1.3 From Diagnosis to Pathogenesis and Treatment: Visualising Sensorimotor Deficits in Cases of Traumatic Hysterical Paralysis

In the two preceding sections, we have analysed how the experimental use of hypnosis enabled Charcot to move beyond a purely nosographic (i.e., descriptive) approach and focus instead on elucidating the potential neurophysiological basis of hysteria. As we will see in the rest of this chapter, hypnosis also played a significant role in the subsequent stages of Charcot's hysteria research. However, my aim in the following two sections is to show that since the mid-1880s, Charcot's hysteria research came to be characterised by a more integrative approach. Specifically, I will argue that, from this point on, Charcot's clinical concerns related to diagnosis and treatments became more closely interwoven with his experimental endeavours.

During this period, Charcot's primary emphasis shifted to the investigation of various somatosensory deficits, which he increasingly regarded as "the principal signs of hysteria."[627] These included different sensory disturbances, some of which had already been the topic of one of Charcot's early clinical lectures on hysteria.[628] Just as significantly, a symptom Charcot designated as hysterical paralysis of traumatic origin began to occupy much of his attention.[629] This symptom entailed the loss of the patient's ability to perform voluntary movement following a physical injury. The actual injury, which often consisted of a contusion caused by a fall or an unexpected blow to the limb, tended to be slight and thus healed quickly. Nevertheless, after the accident, the patient developed a seemingly inexplicable paralysis, typically accompanied by anaesthesia.[630] As I intend to show, while investigating such concurrent sensory and motor loss in his hysteria patients, Charcot managed to aptly bring together and considerably expand several disparate aspects of his previous research.

My analysis will focus on three consecutive clinical lectures Charcot delivered from the beginning of May until mid of July 1885.[631] The topic of these lectures was one-sided upper limb paralysis of traumatic origin in two male hysteria patients. Of central interest for our discussion is that Charcot achieved three things in these lectures. First, he introduced innovations in the diagnosis of traumatic hysterical paralysis. Second, he posited a novel hypothesis about the mechanism underlying the symptom's formation.[632] Third, he developed a new treatment for hysterical limb paralysis. In what follows, I will delineate these three aspects of Charcot's research while carefully tracing their mutual epistemic interactions.

The first section will discuss the new visual tools Charcot developed for diagnosing hysteria. These tools, I will argue, allowed him to increasingly focus on mapping the physiological aspects of hysteria that were inaccessible to the unaided eye. The second

627 Charcot and Marie, "Hysteria," 632.

628 For a discussion of Charcot's early lecture on hysterical hemianaesthesia, see section 1.1.1.

629 See, e.g., Charcot, "Lecture 20: Brachial Monoplegia."

630 See, e.g., Charcot, "Lecture 19: Six Cases," 253–54.

631 See Charcot, "Lecture 20: Brachial Monoplegia"; Charcot, "Lecture 21: Brachial Monoplegia"; and Charcot, "Lecture 22: Brachial Monoplegia."

632 See Charcot, "Lecture 22: Brachial Monoplegia," 305–7.

section will examine how the combined use of such diagnostic tools and hypnosis enabled Charcot to generate new insight into the potential pathogenesis of traumatic hysterical paralysis, thus pinpointing the cause and the course of the symptom's development. We will see that, at this point, Charcot finally succeeded in tentatively defining the nature of the hypothetical functional brain lesion in cases of hysterical paralysis. Finally, I will conclude this chapter by analysing how Charcot drew on his insights into the potential nature of the underlying functional lesion to develop and test a simple yet effective physiological treatment for hysterical paralysis. Throughout, I will highlight the epistemic functions that various kinds of images played at each step.

1.3.1 Using Images to Redefine the Diagnosis of Hysteria

In May 1885, Charcot gave the first of his three mutually related clinical lectures on brachial monoplegia of traumatic origin or, in other words, paralysis limited to a single arm that developed following a physical injury.[633] In such cases, patients lost voluntary control over the affected arm, which hung flaccidly by the side "as an inert body" and fell down heavily if lifted by a physician.[634] In the opening sentence of his lecture, Charcot foregrounded the difficulties entailed in diagnosing this symptom. These difficulties, as Charcot elaborated, consisted in establishing the symptom's actual nature by answering the following set of questions. Can the symptom be attributed to a lesion of the peripheral nerves caused "by a contusion or a shock to the brachial plexus?"[635] Alternatively, "[d]oes it relate to any spinal lesion? Or a focal cerebral lesion?"[636]

Put simply, when faced with a patient who developed limb paralysis after a physical injury, the physician had to perform a so-called differential diagnosis.[637] His task was to determine whether the paralysis arose from physical damage to the nervous system that may have occurred during the accident or if, on the contrary, "the patient must be considered to be hysterical."[638] However, the nineteenth-century physician had no means of directly examining in vivo the paralysed patient's nervous system to localise a potential lesion. Instead, he could only make inferences about the presence and nature of the underlying neural damage or dysfunction by systematically investigating various physiological features that characterised the symptom in question. As I intend to show in this section, it was to enable such indirect, inferential insights into the neurophysiological nature of traumatic hysterical paralysis that Charcot introduced new visual diagnostic tools. Moreover, I will argue that through his targeted use of images as diagnostic tools, Charcot succeeded in determining distinct physical features of hysterical paralysis and thus established this symptom as a clinical entity in its own right.

633 Charcot, "Lecture 20: Brachial Monoplegia," 261.

634 Charcot, 264.

635 Charcot, 266.

636 Charcot, 266.

637 Charcot and Marie, "Hysteria," 634.

638 Charcot, "Lecture 21: Brachial Monoplegia," 283. The medical term for the patients' one-sided arm paralysis was brachial monoplegia, hence the title of Charcot's lecture.

To demonstrate the efficacy of his step-by-step diagnostic procedure in which, as we will see shortly, images had key epistemic functions, Charcot presented two male patients to his audience: Porcz— and Deb—. On superficial examination, both patients seemed to exhibit an identical symptom of flaccid arm paralysis accompanied by a concurrent anaesthesia. Charcot also emphasised that the circumstances under which the two patients developed arm paralysis were strikingly similar.[639] Porcz—, who worked as a coachman, had been thrown off his carriage by a restless horse. He fell onto the pavement and landed on the backside of his right shoulder. Deb—, a labourer, also experienced an accident at work. He had been hit on the backside of his left shoulder by a large iron beam. As a result of this blow, he fell face forwards to the ground.

Having pointed out the similar circumstances that led to their paralysis, Charcot then enumerated the differences between the patients. Porcz— could neither lift his right shoulder nor move his right upper arm or forearm. He nevertheless retained a partial ability to move the fingers of his right hand.[640] Additionally, his tendon reflexes at the affected elbow were slightly exaggerated. Somewhat surprisingly, despite his paralysis having existed for more than four months, Porcz— showed "no appreciable atrophy or diminished consistency of the paralysed muscles."[641] Just as importantly, his paralysed muscles exhibited normal reactions to electrical stimulation, indicating that there were no noticeable signs of muscular degeneration.[642] By contrast, Deb— was still able to lift his shoulder but lost all mobility in the rest of his left arm, including the hand and the fingers. The tendon reflexes in his affected arm were abolished. Furthermore, his paralysed muscles were "extremely atrophied" and irresponsive to electrical stimulation, thus suggesting excessive functional degeneration.[643]

Such differences in the loss of motor function between the two patients appeared to indicate that Porcz— and Deb— did not suffer from the same type of brachial monoplegia. But, in Charcot's view, the features enumerated so far did not provide a sufficient basis for a clear-cut differential diagnosis.[644] Hence, in the next step, Charcot drew the attention of his audience to the importance of investigating the disturbances of sensibility that accompanied each patient's limb paralysis. He emphasised that particular forms of anaesthesia should be regarded as nothing less than "signs decisive for the diagnosis of hysteria in doubtful cases."[645] Yet, such signs were not immediately

639 Charcot, "Lecture 20: Brachial Monoplegia," 267.

640 Charcot, 263.

641 Charcot, 264.

642 Charcot, 266.

643 Charcot, 272.

644 The reason for the diagnostic inconclusiveness at this point was the following. As Charcot explicitly stated in another article, various degenerative changes of the muscular tissue, including atrophy (i.e., the wasting of the muscles), were "scarcely in according with the idea" of hysteria as a functional disorder. Thus, in theory, degenerative changes were viewed as pointing to potential organic damage as the underlying cause of the paralysis in question. Charcot and Marie, "Hysteria," 634. However, in actual clinical practice, for reasons Charcot was unable to explain, muscular atrophy was "not at all rare" in cases of hysterical paralysis. Ibid. Hence, in itself, the presence or absence of muscular degeneration was not a sufficient criterion for differential diagnosis.

645 Charcot and Marie, 631.

apparent. Instead, Charcot underscored that they had to be systematically searched for through meticulous clinical exploration. The modalities of anaesthesia that were regularly and methodically tested at the Salpêtrière comprised the loss of sensibility to touch, pain, heat, and cold.[646] Moreover, Charcot and his team also examined whether the loss of a particular mode of sensibility was limited to the patient's skin and mucous membranes, thus resulting in so-called cutaneous anaesthesia, or if it also affected deeper structures such as muscles, tendons, joints, and the nerve trunks.[647]

To facilitate the clinical exploration of different modalities of anaesthesia, the Salpêtrians used a range of targeted procedures. For example, to determine the distribution of the loss of sensibility to touch, the physician systematically pressed his index finger across the surface of the patient's body. The patients submitted to such examination were instructed to start counting aloud as soon as they felt any contact upon their skin.[648] During the examination of analgesia (i.e., the loss of sensibility to pain), the physician either pinched the patient's skin or pricked it with a thin sharp needle. To test the sensibility to cold, a block of ice wrapped in a woollen cloth was placed on various areas of the patient's body.[649] In contrast, the sensibility to heat was measured using a special thermometer that could be preheated to a chosen temperature and then applied to the patient's skin.[650] Finally, the extent to which the anaesthesia invaded deeper structures below the skin was evaluated by energetically twisting and stretching the patients' limbs or by exposing their peripheral nerves to intense electrical stimulation.[651] Since such interventions would have been painful under normal conditions, the patients were closely monitored during the examination to establish if they showed any signs of experiencing pain. Throughout the entire procedure, the patients were blindfolded to prevent them from seeing the interventions to which they were exposed.[652] Not being able to rely on their sight, the patients were made to focus exclusively on their ability to perceive a particular type of sensation that was being tested.

It should be emphasised that far from being invented by Charcot and his team, the exploration of hysteria patients' loss of sensibility had a long history.[653] However, besides standardising the testing procedures described above, Charcot introduced one other key innovation. Unlike their predecessors, the Salpêtrians did not merely

646 Charcot and Marie, 631. See also Tourette, *Traité clinique*, 139.

647 Charcot, "Lecture 21: Brachial Monoplegia," 294.

648 For a more detailed description of such an examination, see Tourette, *Traité clinique*, 140–41.

649 Tourette, 150.

650 Charcot himself designed this thermometer to minimise the danger of burning the patients' skin while examining their sensibility to heat, which occasionally happened when using alternative methods. For details, see Tourette, 149.

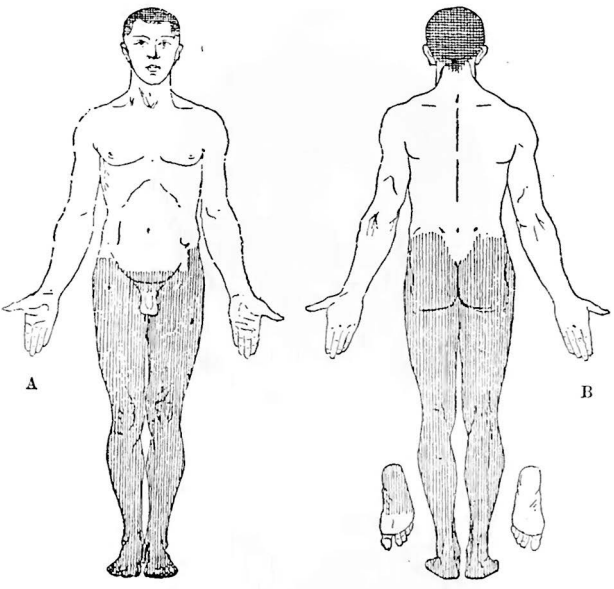
651 Charcot, "Lecture 21: Brachial Monoplegia," 294.

652 Tourette, *Traité clinique*, 140.

653 Charcot's collaborator Gilles de la Tourette compiled a historical overview of both medical and non-medical explorations of hysterical anaesthesia over the centuries leading up to the commencement of the Salpêtrian research. According to this account, the most systematic non-medical exploration of hysterical anaesthesia had taken place in the context of medieval witch trials. For details, see Tourette, 127–38.

document the results of hysteria patients' sensory examinations in the form of written descriptions.[654] Instead, they systematically visualised them in the form of diagrams. While minutely examining various parts of the patient's body, the Salpêtrians registered the findings thus obtained on one of the standardised body schemes. The diagrams the Salpêtrians used had been designed by Paul Richer specifically for this purpose and existed in several variations.[655] They consisted of a pair of schematic drawings that showed an entire generic human body or a particular anatomical segment of interest, such as an arm, a hand, a foot, or the head (figs. 1.20 and 1.21).[656] Typically, the drawing on the left displayed the front, whereas the drawing on the right showed the back view of the body. Moreover, Richer designed a male and a female version of the body maps.[657]

Figure 1.20. Body map of cutaneous and deep anaesthesia in a patient with hysterical leg paralysis. On the head is a large patch of hyperaesthesia. From: Charcot, Diseases of the Nervous System, vol. 3, 380, fig. 84.



654 Tourette, 141.

655 Tourette, 142n.

656 See, e.g., Charcot, *Leçons du mardi*, vol. 1, 2nd ed., 216, 217, 226, 284, 285, 290, 368.

657 The differences between the male and female versions of the diagram mainly concerned schematic visualisations of the primary and secondary sexual characteristics. These included the genital organs, breasts, and the more pronounced muscularity in the male. See, e.g., Charcot, 97, 255, 368.

By filling in such a diagram during the process of sensory examination, Charcot and his team were able to produce body maps that disclosed the exact anatomical distribution of each patient's various disturbances of sensibility. Owing to this translation, an essentially invisible symptom obtained a distinct visual form. Thus visualised, the salient features of anaesthesia could now be "grasp[ed] at a single glance" by a medical expert who knew how to 'read' the resulting body maps.[658] To facilitate the ease of reading of such maps, the Salpêtrians introduced certain notational rules. For example, zones of decreased sensibility were always marked by a pattern of parallel lines. Crosshatching was used to denote anatomical areas of increased sensibility, whereas black spots indicated the locations of the patient's hysterogenic zones.[659] The boundaries of the anatomical areas with disturbed sensibility were designated either by a dashed or a solid line.[660] If a physician chose to deploy any additional graphic elements, he was obliged to clarify their meaning in an accompanying caption.

Notably, Charcot was not the first physician to use schematic diagrams for mapping anaesthesia. Several late-nineteenth-century neurologists used similar schematic diagrams of the human body, or its parts, to map the anatomical distribution of anaesthesia caused by organic nerve damage.[661] In such cases, the diagrams served to relate a particular topographic pattern of the resulting anaesthesia to the anatomical locations of the damaged sensory nerves.[662] In other words, in cases of organic anaesthesia, the distinct purpose of body maps of sensory loss was to provide insights into the neurological basis of this symptom. The novelty of Charcot's approach was that he adopted this mapping procedure from the context of research into organic disturbances and applied it to hysteria. In my opinion, Charcot's motives for repurposing this mapping procedure went beyond its apparent clinical utility. Charcot's repurposing, I suggest, was rooted in the implicit proposition that the anatomical patterns of hysterical anaesthesia were not random but determined by some, at the time still unknown, underlying physiological regularities. As my analysis will show, body maps of anaesthesia were particularly suited to articulating such a proposition.[663]

Importantly, the epistemic usefulness of body maps was not limited to providing an easily graspable overview of the spatial distribution of a single patient's hysterical anaesthesia at a given moment. Instead, additional insights could be gained by comparing body maps produced at different times and for different individuals. For example, by repeatedly producing body maps at chosen intervals, the Salpêtrians could determine if and how each patient's spatial distribution of hysterical anaesthesia changed over time and thus monitor potential fluctuations in the severity of this

658 Tourette, *Traité clinique*, 141.

659 Tourette, 144.

660 I could not find out whether the Salpêtrians had any fixed rule on when to use a dashed and when a solid line or if the choice was purely arbitrary.

661 See, e.g., Ross, "Distribution of Anaesthesia," 68, 72; Mitchell, "Neurotomy," 325, 329; and Létiévant, *Sections Nerveuses*, 42, 105, 147.

662 See, e.g., Ross, "Distribution of Anaesthesia," 63–65, 68–70, 73–74.

663 I am using the terms proposition and articulation in Latour's sense. See Latour, *Pandora's Hope*, 141–44.

symptom.[664] Furthermore, body maps allowed Charcot to compare various topographic distributions of anaesthesia across multiple clinical cases, and thus search for potentially salient similarities and differences among various disorders. As we are about to see, this latter type of comparison enabled Charcot to make a key diagnostic discovery about the distribution of sensory losses in patients with traumatic hysterical paralysis.[665] To analyse how Charcot arrived at new insights, we now need to return to his clinical lecture on brachial monoplegia and take a look at the body maps of his two patients, Porcz— and Deb—.

The body maps of Porcz— and Deb— which Charcot presented to his audience and later included in the printed version of his lectures did not entail any whole-body views. Instead, they consisted of the front and back views of the anatomical segment of interest: the affected arm and shoulder. Porcz—'s map, as Charcot explained, showed that the zone in which the sensibility to touch, pain, and cold was "completely and absolutely abolished" occupied only those "parts of the extremity where there is motor paralysis."[666] The zone of cutaneous insensibility encircled the patient's entire shoulder, extending to all segments of his upper and lower arm and the wrist (fig. 1.21). But large areas of Porcz—'s hand and all of his fingers retained normal cutaneous sensibility. Through additional clinical examination, Charcot also determined that the insensibility of the deeper parts (i.e., muscles, ligaments, joints, and nerves) extended over the same areas as the cutaneous anaesthesia. Moreover, in all anaesthetic regions of his right arm, but not in the fingers, Porcz— had lost the muscular sense.[667] Hence, the areas in the map graphically highlighted by a pattern of parallel lines designated the anatomical segments in which multiple sensory modalities were lost simultaneously.

As Charcot pointed out, the particular anatomical distribution of the patient's anaesthetic zones was not the only potentially salient clinical fact disclosed by the map. Another particularly interesting and previously unknown aspect of Porcz—'s anaesthesia was the "singular disposition" of its outline.[668] The map revealed that the anaesthesia did not end at the shoulder or the wrist. Instead, it spread a few inches beyond each paralysed joint. But the key point was the following. At each end, the lines that delimited the anaesthetic segment from the anatomical parts that retained their sensibility had a distinctly circular form. Charcot emphasised that both of these circular lines occupied a distinct position. Each line was located in an imaginary plane that was perpendicular to the main axis of the affected limb.[669] These topographic characteristics of Porcz—'s anaesthesia could now be perceived as potentially salient clinical facts only because they had been made visible by the body map.

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664 Tourette, *Traité clinique*, 141–42. I will analyse such use of body maps in more detail in the following section.

665 See Tourette, 155–58.

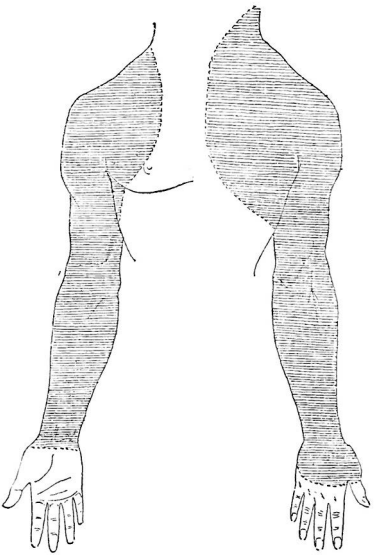
666 Charcot, "Lecture 20: Brachial Monoplegia," 264.

667 In Charcot's words: "When his eyes are shut, he does not know whether one bends his wrist, his elbow, or his shoulder. But under like conditions he knows perfectly well when the same act is practised on his fingers, and which one is experimented upon." Charcot, 265.

668 Charcot, 264.

669 Charcot, "Lecture 21: Brachial Monoplegia," 282n5.

Figure 1.21. Body map of cutaneous and deep anaesthesia in Porcz—. From: Charcot, Diseases of the Nervous System, vol. 3, 268, figs. 54 and 55.



Next, Charcot introduced the body map that displayed the topographic distribution of Deb—'s anaesthesia (fig. 1.22).[670] Also in this map, the areas graphically highlighted by a pattern of parallel lines designated the simultaneous loss of cutaneous and deep sensibility, as well as the muscular sense. However, even a superficial glance at this map sufficed to make evident the considerable differences in the spatial distribution of anaesthesia between Porcz— and Deb—. In Deb—'s case, the anaesthesia occupied the entire hand and fingers. Nevertheless, the total area affected was considerably smaller than in Porcz—. As the map showed, Deb—'s shoulder and large areas of his upper arm retained their normal sensibility. Even more importantly, in Deb—'s case, the limit of the anaesthetic zone did not have a circular but instead an irregular, zigzag shape.

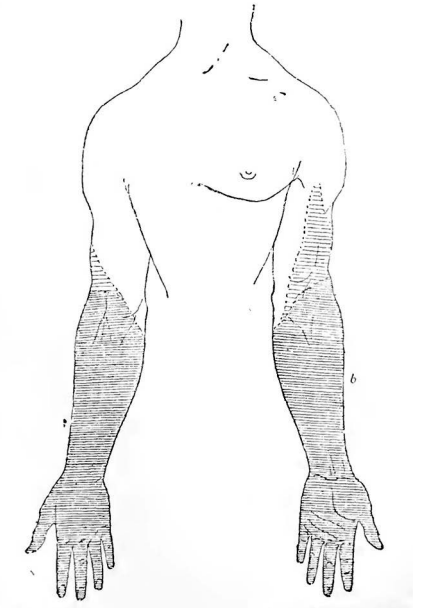
At this point, Charcot moved beyond the mere comparison of the two maps and started to make interpretational claims about the visual patterns that each map displayed. First, Charcot turned to Deb—'s map. He declared that such an apparently irregular topographic distribution of anaesthesia was known to occur when the brachial plexus—i.e., the network of peripheral nerves running from the spine into the arm—had been "severely injured, or even torn across completely."[671] To support this

670 In this particular map, the backside view is shown on the left and the frontside view on the right. I presume that the purpose of this inversion was to visually accentuate the fact that, unlike Porcz—who had right-sided paralysis, Deb—'s affected hand was on the left side of his body.

671 Charcot, "Lecture 20: Brachial Monoplegia," 270.

claim, Charcot quoted an article published in the scientific journal *Brain* by the Scottish neurologist James Ross. In his article, Ross gave a clinical account of a patient with a ruptured brachial plexus and accompanied it by a body map of the resulting organic anaesthesia.[672] The map of Ross's patient, which Charcot presented to his audience, showed "exactly the same" distribution of the complete anaesthesia as the one observed in Deb—.[673]

Figure 1.22. Body map of cutaneous and deep anaesthesia in Deb—. From: Charcot, Diseases of the Nervous System, vol. 3, 269, figs. 56 and 57.



Based on the comparison with Ross's findings, Charcot concluded that the irregular shape of Deb—'s anaesthesia was determined by the anatomical distribution of the peripheral nerves of the arm. This, in turn, enabled Charcot to attribute both Deb—'s anaesthesia and the concurrent motor paralysis of the arm to "deep and destructive organic lesions affecting all the motor and the sensory branches of the brachial plexus."[674] Deb— received a diagnosis of incurable brachial monoplegia of organic origin and was allowed to retire. By contrast, Charcot stated that the distinct circular limits of Porcz—'s anaesthesia did not at all accord with the anatomical distribution of

672 Ross, "Distribution of Anaesthesia," 70–74.

673 Charcot, "Lecture 20: Brachial Monoplegia," 270. See also Ross, "Distribution of Anaesthesia," 70–74.

674 Charcot, "Lecture 20: Brachial Monoplegia," 270.

the sensory nerves of the arm.[675] Charcot thus dismissed the possibility that Porcz—'s paralysis had been caused by physical damage to his peripheral nerves. He conjectured instead that the seat of Porcz—'s "disease had to be sought for elsewhere in the nerve centres."[676]

In the next step, Charcot turned to examining the possibility that Porcz—'s monoplegia arose from an organic lesion situated either in the spinal cord or in one of the cerebral hemispheres. To this end, he systematically considered several likely anatomical locations in the spinal cord and the brain, which, if physically damaged, could have given rise to the flaccid one-sided paralysis of the arm with the clinical features seen in Porcz—.[677] In doing so, Charcot drew both on his clinical experience and multiple studies recently published by his medical colleagues, including David Ferrier.[678] Crucially, Charcot's reasoning throughout this process was informed by the localisationist paradigm. As mentioned previously, in this paradigm, a particular sensory and motor function was attributed to the activity of a specialised anatomical region of the brain or the spinal cord.[679] Consequently, the loss of a particular function was understood to arise from a lesion localised in a designated anatomical region of the central nervous system, which in the healthy state presided over that function.

Drawing on the paradigm of cerebral localisation, one by one, Charcot rejected each of the possible organic lesions of the central nervous system that he had considered. He argued that several organic lesions of the brain could have resulted in a flaccid brachial monoplegia of the extent and severity that Porcz— had. Yet, based on the studies of cerebral localisation published by his colleagues, Charcot conjectured that no known organic lesion would have led to the topographic distribution of the anaesthesia seen in Porcz—'s map.[680] Hence, it was because of the distinct geometric shape of Porcz—'s anaesthesia that Charcot was able to dismiss the possibility that, in this case, the brachial monoplegia was caused by an organic lesion of the cerebral cortex. With no other diagnostic options left, Charcot could now plausibly suggest that Porcz—'s symptoms were of hysterical origin.

It is worth noting that, up to this point, Charcot had been performing a particular type of differential diagnosis, whose aim was to exclude all potential organic causes of Porcz—'s symptoms. Using such a diagnostic approach was necessary at the time because there were no known clinical features of hysterical paralysis or the accompanying anaesthesia that were considered specific to these symptoms. This meant that hysteria was defined in purely negative terms as "an assemblage of odd [and]

675 Charcot, 271–72.

676 Charcot, 273.

677 For details on various organic lesions that Charcot considered and then dismissed, see Charcot, "Lecture 21: Brachial Monoplegia," 275–78.

678 Charcot, 277–78.

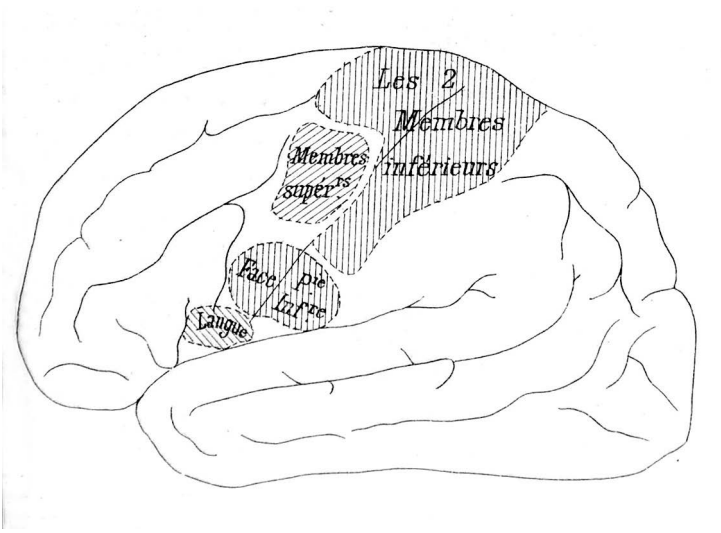
679 We have already discussed the paradigm of cerebral localisation (see sections 1.1 and 1.1.1) and will return to it in the following section. Furthermore, later in this enquiry, we will see that contemporary functional neuroimaging studies of hysteria are informed by a comparable view, according to which particular cognitive functions can be mapped onto the brain's anatomical structure.

680 Charcot, "Lecture 21: Brachial Monoplegia," 277–78.

incoherent" symptoms that remained after the physician had successfully eliminated all known organic diseases as possible diagnostic alternatives.[681] As my analysis will show, Charcot was about to change that.

Remaining firmly embedded in the paradigm of cerebral localisation, in the next step, Charcot stated that Porcz—'s symptoms could be easily explained by positing the existence of "a dynamic hysterical lesion" of cortical origin.[682] He admitted that, for the time being, he could not determine the exact nature of this hypothesised brain lesion since it escaped the means of empirical investigation available to him. Yet, he asserted that the hysterical lesion had to be categorically different from "a circumscribed organic lesion of a destructive nature," such as the ones he had already considered and dismissed during his lecture.[683] In effect—and this is crucial—Charcot argued that hysterical paralysis did not arise from permanent damage to brain structure but from a localised and potentially transient disruption of brain function. Moreover, at this point, Charcot also proposed that, based on the distinct clinical features of Porcz—'s symptoms, it was possible to infer the anatomical location of the specialised cerebral centres that were affected by the hypothesised dynamic brain lesion in cases of one-sided hysterical arm paralysis. This inference was decidedly informed by Charcot's empirical studies into the cerebral localisation of motor function (fig. 1.23).

Figure 1.23. Brain map displaying the anatomical locations of specialised cerebral motor centres that, according to Charcot, controlled voluntary movements of the arms, legs, face, and tongue, respectively. From: Charcot, Leçons du mardi 1:139.



681 Charcot, "Lecture 1: Introductory," 12.

682 Charcot, "Lecture 21: Brachial Monoplegia," 281.

683 Charcot, 278.

First, Charcot reasoned that the lesion causing Porcz—'s monoplegia was situated "in the grey matter of the cerebral hemisphere on the side opposite the paralysis, and more precisely in the motor zone of the arm."[684] Further, by taking into account the distribution, the distinct geometrical shape, and the intensity of the patient's anaesthesia, Charcot posited that the disruption of function could not be limited to the motor zone of the arm. Instead, he conjectured that it had to extend "behind the medial convolutions to the adjacent part of the parietal lobe."[685] Put differently, in Charcot's view, the dynamic lesion occupied both the motor and the sensory brain centres that jointly controlled the sensorimotor functions of the arm affected.

It should be emphasised that, from this point onward, Charcot no longer used Porcz—'s map of anaesthesia merely as a tool of differential diagnosis that allowed him to exclude potential organic causes of the brachial monoplegia in his patient (see fig. 1.21). Instead, as I have shown, Charcot began using Porcz—'s body map in an epistemically innovative way to make inferences about the type (i.e., functional) and the potential anatomical location of the underlying brain disturbances to which he then causally attributed the symptom. Hence, it can be said that, at this point, Charcot started deploying Porcz—'s body map as an active epistemic tool with which he generated new insights into a potential neurophysiological basis of hysterical one-sided arm paralysis.

Having posited a distinct neurological cause for the "particular mode of distribution and limitation" of Porcz—'s anaesthesia, Charcot then asserted that its distinct geometric pattern was by no means accidental but instead represented a feature specific to hysteria.[686] Simply put, Charcot contended that the distinctive visual form of the anaesthesia displayed by Porcz—'s body map was already an unequivocal sign of the hysterical origin of this particular symptom. To support this far-reaching claim with additional empirical evidence, Charcot presented to his audience another patient named Pin—. This patient had also developed a long-standing brachial monoplegia following an accident at work.[687] Yet, unlike Porcz—, who neither experienced any hysterical attacks nor had any traceable hysterogenic zones, Pin— represented a more 'classic' case of hysteria.[688] As Charcot pointedly declared, Pin— had several clearly

684 Charcot, 278. It is important to note that, according to Charcot, the motor zone of the brain, which presided over the accomplishment of voluntary movements, was not functionally homogenous. Instead, based on his localisation studies, Charcot argued that this zone consisted of multiple specialised motor centres, each controlling the voluntary movements of a particular muscle group or a body part. See Charcot and Pitres, *Les centres moteurs*, 192–95. The topographic distribution of these different motor centres is visualised in the hand-drawn brain map seen in fig. 1.23. Charcot presented this map during the Tuesday lecture he held on 24th January 1888 while repeating his hypothesis that hysterical arm paralysis arose from a dynamic brain lesion situated in the cerebral motor centre of the arm. See Charcot, *Leçons du mardi* 1:139–41.

685 Charcot, "Lecture 21: Brachial Monoplegia," 278.

686 Charcot, 282.

687 Pin— had been working as a mason's apprentice when he fell from a height of about two metres. He thereby sustained a contusion of his left shoulder. See Charcot, "Lecture 19: Six Cases," 253.

688 Although Charcot and his team submitted Porcz— to a systematic examination, they could not detect any hysterogenic zones on his body. They thus considered Porcz— to be an atypical case of hysteria. See Charcot, "Lecture 21: Brachial Monoplegia," 286–87.

delineated hysterogenic zones. The patient also suffered from repeated hysterical attacks that had all the characteristics of the 'complete and regular' four-stage model.[689]

However, what mattered even more were the striking similarities in the brachial monoplegia developed by both patients. As effectively demonstrated by the body map Charcot displayed to his audience, the shape of the cutaneous and deep anaesthesia that accompanied Pin—'s arm paralysis (see fig. 1.21) was almost "identical" to that of Porcz— (fig. 1.24).[690] Admittedly, in Pin—'s case, the anaesthesia was slightly more widespread as it also affected his hand and the fingers. Yet, as Charcot emphasised by directly comparing the two patients' body maps, the key point was that, in both patients, the anaesthesia was "limited exactly in the same manner at the shoulder."[691] In both Pocz— and Pin—, the anaesthesia encircled their shoulder and was marked off by a circular line positioned at the right angle to an imaginary axis running through each patient's extended arm. That is, the same highly specific geometric pattern characterised the anaesthesia not just in Pocz—, who due to the absence of hysterical attacks represented a less typical case, but also in Pin—, who was considered a classic case of *grande hystérie*.[692]

Finally, Charcot posited that the same distinctive form of anaesthesia must also be valid in "ordinary cases of hysteria," which, by definition, had to fall somewhere between Porcz—'s atypical and Pin—'s classic case.[693] At this point, Charcot declared this particular circular delimitation of the accompanying anaesthesia to be a decisive diagnostic sign of hysterical paralysis of the arm.[694] Just as importantly, in a lecture he gave in May 1886, Charcot extended the same diagnostic principle to the hysterical paralysis of lower limbs.[695] Once again, relying on the analysis of body maps, he determined that the accompanying anaesthesia in hysterical leg paralysis of traumatic origin was delimited by an equally characteristic circular line (see fig. 1.20). In cases of hysterical leg paralysis, the boundary line was typically located at the level of the abdomen.[696]

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689 According to Charcot's description, Pin—'s hysterical attacks were "absolutely classic; to the epileptoid phase immediately succeeded that of the greater movements. These were of an extreme violence; the patient, in the movements of salutation, went so far as almost to strike his face against his knees. Shortly afterwards he tore the sheets, the curtains of his bed, and turning his fury against himself, he bit his left arm. The phase of passionate attitudes immediately followed, and P— became a prey to a furious delirium; he became abusive, and cited imaginary persons to murder,—'Hold! Take you knife… Quick… Strike!' Ultimately he came to himself, and he affirmed that he had no remembrance of what had occurred." Charcot, "Lecture 19: Six Cases," 257–58. For a detailed discussion of Charcot's four-stage model of the major hysterical attack, see section 1.1.3.

690 Charcot, "Lecture 21: Brachial Monoplegia," 287.

691 Charcot, 284.

692 Charcot, 287.

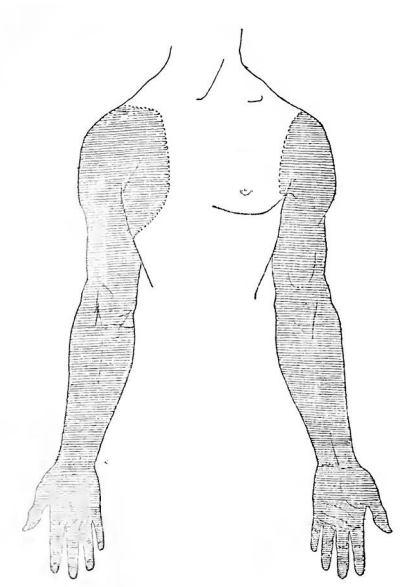
693 Charcot, 287.

694 Charcot and Marie, "Hysteria," 633–34.

695 Charcot, "Appendix 1: Hystero-Traumatic Paralysis," 374–82.

696 In front, the boundary line "passes along the fold of the groin, excluding the genital organs, and reaching to the iliac spine; and behind the boundary line follows the origin of the gluteal muscles, excluding a v-shaped space in the centre which corresponds to the posterior surface of the sacrum." Charcot, 381.

Figure 1.24. Body map of cutaneous and deep anaesthesia in Pin—. From: Charcot, Diseases of the Nervous System, vol. 3, 285, figs. 60 and 61.



To summarise my discussion so far, it can be argued that, in his lectures on brachial monoplegia, Charcot succeeded in establishing distinct diagnostic criteria for hysterical limb paralysis. Charcot achieved this by analysing and comparing diagrams that visualised the topographic distributions of anaesthesia accompanying the loss of motor function in multiple patients. Once Charcot was able to identify the particular geometric shape and the circular delimitation of the anaesthetic zones both in a 'less complete' and a more 'classic' case of hysteria, the body maps he used for this purpose effectively acquired the status of diagnostic tools (figs. 1.20, 1.21, and 1.24). This meant that in subsequent cases of traumatic limb paralysis, it was no longer necessary to perform an elaborate diagnosis of exclusion to determine if the patient suffered from hysteria. Instead, according to Charcot, it sufficed to map the distribution of the patient's cutaneous and deep anaesthesia.[697] If the resulting map displayed the characteristic circular limits of the anaesthetic zones, the physician could reliably diagnose the patient with hysterical paralysis based on the body map alone. Charcot thus radically refashioned the diagnosis of hysterical paralysis into a clinical procedure that, from that point on, centred on identifying the symptom's distinct disorder-specific physical features.

697 Charcot and Marie, "Hysteria," 633–34.

However, while mapping patients' loss of sensibility proved to be an effective diagnostic tool concerning hysterical paralysis, it did have two caveats. First, as stated by Charcot, cutaneous and deep anaesthesia, "although extremely frequent, may in some cases be absent" and could, therefore, not be considered "an absolutely constant symptom."[698] Second, the diagnostic significance of a particular shape of anaesthesia was limited to patients with hysterical paralysis. Hence, in clinically more ambiguous cases and those without hysterical paralysis, an alternative diagnostic strategy was required. With this aim in mind, Charcot additionally focused on systematically monitoring and studying hysteria patients' various impairments of vision to which he attributed particular diagnostic significance.[699]

To begin with, Charcot underscored that all hysterical visual disturbances were purely functional. This meant that despite the most meticulous ophthalmological examination, no structural pathological alterations of the eye could be discovered in hysteria patients.[700] Nevertheless, hysteria patients suffered from a surprisingly wide range of visual problems. These included double vision (polyopia), derangements of visual acuity (amblyopia), loss of colour vision (achromatopsia), as well as partial and total blindness (amaurosis).[701] Different functional visual defects were regularly examined and systematically studied in the Salpêtrian ophthalmological laboratory. However, one particular category of visual disturbances stood at the centre of Charcot's research. From the perspective of differential diagnosis of hysteria, Charcot attached prime importance to mapping various distortions of the patients' fields of vision.[702]

The term 'field of vision' designates an area that an individual can visually perceive while their gaze is fixed on a steady point in front of them.[703] The size of the visual field is determined by the extent of the individuals' peripheral vision, or in other words, their ability to perceive objects beyond the point of fixation. In effect, by systematically measuring and visualising hysteria patients' visual fields, Charcot monitored the potential distortions of their peripheral vision. To identify the extent and the shape of their patients' visual fields, the Salpêtrians used an instrument called the perimeter.[704] It consisted of a metal arc that could be rotated in different directions, thus describing an imaginary half-sphere in space. The inner side of the arch was black. On its outer side, a numerical scale was attached. The numbers on the scale ranged from 0 in the middle of the arc to 90 at each outer end. Each number designated the angle of the arc at a given point.

The patient whose visual field was assessed had to sit still in front of the device and fix their gaze on the point in the centre of the arc. While one eye was examined, the other was covered with a blindfold. Depending on whether they were interested in assessing

698 Charcot and Marie, 634.

699 Charcot, "Lecture 6: On Hysteria in Boys," 72.

700 Charcot, 75–76.

701 See Charcot and Marie, "Hysteria," 632; Charcot, "Lecture 6: On Hysteria in Boys," 72–73; Charcot, "Lecture 21: Brachial Monoplegia," 280-81; and Tourette, *Traité clinique*, 321–81.

702 Charcot and Marie, "Hysteria," 631. Charcot used the terms 'field of vision' and 'visual field' interchangeably, as I also will.

703 Tourette, *Traité clinique*, 333.

704 For details, see Tourette, 332–34.

the patient's vision for white light or a particular colour, the Salpêtrians used white or coloured pieces of paper as visual stimuli.[705] The physician placed a piece of paper on the outer limit of the instrument's arc and then slowly moved it towards the centre until reaching the point at which the patient was able to perceive the stimulus.[706] The physician then determined the position of that point by reading the numerical value of the angle on the instrument's scale. This point indicated the limit of the patient's visual field in the given direction. By rotating the arc and performing the same operation from multiple directions, Charcot and his team were able to determine the exact extent of the patient's peripheral vision from all sides. The perimeter thus enabled the Salpêtrians to quantify each patient's extent of peripheral vision.

Significantly, the Salpêtrians registered the numerical results obtained through the perimetric examination of each patient on a standardised diagram. The diagram was composed of nine mutually equidistant concentric circles, whose joint centre denoted the fixation point. The outer circle of this diagram designated the external limits that were measurable by the instrument and was thus larger than the normal visual field of a healthy subject.[707] Typically, the perimetric map consisted of two such diagrams, one for each eye. In addition to inscribing the exact limits and the spatial distribution of the patient's visual field, the Salpêtrians also always graphically highlighted the extent of the normal field of vision on each perimetric map (figs. 1.25 and 1.26).[708] In effect, the perimetric map was a visual tool specifically designed to enable the Salpêtrians to determine at a glance how and to what extent the patient's visual field deviated from the normal field of vision. Similarly to the body maps of anaesthesia, the perimetric diagrams disclosed hysteria patients' functional sensory disturbances that were inaccessible to unaided observation. Hence, to become an object of medical analysis, a potential distortion of hysteria patients' fields of vision first had to be made accessible through the process of targeted measurement and subsequent visualisation of the thus obtained numerical data.

By systematically submitting his hysteria patients to perimetric examinations and then comparing the resulting maps, Charcot made several important discoveries. On the whole, the accumulated empirical data disclosed that hysteria patients tended to exhibit highly specific disturbances of the visual field. Moreover, the map revealed that each of these disturbances was characterised by a distinct pattern of regularities. For example, one of the most frequently observed disturbances discovered through the analysis of multiple perimetric maps was what Charcot designated as the concentric narrowing of the field of vision (fig. 1.25).[709] Such narrowing meant that hysteria patients lost much of their peripheral vision in the affected eye, retaining only the ability to see what was directly in front of them. Notably, Charcot established that the retraction of

705 Tourette, 333.

706 Tourette, 333.

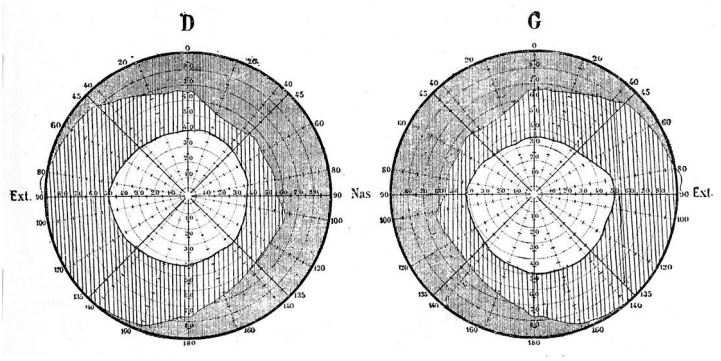
707 Tourette, 334. According to the Salpêtrians, the normal visual field extended approximately 55 degrees toward the nose, 90 degrees outwards, 55 degrees upwards, and 60 degrees downwards. Ibid., 331.

708 For additional examples, see, e.g., Charcot, *Leçons du mardi*, 2:9, 31, 124.

709 Charcot and Marie, "Hysteria," 631–32.

the peripheral vision in hysteria patients progressed symmetrically in all directions. This meant that, as the visual field shrank, it retained a distinctive circular shape. The result was the so-called tunnel vision. As Charcot pointed out, the geometric regularity of the constricted visual field was a decisive sign of hysteria because, in disorders that arose from structural damage to the optic nerve, the visual field retracted in a highly irregular manner.[710] In some cases of hysteria, the concentric loss of the peripheral vision was either limited to or more pronounced in one eye.[711] But more often, as in the case of Porcz—, both eyes were equally affected, resulting in bilateral tunnel vision.[712]

Figure 1.25. Perimetric map showing a bilateral narrowing of the visual field in a hysteria patient. The area shaded with vertical parallel lines designates the normal visual field. The inner white area shows the size and the distribution of the patient's retracted visual field. From: Charcot, Leçons du mardi, vol. 2, 159, fig. 34.



Apart from the general narrowing of the visual field, Charcot attached even greater diagnostic significance to hysteria patients' disturbances of colour perception.[713] He determined that some of his hysteria patients lost all sense of colour so that everything they saw appeared grey, as in "an uncoloured photograph seen through a stereoscope."[714] Yet, Charcot discovered that, more often, patients tended to retain the ability to perceive some colours. To investigate the variations in hysteria patients' loss of colour perception, the Salpêtrians produced perimetric maps that simultaneously displayed multiple visual fields for different colours (fig. 1.26). Producing such maps was time-consuming since the visual field for each colour had to be measured separately. However, it was also epistemically insightful. By inscribing the measurement results within a single diagram, the Salpêtrians could determine how the visual field for each

710 See Charcot, *Leçons du mardi*, 2:165.

711 Charcot and Marie, "Hysteria," 632.

712 Charcot, "Lecture 21: Brachial Monoplegia," 281, 285n.

713 Charcot, "Lecture 6: On Hysteria in Boys," 72.

714 Charcot, 73.

colour retracted. Even more importantly, such a diagram allowed them to explore relative spatial relations among the losses of peripheral vision for different colours.

Based on the analysis of such perimetric maps, Charcot discovered that, in most hysteria patients, there was a specific order in which the disappearance of particular colours took place as the illness progressed.[715] According to Charcot, this order was determined by the same physiological laws that governed the perception of colours in healthy individuals. He explained that, under normal physiological conditions, the visual field was the widest for blue, followed by a narrower field for yellow, a yet smaller one for red, and then green. Finally, violet was "only perceived by the most central part of the retina."[716] The comparison of perimetric maps obtained for multiple patients disclosed that the visual fields for different colours tended to retract concentrically, while maintaining their relative spatial positions and proportions. As a result, in most cases, hysteria patients first ceased perceiving violet and then also green and red. But on average, they tended to retain the ability to perceive yellow and blue much longer than the other colours.[717] Charcot declared this successive disappearance of the ability to distinguish different colours to be another distinctive feature of hysteria.

There was one caveat, however. Charcot admitted that both the concentric retraction of the visual field for white light and the successive loss of colour perception could also "be met with in central [organic] lesion of the brain occupying [the region called] the internal capsule."[718] In short, these two types of visual disturbance were not entirely hysteria-specific. Still, if unsure, a physician could use one significant diagnostic distinction as a point of orientation. In cases of the organic lesion of the internal capsule, the visual disturbances were always accompanied by a complete hemianaesthesia, i.e., the loss of all modes of sensibility on one side of the body.[719] Conversely, complete hemianaesthesia was not necessarily present in cases of hysterical visual field disturbances. Hence, the absence of accompanying complete hemianaesthesia indicated that the visual disturbances were of hysterical origin.

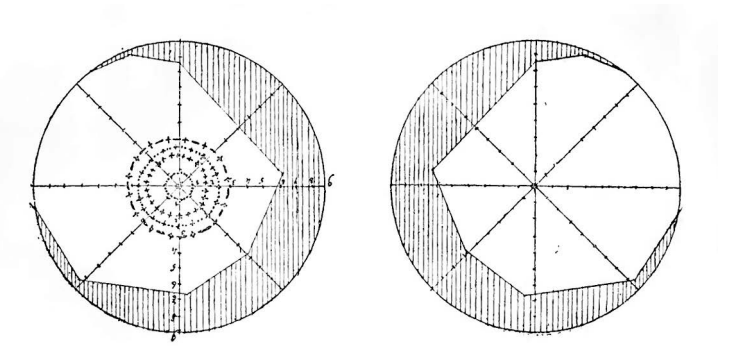
But even more conveniently, through perimetric mapping, Charcot discovered one particular anomaly, which he declared to be exclusive to hysteria, as it appeared in no other clinical context.[720] In this anomaly, the visual field for red contracted to a lesser degree than visual fields for other colours. As a consequence of this relative disproportion in the shrinking across different colours, the visual field for red became larger than the respective fields for other remaining colours (fig. 1.26). Charcot referred to this particular visual field disturbance as the "transposition of the red circle."[721] In more pronounced cases, patients gradually lost the perception of all other colours except red. Charcot argued that this particular disturbance of vision deserved to "be classed among the principal signs" of hysteria.[722] Put differently, in Charcot's view, there could

715 Charcot and Marie, "Hysteria," 632.
716 Charcot, "Lecture 6: On Hysteria in Boys," 72.
717 Charcot, 73.
718 Charcot, 74.
719 Charcot, *Oeuvres complètes*, 1:432.
720 Charcot and Marie, "Hysteria," 632.
721 Charcot, "Lecture 21: Brachial Monoplegia," 281.
722 Charcot and Marie, "Hysteria," 632.

be no doubt whatsoever that patients who exhibited this particular symptom should receive the diagnosis of hysteria.

Notwithstanding the peculiar specificity of 'the transposition of the red circle,' Charcot nevertheless insisted that all forms of concentric narrowing of the visual field—for white light, as well as for particular colours—belonged to the most constant and "most typical symptoms of hysteria."[723] These symptoms were significant for Charcot for several reasons. First, in Charcot's view, the fact that the retraction of the visual field always progressed concentrically indicated a distinct physiological basis of this disturbance. As mentioned above, Charcot observed a similar concentric retraction of the field vision in patients with an organic lesion of the subcortical brain structure called the internal capsule, to which he referred as "the sensory crossroad."[724] Drawing on this similarity in the symptom manifestations, Charcot argued that, in cases of hysterical visual disturbances, a lesion, albeit of a purely dynamic nature, must occupy more or less the same anatomical location. Specifically, he conjectured that the dynamic lesion causing the hysterical loss of peripheral vision was "likely to be located either in the very fibres crossing the sensory crossroad, or in their extension towards the brain surface, or in all these different parts at once."[725] Hence, similarly to body maps of anaesthesia, Charcot also used perimetric maps to make inferences about the nature and location of the functional brain disturbances that could have given rise to the hysterical symptoms in question.

Figure 1.26. Perimetric map of Pin— showing both the general narrowing of the visual field for white light and the transposition of the limits of the visual field for red in the left eye. The white area designates the distribution of the normal visual field. The patient's visual field in the right eye is normal. From: Charcot, Diseases of the Nervous System, vol. 3, 287, figs. 62 and 63.



723 Charcot and Marie, 631.

724 Charcot, *Oeuvres complètes*, 1:432.

725 Charcot, 432 (my translation).

Second, Charcot argued that, due to the highly specific features of hysterical visual field defects, which could only be fully determined through the perimetric examination, patients could neither convincingly simulate nor wilfully exaggerate such symptoms.[726] Hence, when diagnosing these particular symptoms, the physician did not need to fear being duped by the patient. Third, Charcot also claimed that various visual field disturbances were often very accentuated, particularly in those hysteria patients whose "troubles of general sensibility may be but little marked" or even absent.[727] Consequently, perimetric maps that displayed either a general concentric narrowing of the patients' visual fields or the transposition of the red circle became for Charcot the most reliable and frequently used visual tools for diagnosing hysteria.[728] He especially relied on these visual tools to identify hysteria in doubtful cases that lacked more 'classic' symptoms, such as the convulsive attack or hysterogenic zones.[729]

Nevertheless, this did not mean that various defects of the visual field were of no interest to Charcot if they appeared in diagnostically less challenging cases. For example, in the lecture on brachial monoplegia, Charcot emphasised that Pin—, whom he had already diagnosed as a 'classic' case of hysteria, exhibited a clear-cut transposition of the red circle in his left eye.[730] Charcot demonstrated this by presenting the patient's perimetric map to his audience (fig. 1.26). This map also disclosed that, in his left eye, Pin—'s visual field for white light was considerably retracted, thus resulting in tunnel vision. Additionally, the map showed that Pin—'s visual field remained normal in his right eye. In Pin—'s case, the perimetric map was not essential for establishing the differential diagnosis of hysteria. Even so, the map was epistemically useful. It provided Charcot with additional clinical insights into the extent and severity of Pin—'s accompanying visual disturbances. The map also revealed that Pin—'s various visual disturbances clustered on the left side of his body.

***

To conclude, I have shown that by introducing the visual tools discussed in this section, Charcot developed a novel approach to diagnosing hysteria. These tools allowed Charcot to shift the clinical focus on those hysterical symptoms whose very presence and the diagnostically salient features were essentially invisible until disclosed through the mutually correlated processes of targeted measurement and visualisation. Moreover, I have argued that by systematically using standardised diagrammatic visualisations to display the topographic distribution of hysteria patients' multiple sensory dysfunctions, Charcot redefined the diagnosis of hysteria in an even more profound sense. Due to the introduction of these visual tools, Charcot was no longer forced to diagnose hysteria based on the mere absence of other organic diseases. Instead, he could now diagnose his patients based on the actual presence of hysteria-specific symptoms. This radically new approach foregrounded the physiologically distinct and diagnostically salient character

726 Charcot, "Lecture 6: On Hysteria in Boys," 72.

727 Charcot, 72.

728 See, e.g., Charcot, *Leçons du mardi*, 2:163, 168.

729 Charcot, "Lecture 21: Brachial Monoplegia," 280.

730 Charcot, 285–86.

of select hysterical symptoms. Charcot thus displaced the diagnosis of exclusion with a diagnosis of inclusion.

Finally, we have also seen that the image-based discovery of hysterical symptoms' unique characteristics was not only significant from the diagnostic point of view. Rather, the particular visual patterns discovered through the process of mapping hysteria patients' different sensorial and sensitive disturbances permitted Charcot to make inferences about the underlying neurological basis of these symptoms. In fact, as the following section will show, the rest of Charcot's lectures on brachial monoplegia in Porcz— and Pin— directly built on these inferences by focusing on experimentally delineating the symptoms' potential neurophysiological basis.

1.3.2 Elucidating the Pathogenesis of Hysterical Paralysis and Developing New Treatment

So far, we have analysed how in his multipart lecture on hysterical arm paralysis of traumatic origin, Charcot actively used images to uncover previously unknown hysteria-specific characteristics of this symptom. We have also seen that Charcot relied on the diagrams of the patients' concurrent anaesthesia to diagnose them with paralysis of hysterical origin and to attribute their symptoms to what he termed a dynamic brain lesion. In what follows, we will examine how in the remaining part of his lectures on hysterical monoplegia, Charcot turned to defining the nature of this presumed lesion and positing a mechanism of its formation. I will argue that, in doing so, Charcot developed a generalisable hypothesis of hysteria's pathogenesis, which he later gradually expanded to other hysterical symptoms. We will also discuss how, by drawing on his insights into the mechanism underlying the formation of hysterical paralysis, Charcot devised a novel treatment. Finally, I will highlight that to demonstrate the efficacy of his new treatment, Charcot once again reverted to images.

By the time he turned his attention to investigating the potential neurophysiological mechanism underlying traumatic hysterical paralysis, Charcot had already firmly subscribed to the view that the aetiology of hysteria was primarily hereditary.[731] According to Charcot, the onset of hysteria was facilitated by so-called occasional causes or precipitating factors. The precipitating factors (*agents provocateurs*) varied considerably from patient to patient and could include physical accidents, intense emotions, fatigue, alcoholism, as well as different organic and infectious diseases.[732] Such diverse external environmental conditions played a crucial role in triggering the onset of hysterical symptoms. Nevertheless, they could only do so in biologically predisposed individuals, who were "born susceptible to hysteria (*hystérisables*)."[733] In other words, in Charcot's view, hysteria did not commence with the clinical

731 See, e.g., Charcot, "Lecture 7: Contracture of Traumatic Origin," 85.

732 For a more detailed list of triggering factors, see Charcot and Marie, "Hysteria," 628. Charcot's pupil, George Guinon, dedicated an entire book to studying different triggering factors. See Guinon, *Les agents provocateurs*.

733 Charcot and Marie, "Hysteria," 628 (emphasis in original). See also Charcot, "Leçon 14: A propos d'un cas d'hystérie masculine," 291–92.

manifestations of its first symptom. Instead, the disease itself "was pre-existent, but was ignored, and it only wanted an opportunity for breaking forth."[734] Charcot insisted that the particular external condition that triggered the onset of a hysterical symptom did not determine the type or characteristics of the resulting symptoms.[735] The types of symptoms each patient developed depended exclusively on their hereditary make-up.

As Charcot further argued, the underlying morbid predisposition of the nervous system to developing hysteria was something that patients inherited from their ancestors. Drawing on the influential doctrines of biological inheritance espoused by the French physician Prosper Lucas,[736] Charcot differentiated between two types of neuropathic heredity concerning hysteria. He designated as the 'heredity of similitude' those cases in which "hysterical parents beget hysterical offspring."[737] By contrast, he stated that in so-called 'heredity by transformation,' the inborn neurological defect underwent an evolution while being transmitted from one generation to another.[738] For instance, if parents had epilepsy, the inherited neurological condition in their children could manifest itself in the form of hysteria.[739] Charcot did not explain how such a transformation took place. He also did not specify what exactly constituted the inherited predisposition to hysteria at the neurological level. In fact, inheritance, "as Charcot and his fellow clinicians as well as most scientists understood it in the era before the recognition of Mendel's laws of genetics, was a nonspecific blending process of descent from ancestors."[740]

As a logical consequence of his hereditarian views, Charcot considered the patients' innate neuropathic susceptibility to hysteria incurable.[741] Yet he insisted that this did not apply to hysterical symptoms triggered by various precipitating factors. Having diagnosed Porcz— and Pin— with brachial monoplegia of hysterical nature, Charcot assured his audience that the patients' loss of motor function could be cured through appropriate therapeutic intervention.[742] However, Charcot also pointed out that the standard therapeutic options used to treat hysterical paralysis were not particularly effective. These "empirical measures" included the application of static

734 Charcot and Marie, "Hysteria," 628.

735 Charcot, *Leçons du mardi*, 2:297.

736 For an analysis of the hereditarian views espoused by Lucas and the influence they had on Darwin's theory of evolution, see Noguera-Solano and Ruiz-Gutiérrez, "Darwin and Inheritance." In addition to Lucas, the leading proponents of the French doctrine of hereditary degeneracy were Benedict Morel and Moreau de Tours. For a more general overview of the widespread acceptance the doctrine of hereditarianism had in the late nineteenth-century French medicine and psychiatry, see Dowbiggin, "Degeneration and Hereditarianism."

737 Charcot, "Lecture 7: Contracture of Traumatic Origin," 85.

738 Further elaborating Charcot's views, his assistant Charles Féré developed the notion of the 'neuropathic family.' In this family, all diseases of the nervous system were mutually related through inheritance. For details, see Féré, "La famille névropatique." Féré's work thus cemented and systematised the Salpêtrian stance that not just hysteria but all diseases of the nervous system had a hereditary nature.

739 Charcot, "Lecture 7: Contracture of Traumatic Origin," 85.

740 Goetz, Bonduelle, and Gelfand, *Charcot*, 263.

741 Charcot, "Leçon 14: A propos d'un cas d'hystérie masculine," 306.

742 Charcot, "Lecture 21: Brachial Monoplegia," 288.

electricity and hydrotherapy.[743] Both measures were unspecifically aimed at "rousing [the patients'] vital energies" so that "their beneficial effects are long deferred."[744] Charcot suggested instead that, for the treatment to be effective, it had to be "founded on a physiological basis."[745] He further contended that it was necessary to understand the neurophysiological mechanism through which precipitating factors gave rise to traumatic hysterical paralysis. Only then could an appropriate therapeutic intervention be developed that explicitly targeted this mechanism to reverse its pathological effects.

Hence, in the remainder of his lecture, Charcot set out to elucidate the mechanisms underlying the production of traumatic hysterical paralysis through the experimental use of hypnosis. With this aim in mind, he presented to his audience a young female hysteria patient named Greuz—. Charcot did not offer much detail about Greuz—. He merely stated that, whereas the entire left side of her body was anaesthetic, her right side was free from any detectable disturbances of sensibility. Consequently, Charcot's experimental interventions in this lecture were strictly limited to the healthy right side of Greuz—'s body. Charcot then plunged Greuz— directly into the state of somnambulism by exercising pressure on her eyeballs for a few seconds. Unlike Charcot's hypnotic experiments that we discussed previously,[746] those we will analyse in this section were all performed in the state of somnambulism.

According to the Salpêtrian tripartite classification of the hypnotic states, only somnambulism was characterised by what Charcot designated "a tendency to the reconstitution of the *ego*."[747] As Charcot elaborated, this meant that, although hypnotised subjects lacked consciousness during somnambulism, they could nevertheless exhibit some resistance to the suggestions that the physician imposed on them.[748] In short, contrary to the cataleptic state, hypnotised subjects no longer behaved as mere automatons during somnambulism. Charcot nevertheless insisted that the physician retained unlimited power over somnambulistic subjects since their initial resistance in the end always yielded "to a little insistence."[749] Through targeted use of suggestion, the physician could induce the somnambulistic subject to perform highly complex actions. Although some of the thus induced actions had the appearance of voluntary acts, Charcot emphasised that somnambulistic subjects had no volitional control over their behaviour.[750] From the experimental point of view, a particularly convenient aspect of somnambulism was that all of the hypnotised subjects' senses were fully functional.[751] Hence, during this hypnotic state, Charcot could induce suggestion in various ways, acting either only on one of the patient's senses or on several

743 Charcot, 288.
744 Charcot, 288.
745 Charcot, 288.
746 For my analysis of Charcot's experiments on hysteria patients in the states of hypnotic lethargy and catalepsy, see sections 1.2.1 and 1.2.2.
747 Charcot, "Lecture 21: Brachial Monoplegia," 292 (emphasis in original).
748 Charcot, 292.
749 Charcot, 292.
750 Charcot, 292.
751 Charcot, 292.

simultaneously. Just as importantly, in the state of somnambulism, hypnotised subjects became responsive to direct verbal injunctions.

It was precisely by using a direct verbal injunction that Charcot commenced his hypnotic experiments on Greuz—. "Your right hand is paralysed," he instructed her.[752] Since she, at first, resisted his suggestion, he further insisted: "You cannot move any part of it, it hangs by your side."[753] After a few minutes, Charcot succeeded in paralysing his patient's right arm through such repeated injunctions. Crucially, the paralysis Charcot artificially produced in hypnotised Greuz— by verbal suggestion turned out to have the same clinical features as the paralysis that Porcz— and Pin— spontaneously developed following their respective accidents. These features included the loss of voluntary movement and muscular sense, absolute flaccidity of all the muscles of the arm, reduction of tendon reflexes, as well as cutaneous and deep anaesthesia in all the parts affected by paralysis.[754] There was only one difference between Greuz— and Porcz—. In his case, both motion and sensibility were preserved in the fingers of the affected arm. In her case, the paralysis and the accompanying anaesthesia also extended to the hand and the fingers.

Next, Charcot set out to test if he could use suggestion by speech to produce in Greuz— "a perfect imitation" of the brachial monoplegia that did not extend to the fingers.[755] To this end, he first "deparalysed" Greuz—.[756] In doing so, he demonstrated that to undo the artificially produced paralysis, it sufficed to expose Greuz— to a new suggestion by merely telling her that she could now move her arm. Once the patient regained the normal function of her right arm, Charcot then proceeded to induce in her a new paralysis. This time, however, he deployed a step-by-step procedure. This procedure involved paralysing separate segments of the patient's arm progressively, from the shoulder downwards, through a series of successive suggestions.[757] Using targeted verbal injunctions, Charcot first produced paralysis strictly limited to the patient's shoulder and upper arm. In the second step, he selectively paralysed the patient's elbow, and in the third step, also her wrist. He left out the fingers.

After each of these steps, Greuz— was submitted to tests to assess her loss of motor function. Additional tests were performed to determine the exact distribution of the accompanying cutaneous and deep anaesthesia. In the end, a body map of the patient's anaesthesia was produced that summarised the results obtained across the three successive experimental steps (fig. 1.27).[758] Showing this map to his audience, Charcot drew their attention to its following visual aspects. First, as he explained, the map demonstrated that the artificially produced isolated paralysis of a particular joint (i.e., shoulder, elbow, or wrist) was in each case superimposed by a complete cutaneous

752 Charcot, 294.

753 Charcot, 294.

754 Charcot, 295.

755 Charcot, "Lecture 22: Brachial Monoplegia," 302.

756 Charcot, 296.

757 Charcot, 297.

758 The areas of the map designated with A and A' became anaesthetic in the first experimental step. Similarly, B and B' referred to the anatomical regions that became anaesthetic in the second step. Finally, C and C' denoted the effects obtained in the third step. See fig. 1.24.

and deep anaesthesia in the respective anatomical segment. Charcot particularly emphasised that the limits of anaesthesia in each of these individual segments had a "distinctly circular" shape with which we are by now familiar.[759] Moreover, the circular lines delimiting these anatomical segments were all situated at the right angle to the long axis of the limb. Second, Charcot explicitly invited the members of his audience to visually compare the maps of anaesthesia produced separately for Greuz— (fig. 1.27) and Porcz— (fig. 1.21). In doing so, they could convince themselves that the two maps were mutually "*superposable*."[760] In both maps, the regions affected by the anaesthesia "have the same extent, present the same configuration." [761] Thus, for Charcot, the map of Greuz—'s anaesthesia provided decisive empirical proof that the spontaneously developed hysterical paralysis and its "designedly produced" hypnotic counterpart were "not only comparable to one another but really perfectly identical."[762]

Yet, at this point, the very question Charcot explicitly set out to answer remained open: Through which specific mechanism was a dynamic brain lesion underlying hysterical paralysis produced? In fact, at a superficial glance, it may appear as if Charcot's experiment so far had not only failed to address the question it undertook to answer but also inadvertently raised an additional one: How could two 'perfectly identical' paralyses be produced through two completely different processes? In Greuz—, the paralysis was induced through a verbal injunction during somnambulism. In Porcz— and Pin—, the paralysis arose after a physical accident during which each patient had sustained a minor injury. Unlike Greuz—, both Porcz— and Pin— were thereby fully awake. These two modes of producing paralysis might seem so substantially different that, to an outside observer, their direct experimental comparison could appear to make little sense. However, what such an outside observer may have dismissed as senseless tinkering was a carefully planned preparatory phase for the upcoming key point of Charcot's systematically structured multipart hypnotic experiment.[763]

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759 Charcot, "Lecture 22: Brachial Monoplegia," 298.

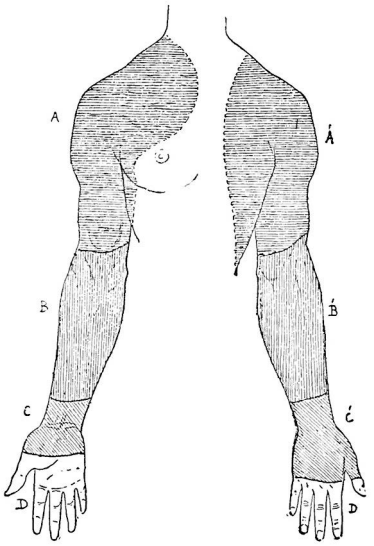
760 Charcot, 302 (emphasis in original).

761 Charcot, 302.

762 Charcot, 304. To substantiate this assertion through additional empirical examples, Charcot presented another female hysteria patient with hemianaesthesia to his audience. Having first hypnotised her, he then used verbal injunctions to produce in this patient the same motor and sensory paralysis of the arm as he had done in Greuz—. Charcot also informed his audience that he had obtained identical results in multiple hysteria patients on his ward using the same procedure. Ibid., 303.

763 As Charcot explicitly stated in the lecture, his experimental investigation of hysterical paralysis of traumatic origin drew on the work of his British colleague, the neurologist John Russell Reynolds. In 1869, Reynolds published an article that dealt with the aetiology, clinical character, and treatment of what he termed paralysis 'dependent on idea.' See Russell Reynolds, "Remarks on Paralysis." According to Russell Reynolds, such paralyses did not arise from organic damage but were caused by 'morbid ideation.' Hence, they were curable. Importantly, Russell Reynolds also insisted that paralyses dependent on ideas had nothing to do with either hysteria or malingering. Ibid., 484. Additionally, he argued that such paralyses were always accompanied by the unimpaired sensibility of the skin, which was in direct opposition to Charcot's cases of hysterical paralysis discussed above. See ibid., 483, 485. Further, as Charcot pointed out, Russell

Figure 1.27. Body map of cutaneous and deep anaesthesia artificially induced in Greuz— through suggestion during somnambulism. From: Charcot, Diseases of the Nervous System, vol. 3, 298, figs. 64 and 65.



Until this point, Charcot focused on demonstrating that he could reproduce all salient features of hysterical paralysis through hypnotic suggestion. Having shown this, he then proceeded to experimentally replicate the precipitating factors that had triggered the onset of paralysis in Porcz— and Pin—. Therefore, in the final stage of his experiment, Charcot no longer used a verbal injunction to induce paralysis of the arm in hypnotised Greuz—. Instead, he now reverted to deploying a targeted physical intervention. This time, he used the palm of his hand to deliver a sharp but not too strong blow to Greuz—'s shoulder. He struck Greuz— on the same shoulder region that Porcz— and Pin— had lightly injured during their accidents. Charcot argued that this latter experimental intervention was "*analogous to that which occasioned the monoplegia both in the case of Pin— and Porcz—, viz. a shock applied on the posterior part of the shoulder.*"[764] Admittedly, the physical blow Porcz— and Pin— had sustained as they fell from a height of about two metres to the ground must have been considerably stronger. Nevertheless, Charcot insisted that, despite such discrepancies in the quantity of their respective

Reynolds could not explain the mechanism underlying either the formation or the disappearance of paralyses dependent on idea. Charcot, "Lecture 21: Brachial Monoplegia," 289. My analysis will show that, instead of merely adopting it, Charcot substantially reworked and expanded Russell Reynold's notion of paralysis dependent on idea.

764 Charcot, "Lecture 22: Brachial Monoplegia," 304 (emphasis in original).

physical impacts, there was no "generic difference" between the blows his two male patients had experienced and the one that Greuz— received during the experiment.[765]

Within a few minutes after Charcot had struck her shoulder, Greuz— developed paralysis of her entire arm. Having examined Greuz—, Charcot was able to confirm that the resulting paralysis had all the clinical features as the one he had previously induced in the same patients through a verbal injunction. Crucially, one of these features also included the distinct distribution of the accompanying cutaneous and deep anaesthesia. Once again, Charcot thus successfully reproduced in his hypnotised patient paralysis 'identical' to those exhibited by Porcz— and Pin—. But this time, the analogy between the artificially induced and spontaneously developed hysterical paralysis was complete because even the experimental mode of production closely replicated the triggering factors to which Porcz— and Pin— had been exposed.

Notably, Charcot's experimental replication had an added benefit. In the short timeframe between the moment he had struck her shoulder and the point at which the paralysis was fully established, Charcot was able to interrogate Greuz— about what she was experiencing. This information was particularly significant because neither Porcz— nor Pin— knew "exactly how the affected member felt at the moment of the accident, nor for some time afterwards."[766] Greuz— reported that she felt "a sensation of enervation, of weight and feebleness" throughout her arm.[767] Moreover, she had a feeling her arm no longer belonged to her, "*that it had become strange to her*."[768] As will soon become apparent, this statement would prove highly significant for Charcot's subsequent interpretation of the mechanism that led to the production of hysterical paralysis.

Drawing together and interpreting various aspects of his multipart experiment on Greuz—, Charcot could finally start to assemble the pieces of the puzzle. In doing so, he managed to posit a distinct neurophysiological mechanism underpinning the formation of both the artificially induced hypnotic and spontaneously developed hysterical paralysis. To begin with, Charcot argued that the paralysis he induced in Greuz— by a verbal injunction and the paralysis he obtained by striking her on the shoulder were both the result of hypnotic suggestion. As discussed previously, Charcot viewed hypnotic suggestion as a fundamentally pathological process. Through this process, an idea that the experimenter had impressed into the subject's mind elicited a reflex response of her brain.[769] Because of "the annihilation of the *ego*" caused by the

765 Charcot, 305.

766 Charcot, 305n2.

767 Charcot, 304. According to the Salpêtrian model, somnambulism was the only stage of hypnosis during which the subjects could communicate with the physician and answer his questions. The Salpêtrians also argued that only during this stage were the hypnotised subjects able to experience sensations and thus verbally describe their experiences. See, e.g. Charcot, "Lecture 21: Brachial Monoplegia," 292. In contrast, during the state of catalepsy, the hypnotised subjects were partly receptive to sensory impressions but remained entirely unaware of these impressions. See section 1.2.2.

768 Charcot, "Lecture 22: Brachial Monoplegia," 304 (emphasis in original).

769 For a detailed discussion of Charcot's views on hypnotic suggestion, see section 1.2.2.

hypnotic state, the subject was unable to suppress this reflex response.[770] Hence, the reflex resulted in a physical action over which the subject had no voluntary control. Charcot claimed that through this process, the idea of motor paralysis he imparted to Greuz— by suggestion led to the formation of an actual physical paralysis. In the first phase of the experiment, he communicated the idea of paralysis directly by telling Greuz— that she could not move her arm. Subsequently, he aroused in her the same idea indirectly by the "traumatic action of a blow on the shoulder, which constituted, as one might say, a veritable *traumatic suggestion*."[771]

It is important to emphasise that throughout his lectures, Charcot consistently used the term trauma in a sense still dominant at the time to denote a physical impact that some external force had on the body.[772] Thus, as my analysis will show, what Charcot meant by a 'traumatic action' in this context referred to purely physical and physiological consequences of the blow he had delivered to his patient's shoulder.[773] To emphasise this point, Charcot explicitly invoked the notion of the "*local shock*" he adopted from the German physician G. H. Groeningen.[774] Groeningen introduced the term "local or peripheral shock" in a monograph published in 1885.[775] According to Groeningen, when some form of a physical "insult" (i.e., trauma) acted on the body, it always caused a local disturbance of physiological functions at the site of the impact.[776] This disturbance, which Groeningen designated as the local shock, was an unavoidable consequence of any trauma, even the one that, like a relatively light blow to the shoulder, did not lead to any actual physical injury. Put simply, in Groeningen's view, the physiological disturbance he referred to as the local shock could either co-occur with an actual physical injury or exist on its own. To underscore this point, Groeningen claimed that a local shock was not caused by any structural damage to the tissue. Instead, the local shock was a direct consequence of the physical commotion and irritation to which the

770 Charcot, "Lecture 22: Brachial Monoplegia," 305 (emphasis in original).

771 Charcot, "Lecture 24: Hysterical Hip-Disease," 335 (emphasis in original).

772 See, e.g., Charcot, *Diseases of the Nervous System*, 1: 41, 73, 87, 107; and 3:26, 32–33, 37, 267. See also Charcot, *Leçons du mardi* 2:534.

773 As pointed out by Ruth Leys, trauma "was originally the term for a surgical wound, conceived on the model of rupture of the skin or protective envelope of the body." Leys, *Trauma: A Genealogy*, 19. Hence, in its original use, trauma was closely linked to the notion of "a physical 'break-in.'" Ibid. This concept was gradually expanded beyond the surgical wound to include other extrinsic agents, such as a more or less violent blow, which did not necessarily rupture the skin. Until the 1870s, the term trauma was used to refer to all "pathological and physical effects" that various extrinsic agents had on the body. Lerner and Micale, "Trauma, Psychiatry, and History," 10. From this point on, the concept of trauma started to slowly shift towards a more psychological meaning that was finally "cemented" by Freud. Leys, *Trauma: A Genealogy*, 18. For a detailed historical study of the concept of trauma, see Fischer-Homberger, *Die traumatische Neurosen*. Notably, Micale, Leys and Fischer-Homberger have all argued that in Charcot's use, the concept of trauma already underwent "a process of psychologization." Micale, "From Medicine to Culture," 123. See also Leys, *Trauma: A Genealogy*, 3–4; and Fischer-Homberger, *Die traumatische Neurosen*, 109–13. However, contrary to their claims, I argue that Charcot's use of the term trauma was firmly embedded in a strictly neurophysiological context.

774 Charcot, "Appendix 1: Hystero-Traumatic Paralysis," 384 (emphasis in original).

775 Groeningen, *Ueber den Schock*, 78.

776 Groeningen, 78.

peripheral nerves were exposed during the physical impact of a trauma.[777] Due to such irritation, the peripheral nerves underwent a temporary decrease in their functioning, which, in turn, resulted in the local shock.

As stated by Groeningen, the symptoms of local shock that arose from a temporary dysfunction of the peripheral nerves at the site of the physical impact consisted of various transitory disturbances of sensibility and movement.[778] If they occurred without a concomitant injury, such sensory and motor disturbances could last from several minutes to an hour. They included the sensations of weight, weakness, and numbness, as well as the feeling that the affected part was either paralysed or even entirely absent. As mentioned earlier, these were precisely the sensations Greuz— had reported experiencing when Charcot asked her how she felt after receiving the blow to the shoulder. Thus, drawing on Groeninger, Charcot designated the particular set of sensations reported by Greuz— as the local shock.[779] Moreover, Charcot explicitly emphasised that, in Greuz—'s case, the local shock was a direct physiological consequence of the traumatic action of the blow delivered to her shoulder. To those familiar with Groeningen's work, the implication of Charcot's statement was clear. The traumatic action of the blow consisted in the physical irritation of the peripheral sensory nerves in the hypnotised patient's arm. Via sensory nerves, this irritation was transmitted to the sensory centres of the patient's brain. Here, it gave rise to the sensations of numbness and feebleness, as well as the impression that her entire limb was absent.

In the next step, Charcot skillfully combined Groeningen's notion of the local shock with his own previously elaborated views on suggestion, understood as a type of cerebral automatism. First, Charcot explained that the sensations entailed in the local shock, which Greuz— experienced upon receiving the blow to the shoulder, called forth an idea of motor and sensory paralysis in her brain.[780] Importantly, Greuz— remained entirely unaware of this idea that arose in a reflex-like manner through a chain of unconscious associations.[781] Up to this point, Greuz—'s physiological responses to the blow she had received were by no means pathological. Instead, similar automatic responses also occurred in healthy individuals.[782] Specifically, an idea of motor and sensory paralysis could unconsciously arise in any individual due to the sensations of numbness and weakness that had been induced by a sufficiently intense contusion of the limb.[783] In a healthy individual, such an idea would pass quickly without being able to produce any lasting physical consequences. However, Greuz— was in the state of

777 Groeningen, 42.

778 Groeningen, 81.

779 Charcot, "Lecture 22: Brachial Monoplegia," 303. See also Charcot, "Lecture 25: Spasmodic Contracture," 344–45; and Charcot, "Appendix 1: Hystero-Traumatic Paralysis," 384.

780 Charcot, "Lecture 22: Brachial Monoplegia," 303. See also Charcot, "Lecture 25: Spasmodic Contracture," 344–45.

781 Greuz— was only aware of the sensations that comprised the local shock but not of the idea of paralysis to which these sensations gave rise through the mechanism of cerebral automatism.

782 Charcot, "Appendix 1: Hystero-Traumatic Paralysis," 384.

783 Charcot, "Lecture 25: Spasmodic Contracture," 344.

hypnotic somnambulism, during which her consciousness was "in abeyance."[784] Hence, the idea called forth by the sensation of limb numbness was free from all control of 'the ego' and could immediately manifest itself in the form of a veritable physical paralysis with concurrent anaesthesia. In other words, in the hypnotised patient, due to the particular nervous state in which she had been placed, the set of sensations induced by the blow (i.e., the local shock) were able to trigger a reflex response of the brain that resulted in a combined motor and sensory paralysis. This was the physiological mechanism that Charcot designated as traumatic suggestion and to which he attributed the formation of paralysis during somnambulism.

Having thus accounted for the production of the artificial limb paralysis in Greuz—, Charcot declared that an analogous physiological process underpinned the formation of hysterical paralysis in Porcz— in Pin—. Yet, two important pieces of the puzzle were still missing. First, as opposed to Greuz—, neither Porcz— nor Pin— were in the state of hypnotic sleep during their respective accidents. This made it difficult to understand how the local shock could lead to paralysis in their cases. Second, whereas Greuz— developed paralysis within minutes after receiving the blow, in neither of the two men did paralysis appear immediately after the accident. In fact, they both initially retained the ability to use their lightly injured arm. It was only several days after the accident that they woke up with arm paralysis.[785] Charcot, however, asserted that the differences between Porcz— and Pin—, on the one hand, and Greuz—, on the other hand, were superficial and could be explained easily. He then proceeded to provide a step-by-step explanation for these apparent differences.

Charcot conjectured that although Porcz— and Pin— had been awake when they received the blow to their shoulders, the accident induced in them a particular "cerebral condition."[786] Charcot designated this condition as the "nervous shock,"[787] deploying the term that had been introduced in the early 1880s by Herbert Page, an English railway company surgeon. Page came up with the notion of the nervous shock while studying cases of functional nervous disturbances similar to hysteria, which were jointly referred to as the railway spine or the railway brain.[788] The symptoms of the railway spine were highly varied. They included different sensory derangements, paralysis, pain in the back, hallucinations, dizziness, loss of memory, mental feebleness and even suicidal thoughts.[789] At the time, such symptoms came to be diagnosed with increasing frequency among victims of railway accidents, especially those who either did not sustain any actual bodily injury or only a very light one.[790] To account for the

784 Charcot, "Lecture 21: Brachial Monoplegia," 292.

785 Charcot, "Lecture 19: Six Cases," 253–54; and Charcot, "Lecture 20: Brachial Monoplegia," 263.

786 Charcot, "Lecture 22: Brachial Monoplegia," 305.

787 Charcot, 305.

788 See Page, *Nervous Shock*.

789 See Page, "Shock from Fright," 1158–59.

790 The railway spine as a medical term was introduced in the 1860s by the London surgeon John Erichsen. For details on Erichsen, see, e.g., Harrington, "Railway Accident," 43–49. Erichsen's initial assumption was that the disorder was due to structural damage to the spinal cord caused by the railway accident. Page vehemently refuted this assumption. See Page, *Nervous Shock*, 58–112. For insightful contemporary studies that trace the gradually changing conception of the railway

discrepancy between the lack of a detectable physical injury and the severity of their symptoms, Page posited that victims of railway accidents suffered from what he termed the nervous shock.[791]

Page defined the nervous shock as "some functional disturbance of the whole nervous balance or tone rather than any structural damage to any organ of the body."[792] Moreover, he stated that "the *primary* seat of this functional disturbance lies in the brain," more specifically "in the centres of conscious volition."[793] According to Page, the nervous shock led to a temporary attenuation or complete annihilation of the higher cerebral faculties. The result was a general weakening of the brain's controlling power over the rest of the body. Crucially, Page argued that in victims of railway collisions, such a dynamic disturbance of the brain was produced "by fright and by fright alone."[794] In individuals who experienced a railway collision, the emotion of extreme fear was inevitably induced by the suddenness of the accident and the imminent danger the accident posed to their lives. In Page's view, such an extreme emotion left a powerful impression on the nervous system, thus disrupting its normal functioning. Importantly, Page asserted that the disruption underpinning the nervous shock was of physiological nature. Yet, at the same time, he explicitly insisted that this disruption was produced by a "purely mental" cause, i.e., a strong emotion.[795] In short, Page emphatically foregrounded the patient's subjective experience of fear as the cause of the nervous shock. As the historian Ralph Harrington has aptly put it, "for Page, the psychological shock suffered by the mind came first, and it produced the physical changes in the nervous system that underlay the subsequent disorders."[796]

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spine in the late-nineteenth-century medicine, see, e.g., Harrington, "Railway Accident"; and Caplan, "Trains and Trauma." Importantly, as explicitly stated in his lecture, Charcot followed with keen interest the work of his "English and American colleagues" on the topic of the railway spine. Charcot, "Lecture 22: Brachial Monoplegia," 305n1. Charcot argued that the disturbances his colleagues referred to as the railway spine and the railway brain were "simply manifestations of hysteria." Charcot, "Lecture 18: Six Cases," 221.

791 See Page, "Shock from Fright," 1157.

792 Page, *Nervous Shock*, 158.

793 Page, 207–8 (emphasis in original). See also Page, "Shock from Fright," 1158. Page did not specify if he regarded the centres of volition to have a designated anatomical location in the brain. Thus, in anatomical terms, it remained unclear what he meant by the primary seat of the functional disturbances underpinning the nervous shock. It should be noted that, in his studies of cerebral localisation, Charcot restricted his empirical efforts to localising only motor and sensory brain centres while steering away from attributing any anatomical seat to higher functions such as the ego, volition, or consciousness. See Goetz, Bonduelle and Gelfand, *Charcot*, 125–34. It, therefore, seems that Charcot subscribed to the view explicitly espoused by David Ferrier: "Intelligence and will have no local habitation distinct from the sensory and motor substrata of the cortex generally. There are centres for special forms of sensation and ideation, and centres for special motor activities and acquisitions, in response to and in association with the activity of sensory centres; and these in their respective cohesions, actions, and interactions form the substrata of mental operations in all their aspects and all then range." Ferrier, *Functions of the Brain*, 2nd ed., 467.

794 Page, *Nervous Shock*, 162.

795 Page, 163.

796 Harrington, "Railway Accident," 51.

But, as I intend to show, in adopting Page's notion of the nervous shock, Charcot significantly expanded and even modified it. First, Charcot contended that the nervous shock elicited by a strong emotion during an accident was, at the physiological level, equivalent to the cerebral condition artificially induced through hypnosis.[797] According to Charcot, both the nervous shock and hypnotic somnambulism were characterised by "the obnubilation [i.e., clouding] of consciousness" and "the dissociation of the *ego*."[798] For this reason, in both of these conditions, "the *will*, or the *judgment*, is more or less suppressed or obscured, and suggestions become easy."[799] Hence, in Charcot's view, the salient point about the nervous shock was that it made hypnotic suggestion possible even during the waking state. It is worth mentioning that, in his later work, Page approvingly quoted Charcot on this point. Even more to the point, Page explicitly credited Charcot for being the first to recognise that "the phenomena of hypnotism are practically identical" with the state of the nervous shock.[800]

Second, unlike Page, Charcot insisted that fear or a similar strong emotion elicited by a physical accident could produce nervous shock only in predisposed neuropathic individuals.[801] Put differently, in Charcot's reinterpretation, the intense emotion served merely as a triggering factor that activated the subject's inherited neurological deficit, which until that point had remained latent. Third, by referencing Darwin, Charcot contended that "a sudden and violent emotion," such as fear, could produce a limb paralysis "without departing, so to speak, from physiological conditions."[802] Hence, contrary to Page, who viewed fear as a purely mental factor, Charcot subscribed instead to a decidedly physiological interpretation of emotions. As discussed above, Page foregrounded the patient's subjective, internal experience of a particular emotion that arose in the context of the accident. In contrast, Charcot argued that the bodily

797 Charcot, "Lecture 22: Brachial Monoplegia," 305.

798 Charcot, "Appendix 1: Hystero-Traumatic Paralysis," 383 (emphasis in original).

799 Charcot, "Lecture 24: Hysterical Hip-Disease," 335 (emphasis in original).

800 Page, "Shock from Fright," 1159.

801 Charcot, "Lecture 22: Brachial Monoplegia," 305; and Charcot, "Lecture 25: Spasmodic Contracture," 344.

802 Charcot, "Appendix 1: Hystero-Traumatic Paralysis," 386. According to Darwin, emotions and their expressions were a consequence of "the direct action of the nervous system." Darwin, *Expression*, 29. Additionally, in his description of the emotion of fear, Darwin focused exclusively on enumeration underlying physiological responses. These included the arousal of the senses of sight and hearing, "disturbed action of the heart," hurried breathing, dry mouth, and "the trembling of all the muscles of the body." Darwin, 290–91. Charcot's contemporary, the English psychiatrist Henry Maudsley was another influential proponent of the view that emotions were primarily physical phenomena. For his detailed analysis of emotions, see Maudsley, *Physiology of Mind*, 348–408. Furthermore, writing in 1884, William James contradicted the generally held view that "the mental perception of some fact excites the mental affection called the emotion, and that this latter state of mind gives rise to the bodily expression." James, "What is an Emotion?," 247. Instead, James proposed that "*the bodily changes follow directly the PERCEPTION of the exciting fact, and that our feeling of the same changes as they occur IS the emotion*." James, 247 (emphasis in original). In this view, emotions were first and foremost physiological reactions to external stimuli, whereas the subjective experience of these physiological reactions was secondary. Although Charcot did not explicitly quote James, his above statement suggests that he shared this view.

processes underpinning a particular emotion gave rise to the nervous shock.[803] To put it more clearly, from Charcot's point of view, what mattered was not how the patients felt during the accident but how their bodies responded to an emotionally charged context.

Next, by aptly combining the different notions of shock he had adopted from his German and British colleagues, Charcot could finally explain how Porcz— and Pin— developed hysterical paralysis in the aftermath of their accidents. As Charcot specified, the accident they had experienced produced in both Porcz— and Pin— two distinct yet simultaneous physiological effects. On the one hand, the blow to the shoulder resulted in the local or traumatic shock. As discussed above, this type of shock consisted in temporary motor and sensory disturbances in the contused limb. Charcot emphasises that the resulting local sensations of weakness and numbness, which had arisen from the local shock, were nothing else but a form of a transient "rudimentary paralysis."[804] This, in turn, meant that the idea of limb paralysis that the local shock called forth was merely "the memory of sensory impressions" of weakness and numbness induced by the blow.[805] On the other hand, in both patients, due to their hereditary predisposition, the accident additionally induced the nervous shock. This other type of shock was triggered by the physiological response of fear that arose in each patient during the accident.

At this point, it is important to highlight two aspects of Charcot's explanation. First, for Charcot, the local (i.e., traumatic) and the nervous shock were two mutually independent yet co-occurring physiological consequences of the accident. Second, in Charcot's view, the joint occurrence of the traumatic and the nervous shock was crucial for the production of hysterical paralysis.[806] As we have seen, the sensation of numbness resulting from the local shock was a necessary point of departure for the idea of paralysis. Yet, this idea could lead to an actual physical paralysis only in a subject who was in the state of nervous shock and whose volitional control (i.e., the ego) was thus suppressed.

Moreover, Charcot argued that the state of dazed consciousness entailed in the nervous shock did not end immediately after the accident but extended "for some days afterwards."[807] During this period, the idea of paralysis, which had originated from the local shock, underwent further elaboration through the process of unconscious cerebration analogous to the one happening during hypnosis.[808] One key difference, however, was that in cases of traumatic hysterical paralysis, this cerebral reflex was not set in operation intentionally through the external influence of a hypnotist. Instead, the cerebral reflex was set off by sensory impressions that "developed spontaneously or accidentally in the patient himself."[809] To emphasise this difference, Charcot designated the latter type of unconscious cerebration as autosuggestion.

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803 In one of his subsequent case studies, Charcot conjectured that anger could also produce a nervous shock. See Charcot, *Leçons du mardi*, vol. 1, 2nd ed., 98.

804 Charcot, "Lecture 25: Spasmodic Contracture," 345; and Charcot and Marie, "Hysteria," 633.

805 Charcot, "Appendix 2: Muscular Sense," 398.

806 Charcot, "Appendix 1: Hystero-Traumatic Paralysis," 385.

807 Charcot, "Lecture 22: Brachial Monoplegia," 305n2.

808 Charcot, "Lecture 25: Spasmodic Contracture," 345.

809 Charcot, "Appendix 1: Hystero-Traumatic Paralysis," 384.

Another significant difference was that autosuggestion, unlike hypnotic suggestion, was a considerably slower process that required "a period of incubation" of several days for a complete paralysis to establish itself.[810] During this period, the sensations provoked by the local shock, which initially represented merely "a sketch, a rudiment, or germ" of a paralysis, gradually developed into what Charcot referred to as a full-blown 'fixed idea' of paralysis.[811] Charcot apparently viewed this 'fixation' as a physiological process that entailed some unknown modification of the nerve cells. Specifically, he claimed that, as a result of this process, the idea of paralysis became "installed in the brain."[812] Once installed, the idea of paralysis took "sole possession" of the patient's mind.[813] Only at this point did the fully established fixed idea acquire "sufficient domination to realise itself objectively in the form of paralysis."[814] In designating the idea of paralyses as 'fixed,' Charcot underscored two of its aspects: first, the pathological dominance that this idea had acquired through the process of autosuggestion; and second, the hypothesised physiological inscription of this idea into the cerebral centres.[815] Charcot insisted that the patient only became aware of the resulting paralysis. In contrast, the entire process underlying the formation of paralysis, including the fixed idea itself, remained entirely unconscious. Charcot thus declared the formation of hysterical paralysis to be "a sort of reflex action, in which the centre of a diastaltic arc is represented by regions of the grey cortex."[816]

Having thus explained why the paralysis did not appear immediately after the accident but only a few days later, Charcot then clarified the mechanism through which the fixed idea of motor weakness produced an actual physical paralysis. Charcot posited that, once it had obtained sufficient dominance, the "fixed idea of motor weakness"

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810 Charcot, 385.

811 Charcot, "Lecture 25: Spasmodic Contracture," 345.

812 Charcot, "Lecture 22: Brachial Monoplegia," 305. Similarly, Carpenter and Ferrier also argued that 'new' ideas stemming from recently experienced sensory impressions needed to physiologically 'imprint' themselves in the sensory centres of the brain. See Ferrier, *Functions of the Brain*, 258–59; and Carpenter, *Mental Physiology*, 470.

813 Charcot, "Lecture 22: Brachial Monoplegia," 305.

814 Charcot, 305. According to Jan Goldstein, the term 'fixed idea' (i.e., idée fixe) was "probably coined by the phrenologists Gall and Spurzheimer in connection with Esquirol's delineation of monomania." Goldstein, *Console and Classify*, 155n21. See also Goldstein, 268. Esquirol was a French psychiatrist who worked at the Salpêtrière in the early nineteenth century. In 1810, Esquirol introduced the diagnostic category of monomania to designate a form of partial insanity that comprised a pathological preoccupation with a single *idée fixe* in an otherwise sound mind. Goldstein, 155–56. In Esquirol's definition, a patient suffering from monomania was well aware of his fixed idea. Several decades later, William Carpenter significantly expanded the original notion of the fixed idea. Carpenter argued that fixed or dominant ideas could also occur in healthy individuals. He also suggested that fixed ideas were especially prevalent during hypnotic states. See Carpenter, *Mental Physiology*, 555–56. Importantly, in Carpenter's reinterpretation, an individual could become 'possessed' by fixed ideas while at the same time remaining entirely unaware of them. Carpenter, 281–82. It appears to me that Charcot's use of the term fixed idea in his research on hysterical paralysis of traumatic origin clearly reflects Carpenter's influence.

815 As we will see shortly, such physiological inscription did not imply any structural modification of the cerebral centres themselves but a change in their mutual interactions.

816 Charcot, "Appendix 1: Hystero-Traumatic Paralysis," 387n.

started to exercise "an inhibitory action over the cortical motor centres."[817] Quoting Wilhelm Wundt, Alexander Bain, David Ferrier, Herbert Spencer, Théodule Ribot, and Henry Maudsley, Charcot argued that to perform a voluntary movement, the subject first had to form an idea or "a mental representation, no matter how summary or rudimentary it may be of the movement to be executed."[818]

Drawing in particular on Wundt, Charcot asserted that the formation of the idea of movement took place in the motor centres of the brain. This idea was "chiefly constituted" by the "nervous discharge" (i.e., the innervation) and was "indispensable to call voluntary movement into operation."[819] Having originated in the "organic substratum" of the motor centres, the nervous current was then directed towards muscles, inducing their coordinated contractions.[820] To further emphasise this point, Charcot additionally quoted Herbert Spencer's view that the mental representation or the idea of movement "is nothing else than the nascent excitation of all the nerves participating" in the actual execution of that voluntary movement.[821] However, as Charcot explained, in Porcz— and Pin—, the idea of the absence of movement had through subconscious cerebration become so dominant (i.e., fixed) as to render the normal formation of the idea of movement in the cortical motor centres impossible.[822] The result was the functional inhibition of the cerebral motor centres, which, in turn, manifested itself in the form of an "objective" physical paralysis.[823]

It is worth reminding ourselves that for Charcot, the fixed idea of paralysis consisted of revived sensations of the previously experienced transitory motor weakness elicited by the local shock during the physical accident. Consequently, in this interpretational framework, the fixed idea of paralysis was constituted by a nervous current at the physiological level. Due to autosuggestion, this nervous current became so morbidly intense as to actually "re-induce the peripheral [sensory] impression" of motor weakness long after the initial event that gave rise to this impression had passed.[824] Quoting

817 Charcot, "Lecture 22: Brachial Monoplegia," 310.

818 Charcot, 309n1. In support of this view, Charcot also quoted James Mill, William Hamilton, Theodor Meynert, Johannes Müller, Salomon Stricker, and Hughlings Jackson. Ibid. But Charcot also admitted that some of the leading neurologists of the time, such as Charlton Bastian, did not share this view. Bastian contested the claim that the formation of the idea of movement took place in the cortical motor centres. Instead, he denied the existence of motor centres and conjectured that voluntary movement was initiated in the sensory centres of the brain. For Charcot's discussion of his colleagues' divergent views on the cerebral basis of voluntary movement, see Charcot, "Appendix 2: Muscular Sense," 396–400. See also Ribot, *Diseases of the Will*, 127–28. For an elegantly written overview of various nineteenth-century theories of the neurophysiological basis of voluntary movement, see Jeannerod, *Brain Machine*, 34–94.

819 Charcot, "Appendix 2: Muscular Sense," 395.

820 Charcot, 395.

821 Charcot, 397.

822 Charcot, "Lecture 22: Brachial Monoplegia," 310.

823 Charcot, 310.

824 Ferrier, *Functions of the Brain*, 259. According to Spencer, Bain, and Ferrier, an idea consisted of "a faint revivification" of previously experienced sensations in the brain's sensory centres. Ibid., 258. Under normal conditions, the "molecular thrill" underlying this revivification was not so strong as to "extend to the periphery" and thus re-induce the actual sensations. Ibid., 258–59. Only fixed

Ferrier, Charcot conjectured that such revival of sensory impressions necessarily took place in the sensory centres of the brain.[825] The crucial point was that, according to Ferrier and Charcot, the execution of all voluntary movements required hierarchical cooperation between the cortical motor and sensory centres. The generation of the motor idea necessary to initiate a voluntary movement took place in the motor centres. Yet the normal accomplishment of the initiated movement required additional coordination with visual sensations and various sensory impressions furnished by the muscular sense.[826] The execution of voluntary movements, therefore, depended on "the organic nexuses [that] are established between the sensory and motor centres."[827] However, due to the organic nexuses that connected them, a faulty nervous discharge in the sensory centres could impinge on the normal excitatory activity of the motor centres, thus causing their inhibition.

Drawing all these elements together, we can now surmise that the inhibition of the motor centres, which in Charcot's view underpinned hysterical paralysis, amounted to a functional disturbance of the excitatory activity in these centres. In other words, the presence of one abnormally strong nervous current (i.e., the fixed sensory idea of paralysis) blocked the formation of another nervous current (i.e., the idea of voluntary movement to be executed). Having thus lost the ability to form the idea of movement in the motor centres of their brain, the patients could no longer execute voluntary movements. In short, in Charcot's interpretation, the underlying cause of hysterical paralysis was a functional disruption in the hierarchical top-down neural processing of voluntary movement formation.

There are two aspects of Charcot's proposed mechanism to which I want to draw particular attention. First, my analysis has foregrounded that Charcot's account of the pathogenesis of traumatic hysterical paralysis remained firmly grounded in a purely somatic framework. To develop this account, Charcot productively combined multiple neurophysiological concepts and theories of his time. These, as we have seen, included the concept of cerebral reflexes, the theory of associationism, the doctrine of hereditary nervous defects, the disparate notions of local and nervous shock and, crucially, the paradigm of cerebral localisation. Just as importantly, I have demonstrated that even

ideas could re-induce peripheral sensations despite the absence of actual sensory stimuli. Ibid., 259.

825 Charcot, "Appendix 2: Muscular Sense," 398.

826 Charcot, 395, 400. In Charcot's view, both the visual image of movement and other sensory impressions intervened "only in a secondary, though very effectual fashion, in order to complete, direct, and so to speak to perfect the movement which is already in process of execution." Ibid., 395.

827 Ferrier, *Functions of the Brain*, 265. Ferrier also argued that precisely because the execution of voluntary movements depended on the establishment of such nexuses, each voluntary movement had to be acquired through repetition and learning. "The individual activity of the various specially differentiated motor centres having once been fairly established at first in response to particular sensations and desires, voluntary acquisition proceeds apace, the centres being free to form new associations and become the means of realisation in action of all the varied simple and complex impulses of the sensory centres. The associating fibres between the one motor centre and the various sensory centres may thus become innumerable" and vary depending on "the degree of complexity and intricacy of the movements." Ibid.

when discussing the roles of mental processes, such as the formation of ideas, volition, unconscious cerebration, and emotional responses, Charcot's interpretation was strictly framed in neurophysiological terms. For the remainder of his medical career, Charcot never deflected from this view. In his subsequent lectures, Charcot continued to insist that all mental operations underpinning the production of hysterical symptoms had their seat in the cerebral cortex and were thus physiologically determined.[828]

Second, it is important to emphasise that by attributing hysterical paralysis to the inhibition of the cortical motor centres, Charcot finally managed to specify the nature of the hypothesised functional brain lesion. As discussed previously, while diagnosing Porcz— with hysterical monoplegia, Charcot already posited the existence of a functional brain lesion, which he then tentatively localised in the motor and sensory centres of the brain. However, at first, he had been unable to define the nature of this lesion, apart from stating that it was neither structural nor permanent. As detailed above, it was only in his third and final lecture on hysterical monoplegia that Charcot causally linked his two patients' arm paralysis to the functional inhibition of their cortical motor centres. The implication of this statement was clear—in hysterical paralysis, the underlying dynamic brain lesion consisted in the functional inhibition of the cerebral motor centres. As my analysis has shown, this inhibition, in turn, comprised what can be termed an excitatory defect, i.e., the inability of the centres to produce a nervous discharge necessary for initiating a voluntary movement.

Moreover, although Charcot did not explicitly state this, it is safe to assume that the hypothesised dynamic lesion of the cerebral sensory centres to which he attributed the paralysed patients' accompanying anaesthesia entailed a similar functional inhibition. Drawing on Charcot's previous statements,[829] we can, therefore, presume that in anaesthesia, the inhibition of the sensory centres consisted in the inability of these centres to register the incoming nervous current delivered by the peripheral afferent nerves. At this point, we also need to recall our discussion of Charcot's earlier hypnotic experiments, in which he linked hysterical contractures to a hypothesised dynamic lesion of the nervous centres in the spinal cord. As analysed previously, Charcot had argued that the hypothesised dynamic lesion which gave rise to hysterical contractures consisted in the functional overexcitability of the spinal nervous centres.[830]

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828 Charcot, *Leçons du mardi*, vol. 1, 2nd ed., 281. See also ibid., 99–100, 347; and Charcot and Marie, "Hysteria," 633.

829 As discussed previously, Charcot argued that under normal conditions, sensory impressions were transmitted via afferent nerves to the "cortical sensitive centres, where their ideal recall can take place." Charcot, "Appendix 2: Muscular Sense," 395.

830 For a detailed discussion, see section 1.2.1. In the late 1880s, Charcot expanded his initial interpretation of the dynamic lesion underpinning the formation of hysterical contractures. He continued to attribute the formation of contractures to the overexcitability of the spinal nervous centres. Yet, he now argued that the motor centres in the spinal cord were connected via the pyramidal tract to the motor centres in the cerebral cortex. Charcot, "L'hypnotisme en thérapeutique," 468–69. He also conjectured that the higher-order cerebral motor centres controlled the reflex activity of the spinal centres by sending them either excitatory or inhibitory impulses via the pyramidal tract. Ibid. He further posited that a dynamic disturbance of the cerebral motor centres or the pyramidal tract suppressed their control over the spinal reflexes. The result was the hyperexcitability of the spinal motor centres, which, in turn, led to the formation

Taken together, all these different elements suggest that, by the mid-1880s, Charcot came to attribute multiple hysterical symptoms to functional disturbances of designated nervous centres that were localised throughout the spinal cord or in the brain cortex. The hypothesised disturbances of function that underpinned hysteria entailed either a pathologically excessive excitatory activity of these centres or their abnormal inactivity. That is, in Charcot's view, such dynamic lesions were equivalent to a faulty inhibition or a faulty disinhibition of the specialised nervous centres, which under normal conditions presided over a particular motor or sensory function that was disturbed in a given hysterical symptom. As my foregoing discussion has demonstrated, Charcot regarded traumatic autosuggestion to be the underlying neurophysiological mechanism that led to the formation of such dynamic lesions in predisposed individuals with innate weakness of the nervous system.

By the end of the 1880s, Charcot gradually expanded this interpretation to other hysterical symptoms. These included different forms of arthralgia (joint pain), mutism (speech loss), astasia-abasia (inability to walk or stand), and hysterical attacks.[831] In each case, Charcot argued that autosuggestion had given rise to a functional lesion of a specialised nervous centre located "in the grey cortex of the cerebral hemispheres."[832] For example, in a lecture he gave in 1890, Charcot attributed hysterical attacks to a transitory 'irritative' lesion of the cortical area called the paracentral lobule.[833] In effect, Charcot thus established the functional brain lesion, understood as a disturbance in the excitatory activity of a given nervous centre, as the underlying cause of all hysterical symptoms. The principle underpinning the formation of such a lesion always remained the same—the aberrant cerebral reflex (i.e., autosuggestion) triggered by some external provoking agent. What changed from symptom to symptom was the hypothesised anatomical location of the resulting lesion.

One final aspect of Charcot's lectures on brachial monoplegia deserves our close inspection. Having come up with a hypothesis about the neurophysiological mechanism through which traumatic hysterical paralysis was produced, Charcot then drew on this mechanism to develop a targeted treatment. He argued that to "deparalyse" Porcz— and Pin—, it was merely necessary to find a way to disinhibit their cerebral motor centres.[834] He further claimed that this could be achieved by reviving in these centres the formation of the idea "which is a necessary preliminary to the motor movement."[835] With this

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of contractures. Ibid., 469. Hence, through this subsequent reinterpretation, Charcot linked the formation of hysterical contractures to combined dynamic lesions that simultaneously affected both the lower-order spinal and the higher-order cerebral motor centres.

831 See Charcot, "Lecture 24: Hip-Disease," 334–36; Charcot, "Lecture 26: Mutism," 372–73; Charcot, *Leçons du mardi*, 2:375–77; and Charcot, "Leçon 14: A propos d'un cas d'hystérie masculine," 304–6.

832 Charcot, "Lecture 26: Mutism," 373.

833 Charcot, "Leçon 24: Epilepsie partielle crurale," 8–9; and Charcot, "Appendice 2: Hémianesthésie hystérique," 465–66. Using the anatomo-clinical method, the Salpêtrians discovered in 1883 that the paracentral lobule in each cerebral hemisphere presided over the movement of the lower limb on the contralateral side of the body. Charcot, "Leçon 24: Epilepsie partielle crurale," 6.

834 Charcot, "Lecture 22: Brachial Monoplegia," 296.

835 Charcot, 310. Charcot interchangeably referred to the 'idea of movement' as the 'motor image' or as the 'mental representation' of movement. See ibid., 309.

aim in mind, Charcot devised a deceivingly simple physical exercise with a mechanical device called the dynamometer. This small hand-held device was used routinely at the Salpêtrière to measure the strength of the patients' grip and thus quantify the loss of their muscular force due to paralysis.[836] Holding the dynamometer in one hand and squeezing its metal handles with his fingers, the patient caused the needle of the instrument to change its position in relation to an integrated numerical scale.[837] The deflection of the needle indicated the amount of muscular force that the patient had exerted. The units of measurement were kilograms.

Charcot's novel therapy consisted in placing the dynamometer in the patient's affected hand and instructing him to squeeze it with all his power. The patient was additionally asked to observe his hand during the exercise, paying particular attention to the movement of the instrument's needle he was causing through squeezing. In submitting Pin— and Porcz— to this exercise, Charcot made use of the fact that in both patients, some rudimentary voluntary movement of fingers "subsisted, though in a feeble degree."[838] Due to the feebleness of their fingers, the patients' results did not seem very promising at the commencement of the treatment. Despite this, Pin— and Porcz— were required to regularly repeat the dynamometric exercise every hour of the day for several weeks. Each time they performed the exercise, both patients were expressly encouraged to focus on progressively increasing the maximum deflection of the instrument's needle that they could obtain.

In this exercise, the changing position of the needle in relation to the numerical scale of the dynamometer served a twofold function. On the one hand, it permitted the Salpêtrians to quantify the maximum muscular force the patients could achieve on each trial. On the other hand, the changing position of the needle also gave the patient real-time visual feedback during the exercise, enabling him to adjust the strength of his grip accordingly. In fact, the visual guidance provided by the instrument's needle had a crucial role in the therapy. This is perhaps best illustrated by the fact that, when asked to perform the same exercise with his eyes closed, Pin— could attain only a fraction of the muscular force compared to when his eyes were open and closely focused on the changing position of the needle.[839] However, although achieving a steady increase in the maximum muscular force was the explicit aim of the dynamometric exercise, Charcot cautioned against any overzealousness. He asserted that, while the exercise had to be performed regularly, it was paramount not to repeat it too frequently or with too

836 See Tourette, *Traité clinique*, 145, 448.

837 In the centre of the instrument was a metal spring, which was attached to the needle. By squeezing the handles of the instrument, the patient pressed the spring, thus causing the needle to change its position. When the pressure was released, the needle returned to its original position. For a detailed description of various models of hand-held dynamometers used in clinical medicine in the second half of the nineteenth century, see Nicola and Vobořil "Collin Dynamometer," 179–202. As stated by Tourette, the Salpetrians used the hand-held dynamometer designed by the French physician Victor Burq. See Tourette, *Traité clinique*, 448.

838 Charcot, "Lecture 22: Brachial Monoplegia," 309. Since the exercise involved squeezing the instrument, this treatment could not be applied to a patient with complete arm paralysis that also affected the fingers.

839 Charcot, 310.

much strain. He warned that overstraining would necessarily result in fatigue "and thus retard the expected results," leading to a temporary decline in the patients' muscular force.[840]

As Charcot explained to his audience, the goal of this simple treatment was to induce the patients to repeatedly and methodically practise forming the mental representation (i.e., the idea) of the hand movement required to perform the dynamometric exercise.[841] Such daily interventions, which had to be performed with unfailing regularity over weeks or even months, were meant to reactivate the patients' cerebral motor centres. The effectiveness of the therapy hinged entirely on the patients' active participation. For this reason, as Charcot emphasised, it was crucial to continually encourage the patients by "affirming in a positive manner" that their paralysis would "certainly be cured" by the treatment.[842]

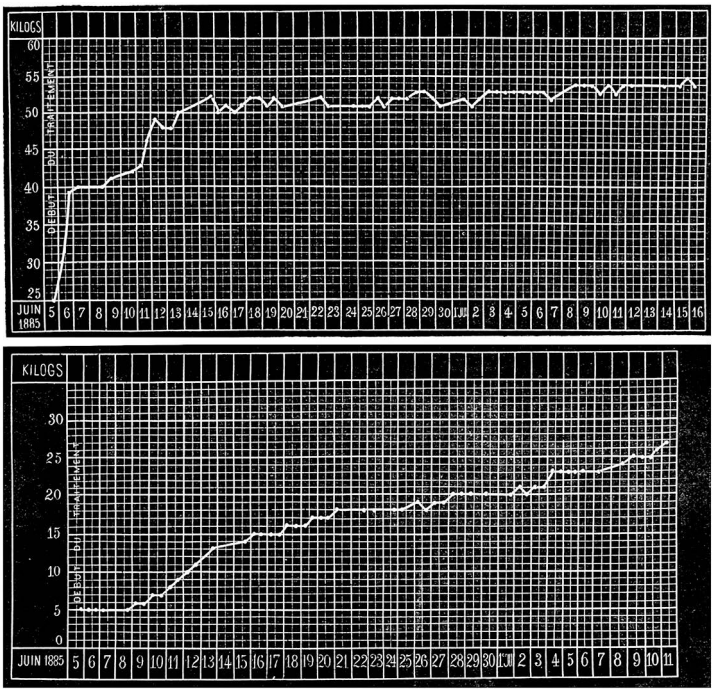
Charcot suggested that additional therapeutic interventions such as massage, electrical stimulation, hydrotherapy, and passive movements of the paralysed limb could all be employed as supportive measures, especially in the early phases of the treatment.[843] Nevertheless, the central part of the therapy was that the patients had to actively initiate voluntary movement under controlled conditions and then closely observe the results of their effort. In doing so, the patients were repeatedly generating "the active nervous current" in the motor centres of their brain and thus gradually suppressing the inhibitory power that the fixed idea of paralysis exercised over these centres.[844] Through such methodical exercise, the patients were slowly re-educating their brains how to execute voluntary movements by re-establishing the normal excitability in the cerebral motor centres.[845] In other words, the explicit aim of the exercise was to retrain the patient's disrupted top-down motor control. Consequently, no passive external intervention could displace the patient's self-initiated performance of movements, which was the key element of the therapy.

To monitor and quantify the effects of the dynamometric therapy, the Salpêtrians registered twice a day the maximum muscular force that Pin— and Porcz— managed to obtain over a period of approximately forty days. The numerical data were then visualised in the form of respective line graphs, which separately charted each patient's progress across this period (fig. 1.28). The resulting ascending lines demonstrated that each patient's muscular force in the affected arm increased considerably over the course of the therapy. This meant that both Pin— and Porcz— were gradually regaining the ability to perform simple voluntary movements with their paralysed hand. Yet, as Charcot admitted, the zigzag shape of the ascending lines also disclosed that the patients' recovery was slow and that, despite daily practice, the increase in the muscular force could stagnate for several days in a row. Nevertheless, the line graphs

840 Charcot, 309.
841 Charcot, 310.
842 Charcot, 308.
843 Charcot, 310.
844 Charcot, 307n2.
845 Charcot, *Leçons du mardi*, 2:377, 380.

provided convincing visual evidence that the progress was "very real" and that the therapy positively affected both patients.[846]

Figure 1.28. Line graphs showing the results of the dynamometric therapy in Pin— (above) and Porcz— (below). Above: daily changes in the maximum dynamometric force obtained by Pin— from June 5 to July 16, 1885. Below: daily changes in the maximum dynamometric force obtained by Porcz— from June 5 to July 11, 1885. From: Charcot, Diseases of the Nervous System, vol. 3, 312, fig. 74; and 313, fig. 75.



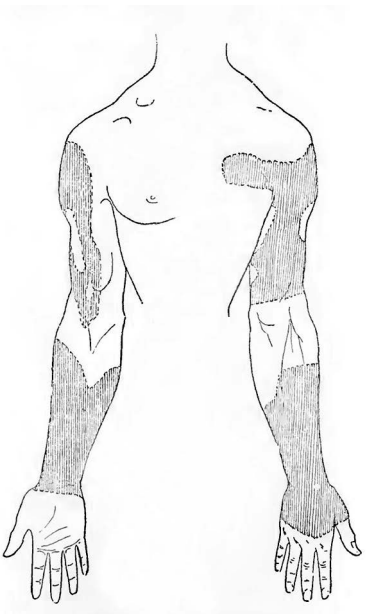
Moreover, Charcot also presented to his audience a body map that visualised the new distribution of Porcz—'s anaesthesia approximately one month after the commencement of the dynamometric therapy (fig. 1.29).[847] Unlike the line graphs, however, the body map did not visualise the temporal progress of the therapy. Instead, it presented what could be called a 'snapshot' of the anatomical distribution of the patient's cutaneous and deep anaesthesia on the day of the measurement. Hence, the clinical meaning of this image had to be established through visual comparison with the body map of the patient's anaesthesia, which had been produced before the therapy started (see fig. 1.21). The comparison of these two maps disclosed that Porcz— had

846 Charcot, "Lecture 22: Brachial Monoplegia," 314.

847 As Charcot informed his audience, during the therapy, the changes in each patient's distribution of the anaesthesia were "noted daily." Charcot, 315.

regained sensibility in the shoulder and the armpit, parts of the elbow, and the upper arm. The changes across the two body maps made apparent that the outcome of the therapy was not limited to the partial restoration of the patient's voluntary movement. The therapy also simultaneously led to a partial restoration of the patient's cutaneous and deep sensibility. The body maps thus indicated that the therapy modified the patient's brain dynamics by weakening the inhibitory effects that the fixed idea of paralysis exercised over the cortical motor and sensory centres. As a consequence of the dynamometric exercise, the fixed idea became less effective in blocking the formation of the idea of movement in the cortical motor centres. In parallel, due to the dynamometric exercise, the fixed idea also became less effective in blocking the formation of normal sensations in the cortical sensory centres.

Figure 1.29. Body map showing the distribution of Porcz—'s anaesthesia after one month of dynamometric therapy. From: Charcot, Diseases of the Nervous System, vol. 3, 311, figs. 72 and 73.



Importantly, when Charcot presented to his audience the images that so effectively charted the two patients' clinical improvement, neither Porcz— nor Pin— were entirely cured of their symptoms. This, I suggest, was all the more reason why Charcot needed the images to prove that his simple therapy had indeed resulted in measurable clinical improvements. Yet, apart from providing empirical proof for the efficacy of Charcot's therapy, the three images had an additional, and perhaps even more far-reaching, epistemic function. As Charcot himself stated, he developed the dynamometric therapy

to target the very mechanism that, according to his hypothesis, underpinned the formation of hysterical paralysis. The unspoken implication of this statement was that, if the therapy worked, Charcot's conjecture about the mechanism underlying hysterical paralysis must be correct.

Therefore, I argue that the images visualising the two patients' therapy-induced partial recovery first and foremost served as indirect visual proof for the validity of Charcot's conjecture about the nature of the underlying dynamic lesion in cases of hysterical paralysis. Although only indirectly, these images effectively reinforced Charcot's claim that the arm paralysis in his two male patients arose from a potentially reversible functional inhibition of their cerebral motor and sensory centres. At this point, we might remind ourselves that the body maps visualising the anatomical distributions of anaesthesia in Porcz—, Pin—, and Greuz— provided the starting point for Charcot as he set out to develop his hypothesis about the mechanism underpinning the production of traumatic hysterical paralysis. As we have seen, based on these maps, Charcot posited the hypothesis about the nature and the anatomical location of the dynamic lesion that caused hysterical paralysis. Fittingly, Charcot once again turned to images to provide indirect empirical evidence for the validity of his hypothesis that causally linked hysterical paralysis accompanied by anaesthesia to reversible functional disturbances of the motor and sensory cerebral centres.

In subsequent years, Charcot continued to expand his hypothesis. He declared that the extent to which the accompanying anaesthesia subsided was the only reliable indicator of a hysteria patient's recovery from hysterical paralysis. Specifically, he argued that even the patients who managed to regain voluntary movement in their previously paralysed limbs through dynamometric therapy should not be regarded as healed as long as the accompanying disturbances of sensibility persisted.[848] If body maps of such patients continued to disclose remaining patches of anaesthesia, the recovery was only partial and temporary. In such cases, hysterical paralysis merely became latent and could reappear in its full intensity on the slightest occasion.[849] For Charcot, only those patients whose body maps showed no remaining disturbances of either cutaneous or deep sensibility were truly cured of hysterical paralysis. Hence, not the apparent re-establishment of the motor function but the body maps of the accompanying anaesthesia became the visual arbiters of hysteria patients' actual recovery. We can thus surmise that Charcot came to regard the body maps of anaesthesia as the most reliable indirect measure of the presence and intensity of the underlying functional brain lesion causing his patients' hysterical symptoms. As a result, the epistemic function of this type of image was further expanded. In addition to their already established diagnostic function,[850] the body maps of anaesthesia also acquired a prognostic function, as they allowed Charcot to assess whether a patient's recovery from hysterical paralysis was merely temporary or not.

848 Charcot, *Leçons du mardi*, vol. 1, 2nd ed., 283. See also ibid., 284, fig. 39; and 285, fig. 40.

849 Charcot, 288–89. See also Charcot, *Clinique des maladies*, 1:45.

850 For a detailed discussion, see section 1.3.1.

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To summarise, I have demonstrated in this chapter that Charcot's decades-long image-based hysteria research was a complex and highly systematic scientific endeavour that generated novel insights into the nature of heterogeneous hysterical symptoms. Far from merely staging "dazzling displays" of his patients,[851] Charcot broke new ground by using hypnosis as an experimental model of hysteria. Such use of hypnosis enabled Charcot to draw conclusions about hysteria's underlying neural basis in the form of anatomically localisable functional disturbances, which he termed dynamic lesions. Just as importantly, I have discussed how Charcot combined detailed clinical observation, physiological measurements, and hypnotic experiments to make conjectures about a particular pathophysiological mechanism responsible for the formation of such dynamic brain lesions. Drawing on these findings, Charcot then developed a simple yet apparently effective treatment for hysterical paralysis and introduced new image-based tools that reshaped the diagnosis of this disorder by foregrounding the hysteria-specific characteristic of its symptoms.

Throughout this chapter, my analysis has highlighted how, far from serving as mere illustrations of preconceived notions, images fulfilled key epistemic functions in all the stages of Charcot's hysteria research. Depending on the particular epistemic goal and the type of symptom he was investigating, Charcot deployed highly diverse kinds of images. These included photographs, schematic drawings, sketches, line graphs, inscriptions generated through Marey's graphic method, as well as perimetric and body maps. Charcot systematically used such images to search for the symptoms' underlying physiological regularities, gain insights into the nature of hysteria's elusive dynamic lesion, develop new diagnostic approaches, and evaluate as well as demonstrate the effectiveness of his novel therapy. Therefore, images were constitutive of Charcot's endeavour to establish hysteria as a genuine neurological disorder characterised by a set of clear-cut clinical signs and a distinct pathophysiological mechanism.

Finally, what has been of particular importance to me was to demonstrate that Charcot's use of images as investigation tools was firmly embedded in the neurophysiological theories of his time and was influenced, in particular, by the paradigm of cerebral localisation. Hence, I have insisted that to understand why Charcot produced specific images in a particular context and how he read and interpreted them, we have to pay close attention to the theories of brain function and human physiology, which decidedly informed Charcot's hysteria research. In fact, as my analysis in the following chapter will show, it was precisely this exclusively neurophysiological orientation of Charcot's image-based investigation of hysteria that came to be challenged by the end of the nineteenth century. However, we will also see that by the beginning of the twenty-first century, Charcot's understanding of hysteria as a brain-based disorder has been taken up by a new generation of scientists, who once again use images, although of a very different kind, to investigate this elusive illness.

851 Scull, *Hysteria*, 114.