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The Speechless Patient: Charcot's Diagnostic Interpretation of Vocal, Gestural, and Written Expressions in Hysterical Mutism

Beyond the medical discourse, the dominant cultural stereotype of a hysteria patient is that of a woman screaming, crying, or producing other emotionally charged vocalisations. Multiple humanities scholars attributed the emergence of this stereotype to the image-based hysteria research that the nineteenth-century neurologist Jean-Martin Charcot conducted at the Parisian hospital Salpêtrière.[1] They argued that the photographs of Charcot's female patients in the throes of hysterical attacks – included in the medical publication *Iconographie photographique de la Salpêtrière* and then widely disseminated – "did much to fix the image of hysteria in the public mind".[2] Such accounts posit that Charcot enticed his female patients into performing the dramatic image of hysteria he imposed on them.[3] Moreover, we are told that Charcot was disinterested in listening to his patients' utterances and focused exclusively on visualising their bodies to produce a "full pictorial record" of hysterical symptoms.[4]

Such dismissive accounts mainly address Charcot's image-based investigation of hysterical attacks in female patients. Admittedly, the hysterical attack stood at the centre of Charcot's early hysteria research, conducted when the Salpêtrière housed only female patients. However, after 1880, Charcot shifted to studying other hysterical symptoms in female and male patients, arguing that hysteria was identical in both genders.[5] One seemingly less dramatic hysterical symptom that

1 Bronfen, 1998, 198; Rose, 2005, 114; Showalter, 2014, 147–150. Besides his highly publicised hysteria research, Charcot concurrently studied and provided groundbreaking insights into many other neurological disorders, such as multiple sclerosis, amyotrophies, locomotor ataxia, Parkinson's disease, and Huntington's chorea (see Goetz, Bonduelle, and Gelfand, 1995, 99–133).

2 Scull, 2009, 123.

3 Bronfen, 1998, 190–203; Didi-Huberman, 2004, 104; Gilman, 1993, 345–346; Scull, 2009, 122–123; Showalter, 2014, 151–154.

4 Showalter, 2014, 155; see also Bronfen, 1998, 199; Rose, 2015, 114.

5 Charcot, 1889a, 221. For Charcot's introduction of male patients into the clinic, see Goetz, Bonduelle, and Gelfand (1995, 200–205). For Charcot's study of diverse hysterical symptoms, see Muhr (2022, chapter 1).

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occupied Charcot's attention in the late 1880s was mutism.[6] Patients with this symptom had lost the ability to speak despite the absence of any detectable organic damage.[7] And although Charcot dedicated multiple clinical lectures to hysterical mutism, humanities scholars have ignored this aspect of his research.

As this chapter will show, examining Charcot's research on hysterical mutism is instructive for two reasons. First, we will gain insights into how Charcot instituted this, at the time, controversial symptom as a clinical entity by determining its diagnostic specificity and postulating its underlying neurophysiological mechanism. Second, through this examination, I will propose a more nuanced view of Charcot's hysteria research. I will do so by challenging the dismissive accounts according to which Charcot's approach was limited to investigating only the visual aspects of hysteria while suppressing the patients' voices. Based on close reading of his lectures, I will argue that Charcot established hysterical mutism as a distinct clinical entity by attentively listening to his mute patients' non-verbal utterances, reading their written answers to his questions, and interpreting their communicative gestures.

This chapter will focus on the seminal 1886 lecture during which Charcot presented a male patient with typical clinical features of hysterical mutism. The transcript of this lecture was published in the third volume of Charcot's *Clinical Lectures on the Diseases of the Nervous System* and accompanied by an extensive appendix that summarised several other cases of mutism from Charcot's previous lectures.[8] Analysing this lecture, I will trace how Charcot attributed diagnostic significance to vocal outputs that mute patients could still produce and those they could not. Finally, I will discuss how, based on the distinctive clinical features of hysterical mutism that he identified, Charcot made inferences about this symptom's neurophysiological basis. But before turning to Charcot's research on hysterical mutism, we must first examine his earlier studies of language disorders of organic origin. As I will demonstrate, these studies are significant for our discussion because they provided a conceptual framework for Charcot's subsequent research into hysterical mutism as an isolated loss of spoken language.

6 Charcot, 1886, 34–35; Charcot, 1889b, 247–252; Charcot, 1889f, 360–373; Charcot, 1892, 257–261, 264–274.

7 Charcot, 1889f, 360.

8 Charcot, 1889f, 360–373; Cartaz, 1889, 410–433.

Charcot's Research into the Neurological Basis of Language

In 1885, when Charcot turned to hysterical mutism, he had already spent the previous two years systematically studying and lecturing on various forms of language disturbances jointly referred to as organic aphasia, a topic he would continue to research throughout the 1880s.[9] But even before 1883, Charcot participated in aphasia studies that had been inaugurated in 1861 by his colleague Paul Broca.[10] In the early 1860s, Broca relied on a series of autopsies to correlate a form of language disturbance, which he initially termed aphemia, with structural brain damage to the third convolution of the left frontal lobe.[11] This particular type of aphasia was characterised by patients' inability to produce articulated speech. Through autopsies, Broca thus identified a circumscribed brain region – i.e., a cerebral centre – that controls the production of articulate speech. His discovery, in turn, gave rise to the paradigm of cerebral localisation in neurological research. According to this paradigm, the brain is not a homogeneous organ but consists of multiple specialised centres that each "possesses its proper function, though each one remains in the most intimate connection with the others".[12]

Charcot shaped the early aphasia research, first by providing Broca with six autopsy cases that confirmed the localisation of the speech centre in the left frontal lobe and then by reporting several autopsy cases that appeared to contradict this localisation.[13] However, from 1865 to the early 1880s, Charcot stopped actively studying aphasia.[14] Instead, during the 1870s, he focused on the localisationist investigation of cerebral motor centres and hysteria research.[15] By the time Charcot returned to aphasia, significant new insights had been won in this vibrant field. In 1874, Carl Wernicke discovered sensory aphasia, "in which speech remains fluent but not meaningful," and linked it to the lesion of the left temporal lobe.[16] Three years later, Adolf Kussmaul divided Wernicke's sensory aphasia into word-

9 Charcot, 1883a; Charcot, 1883b; Charcot, 1883c; Charcot, 1884; Charcot, 1889c; Charcot, 1889d; Charcot, 1889e. For a succinct overview, see Anonymous (1884, 593–595) and Marie (1888, 81–84).

10 Jacyna, 2000, 14. For a detailed discussion of the different phases of Charcot's decades-long aphasia research, see Gasser (1995, 119–214).

11 Finger, 2005, 137–154. In 1864, Armand Trousseau introduced the term aphasia to designate language disturbances (Gasser, 1995, 119).

12 Charcot, 1889e, 162–163.

13 Gasser, 1995, 119–134.

14 Gasser, 1995, 134.

15 Goetz, Bonduelle, and Gelfand, 1995, 128–129.

16 Finger, 2005, 150.

blindness and word-deafness, and in 1881 Sigmund Exner identified a cerebral 'centre for writing'.[17]

In his 1883 lectures, drawing on the paradigm of cerebral localisation, Charcot combined and expanded his colleagues' different findings to develop a general theory of aphasia and of the neurological basis of language.[18] First, synthesising the work of other researchers, Charcot posited the existence of four "fundamental forms" of aphasia, each of which, in theory, entailed a selective loss of a particular language faculty.[19] Besides Broca's motor aphasia (the loss of spoken language), the other forms included agraphia (the inability to write), verbal blindness (the inability to read despite intact vision), and verbal deafness (the inability to comprehend spoken language despite intact hearing).[20] Next, based on clinical observations of his aphasic patients, postmortem analyses of their brains, and the review of findings published by other neurologists, Charcot tentatively linked each of the four basic forms of aphasia to a structural lesion of an anatomically distinct brain region.[21]

Charcot thus postulated the existence of four independent yet mutually physically interconnected cerebral language centres that jointly controlled the production and comprehension of language (Fig. 1). He conjectured that in each of these centres, specialised partial memories – or commemorative mental images – of a particular motor or sensory aspect of words were physiologically engraved.[22] The "commemorative auditive image" acquired by repeatedly hearing a particular word had its seat in the auditory centre for words that controlled the comprehension of the spoken language.[23] Visual memories of words, which underpinned the individual's ability to comprehend hand-written and printed texts, were imprinted in the specialised visual centre for words. Apart from these two sensory language centres, Charcot distinguished two motor language centres – Broca's centre of articulated language and a centre of written language – that stored motor images of articulation and graphic motor images, respectively. The first type of motor image was "developed by the repetition of the movements of the tongue necessary to pronounce a word, the latter by a repetition of the movement of the hand and fingers necessary in writing".[24]

17 Gasser, 1995, 137–138.

18 Gasser, 1995, 134.

19 Charcot 1889c, 131.

20 Charcot, 1892, 266. Whereas Kussmaul introduced word-blindness and word-deafness as medical categories, the term agraphia was first used by William Ogle in 1867 (Gasser, 1995, 136).

21 Marie, 1888, 82–83. For alternative models of aphasia by Wernicke, Kussmaul and others, see Hakosalo (2006, 276–310).

22 Charcot adopted the concept of partial memories from the French philosopher and psychologist Théodule Ribot (see Charcot, 1889e, 151–152).

23 Charcot, 1889e, 161.

24 Charcot, 1889e, 161.

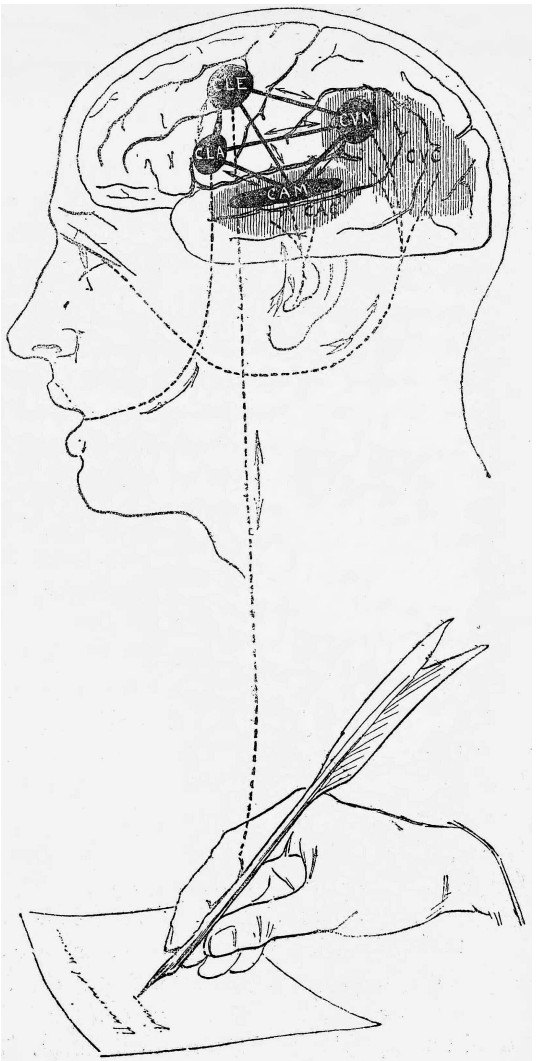

Fig. 1: The tentative localisation of the cerebral language centres according to Charcot: CVM – visual centre for words; CAM – auditory centre for words; CLA – centre of articulated language; and CLF – centre of written language. Also visualised in the figure are: CVC – centre of vision; and CAM – centre of hearing. From: Marie (1888, 82).

According to Charcot, the coordinated activity of the four language centres underpinned each individual's ability to translate their thoughts into words and then communicate them. Conversely, in theory, the "isolated suppression" of one of the four forms of verbal memory resulted in one of the four fundamental forms of

aphasia – motor aphasia, agraphia, verbal blindness, or verbal deafness.[25] However, Charcot repeatedly emphasised that cases of selective language loss were extremely rare in clinical praxis. Instead, most patients exhibited mixed forms of aphasia in which all four language faculties were affected simultaneously, although to a different degree.[26]

Moreover, Charcot contended that, despite being robbed of language, aphasic patients could still form thoughts in a neurophysiologically separate process of ideation, which transpired in its designated centre.[27] But because of the illness-induced language suppression, these ideas could not be associated with their verbal representations, so the patients' thoughts lacked a concrete form.[28] Without words, thoughts remained vague and imprecise. In Charcot's interpretation, language loss unavoidably affected the patients' abilities to exercise their intellectual faculties. Unsurprisingly, Charcot claimed that organic aphasia correlated with some weakening of the patient's intellectual power.[29] Thus, although thinking and speaking were two neurophysiologically distinct processes for Charcot, he regarded them as functionally interrelated. Disturbances of one process were linked to disturbances of the other.

Establishing Hysterical Mutism as a Genuine Affliction

Whereas by 1885, organic aphasia was an acknowledged medical category, hysterical mutism had not yet been established as a distinct diagnostic entity. Charcot emphasised that cases of hysterical mutism were not rare as they were "mentioned in all writings devoted to hysteria".[30] But unlike aphasia, in hysterical mutism, no structural brain lesion could be identified through postmortem analyses. Hysterical mutism thus appeared to lack a physiological basis, a view that Charcot contested.[31]

25 Charcot, 1889e, 152.

26 Charcot, 1889c, 140.

27 Charcot, 1883c, 523. Charcot argued that the four independent language centres were hierarchically subordinated to the higher-order centre of ideation whose anatomical location he did not specify (Anonymous, 1884, 593).

28 Charcot, 1883c, 523.

29 Charcot, 1883c, 523.

30 Charcot, 1889f, 362.

31 Charcot, 1889f, 360.

Contrary to his colleagues, Charcot did not regard hysterical mutism as a single symptom. Instead, he argued that it was a clinical syndrome consisting "of a very characteristic group of [simultaneous] symptoms" whose distinguishing diagnostic features were unrecognised in the medical community.[32] Due to their ignorance, doctors thus either dismissed patients with hysterical mutism as malingerers or erroneously attributed their symptoms to speech disturbances caused by permanent organic lesions of the central nervous system.[33] Typically, doctors confused hysterical mutism with organic aphasia or with bulbar palsy, a progressive form of labio-glosso-laryngeal paralysis arising from permanent damage to the motor centres in the brain stem.[34] In his seminal 1886 lecture on hysterical mutism, Charcot set out to resolve this problem by delineating the chief clinical features that distinguished hysterical mutism from malingering and from speech loss due to structural brain damage.

To that end, Charcot presented to his audience a thirty-three-year-old male patient who had suddenly become mute after "a laryngitis of only a few hours' duration".[35] According to Charcot, this patient presented all the classical features of hysterical mutism, including the sudden onset of speech loss. However, Charcot also asserted that this case might appear unusual since the mutism was the patient's only hysterical symptom. Typically, hysteria patients had multiple concurrent symptoms, such as seizures, contractures, tremors, visual and sensorial disturbances, and pharyngeal anaesthesia.[36] But Charcot explained that hysterical mutism "may sometimes be met with completely isolated" as a monosymptomatic manifestation of hysteria.[37] Such cases were considered challenging, yet Charcot insisted that doctors familiar with the distinctive features of hysterical mutism could easily make an accurate diagnosis.

First, to demonstrate how to differentiate hysterical mutism from intentional simulation, Charcot prompted the patient to speak. The patient, however, was unable to comply. After Charcot's continued prompting, the patient gestured with his hand towards his throat "as though he would tell us that the difficulty lies there".[38] Charcot asserted that this gesture was characteristic since many patients with hysterical mutism have a subjective feeling of constriction in their throat,

32 Charcot, 1889f, 360.
33 Charcot, 1889f, 368.
34 Charcot, 1889f, 360.
35 Charcot, 1889f, 370.
36 Charcot, 1889f, 367.
37 Charcot, 1889f, 367.
38 Charcot, 1889f, 370.

which they think causes their mutism.[39] Since simulators could not know this, they were more likely to gesture towards their mouth or head. Charcot thus instituted a simple hand gesture with which hysteria patients communicated their inability to speak into a differential diagnostic sign.

However, Charcot warned his colleagues not to rely on a single diagnostic sign to exclude the possibility of simulation. Instead, he instructed them to attentively examine multiple aspects of the patient's communicative behaviour during the clinical interview. He underscored that a "legitimate mute" always remained completely silent whatever the doctor said or did.[40] Even if startled or made to laugh, the patient would not emit a single sound. Just as importantly, patients with hysterical mutism were fully aware of their speech loss and thus tended not to waste time on "useless attempts" at verbal articulation.[41] Instead, if asked a question, they responded without hesitation using either non-verbal gestures or, if given the opportunity, by writing down their answers on paper. Charcot stressed that the patients avoided unnecessary gestures and aimed to communicate as clearly and efficiently as possible. Conversely, simulators tended to add "all sorts of embellishments" by performing superfluous, meaningless gestures or producing inarticulate sounds, thus failing to remain silent.[42]

Notably, the salient diagnostic features that, according to Charcot, differentiated hysterical mutism from malingering could not be identified through physiological measurements. Instead, to exclude simulation, doctors had to judge the intersubjective, relational aspects of the speechless patient's behaviour during the clinical encounter. What mattered was the quality of the patient's silence. But the silence in itself was not sufficient for diagnosis. The doctor also had to interpret the patient's non-verbal gestures. The unspoken implication of Charcot's instructions was that the crucial diagnostic difference between hysteria patients and malingerers lay in the disparate motives underpinning their non-verbal gestures. The malingerers' exaggerated gestures were meant to convince the doctor that their fake muteness was real. Their gestures thus focused on demonstrating their purported speechlessness. Contrary to this, genuine patients experienced their mutism as an inconvenience they tried to circumvent by communicating their thoughts as economically and as distinctly as possible through non-verbal gestures and writing. Their gestures focused on the message they wanted to convey. Hence, to diagnostically distinguish hysterical mutism from malingering, doctors not only had to assess the quality of the patients'

39 Charcot, 1889f, 369.
40 Charcot, 1889f, 369.
41 Cartaz, 1889, 431.
42 Charcot, 1889f, 368.

silences, but also to infer the communicative motives underpinning the patients' non-verbal gestures.

Differentiating Hysterical Mutism from Similar Organic Disorders

Having excluded malingering, the doctor still had to determine if his speechless patient was suffering from an organic disease. To show how this is done, Charcot introduced a seventy-one-year-old male patient with advanced bulbar palsy. He then compared him to the thirty-three-year-old patient with hysterical mutism to "accentuate the contrast and to bring out the distinctive clinical characters of the two afflictions".[43] But before highlighting the differences between the patients, Charcot first delineated their shared features. Although deprived of speech, each patient "preserved the power of conversing by gesture to perfection".[44] Moreover, both understood everything that was said to them and could write.

Beyond these resemblances, there were multiple diagnostically salient differences between the patients. Unlike the sudden onset of complete speech loss in hysterical mutism, speech difficulties caused by bulbar palsy developed slowly and progressively.[45] Moreover, patients with bulbar palsy, which was always fatal, never entirely lost the ability to articulate words. As Charcot demonstrated, his seventy-one-year-old patient with advanced palsy could still feebly "pronounce some indistinct words".[46] This indicated that his speech difficulties were due to paralysis of the lips and tongue. The paralysis, in turn, had been caused by the destruction of the motor centres in the brain stem.[47] In other words, the patient with palsy still knew how to articulate words, but could not move his lips and tongue. To support this claim, Charcot drew attention to the patient's facial expression: his mouth was widened, his tongue immobile and atrophied, and his lips hung loosely.

By contrast, the patient with hysterical mutism retained the ability to move his tongue and lips "perfectly in every direction".[48] Charcot tested this by asking the patient to put out his tongue, then purse his lips and blow out the air. The thirty-three-year-old hysteria patient fulfilled these requests without difficulty. Charcot noted

43 Charcot, 1889f, 361.
44 Charcot, 1889f, 361.
45 Charcot, 1889f, 371.
46 Charcot, 1889f, 371.
47 Charcot, 1889f, 371.
48 Charcot, 1889f, 371.

that another efficient way to test the integrity of patients' lip movements was to ask them to whistle. Patients with hysterical mutism could whistle, whereas those with bulbar palsy could not.[49] Thus, in this context, a seemingly meaningless non-vocal output, such as whistling, acquired diagnostic salience by allowing the doctor to distinguish hysterical mutism from bulbar palsy.

Yet, unlike the patient with bulbar palsy, the hysteria patient could not pronounce a single word despite the unrestricted mobility of his lips and tongue. Furthermore, even when trying his best, the hysteria patient could not "imitate the movements of articulation which he sees [being performed] before him".[50] Drawing these facts together, Charcot concluded that the patient with hysterical mutism was unable to perform the coordinated movements required to produce articulated speech. Simply put, the hysteria patient seemed not to know how to move his lips and tongue in order to speak. Although a comparable inability to execute specialised movements underpinning the speech production was a recognised characteristic of Broca's motor aphasia of organic origin,[51] Charcot assured his colleagues that it was just as easy to diagnostically distinguish hysterical mutism from organic aphasia as it was from bulbar palsy.

To articulate the diagnostic differences, Charcot stated that patients with hysterical mutism were mute in a stricter sense than those with organic aphasia. "[E]ven in the most complete organic motor aphasia the patient is able to call out, to enunciate a few syllables in a loud voice, even to pronounce a few words, albeit not appropriate ones, but perfectly distinct".[52] However, patients with hysterical mutism could pronounce neither a single word nor a syllable. Furthermore, Charcot declared that, compared to aphasic patients, those with hysterical mutism were even "*more than mute*".[53] Aphasic patients could shout and produce loud, inarticulate sounds with their larynx.[54] Yet patients with hysterical mutism were entirely aphonic – they lost their voice. As his thirty-three-year-old patient demonstrated, the only sound he could emit was a short, feeble grunt. Charcot foregrounded the hoarseness of this restrained grunt and how effortful it was for the patient to make it.[55] Hence, both the sonic quality of the patients' inarticulate vocal outputs and the physical effort required for their production attained diagnostic significance.

49 Charcot, 1889f, 363, 366.
50 Charcot, 1889f, 363.
51 Charcot, 1889f, 364.
52 Charcot, 1889f, 364.
53 Charcot, 1889f, 363, emphasis in original.
54 Charcot, 1886, 34.
55 Charcot, 1889f, 363.

However, Charcot cautioned his colleagues against reducing hysterical mutism to aphonia by assuming that the "patient is mute because he has no voice, because the larynx and the vocal cords do not vibrate properly".[56] Instead, he noted that aphonia caused by the weakness of the larynx was a frequent symptom of hysteria, even in the absence of mutism. But in such cases, although they could no longer produce loud sounds, patients could talk in a low voice or whisper. Charcot clarified that during whispering, the formation of vowels and consonants depended on the coordinated movements of the tongue and lips, whereas the larynx and vocal cords remained motionless.[57] Thus, in cases of simple aphonia, hysteria patients were voiceless but not speechless. Conversely, the distinctive feature of hysterical mutism was that patients were both voiceless and speechless, thus also unable to whisper.

Next, drawing on his previous research into organic aphasia, Charcot postulated that patients with organic motor aphasia and those with hysterical mutism could neither talk loudly nor whisper because they were deprived of the motor images (i.e., partial memories) "necessary for the calling into play of articulate speech".[58] In both afflictions, the loss of the designated mental images resulted in patients' inability to execute specialised movements required to articulate words. The implication entailed in this statement, which, as we will see later, Charcot elaborated at the end of his lecture, was that disturbances of the cerebral centre of articulated language underpinned both afflictions.

But at this point in his lecture, Charcot focused on further explaining how to diagnostically differentiate hysterical mutism from organic aphasia. He reminded his audience that, as stated in his previous lectures, cases of pure motor aphasia were exceedingly rare. In most clinical cases, the loss of articulate speech was accompanied by the patient's inability to communicate by gestures and some level of verbal blindness and verbal deafness.[59] Moreover, most aphasic patients could not write or did so only imperfectly and effortfully. By contrast, patients with hysterical mutism had no difficulties understanding the spoken or written language and were apt at expressing themselves through non-verbal gestures.

Even more characteristically, patients with hysterical mutism had a perfectly preserved writing faculty. Charcot emphasised that they wrote quickly, effortlessly, and with "singular readiness".[60] All this could be established by observing the patients while they wrote. However, Charcot did not stop at that. He carefully

56 Charcot, 1889f, 363.
57 Charcot, 1889f, 363–364.
58 Charcot, 1889f, 364.
59 Charcot, 1889f, 364.
60 Charcot, 1889f, 365.

analysed the content, orthography, and style of hysteria patients' written responses to his questions, noting such detail as the presence of humour.[61] He also compared the patients' written responses to examples of their writing from before they had fallen ill. He thus concluded that, unlike aphasic patients, individuals with hysterical mutism remained capable of rendering "their thoughts in writing exactly as they could before the development of the disease; that is to say in a style and with an orthography quite in keeping with the education" they had received.[62]

Based on the quality and complexity of their written responses and the clarity and communicative efficacy of their non-verbal gestures, Charcot forcefully argued that patients with hysterical mutism had "lost nothing, absolutely nothing" of their intelligence.[63] In other words, Charcot claimed that whereas some weakening of intellectual power accompanied organic aphasia, the intelligence of patients with hysterical mutism remained unaffected by their disease. While aphasic patients had problems associating their thoughts with verbal representations, patients with hysterical mutism did not. One Charcot's patient pointedly expressed this by reporting that he understood everything others said, and the words immediately came to him to answer, yet his tongue refused to move.[64] In Charcot's view, the swiftness with which hysteria patients translated their thoughts into words was hysterical mutism's most distinctive diagnostic feature.[65]

In sum, by systematically comparing hysterical mutism to malingering, bulbar palsy, and organic aphasia, Charcot demonstrated that this surprisingly complex manifestation of hysteria had a distinct clinical character. In the process, he taught his colleagues that to diagnostically identify hysterical mutism, they had to listen to the quality and extent of the patients' silence, assess the materiality of the seemingly senseless noises, analyse the patients' facial expressions and non-verbal gestures, and pay attention to the accuracy, consistency, and style of their written responses. Based on such analysis, Charcot identified six principal symptomatic features that, as he argued, jointly defined hysterical mutism as a distinct clinical syndrome. These comprised: first, complete loss of articulate speech, including the ability to whisper; second, complete loss of voice; third, preservation of the general movements of the lips and tongue; fourth, absence of verbal blindness and verbal deafness together with the preserved ability to communicate by gestures; fifth, fluency in writing; and sixth, intact intelligence.

61 Cartaz, 1889, 417.
62 Charcot, 1889f, 361.
63 Charcot, 1889f, 365.
64 Charcot, 1892, 293.
65 Cartaz, 1889, 431.

In effect, Charcot's description of this distinctive symptom clustering revealed the paradoxical character of hysterical mutism. Patients with hysterical mutism suffered a more excessive loss of both voice and spoken language than patients with comparable language disorders of organic nature. However, the language loss in hysterical mutism was selectively limited to speech, whereas other language faculties – and the patients' intellect – remained intact. As Charcot repeatedly insisted, such clear-cut dissociation of otherwise mutually interrelated language faculties rarely occurred in organic diseases. Yet, as we are about to see, instead of regarding this paradoxical aspect of hysterical mutism as medically inexplicable, Charcot drew on it to postulate a neurophysiological mechanism underpinning hysterical mutism.

Linking Hysterical Mutism to a Reversible Localised Brain Dysfunction

While Charcot's seminal lecture on hysterical mutism focused on identifying this syndrome's diagnostic specificity and establishing it as a clinical entity, the insights won in this process had an added benefit. They allowed Charcot to make inferences about the potential neurological basis of hysterical mutism. Thus, the unambiguous diagnostic differentiation between hysterical mutism and malingering was epistemically significant because it indicated that hysterical mutism, as a genuine affliction, had to have a physiological basis. But to identify the syndrome's underlying neurophysiology, Charcot reverted to comparing hysterical mutism to bulbar palsy, organic aphasia, and hysterical paralysis.

By contrasting clinical features of hysterical mutism and bulbar palsy, Charcot posited that the loss of articulate speech in these two disorders relied on different mechanisms.[66] He thus excluded the possibility that, like bulbar palsy, hysterical mutism was caused by the paralysis of the facial nerves. Next, as mentioned earlier, Charcot conjectured that in organic aphasia and hysterical mutism, patients suffered from the suppression of partial memories of speech articulation. Since, according to Charcot, such partial memories had their seat in the specialised language centre of the brain, he thus effectively designated hysterical mutism as a brain-based disturbance. He then turned to specifying the presumed nature and location of the underlying brain disturbance.

66 Charcot, 1889f, 371.

To that end, Charcot outlined the syndrome's peculiar temporal dynamics. Whereas the speech loss in organic aphasia was permanent, in hysterical mutism, it was not. Not only was the onset of hysterical mutism sudden, but also its duration varied considerably.[67] In some patients, the mutism lasted a few hours; in others, several days, months, or even years. In most patients, hysterical mutism disappeared as suddenly as it had appeared, although the speech recovery was initially partial. Before fully regaining their speech, patients underwent a transitional period of a few days or weeks. During this period, the recovered speech lacked fluency and was characterised by "a peculiar stammering consisting of the frequent repetition of the same syllable".[68] Yet even after complete recovery, relapses were frequent, with many patients experiencing recurring spells of mutism.

Because of the volatile, transitory nature of hysterical mutism, which all hysterical symptoms had in common, Charcot conjectured that the underlying brain disturbance had to be "of a purely dynamic order".[69] Unlike structural damage to the cerebral language centres, which caused different forms of organic aphasia, the "dynamic lesion" in hysterical mutism was unrelated to any permanent pathological changes of the brain tissue.[70] Instead, Charcot posited that the dynamic lesion in hysterical mutism – similarly to dynamic lesions that, as he claimed, underpinned other hysterical symptoms – consisted of a temporary disturbance of function of a particular brain centre. In a series of lectures he held in 1885, Charcot attributed hysterical arm paralysis to such a dynamic lesion, which he argued was located in the cortical motor centre of that arm.[71] According to Charcot, in hysterical arm paralysis, the dynamic lesion was equivalent to the functional inhibition of this centre, which rendered the centre incapable of producing the mental image of movement necessary for initiating voluntary movements.[72] A year later, Charcot extended this hypothesis by claiming that a comparable functional inhibition of the designated brain centre underpinned hysterical mutism.[73] Because of this functional inhibition, the patient could not recall the memorial images of words and became speechless.

As discussed previously, Charcot had already attributed hysterical mutism to selective suppression of the motor images of articulation. In his aphasia research, Charcot had argued that motor images of articulation had their seat in Broca's

67 Charcot, 1889f, 362.
68 Charcot, 1889f, 363.
69 Charcot, 1889f, 360.
70 Charcot, 1889f, 373.
71 Charcot, 1889a, 278.
72 Charcot, 1889a, 310. See also Muhr, 2022, 156–178.
73 Charcot, 1889f, 373.

centre, which occupied the third convolution of the left frontal lobe. Drawing these insights together, Charcot suggested that the dynamic lesion that caused hysterical mutism was limited to Broca's centre without affecting any other cerebral language centres.[74] He emphasised that such functional selectivity was a typical feature of dynamic lesions, whereas structural lesions usually occupied multiple neighbouring brain centres, regardless of their different functional specialisations.

Notably, because the dynamic lesion amounted to the centre's disturbance of function without any accompanying destruction of anatomical structure, it was potentially reversible. Put simply, in hysterical mutism, the motor images of articulation were not obliterated from Broca's centre, but merely inaccessible to conscious recall. Thus, through the influence of other cerebral centres to which it was structurally connected, Broca's centre could spontaneously become disinhibited, and the patient would immediately regain the ability to recall the motor images of articulation. After a transitional period, during which the motor images of articulation were sufficiently revived through repeated recall, the patient recovered fluent speech. Thus, although in clinical terms, hysterical mutism entailed a more extensive loss of articulate language than organically caused mutism, in Charcot's final analysis, this loss was temporary. The patient could regain speech because the underlying neural basis of hysterical mutism was a highly selective, transitory disturbance of brain function.

Conclusion

In sum, I have shown that to establish hysterical mutism as a distinct diagnostic category, Charcot attentively listened to his patients' silences and to every type of acoustic utterance they could produce. He systematically tested whether his speechless patients could enunciate single syllables, pronounce consonants, utter loud sounds, or whisper. In this context, seemingly meaningless noises, such as feeble cries, low-pitched grunts, and whistling, acquired diagnostic relevance. But to become diagnostically relevant, non-verbal acoustic outputs and instances of silence had to be interpreted in relation to the patients' facial expressions, communicative gestures, and other salient aspects of their behaviour, such as the preserved ability to understand spoken words and to write.[75]

74 Charcot, 1892, 267–268.

75 Because the patients discussed in this chapter were all men, some might suggest that Charcot's willingness to listen was limited to male patients. However, due to his conviction that hysteria was identical in men and women, Charcot applied the same diagnostic categories and

I have highlighted how, during his diagnostic encounters with mute patients, Charcot assessed the material quality of patients' vocal outputs, the expressive quality of their non-verbal gestures and written responses, and the physical effort they invested into communicating. He also evaluated the content and consistency of the patients' non-vocal responses, the accuracy and style of their writing, and their willingness and motives to communicate with him. In doing so, he took note of their humour, appraised their intelligence, and inferred their education level. Based on such comprehensive analysis and interpretation of the patients' missing as well as preserved language abilities, Charcot identified the distinctive diagnostic features of hysterical mutism and postulated the syndrome's neurophysiological basis. Moreover, he demonstrated that patients with hysterical mutism were neither malingering nor was their speech loss necessarily permanent. As suddenly as it appeared, the underlying localised brain dysfunction that caused hysterical mutism might spontaneously disappear, and the patient could speak again.

Charcot made these wide-ranging discoveries by jointly interpreting his patients' silences, seemingly meaningless noises, gestural expressions, and written statements. Admittedly, his listening did not encompass enquiring about his patients' subjective experiences of their illness. Such information did not seem diagnostically relevant in his neurophysiological approach to hysterical mutism, which, as we have seen, was informed by his previous aphasia research. While listening, Charcot thus selectively focused on those vocal, gestural, and written expressions that he could incorporate into this interpretative framework. Despite its limitation, his approach was epistemically productive as it generated new medical insights into a previously contested affliction.

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procedures to both genders. He thus diagnosed and photographed hysterical attacks of male patients according to the criteria he had initially developed for female patients (see Muhr, 2022, 56–87). To diagnose hysterical mutism, Charcot used the same procedure, which entailed attentive listening and communication assessments, with male and female patients (see Charcot, 1892, 273; Charcot, 1889b, 247–250; Charcot, 1889f, 369–370).

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