words, "the extent to which the stimulus predicts an impending threat or reward."[485] Thus, in line with more recent findings, instead of being linked only to the processing of fear, the amygdala is currently regarded to have a broader relevance as "a key structure for the appraisal of events that are relevant to the organism."[486]

Searching for the potential role of the amygdala in hysteria patients' motor symptoms, Voon et al. first computed the neural responses to happy versus neutral and fearful versus neutral faces in both groups of subjects separately. Interestingly, both contrasts in each group were "associated with an increase of amygdala activity."[487] Thus, at this stage, Voon et al. found no differences between patients and healthy controls. It was only by directly computing how these two contrasts (happy versus neutral and fearful versus neutral faces) changed across the groups that the researchers managed to discover a differential pattern of amygdala activity between patients and controls.[488] In the latter analysis, the healthy participants showed increased response in the right amygdala to the fearful compared to the happy condition. This increased response was taken to reflect the right amygdala's "crucial role in determining biologically salient or threatening stimuli in the environment."[489] The patients, however, showed an abnormal pattern of activation. Specifically, they lacked the expected asymmetrical response in the right amygdala to fearful relative to happy faces.

Zooming further in on the amygdala's activity, Voon et al. performed additional fMRI analyses. The map derived from the functional connectivity analysis identified

482 Voon et al., "Emotional Stimuli," 1528.

483 George, "Facial Expressions," 178.

484 George, 178.

485 Barrett and Wager, "Structure of Emotion," 81.

486 George, "Facial Expressions," 179.

487 Voon et al., "Emotional Stimuli," 1530.

488 In other words, Voon et al. deployed the factorial design (see section 3.1.2) and, at this point, calculated the neural effects induced through the interaction of their factors. See Voon et al., 1530.

489 Voon et al., 1533.

a statistically significant increase in the interaction between the amygdala and the supplementary motor area (SMA) in response to happy faces in patients, but not in controls.[490] Moreover, the analysis of the BOLD signals' time courses disclosed that the intensity of the amygdala's responses to all emotional stimuli in healthy subjects gradually decreased over time. According to Voon et al., this decrease in the signal's intensity demonstrated a normal pattern of the amygdala's habituation to emotionally salient stimuli.[491] In patients, however, the amygdala failed to habituate to the repeated exposure to happy faces, whereas a similar trend for fearful faces did not reach the level of statistical significance.[492] The discovery that the right amygdala in the patient sample appeared to overrespond to happy faces was surprising. Unable to explain this particular anomaly on its own, Voon et al. suggested instead that, on the whole, their imaging findings could be attributed to "a general effect of arousal."[493] They conjectured that hysteria patients' aberrant emotion processing was twofold. It entailed not just the amygdala's excessive responsiveness but also its impaired habituation to emotional stimuli in general. Voon et al. also argued that, during the resulting state of emotional arousal, the amygdala exhibited increased downstream influence on the preparatory motor regions (the SMA), thus possibly leading to either "the onset or exacerbation" of hysterical motor symptoms.[494]

In a study published in 2015, Aybek et al. deployed the same type of task but compared the implicit emotion processing between twelve patients with hysterical paralysis and a group of healthy volunteers.[495] As their emotional stimuli, they used standardised images of fearful, sad, and neutral faces from Ekman's set. Notably, whereas Voon et al. failed to find group-specific differences by contrasting the responses to fearful versus neutral faces between patients and controls, Aybek et al. identified several. Compared to controls, patients in the Aybek et al. study showed increased activity in the left (but not the right) amygdala to both fearful versus neutral and sad versus neutral faces in separately computed fMRI maps.[496] In another opposition to Voon et al., Aybek et al. found that the amygdala's lack of habituation in patients was highly specific to fearful stimuli and did not extend to sad faces.[497] Additionally, in patients but not in controls, the hyperactivation of the amygdala in response to fearful faces was accompanied by significantly increased activity of the brain regions involved in motor planning. These included the PAG (the periaqueductal grey matter) and the SMA (the supplementary motor area).

Based on "robust evidence from animal models," which suggested that the PAG is "a key region in the 'freeze response' to threat," and the patients' apparent inability

490 Voon et al., 1530–31.

491 Voon et al., 1533.

492 Voon et al., 1533.

493 Voon et al., 1533. It appears to me that a possible alternative interpretation, which Voon et al. ignored, is that the patients' amygdalae did not overreact to happy faces but instead had a blunted response to fearful ones.

494 Voon et al., 1535.

495 Aybek et al., "Emotion-Motion Interactions."

496 Aybek et al., 7–8, e0123273.

497 Aybek et al., 8, e0123273. Compare Voon et al., "Emotional Stimuli,"1530.

to habituate to fearful faces, Aybek et al. came to the following conclusion.[498] They postulated that the dysfunction of emotion processing in hysteria patients consisted in abnormal hypersensitivity only to stimuli perceived as threatening. It is worth noting that the hypothesis Aybek et al. postulated directly contradicted the finding of the Voon et al. study. Admittedly, both studies stated that the hysteria patients' underlying disturbance comprised the hypersensitivity to emotional stimuli. However, Voon et al. postulated that patients were generally overresponsive to all types of emotional events, both positive and negative. In contrast, Aybek et al. claimed that the patients' aberrant processing was limited exclusively to fear-inducing stimuli.

Subsequent fMRI studies that used the same type of implicit emotional task to examine aberrant emotion processing underlying different hysterical symptoms complicated the picture even further. For example, Lemche et al. measured neural responses to sad, happy, and neutral facial expressions in patients with multiple concurrent somatic symptoms.[499] But unlike the studies analysed above, Lemche et al. did not compare responses between hysteria patients and healthy subjects. In fact, they did not even recruit any healthy control subjects. Instead, they computed fMRI maps that identified brain regions in which the magnitude of the task-induced activity correlated with the self-reported severity of the patients' symptoms. The resulting map showed that the anatomical region called precuneus was activated by both happy and sad facial stimuli.[500] Employing reverse inference, Lemche et al. suggested that, since the precuneus is thought to mediate "self-referential functioning," "autobiographic memory," and "sensorimotor control," the aberrant emotion processing in hysteria patients entailed mental rumination and dysfunctional cognitive filtering of bodily sensations.[501] Although the findings by Lemche et al. implicated a different brain region than the Voon et al. study, there was nevertheless one point in common. The authors of both studies argued that hysteria patients had aberrant neural processing of positive as well as negative emotional stimuli.

Moreover, in three separate studies—one conducted by Szaflarski et al. and the other two by Espay et al.—researchers used the gender-identification task with happy, sad, fearful, and neutral faces to examine aberrant emotion processing in non-epileptic seizures, hysterical tremor, and functional dystonia, respectively.[502] There was one significant methodological novelty—in each of these studies, hysteria patients were not only compared to healthy control subjects. Instead, hysteria patients were additionally compared to patients with clinically similar symptoms that had a detectable somatic

498 Aybek et al., "Emotion-Motion Interactions," 8, e0123273.

499 Lemche et al., "Somatization Severity." The official designation for this multisymptomatic form of hysteria when the study was conducted was somatisation. In the meantime, this term has been displaced in the *DSM-5* by the somatic symptom disorder. See section 2.4.2 for details.

500 Lemche et al., "Somatization Severity," 3, article 1032.

501 Lemche et al., 3, article 1032.

502 See Espay et al., "Functional Dystonia"; Espay et al., "Functional Tremor"; and Szaflarski et al., "Emotion Processing." Functional dystonia refers to "excessive posturing or twisting" of a limb and thus denotes a set of symptoms Charcot called contractures. Espay et al., "Functional Dystonia," 136. In all three studies, the researchers used standardised photographs of facial expressions from the NimStim set.

aetiology. The patients with non-hysterical symptoms in these three studies were diagnosed with epilepsy, essential tremor, and primary organic dystonia, respectively. Thus, in the Szaflarski et al. study, the researchers calculated different fMRI activation maps by comparing patients with non-epileptic seizures to healthy controls and then to epilepsy patients.[503] The comparisons were computed separately for each category of emotional faces.

Notably, Szaflarski et al. introduced an additional methodological twist. In the studies analysed so far in this section, the standardised neutral facial expression was consistently used as a baseline condition. Simply put, the neutral expression served as a purportedly non-emotional stimulus in relation to which the neural responses to the emotional content of all other facial expressions (happy, fearful, or sad) were determined through subtraction.[504] Hence, in the Voon et al., Aybek et al., and Lemche et al. studies, the effect of the neutral facial expression was not of interest in its own right. By contrast, in the Szaflarski et al. study, the absence of facial stimuli served as a baseline, whereas the neutral facial stimuli were treated as a condition of interest on an equal footing with the expressions of happiness, fear, and sadness.[505]

The fMRI maps Szaflarski et al. calculated for each facial expression displayed patterns of activations that extended across multiple and functionally diverse brain regions.[506] The anatomical distributions of these patterns differed among the three subject groups and across the distinct emotional categories. Yet, interestingly, none of the aberrant patterns included the amygdala, the region that had been implicated in the Voon et al. and Aybek et al. studies. Summarising their activation maps, Szaflarski et al. emphasised that hysteria patients, as opposed to those with epilepsy, "exhibited increased fMRI response to happy, neutral, and fearful faces in visual, temporal, and/or parietal regions and decreased fMRI response to sad faces in the putamen bilaterally."[507] But the interpretation the researchers posited for these differential patterns of responses was cryptic and circular. Szaflarski et al. merely stated that, apart from the putamen, which had a role in motor control, the other regions had "been previously described to be involved in emotion processing."[508] The researchers stated neither which specific aspects of emotion processing were disturbed in patients with non-epileptic seizures nor how.

Next, Szaflarski et al. computed additional connectivity fMRI maps to explore the mutual interactions among the aberrantly activated brain regions. These maps showed that, only in hysteria patients, each of the differentially activated areas also displayed stronger neural interactions with multiple other brain regions.[509] Based on these findings, Szaflarski et al. claimed that they had identified the neural circuitry involved in the distinctly different emotion processing in hysteria patients as opposed

503 Szaflarski et al., "Emotion Processing," 193.

504 See, e.g., Voon et al., "Emotional Stimuli," 1529.

505 Szaflarski et al., "Emotion Processing," 196. During the baseline condition, the subjects viewed "a screen with a '+' [i.e., a fixation cross] in the center." Ibid.

506 See Szaflarski et al., 197, table 2.

507 Szaflarski et al., 193.

508 Szaflarski et al., 199.

509 For detail, see Szaflarski et al., 201–2.

to both healthy controls and epilepsy patients.[510] Yet Szaflarski et al. remained curiously tacit about what such differences actually meant in cognitive terms. Apparently, they were unable to interpret the aberrant patterns displayed by their fMRI maps in terms of any clear-cut neurocognitive mechanisms.

Despite such limitations, a methodologically innovative aspect of the Szaflarski et al. study should be highlighted. This is the only fMRI study to date that explicitly tested hysteria patients' ability to recognise the emotional content of the standardised facial stimuli. Immediately after the scanning, the subjects were asked to identify the stimuli to which they had been exposed in the scanner. While viewing the stimuli, the subjects could choose among the following labels: happy, fearful, sad, neutral, and unknown. Guided by these constrained choices, all three subject groups showed a similarly high degree of accuracy in identifying each emotional expression.[511] The Szaflarski et al. study thus delivered empirical evidence that the hysteria patients in their sample could explicitly identify the emotional facial expressions shown in the stimuli.

Finally, Espay et al. applied the same basic emotion task as Szaflarski et al. first to hysteria patients with contractures and then—in a separate study—to hysteria patients with tremor.[512] Surprisingly, however, in each of these two studies, Espay et al. reported fMRI responses for very different comparisons of emotional faces. As I see it, this inconsistency suggests that during their data analyses, Espay et al. tested a variety of possible contrasts, including those that did not have any clear cognitive meaning. It is conceivable that they deployed this problematic strategy to search for any contrast that would reveal differential neural responses to emotional stimuli between the different patient groups and healthy subjects.

For example, the first Espay et al. study involved patients with both hysterical and organic contractures, as well as healthy controls. In this study, "differences at the group level were examined for emotional faces (happy, sad, fearful) versus neutral faces, fearful faces versus neutral faces, and all faces (happy, sad, fearful, neutral) versus a fixation cross."[513] Only the last contrast (i.e., all faces versus the fixation cross) enabled the researchers to identify an altered activations pattern in hysteria patients relative to the other two participant groups.[514] Yet, since the researchers did not perform any correction for multiple comparisons,[515] it remains questionable how much of this

510 Szaflarski et al., 202.

511 Szaflarski et al., 194.

512 Espay et al., "Functional Dystonia"; and Espay et al., "Functional Tremor." Apart from the basic emotion task, both Espay et al. studies contained two additional tasks. These were, first, the finger-tapping motor task; and second, a so-called 'intense-emotion' task. The intense-emotion task used "a series of offensive or disgusting images" from the IAPS to induce implicit emotion processing. Espay et al., "Functional Tremor," 180. All three tasks were analysed separately, and the authors failed to provide an overarching interpretation that would have integrated the disparate results. For this reason, I will only focus on discussing the basic emotion tasks in these two studies.

513 Espay et al., "Functional Dystonia," 139.

514 To be more exact, hysteria patients "showed areas of decreased activation in the right middle temporal gyri and bilateral precuneus and increased activation in the right inferior frontal gyrus, bilateral occipital cortex and fusiform gyrus, and bilateral cerebellar hemispheres." Espay et al., 139.

515 Espay et al.,139.

pattern comprised false-positive activations. Based on reverse inference, Espay et al. concluded that hysteria patients showed aberrant activation "in networks involved in motor preparation and execution, spatial cognition, and attentional control."[516] However, in my opinion, it remained unclear what kind of cognitive processes the researchers intended to isolate through the contrast between all emotional faces (happy, sad, fearful, and neutral) versus the fixation cross. In effect, this poorly defined contrast merely conflated various categories of basic emotions together with a purportedly non-emotional (i.e., 'neutral') expression.

Conversely, in the second Espay et al. study, in which the researchers also apparently tested all possible contrasts, only the comparatively straightforward contrast between sad and neutral faces disclosed statistically significant results. The activation maps computed for this contrast showed regional differences between hysteria patients and healthy controls, as well as between hysteria patients and patients with organic tremor.[517] All other comparisons of neural responses to various facial expressions stimuli did not significantly differ across the participant groups. Interestingly, in this study, Espay et al. did perform an appropriate correction for multiple comparisons.[518]

But perhaps most surprisingly, although the aberrantly activated brain areas across the two Espay et al. studies did not overlap and were, as we have seen, derived from randomly chosen contrasts, in each case, the authors resorted to the same overarching interpretation. In both studies, Espay et al. suggested that the respective fMRI responses to the facial expressions stimuli represented "the neurobiological correlate of alexithymia, the inability to identify and describe emotions."[519] I suggest that this conclusion was purely speculative because the researchers neither explicitly evaluated the patients' purported alexithymia nor assessed the patients' ability to discriminate between the facial expression stimuli they had viewed. Instead, Espay et al. made this reverse inference based exclusively on the imaging results.[520] Also, this conclusion appears to contradict the incidental finding made by Szaflarski et al. that hysteria patients in their sample were able to accurately identify the emotional content of the standardised facial expressions stimuli.

To summarise my analysis so far, although the researchers deployed standardised visual stimuli and used the same type of implicit emotional task, no specific brain region was consistently activated across the six fMRI studies discussed above. In fact, the endeavour to identify the neural basis of hysteria patients' aberrant emotion

516 Espay et al., 136.

517 Specifically, hysteria patients "showed increased activation in the paracingulate gyrus and left Heschl's gyrus compared with HC [healthy controls] and decreased activation in two regions in right precentral gyrus when compared with" patients with organic tremor. Espay et al., "Functional Tremor," 182.

518 Espay et al., 182.

519 Espay et al.,185. See also Espay et al., "Functional Dystonia," 144.

520 See Espay et al., "Functional Tremor," 185. By contrast, in another fMRI study whose authors hypothesised that alexithymia "might be a factor potentially contributing to emotional dysregulation" in hysteria patients, this trait was explicitly evaluated. Sojka et al., "Processing of Emotions," 3, article 861. To measure the patients' alexithymia, Sojka et al. used the Toronto Alexithymia Scale. For details, see Sojka et al., 3, article 861.

processing of discrete and purportedly hardwired emotional categories such as fear, happiness, and sadness resulted in highly disparate activation and connectivity patterns spread throughout the entire brain. A possible explanation for such disparities could be provided by the assumption that the underlying disturbances in emotion processing vary across different hysterical symptoms. However, this assumption does not account for the disparities in the imaging results between the Voon et al. and Espay et al. studies,[521] both of which focused on patients with tremor. Nor can this assumption explain why the researchers, as detailed above, often struggled with finding unambiguous interpretations at the cognitive level for the isolated patterns of neural activity.

My analysis has also underscored that the researchers sometimes indiscriminately tested various contrasts, searching for statistically significant patterns of differential activations between hysteria patients and control subjects. For example, they compared happy to neutral but also happy to sad faces. Additionally, they also directly contrasted the combined reactions induced by all emotional faces, on the one hand, with the complete absence of facial stimuli, on the other. Sometimes they used the neutral face as a purportedly non-emotional baseline while, at other times, as an emotional condition of interest. Yet, it appears debatable what type of emotional response the neutral facial expression stimulus was meant to induce when used as an experimental condition in its own right. Due to such vaguely defined experimental contrasts, I contend that, in many cases, it remained unclear which particular aspect of emotion processing was meant to be isolated through various comparisons across emotional stimuli. Unsurprisingly, the result of such often arbitrary comparisons were fMRI maps whose meaning was ambiguous. Moreover, as I have shown, researchers occasionally posited rather speculative interpretations of the imaging results that relied exclusively on reverse inference without being grounded in behavioural data.

Therefore, I argue that the discrepancies in the imaging results analysed above were due to the following fact. By comparing the purportedly automatic neural responses between patients and control subjects to the posed facial expressions of the basic emotions, the researchers failed to isolate the aberrant emotion processing specific to hysteria. From the methodological perspective, it is conceivable that by exposing their subjects to sequences of decontextualised images of supposedly prototypical, pan-culturally recognisable emotional expressions, the researchers inadvertently induced a variety of confounding cognitive processes, which possibly varied across individual subjects. Such potential differences introduced uncontrollable ambiguity into the fMRI data. Even more problematically, none of the potential differences could be accounted for within the context of the basic emotions approach, which postulates a fixed, hardwired reaction to each standardised facial expression. The basic emotions approach thus a priori disregards the very possibility that subjects could attribute disparate meanings to the standardised facial expressions.

Based on my analysis above, I suggest that the epistemic adequacy of using the implicit emotional task with the standardised facial expressions to investigate emotion processing in hysteria is questionable. The main drawback of this approach, I think, is

521 See Voon et al., "Emotional Stimuli"; and Espay et al., "Functional Dystonia."

that it imposes a problematic and exceedingly rigid conceptual framework onto a group of patients whose multiple and highly heterogeneous symptoms might not necessarily be associated with a uniform or even fixed disturbance in emotion processing. Instead, it appears to me more likely that hysteria patients' potential disturbances in emotion processing are dynamic and context-dependent.

Importantly, I do not mean to imply that hysteria patients' potential disturbances in emotion processing are entirely beyond the reach of fMRI research. In my view, what appears epistemically more promising is an alternative approach to studying the deficits of emotion processing in hysteria that can be gleaned from two recent fMRI studies. The two studies I have in mind were authored by Blakemore et al. and Morris et al.[522] As we will see shortly, these two studies set out to answer two very different research questions by deploying mutually disparate experimental tasks. Despite such differences, the two studies had two important things in common. First, both studies were informed by the dimensional view of emotions I have introduced at the beginning of this section. Thus, instead of standardised photographs of facial expressions, Blakemore et al. and Morris et al. used affective images from the IAPS, which had been rated according to their valence (i.e., level of pleasantness) and arousal (i.e., perceived intensity).[523] This choice of stimuli already indicated that the authors' aim was not to induce distinct emotional categories in their experimental subjects but more general positive or negative affective states.

Second, and even more significantly, unlike the studies analysed so far in this section, Blakemore et al. and Morris et al. did not focus on hysteria patients' aberrant emotion processing in isolation. Rather, the researchers chose to examine how the abnormalities in emotion processing modify hysteria patients' goal-directed behaviour at the neural level. One of the studies focused on voluntary movement, whereas the other on the cognitive phenomenon called avoidance learning.[524] In what follows, I will show that to enable such investigations, the select emotional stimuli were neither attributed fixed, pre-established meanings nor shown in context-free sequences. Instead, in the Blakemore et al. and Morris et al. studies, emotional stimuli were embedded in sophisticated experimental tasks.

In a study published in 2016, Blakemore et al. set out to test whether a negative emotional context would affect the execution of voluntary movement in hysteria patients with mixed motor symptoms compared to healthy controls.[525] More specifically, the researchers wanted to determine if the patients' potentially defective processing of aversive stimuli directly interacted with the neural circuitry underpinning their motor symptoms. To this end, ten patients and ten healthy subjects were placed in the MRI scanner and asked to hold a force-measuring device in their hands. The subjects were instructed to pinch the device between their thumb and index finger to produce a sustained contraction at 10% of their maximum force. While maintaining this voluntary contraction, the subjects viewed the visual feedback on the screen, which indicated

522 Blakemore et al., "Aversive Stimuli"; and Morris et al., "Avoidance."

523 See Blakemore et al., "Aversive Stimuli," 231; and Morris et al., "Avoidance," 287.

524 See Blakemore et al., "Aversive Stimuli," 230; and Morris et al., "Avoidance," 286–87.

525 Blakemore et al., "Aversive Stimuli," 229.

the intensity of their force output. Occasionally, the visual feedback was displaced by either pleasant or unpleasant IAPS images. The subjects' force output was continually registered parallel to the fMRI data acquisition.[526] Additionally, in a post-scanning session, each participant was asked to subjectively rate the affective content of the IAPS images they had seen in the scanner. In both groups, the subjective assessment of valence and arousal was similar to the IASP's normative ratings.[527]

The analysis of the behavioural data showed that the healthy control subjects managed to maintain the target level of force only during visual feedback. By contrast, their force output gradually decayed while viewing both pleasant and unpleasant images, though in the latter case in a slightly attenuated form.[528] The patients' force output showed a comparable decay during the exposure to pleasant images. Yet, unexpectedly, the grip force remained at the target level not only during visual feedback but also while the patients viewed unpleasant images. This behavioural finding indicated a significantly "more pronounced influence of negative emotional signals on voluntary force control" in patients.[529] Based on this finding, Blakemore et al. conjectured that the patients' maintenance of force in the emotionally aversive condition represented an excessive defensive motor reaction "akin to freezing behaviour."[530] In other words, the behavioural data pointed to an abnormal interaction between the processing of negative emotions and the motor control in hysteria patients.

Crucially, the behavioural differences between the two groups were also reflected in the imaging results. The fMRI maps calculated for the contrast between the unpleasant and pleasant conditions showed differential activations unique to each group. Control subjects but not patients "engaged several prefrontal cortical areas, most notably the medial and inferior frontal gyrus."[531] As pointed out by Blakemore et al., these brain regions are known to be "involved in motor preparation and behavioural control."[532] In the patients, however, higher responses to unpleasant images were situated in the cerebellum, a structure "involved in regulating motor process in emotional (particularly fear-related) contexts."[533] The patients also showed greater activity during the unpleasant condition in parts of the limbic network (the hippocampus and the posterior cingulate cortex). The limbic areas are thought to be "critically implicated in the integration of emotion and memory."[534]

Combining their behavioural and imaging data, Blakemore et al. postulated a neurocognitive mechanism through which aberrant emotion processing in hysteria patients could modulate voluntary movement in an automatic, non-conscious way. In short, they suggested that the "presentation of unpleasant images could possibly engage associations stored in long-term memory," thus "tagging stimuli with threat-related

526 For details, see Blakemore et al., 230–31.
527 Blakemore et al., 233.
528 Blakemore et al., 233, 235.
529 Blakemore et al., 235.
530 Blakemore et al., 235.
531 Blakemore et al., 233.
532 Blakemore et al., 237.
533 Blakemore et al., 237.
534 Blakemore et al., 238.

information or personal relevance."[535] This aberrant threat-related tagging, in turn, led to an "abnormal translation of negative affective signals into dysfunctional motor commands and excessive freezing-like behaviour."[536] The authors also pointed out that, contrary to some previous studies, their findings did not support the hypothesis of hysteria patients' "physiological reactivity to both negative and positive emotions."[537] Instead, Blakemore et al. argued that only negative affective information could directly modulate voluntary movement in hysteria patients, thus leading to impaired motor function. Pertinently, Blakemore et al. emphasised that, although their study indicated "a prominent role of emotion" in hysteria, it nevertheless did not demonstrate its causal involvement in the symptom formation.[538]

Whereas Blakemore et al. probed functional links between emotion processing and motor control in hysteria, in an equally fine-grained study, Morris et al. investigated how a negative affective context impacts the patients' cognitive ability necessary "for the selection of appropriate behaviour and environmental adaptation."[539] More specifically, Morris et al. chose to examine the assumption that hysteria patients unconsciously develop their symptoms as a means of escaping stressful life events. Morris et al. argued that if this assumption holds, then hysteria patients should exhibit an enhanced behavioural tendency to avoid harm in general.[540] Hence, they decided to explore whether this was indeed the case and, if so, then how such purportedly enhanced harm avoidance was "expressed neurally."[541] With this purpose in mind, twenty-five patients with heterogeneous hysterical symptoms and twenty healthy volunteers underwent fMRI data acquisition while performing a so-called "aversion learning task."[542] This task was developed to test the participants' ability to learn to avoid adverse outcomes.

Interestingly, in the Morris et al. study, the participants were not exposed to images with explicit affective content during the fMRI data acquisition but only in the pre-scan conditioning phase. During the conditioning phase, both patients and healthy subjects viewed visual stimuli consisting of various abstract geometric shapes. While appearing on the screen, each abstract shape was paired either with an unpleasant or neutral IAPS image.[543] Through such conditioning, each abstract shape was meant to acquire the same emotional salience as the IAPS image with which it had been paired. During the subsequent task, which they performed inside the MRI scanner, the subjects were

535 Blakemore et al., 238.

536 Blakemore et al., 239.

537 Blakemore et al., 235. At this point, Blakemore et al. directly contradicted the hypothesis posited by Voon et al. about the general arousing effect of all emotions, which we have discussed earlier in this section. See Voon et al., "Emotional Stimuli," 1533.

538 Blakemore et al., "Aversive Stimuli," 239.

539 Morris et al., "Avoidance," 287.

540 Morris et al., 287.

541 Morris et al., 287.

542 Morris et al., 288.

543 See Morris et al., 287. Interestingly, this is the only fMRI study of aberrant emotion processing in hysteria in which the visual stimuli were additionally combined with sounds. During the conditioning phase, the unpleasant IAPS images were paired with "high pitched screaming and nails scratching a blackboard," whereas the neutral IAPS images were accompanied by "a neutral sound from a musical instrument." Ibid.

presented with and could choose between two abstract visual shapes. One of these shapes was previously conditioned, whereas the other represented a novel stimulus. Choosing either a neutrally or negatively conditioned stimulus was associated with a higher chance of a negative outcome, which entailed a symbolic monetary loss. By contrast, choosing a novel stimulus was more likely to result in no monetary loss. Each outcome was immediately communicated to the participant in the form of visual feedback.[544] In effect, while performing the task, the participants were expected to learn to associate both neutrally and negatively conditioned stimuli with punishment and to increasingly avoid choosing them. The fMRI and behavioural data were acquired both during the choice and the feedback phase of the task. The task consisted of 180 trials for each participant.

First, Morris et al. analysed the behavioural data for each subject group separately. This analysis showed that the control subjects learnt to avoid losses, thus exhibiting the goal-directed behaviour referred to as "increased harm avoidance."[545] The patients, however, exhibited disrupted avoidance learning "by persisting to choose the option that resulted in a negative outcome."[546] In fact, it was particularly in response to negatively conditioned visual stimuli that the patients displayed a "trend towards impaired learning and greater noise or randomness of choice behaviour."[547] Additionally, the fMRI maps disclosed increased amygdala activity in patients relative to controls in response to receiving the feedback of negative outcomes.[548] Morris et al. conjectured that the amygdala's abnormally heightened sensitivity to adverse environmental cues "can impair goal-directed decision making" and thus interfere with hysteria patients' ability to learn to avoid harm.[549] Taken together, both the behavioural and imaging results of the Morris et al. study have empirically challenged the assumption that the symptom formation in hysteria patients "has a purpose and is used to solve a problem."[550] In doing so, the Morris et al. study directly contradicted the very assumption that provided the axiomatic starting point of the Aybek et al. study we discussed in the previous section.

Thus, using standardised affective visual stimuli, both Morris et al. and Blakemore et al. succeeded in generating surprising, though still preliminary new empirical insights into potential neural disturbances of emotion processing specific to hysteria. However, in my opinion, what is even more significant about these two studies is not limited to their particular empirical findings. At a more general level, these two studies have developed a new, more dynamic approach to examining hysteria patients' potential disturbances in emotion processing. In doing so, they have moved away from trying to shoehorn the investigation of hysteria patients' emotion processing into the rigid and predefined categories of the basic emotions. Instead, Morris et al. and Blakemore

544 An image of a crossed-out coin signified monetary loss, whereas a grey square represented a 'neutral' no-loss outcome. Morris et al., 288.

545 Morris et al., 286.

546 Morris et al., 286.

547 Morris et al., 290.

548 Morris et al., 289.

549 Morris et al., 293.

550 Morris et al., 290.

et al. have deployed visual stimuli based on their broader affective relevance—i.e., pleasantness or aversiveness.

The purpose of the IAPS images was to enable a targeted creation of either positively or negatively charged affective situational contexts within which the study participants performed specially tailored experimental tasks. The tasks were devised to engage either a chosen hysteria-specific deficit (i.e., impaired motor control in the Blakemore et al. study) or a hysteria-specific dysfunctional behavioural pattern (i.e., disrupted adaptive behaviour in the Morris et al. study). The researchers then examined how the externally determined changes in the affective context, which they induced through the exposure to images of different valences, influenced the participants' task performance. This approach allowed the researchers to explore how patients' aberrant emotion processing interacts with and modulates additional cognitive deficits entailed in hysteria to give rise to the disorder-specific symptom manifestations or behavioural patterns. Due to its explicit focus on identifying hysteria-specific impairments in emotion processing, this approach appears to me far more epistemically productive than the arbitrary use of the implicit emotional task discussed earlier in this section.

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In sum, a decade of the gradually intensifying fMRI-based endeavour to delineate hysteria patients' dysfunctional emotion processing by using mostly decontextualised standardised visual stimuli to induce purportedly controlled emotional states within laboratory settings has brought surprisingly little progress. As we have seen, much of this research has been informed by the basic emotion approach. Hence, most studies have focused on mapping the patients' aberrant and supposedly automatic neural responses to photographs displaying facial expressions of discrete emotional categories such as fear, sadness, and happiness. We have discussed how different combinations and contrasts of these responses across various fMRI studies delivered ambiguous and often mutually conflicting results.

On a more promising note, I have also outlined a more recent development exemplified by two fMRI studies that have shifted the focus away from examining dysfunctional emotion processing in isolation. Instead, at the centre of this new approach is the investigation of how deficits in emotion processing are functionally linked to hysterical symptoms. As foregrounded by my analysis, the studies representative of this new approach have used the action-guiding concept of emotion processing to pose more specific and clearly defined questions about hysteria. Just as importantly, these studies have moved beyond the restrictive basic emotion approach. But although they hold the potential to generate relevant new insights into hysteria, such studies remain rare.[551] This can probably be attributed to the conceptual challenges

551 Two studies of this type were published in 2019. Both focused on hysteria patients with aberrant movements. See Sojka et al. "Processing of Emotions"; and Allendorfer et al., "Psychological Stress." The Sojka et al. study tested hysteria patients' spontaneous emotion regulation strategies by exposing them to negative and neutral IAPS pictures inside the MRI scanner and then asking them to voluntarily down-regulate their negative affective responses to these pictures. See Sojka et al. "Processing of Emotions," 3–4, article 861. Interestingly, in the latter study, the

entailed in developing more complex and sophisticated experimental tasks that need to be specially tailored to hysterical symptoms.

Such challenges notwithstanding, the potential role of emotions in the formation and maintenance of hysterical symptoms appears to be a topic of increasing interest in the current hysteria research. It is, therefore, safe to assume that the development towards designing more complex and symptom-specific fMRI studies will continue in the near future. Yet, one thing with which, in my opinion, future studies will have to deal with more systematically is clarifying if the distinct experimental interventions they are deploying are capable of inducing sufficiently clear-cut and controllable emotional and affective responses. To achieve this goal, however, researchers will perhaps first need to more clearly delineate the concepts of 'emotion' and 'affect' with which they operate. As highlighted by my analysis, these two concepts have so far remained vaguely defined in fMRI-based hysteria research. Sometimes they are used interchangeably as mere synonyms,[552] whereas at other times, their deployment implies mutually opposing theoretical frameworks. Such conceptual inconsistencies lead to the production of results that are difficult to compare across studies and impossible to unify into an overarching interpretation regarding hysteria patients' potential deficit in emotion processing. It appears to me that as long as such conceptual inconsistencies remain unaddressed, they will continue to impede future research.

4.4 Identifying Symptom-Related Alterations in the Intrinsic Dynamic Organisation of Hysteria Patients' Brains

Apart from the emotion processing analysed in the section above, two other action-guiding concepts have attained increasing epistemic importance in the fMRI hysteria research in the second decade of the twenty-first century. These two concepts are resting-state functional connectivity and functional neuroplasticity.[553] Both concepts

researchers did not use affective visual stimuli but instead chose to investigate how patients with non-epileptic seizures "respond to acute emotional and psychological stress." Allendorfer et al., "Psychological Stress," 2, article 101967. To experimentally induce acute emotional stress in their study participants, the researchers used negative verbal feedback. The participants were asked to perform a so-called 'stress math task' inside the scanner. Regardless of their actual math performance, during the task, the participants were exposed to pre-recorded auditory feedback repeatedly telling them that they were too slow and thus failing the task. Allendorfer et al., 3, article 101967. Finally, an additional study worth mentioning is Luo et al., "Pain Processing." In this fMRI study, published in 2016, the researchers examined "the association between emotion and pain-related brain activities" in patients with chronic somatoform pain disorder. Luo et al., 969. To do so, Luo et al. scanned their patients' brain activity while exposing them to painful pinprick stimuli and simultaneously asking them to view a series of pleasant, unpleasant and neutral pictures from the IAPS. In short, Luo et al. investigated how changing affective context modulates the patients' perception of pain at the neural level.

552 See, e.g., Aybek et al., "Emotion-Motion Interactions," 3–4, e0123273.

553 See, e.g., Diez et al., "Fast-Tracking"; LaFaver et al., "Before and After"; Otti et al., "Chronic Pain"; Wegrzyk et al., "Functional Connectivity"; Wei et al., "Default-Mode Network"; and Roy et al., "Dysphonia."