

Jean-Martin Charcot (1825–1893)

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Summary

Jean-Martin Charcot (1825–1893), son of a Parisian craftsman, went on to a brilliant university career and worked his way to the top of the hospital hierarchy. Becoming a resident in 1858 at the women’s nursing home and asylum at La Salpêtrière Hospital, he returned there in 1868 as chief physician. Observing more than 2,000 elderly women, he first worked as a geriatrician–internist, leading him to describe thyroid pathology, cruric pulmonary embolism, and so forth. To deal with the numerous nervous system pathologies, he applied the anatomoclinical method with the addition of microscopy. In less than around 10 years, his perspicacious clinical eye enabled him to describe Parkinson’s disease, multiple sclerosis, amyotrophic lateral sclerosis, and tabetic arthropathy and to identify medullary localizations, for example. Already aware of functional neurological disorders, at that time referred to as hysteria and frequent to this day, Charcot used hypnosis to try to decipher the pathophysiology. His thinking gradually evolved from looking for lesions to recognizing triggering psychological trauma. This prolonged search, misinterpreted for years, opened the way to fine, precise clinical semiology, specific to neurology and psychosomatic medicine. Charcot knew how to surround himself with a cohort of brilliant clinicians, who often became as famous as he was, notably Pierre Marie (1853–1940), Georges Gilles de la Tourette (1857–1904), Joseph Babiński (1857–1932), and Pierre Janet (1859–1947). This cohort and the breadth of Charcot’s innovative work define what is now classically called the “Salpêtrière School.”

Keywords: Jean-Martin Charcot, history of neurology, Parkinson’s disease, multiple sclerosis, Charcot disease, Charcot–Marie–Tooth disease, Charcot foot

Subjects: Disorders of the Nervous System, Motor Systems

“The history that will be presented to you today is that of a very simple and very glorious life; it is quite beautiful to recount” (Marie, 1925). When Jean-Martin Charcot was born on November 29, 1825, his parents lived at 1 rue du Faubourg–Poissonnière in Paris’s ninth district. He was the eldest son of Simon Pierre Charcot (1798–1863), a saddler–coach builder, and Jeanne-Georgette Saussier (1808–1839). He had four brothers: Eugène Charcot, born on December 23, 1826, died after only 15 days; Pierre-Martin Charcot (1828–1906) took over his father’s business; Émile-Martin Charcot (1830–1899) devoted himself to a military career, becoming an officer and captain of the infantry; and lastly, Jean-Eugène Charcot (1831–1869), also in the military, died in Senegal, probably of malaria.

Charcot passed his “baccalauréat” exam on August 31, 1843. His father had announced during a family meal he could only afford long costly studies for one of his children, in principle the eldest, and thus Jean-Martin.

Medical Training

After passing the competitive exam for external hospital students (*externe*) in December 1845, Charcot was named a temporary resident in 1847, then passed the competitive residency exam (*interne*) for the Hospitals of Paris on December 18, 1848, along with Alfred Vulpian (1826–1887), who became his inseparable friend. He was successively the resident of Louis Béhier (1813–1876) and Pierre Rayer (1793–1867) at the famous La Charité Hospital, of Pierre-Adolphe Piorry (1794–1879) at the old La Pitié Hospital, and of Eugène Cazalis (1808–1883) in his fourth year of residency at La Salpêtrière Hospital: “[At La Salpêtrière,] he gathered the elements of his inaugural thesis, knew how to appreciate the heaps of inexhaustible resources in this women’s nursing home and asylum, and resolved to return there as a physician” (Joffroy, 1893). Piorry, a professor of clinical medicine who had already promoted the use of the microscope, offered Charcot a position as his senior resident (*chef de clinique*) in 1853 and 1854. But it was Rayer, future physician to Napoléon III, who would have the most decisive influence on Charcot’s career. Working alongside Rayer, Charcot bolstered his experience as a powerful leader who remained attentive to his students. Before he allowed him to pass the “agrégation” exam (opening the way to an associate professorship), Rayer had him join the French Society of Biology, which he presided over at the time and had founded with Claude Bernard (1813–1878) and Charles Robin (1821–1885). Charcot became a full member in 1851, “a young man and colleague of eminent personalities” (Goetz et al., 2005), such as Claude Bernard, Charles Brown-Séquard (1817–1894), François Magendie (1783–1855), and Émile Littré (1801–1881). They discussed advances in clinical science and laboratory research and were strongly shaped by the positivist philosophy of Auguste Comte (1798–1857). By belonging to this society, Charcot was able to publish his own work very early on, and by remaining faithful to it throughout his career, he in turn did not fail to sponsor the entry of most of his residents (Figures 1 and 2).

JEAN MARTIN CHARCOT.



AGE 35. 1860.



AGE 38. 1863.



AGE 43. 1868.



AGE 47. 1872.



AGE 54. 1879.



AGE 55. 1880.

Figure 1. Jean-Martin Charcot at different stages of his life (private collection of the author).

Charcot's thesis was his first nosographic work. In it, he distinguished gout from chronic rheumatism (considered today as rheumatoid polyarthritis and degenerative arthritis, respectively). The importance he placed on references to the English and German literature not only indicated his erudition and mastery of foreign languages but also established an innovative approach to this kind of personal work. He defended his thesis on March 16, 1853, with Piorry presiding over the jury (J.-M. Charcot, 1853). His talents as a draughtsman were apparent in the drawings of hands with deformed fingers that illustrated his thesis.

Rayer has also helped him develop a private clientele. It was on his recommendation that the family of the banker to Napoléon III, Achilles Fould (1800–1867), went to Charcot for medical care, providing him with the necessary network to become the physician of Paris's high bourgeoisie. At this point, he no longer had any financial worries.



Figure 2. Jean-Martin Charcot with his wife around 1890 (private collection of the author).

In 1856, Charcot passed the competitive exam for the “Central Office”; that is, he acquired the status of a hospital physician. On April 17, 1857, at his first attempt to pass the “agrégation” exam (to become an associate professor), he was given a thesis subject that hardly inspired him: “expectation in medicine.” He failed after having expressed numerous doubts about the approach, commonly practiced at the time, whereby no treatment was provided during pneumonia. He admitted having observed “the medicative power of nature” in this disease but went on to say that “in general, [it] imperiously calls for active and energetic medication.” The lack of conviction and thus of a clear conclusion worked against him.

In 1860, when he became vice president of the French Society of Biology, he also successfully passed the “agrégation” exam (to become an associate professor), as did his friend Vulpian. This “agrégation” thesis “on chronic pneumonia” was a compilation of works mostly focused on febrile prolonged progression, from acute lobar pneumopathy to a serious degradation in general condition and ultimately to death. The autopsy found gangrene in the lungs, with wide, nonfunctional fibrous segments, sometimes with cavities but without tubercles. The etiology remained mysterious at a time when bacteriology was still unknown.

Ten years after having been a resident at the women’s nursing home and asylum at La Salpêtrière Hospital, Charcot was appointed chief physician there in 1862, taking over the Pariset division from Cazalis. Charcot was 37 years old. His friend Vulpian was appointed to head the second entity, the Pinel division. On July 1, 1862, the two friends were in charge of 2,635 patients (Husson, 1862). At a time when the nosography of chronic pathology, notably that affecting the nervous system, was in its infancy,

the two young “agrégés” (associate professors) could be seen working together from room to room of this immense asylum, examining all the patients, gathering all the observations, and compiling an enormous dossier that gradually expanded to include autopsies and histological studies and the precious contribution of laboratory research.

(Joffroy, 1893, p. 579)

They were applying the anatomoclinical method. In France and England, the first half of the 19th century saw the development of this method, which compared the examination of the patient, as objectively as possible, with the anatomical lesions found at autopsy. René Théophile Laënnec (1781–1826), who combined medial auscultation with the use of a stethoscope to study macroscopic lesions of the lung and heart, remains the emblematic figure of this period. But in neuropathology, the real master was Jean Cruveilhier (1791–1874), the first to hold the chair of anatomicopathology at the Paris Medical School. After completing his residency with Guillaume Dupuytren (1777–1835), he defended his thesis in 1816—*Essai sur l’anatomie pathologique en général* (Essay on Pathological Anatomy in General)—in which he noted that “all good minds in France today are driven by an ever-increasing ardour” toward pathological anatomy and physiology, closely linked to medicine. In the mid-19th century, the advent of the achromatic optical microscope and the cell theory introduced by Mathias Schleiden (1804–1881) and Theodor Schwann (1810–1882), which was perfected by Rudolph Virchow (1821–1902), led to a revolution, given that the macroscopic pathological anatomy of the early 19th century was purely

macroscopic (Poirier, 1999). In his first lesson on the diseases of the elderly, in 1874, Charcot underscored this profound revolution in medicine wrought by “histology armed with the microscope” (J.-M. Charcot, 1867), and at Vulpian’s funeral, he emphasized that

macroscopic pathological anatomy had, in Cruveilhier’s hands, the highest possible degree of perfection, but it was no longer enough. On the other side of the Rhine, Virchow had paved the way to the study of cellular lesions. In France, Vulpian was to be the man of this radical shift.

(J.-M. Charcot, 1887, p. 451)

In only 8 years, from 1862 to 1870, the two friends enriched medical nosography, adding to it the clinical features of multiple sclerosis and Parkinson’s disease and describing tabetic arthropathy and medullary localizations, among others. In 1866, Charcot began teaching, in his department and outside the Paris Medical School, mixing theory and clinical elements of chronic disease, notably in the elderly (J.-M. Charcot, 1867), then in nervous system diseases. He quickly became known for the originality and quality of his lessons, compiled by Benjamin Ball (1833–1893). They attracted more and more attendees, notably foreigners visiting Paris, and thus laid the foundation for the Salpêtrière School.

The Franco-Prussian War, the revolutionary Commune, and the defeat hindered Charcot but did not stop him from pursuing his career. He remained in Paris but sent his wife and two children first to Dieppe, then to London, when the Germans were approaching the English Channel. Wearing a Red Cross armband that allowed him to get past the barricades of the Commune, he reached, not without risk, the women’s nursing home and asylum, situated in east Paris, whereas he resided at 6 avenue du Coq, situated in west Paris, near the new Saint-Lazare train station (J.-B. Charcot, 1926). At La Salpêtrière, he treated not only wounded soldiers but also numerous victims of smallpox and cholera epidemics, which interrupted his research and his private practice.

Once peace was reestablished, in 1872, he was appointed to the chair of pathological anatomy, replacing Vulpian, who went on to hold the chair of experimental pathology. Whereas Charcot tended to be reticent on political subjects, the ideology of the young Third Republic was in keeping with his positivism and anticlerical views. In 1873, Charcot was elected to the French Academy of Medicine, then in 1883 to the French Academy of Sciences.

A dozen of “free” lectures, given in a refectory of the department, were published in various journals and then grouped together in 1872 in a book entitled *Leçons sur les maladies du système nerveux* (Lessons on Nervous System Diseases).

Physician at La Salpêtrière Hospital

From his beginnings at La Salpêtrière, Charcot had his medical staff systematically take residents' temperature using a mercury thermometer and not simply with their hand. This was an innovation. His 1868 resident, Désiré-Magloire Bourneville (1840–1909), made this the subject of his thesis and later publications (Bourneville, 1870,1873).

Charcot was initially a geriatrician. In 1856, he wrote the first dissertation summarizing the symptomatology of Graves' disease, unknown at that time in France. He insisted that the heart should not be considered the pathology's cause but remarked on the modifications to the structures and dimensions of the thyroid arteries. He put forth the hypothesis of an increase in the gland's activity by stimulation of the vasomotor nerves; auscultating the goiter, he noticed "a continuous blowing sound." For him, the increase in the volume of the acini was the result (J.-M. Charcot, 1856b).

François-Amilcar Aran (1817–1861) focused his 1853 "agrégation" thesis (to become an associate professor) on causes of sudden death, noting pulmonary embolism but without indicating the point of departure. Ball and Charcot showed in 1858 that a clot blocking the pulmonary artery was caused by venous phlebitis in a limb (Charcot & Ball, 1858).

It is important to remember Charcot's description of octahedral crystals seen in the blood of a patient with leukemia, which Ernst Victor von Leyden (1832–1910) would find again in 1872 in the spital of patients with asthma, hence the eponym Charcot–Leyden crystals. These crystals are made of an enzyme, lysophospholipase, which is synthesized by eosinophil granulocytes, blood cells not yet described in Charcot's time (Walusinski, 2022).

From his early days at La Salpêtrière, Charcot multiplied his publications on various subjects: endocarditis, arsenic intoxication, canities, fever, thrombotic complications, cancer, and cholera, among others. His book on the diseases of the elderly was published in 1867, attesting to his activity as a geriatrician–internist before the specialty existed.

It is not possible to cover here Charcot's entire neurological oeuvre. But this article reviews his main works, intertwined over time, with some research subjects put aside to publish other works, then a later return to the initial research.

Multidisciplinary Study of Neurological Illness

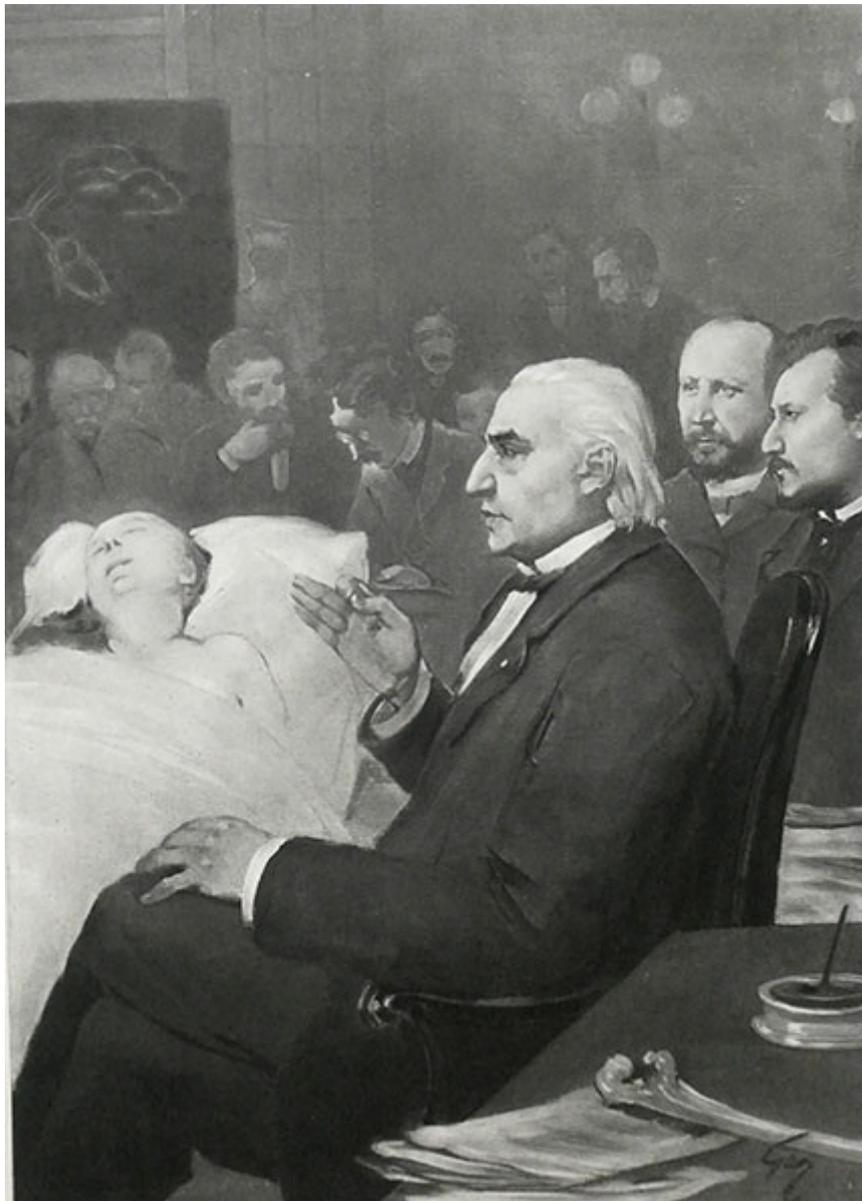


Figure 3. An example of a handwritten letter and drawings sent by Charcot to his wife, who remained in Paris while he was traveling in Russia (private collection of the author).

Charcot's visual perception and memory were extraordinary, and he possessed special artistic gifts as well (Figure 4). They were first a hobby for him and later a professional tool (Meige, 1898). He always surrounded himself with dedicated students and colleagues who helped him with his research and its dissemination. Bourneville founded the journals *Progrès médical* and *Iconographie photographique de La Salpêtrière*, not to mention his publication of collections of the master's lessons and his students' theses (Poirier et al., 1991). Richer illustrated hysteria at La

Salpêtrière and was influenced by the art of previous centuries. He also created statuettes for educational purposes (*La Parkinsonienne*; Walusinski, 2023). Albert Londe photographed patients and anatomical parts (Walusinski, 2021).

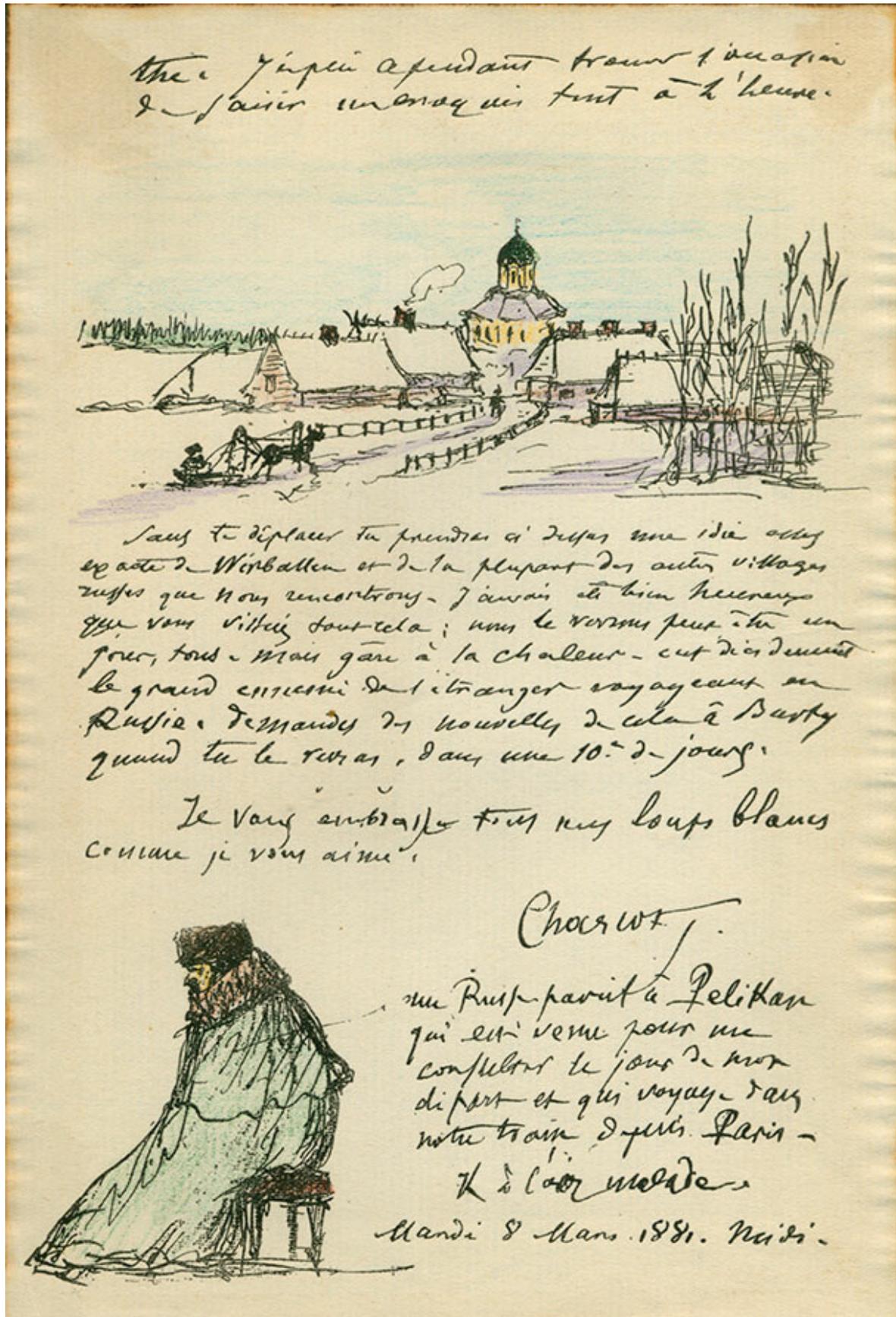


Figure 4. Jean-Martin Charcot teaching under the watchful eyes of Adolph Dutil (1862-1940) and Paul Richer (1849-1933; private collection of the author).

According to Joseph Babiński (1857–1932), Charcot had

faculties worthy of the great observers enabling him to discern hitherto unnoticed facts or those for which only a facet had been described. He had the capacity to concentrate his attention on them and see them from another point of view.

(Babiński, 1925, p. 748)

The frequency of paralysis and abnormal movements in the women at the La Salpêtrière nursing home and asylum, some of whom had been hospitalized for many years, naturally led him to perfect the body of knowledge of the diseases of the nervous system, at that time still poorly understood. He also became a great teacher in this area (Figure 3).

Parkinson's Disease

In the November 29, 1861, issue of *La Gazette hebdomadaire de Médecine et de Chirurgie*, Charcot and Vulpian published their first collaborative article called “De la paralysie agitante” (on paralysis agitans; Charcot & Vulpian, 1861). The didactic goal of this article, a quasi-seminal description in French (Trousseau, 1859), is manifest. Charcot would introduce soon thereafter the denomination Parkinson's disease, a substitute for the inadequate term of *paralysis agitans*. The chapter on symptoms, mode of progression, and prognostics gave an accurate description of shaking, “the feeling of muscular stiffness,” “the irresistible propulsion,” and slow speech, despite “very clear and very accurate comprehension,” but “later, in general, psychological faculties decisively weaken.” The prognosis “is very sad” due to “the weakening and especially the motor paralysis, along with the debilitation of memory and intelligence, which demonstrate that the reach of the disease is increasingly profound.” “Therapy is more or less powerless against the disease's progression.” It should be noted that Charcot was the first to distinguish bradykinesia in Parkinson's disease and separate it from rigidity or weakness:

Yet, long before rigidity actually develops, patients have significant difficulty performing ordinary activities; this problem relates to another cause. In some of the various patients I have presented to you, it is easy to recognise how difficult it is for them to do things, even when rigidity or tremor is not the limiting feature. Instead, even a cursory examination demonstrates that their problem relates more to slowness in execution of movement rather than to real weakness. In spite of tremor, a patient is still able to do most things, but he performs them with remarkable slowness. One would think neural activity can only be effected after remarkable effort.

(J.-M. Charcot, 1892, p. 169)

Cerebral Vascular Pathology

Several influences led Charcot to take an interest in vascular pathology. During his residency under Cazalis at La Salpêtrière Hospital in 1852, he observed frequent cases of chronic gangrene in the lower limbs (J.-M. Charcot, 1856a). He was also struck by the number of patients at the women's nursing home and asylum with hemiplegia (Lellouch, 1992). Interested in the work published in 1847 by Virchow on thromboembolism (Schiller, 1970), he accepted Virchow's findings based on his own clinical and anatomicopathological observations. The examples at that time were almost exclusively cases of rheumatic endocarditis and syphilitic arteritis and, less frequently, artery-to-artery embolism (Paciaroni & Bogousslavsky, 2009). When Charcot was a resident, the debate that had begun at the beginning of the 19th century between the advocates of the inflammatory theory of apoplexy and those of the vascular theory was nearly dead. For him, there was no doubt that the origin of the cerebral lesion was “nutritive,” that is, ischemic. The process is common to all localizations of arterial pathology, whether it develops in the arteries of the limbs or in the cerebral arteries. Charcot's observation of claudication in a lower limb, in 1859, bears this out (J.-M. Charcot, 1859).

Cerebral vascular pathology brings together the names of Charcot and Charles Bouchard (1837–1915), his resident in 1864 and 1866, in the eponym Charcot–Bouchard aneurysm, a rare pathology that causes cerebral hemorrhage (Bouchard, 1866; Charcot & Bouchard, 1868). Current studies confirm the reality of microaneurysms, mainly in lenticulostriate arteries. To explain cerebral hemorrhage, the rupture of saccular aneurysms is possible but also the rupture of an arteriole without aneurysm or arteriolar dissection (Dubas, 2006). Charcot minimized the role of “exaggerated blood pressure in the vessels of the encephalon,” which referred to arterial hypertension, but he lacked the means to measure it; he seemed to prefer “the decreased resistance of vessels following the degradation of their walls” to explain the bleeding.

The observations that Charcot compiled in the 1860s show that he had elucidated very early on the progressive pathophysiology of cerebral infarct. Using the term *cholestérine* (cholesterin), the name of cholesterol at the time, he identified the biological nature of atheromatous plaques (Walusinski, 2019). He had meticulously described its ulceration at the intima of an artery, on which a clot aggregated, causing obstruction of the vessel or emboli flowing downstream, leading to cerebral ischemia and the resultant parenchymal lesions. Ivan Poumeau (1839–1878) revisited these observations in his famous 1866 thesis (Poumeau, 1866).

Locomotor Ataxia or Tabes

In 1858, Guillaume Duchenne de Boulogne (1806–1875) published the description of “a disease characterised especially by general disturbances of movement coordination” (Duchenne de Boulogne, 1858); this was the first description of progressive locomotor ataxia. The German Moritz von Romberg (1795–1873) had given a first description in 1851, referring to “tabes dorsalis” (from the Greek for “melt, liquefy”); for Charcot, this was “an outline” (von Romberg, 1851). The eponymous sign was clearly described: exaggerated imbalance when the eyes are

occluded. In 1862, Charcot and Vulpian published three articles on this disease (Charcot & Vulpian, 1862a,1862b,1863), which Armand Trousseau (1801–1867) had featured in three memorable lessons in January 1861 (Trousseau, 1861). With new accuracy, Charcot and Vulpian described the searing pain, “like being struck by lightning,” that characterized this illness; they also described its modes of progression and the detailed histopathological exams of the entire nervous system, demonstrating damage to the posterior medullary tracts and examining the possibility of initial microarterial damage. They covered the pathophysiology of “gastric attacks” occurring in this disease by “damage to the posterior tracts and atrophy of the posterior roots” (J.-M. Charcot, 1872–1873) as Hippolyte Bourdon (1814–1892) had already reported in 1861 (Bourdon, 1861), before Georges Delamare (1842–1911) did so in this thesis defended on August 25, 1866 (Delamare, 1866), then Paul Dubois in his thesis in 1868 (Dubois, 1868).

In 1868, Charcot described arthropathy in progressive locomotor ataxia (J.-M. Charcot, 1868). At first contested, this clinical picture, which he enriched with photos and anatomical items held at the Dupuytren Museum, gradually gained ground. Invited to the International Medical Congress in London in 1881, he achieved a triumph with his presentation entitled *Demonstration of Arthropathic Affections of Locomotor Ataxia*. James Paget (1814–1899), the president of the congress, brought into common usage the eponyms Charcot foot and Charcot arthropathy, which since then have been used to name arthropathy of neurological origin, like that complicating diabetes.

Multiple Sclerosis

In addition to the clinical picture of paralysis agitans (Parkinson’s disease), Charcot wrote the seminal description of a new pathology, multiple sclerosis, by differentiating between the two clinical pictures based on the type of shaking. Charcot wrested multiple sclerosis from the chaos of the various forms of “chronic myelitis” based on its histopathological specificity, explained during a memorable lesson on September 1, 1868, recorded by Bourneville, his resident at the time: “I have briefly examined its fortune [the word *sclerosis*] and, in my opinion, it corresponds to natural morbid types, characterised by marked anatomical lesions and a set of symptoms sufficiently determined to lead to an accurate diagnosis” (Charcot & Bourneville, 1868). Before this, Vulpian had presented on May 9, 1866, a “note on multiple sclerosis of the spinal cord” (Vulpian, 1866) to the French Medical Society of the Paris Hospitals; then it was Charcot who presented on March 14, 1868, to the French Society of Biology “anatomical parts relative to a case of generalised multiple sclerosis in the brain and spinal cord” (J.-M. Charcot, 1869). Henry Liouville (1837–1887), the first husband of Marie Durvis (1854–1936), who was Mme Charcot’s eldest daughter, published in the same year an extensive, detailed study on multiple sclerosis with Vulpian’s help (Liouville, 1869). The microscopic study showed the disappearance of the myelin sheath with the conservation of “cylindraxes,” surrounded by a fibrillary proliferation. Charcot highlighted that, in contrast with the rest tremor of Parkinson’s disease, in multiple sclerosis, the patients developed predominantly an action tremor, clustered with signs of weakness, sensory abnormalities, visual defects, and, frequently, nystagmus.

Amyotrophic Lateral Sclerosis, or Charcot Disease

One of Charcot's lessons given in June 1868 was published in 1869 with the help of Alix Joffroy (1844–1908), his resident that year. It focused on “two cases of progressive muscular atrophy” (Charcot & Joffroy, 1869), the first milestone in the seminal description of amyotrophic lateral sclerosis, or Charcot disease. The 1850 dissertation of François-Amilcar Aran (Aran, 1850) and the autopsy published in 1853 by Jean Cruveilhier (1791–1874; Cruveilhier, 1852–1853) were Charcot's initial guides along with infantile acute spinal paralysis (future poliomyelitis, or Heine–Medin disease; Charcot & Joffroy, 1870). He compared their descriptions and his own findings: “In the grey matter, these lesions occupy almost exclusively the anterior horns, where they are indicated especially by deep atrophy and even by the disappearance of many large nerve cells”; this explained the muscular atrophy that caused paralysis and muscular fasciculations. Simultaneously, he observed sclerosis in the white matter of the anterolateral tracts along much of the spinal cord's height, which he associated with contraction.

In 1870, Charcot revisited the initial 1860 publication of Duchenne de Boulogne (Duchenne de Boulogne, 1860), adding “glosso-laryngeal paralysis” as a clinical form localized in the brain stem that had the same pathology (J.-M. Charcot, 1870). The Franco-Prussian War interrupted his publications, to which he did not return until 1874. In the complete works published in 1894, the complete description of amyotrophic lateral sclerosis was the focus of three lessons (XI, XII, XIII) that brought together the clinical picture, the various localizations, the progressive forms, and the pathological anatomy (J.-M. Charcot, 1894). The search for the etiology was postponed, which it continues today.

When he coined the term *amyotrophic lateral sclerosis*, Charcot associated the clinical and anatomical aspects: amyotrophy (i.e., gray matter involvement) and lateral sclerosis (i.e., white matter damage). He wisely pointed out to his students,

I do not think that elsewhere in medicine, in pulmonary or cardiac pathology, greater precision can be achieved. The diagnosis as well as the anatomy and physiology of the condition “amyotrophic lateral sclerosis” is one of the most completely understood conditions in the realm of clinical neurology.

(J.-M. Charcot, 1887–1888, p. 151)

Muscle Pathology

Charcot's lessons also reviewed other forms of muscle atrophy: pseudohypertrophy in Duchenne muscular dystrophy (1868), the juvenile form of progressive muscular atrophy of Heinrich Erb (1840–1921), and the hereditary facioscapulohumeral muscular dystrophy of Louis Landouzy (1845–1917) and Jules Dejerine (1849–1917).

In 1886, Charcot and Pierre Marie published five observations of “a special form of progressive muscular atrophy first invading the feet and legs and only found in the upper limbs (hands first then forearms) several years later; hence with slow progression. Existence of fibrillary

contractions in atrophying muscles” (Charcot & Marie, 1886). They were unaware that in England, Howard-Henry Tooth (1856–1925) simultaneously described the same disease in his thesis defended at the University of Cambridge and entitled *The Peroneal Type of Progressive Muscular Atrophy* (Tooth, 1886), resulting in an eponym that combined their three names.

Medullary Localizations

“The principle characteristic of M. Charcot’s studies on the pathology of the spinal cord was to make two entities walk in step, so to speak, by the close connection between clinical practice and pathological anatomy, in light of physiological knowledge”; thus began what Charcot wrote in 1883 in his *Exposé des titres scientifiques* (Presentation of Scientific Titles), introducing his “theory of localisations in spinal disease” (J.-M. Charcot, 1883). Charcot described the pyramidal tract (mobility) by studying degeneration secondary to lesions on its pathway; similarly, he predicted lesions of the lateral tracts, “symmetrical fascicled sclerosis,” for what he temporarily named “spasmodic dorsal tabes.” This was not tabes but “spastic spinal paralysis,” or “Erb-Charcot paralysis,” now recognized as one of the forms of hereditary spastic paraplegia (Strümpell–Lorrain disease; Walusinski, 2020). Charcot’s anatomicopathological studies on the spinal cord in progressive locomotor ataxia and all his additional research enabled him to elucidate medullary physiology as no one before him in France had done. In addition, his research allowed him to establish, for the first time in neurological pathology, the precise anatomical location of a clinical problem while the patient was still alive.

Cerebral Localizations

Charcot revisited the anatomoclinical method, so fruitful for studying the spinal cord, to elucidate a contemporary issue from 1875 onward: “cerebral localisations” (Gasser, 1994). Whereas Pierre Flourens (1794–1867), in his criticism of phrenology in 1842 (Flourens, 1842), concluded that the brain functioned holistically, Jean-Baptiste Bouillaud (1796–1881), in keeping with Franz-Joseph Gall (1758–1828), localized language in the frontal lobes (Bouillaud, 1825), then Paul Broca (1824–1880) in the basal region of the third left frontal gyrus in 1861 (Broca, 1861). In 1870, Eduard Hitzig (1838–1907) and Gustav Fritsch (1837–1927) showed that localized cortical electric stimulation determined the movements on the contralateral side of the body (Fritsch & Hitzig, 1870). David Ferrier (1843–1928) then confirmed these data in 1874 (Ferrier, 1874). In 1875, Charcot undertook a series of 27 lessons on cerebral localizations:

The encephalon does not represent a homogeneous, unitary organ, but rather an association, or, if you prefer, a federation made up of a number of diverse organs. Each of these organs could be physiologically linked to distinct properties, functions, and faculties. Once the physiological properties of each of these parts is known, it should be possible to deduce the pathologic situation, since this would be only a modification, mild or marked, of the normal state, without the intervention of new law.

(J.-M. Charcot, 1876, p. 3)

Charcot set out to show the relevance of the anatomicopathological examination of lesions found in the cortex after paralysis or partial convulsions. In 1883, with his resident in 1876 Albert Pitres (1848–1928), Charcot updated the data that had been published in France and abroad since his first lectures at the end of the preceding decade; the resultant book would remain a reference for several years (Charcot & Pitres, 1883). Charcot localized in this work the cortical motor area: the paracentral lobule and the upper two thirds of the ascending convolutions for both contralateral limbs and the lower third of the ascending convolutions for the lower part of the face. He established that cortical lesions in the motor area and those in the internal capsule, and those alone, determined secondary spinal degeneration. In contrast, his conjectures delimiting the cortical areas of sensitivity were not all validated later on (Jeannerod, 1994).

On December 18, 1875, at the meeting of the French Society of Biology, Charcot demonstrated how he was able to argue against those who tried to contradict his propositions. As he put it, “I do not think that experimental physiology can be considered, in and of itself, capable of revealing the functions of the various departments of the nervous system.” To which Brown-Séguard replied, “I unfortunately disagree with M. Charcot regarding the role of experimental physiology. . . . Cerebral localisations, as they are currently conceived of, are false and others must be established.” For him, all lesions had repercussions at a remove, which could vary for the same primary lesional localization. Charcot replied in turn,

I cannot consider the observations put forth by M. Brown-Séguard as convincing. Pathological anatomy is so incomplete that it is impossible to base anything on such descriptions. All these observations can be contested from other points of view, and there would be no end to the lacunae were I to try to illustrate them all.

(Charcot & Brown-Séguard, 1875, p. 423)

Case of Aphasia

Aphasia is a typical pathology giving rise to research and theories about localization. Charcot’s first personal study presenting the case of a patient with hemiplegic aphasia was published in July 1863 (J.-M. Charcot, 1863). His patient had a lesion occupying part of the temporal lobe and the insula, with no apparent lesions in the third frontal gyrus at her first examination. Charcot concluded, “The seat of the central organ of articulated language, if such an organ does exist, remains to be determined.” In 1875, Charcot helped his 1867 resident, Raphaël Lépine (1840–1919), to write his “agrégation” thesis (to become an associate professor) on “localisation in cerebral disease.” In this compilation study summarizing the state of knowledge at the dawn of the last quarter of the 19th century, Lépine reiterated the doubts about the localization of language proposed by Broca, notably arguing that the insula had not been taken into account.

It was only 20 years later, in 1883, that Charcot took a real interest in aphasia and covered it in five Friday lessons, published in *Le Progrès médical* following their transcription by Charles Féré (1852–1907; Charcot & Féré, 1883). Pierre Marie revised and completed them in 1885 (Charcot & Marie, 1883).

Charcot presided over the jury for two theses on aphasia. The first was that of his Russian student Nadia Skwortzoff (1852–?), defended on April 5, 1881, and covering cases of aphasia with “blindness and verbal deafness,” which she compared to cases already reported by Carl Wernicke (1848–1905), Adolf Kussmaul (1822–1902), Otto von Kahler (1849–1893), and Arnold Pick (1851–1924).

The second thesis, defended on January 9, 1885, was that of Antoine–Désiré Bernard (1853–1888), Charcot’s resident in 1883 (Bernard, 1885). In his thesis, Bernard condemned the illusion of his times whereby “to say aphasia in the language of Trousseau was to say Broca’s gyrus, making Trousseau the first guilty party in the serious confusion that followed. Everybody was guilty after him, despite whatever effort Broca made to avoid conflict.” He himself presented studies identifying the existence of sensory aphasia based on the publications of Wernicke and Kussmaul. He “most disagreeably” refuted the objections to his work, made notably by Louis–Lucien Dreyfus–Brisac (1849–1903) (Dreyfus–Brisac, 1881), but lauded Charcot’s 1883 and 1884 lessons, which he reused extensively. He reproduced Charcot’s famous explanatory drawing, “the bell,” which appeared for the first time in a book by Gaetano Rummo (1853–1917; Rummo, 1884). Basing his deduction on the research of Henry Duret (1849–1921) about the distribution of arterial branches of the middle cerebral artery, Bernard was the first to hit upon an anatomoclinical relationship between the localization of cerebral infarct, the location of the arterial occlusion, and the type of aphasia (Walusinski & Courrivaud, 2014).

The literary style of his thesis is particularly refined and full of humor. For example, after noting that Charcot gave his first observations of patients with aphasia to Broca, he added ironically, “in order that Broca be credited for the discovery and held responsible for the error.” During his residency, Bernard reported on one of Charcot’s lessons describing the first case of loss of mental imaging, or visual agnosia, which, for the master of La Salpêtrière, meant that memory was not singular but rather comprised “partial memories” (Charcot & Bernard, 1883).

Faced with the complexity of the various types of aphasia and the insufficiency of the anatomoclinical method to explain them all, Charcot turned to psychology with the help of Théodule Ribot (1839–1916):

The function or functions that allow us to communicate with our fellow man must be associated with the most advanced operations of our central system. Properly speaking, although these functions are not fully part of intelligence itself, under normal and pathological states, they certainly have the most decisive influence on the exercise of intelligence.

(J.-M. Charcot, 1892, p. 154)

The creation of the French Society of Physiological Psychology under the auspices of Ribot and Charcot was probably the result (Mercier, 1897). Ribot’s presence in the painting of André Brouillet (1857–1914), *Une leçon clinique à La Salpêtrière*, bears witness to this collaboration (Walusinski, 2021).

Hystero-Epilepsy

Faced with the imminent collapse of the building that housed the Sainte-Laure ward, the hospital administration decided in 1869 to transfer the patients with epilepsy and hysteria treated by Louis Delasiauve (1804–1893) to Charcot's department. Bourneville, Charcot's resident in 1868, was responsible for treating these patients when he was a resident under Delasiauve. He initiated Charcot into examining these poor women, who experienced such a variety of crises. But also previously, in 1869, Charcot had attended the Congress of the British Medical Association in Leeds and listened to the presentation of John Russel Reynolds (1828–1896) entitled *Paralysis and Other Disorders of Motion and Sensation Dependent on an Idea*. He would often cite this article during his later lessons (Reynolds, 1869); Reynolds probably played a decisive role in his new commitment to studying hysteria. From 1870 to 1893, Bourneville, the resident–artist Paul Richer (1849–1933), Georges Gilles de la Tourette (1857–1904), and numerous other students represented by Brouillet, would follow in Charcot's footsteps to explore and describe hysteria, notably through hypnosis. The fruitless search for a cerebral lesion causing the disturbances gradually, over 20 some years, led Charcot to substitute a psychological etiological paradigm for a lesional one, which had been in place for a long time. This new model improved neurological clinical examination, which was the basis of neurological semiology (analysis of tendon and cutaneous reflexes, Babiński sign, etc.). This gives Charcot's study of hysteria legitimate recognition, denied for too long.

A meeting with Pierre Janet (1859–1947) in 1885 was the most notable event in Charcot's last productive period, leading to the creation of a "laboratory of psychology" in 1890 within the Clinic of Nervous System Diseases, the first milestone in the brilliant career of one of Charcot's last students. Janet was the first to assert a link between the subject's lived experience and "a traumatic event," which generated his understanding of hysteria and led him to this definition: "Hysteria is a set of diseases by representation." This foundational work cannot be summarized here, but it should be noted that Janet introduced the following concepts: "doubling of personality" and "shrinking of the field of consciousness," along with subconsciousness and dissociation. These ideas can be found in the conclusion to his thesis, with Charcot presiding over the jury, defended 2 weeks before the latter's death: "Hysteria is a form of mental disintegration characterised by the tendency toward permanent and complete doubling of personality"; also: "A banished idea, like a psychological parasite, causes all accidents of physical and mental diseases" (Janet, 1893). In 1886, Janet did not yet have the eminence in Charcot's orbit that he would acquire by 1890, which probably explains his absence in Brouillet's painting.

In Conclusion

This survey of Charcot's immense oeuvre cannot be exhaustive. The book by Christopher Goetz, Michel Bonduelle, and Toby Gelfand, *Charcot Constructing Neurology*, remains the indispensable reference for a more detailed, in–depth look at the information presented here (Goetz et al., 2005).

In his homage to Charcot on May 25, 1925, at the centennial of the master's birth, Babiński concluded his speech with terms that perfectly situate the oeuvre that originated at La Salpêtrière:

To cut neurology off from Charcot's acquisitions would make it unrecognisable. The truth is that in neurology departments, not a day goes by when we do not apply the notions he introduced; his way of thinking is always with us, always present.

(Babiński, 1925, p. 756)

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