***

To sum up, my analysis has shown that during the mid-to-late 1870s, Charcot and his team used photography as an experimental condition in their research into the hysterical attack. Such exploratory use of photography enabled them to produce new empirical insights into the hysterical attack's repetitive visual features, temporal development, and most common variations. I have underscored how the epistemic efficacy of photography was contingent on its embeddedness into a specific experimental system and the coordination with physiological measurements, written observations, and sketching. Regardless of whether or not the thus obtained photography-based insights could stand the test of time, they were epistemically significant because they led to Charcot's reconfiguration of the initial tripartite into a new four-stage model of the hysterical attack. Moreover, we have discussed how through the process of intermedial transcription, Regnard's heterogeneous photographs provided the basis for the subsequent development of the synoptic table of the hysterical attack. By creating the synoptic table, Richer succeeded in mapping the fundamental type of the hysterical attack and its multiple incomplete variations within a single diagrammatic visualisation. The synoptic table thus became an effective diagnostic tool that trained the physician how to look at chaotic convulsive fits and recognise in them a hysterical attack.

But, as Charcot repeatedly pointed out, the synoptic table had an additional benefit apart from its diagnostic value. For Charcot, this multipart visualisation also demonstrated "that in the attack," and all the other clinical manifestations of hysteria, "nothing is left to chance, everything follows definitive rules."[334] Put simply, the synoptic table provided admittedly indirect but visually compelling evidence that, despite the lack of any detectable anatomical lesion, the hysterical attack, in particular, and hysteria, in general, were governed by strict physiological laws.[335] Consequently, as soon as the basic tenets of the new conception of the hysterical attack had emerged in 1878, Charcot began to redirect his research away from purely nosographic concerns. From this point, his research focused increasingly on elucidating the underlying neurophysiological basis of hysteria. And as the following sections will show, in this process, symptoms other than the hysterical attack came to occupy much of Charcot's attention.

1.2 Hypnotic Experiments: Image-Based Search for the Neurophysiological Basis of Hysteria

So far, we have discussed how the targeted use of various visualisation techniques enabled Charcot and his team to articulate underlying regularities of symptoms such as hysterical attack and ischuria, and thus establish these manifestations of hysteria as clearly defined diagnostic entities. None of the resulting visualisations provided

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

335 Charcot, 13.

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Charcot with any direct information about the hypothesised neurophysiological basis of the symptoms under study. Nevertheless, by drawing on the patterns of underlying regularities that started to emerge from his image-based research, as well as the lack of any detectable anatomical brain lesion, Charcot conjectured that hysteria could only arise from "some [aberrant] action of the nervous system."[336] But at first, he had to admit that, for the time being, he could neither determine the exact nature nor the potential anatomical location of this presumed neural dysfunction.[337]

Searching for new ways of identifying hysteria's unknown neurophysiological basis, in 1878, Charcot and his team started to focus on the experimental use of hypnosis.[338] At the time, hypnosis was vaguely understood and, therefore, routinely equated with charlatanry and deception.[339] Despite its bad reputation, hypnosis was of interest to Charcot because it could be used to artificially induce changes in the subject's motor and sensory functions in ways that closely resembled hysterical symptoms. As Richer pointed out, hysterical symptoms and their hypnotically induced counterparts were so similar in their surface manifestations that the only apparent difference between them was their origin.[340] Whereas hysterical symptoms developed spontaneously, their hypnotic counterparts had to be provoked artificially.

Conveniently, this also meant that whereas hysterical symptoms were entirely uncontrollable, their hypnotic counterparts were not. But to be able to produce hypnotic counterparts of hysterical symptoms, the physician first had to induce the experimental subject into a hypnotic state, which Charcot designated as a form of artificial sleep.[341] Charcot and his team used a variety of methods to induce the hypnotic state. These included fixating the subjects' gaze on a bright object placed slightly above their eyes, applying light pressure on their eyeballs, exposing them to bright light or loud noises, or verbally instructing them to fall asleep.[342] Once the subject was in artificial sleep, various somatic and psychological phenomena could be produced "at the discretion" of the experimenter.[343] These included limb paralysis, contractures, different forms of anaesthesia, and diverse visual and auditory hallucinations. Additionally, hypnotised subjects could be made to perform various actions because, as Charcot explained, "their brains assent[ed] with singular accommodation to all the suggestions coming from the experimenter."[344] For instance, hypnotised patients could be made to drink wine that

336 Charcot, "Lecture 9: Hysterical Ischuria," 242.

337 See Charcot, 244. See also Charcot, "Lecture 21: Brachial Monoplegia," 278.

338 See Charcot, "Études physiologiques," 297. For a historiographic analysis of how Charcot's hypnosis research related to the earlier practice of Antoan Mesmer's animal magnetism and was even more closely linked to Victor Burq's metalloscopy (i.e., an approach to treating hysteria and other ailments through the application of metals), see Harrington, "Metals and Magnets."

339 See, e.g., Bourneville and Regnard, *Iconographie photographique*, 3:149.

340 Richer, *Études cliniques*, 2nd ed., 505.

341 Charcot and Richer, "L'hypnotisme chez les hystériques," 309.

342 See Charcot and Tourette, "Hypnotism in the Hysterical," 606–7. As explicitly stated by the Salpêtrians, they adopted many of these induction methods from the Scottish surgeon James Braid, whom they viewed as a pioneer of scientific research on hypnosis. See Bourneville and Regnard, *Iconographie photographique*, 3:156.

343 Charcot and Richer, "L'hypnotisme chez les hystériques," 310 (my translation).

344 Charcot and Tourette, "Hypnotism in the Hysterical," 608.

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did not exist, dance to music that nobody else heard, or pick and smell flowers that were not there.[345] Such experiments were ended by "lightly blowing on the eyes of the subject" to awaken them from their artificial sleep.[346]

Crucially, Charcot asserted that both the hypnotic state (i.e., artificial sleep), as well as all the subsequent somatic and psychological phenomena that could be induced in the subject during this state, should be viewed as unequivocal signs of pathology. In short, he argued that hypnosis was a "morbid condition," albeit an artificially provoked one.[347] Moreover, he posited that this morbid condition, which lacked any detectable anatomical brain lesion, must be caused by some unknown disturbance in the normal functioning of the nervous system.[348] To put it plainly, in hypnosis, just like in hysteria, Charcot hypothesised the existence of an unknown functional lesion of the nervous system. Emphasising this point, Charcot designated hypnosis as an artificial or experimental neurosis (*nevrosé*).[349] In doing so, he placed hypnosis in the same category of neurological disorders as hysteria.

Far from stopping at this point, Charcot claimed to have identified further explicit links between hypnosis and hysteria, which went beyond the mere visual similarity of the two phenomena's surface manifestations. Specifically, Charcot insisted that hypnotic phenomena "*in their totality*" could only be induced in hysteria patients.[350] He admitted that there were some exceptions. First, not all hysteria patients appeared to be susceptible to hypnosis.[351] Nevertheless, those hysteria patients who were entirely resistant to hypnosis were rare. Second, Charcot claimed that hypnotic susceptibility was uncommon among healthy individuals who did not exhibit any hysterical symptoms. He also argued that if susceptibility to hypnosis was found in apparently healthy individuals, it was a clear sign of latent hysteria, which had yet to manifest itself.[352] Hence, on the whole, Charcot regarded hypnosis as the experimental analogue of hysteria. This hypothesised analogy allowed Charcot to use hypnosis to experimentally model and study hysteria.

One key benefit of using hypnosis to experimentally model hysteria was that the symptoms thus induced could be "carried to the highest degree, and occur, moreover, under conditions which are more accessible to analysis."[353] For example, using hypnosis, Charcot could induce either an isolated symptom or combine several symptoms to fit his research purposes. Additionally, he could determine and even controllably vary the type, the intensity, and the anatomical location of each such artificially produced symptom. Another no less significant benefit was

345 See, e.g., Richer, *Études cliniques*, 2nd ed., 727.

346 Charcot and Tourette, "Hypnotism in the Hysterical," 607.

347 Charcot and Tourette, "Hypnotism in the Hysterical," 606. For details, see also Charcot and Richer, "L'hypnotisme chez les hystériques," 310.

348 See, e.g., Charcot and Richer, "L'hypnotisme chez les hystériques," 310; and Charcot and Tourette, "Hypnotism in the Hysterical," 606.

349 Charcot and Tourette, "Hypnotism in the Hysterical," 606.

350 Charcot and Tourette, 606 (emphasis in original).

351 Charcot and Tourette, 606.

352 Charcot and Tourette, 606.

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

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that hypnosis allowed Charcot to frame his experimental research into hysteria in decidedly neurophysiological terms. The basis for this framing was Charcot's aforementioned tenet that all hypnotic phenomena arose from an, at that point, still unknown modification of the normal functioning of the nervous system. Drawing on this tenet, Charcot argued that all hypnotic phenomena had to be determined by strict neurophysiological laws.[354] Some variations in how subjects responded to hypnosis were unavoidable. They arose from individual differences in each subject's "temperament and special nervous dispositions."[355] Yet, Charcot insisted that both the scientific study and the experimental use of hypnosis had to disregard such essentially irrelevant variations. Instead, the primary scientific aim was to identify and experimentally manipulate the underlying physiological regularities of hypnosis.

To achieve this, the research had to focus primarily on what Charcot termed "generic" physical manifestations of hypnosis.[356] Such generic manifestations, which I will list shortly, comprised various disturbances of motor and sensory functions that developed "spontaneously" in all hysteria patients as soon as they were inducted into a hypnotic state.[357] Importantly, Charcot and his team insisted that neither the experimenter nor the hypnotised subject could influence the features of the generic manifestations of hypnosis because these features were physiologically determined.[358] Further, Charcot asserted that hypnosis was not a unitary condition but a series of different morbid states of the nervous system.[359] Each of these distinct states could be induced separately and was characterised by a particular set of generic somatic manifestations. Based on these differences, Charcot divided hypnosis into three distinct phases: lethargy, catalepsy, and somnambulism.

According to Charcot, during the state of lethargy, the subjects were "plunged into the most complete coma."[360] This state was characterised by the abolition of all senses, loss of skin sensibility, and absolute "mental inertia."[361] With their eyes closed and limbs hanging, the subjects were entirely unresponsive. It was, therefore, "impossible to enter into relation" with them.[362] Even more significantly, in addition to exalted tendon reflexes, the subjects also exhibited an unusual "aptitude of muscles to contract under a simple mechanical excitation."[363] Charcot designated this curious aptitude as neuromuscular hyperexcitability.[364] He considered this aptitude to be the chief generic manifestation of hypnotic lethargy or, in other words, its 'objective' physiological sign.

354 See Charcot and Tourette, "Hypnotism in the Hysterical," 606. See also Richer, *Études cliniques*, 2nd ed., 512.

355 Richer, *Études cliniques*, 2nd ed., 512.

356 Charcot, "Études physiologiques," 299. See also Richer, *Études cliniques*, 2nd ed., 514.

357 Richer, *Études cliniques*, 2nd ed., 514.

358 Richer, 512, 514.

359 Charcot and Richer, "Cerebral Automatism," 2. See also Charcot and Tourette, "Hypnotism in the Hysterical," 607–8; and Charcot, "Études physiologiques," 300–4.

360 Charcot and Tourette, "Hypnotism in the Hysterical," 607.

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

362 Charcot, 290.

363 Charcot, "Études physiologiques," 305.

364 Charcot, 305.

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Contrary to lethargy, in the cataleptic state, the subjects' tendon reflexes were abolished, and the mechanical excitation of muscles resulted in paralysis and not a contracture.[365] Moreover, the activity of some of the subjects' senses was partly restored.[366] But the most defining generic physiological signs of this state were the suppleness of the subjects' limbs and their immobility.[367] As a result, the experimenter could place the cataleptic subjects' bodies into a range of different positions in which they would remain for a long time "as if petrified."[368] Finally, in the state of somnambulism, hypnotised subjects exhibited normal tendon reflexes, and their limbs ceased to be pliable. However, their skin and sense organs exhibited increased sensitivity to stimuli.[369] During this state, hypnotised subjects became responsive to the experimenter's verbal injunctions and could be made to perform various complex acts.[370]

Importantly, Charcot and his team insisted that all the characteristics listed above were fully developed only in what they referred to as the *grand hypnotism*, a form of hypnosis that could be induced exclusively in patients suffering from major hysteria (i.e., *grande hystérie*).[371] Hence, in their hypnosis research, the Salpêtrians focused only on those exceptional clinical cases in which both hysterical symptoms and hypnotic responsiveness were developed in an accentuated form.[372]

The following two sections will examine how Charcot and his team sought to elucidate the neurophysiological basis of hysteria by systematically inducing and studying the key generic manifestations of lethargy and catalepsy.[373] I will demonstrate that, just as in the preceding nosographic stage of his research, also in Charcot's hypnotic experiments, images played crucial epistemic roles. Yet, I will argue that in their hypnotic experiments, Charcot and his team used photography in distinctly different ways than in their investigation of the hysterical attack. Apart from photography, I will also analyse how the Salpêtrians implemented the graphic method, which they adopted from Étienne-Jules Marey, to study the aspects of hypnotic phenomena inaccessible to human vision.

Moreover, to underscore how the use of photography and the graphic method could generate new insights into hypnosis and hysteria, my analysis will focus, in particular, on neurophysiological theories that, as I intend to show, had informed both the production and interpretation of images in Charcot's hypnotic experiments. The first section will look into how Charcot and Richer attributed hysterical contractures

365 Richer, *Études cliniques*, 2nd ed., 612.

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

367 Charcot and Richer, "Cerebral Automatism," 3.

368 Charcot and Richer, 3.

369 Charcot and Tourette, "Hypnotism in the Hysterical," 608.

370 Charcot, "Études physiologiques," 303–4.

371 Richer, *Études cliniques*, 2nd ed., 513.

372 Charcot, "Études physiologiques," 299. As stated by Charcot, only one in four to five of his patients exhibited *grande hystérie*. In the rest of his patients, the hypnotic phenomena could only be induced in an attenuated form. See Charcot and Richer, "L'hypnotisme chez les hystériques," 386.

373 Later in this chapter, I will show that the state of hypnotic somnambulism played a crucial role in subsequent stages of Charcot's hysteria research. See section 1.3.2.

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to a morbid exaggeration of spinal reflexes as a result of their systematic study of neuromuscular hyperexcitability. The subsequent section will then analyse how by drawing on the result of their cataleptic experiments, Charcot and Richer linked hysteria to higher-order brain reflexes.

1.2.1 Attributing Hysterical Contractures to Exaggerated Spinal Reflexes

In the early phase of Charcot's use of hypnosis as an experimental neurosis, one hypnotic phenomenon, in particular, stood in the focus of his research. Charcot initially named this phenomenon muscular hyperexcitability.[374] However, by 1881, he referred to it as neuromuscular hyperexcitability.[375] This renaming reflected Charcot's new insights into the neural basis of this phenomenon, which we will analyse in this section. In Charcot's use, neuromuscular hyperexcitability designated the ability to artificially induce in a hypnotised patient a localised contracture (i.e., a permanent contraction) of a muscle through simple mechanical excitation, such as kneading, light pressure, or massage. According to the Salpêtrians, two conditions were thereby necessary. First, the hypnotised patient had to be in the state of lethargy since this peculiar somatic phenomenon existed neither during catalepsy nor somnambulism. Second, to induce a contracture, the mechanical excitation had to go beyond skin limits and reach the subcutaneous tissue.[376]

The preliminary experiments investigating neuromuscular hyperexcitability were already presented and discussed in the third volume of the *Iconographie photographique*.[377] But the most systematic overview of the Salpêtrian research into neuromuscular hyperexcitability and a detailed examination of how this phenomenon related to spontaneously developed hysterical contractures can be found in a one-hundred-page-long study Charcot jointly authored with Richer.[378] This study is the focus of my analysis in the current section. I aim to demonstrate that, in this study, Charcot and Richer succeeded in elucidating the neurophysiological basis of neuromuscular hyperexcitability and then used this finding to explain the nature of spontaneous hysterical contractures. The study itself comprised a description of a long series of experiments, with each experiment building upon the finding of those preceding it.[379] My analysis will outline how, through this series of experiments,

374 See Bourneville and Regnard, *Iconographie photographique*, 3:20, 27. See also Richer, *Études cliniques*, 368, 382, 431.

375 See, e.g., Charcot and Richer, "L'hypnotisme chez les hystériques," 309; and Richer, *Études cliniques*, 2nd ed., 539.

376 Richer, *Études cliniques*, 2nd ed., 538. As mentioned earlier, during lethargy, the sensibility of the hypnotised patient's skin was entirely abolished.

377 See, e.g., Bourneville and Regnard, *Iconographie photographique*, 3:20, 217, 219.

378 The study initially appeared in several instalments in the medical journal *Archives de neurologie* from 1881 to 1883. See Charcot and Richer, "L'hypnotisme chez les hystériques," 309n1. It was later republished in the ninth volume of Charcot's *Oeuvres complètes*, which is the source I am using here. See Charcot and Richer, "L'hypnotisme chez les hystériques," 309–421.

379 The experiments were conducted from 1878 to 1881. In their study, Charcot and Richer did not present the experiments in their chronological order, which makes for difficult reading. My analysis reconstructs the order in which the experiments were conducted.

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Charcot and Richer gradually articulated the view that hysterical contractures arose from a disturbance of the reflex activity of the spinal cord.[380] Importantly, I will argue that the articulation of this view was facilitated by the targeted use of photography and Marey's graphic method. Moreover, I will show that, in the process, Charcot and Richer drew on Duchenne de Boulogne's experiments investigating the neurophysiological basis of bodily movements and facial expressions, as well as Wilhelm Erb's research on tendon reflexes.[381]

Charcot's experiments on neuromuscular hyperexcitability started in 1878. Initially, he focused on using this phenomenon to artificially reproduce various contractures his hysteria patients developed spontaneously in their waking state. For example, Charcot determined that by mechanically stimulating the so-called flexor muscles on the inner side of a hypnotised patient's forearm, he could produce a particular contracture. The result was the bending of the patient's arm towards the body and the concurrent flexing of the hand and fingers.[382] Furthermore, the Salpêtrians also established that artificially produced contractures remained permanent unless resolved through an additional experimental intervention, which had to be performed while the patient was still in the state of lethargy. This intervention involved mechanically exciting the antagonist muscles that performed the opposite movement of those initially excited.[383] Hence, to dispel the contracture of the arm described above, which entailed a flexion (i.e., stretching), Charcot merely had to mechanically stimulate the extensor muscles situated on the backside of the patient's forearm.[384] According to Charcot, the fact that, without such intervention, the artificially induced contractures remained permanent even after the patient woke up from hypnosis was highly significant. It proved that spontaneously developed hysterical and artificially induced hypnotic contractures were mutually analogous.[385]

By systematically kneading and pressing muscles on different parts of their hypnotised patients' bodies, Charcot and his team experimented with inducing and resolving a wide range of contractures. The resulting contractures entailed various defective attitudes of the patients' upper and lower limbs, hands, feet, trunk, and neck.[386] In each case, the muscle to which the mechanical excitation was applied

380 See Charcot and Richer, "L'hypnotisme chez les hystériques," 411. I am using the term articulation here in Latour's sense. See Latour, *Pandora's Hope*, 142–44.

381 See, in particular, Duchenne de Boulogne, *L'électrication localisée*; Duchenne de Boulogne; *Physiologie des Mouvements*; Duchenne de Boulogne, *Facial Expression*; and Erb, "Ueber Sehnenreflexe."

382 See Bourneville and Regnard, *Iconographie photographique*, 3:20.

383 Bourneville and Regnard, 20. See also Charcot and Richer, "L'hypnotisme chez les hystériques," 377–78.

384 The effectiveness of this kind of intervention indicated that hysterical contractures entailed a disbalance in the motor activity of mutually antagonistic muscular groups, such as flexors and extensors. Charcot kept returning to this point in his subsequent studies and lectures. See, e.g., Charcot, "Lecture 7: Contracture of Traumatic Origin," 87, 89; and Charcot, "Lecture 25: Spasmodic Contracture," 351. See also Charcot and Richer, "On a Muscular Phenomenon."

385 Charcot and Richer, "L'hypnotisme chez les hystériques," 379.

386 See Bourneville and Regnard, *Iconographie photographique*, 3:204.

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contracted, thus "producing the movement which naturally belongs to it."[387] Having reached the end of this movement, the muscle then remained immobilised in the attitude of its maximal contraction even after the mechanical stimulation had stopped. Several photographs that documented the artificial contractures thus obtained were published in the third volume of the *Iconographie photographique*.[388]

At first, the Salpêtrians focused on experimenting with large muscles easily accessible to mechanical excitation, such as the sternomastoid muscle, which is located on the side of the neck.[389] Soon, they discovered that to obtain a permanent contracture of this large muscle, it was not necessary to knead or massage its entire surface. It turned out that using a blunt end of a small wooden stick to exert light pressure on any single point along one of its many fibres sufficed to produce an energetic contracture of the whole sternomastoid muscle. In their joint study, Charcot and Richer reproduced a photograph of this particular experiment and explicitly referred the reader to consult this image (fig. 1.10).[390] As they explained, the image showed that the resulting contracture entailed a tilting of the patient's neck and the rotation of her face away from the point of excitation. Charcot emphasised that this rotational movement of the patient's neck was entirely in accordance with the normal physiological function of the sternomastoid muscle.[391] What was out of the ordinary was the disproportionate intensity of the muscular reaction to minimal stimulation.

Significantly, I argue that, in this specific experiment, photography had a distinctly different function than in the cases discussed so far. The function of this particular image was neither to illustrate a chosen feature of a previously diagnosed manifestation of hysteria nor to provide initially ambiguous empirical data about a symptom of interest. Rather, the image served to establish a clear visual correlation between the experimental manipulation (i.e., the experimenter's hand holding a stick that touched a point on the patient's neck) and its physiological consequences (the visibly protruding muscle and the tilted position of the patient's head). Notably, the resulting contracture persisted after the cessation of the direct mechanical excitation. This means that the contracture could also have been photographed without the presence of the experimenter's hand. Therefore, it appears to me that instead of merely intending to document the result of the experiment, Charcot and Richer deliberately chose to have a photograph taken that simultaneously visualised both the experimental manipulation and its effect. Hence, the intended function of this photograph was to provide empirical evidence of Charcot's novel experimental finding. The image effectively demonstrated that, during the hypnotic lethargy, even a minimal mechanical excitation limited to a single anatomical point produced a spasmodic contracture of an entire sizeable muscular mass.[392]

387 Charcot and Tourette, "Hypnotism in the Hysterical," 608.

388 See Bourneville and Regnard, *Iconographie photographique*, vol. 3, plates 12, 19, 21, and 31.

389 See Charcot and Richer, "L'hypnotisme chez les hystériques," 349.

390 See Charcot and Richer, 349.

391 Charcot and Richer, 349.

392 Charcot and Richer, 350.

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Figure 1.10. Photograph of a permanent contracture of the sternomastoid muscle induced through simple mechanical excitation during hypnotic lethargy. From: Charcot, Oeuvres complètes, vol. 9, plate 5, fig. 1.

Through continued experiments, Charcot soon identified another peculiar feature of neuromuscular hyperexcitability. He established that, in some anatomical regions, although the mechanical excitation was applied to the body of a single muscle, the result he obtained was not a localised contracture. Instead, the excitation led to simultaneous contractures of several so-called synergistic muscles.[393] Synergistic muscles—whose discovery was made by Duchenne de Boulogne—are groups of functionally connected muscles.[394] These muscles are located in different parts of the body yet work together to enable the execution of a particular movement in healthy individuals. Thus, for example, Charcot's experiments showed that pressing the wooden stick on a hypnotised patient's shoulder muscle (i.e., the deltoid) always additionally elicited concurrent contractures of two large muscles in the patient's back and trunk (i.e., the trapezius and serratus). The concurrent contractures arose, although the latter two muscles had not been directly

393 Charcot and Richer, 350.

394 See Duchenne de Boulogne, *Physiologie des Mouvements*, viii; Duchenne de Boulogne, *L'électrication localisée*; and Duchenne de Boulogne, *Facial Expression*, 18–19.

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stimulated.[395] According to Duchenne, these three muscles (i.e., the deltoid, trapezius, and serratus) were functionally connected since they always worked in synergy to move the shoulder in healthy subjects.[396] Drawing on Duchenne, Charcot concluded that, during hypnotic lethargy, mechanical excitation propagated in conformity with physiological laws because it led to joint contractures of the muscles that acted together in a healthy state.

Based on the two novel findings discussed so far, Charcot conjectured that the contractures induced during hypnotic lethargy could not be attributed to any direct effect of mechanical excitation on the muscular fibres.[397] Specifically, he argued that the direct excitation of muscular fibres accounted neither for the simultaneous contractures of synergistic muscles nor for the fact that entire muscle masses contracted in response to a slight punctual stimulation. Charcot reasoned instead that the mechanical stimulation had spread from the muscles to their tendons and nerves, inducing a reaction in all these different elements of the neuromuscular system, which then jointly produced the contracture.[398] In other words, Charcot proposed at this point that the phenomenon he had initially designated as muscular hyperexcitability was based on some yet unknown action of the nervous system.[399] To test this proposition and uncover the phenomenon's underlying neural basis, Charcot and Richer devised a long series of mutually interrelated experiments. As my analysis will show, these experiments allowed Charcot and Richer to decompose neuromuscular hyperexcitability into its neurophysiological components and thus isolate the distinct roles that muscles, nerves, and tendons had in producing contractures.

Importantly, the starting point for Charcot's investigation of how isolated muscles and nerves responded to mechanical excitation during hypnotic lethargy was Duchenne de Boulogne's decades-long electrophysiological research into the mechanisms of human movement.[400] In fact, both the discovery of muscular synergies and the studies of emotional facial expressions we discussed previously were part of Duchenne's broader research into the neurophysiological basis of movement. Therefore, understanding some of the basic tenets of Duchenne's electrophysiological research is crucial for our further discussion. For this reason, in what follows, we will examine those aspects of Duchenne's research that Charcot and Richer used as the basis for their hypnotic experiments.

Aiming to study human movement by delineating individual actions of different muscles that partook in it, Duchenne developed a method he called localised faradisation.[401] The method entailed applying electrodes to the surface of the body to direct the electrical current through the skin "and concentrate its action in one muscle or in a muscle bundle, in a nerve trunk or in a nerve branch."[402] In Duchenne's

395 Charcot and Richer, "L'hypnotisme chez les hystériques," 350.

396 See Duchenne de Boulogne, *Facial Expression*, 18–19.

397 Charcot and Richer, "L'hypnotisme chez les hystériques," 312.

398 Charcot and Richer, 312.

399 Again, I am using the term proposition here in Latour's sense. Latour, *Pandora's Hope*, 141.

400 See Charcot and Richer, "L'hypnotisme chez les hystériques," 351–52.

401 For details, see Duchenne de Boulogne, *L'électrication localisée*, 27–58.

402 Duchenne de Boulogne, *Facial Expression*, 10.

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experiments, the electricity served as a stimulating agent "analogous to the nervous fluid" or, in other words, the nerve impulse.[403] Through this intervention, Duchenne was able to provoke targeted contractions of either single muscles or select groups of muscles. The resulting contractions permitted Duchenne to determine the action that each muscle performed under normal physiological conditions. Over the years, using this method, Duchenne systematically mapped the functions of various muscles and nerves in the human limbs, trunk, and face.[404]

In the initial phase of his research, Duchenne first focused on delimiting the action of several large nerve trunks in the arm.[405] Relying on his knowledge of anatomy to identify the points on the skin at which the ulnar, medial, and radial nerves were accessible to his electrodes, Duchenne induced simultaneous contractions of all muscles that each of these nerves control.[406] He thus succeeded in determining which muscles of the arm were controlled by which of the three main nerve branches. But to induce a clearly isolated movement of individual muscles of the arm, Duchenne had to find a way of activating each muscle separately. This, at first, proved challenging due to the muscles' anatomical vicinity. Yet, through trial and error, Duchenne soon made the empirical discovery that the partial excitation of a single muscle was most easily and clearly obtained if the electrodes were applied to a particular location on the skin above the muscle of interest.[407] Systematically, he identified such points in the limbs, trunk, and face. He later referred to these locations as the election points.[408]

Duchenne believed that by applying his electrodes to the election points, he was directly stimulating the fibres of the muscles.[409] However, by the late 1850s, two German physicians, Robert Remak and Hugo von Ziemssen, determined that Duchenne's election points were, in fact, anatomical locations at which the muscular nerves entered into the body of the respective muscle.[410] Hence, Remak and Ziemssen opposed Duchenne's claim that the localised contractions of individual muscles in his experiments were caused by the direct stimulation of the muscular fibres. Instead, they argued that the contractions arose from the electrical excitation of the muscular nerves at their point of entry into the respective muscles.[411] It was this explanation by Remak and Ziemssen that Charcot supported and quoted in a series of hypnotic experiments, which he devised together with Richer to study neuromuscular hyperexcitability. As

403 Duchenne de Boulogne, 9.

404 Duchenne de Boulogne, *Physiologie des mouvements*; and Duchenne de Boulogne, *L'électrication localisée*, 171–401.

405 Duchenne de Boulogne, *L'électrication localisée*, 45.

406 Duchenne de Boulogne, 45.

407 Duchenne de Boulogne, 47, 58.

408 See, e.g., Duchenne de Boulogne, *L'électrication localisée*, 3rd ed., 81.

409 Duchenne de Boulogne, *L'électrication localisée*, 47.

410 See Remak, *Methodische Electrisirung*, 14; and Ziemssen, *Die Electricität in der Medicin*, 4–6.

411 Somewhat confusingly, on different occasions, Duchenne took entirely inconsistent stances on this view. For example, in some of his subsequent publications, Duchenne appeared to accept the explanation posited by Remak and Ziemssen. See, e.g., Duchenne de Boulogne, *Facial Expression*, 48. By contrast, in other publications, Duchenne vehemently opposed Remak's views. See, e.g., Duchenne de Boulogne, *L'électrication localisée*, 3rd ed., 73–75, 82–85.

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we are about to see, Charcot's and Richer's hypnotic experiments explicitly recreated Duchenne's electrophysiological studies.[412]

In their research into neuromuscular hyperexcitability, Charcot and Richer first turned to recreating those of Duchenne's experiments in which he had applied localised electricity to the large nerve trunks in the arm.[413] In their version, the experimental subjects were not fully awake individuals but hysteria patients in the state of hypnotic lethargy. Moreover, Charcot and Richer displaced electricity with mechanical stimulation. They either pressed their finger or a small wooden stick onto the same anatomical location on the patient's arm to which Duchenne had applied his electrodes.[414] For example, by pressing a spot on the inner side of a patient's elbow, Charcot mechanically excited the ulnar nerve. Due to this intervention, the hypnotised patient's hand assumed a peculiar attitude Charcot referred to as the ulnar deformity (*griffe cubitale*).[415] As Charcot explained, this artificially induced attitude arose from the simultaneous contractures of all the muscles in the forearm and hand, which according to Duchenne's electrophysiological findings, were innervated by the branches of the ulnar nerve.[416] Using the same procedure, Charcot and Richer then successfully reproduced two other typical attitudes of the hand Duchenne had induced through the localised faradisation of the median and radial nerves, respectively.[417] Based on these results, Charcot and Richer were able to claim that the mechanical stimulation deployed during hypnotic lethargy produced the same effects on the nerve trunks as the faradisation in the waking state.[418] This, in turn, allowed them to posit a relation of analogy between these two types of intervention in the given contexts.

Drawing on the thus established analogy, in the next step, Charcot and Richer proceeded to recreate with their hypnotised patients the experiments in which Duchenne had induced the isolated action of individual muscles of the arm through faradisation.[419] Again, Charcot and Richer deployed mechanical excitation and not electricity. And once again, they took great care to exert pressure on the same election points Duchenne had used in his experiments.[420] However, transposing this set of experiments into the context of hypnotic lethargy proved challenging. Despite considerable efforts they had invested in these experiments, Charcot and Richer succeeded in producing only a few clearly delineated contractures of individual muscles

412 Charcot and Richer, "L'hypnotisme chez les hystériques," 352. The importance of the finding that Remak and Ziemssen made about the nature of the election points will become apparent in the course of my analysis.

413 See Charcot and Richer, 336–48.

414 Charcot and Richer, 336.

415 In this characteristic hand attitude, the index and middle fingers were extended, the ring and little fingers were completely bent, and the thumb pressed upon the last two fingers. See Charcot and Richer, 337.

416 Charcot and Richer, 338–40.

417 Charcot and Richer, 342–48.

418 Charcot and Richer, 355–56.

419 Charcot and Richer, 348–55.

420 Charcot and Richer, 354–55.

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in the fingers.[421] In the rest of the arm, they obtained unclear and ambiguous results. The problem was, they argued, that the muscles of the arm were grouped tightly together, had many synergistic actions, and were innervated by widespread nerve branches.[422] Under such conditions, the mechanical excitation failed to remain isolated to the election points to which it was directly applied. Instead, the excitation spread to neighbouring muscles and nerves, leading to multiple simultaneous contractures. Charcot and Richer regarded such effects as errors since their explicit aim was to obtain isolated actions of single muscles through the localised excitation of their designated election points. Hence, despite the apparent analogy of the methods, mechanical excitation turned out to be anatomically less precise than the stimulation by means of electrodes.

Nevertheless, Charcot and Richer were not willing to give up. To solve the problem, they switched from the muscles of the arm to the face. In other words, they shifted the focus of their research onto recreating the electrophysiological experiments that constituted Duchenne's study of facial expressions. As Charcot explained, the conditions for experimenting on the facial muscles were less complex. "The muscles are superficial, usually arranged in a single layer, and, therefore, easily accessible to mechanical excitation. Moreover, there are no tendons whose indirect excitation can thwart, mask or even completely hinder the desired result."[423] In my opinion, what was even more significant for Charcot's purpose of inducing isolated muscular action in the state of lethargy was a particular feature of facial muscles Duchenne had discovered in his experiments. To delineate this feature, we need to take a look at Duchenne's experiments on facial expressions.

In his study of facial expressions of emotions, Duchenne used the same approach as in his broader electrophysiological research into bodily movements. In short, he applied electrodes to the election points of different muscles of the face to induce the isolated contractions of the muscles of interest and thus study their movement.[424] As in his previous studies, Duchenne proceeded systematically. He first elicited contractions of each facial muscle in isolation. He started by manipulating the muscle of interest only on one side of the face and then on both sides of the face simultaneously. Next, he proceeded to test various combinations of muscular contractions "two by two and three by three."[425] Contrary to his previous studies of bodily motion, here he was interested in one particular effect of muscular movement—how it gave rise to recognisable facial expressions of distinct categories of emotion.[426] As mentioned earlier, this aspect of Duchenne's research was guided by the premise that facial expressions of distinct emotional categories were physiologically determined and, therefore, universal. He argued that facial expressions were "under the control of instinctive or reflex muscular contractions" and that, therefore, the "patterns of expression of the human face cannot

421 Charcot and Richer, 353–54.

422 Charcot and Richer, 356–58.

423 Charcot and Richer, 359.

424 See Duchenne de Boulogne, *Facial Expression*, 1, 3, 9–11.

425 Duchenne de Boulogne, 12.

426 Duchenne de Boulogne, 9.

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be changed, whether one simulates them or actually produces them by an action of the soul."[427]

Working under this premise, Duchenne aimed to identify the facial muscles whose combined contractions underpinned the expressions of distinct categories of emotions. Unexpectedly, he observed that facial muscles behaved differently than the muscles in the limbs and the trunk.[428] More specifically, based on his experiments, Duchenne determined that whereas all movements of the body required "simultaneous (synergistic) contraction of a more or less large number of muscles,"[429] facial expressions did not. In fact, he established that several facial muscles, which he labelled 'completely expressive,' could "produce an expression of their own by their isolated action."[430] Duchenne identified four such 'completely expressive' muscles. He stated that each of these muscles expressed through their individual action "in a most complete way" one of the four emotions: pain, aggression, reflection, and attention.[431]

However, apart from this significant peculiarity, Duchenne also discovered that facial expressions of all other emotions—such as joy, sadness, fear, or disgust—required combined contractions of two other types of muscles. He referred to one of these types as 'incompletely expressive' and the other as 'expressive in a complementary way.'[432] According to Duchenne, the 'incompletely expressive' muscles were "uniquely representative" of a particular emotion, yet unable to fully express this emotion on their own.[433] If activated in isolation, these muscles produced facial expressions that did not appear 'natural.' By contrast, the muscles designated as 'expressive in a complementary way' were entirely "inexpressive in isolation."[434] They merely served to complement the action of the 'incompletely expressive' muscles. Importantly, muscles belonging to these different types (i.e., completely expressive, incompletely expressive, expressive in a complementary way) could combine in various ways to give rise to a range of emotional expressions. In effect, this meant that even when various facial muscles acted together, there were no fixed, anatomically determined synergistic relations among them.[435] Hence, unlike the rest of the body, a contraction of one facial muscle did not necessarily spread to other muscles in the face. In my opinion, this particular functional feature of facial muscles was crucial for Charcot, as it allowed him to

427 Duchenne de Boulogne, 30.

428 Duchenne de Boulogne, 12–15.

429 Duchenne de Boulogne, 9.

430 Duchenne de Boulogne, 12.

431 Duchenne de Boulogne, 24. These four muscles were the frontalis ('muscle of attention'), the orbicularis oculi ('muscle of reflection'), the corrugator supercilii ('muscle of pain'), and the procerus ('muscle of aggression'). See ibid.

432 Duchenne de Boulogne, 24.

433 Duchenne de Boulogne, 24.

434 Duchenne de Boulogne, 24.

435 As Duchenne explained, the synergistic contractions in the rest of the body were "necessitated by the laws of mechanics." Duchenne de Boulogne, 19. Whereas one muscle performed the actual movement, those synergistically related to it acted to stabilise the body. Such a "need for mechanical equilibrium" did not "apply to the expressive movements of the face." Ibid.

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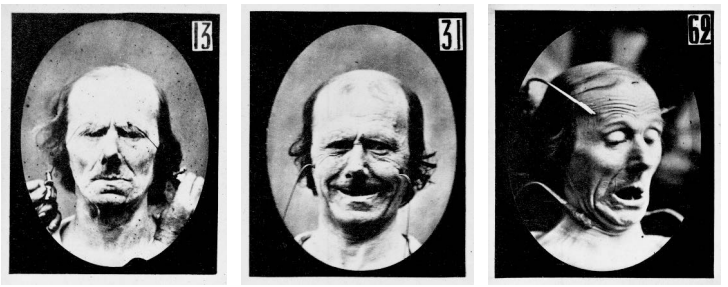
avoid the uncontrolled spreading of the effects of mechanical excitation with which he struggled in his experiments on the muscles of the arm.

Before we return to Charcot, we need to consider another aspect of Duchenne's experiments. Using the electrodes to induce both isolated and combined contractions of various facial muscles, Duchenne artificially produced expressions of more than thirty different categories of emotion in his experimental subjects.[436] Inconveniently, the electrically induced muscular contractions turned out to be transient. They lasted a maximum of a few seconds and only as long as the electrodes were applied to the face. Arguing that his findings "on the mechanisms of facial expression can only be judged by seeing them," Duchenne used photography to visually fix and later disseminate his experimental results (fig. 1.11).[437] As we are about to see, these photographs represented key points of reference for Charcot and Richer in their transposition of Duchenne's experiments into the context of hypnotic lethargy.

Figure 1.11. Photographs of emotional facial expressions induced by Duchenne de Boulogne through electrical stimulation of the designated election points. Left: mental concentration. Middle: false laughter. Right: terror. From: Duchenne de Boulogne, Mécanisme de la physionomie humaine, figs. 13, 31, and 62.



In their version of the experiments on facial muscles, Charcot and Richer once again displaced Duchenne's electrodes with a small wooden stick. They used the blunt end of the stick to apply light pressure to the same election points of the facial muscles that Duchenne had identified in his electrophysiological experiments (fig. 1.12).[438] However, they discovered that, during hypnotic lethargy, the facial muscles responded slightly differently to mechanical excitation than the rest of the body. Although the facial muscles proved to be susceptible to mechanical stimulation, their excitation did not produce a lasting contracture. Instead, the excitation led to a muscular contraction that lasted only while the stick was pressed to the election point.[439]

436 For a list of these emotions, see Duchenne de Boulogne, 26–28.

437 Duchenne de Boulogne, 36.

438 Charcot and Richer, "L'hypnotisme chez les hystériques," 369.

439 Charcot and Richer, 359–61.

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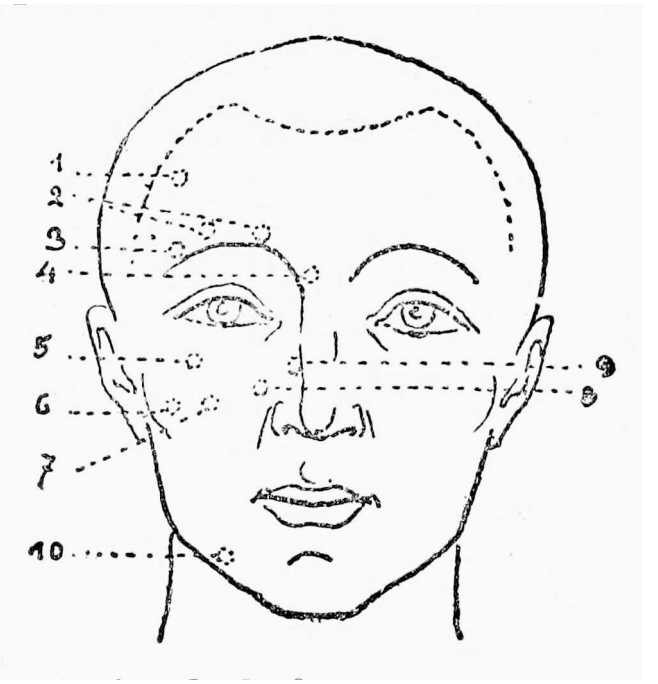
Figure 1.12. Map of the election points of ten facial muscles derived from Duchenne de Boulogne's electrophysiological experiments. From: Charcot, Oeuvres complètes, vol. 9, 363, fig. 16.



To facilitate the fixation of their experimental results and thus be able to compare them to those obtained by Duchenne, Charcot and Richer had to produce photographs of the resulting muscular contractions. Importantly, a direct visual comparison of their results with Duchenne's was the very aim of these experiments.[440] Yet, such a comparison would not have been possible without the aid of photography. It can, therefore, be said that photography once again became a constitutive element of the Salpêtrian experimental setup, attaining the function of an "experimental condition."[441] But in the hypnotic experiments, the role of photography was no longer to generate initially ambiguous empirical data, as was the case in the Salpêtrian exploration of the hysterical attack.[442] As will become apparent in what follows, in the context of hypnotic research, the role of photography shifted to generating empirical evidence of the outcomes obtained intentionally through targeted experimental interventions.

A particularly instructive aspect of how Charcot and Richer set about recreating Duchenne's experiments on facial expressions of emotions was the selectivity of their approach. Rather than aiming to reproduce on the faces of their hypnotised patients

440 Charcot and Richer, 362.

441 Rheinberger, *History of Epistemic Things*, 28.

442 See section 1.1.2.

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Duchenne's entire catalogue of emotional categories, Charcot and Richer chose a different focus. As the following examples will show, at the centre of their interest was testing, in a step-by-step procedure, if they could induce isolated actions of the three different types of facial muscles as classified by Duchenne. With this aim in mind, Charcot and Richer first used mechanical excitation to separately induce an isolated contraction of the muscles Duchenne had designated as 'completely expressive' due to their ability to display distinct emotions through their individual action.[443] One of these muscles was the frontalis, which Duchenne had termed 'the muscle of attention.' The other was the orbicularis oculi or, in Duchenne's terminology, 'the muscle of reflection.'

By separately stimulating these muscles, Charcot and Richer were able to obtain their isolated contractions and thus reproduce in the hypnotised patients the respective expressions of 'attention' and 'reflection' (fig. 1.13, left).[444] But whereas Duchenne unfailingly foregrounded the emotionally expressive aspects of his experimental results in the accompanying narrative description,[445] Charcot and Richer did not. They focused instead on describing the temporary modifications in the physiognomy that arose from the artificially induced muscular contractions. These modifications included, for example, the "lowering of the eyebrows," the appearance of the "curvilinear frontal folds," and "the smoothing of the wrinkles on the forehead."[446]

After this initial success, Charcot and Richer proceeded to induce the individual contractions of several muscles, which, according to Duchenne's classification, were incompletely expressive and, if activated in isolation, resulted in emotional expressions that appeared artificial.[447] One such example that Charcot and Richer chose to recreate was the facial expression Duchenne termed an insincere or false smile. This expression entailed an isolated flexion of the sides of the mouth, or in medical terms, the contraction of the zygomaticus major muscle (fig. 1.11, middle).[448] Having obtained the desired results (fig. 1.13, middle), Charcot and Richer then focused on recreating the expressions that, as stated by Duchenne, required the combined contractions of 'inexpressive' and 'expressive' muscles. For example, by simultaneously exposing the muscles in the forehead and the neck to separate mechanical excitations, Charcot and Richer induced in their patient the expression of fear (fig. 1.13, right).[449] In all these cases, their descriptions of the facial expression thus obtained remained focused on detailing the purely physical effects of the muscular contractions.[450]

Throughout the text that detailed their targeted experimental interventions, Charcot and Richer expressly referred their reader to the photographs of the obtained results, which were appended to the study. The photographs, as Charcot emphasised, confirmed that the outcomes of his experiments on hypnotised patients in the state

443 Charcot and Richer, "L'hypnotisme chez les hystériques," 363–66.

444 Charcot and Richer, 363–64.

445 Duchenne de Boulogne, *Facial Expression*, 49, 52.

446 Charcot and Richer, "L'hypnotisme chez les hystériques," 364.

447 Charcot and Richer, 366.

448 Duchenne claimed that a 'genuine' smile entailed simultaneous contractions of the zygomaticus major muscle and the corners of the eyes. See Duchenne de Boulogne, *Facial Expression*, 72–73.

449 Charcot and Richer, "L'hypnotisme chez les hystériques," 372–73.

450 See Charcot and Richer, 367–68, 370.

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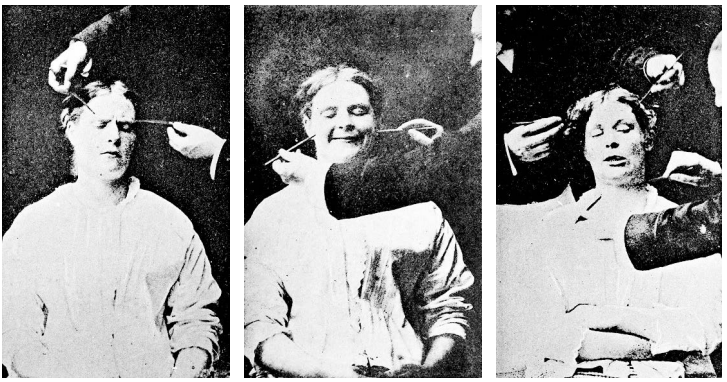
of lethargy were "absolutely identical" to the results obtained by Duchenne.[451] In other words, according to Charcot, the photographs demonstrated that using simple mechanical excitation, he was able to elicit in his hypnotised patients the same isolated contractions of the facial muscles Duchenne had induced in his waking subjects through electricity. Yet, why did Charcot make such an elaborate effort to translate Duchenne's experiments on facial expressions into the context of hypnotic lethargy and thus obtain what he regarded as absolutely identical visual results?

Figure 1.13. Photographs of targeted facial contractions induced through simple mechanical excitation during the hypnotic state of lethargy. Left: bilateral contraction of the orbicularis oculi muscle ('attention'). Middle: bilateral contraction of the zygomaticus major muscle ('false laughter'). Right: simultaneous contractions of the platysma and frontalis muscles ('terror'). From: Charcot, Oeuvres complètes, vol. 9, plate 5, fig. 4; plate 7, fig. 1; and plate 9, fig. 1.



To answer this question, I argue that we must first uncover the new meaning that the photographs of the artificially induced facial expressions acquired in Charcot's hypnotic experiments. We have discussed previously that Duchenne's aim in experimentally inducing and then photographing various combinations of muscular contractions in the face was to determine which and how many individual muscles gave rise to a particular emotional expression. Duchenne, therefore, regarded the muscular contractions captured by the photographs as "the characteristic signs of the emotions," even when such contractions were artificially induced.[452] By contrast, I have shown that

451 One striking visual difference, as Charcot admitted, was that in the photographs of his hypnotic experiments, the eyes of the subjects were always closed. This was an unavoidable feature of hypnotic lethargy. See Charcot and Richer, 373. In one experiment, Charcot opened the patient's eyes to complete the expression of terror he had induced in her face through mechanical excitation. Due to this intervention, the patient immediately shifted to the state of catalepsy. Nevertheless, as Charcot claimed, her expression remained unaltered. See ibid., 373, and plate 9, fig. 2.

452 Duchenne de Boulogne, *Facial Expression*, 19.

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Charcot had little interest in the emotionally expressive aspects of the experimentally induced actions of the facial muscles. Instead, I have already suggested that the face was primarily of interest to Charcot because it allowed him to avoid complex anatomical relations and synergistic connections that characterised the muscular activity in the rest of the body. Even more importantly, the fact that he was able to induce the same facial expressions as Duchenne had meant for Charcot, first and foremost, one thing. It confirmed that he succeeded in producing clearly isolated mechanical excitations of each facial muscle's designated election point without affecting any of the neighbouring tissue (see fig. 1.12).

To understand why this, in turn, was so important for Charcot, we have to remind ourselves of the discovery Remak and Ziemssen had made about the nature of the election points. As mentioned earlier, Remak and Ziemssen claimed, and Charcot agreed, that peripheral nerves entered into the body of the respective muscle at the election points. By taking this into account, the following can be said about the photographs of the artificially induced facial expressions of Charcot's patients in the state of lethargy. These photographs, I argue, demonstrated that the resulting muscular contractions arose from the isolated excitation of the peripheral nerves that entered into each of these muscles at their respective election points. Hence, the photographs delivered empirical support for Charcot's initial conjecture that neuromuscular hyperexcitability was not a direct effect of the mechanical excitation of the muscles but instead of the muscular nerves. Put differently, these photographs were Charcot's most explicit evidence that the phenomenon of neuromuscular hyperexcitability had a distinct neural basis. However, as underscored by my detailed analysis, this evidence was highly mediated since it was generated through elaborate and protracted procedures of intermedial and intramedial transcriptions.[453] Specifically, I have shown that, on the one hand, the neurological meaning of these photographs was constructed through intramedial references to images stemming from Duchenne's experiments. On the other hand, the ascription of a distinct neurological meaning to Charcot's photographs hinged on the intermedial references to the findings made by Remark and Ziemssen about the nature of Duchenne's election points.

Having thus indirectly demonstrated the neural nature of contractures induced through simple mechanical excitation during hypnotic lethargy, Charcot and Richer were nevertheless one step away from their stated goal. At this point, they were still unable to identify what kind of functional neurological disturbance gave rise to neuromuscular hyperexcitability. Therefore, in the next step, Charcot and Richer focused on elucidating the neurophysiological basis of neuromuscular hyperexcitability. As a starting point in this segment of their enquiry, Charcot and Richer introduced a proposition that neuromuscular hyperexcitability and increased tendon reflexes could be mutually related.[454] Not only did these two phenomena typically co-occur during hypnotic lethargy, but they also both involved a pathological modification of motor function. Moreover, in 1875, the German neurologist Wilhelm Erb had posited that

453 See Jäger, "Transcriptivity Matters," 53–54.

454 Charcot and Richer, "L'hypnotisme chez les hystériques," 313–14. I am using the term proposition here in Latour's sense. See Latour, *Pandora's Hope*, 141–44.

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all tendon reflexes in the normal state arose from the automatic action of the spinal cord.[455] This was of interest to Charcot as he already assumed that the spinal cord might be implicated in the production of contractures. Charcot based this assumption on two things. First, he drew on the widely accepted view that the normal muscular tone (i.e., the residual tension that all healthy muscles had at rest) was controlled by the automatic action of the spinal cord.[456] Second, based on his multiple clinical observations, Charcot began to suspect that a contracture was nothing else but a pathological exaggeration of the affected muscles' normal tone.[457]

To articulate their proposition about the potential relation between neuromuscular hyperexcitability and increased tendon reflexes during hypnotic lethargy, Charcot and Richer devised another series of experiments. The purpose of these experiments was to test if they could produce artificial contractures by using a percussion hammer to elicit various tendon reflexes in their hypnotised patients. In healthy individuals, a light but sharp tap with a percussion hammer on the designated tendon in the knee, ankle, wrist or elbow provoked a single involuntary jerk (i.e., contraction) of the respective muscle in the arm or leg.[458] The jerk was then immediately followed by the relaxation of the contracted muscle. However, as mentioned earlier, Charcot had already established that the exaggeration of tendon reflexes was one of the typical features of hypnotic lethargy.[459] This meant that, during lethargy, muscular contractions elicited by light blows to the patients' tendons either lasted longer or were more intense than in their waking state. Charcot and Richer conjectured that such a modification of the muscular action during lethargy possibly indicated a latent tendency towards contracture. They, therefore, decided to test if by increasing either the number or the intensity of the blows, they could produce an actual contracture. Importantly, to be able to compare and thus analyse the distinct effects their targeted manipulations of the tendon reflexes had on the resulting muscular action, Charcot and Richer once again reverted to visualising the effects of their experimental interventions.

With this aim in mind, Charcot and Richer deployed Marey's myograph. Using this device, which Étienne-Jules Marey had developed in the late 1860s, Charcot and Richer were able to mechanically translate experimentally induced changes in the intensity

455 Erb, "Über Sehnenreflexe," 794–97. I will return to this point later in this section.

456 See Charcot and Richer, "L'hypnotisme chez les hystériques," 416.

457 See Charcot, "L'hypnotisme en thérapeutique," 467. For details regarding the late-nineteenth-century views on the physiological basis of the muscular tone, see, e.g., Ferrier, *Functions of the Brain*, 22.

458 Charcot and Richer, "L'hypnotisme chez les hystériques," 314–15. As Charcot explained, several conditions were required to produce a tendon reflex in the normal state. First, the muscle to be acted upon had to be placed in a state of moderate tension. Second, the excitation on the tendon had to be elicited by a sudden yet light blow (i.e., percussion). Finally, reflex muscle contractions could not be produced by any electrical or mechanical excitation other than percussion. Ibid., 314. These conditions for inducing and testing tendon reflexes were first defined independently of each other by Wilhelm Erb and Carl Westphal in 1875. See Erb, "Über Sehnenreflexe," 793; and Westphal, "Bewegungs-Erscheinungen," 803–6.

459 Charcot and Richer, "L'hypnotisme chez les hystériques," 315.

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of the patients' muscular contractions into graphic inscriptions.[460] Marey's myograph was composed of several parts. The part of the device called the myographic drum was directly attached to the muscle of interest. This drum registered the changes in the muscular contractions and transmitted the resulting movement to another drum with which it was connected via a rubber tube.[461] The other drum was equipped with a stylus, which inscribed the transmitted movement onto a uniformly rotating cylinder covered with a smoke-blackened paper. As a result of this configuration, the changes in the muscular contraction were translated into an undulating, continuous curve.[462]

A rise in the curve indicated an increase in the muscle's contraction. Conversely, the curve's subsequent ascent to the baseline level signified muscular relaxation. A visual indication that a contracture had taken place was a curve that ascended to a peak and then remained more or less flat at this elevated level.[463] That is, in the case of a contracture, the curve exhibited a plateau instead of returning to the baseline. Depending on the temporal duration of such a plateau, Charcot and Richer differentiated between a permanent contracture and a more transient one, which they called a "sketch of a contracture."[464] Moreover, the height of the plateau relative to the baseline provided information about the intensity of the contracture. Hence, myographic inscriptions enabled Charcot and Richer to precisely trace and quantify the effects of their experimental interventions.

Applying the myographic drum to their hypnotised patients' forearms and then tapping their tendons at the level of the elbow or slightly below the wrist, Charcot and Richer generated multiple graphic tracings.[465] Based on the visual analysis of such tracings, Charcot and Richer established that several very light blows repeated in a row were sufficient to gradually produce a permanent contracture of the arm (fig. 1.14).[466] It is worth emphasising the following point. The resulting curves provided Charcot and Richer with a continuous recording that visualised the entire dynamic process of the contracture production. This continuous recording, in turn, enabled them to analyse the extent to which each percussion blow contributed to the formation of the resulting contracture. By reading the curves, Charcot and Richer concluded that the first tap of the hammer already induced a slightly prolonged contraction or a 'sketch of a contracture.'[467] The curves thus provided clear-cut empirical evidence for their

460 For detailed descriptions of different versions of myographs and their experimental uses, see Marey, *Méthode graphique*, 192–202, 508–38. For a succinct analysis of various inscription devices Marey developed and then systematically applied in his physiological studies, see Rabinbach, *Human Motor*, 84–103.

461 See Marey, *Méthode graphique*, 201–2. The drum consisted of an air-filled metal capsule covered by a thin rubber membrane. Movements of the limb to which this tambour was attached caused a change in the pressure on the rubber membrane. Thus, the bodily motion was translated into the vibrations of the tambour's membrane. For details, see also Braun, *Picturing Time*, 20–22.

462 Charcot and Richer, "L'hypnotisme chez les hystériques," 317.

463 Charcot and Richer, 320.

464 Charcot and Richer, 320.

465 Charcot and Richer, 317–28.

466 For additional curves obtained through this intervention, see Charcot and Richer, 323, 326.

467 Charcot and Richer, 320.

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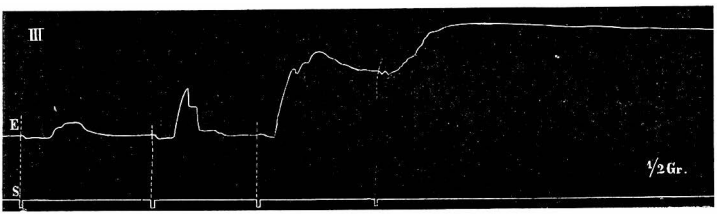
previously posited conjecture about the hypnotised patient's latent tendency towards developing a contracture. The curves also showed that the subsequent blows of the hammer had a more significant effect on producing the contracture than the initial ones, suggesting "a sort of accumulation of force and successive addition of each partial excitation."[468] Building upon these image-based insights, Charcot and Richer devised further experimental interventions, which led to additional discoveries. For example, by increasing the tapping intensity and analysing the curves they obtained, Charcot and Richer established that a contracture could be induced more quickly with more vigorous blows.[469]

Figure 1.14. Graphic tracing showing the production of a permanent contracture of a muscle through four successive blows with a percussion hammer on a patient's tendon during hypnotic lethargy. Dashed vertical lines denote the moments at which each blow was dealt. From: Charcot, Oeuvres complètes, vol. 9, 324, fig. 4.



However, both the increase in the intensity and the number of blows required to induce a permanent contracture had one unwanted side effect. Both interventions led to a diffusion of excitation, thus eliciting uncontrolled contractions and contractures in other parts of the patient's body.[470] Charcot regarded such uncontrolled indirect effects as noise in his experimental setup. To avoid them, he decided to dispense with the percussion hammer and instead apply continuous light pressure to his patients' tendons using a stick.[471] Yet, this also meant that, from the operational point of view, the phenomenon he was now inducing was not a tendon reflex.[472] Instead, in this latter case, Charcot was eliciting a muscular response to a prolonged mechanical excitation of the tendon.

Revealingly, the shape of the resulting myographic curve showed that light pressure on the patient's tendon at the wrist level led to a swift formation of a high-intensity permanent contracture of the forearm (fig. 1.15). In effect, this curve visualised a clear-

468 Charcot and Richer, 321.

469 Charcot and Richer, 321.

470 Charcot and Richer, 321. The diffusion of excitation was demonstrated by simultaneous graphical recording Charcot generated by applying multiple myographs to his patients' legs and arms. See ibid., 326, 328.

471 Charcot and Richer, 333.

472 According to the definition posited by Erb and Westphal, tendon reflexes could only be elicited by a light yet sharp blow and not through prolonged pressure. See Erb, "Über Sehnenreflexe," 793.

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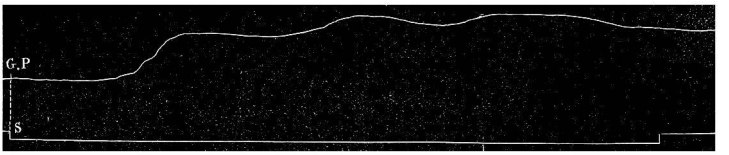
cut manifestation of the phenomenon of neuromuscular hyperexcitability. In other words, it visualised the production of a contracture that was induced through indirect mechanical stimulation of a muscle via its tendon.[473] More importantly, this curve provided a novel insight that, during hypnotic lethargy, a simple pressure on the tendon produced the same muscular action as the repeated swift blows with the percussion hammer.[474] Crucially, with the two curves (figs. 1.14 and 1.15), Charcot and Richer succeeded in articulating their initial proposition that neuromuscular hyperexcitability and exalted tendon reflexes were two mutually related phenomena. Based on the visual similarity of the two curves, Charcot concluded that more than merely being related, neuromuscular hyperexcitability and exalted tendon reflexes were "phenomena of the same order."[475] The thus posited equivalence, in turn, allowed Charcot to claim that exalted tendon reflexes and neuromuscular hyperexcitability shared the same neurophysiological mechanism.[476] It is difficult to overstate the importance of this claim since, in the next step, it enabled Charcot to postulate a neurophysiological mechanism underlying the production of hysterical contractures.

Figure 1.15. Graphic tracing showing the production of a permanent contracture of a muscle through prolonged light pressure on a patient's tendon during hypnotic lethargy. From: Charcot, Oeuvres complètes, vol. 9, 333, fig. 7.



In doing so, Charcot drew on the explanation the German neurologist Wilhelm Erb put forth in 1875 concerning the nature of the knee jerk and all other muscular contractions elicited by a slight blow to a tendon.[477] Erb argued that all such contractions arose from the reflex action of the spinal cord and, therefore, represented automatic, involuntary responses of the nervous system to external stimuli.[478] In

473 Charcot and Richer, "L'hypnotisme chez les hystériques," 331.

474 Charcot and Richer, 333.

475 Charcot and Richer, 334.

476 Charcot and Richer, 409.

477 Charcot and Richer, 409. Wilhelm Erb was the first to introduce the term tendon reflexes to designate the thus elicited muscular contractions. See Erb, "Ueber Sehnenreflexe," 792.

478 Erb, "Ueber Sehnenreflexe," 793–95. By contrast, Erb's colleague Westphal maintained that a muscular contracture induced by a blow to a tendon resulted from the direct propagation of the irritation from the tendon to the muscle fibre. In other words, Westphal claimed that tendon reflexes did not involve any action of the nervous system. See Westphal, "Bewegungs-Erscheinungen," 809–10. Erb's and Westphal's opposing views led to a protracted debate in the scientific community. This debate was resolved in 1891 by the English neurologist Charles Sherrington, who demonstrated the validity of Erb's view. See Finger, *Minds Behind the Brain*, 222–23.

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neuroanatomical terms, Erb's explanation built upon and expanded the notion of the diastaltic arc. Initially, the notion of the diastaltic arc was introduced in the 1830s by the British physiologist Marshall Hall to designate a distinct neural pathway that underpinned all spinal reflexes.[479] Significantly, in Hall's view, the reflex action of the spinal cord was the fundamental neurophysiological principle that informed the entire functioning of the nervous system. Consequently, Hall insisted that "all muscular system function, other than that owing to volition, respiration, or irritability, and excluding cardiac action, were dependent" on reflex activity.[480]

According to Hall, the diastaltic arc was made up of two types of peripheral nerves that converged in the nervous centres located in the spinal marrow.[481] Specifically, the arc consisted of the afferent (i.e., sensory) nerves that sent a signal about an external stimulus being detected in one part of the body to the designated nervous centres in the spinal cord. The spinal nervous centres then initiated a response, which was sent via the efferent (i.e., motor) nerves to a muscle at the site of the excitation, thus eliciting its contraction. The crucial point was that because the resulting reflex movement was initiated through the autonomous action of the spinal nervous centres and without any participation of the brain, it occurred independently of the subject's will.[482] Moreover, Hall emphasised that the stimulus which triggered a reflex response could not induce any conscious sensation because the sensory information about its presence was not relayed to higher centres of the brain.[483] Hence, in this view, spinal reflexes were purely mechanical motor responses to external excitation, independent of the will, sensation, and consciousness.

Notably, in Hall's account, the afferent segment of the diastaltic arc consisted exclusively of the sensory nerves of the skin.[484] Conversely, based on his research into tendon reflexes, Erb posited the existence of two distinct, functionally entirely independent diastaltic arcs.[485] One of these arcs entailed the sensory nerves of the skin. Thus, this arc was responsible for spinal reflexes that arose in response to the stimulation of the skin. The afferent segment of the other diastaltic arc consisted of the sensory nerves originating from the muscles and tendons. According to Erb, it was the autonomous activity of this latter arc that underpinned all tendon reflexes.[486]

Drawing on Erb, Charcot conjectured that the proposed mechanism of "the muscular diastaltic arc" could be invoked to explain both normal and exaggerated

479 Clarke and Jacyna, *Origins*, 116.

480 Clarke and Jacyna, 117. For a detailed analysis of the historical evolution of the concept of reflex action in the nineteenth century and the role Marshall Hall played in it, see ibid., 101–24. For a comprehensive analysis of the historical development of the concept of reflex action from the seventeenth century onwards, see Fearing, *Reflex Action*.

481 Hall, *Diastaltic Nervous* System, 35.

482 Hall, *Memoires on the Nervous System*, 10.

483 Hall, 10.

484 Hall, 47. See also Hall, *Diastaltic Nervous* System, 35.

485 Erb, "Ueber Sehnenreflexe," 802.

486 Based on his experimental results, Erb showed that tendon reflexes could not be elicited through mechanical stimulation of the skin. See Erb, 794–96. He thus delivered empirical proof that the sensory nerves of the skin could not participate in the production of tendon reflexes.

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tendon reflexes, as well as the equivalent phenomenon of neuromuscular hyperexcitability.[487] More specifically, Charcot asserted that the only difference between neuromuscular hyperexcitability, on the one hand, and the normal reflex action, on the other hand, consisted in a functional pathological modification that the nervous centres in the spinal cord underwent during the state of lethargy.[488] Notably, Charcot could not provide any direct evidence for the existence of such a functional modification, which he designated as a dynamic lesion to emphasise its presumed non-organic character. Instead, by summarising the findings of his hypnotic experiments, Charcot hypothesised that this functional modification consisted in excessive excitability of those nervous centres in the spinal cord, which presided over tendon reflexes.[489]

In support of his conjecture, Charcot argued that because the spinal nervous centres controlled the normal muscular tone, their excessive excitability could explain why even the slightest mechanical excitation of muscles or tendons during the hypnotic lethargy led to the formation of enduring spasmodic contractures.[490] Furthermore, Charcot pointed out that, under normal conditions, the same spinal centres also regulated a balanced and mutually coordinated activity of both synergistic and antagonistic muscles. Hence, the exaggerated excitability of these centres could be responsible for two particular effects demonstrated by his experiments. First, the existence of a dynamic lesion of the spinal cord explained why the excitation applied to a single muscle induced concurrent contractures in several other synergistic muscles.[491] Second, it was because of functional connections between antagonistic muscles in the spinal cord that it was possible to resolve a contracture by applying moderate pressure to the muscles antagonistic to those that were permanently contracted.[492] In short, according to Charcot, a hypothesised dynamic lesion of the spinal cord, which consisted in the abnormal irritability of its nervous centres, could account for all the experimental results discussed in this section.

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487 Charcot and Richer, "L'hypnotisme chez les hystériques," 421. Charcot used the term 'muscular diastaltic arc' to refer to the neural pathway understood to underpin the tendon reflexes. This arc entailed: first, the sensory nerves of the muscles and tendons; second, the nervous centres in the spinal marrow; and third, the motor nerves. See ibid., 411. Erb's introduction of a distinction between skin and tendon reflexes was crucial for Charcot. As mentioned previously, Charcot insisted that the patient's skin sensibility was entirely abolished during lethargy. The absence of skin sensibility, in turn, meant that, while in this hypnotic state, the patient could not have any skin reflexes. Since skin and tendon reflexes were entirely independent of each other, if one type was absent, the other could nevertheless continue to exist or even be exalted. Ibid., 421. In effect, Charcot posited that, in the state of hypnotic lethargy, mechanical excitation applied to a muscle or its tendon became registered by their designated sensory nerves and then communicated to the nervous centres in the spinal cord. Here, the sensory impression elicited a reflex response. This response was then conveyed to the muscle, which had been exposed to the mechanical excitation, causing the muscle to contract. Ibid., 417.

488 Charcot and Richer, 411.

489 Charcot and Richer, 411.

490 Charcot and Richer, 407.

491 Charcot and Richer, 409.

492 Charcot and Richer, 408.

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Finally, Charcot stated that he had made another critical discovery in the course of his experiments. He established that many of his hysteria patients exhibited an indication of neuromuscular excitability even in their waking state.[493] This was demonstrated by the fact that a sudden movement, prolonged massage, or a light blow often sufficed to produce permanent contractures of their limbs.[494] In other words, Charcot asserted that even hysteria patients who did not have an actual contracture nevertheless exhibited an inherently pathological tendency to develop contractures, which he termed 'contracture diathesis.' The contracture diathesis was nothing else but a continually present, attenuated form of neuromuscular excitability, which then merely became artificially intensified during the state of hypnotic lethargy.[495] With this statement, Charcot declared neuromuscular excitability, albeit in its attenuated form, a permanent symptom of hysteria. At the same time, he also effectively declared the hypothesised functional lesion of the spinal cord, which underpinned neuromuscular excitability, to be the underlying neurophysiological mechanism of all hysterical contractures. In the process, Charcot redefined hysterical contractures as excessive reflex responses of the overexcited spinal nervous centres to even the slightest external stimuli.

Furthermore, it appears to me that Charcot's claim about hysteria patients' muscles and nerves being in the state of permanent over-responsiveness to external stimuli had broader implications. Although Charcot did not explicitly state this, it is conceivable that he held the same functional lesion of the spinal cord responsible for various 'illogical' spasmodic convulsions, which took place during the hysterical attack. In effect, such 'illogical' convulsions were nothing else but a combination of multiple involuntary contractions that simultaneously affected different parts of the patient's body. Just as importantly, Charcot and his team repeatedly and explicitly linked both the occurrence and the sudden disappearance of permanent contractures to the onset of the patients' hysterical attacks.[496] This suggests that, in their view, convulsive aspects of the hysterical attack and permanent contractures were two mutually related phenomena. Hence, it is safe to assume that they regarded these two phenomena to rely at least in part on a shared neural basis.

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To summarise, in this section, I have traced the process through which Charcot arrived at his novel conceptualisation of hysterical contractures as abnormal reflex responses of the spinal cord. We have seen that this new insight was obtained through a systematic step-by-step experimental decomposition of the phenomenon of neuromuscular hyperexcitability into its constituent neurophysiological components. This decomposition first focused on demonstrating the fundamentally neurological nature of contractures artificially produced during hypnotic lethargy. To achieve this

493 Charcot and Richer, 406.

494 Charcot, "Lecture 8: Contracture of Traumatic Origin," 90.

495 Charcot and Richer, "L'hypnotisme chez les hystériques," 406.

496 See Charcot, "Lecture 12: Hysterical Contracture," 288–89; and Bourneville and Regnard, *Iconographie photographique*, 1:21, 60, 63, 83, 93.

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goal, Charcot and Richer deployed photography as an experimental condition and drew extensively on the neurophysiological experiments of their older colleague Duchenne de Boulogne. Having used photography to provide indirect empirical evidence for the neural nature of muscular contractions and contractures in the state of lethargy, Charcot and Richer then proceeded to the next experimental stage. Based on the experiments in which they used Marey's graphic method, Charcot and Richer were finally able to link hypnotically induced, and by analogy, also spontaneously developed hysterical contractures to a functional disturbance of the spinal cord. This, I suggest, was a crucial milestone in Charcot's image-based hysteria research. It marked his initial success in developing an admittedly tentative yet plausible neurophysiological explanation for the somatic basis of a hysterical symptom. Moreover, in the course of the experiments discussed in this section, Charcot's initially abstract notion of functional lesion began to take a more concrete shape. At least concerning hysterical contractures, the lesion now attained a location within the nervous centres of the spinal cord and became defined in functional terms as a permanent state of hyperactivity.

1.2.2 Linking Hysteria to the Aberrant Reflex Action of the Brain

In the previous section, we have discussed how by systematically visualising and analysing hysteria patients' neuromuscular responses to various experimental interventions during hypnotic lethargy, Charcot causally linked hysterical contractures to overexcited spinal reflexes. Importantly, we have also seen that such reflexes were understood to be entirely automatic responses of the spinal cord to external stimuli, which happened without any involvement of the brain. Having attributed hysterical contractures to a disturbance of spinal reflexes, Charcot thus effectively foregrounded the involuntary nature of this symptom. In what follows, I will show that a series of experiments Charcot conducted on his patients during hypnotic catalepsy had comparable although somewhat broader epistemic aims. In this case, instead of focusing on a single symptom, Charcot aimed to link more complex physical manifestations of hysteria to functional disturbances of higher-order brain centres. Another equally important aim of Charcot's experiments on cataleptic patients, I will argue, was to emphasise, albeit implicitly, the involuntary nature of hysteria, on the whole. With a view to achieving these aims, Charcot once again deployed photography and Marey's graphic method. To reveal how the resulting images were able to fulfil their intended epistemic functions, my analysis will reconstruct the neurological concepts and theories that informed the ways in which the Salpêtrians produced and interpreted these images. But before turning to the analysis of the experiments, we first need to take a look at how Charcot defined the state of hypnotic catalepsy.

In many ways, catalepsy and lethargy were two mutually contrasting hypnotic states. Charcot insisted that, contrary to lethargy, both the exaggerated tendon reflexes and neuromuscular hyperexcitability were absent during catalepsy.[497] This already indicated that the mechanism of spinal reflexes, which Charcot had declared to underpin the neuromuscular hyperexcitability, could not be responsible for any of the

497 Charcot and Richer, "Cerebral Automatism," 3.

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hypnotised patients' muscular responses during catalepsy. Moreover, during lethargy, the patients' limbs were rigid and fell down if forcefully lifted by the experimenter. In contrast, during catalepsy, all of the patients' body parts became light and flexible and offered no resistance to passive movements the experimenter wished to impose on them.[498] Hence, the experimenter could easily place cataleptic patients into any posture he chose. The patients then remained in this posture until the experimenter decided to reposition their bodies. Charcot declared such immobility "to be the most pronounced characteristic of the cataleptic state."[499] He even emphasised that the cataleptic immobility—i.e., the reduction of muscular activity—affected all of the patients' physiological functions. They winked only infrequently during the cataleptic state, their pulse was low, and their breathing was slow and shallow.[500]

Finally, although the skin of cataleptic patients remained as insensible to impressions as it was during lethargy,[501] the activity of their senses was partially awoken. As a result, some patients became more or less responsive to impressions they received through the senses of sight, hearing, or smell.[502] However, one feature most patients had in common during catalepsy was that their muscular sense regained almost all of its activity.[503] The notion of the muscular sense as the "sixth sense" (in addition to sight, hearing, touch, taste, and smell) was introduced by the Scottish physiologist Charles Bell in the 1820s.[504] As we will see later in this section, the muscular sense played a central role in Charcot's experiments on cataleptic patients. It is, therefore, necessary for our subsequent discussion that we examine how the muscular sense was understood in the 1880s when Charcot performed his experiments.

As defined by Bell, the muscular sense was a sense in its own right that yielded information about the position and movements of our body. Bell posited its existence based on his discovery that, apart from a motor nerve, which "*conveys the influence from the brain to the muscle*," each muscle also had a designated sensory nerve.[505] In Bell's view, the muscular sensory nerves were anatomically and functionally distinct from the sensory nerves of the skin. Therefore, muscular sensory nerves could not provide tactile impressions. Rather, Bell conjectured that the muscular sensory nerves conveyed to the brain the information about "the degree of action" of muscles, such as, for example, different intensity of their contractions.[506] In effect, Bell thus introduced a distinction between the senses that registered external stimuli (e.g., touch or sight) and the muscular sense as the source of awareness about the internal conditions of

498 Charcot and Richer, 3.

499 Charcot and Richer, 3.

500 Charcot and Richer, 3. See also Charcot and Tourette, "Hypnotism in the Hysterical," 607.

501 Charcot and Tourette, "Hypnotism in the Hysterical," 607.

502 Richer, *Études cliniques*, 2nd ed., 662.

503 Richer, 662.

504 Bell, *Hand*, 195. For a contemporary account of the history of the muscular sense, see Smith, "Sixth Sense."

505 Bell, "Nervous Circle," 170 (emphasis in original). Incidentally, Bell's discovery of the functional distinction between sensory and motor nerves served as the basis for the theories of reflex action discussed in the previous section. For details, see Clarke and Jacyna, *Origins*, 110–12.

506 Bell, *Hand*, 188.

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the muscles. By the 1830s, the existence of the muscular sense, understood as the "sense, whose objects are sensations attached to the movements of the body, or to the action of the muscles," became widely accepted in scientific circles.[507] But apart from this general designation, there was little agreement among leading nineteenth-century physiologists about any other aspect of the muscular sense. Hence, throughout the nineteenth century, a heated debate persisted about the neurological basis of the muscular sense.[508]

On one side of this debate, the German physiologist Wilhelm Wundt and the Scottish philosopher Alexander Bain rejected Bell's conjecture that the muscular sense was derived from impressions passing from the contracted muscles to the brain. Instead, Bain suggested that since "the [voluntary] muscular movements are stimulated from the brain and nerve centres, our safest assumption is, that the sensibility accompanying muscular movement coincides with the *outgoing* stream of nervous energy" by which the muscles were induced to act.[509] Similarly, Wundt attributed the muscular sense to sensations that, as he claimed, accompanied the discharge of the nervous current (i.e., "the innervation") from the motor centres of the brain in which a voluntary movement had been initiated.[510] Simply put, both Bain and Wundt conjectured that the origin of the muscular sense was not in the muscles and their afferent (i.e., sensory) nerves but in the motor centres of the brain and the efferent (i.e., motor) nerves. This had two significant consequences. First, in this view, the muscular sense was linked exclusively to voluntary movements. Understood in this way, the muscular sense was purported to play no role in passive movements or any motion that was not initiated by what Wundt called a volitional impulse ("Willensimpuls").[511]

Second, both Bain and Wundt detached the muscular sense from any physical sensation that arose from muscular action. They tied it instead to a consciousness of voluntary effort that accompanied an active initiation of movement. According to Wundt, the subjective awareness of effort consisted in the sensation of the force that the subject exerted to initiate the volitional impulse. Thus defined, the sense of effort was independent of the actual performance of a movement.[512] In support of this claim, Wundt argued that even patients with paralysis experienced effort when they tried but failed to move their affected limbs. Similarly, Bain attributed the experience of effort to the mind's ability to discriminate "the degree of energy of the motor current, or the force poured out from the brain in voluntary movement."[513] To sum up, in this

507 Ribot, *English Psychology*, 199. See also Smith, "Sixth Sense," 233.

508 See, e.g., Smith, "Sixth Sense," 259–62.

509 Bain, *Sense and Intellect*, 76–77. Similar views were also held by the influential German physiologist Johannes Müller and the English neurologist Hughlings Jackson. For a succinct overview of their views, see James, "Feeling of Effort," 152–53.

510 Wundt, *Grundzüge*, 1:375. Wundt introduced the term "Innervationsempfindung" (i.e., the sensation of innervation) to designate a purported awareness that accompanied the efferent discharge of the motor centres of the brain. Ibid.

511 Wundt, 376. See also ibid., 2:17; and Bain, *Sense and Intellect*, 77. Passive movements are imparted to a subject by another person and are devoid of any voluntary intervention on the subject's part.

512 Wundt, *Grundzüge*, 1:375.

513 Bain, *Sense and Intellect*, 77–78.

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interpretation, the muscular sense did not provide information about the changing physical conditions of the muscles. Instead, it hinged on the feeling "of power going out of us" during intended voluntary action, regardless of whether an actual movement took place or not.[514]

On the other side of the debate, the neurologists David Ferrier and Charlton Bastian, and the philosopher William James contested that we could be conscious of the efferent discharge of the nervous current from our cortical motor centres.[515] In contrast, they insisted that the muscular sense was derived from afferent impressions that were "a consequence and not an antecedent of the movement itself."[516] But far from merely restating Bell's initial views, they declared that the muscular sense consisted of a complex assemblage of various kinds of peripheral sensory impressions induced by a movement. In their view, in addition to the afferent impressions coming from the muscles, the muscular sense also comprised sensory impressions arising from the accompanying "stretching of tendons, ligaments, and skin, and the rubbing and pressing of joints."[517] Ferrier posited that all such peripheral impressions were transported via afferent nerves to the brain's sensory centres, where they jointly gave rise to the conscious discrimination of the movement performed.[518]

Understood as being dependent on complex incoming sensory impressions and not an outgoing nerve current, the muscular sense was no longer limited to voluntary movements. Thus reinterpreted, the muscular sense could also play a role during passive movements by yielding sensory information about the externally imposed changes in one's posture.[519] This reinterpretation, as I will show at a later point, was significant for Charcot's experiments. Just as importantly for Charcot, both Ferrier and James continued to explicitly link the activity of the muscular sense to the subjective experience of effort, but only in voluntary movements. Yet, unlike Bain and Wundt, Ferrier and James asserted that the consciousness of muscular exertion (i.e., effort) "must be an afferent [i.e., incoming] and not an efferent [i.e., outgoing] sensation."[520] Ferrier and James forcefully argued that the experience of effort was "impossible without a movement *effected somewhere*."[521]

514 Bain, 79.

515 See Ferrier, *Functions of the Brain*, 219–22; Bastian, *Organ of Mind*, 541–44, 554–57, 691–700; and James, "Feeling of Effort," 152–80. James explicitly stated that "the motor discharge ought to be devoid of sentience." James, "Feeling of Effort," 157. He even went so far as to designate Wundt's concept of the sensation of innervation ('Innervationsempfindung') "as a pure encumbrance." James, 159.

516 James, "Feeling of Effort," 168.

517 James, 159. See also Ferrier, *Functions of the Brain*, 218; and Bastian, *Organ of Mind*, 543, 695.

518 Ferrier, *Functions of the Brain*, 226–27. Unlike Ferrier, Bastian claimed that only the sensory components derived from the skin, ligaments and joints were conscious, whereas the afferent inputs from muscles always remained unconscious. Bastian, *Organ of Mind*, 543. Moreover, Bastian and Ferrier disagreed about the exact anatomical localisation of the sensory centres in which the various impressions comprising the muscular sense were supposed to be registered. See Bastian, 543.

519 See, e.g., Maudsley, *Physiology of Mind*, 488.

520 James, "Feeling of Effort," 168.

521 James, 167–68 (emphasis in original).

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To prove his point, Ferrier asked his reader to perform a simple experiment. The reader was instructed to extend his right arm and hold "his forefinger in the position required for pulling the trigger of a pistol" but to refrain from actually moving the finger.[522] Ferrier contended that "by simply making believe" that he was moving his finger, the reader would experience a sense of effort even without any contraction of the muscles in the hand taking place.[523] However, if the reader were to "pay careful attention to the condition of his respiration, he will observe that his consciousness of effort coincides with a fixation of the muscles of his chest, and that in proportion to the amount of energy he feels he is putting forth, he is keeping his glottis closed and actively contracting his respiratory muscles."[524] In other words, Ferrier claimed that, whether we actually execute a voluntary movement or merely imagine performing it, we always automatically arrest our breathing by contracting the muscles of the chest. He then posited that the sensory impressions arising from "this essential and ever present respiratory factor" were "the basis of the general sense of effort in all its varying degrees."[525] As will become apparent in the course of this section, Ferrier's linking of voluntary effort to what he termed the respiratory factor was of central importance for one of Charcot's crucial experiments on cataleptic patients.

Charcot did not explicitly participate in the debate on the muscular sense, which remained unresolved when he performed his experiments on cataleptic patients.[526] But based on his statements about the nature of the muscular sense, he apparently subscribed to Ferrier's views. In agreement with Ferrier, and unlike Wundt and Bain, Charcot referred to the muscular sense as consisting of afferent "impressions coming from the periphery, namely, from the skin, muscles," tendons, and joints.[527] Moreover, like Ferrier, Charcot also contended that all these various impressions became jointly registered in the sensory centres of the cerebral cortex.[528]

The fact that Charcot held this view on the muscular sense had significant consequences for his interpretation of hypnotic catalepsy. A particularly significant aspect was that, according to this view, the muscular sense (as well as the senses of sight, hearing, and smell) entailed the activity of the higher cerebral centres. In effect, the revival of the muscular senses during catalepsy meant that hypnotised patients were no longer in a state of complete mental stupor as during lethargy. Instead, Charcot conjectured that the presence of some degree of sensorial activity during catalepsy testified to "a sort of partial waking" of the brain as "the organ of the psychic

522 Ferrier, *Functions of the Brain*, 223

523 Ferrier, 223.

524 Ferrier, 223.

525 Ferrier, 223–24. If a voluntary movement was merely imagined, Ferrier attributed the experience of effort exclusively to the contraction of the respiratory muscles. If the intended movement took place, both the contraction of the chest and the contraction of the muscles performing the voluntary movement contributed to the sense of effort. See ibid., 223.

526 The debate was resolved in the first decade of the twentieth century by the English physiologist C. S. Sherrington. See Smith, "Sixth Sense," 261–62.

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

528 Charcot, 395.

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[i.e., mental] faculties."[529] Consequently, the experimental use of catalepsy permitted Charcot to focus on investigating the aberrant functioning of hysteria patients' higher brain centres. That is, Charcot was no longer limited to using simple mechanical excitation of muscles and tendons as in the hypnotic experiment discussed in the previous section. As he claimed, he could now act on the cataleptic patients' minds by using experimental interventions to produce targeted sensory impressions. The resulting sensory impressions, in turn, induced the patients to perform "more or less complex, and perfectly coordinated" actions to whose analysis we will turn shortly.[530]

However, by claiming that the cataleptic subjects' mental functions were partly restored, Charcot could no longer a priori exclude the possibility that, while in this hypnotic state, his patients were capable of simulation. Hence, Charcot's first experiment focused on proving that a genuine cataleptic state could be reliably differentiated from a wilful simulation.[531] At the centre of this experiment was the aforementioned ability of cataleptic subjects to maintain a posture the experimenter had imposed on them for a long time. According to Charcot, a cataleptic patient whose arm was extended horizontally could keep this position for about ten to fifteen minutes.[532] After this period, his arm would begin to descend, gradually resuming its initial vertical position. But Charcot emphasised that these were "the limits of endurance" that "a vigorous man, endeavoring to preserve the same position" could also attain.[533] Charcot, therefore, warned that based on unaided observation alone, it was impossible to differentiate reliably between a genuine cataleptic subject and a simulator. His solution to this conundrum was to deploy Marey's graphic method.[534]

Specifically, Charcot suggested that to establish a distinction between a cataleptic patient and a simulator, it was necessary to measure the underlying changes in their physiological functions while their arms remained outstretched in the horizontal position. To this end, Charcot developed an experimental setup that entailed a simultaneous use of two of Marey's registering instruments (fig. 1.16). First, Marey's myographic drum, a device already familiar to us from Charcot's previous hypnotic experiments, was attached to each subject's outstretched arm. In this setup, the myograph was meant to register even the smallest oscillations of the subjects' arms.[535] Once registered, the oscillations were transmitted via a rubber tube to a stylus that inscribed them onto a steadily revolving cylinder covered with a smoke-blackened paper. Second, a pneumograph was attached to each subject's chest and, via a rubber tube, connected to a separate stylus. This device had been designed by Marey to measure the rhythmical movement of the chest during breathing and translate it into a curve that provided information about the subject's respiratory pattern.[536] As Marey

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

530 Charcot and Richer, "Cerebral Automatism," 4.

531 Charcot and Richer, 4.

532 Charcot and Richer, 4.

533 Charcot and Richer, 4.

534 Charcot and Richer, 4.

535 Charcot and Richer, 5.

536 For a detailed description of the pneumograph and its use, see Marey, *Méthode graphique*, 202–5, 539–58.

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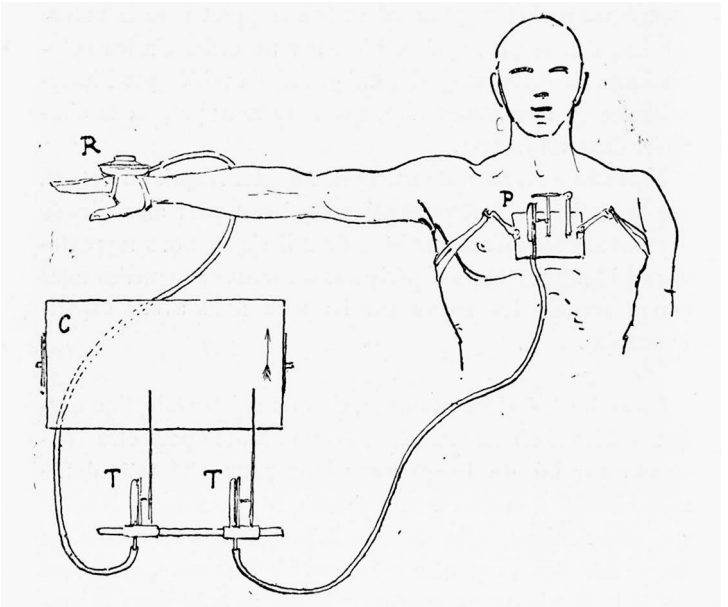
explained, in a curve obtained by his pneumograph, a rising line denoted exhalation and a descending line inhalation.[537] In Charcot's experimental setup, both devices were mutually synchronised so that their respective styli simultaneously inscribed parallel curves onto the same paper. Hence, both measurements were assembled into a single diagram for each subject. The choice of such a setup already implied that Charcot was interested in using the graphic data to visually explore potential correlations between the subjects' trembling of the outstretched arm and their respiratory patterns.

Figure 1.16. Diagram showing the arrangement of the apparatus in the experiment on cataleptic immobility. R: Marey's myographic drum; P: pneumograph; C: revolving cylinder; TT: recording styli. From: Charcot and Richer, "Cerebral Automatism," 5, fig. 1.



The resulting sets of curves disclosed considerable physiological differences between the cataleptic patient and the simulator. The myographic drum applied to the arm of the cataleptic patient traced a continually straight line (fig. 1.17, left, section II). The shape of this line indicated that the patient's arm had remained outstretched without even the slightest tremor. Similarly, the tracing obtained by the pneumograph consisted of an ever so slightly undulating line (fig. 1.17, left, section I). It showed that the patient's breathing was slow and superficial.[538] Moreover, a detail Charcot particularly emphasised was that, in the case of the cataleptic patient, the end of each tracing

537 Marey, 542.

538 As mentioned previously, Charcot regarded such slowing down of the breathing pattern as one of the distinguishing features of the cataleptic state.

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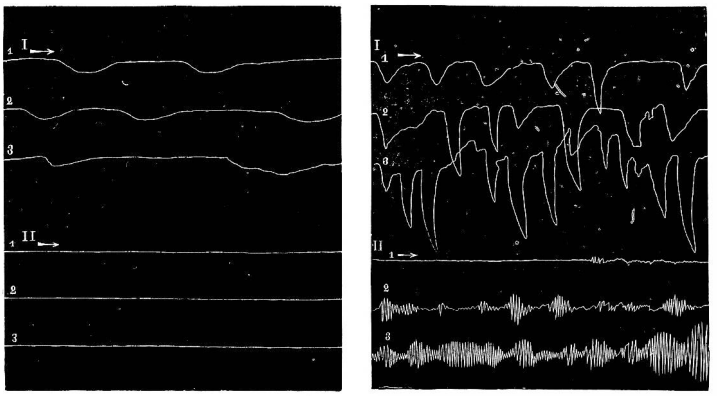
resembled its beginning.[539] Put simply, the shape of the patient's curves remained uniform during the entire experiment.

In contrast, the set of curves obtained for the healthy subject who simulated the cataleptic attitude charted a very different temporal development of the underlying physiological processes. The initial portion of the simulator's myographic tracing was similar to that of the cataleptic patient. However, very quickly "the straight line changes into a line sharply broken and characterized by instants of large oscillations arranged in series" (fig. 1.17, right, section II).[540] These oscillations disclosed the presence of tremors of gradually increasing intensity in the simulator's outstretched arm. Significantly, the simulator's pneumographic curve displayed a correlated visual pattern (fig. 1.17, right, section I). This curve showed that, in the beginning, the simulator's breathing was "regular and normal."[541] But, at the exact moment the tremor set in, the subject's breathing pattern also changed considerably, indicating what Charcot termed the disturbance of the respiratory rhythm.[542] The disturbance consisted in the prolongation and intensification of respiratory movements. The flat-topped sections of the curve disclosed that the subject was repeatedly holding his breath and then, as shown by the dips in the curve, inhaling deeply and rapidly.

Figure 1.17. Left: tracings obtained from a hysteria patient in the state of hypnotic catalepsy. I: pneumographic tracing; II: myographic tracing. Right: tracings obtained from a healthy subject who attempted to maintain the cataleptic attitude. I: pneumographic tracing; II: myographic tracing. Read from left to right in order 1, 2, 3. From: Charcot and Richer, "Cerebral Automatism," 6, fig. 2; and 7, fig. 3.



539 Charcot and Richer, "Cerebral Automatism," 6.

540 Charcot and Richer, 6.

541 Charcot and Richer, 7

542 Charcot and Richer, 7.

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Drawing these results together, Charcot triumphantly concluded that "when submitted to this double test," the simulator was simultaneously "betrayed" by the tracing of the tremor in his arm and by a distinct shape of his pneumographic curve.[543] Even a superficial visual comparison sufficed to make evident the pronounced differences between the two sets of curves produced separately for the cataleptic patient and the simulator. At this point, one might argue that based on close observation alone, the physician could also have noticed the changes in the simulator's breathing rhythm or the tremor of his hand. Yet, first of all, Charcot explicitly chose to use the myograph because this device could "record with mathematical precision" the kind of tremor that was "barely perceptible to the eye."[544] And even more significantly, the synchronised deployment of the myograph and the pneumograph enabled Charcot to determine that the tremor and the breathing irregularity in the simulator developed simultaneously and intensified over time in correlation to each other. Moreover, the curves of the cataleptic subject disclosed with equal 'mathematical precision' the lack of any temporal changes in either his muscular action or his breathing pattern. These specific patterns and relations were not accessible to analysis before their translations into graphic inscriptions. Hence, it can be said that through the combined use of Marey's two inscription devices, Charcot succeeded in making visible clear-cut differences between the cataleptic subject and the simulator, which as such could not have been obtained through unaided observation. The graphic inscription thus delivered decisive empirical proof that hypnotic catalepsy was distinguishable from simulation.

However, this experiment had greater significance in Charcot's hysteria research than it might appear at a superficial glance. I suggest that the reason for this is twofold. First, Charcot contended that the myographic and pneumographic curves could be used effectively for diagnostic purposes, which went beyond mere differentiation between genuine hypnotic catalepsy and intentional simulation. Based on his by now familiar claim that hypnosis and hysteria were mutually analogous morbid conditions, Charcot argued that the same experimental setup could also be deployed to reliably diagnose hysteria by eliminating any suspected "artifice of the patient."[545] To exclude the possibility of simulation, patients merely had to be inducted into the state of catalepsy and submitted to the 'double test.' Based on the analysis of the resulting myographic and pneumographic curves, the physician could then easily and reliably distinguish between genuine hysteria patients and simulators. Charcot primarily foregrounded the clinical diagnostic value of this experiment when he presented it in full detail in the programmatic lecture with which he inaugurated his new professorship in diseases of the nervous system in 1882.[546]

Second, I argue that, in addition to its diagnostic utility, this experiment was also important to Charcot because it enabled him to draw inferences about the higher-order mental processes underpinning intentional simulation, on the one hand, and cataleptic immobility, on the other. This becomes apparent when we take a look at Charcot's

543 Charcot and Richer, 8.

544 Richer, *Études cliniques*, 2nd ed., 616.

545 Charcot, "Lecture 1: Introductory," 18.

546 Charcot, 15–18.

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tersely formulated interpretation of his experimental findings. To begin with, Charcot stated that the irregularities in the myographic tracing of the simulator's extended arm were "indications of muscular fatigue."[547] Charcot then went on to claim that the simulator's accompanying disturbance of respiration expressed "the effort devoted to masking the effects of his muscular fatigue."[548] By contrast, the curves of the cataleptic patient, according to Charcot, gave "no evidence of fatigue."[549] Instead, they showed that the patient's "muscles yield, but without effort, and without the concurrence of the volition."[550] Due to Charcot's cryptic formulation, it is easy to overlook the significance of this last statement. With it, Charcot effectively declared cataleptic immobility to be involuntary. Moreover, since Charcot used the same experiment to differentiate hysteria from simulation, the thus established involuntary character applied not only to cataleptic immobility but also, at this point, at least implicitly, to hysterical symptoms in general.[551]

To a contemporary reader, it may appear surprising that Charcot did not offer any explanation for his interpretation of the myographic and pneumographic curves, which I have just quoted. From the current perspective, it is far from apparent how these tracings (fig. 1.17) could have been taken to indicate either the presence or the absence of muscular fatigue and effort. It is even less evident how these tracings could signify either the involvement or the lack of the subjects' voluntary intervention. However, the matter-of-factness with which Charcot delivered his statements seems to imply that the medical audience he was addressing was well acquainted with the theoretical framework in which his interpretation of the curves was tacitly embedded. Although Charcot did not provide any explicit references, we can reconstruct the theoretical framework that informed his interpretation. To do so, we have to revisit our preceding discussion of David Ferrier's views on the sense of effort. Additionally, we also need to examine how the English physiologist William Carpenter linked the occurrence of muscular fatigue to the investment of voluntary effort and how he attributed the lack of fatigue to what he referred to as automatic actions.[552]

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547 Charcot and Richer, "Cerebral Automatism," 7. It is worth noting that Charcot's experiment, which he for the first time presented in 1882, predated Angelo Mosso's famous physiological research into human fatigue. In 1884, Mosso invented the ergograph, a device with which he systematically generated the so-called fatigue curves of human subjects. See Mosso, *Fatigue*. For a succinct analysis of the nineteenth-century physiological research into fatigue, including the early myographic experiments that Hermann von Helmholz and É.-J. Marey performed on isolated muscles of dead frogs, see Felsch, "Nach oben." For a wide-ranging study of the late-nineteenth and early-twentieth-century conceptions of fatigue, see Rabinbach, *Human Motor*.

548 Charcot and Richer, "Cerebral Automatism," 8.

549 Charcot and Richer, 7.

550 Charcot and Richer, 7–8.

551 Several years later, Charcot used a slightly modified version of this experiment to diagnose a case of hysterical contracture. See Charcot, "Lecture 8: Contracture of Traumatic Origin," 95–98. This time, while interpreting the pneumographic curves, he explicitly stated that in genuine hysterical symptoms, "the will of the patient counts for nothing, absolutely nothing." Ibid., 98.

552 We are already familiar with Carpenter, whom Charcot quoted in his 1872 lecture on hysterical hemianaesthesia. See section 1.1.1. Although Charcot did not quote Carpenter in his hypnosis

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As mentioned earlier, Ferrier defined the sense of effort as an assemblage of conscious sensory impressions induced by the active muscular exertion entailed in a voluntary execution of movement. We also saw that Ferrier explicitly linked the sense of effort to what he termed the respiratory factor, which involved the contraction of the chest muscles. In short, Ferrier argued that volitional acts were typically accompanied by the act of breath-holding, which, in turn, gave rise "to the general sense of effort."[553] If we now take another look at the simulator's respiratory curve, we will see that, for the most part, it disclosed a pattern in which the breath-holding alternated with deep, short inhalations (fig. 1.17, right, section I). This particular pattern is what Charcot designated as "the disturbance of respiration that accompanies the phenomena of effort."[554] Therefore, it appears to me that Charcot's interpretation of this curve was rooted in Ferrier's notion of the respiratory factor as the physiological basis of conscious effort. In this context, it also becomes clear why Charcot attributed the continually uniform breathing pattern of the cataleptic subject to the lack of conscious effort. Since, as we have seen, Ferrier linked the sense of effort to voluntary movement,[555] the absence of effort, in turn, could be taken to signify that the cataleptic subject kept his arm extended without any voluntary intervention.

Further, both Ferrier and Carpenter contended that as "a direct consequence of strained attention and conscious effort" he was investing, a subject performing a volitional act soon experienced a painful sensation of fatigue.[556] The source of this sensation was the physical condition of the overstrained muscles of which the subject became aware through his muscular sense.[557] As stated by Carpenter, once the sensation of fatigue had set in, the subject had to keep increasing his conscious effort to continue executing the voluntary action already in progress.[558] Charcot's claim that the simulator's effort was "devoted to masking" the effects of his muscular fatigue seems to reflect Carpenter's statement.[559] However, as Carpenter further elaborated, the increased effort necessarily led to an even stronger sensation of fatigue. As a result, the subject soon found himself "unable to evoke a respondent movement" from his exhausted muscles.[560] If we apply Carpenter's description to Charcot's experiment, it follows that the continual voluntary effort the simulator had to invest to keep his arm extended resulted in muscular fatigue. Once fatigued, his muscles could no longer maintain the intensity of voluntary contractions necessary for the arm to remain still in the outstretched position. This, in turn, led to unintentional fluctuations in the intensity

research, in what follows, I intend to show that he drew extensively on the views of his English colleague.

553 Ferrier, *Functions of the Brain*, 223.

554 Charcot and Richer, "Cerebral Automatism," 7.

555 Carpenter held a similar view. He argued that the volitional power is "the power exerted by the Ego not only with a distinct purpose, but with a consciousness of effort, the strength of which is the mark and measure of its exercise." Carpenter, *Mental Physiology*, xxx.

556 Ferrier, *Functions of the Brain*, 113. See also Carpenter, *Mental Physiology*, 264, 388.

557 Ferrier, *Functions of the Brain*, 51.

558 Carpenter, *Mental Physiology*, 18.

559 Charcot and Richer, "Cerebral Automatism," 8.

560 Carpenter, *Mental Physiology*, 18.

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of the muscular contractions, which manifested themselves in the form of gradually intensifying tremors.

But, what at this point remains unexplained, is the cataleptic patient's ability to maintain a position imposed on his limb without investing any effort or showing any physiologically measurable signs of fatigue. To account for the puzzling cataleptic immobility, Charcot merely made an off-hand reference to cerebral automatism.[561] The notion of cerebral automatism was introduced by William Carpenter and is important for understanding the current and all of the subsequent Charcot's experiments on cataleptic patients. Hence, in what follows, we will examine this notion in some detail.

Carpenter viewed all mental activity in strictly physiological terms as correlated with underlying brain processes.[562] Moreover, he argued that a great deal of mental activity took place outside our conscious awareness and "without the control and direction of the Will."[563] He coined the term "unconscious cerebration" to designate the portion of mental activity that "is essentially *automatic*, and may be described in Physiological language as the *reflex action of the Cerebrum* [i.e., the brain]."[564] In effect, Carpenter claimed that a physiological mechanism analogous to the one underpinning the reflex sensorimotor responses executed by the spinal cord (i.e., the diastaltic arc we discussed in the previous section) also influenced the functioning of the brain.[565] Put more simply, Carpenter posited that the brain could act upon external sensory impressions in a purely automatic way. According to Carpenter, a proponent of the so-called theory of associationism, the brain's automatic response consisted of "a succession of Mental states, of which each calls forth the next" through a process of involuntary association

561 Charcot and Richer, "Cerebral Automatism," 4.

562 Carpenter, *Mental Physiology*, 14. See also ibid., 12–28.

563 Carpenter, "Influence of Suggestion," 153. For Carpenter's detailed description of what he explicitly termed the correlation between mental activity and underlying neural processes, see Carpenter, *Mental Physiology*, 12–14.

564 Carpenter, *Mental Physiology*, 515 (emphasis in original).

565 As pointed out by Carpenter, it was his colleague Thomas Laycock "who first extended the doctrine of reflex action to the Brain." Carpenter, "Influence of Suggestion," 152. Before Laycock, reflex action was understood to be limited to the spinal cord. Simultaneously and entirely independently of Laycock, the German psychiatrist Wilhelm Griesinger also developed a similar concept of cerebral reflexes in the 1840s. For details on both Laycock and Griesinger, see Clarke and Jacyna, *Origins*, 127–47. In 1863, the Russian physiologist Ivan Sechenov, who was apparently unaware of either Griesinger's or Laycock's work, also independently developed similar views on the reflexes of the brain. For details, see Smith, *Inhibition*, 96–112. Importantly, as Peter Amacher showed in his incisive analysis, by extending the concept of the reflex action to the brain, both Laycock and Secehenov "eliminated the potency of mind" since they effectively declared all human action to be a mere automatic response to external stimuli. Amacher, "Reflex Arc Concept," 183. In contrast, Carpenter's contribution was that he expanded the notion of the cerebral reflex action into the primary function of the nervous system without denying the existence of the volitional control over various human actions. In his view, cerebral reflexes influenced all mental activities, including intellectual elaboration, imagination, and artistic creation. See Carpenter, *Mental Physiology*, 515–43. Yet, unlike Laycock and Sechenov, Carpenter nevertheless insisted that human beings "are *not* mere thinking Automata," since "*we have within us a self-determining Power which we call Will*." Carpenter, 27, 28 (emphasis in original). Moreover, like later Charcot, Carpenter explicitly linked brain reflexes to hypnotic states. See Carpenter, xxvi–xxvii.

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of ideas.[566] Carpenter designated such involuntary association of ideas as 'suggestion,' a point to which we will return later when discussing Charcot's experiments.[567]

However, Carpenter also contended that, despite their shared physiological mechanism, there were two significant differences between the more primitive spinal and higher cerebral reflexes. First, to prompt a cerebral reflex, external impressions transmitted by the afferent nerves had to pass upwards of the spinal cord and reach the brain's sensory centres. Hence, the seat of cerebral reflexes was in the "expanded layer of Cortical substance."[568] Here, the incoming sensory impressions "successively produce[d] sensations, ideas, emotions, and intellectual processes," which then, in turn, gave rise to what Carpenter referred to as "truly automatic" actions.[569] Importantly, all stages of this process were carried out without the subject's conscious awareness.[570] Second, as opposed to comparatively simple motor responses induced through spinal reflexes, those called forth by the cerebral automatism could vary considerably in their complexity, often resembling voluntary actions.

In fact, Carpenter asserted that many cerebral reflexes were initially voluntary actions, which through frequent repetition and acquired habit came to be performed in an automatic manner.[571] He insisted that both voluntary and automatic actions were executed by the same neuromuscular system. The key distinction, however, was that voluntary actions had to be "called forth by a distinct effort of Will."[572] Voluntary

566 Carpenter, *Mental Physiology*, 15. The theory of associationism had its roots in the works of the seventeenth-century English philosopher John Locke and the eighteenth-century Scottish philosopher David Hume. It was initially formulated by the eighteenth-century English philosopher David Hartley and the early-eighteenth-century philosopher James Mill. In the nineteenth century, associationism was taken up and further developed by Alexander Bain, Herbert Spencer, John Stewart Mill, William Carpenter, David Ferrier, and Henry Maudsley, among others. For a detailed historical account of the development of associationist psychology, which Charcot quoted in his lectures, see Ribot, *English Psychology*. The basic tenet of associationism was that the phenomenon designated as the association of ideas was the fundamental principle, which governed the working of the human mind, underpinning its "various faculties, *senses, memory, imagination, understanding, affections, and will*." Ribot, 39 (emphasis in original). Specifically, in this view, sensory impressions of external stimuli first produced sensations in the mind, which, in turn, gave rise to simple ideas. A simple idea was nothing else but "a copy, an image of the sensation, sometimes a representation or a trace of the sensation." Ribot, 48. Such simple ideas then merged through the process of association into complex ideas. But far from being limited to simple ideas, associations could also take place "between complex ideas, which melt together so as to form an idea which appears simple." Ribot, 50. The ideas tended to form associations either according to the principle of temporal contiguity (i.e., co-occurrence and succession) or the principle of resemblance. Ribot, 216–17. Once linked through association, ideas became "inseparable in consciousness." Ribot, 115. Importantly, proponents of associationism regarded the association of ideas to be a physiological process that took place "in the cerebral hemispheres." Ribot, 217. Charcot explicitly subscribed to the theory of associationism. See, e.g., Charcot, "Lecture 21: Brachial Monoplegia," 290–91; and Charcot, "Appendix 2: Muscular Sense," 397–98.

567 Carpenter, *Mental Physiology*, 15.

568 Carpenter, 105.

569 Carpenter, "Influence of Suggestion," 152.

570 Carpenter, 153. See also Carpenter, *Mental Physiology*, 15.

571 Carpenter, *Mental Physiology*, 16.

572 Carpenter, 16.

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actions were, therefore, "guided by a distinct conception of the object to be attained, and by a rational choice of the means employed."[573] By contrast, automatic actions were independent of any preformed intention since external sensory impressions prompted them. As such, they were executed "mechanically" without any voluntary intervention.[574] Carpenter contended that because automatic actions did not entail any voluntary effort, they were "followed by comparatively little fatigue."[575] The effects of fatigue would only occur after "a period many times as long" as when the same action was executed voluntarily.[576]

It now becomes clear how by attributing the cataleptic patient's immobility to "the facts of automatism," Charcot was able to account for the apparently puzzling lack of both effort and fatigue that the graphic inscriptions had disclosed.[577] Drawing on this interpretational framework, we can posit the following explanation. By placing the cataleptic's arm into a horizontally extended position, the experimenter induced a change in the tension of the patient's muscles. The sensory consequences of this passively imposed attitude were communicated via the muscular sense to the patient's brain. Here they excited an automatic motor response, which was then communicated via efferent nerves to the muscles of the arm. As a result of this entirely automatic cerebral response, the patient's arm remained in the position the experimenter had placed it. Moreover, due to the involuntary character of the patient's muscular action, the onset of fatigue was considerably postponed and, as far as we can judge from the curves, did not occur during the experiment.

My analysis so far has aimed to show that the experiment in which Charcot used the graphic method to compare the physiological functions of a cataleptic patient and a simulator fulfilled multiple epistemic functions. This experiment enabled Charcot to generate visual evidence for his claim that hypnotic catalepsy was a genuine neurophysiological state distinct from simulation. I have also highlighted how this experiment allowed Charcot to posit the fundamentally involuntary nature of hysteria patients' motor responses during catalepsy. But far from stopping at this point, Charcot collaborated with Richer to devise experiments that provided further empirical evidence for the role of cerebral automatism in catalepsy. The aim of these experiments, as we will see, was to induce in cataleptic patients considerably more complex automatic responses.

In the first series of their jointly conceived experiments on cataleptic patients, Charcot and Richer set out to explore what they termed "the influence of gesture upon the expression of the face."[578] To achieve this, Charcot and Richer first plunged their subjects into catalepsy and then imparted passive movements onto their immobile yet highly pliable bodies. They began by imposing onto their patients' bodies a range of gestures that were meant to unambiguously express particular categories of emotions.

573 Carpenter, "Influence of Suggestion," 151.
574 Carpenter, *Mental Physiology*, 16.
575 Carpenter, 388.
576 Carpenter, 389.
577 Charcot and Richer, "Cerebral Automatism," 4.
578 Charcot and Richer, 8.

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In response to this experimental manipulation, the subjects' faces automatically assumed an expression. According to Charcot, the resulting facial expression was always "in harmony with" the gesture the experimenter had imposed on the patient.[579] For example, he described that "a tragic attitude imparts a severe air to the physiognomy, and the eyebrows contract." In contrast, "if the open hands are carried to the mouth, as in the act of throwing a kiss, a smile immediately appears upon the lips."[580] Once such automatic coordination between the gesture and the facial expression had taken place, the patient remained as if frozen in the resulting attitude, akin to an "expressive statue."[581] But in performing such experiments, Charcot and Richer soon encountered what they perceived as limitations. As Charcot explained, "perfectly expressive movements are difficult to impart to a mannikin, however docile it may be, and the number of communicable attitudes fully adequate to express a given sentiment or feeling is relatively restricted."[582] Insufficiently expressive gestures still produced changes in the patient's physiognomy, but the resulting facial expressions were ambiguous. Charcot viewed such results as noise and discarded them.

Aiming to circumvent these limitations, Charcot and Richer decided to invert the experimental procedure. In a separate set of experiments, they systematically modified cataleptic patients' facial expressions and then examined the effects that these modifications had on the patients' bodily gestures. In doing so, Charcot and Richer once again took recourse to Duchenne's neurophysiological studies of emotional facial expressions, which, as discussed previously, had already served as the key reference point in their experiments on patients in the state of hypnotic lethargy. Yet, in this case, Charcot and Richer could no longer use mechanical excitation to modify their cataleptic patients' facial expressions.[583] Instead, to artificially inscribe chosen emotional expressions onto the subjects' faces, Charcot and Richer had to use localised electricity (i.e., the faradisation).

Hence, by applying electrodes to the faces of cataleptic patients, Charcot and Richer started to selectively induce contractions of those facial muscles that Duchenne had codified as expressive of particular emotions. They primarily focused on reproducing the expressions that "according to the rule established by Duchenne" required either an isolated contraction of a single, so-called 'completely expressive' muscle or a simultaneous contraction of two 'incompletely expressive' muscles.[584] This procedure was meant to enable Charcot and Richer to increase the precision of their experimental intervention concerning the clarity of emotional expressions they were imprinting onto the patients' muscles. The underlying assumption was that facial expressions were less ambiguously attributable to particular categories of emotion than bodily gestures. Moreover, focusing on the face allowed them to induce a considerably wider range of

579 Charcot and Richer, 8.
580 Charcot and Richer, 8.
581 Charcot and Richer, 9.
582 Charcot and Richer, 8.
583 This is because, as mentioned earlier, neuromuscular excitability did not exist during catalepsy. Thus, the patients' muscles did not contract involuntarily in response to light pressure.
584 Charcot and Richer, "Cerebral Automatism," 10.

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emotional expressions than in previous experiments that used gestures as the starting point. Using the electrodes, Charcot and Richer thus managed to imprint onto their patients' faces various emotional expressions such as anger, astonishment, joy, sadness, fear, contempt, pain, and horror.[585]

Crucially, Charcot and Richer established that during the process of faradisation, the patient's "entire body, spontaneously as it were, entered into action, and completed by its attitude the expression of the face."[586] This reaction started happening as soon as the facial expression of a particular emotion had been induced with sufficient clarity.[587] For example, once the expression of anger had been imprinted on her face, the patient's fists started to clench, and her arms gradually assumed "a fixed position of aggression" (fig. 1.18, right).[588] Due to their cataleptic immobility, the patients retained both the experimentally imprinted facial expressions and the spontaneously developed accompanying bodily gestures even after the electrodes had been removed from their faces. It was at this point that the cataleptic patients were photographed.[589] I argue that the function of the resulting photographs was twofold.

First, as in the hypnotic experiments we discussed earlier, also in this context, photography enabled the fixation of the ephemeral experimental results.[590] Owing to such use of photography, the experimental results were made available for subsequent visual analysis and could be compared across multiple trials and different patients. The visual comparison of accumulated results, in turn, enabled the Salpêtrians to generate new insights. For example, through such analysis, Richer established that in a single subject, the experimental induction of a particular facial expression always led to the production of the identical gesture across multiple trials.[591] By contrast, Richer also

585 Richer, *Études cliniques*, 2nd ed., 673–79.

586 Charcot and Richer, "Cerebral Automatism," 9.

587 Richer warned that the clarity with which a particular emotional expression was induced also depended on the intensity of the current applied to a particular muscle. This was because some muscles, such as the frontalis, participated in expressing very different emotions (attention, ecstasy, and astonishment), depending on the degree of their contraction. Richer, *Études cliniques*, 2nd ed., 674.

588 Charcot and Richer, "Cerebral Automatism," 11.

589 Richer, *Études cliniques*, 2nd ed., 671. My following analysis of the function of photography is limited to the original set of Charcot's and Richer's experiments on cataleptic patients. Subsequently, Richer and Londe developed a variation of these experiments by modifying the operating procedure. In the novel set of experiments, Richer attached small electrodes to a malleable metal rod that was fixed directly to the patient's head, thus remaining in place during the entire experiment. By varying the intensity of the current, Richer was able to induce continuous changes in the patients' facial expressions of different emotions, which led to gradual changes in their gestures. Londe then used the photographic camera to capture and explore consecutive phases of progressive concurrent changes in the patient's facial expressions and gestures. See ibid.; and Londe, *La photographie médicale*, 92–93, and plate 6. However, since Charcot neither discussed these subsequent experiments in his lectures nor used the resulting photographs in his publications, I will disregard them in my analysis.

590 Interestingly, Charcot emphasised that the immobility of the attitudes and facial expressions he artificially provoked in his cataleptic patients was "eminently favorable to photographic reproduction." Charcot and Richer, "Cerebral Automatism," 9.

591 See Richer, *Études cliniques*, 2nd ed., 684.

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discovered that in response to the faradisation of precisely the same facial muscles, each patient assumed a slightly different bodily attitude. In each case, the resulting gesture appeared to harmonise sufficiently with the experimentally induced facial expression. Yet, Richer emphasised considerable differences across subjects concerning what he referred to as the expressive "quality" of their gestures.[592] In some patients, the resulting emotional gestures were more expressive, in others less. The emergence of such insights hinged on the use of photography. Therefore, we can say that, also in this context, the Salpêtrians deployed photography as an active epistemic tool.

Figure 1.18. Photographs by Albert Londe of expressive gestures indirectly induced in a hysteria patient during catalepsy through suggestion by the muscular sense. Left: laughter. Right: anger. From: Charcot, Oeuvres complètes, vol. 9, plates 12 and 13.

Second, Charcot included "several of the most interesting" photographs that documented the results of the cataleptic experiments in his publications (fig. 1.18).[593] He explicitly invited his readers to visually examine the images and thus verify that appropriate gestures spontaneously complemented the expressions he had artificially imparted onto the patients' physiognomy.[594] Therefore, I suggest that Charcot used these particular photographs as empirical evidence for the physical reality of what he termed the cataleptic "suggestion by the muscular sense."[595] Charcot introduced this term to designate the automatic and "reciprocal" coordination between cataleptic

592 Richer, 684.

593 Charcot and Richer, "Cerebral Automatism," 10. For additional figures, see ibid.; and Charcot, *Oeuvres complètes*, vol. 9, plates 9–13.

594 Charcot and Richer, "Cerebral Automatism," 10–11.

595 Charcot and Richer, 1.

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patients' gestures and facial expressions, which the experiments he conducted with Richer so effectively demonstrated.[596] By introducing this term, he explicitly attributed the coordination of bodily responses during catalepsy to the "intermediation of the muscular sense."[597] In doing so, Charcot aimed to provide a plausible physiological explanation for the phenomena that admittedly appeared "singular and unexpected."[598]

As part of his explanation, Charcot specified that all the various instances of the seemingly puzzling coordination between cataleptic patients' gestures and facial expressions were purely automatic acts. Moreover, he argued that these automatic acts were "developed by the influence of excitation conveyed to nervous centres by means of the muscular sense."[599] The photographs served to reinforce this claim with which Charcot placed the behaviour of cataleptic patients into a strictly neurophysiological framework. The photographs fulfilled this function by providing visual evidence that the automatic acts experimentally induced through suggestion by the muscular sense resulted in clear-cut and reproducible physical effects.

Yet once again, to understand what Charcot meant under the suggestion by the muscular sense, we must unpack his cryptic explanation. To this end, we need to synthesise and further expand the insights we have won through our previous discussions about Ferrier's views on the muscular sense and Carpenter's notion of cerebral automatism. First, by drawing on Ferrier, we can reason that the artificially induced contractions of the facial muscles resulted in multiple peripheral sensory impressions. These impressions were then communicated via the afferent nerves to the sensory centres of the patients' brains, where they gave rise to the sensory idea of a particular emotion.[600] Importantly, this idea was merely a revival of an entire set of sensory impressions, which had been repeatedly registered in the same cerebral centres on all previous occasions when the patient made that particular facial expression.[601] Furthermore, since a particular combination of a facial expression and a bodily gesture tended habitually to co-occur in the same emotional context, their accompanying sensory impressions became "connected together by previous associations."[602] This meant that the memories of these two distinct sets of sensory impressions became

596 Charcot and Richer, 10.

597 Charcot and Richer, 4.

598 Charcot and Richer, 12.

599 Charcot and Richer, 11.

600 See Charcot, "Lecture 21: Brachial Monoplegia," 291.

601 Charcot's use of the term 'idea' was firmly grounded in the physiological context. When discussing the muscular sense, he explicitly quoted Ferrier. See Charcot, Appendix 2: Muscular Sense," 398. According to Ferrier, a complex stimulus—an object or a movement—gives rise to a set of sensory impressions in the sensory centres of the brain. Each of these impressions induces physiological cell modifications in the sensory centres, which then form "the organic basis of the memory of such impressions." Ferrier, *Functions of the Brain*, 258. "When the same cell modifications are again excited" through the renewed sensory impressions, the 'idea' of the original stimulus is revived in the sensory centres. Ferrier, 258. "The sensory centres, therefore, are to be regarded not merely as the organs of consciousness of immediate sensory impressions, but as the organic register of special sensory experiences. This organic memory is the physical basis of Retentiveness, and the property of re-excitability is the organic basis of Recollection and Ideation." Ferrier, 258.

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

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organically welded in the sensory centres, thus becoming part of the same sensory idea.[603] Due to the resulting "organic cohesion,"[604] a re-excitation of the sensory impressions that accompanied a particular facial expression inevitably led to an automatic 'ideal recall' of the associated set of sensory impressions, which in the past had always arisen when the correlated bodily gesture was performed.

But the chain of associations did not end there. Next, the recall of the sensory impressions associated with a particular bodily gesture, in turn, called up in the brain's motor centres the idea of the movement entailed in the execution of that particular bodily gesture.[605] Such sequencing of ideas, which Carpenter had designated as suggestion, was involuntary (i.e., automatic) and unconscious.[606] As we have seen, this sequencing was physiologically determined by the structural connections in the brain, which had been established through the patient's previous experiences and habits.[607] Charcot foregrounded the physiological basis of this process by stating that suggestion by the muscular sense was "intimately connected with the normal action of the nervous system."[608] However, there was one critical distinction between cataleptic patients and healthy subjects concerning cerebral reflexes. According to Carpenter, although all automatic actions of the brain were executed without any involvement of the will, under normal conditions, "the human Ego" was nevertheless able to "exercise a rational control" over this automatism.[609] In other words, even healthy subjects could not avoid the automatic arousal of a sequence of mutually associated ideas in response to an external stimulus. But healthy subjects could choose whether or not to act on the ideas provoked by external circumstances. In contrast, Charcot argued that cataleptic patients could not make such decisions.

In healthy subjects under normal conditions, all senses were equally awake, thus delivering a variety of impressions to the brain's sensory centres. In these centres, such diverse impressions were brought into relation to one another and synthesised into a set of mutually interconnected ideas and sensations.[610] But during hypnotic catalepsy, due

603 Ferrier conjectured that such associative connections consisted of actual structural links within the sensory centres of the brain. Ferrier, *Functions of the Brain*, 258.

604 Ferrier, 258.

605 As stated by Ferrier, we "have a memory of sensations and a memory of movements, organically distinct from each other; but, by association, a memory of sensations combined with movements." Ferrier, 225. Further, the "ideal associated movement is thus made to arise in consciousness, when the corresponding sensation is artificially re-excited." Ibid.

606 Carpenter, *Mental Physiology*, 15.

607 Charcot, "Lecture 21: Brachial Monoplegia," 290. See also Charcot and Tourette, "Hypnotism in the Hysterical," 609. One added benefit of Charcot's explanation was that it could account for the individual difference in the expressiveness of resulting gestures across patients we discussed previously. Such variations across subjects could now be attributed to their different habits. In other words, in this view, the level of expressiveness of each patient's artificially induced emotional gesture during catalepsy depended on how expressively she tended to physically manifest her feelings during the waking state.

608 Charcot and Richer, "Cerebral Automatism," 12.

609 Carpenter, "Human Automatism," 414. See also Carpenter, *Mental Physiology*, 106; and Ferrier, *Functions of the Brain*, 282–84.

610 For details, see Richet, "Des mouvements," 612–15.

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to the patient's mental inertia, such synthesis could not occur. Instead, the ideas called forth by suggestion remained entirely isolated, "without diffusion, and fixed," hence acquiring an enormous force and dominance.[611] As Charcot explained, these ideas were "free from the control of that large collection of personal ideas long accumulated and organised, which constitute the conscience properly so-called, the *ego*."[612] In short, in a cataleptic patient, the ideas induced externally through suggestion remained isolated from the patient's conscious control. As a result, these ideas automatically manifested themselves in the form of "corresponding motor phenomena."[613]

Hence, it was part of the normal process of 'unconscious cerebration' that a particular facial expression imprinted onto a cataleptic patient's face through faradisation led to a revival of the idea of movement entailed in the 'harmonising' bodily gesture. The pathological aspect was that, as soon as this idea of the movement arose in the brain's motor centres through a cerebral reflex, the patient automatically executed the idea. This motor reaction demonstrated that she had no voluntary control whatsoever over her responses to external stimuli. In effect, in Charcot's interpretation, the muscular action underlying the coordination of facial expressions and bodily gestures in cataleptic patients was understood to be a direct consequence of abnormally unrestrained cerebral reflexes.[614] The unrestrained cerebral reflexes, in turn, were understood to arise from a disruption in the hierarchical functioning of the nervous system, which in normal circumstances, was under the control of the conscious self (i.e., the ego).

Charcot's neurophysiological explanation for the coordination between the cataleptic patients' emotional expressions and gestures had two consequences. First, in the context of hypnosis and, by analogy, in hysteria in general, Charcot redefined suggestion as a fundamentally "pathological phenomenon" that was exempt from the normal restraining control of 'the ego.'[615] It is important to note that Charcot used the term suggestion in two distinct yet mutually related ways. On the one hand, suggestion referred to a process through which external sensory impressions triggered unrestrained reflex responses of the brain, thus giving rise to involuntary actions of a purely 'mechanical' character.[616] On the other hand, suggestion also referred to targeted procedures through which the experimenter acted on the patient to induce such reflex

611 Charcot, "Lecture 21: Brachial Monoplegia," 290–91.

612 Charcot, 290 (emphasis in original). A similar definition of the ego (i.e., the self) was offered by Carpenter: "Thus each Human *Ego*, at any one moment, may be said to be the *general resultant* of his whole Conscious Life; the direction of which has been determined in the first instance by his congenital Constitution, second by the education he has received from the Will of others or from the discipline of circumstances, and thirdly by the Volitional power he has himself exercised." *Mental Physiology*, 106 (emphasis in original). In the French original, Charcot used the term "le moi" (the self) for what his English translator designated as the ego. Charcot, *Oeuvres complètes*, 3:337.

613 Charcot, "Lecture 21: Brachial Monoplegia," 289.

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

615 Charcot and Tourette, "Hypnotism in the Hysterical," 606.

616 See, e.g., Charcot, "Lecture 22: Brachial Monoplegia," 305; and Charcot, "Appendix 1: Hystero-Traumatic Paralysis," 385.

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responses.[617] Second, a hysteria patient in the state of catalepsy came to be viewed as a mere "automaton without any consciousness or spontaneity [i.e., will], who moves only under the influence of external sensory excitations."[618] Put simply, the Salpêtrians regarded the cataleptic patient to be a passive neurological machine whose actions were entirely determined by external circumstances. This was precisely the point that the photographs of the 'harmoniously' coordinated facial expressions and gestures induced through 'the suggestion by the muscular sense' were meant to demonstrate (fig. 1.18).

Richer took this latter implication a step further. He decided to prove that "despite the striking truthfulness of the external manifestations" it produced, the suggestion by the muscular sense did not affect the cataleptic patient's "inner being."[619] With this aim in mind, he applied a pneumograph to the chest of several cataleptic patients to trace if the artificially imposed expressions of emotions led to corresponding changes in their breathing patterns. The resulting respiratory traces showed that even when clear-cut expressions of various emotions were artificially imprinted on the patients' faces or bodies, their breathing patterns underwent only a mild and temporary disturbance. After one or two respiratory movements, the curves resumed their uniform shape, showing that the cataleptic patient's breathing remained slow and shallow for the remainder of the experiment (fig. 1.19).[620] As Richer explained, the curves thus delivered empirical evidence that the patients did not experience any of the emotions that were externally so clearly manifested in their mutually coordinated facial features and bodily gestures.[621] Compellingly, this finding provided further support to the stance that all of the cataleptic patients' actions were mere cerebral reflexes of which they had no conscious awareness and no voluntary control.

Finally, Richer additionally extended the range of cataleptic experiments by shifting the focus away from the muscular sense and placing it instead on the senses of hearing and sight.[622] The details of his numerous experiments remain beyond the scope of this enquiry. However, what is of interest for our discussion is the following. Richer established that by exposing cataleptic patients to various noises, he could induce in them complex hallucinations.[623] Once provoked, such hallucinations were then enacted through the cataleptic patients' gestures, facial expressions, and verbal utterances. Richer argued that both the resulting "mimed and spoken scenes" and the correlated

617 See, e.g., Charcot, "Lecture 19: Six Cases," 258; and Charcot, "Lecture 21: Brachial Monoplegia," 289.

618 Richer, *Études cliniques*, 2nd ed., 789.

619 Richer, 680.

620 Richer, 679–81.

621 Notably, Charcot and Richer held the view that in healthy individuals, "the expressive movements of the physiognomy or of the entire body" necessarily produced corresponding mental and emotional effects. To emphasise this view, they quoted the Scottish philosopher Dugald Stewart: "As every motion of the mind produces a sensible effect on the bodily appearance, so, upon the other hand, when we assume any strongly expressive look, and accompany it with appropriate gestures, some degree of the correspondent emotion is apt to arise within us." Charcot and Richer, "Cerebral Automatism," 13. It was precisely this 'normal' emotional reaction that was absent in cataleptic patients.

622 Richer, *Études cliniques*, 2nd ed., 686–711.

623 Richer, 679. Richer did not specify which noises he used to induce such hallucinations.

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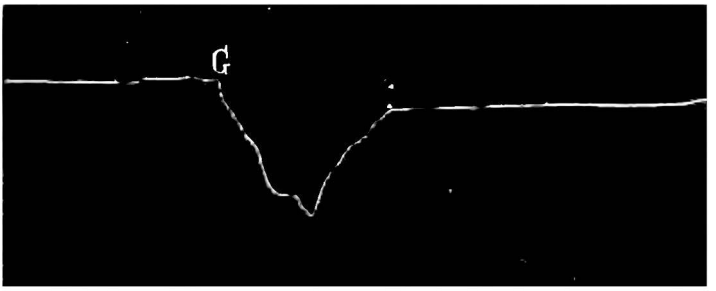
hallucinations these scenes expressed were merely physiological manifestations of the patients' unrestrained cerebral reflexes.[624] He noted that the hallucinatory scenes induced during catalepsy varied considerably from patient to patient. Yet, he insisted that the content of the induced hallucinations was "very similar" to those hallucinations the same patients enacted during the third period of their hysterical attacks termed the passionate attitudes.[625]

Figure 1.19. Respiratory curve of a patient in the state of catalepsy. G designates the moment at which the smile was indirectly induced in the patient by bringing her hands close to her mouth in a gesture that imitated the act of giving a kiss. From: Richer, Études cliniques, 2nd ed., 681, fig. 159.



624 Richer, 697. As Richer explained, the only difference between the thus provoked hallucination and the simple cataleptic immobility was the level of complexity of the underlying associations. In this interpretation, the induction of hallucinations presumed the re-activation of multiple and far more complex associative connections among a large number of 'nervous elements,' which had been established through the patient's previous experience and habits. See ibid., 698, 754.

625 Richer, 697. Interestingly, this line of experimentation was taken up and further developed by another of Charcot's assistants, George Guinon. In 1891, working with Sophie Woltke, Guinon devised two parallel series of experiments. First, Guinon and Woltke systematically exposed two cataleptic patients to various colours, smells, and sounds. In response to such varying sensory stimuli, the patients experienced different hallucinations. They manifested the emotional content of the resulting hallucinations through particular gestures and facial expressions, which the researchers documented through photographs. See Guinon and Woltke, "Excitations sensitives et sensorielles." Subsequently, Guinon and Woltke repeated the same experimental procedures with hysteria patients during the passionate attitudes period of the hysterical attack. See Guinon and Woltke, "Excitations des organs des sens." Similarly to Richer, Guinon and Woltke concluded that, both during catalepsy and the passionate attitudes period, simple sensory excitations induced hallucinations that were always the same in a single individual yet differed considerably from patient to patient. They further conjectured that the emotional content of hallucinations was highly idiosyncratic because they were determined by each patient's "personal habits, her way of life, her memories, in short, her own personality." Guinon and Woltke, "Excitations des organs des sens," 55 (my translation). See also Guinon and Woltke, "Excitations sensitives et sensorielles," 87.

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In effect, Richer thus established a relationship of equivalence between cataleptic hallucinations and the passionate attitudes period of the hysterical attack. The only difference, as Richer claimed, was that during the period of passionate attitudes, the hallucinations arose spontaneously. By contrast, in the cataleptic state, the hallucinations had to be elicited through experimental intervention.[626] The key implication was that the hallucination hysteria patients experienced during the passionate attitudes period of the hysterical attack, as well as the bodily actions through which they enacted these hallucinations, now came to be viewed by the Salpêtrians as a consequence of the aberrant cerebral reflexes. At least indirectly, a significant segment of the hysterical attack was thus linked to a distinct functional disturbance of the brain.

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To conclude, my analysis in this and the previous sections has shown that Charcot and his team viewed hypnosis as an artificially induced, selective intensification of the neurophysiological characteristics latently already present in hysteria patients during their waking state. Drawing on this assumption, Charcot used lethargy and catalepsy to isolate, experimentally model, and indirectly explore the underlying neurophysiological basis of hysteria. As we have seen, his experiments systematically focused on what he perceived as the two key characteristics of lethargy and catalepsy—neuromuscular hyperexcitability and cerebral automatism. I have argued that, through the series of experiments we have analysed in detail, Charcot succeeded in attributing multiple hysterical symptoms either to overactive lower-order spinal or to uncontrolled higher cerebral reflexes. This attribution, in turn, had broader consequences for Charcot's understanding of hysteria on the whole. Across these different experiments, hysteria was gradually redefined as a disorder whose various symptoms appear to arise from a pathologically heightened reflex activity of the nervous system.

Taken together, Charcot's hypnotic experiments not only foregrounded the involuntary nature of hysterical symptoms but also began to link them to distinct neurophysiological processes. Admittedly, this linking was still very fragmentary and tentative. Charcot could not explain why a specific kind of reflex (i.e., spinal or cerebral) became activated in a given context. His experiments also failed to clarify how cerebral reflexes gave rise to particular symptoms, such as the hysterical attack. Yet, despite this lack of specificity and the fact that many questions remained open, Charcot nevertheless achieved one important goal. He effectively embedded hysteria in a neurological context. Throughout my analysis, I have emphasised how this embedding hinged on the systematic use of photography and Marey's graphic method. Moreover, I have strived to demonstrate that to understand why Charcot produced particular images, as well as how he read and interpreted them, we must reconstruct the broader neurophysiological discourse of the time, which both explicitly and implicitly informed his hypnosis research.

626 Richer, *Études cliniques*, 2nd ed., 697.

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