



Little-known aspects of the work of Jean-Martin Charcot

(History of Neurology)

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Abstract

Jean-Martin Charcot, recognized as one of the founding fathers of neurology, began his hospital career as an internist and geriatrician responsible for some 2,500 elderly women at what was originally a nursing home and asylum, the Hospice de la Vieillesse Femmes at La Salpêtrière Hospital. For some twenty years, he thus had the opportunity to introduce pathologies identified in England and Germany to French physicians, such as Graves' disease and Addison's disease. In cardiology, he worked on the pathophysiology of intermittent claudication and pulmonary embolism. He then extended the concept of embolism to infectious pathology. Over many years, his clinical and metabolic research on gout led him to conduct multiple studies on rheumatology, notably chronic forms of rheumatoid arthritis. This summary is only a limited overview of the nearly 100 publications on various subjects covering all medical fields. Charcot's contributions may thus be considered as significant as those he made to neurology.

Keywords Jean-Martin Charcot, Geriatrics, Gout, Pulmonary embolism, Intermittent Claudication

Introduction

Jean-Martin Charcot (1825–1893) is unanimously recognized as one of the founding fathers of neurology (Figure 1). The numerous historical works devoted to him have analyzed in detail his decisive contributions to this emerging specialty. When he returned to the La Salpêtrière hospice on July 1862, to take over the department headed by Eugène Cazalis (1808–1883), whom he succeeded after having been an intern there ten years earlier, he took charge of 2,635 women, the majority of whom were elderly. This explains why his early publications were those of a geriatrician and internist (*Husson, 1862*). To illustrate this more precisely, I propose to review his numerous articles published from the time of his dissertation until 1867, the year in which his work began to focus exclusively on diseases of the nervous system (*Lellouch, 1992*).



Figure 1. A little-known photograph of Charcot from around 1870, from Françon F. Le centenaire de la thèse de Charcot (1853-1953). *Rhumatologie* 1954;6(1):27-31. (Collection OW).

Brief Biographical Note

Jean-Martin Charcot, born in Paris on November 29, 1825, was the eldest son of a saddler and coachbuilder. Of his four other brothers, Eugène, born thirteen months after him, died at the age of 15 days; Pierre would take over the family business; Émile and Jean-Eugène would become soldiers. After passing his baccalaureate on August 31, 1843, he began his medical studies. After passing the externship exam in December 1845, Charcot was appointed provisional intern in 1847, then admitted on December 18, 1848, to the internship program at the Paris Hospitals alongside Alfred Vulpian (1826–1887) and Alexandre Axenfeld (1825–1876). He served successively as an intern under Louis Béhier (1813–1876), Pierre Rayer (1793–1867), Pierre-Adolphe Piorry (1794–1879), and, in his fourth year, under Cazalis at La Salpêtrière: “there he gathered the material for his inaugural thesis, learned to appreciate the inexhaustible resources amassed in that hospital, and resolved from that point on to return there as a physician” (*Joffroy, 1893*). Piorry, a professor of clinical medicine who was already advocating the use of the microscope, offered him the position of chief resident in 1853 and 1854. But it was Rayer who would have a decisive influence on Charcot’s career. Working alongside him, Charcot absorbed the role of a powerful mentor attentive to his students. Before allowing him to take the agrégation exam, Rayer admitted him to the Société de Biologie, which he had helped found and was then presiding over. Charcot became a full member in 1851, “a young man, a colleague of eminent figures” such as Claude Bernard (1813–1878), Charles Brown-Séquard (1817–1894), François Magendie (1783–1855), and Émile Littré (1801–1881). These leading figures discussed advances in clinical science and laboratory research, strongly influenced by the positivist philosophy of Auguste Comte (1798–1857). By belonging to this society, Charcot was able to publish his own work at a very early stage. He remained loyal to it throughout his career, ensuring that he, in turn, introduced most of his residents to it (*Bonduelle et al., 1996*).

His friend Vulpian was appointed head of the second department. At a time when the nosography of chronic pathologies—particularly those affecting the nervous system—was still in its infancy, “we saw the two young professors, traversing together all the wards of this immense asylum, examining all the patients, gathering all the observations, and compiling a colossal dossier that was to be gradually supplemented by postmortem and histological studies, or by the invaluable contributions of laboratory research” (*Joffroy, 1893*). There, they put into practice the anatomo-clinical method that had established the Paris School’s reputation since the turn of the century, perfecting it through the systematic use of the microscope. Another innovation introduced by Charcot was the systematic measurement of the residents’ temperature using a mercury thermometer rather than simply placing a hand on their forehead. His resident in 1868, Désiré-Magloire Bourneville (1840–1909), would make this the subject of his thesis and subsequent publications (*Bourneville, 1870*) (*Bourneville, 1873*).

The thesis, a pioneering nosographic work

Charcot’s thesis was his first nosographic work. The defense took place on March 16, 1853, presided over by Piorry (*Charcot, 1853*). In this monumental work, he distinguished gout from chronic rheumatism (now recognized as rheumatoid arthritis on the one hand and osteoarthritis on the other) (Figure 2).



Figure 2. A drawing by Charcot illustrating his thesis. (BIUsanté, Paris Cité University)

The importance he places on references from English and German literature not only attests to his erudition and mastery of foreign languages but also represents an innovation for this type of personal work. His talents as a draftsman are evident in the plate of hands with deformed fingers illustrating his thesis. Charcot would continue to take an interest in gout throughout his career. For example, in 1860, he provided a detailed description of tophaceous deposits on the earlobes: “tophaceous concretions of the external ear appear to occupy an important place in the clinical history of gout” (*Charcot, 1861 c*). In 1867, he translated into French the treatise on gout by the Englishman Alfred Baring Garrod (1814–1917) (*Garrod, 1867*). Charcot would always keep abreast of advances in the understanding of rheumatic diseases and gout. Thus, in 1863, he reported an observation made by Garrod regarding the high frequency of gout patients who had previously exhibited signs of lead poisoning (*Charcot, 1863*). However, he had not previously noted this association, which was attributed to a toxic effect of lead on the kidneys preventing the normal elimination of uric acid. He encouraged physicians to investigate this association, which was likely not coincidental, in order to clarify the matter. In 1866, as he began teaching courses on diseases of the elderly, Charcot devoted several lectures to gout—its clinical presentation, pathological anatomy, and emerging biology—which were published in *La Gazette des Hôpitaux* in 1866 and 1867.

Early Years at the Society of Biology

As early as 1852, Charcot gave several presentations at the Société de Biologie addressing a variety of topics: valvular lesions in endocarditis (Figure 3), hydatid cysts of the pelvis, predisposing factors (eclampsia) for idiocy and imbecility, fetal smallpox, gout, pyelonephritis

due to pyeloureteral obstruction (ureteropelvic junction syndrome), biliary peritonitis, gastric ulcer followed by pyloric stenosis, and rupture of the spleen in a fetus.

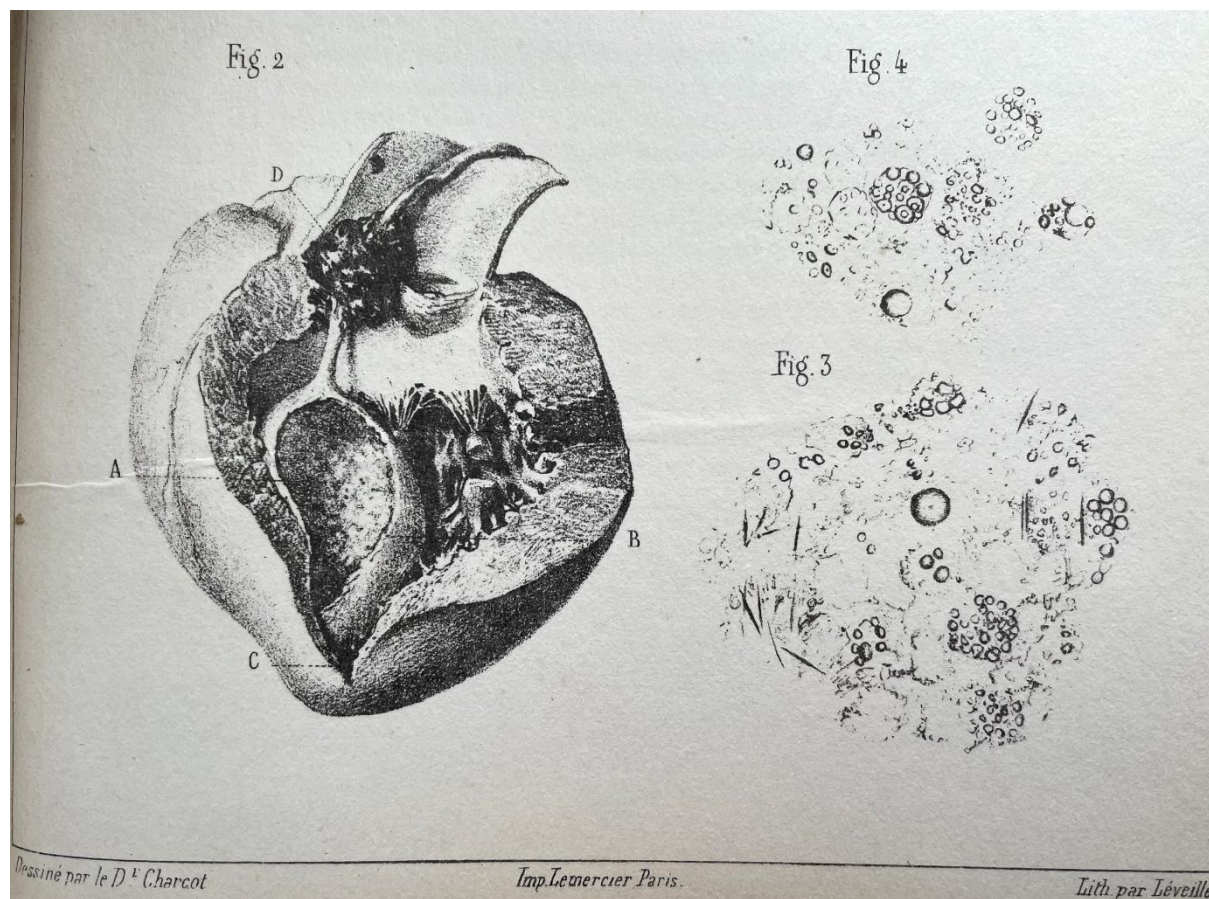


Figure 3. Engraving taken from: Charcot JM. Cas de tumeurs fibrineuses multiples contenant une matière puriforme situées dans l'oreillette droite du cœur. *Comptes Rendus des séances et Mémoires de la Société de Biologie* (année 1851). Paris: Au bureau de la Gazette médicale 1852;3:189-200. (Collection OW).

In May 1856, the year he was admitted to the Central Bureau (hospital physician), Charcot wrote the first French-language treatise devoted to Graves' disease, a condition previously unrecognized in France (*Charcot, 1856*). Inspired by the Irish physician Robert James Graves (1797–1853), the first to describe the clinical picture, Charcot presented French physicians with the cardinal signs—goiter, tachycardia, exophthalmos, and agitation—as he had observed them at La Charité Hospital in young women. He insisted that the heart should not be considered the source of the pathology but noted changes in the structure and dimensions of the thyroid arteries. He hypothesized increased gland activity due to stimulation of the vasomotor nerves, as, upon auscultating the goiter, he observed “a continuous murmur.” For him, the increase in the volume of the acini was the consequence. Death occurred in nearly half of the identified cases! In the same vein, in 1859, Charcot reported on his German readings. He recounted the observation made by August Hirsch (1817–1894) of Königsberg regarding a fatal subarachnoid hemorrhage marking the culmination of a case of Graves' disease, and those of the ophthalmologist Albrecht von Graefe (1828–1870) of Berlin, highlighting the risk of severe

corneal changes secondary to exophthalmos (*Charcot, 1859a*). In concluding this article, Charcot agreed with the proposal put forward by Hirsch to name this disease Basedow's disease (Karl Adolph von Basedow 1799–1854), an eponym still in use today (*Déchy, 2021*).

An interest that would remain unwavering throughout his career, Charcot took an interest in art and medicine as early as 1857, describing with Amédée Dechambre (1812–1886), the journal's editor-in-chief, "some ancient marbles pertaining to anatomical studies" (*Charcot and Dechambre, 1857*).

On September 18, 1857, Charcot published an article in the *Gazette hebdomadaire de Médecine* devoted to melanosis: "The alteration of the blood consists essentially in the presence within this fluid of a more or less considerable number of granules, corpuscles, or pigment cells" (*Charcot, 1857*). First described in Germany by Heinrich Meckel von Hemsbach (1821–1855) in 1847 (*Meckel, 1847*), this blood abnormality was linked to intermittent fevers, specifically malaria. For Charcot, these circulating colored corpuscles "belong to that group of substances that derive their origin from the dissolution, or the regressive metamorphosis undergone by red blood cells, under certain pathological circumstances." Charcot reports on the numerous German and Italian publications related to the discovery of melenemia. He attributes its origin first to the activity of the spleen, and then to that of the liver. While he recognizes renal melanemia associated with proteinuria as the cause of nephritis secondary to bouts of malaria, he rejects this melanemia as the cause of neuropsychiatric disorders (delusions, convulsions, etc.) occurring during malaria. Charcot wondered about the nosological classification of melanemia: was it a symptom or a disease? He was unaware of causes other than fevers.

Together with Vulpian, Charcot presented an observation of Addison's disease to the Société de Biologie in 1857. Macroscopically, the adrenal glands appear normal, but microscopic examination reveals degeneration: "Following the typical symptoms of Addison's disease, the adrenal capsules would be deemed healthy without having been examined under a microscope or treated with appropriate reagents. From a theoretical standpoint, this must be considered null and void." This text reveals the commitment to rigor that drove the two colleagues at La Salpêtrière (*Charcot, 1878*).

On February 26, 1858, Charcot reviewed a series of articles by German authors, notably Rudolf Virchow (1821–1902), to discuss the amaurosis affecting many patients with "albuminuric nephritis" and during the course of Bright's disease, that is, chronic renal failure, as he had observed while serving as Rayer's intern. Whereas previously, macroscopic pathological anatomy had revealed no lesions, the practice of examining the fundus while the patient was alive and microscopic examinations made it possible to identify retinal damage and central retinal artery thrombosis, in particular. The concept of hypertension was still unknown. Charcot thus became a champion of fundus examination, a new diagnostic technique invented by Hermann von Helmholtz (1821–1894) in 1851.

Together with Casimir Davaine (1812–1882), the biologist and bacteriologist who discovered the anthrax bacterium, Charcot presented to the Société de Biologie an observation of multiple hydatid cysts in the liver and pelvis, as he had already done in 1852 (*Charcot and Davaine, 1858*). The patient presented with a severely deformed abdomen due to an enormous mass on the right flank. "This tumor is fluctuant; no hydatid quivering is perceptible." They punctured the cyst, draining three liters, then administered several successive injections of caustic agents. Examination of the fluid revealed echinococci. A fistula developed, continuing to discharge parasites. After the patient's death, the autopsy noted widespread dissemination of the parasitic infection. It should be noted that the discovery of the parasite's life cycle by the Bavarian Karl

Theodor Ernest von Siebold (1804–1885) was still very recent, in 1853, but was evidently known to Charcot and Davaine (*von Siebold, 1853*).

François-Amilcar Aran (1817–1861) devoted his 1853 agrégation thesis to the causes of sudden death, among which he identified pulmonary embolism, though without specifying its pathophysiology. Benjamin Ball (1833–1893) and Charcot demonstrated in 1858 that the clot obstructing the pulmonary artery originated from venous phlebitis in a lower limb (*Charcot and Ball, 1858*). Charcot recognized elevated fibrin levels as a factor promoting clot formation, even though the physiology of hemostasis was still poorly understood. He noted the frequency of these incidents among young women and those who had recently given birth. This pioneering description, inspired by the work of Rudolf Virchow (1821–1902), demonstrates the vast medical knowledge of a 33-year-old Charcot who read English and German authors. Having been a hospital physician for two years, he was filling in for colleagues. It was while filling in for Louis-Alfred Becquerel (1814–1862) that he met Benjamin Ball, then an intern in that department at La Pitié. Ball would defend his thesis on the same subject before Armand Trousseau (1801–1867) four years later, on January 3, 1862. Charcot would recall this fruitful collaboration when he successfully nominated Ball for the Chair of Mental and Brain Diseases in April 1877.

The collaboration between Charcot and Ball continued with the 1860 publication of a work that now holds only historical value, titled: “On the Coincidence of Visceral Gangrene and External Gangrenous Conditions; Gangrenous Erysipelas and Gangrenous Dissecting Pneumonia” (*Ball and Charcot, 1860*). A fifty-seven-year-old woman with heart failure at the anasarca stage, due to mitral stenosis, underwent an ascites tap. An incomplete tap drained eight liters but was complicated by erysipelas at the puncture site, rapidly progressing to gangrenous sphacelus and death. At autopsy, the base of the right lung was occupied by a gangrenous sequestrum, an exceptional finding rarely described previously. The term “*gangrenous dissecting pneumonia*” was introduced by the German Hermann Lebert (1813–1878) in 1845 in the French edition of his treatise on pathological physiology (*Lebert, 1845*). Ball and Charcot mention “septic fluids originating from the primary focus, or even blood clots laden with gangrenous pus and formed initially in the veins passing through the focus,” which they suppose spread the gangrene; that is, they considered septic emboli before the era of microbiology! Were these cases of gas gangrene caused by *Clostridium perfringens*?

Following a presentation to the Society of Biology, Charcot was the first to publish, in 1859—and quite originally, in a veterinary medical journal, the *Recueil de médecine vétérinaire* (Figure 4), and then in *La Gazette médicale de Paris* (*Charcot, 1859d*)—the observation that allowed him to describe claudication of arterial origin: “This report seems to us to be closely related to cases of intermittent lameness due to arterial obliteration, now well known in equine pathology, and to which our colleagues Ms. Goubaux and H. Bouley—to whom we owe important work on this subject—have repeatedly drawn the Society’s attention” (*Charcot, 1859c*). Armand Goubaux (1820–1890), who would later become director of the National Veterinary School of Maisons-Alfort, described “paralysis in horses caused by occlusion of the posterior aorta” in 1846 (*Goubaux, 1846*). Henri Bouley (1814–1885), known as “the Great Bouley,” was the first to draw attention to this form of lameness in horses in 1830 (*Bouley, 1830*). After the clinical description of the exertional lameness experienced by his patient, which appeared nearly three decades after a war injury, Charcot discovered, during the autopsy, the presence of a bullet compressing the primary iliac artery, reproducing in a quasi-experimental manner what veterinarians had observed in horses. Charcot thus elucidated the pathophysiological mechanism of ischemia: “As we can see, physiology can provide us, to a certain extent, with

the key to the main phenomena observed during an episode of claudication.” Knowing that arterial narrowing causes transient or permanent ischemia, he could extend this explanation to the various arterial territories and attribute the narrowing of the arterial lumen to atheroma. Did he have the same thought in 1893 when he himself was suffering from angina attacks?

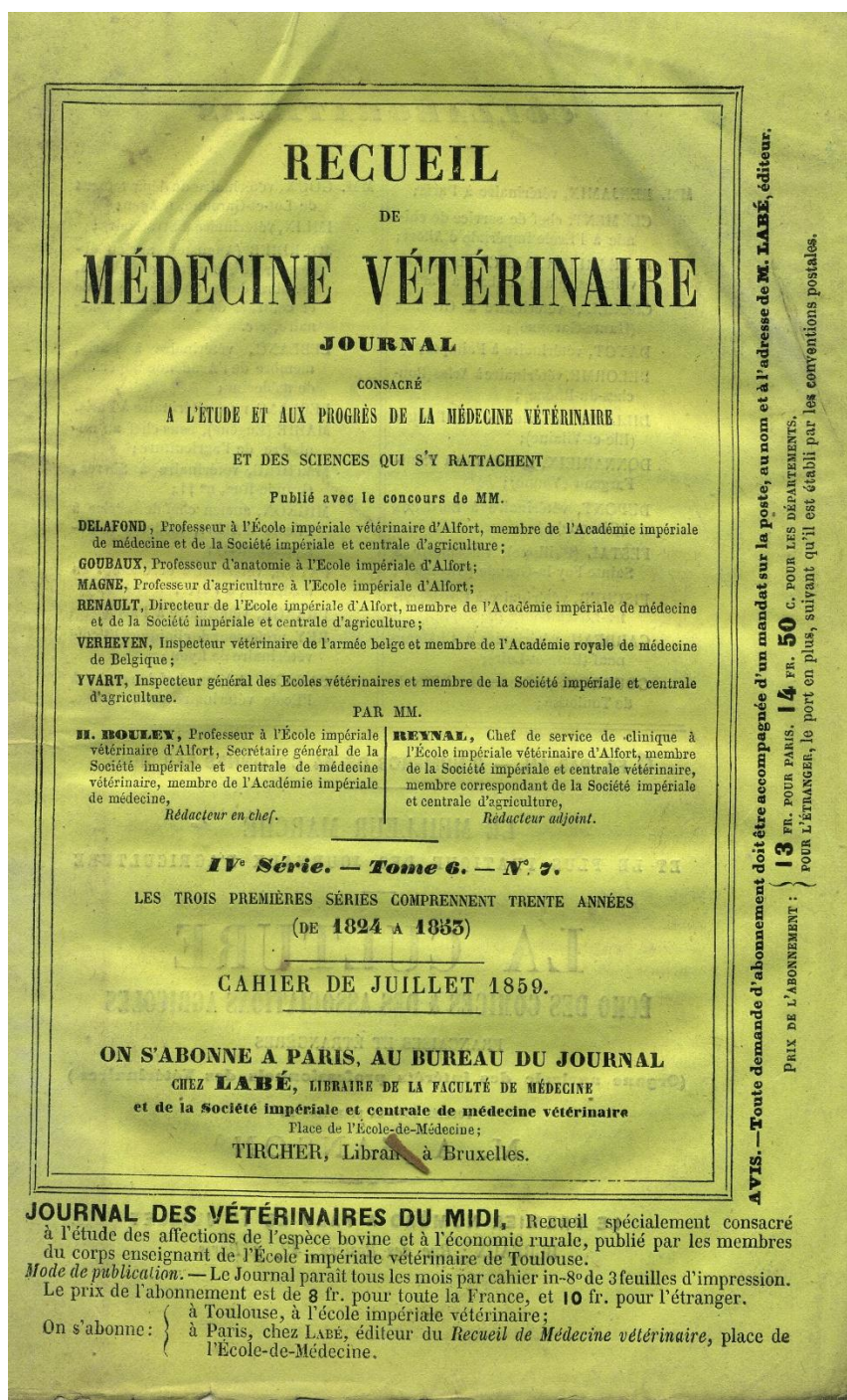


Figure 4. Front page of the journal Recueil de médecine vétérinaire July 1859. (Collection OW).

Charcot, after examining two chemists exposed to “experiments on the fusion and vitrification of certain substances by the action of an electric battery,” on February 14, 1858, described photokerato conjunctivitis, since then known as “arc flash” or snow blindness, accompanied by facial erythema, even though UV radiation would not be discovered until 1893. He does not fail to compare this burn to a sunburn, even though the experimenters had not perceived any excessive heat (*Charcot, 1859b*).

By the time he became vice president of the Society of Biology, 1860 marked his success in the agrégation exam on his second attempt. His thesis “On Chronic Pneumonia” is a compilation of studies, most of them (*Charcot, 1860*) describing a prolonged febrile course of acute lobar pneumonia, leading to a severe deterioration in general health and ultimately death. The autopsy revealed gangrenous changes in the lung, with large non-functional fibrous segments, sometimes cavitary, but not tuberculous. The etiology remained a mystery at that time, as bacteriology was still unknown.

Charcot-Leyden crystals

In 1853, Charcot and Charles Philippe Robin (1821–1885) presented a case of acute leukemia to the Société de Biologie, a condition then largely unknown in France but already described in Germany by Virchow (*Virchow, 1845*) and in England by John Hughes Bennett (1812–1875) (*Bennett, 1845*). The patient, who was pale and had significant splenomegaly, died after throwing himself down the hospital stairs. During the autopsy, blood was examined from the contents of the ventricles. A significant increase in white blood cells and a decrease in red blood cells were observed. Robin added: “In the blood of the right ventricle, mixed with the white blood cells, a large quantity of rhomboid crystals, very regular in shape, and slightly yellowish-red in color. These same crystals are extremely abundant in the spleen tissue, where they form considerable clusters, though visible only under a microscope” (*Charcot and Robin, 1854*). Charcot and Vulpian, in 1860, described these crystals in relation to another case of leukemia: “very elongated octahedrons, of very regular shape and fairly uniform,” which they measured at 6 to 8 μ (*Charcot and Vulpian, 1860*) (figure 5). Friedrich Albert von Zenker (1825–1898) is credited with first describing this type of crystal during his identification of trichinosis (*Zencker von, 1862*). The eponymous Charcot-Leyden crystals, still in use today, pay homage to the description by Charcot and Robin in 1853, adding the name of Ernst Victor von Leyden (1832–1910), who identified them in the sputum of asthmatics in 1872 (*Leyden von, 1872*). These colorless, double-pointed, and often needle-shaped crystals, observed in serous fluids such as bronchial secretions in asthma and in stool in certain cases of intestinal parasitism, consist of an enzyme, lysophospholipase, which is synthesized by eosinophils, blood cells that were not yet identified in Charcot’s time.

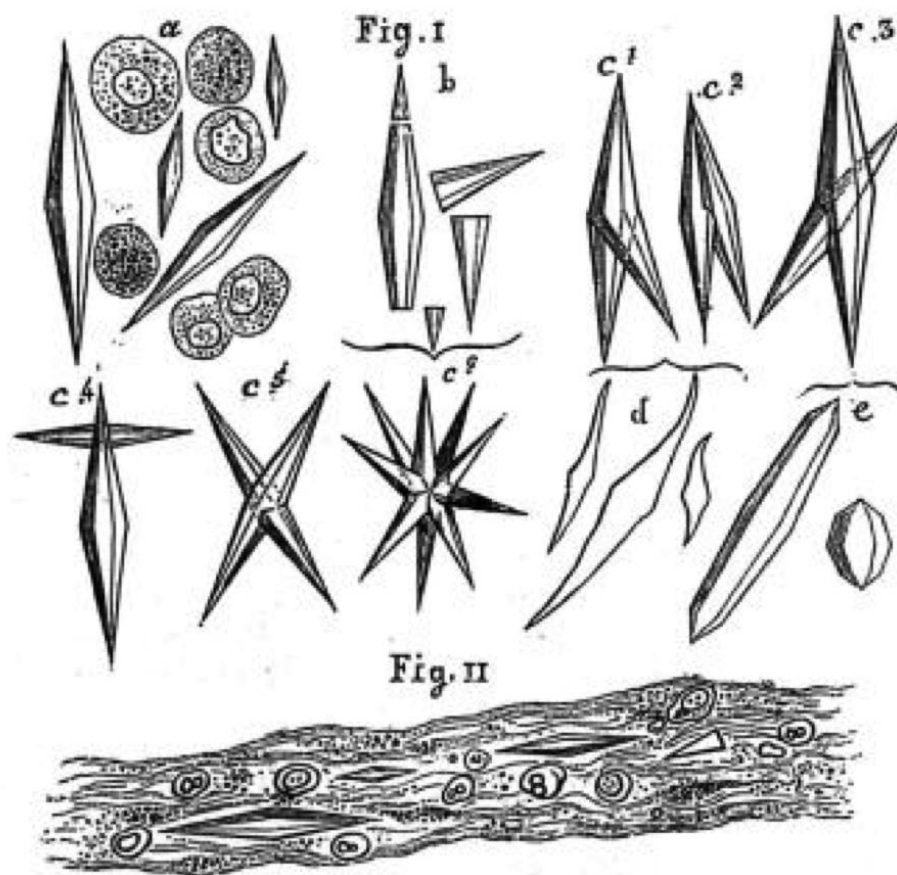


FIG. 4. — Cristaux octaédriques trouvés dans le sang d'un sujet leucémique.
a. Cristaux et globules blancs représentés à l'aide de la chambre claire, avec un grossissement de 450 diamètres.
b. Cristaux rompus : modes de rupture les plus ordinaires.
c¹, c², c³, c⁴, c⁵, c⁶. Cristaux groupés.
d. Cristaux modifiés par l'acide azotique.
e. Cristaux représentés, soit au moment où la chaleur commence à les dissoudre, soit au moment où ils se reforment après leur dissolution.

FIG. 2. — Concrétion fibrineuse expectorée par un sujet non leucémique, et dans laquelle on a trouvé des leucocytes et des cristaux analogues aux précédents.

Figure 5. Charcot-Leyden crystals drawing by Charcot from *Gazette hebdomadaire de médecine et de chirurgie* 1860;7:755-758. (Collection OW).

An insatiable curiosity in all fields

To illustrate Charcot's insatiable curiosity, consider his translation of an article from the English journal *London Medical Times* describing a case of sudden graying in 1861 (*Charcot, 1861a*). Seeking to refute an irrational explanation of a supernatural nature, he traces the history of this issue—a testament to his vast medical knowledge—by compiling clinical cases gathered from European literature. He correctly attributes this phenomenon to stress and distinguishes it from vitiligo, lamenting that “the physiological cause still eludes us.” How could he have conceived of an autoimmune phenomenon triggered by the activation of the hypothalamic-pituitary-

adrenal axis, leading to the disappearance of melanocytes that darken hair follicles and precipitate hair loss, while hair that is already white—lacking melanocytes—remains alone and gives the hair a gray-white appearance (Sikkink et al., 2020).

In 1861, Charcot turned his attention once again to gangrene, and more specifically to the form that complicates diabetes (*Charcot, 1861b*). In his review of the literature, he pays tribute to the original description by Charles-Jacob Marchal de Calvi (*Walusinski, 2023*) in 1852 (*Marchal, 1852*) and 1856 (*Marchal, 1856*). Charcot divides his study into three categories, based on cases he observed and others reported in the literature: diffuse phlegmons, so-called senile gangrene, boils, and anthrax. The concept of microbial infection is obviously still unknown to him. He does not specify the state of arterial circulation or the presence of sensory disturbances, unlike Marchal. His lecture on Tuesday, December 13, 1887, devoted to intermittent claudication in diabetics, attests to the progress made in understanding the pathophysiology of these diabetic complications between 1861 and 1887 (*Charcot, 1887*).

Starting in 1862, the year he became head of the department at La Salpêtrière, neurological topics became more frequent, while those from other medical fields became less common.

In response to a column by Sigismond Jaccoud (1830–1913) reporting on a presentation made to the Royal Society of Medicine and Surgery in London regarding the expected benefits of treatment with potassium bicarbonate to alleviate an acute attack of rheumatoid arthritis (*Jaccoud, 1862*), Charcot discusses his own therapeutic trials with high-dose sodium bicarbonate, conducted at Lariboisière Hospital while he was replacing Claude-Hermann Pidoux (1808–1882)—trials carried out in collaboration with Vulpian at the Hôtel-Dieu (*Charcot, 1862*). They used sodium bicarbonate—known for its anti-inflammatory properties—with real benefits for the patients. Thus, Charcot demonstrated his continued interest in the study of rheumatism, building on his memorable doctoral thesis.

Also in 1862, Charcot recounts the story of a young woman he examined alongside Armand Trousseau (1801–1867). Suffering from a severe form of Graves' disease, and with none of the modest treatments offered having curbed her hyperthyroidism, the onset of pregnancy led to the disappearance of all the alarming symptoms she had been experiencing. This return to near-normalcy persisted four months after childbirth. For Charcot, and supported by other similar cases, this outcome was no mere coincidence. Pregnancy can reduce goiter, exophthalmos, tachycardia, and weight loss, and should be recommended whenever possible! This empirical clinical observation had no pathophysiological explanation at the time. The child appears to have shown no pathological symptoms at birth. Thyroxine (T4) was isolated in 1910 by Edward Calvin Kendall (1886–1972).

Based solely on clinical findings, without yet knowing bacteriology, it was very difficult to distinguish typhoid fever from septicemia or endocarditis. For example, in 1862, Charcot and Vulpian reported a case of ulcerative endocarditis of the tricuspid valve (*Charcot and Vulpian, 1862*). They note the higher frequency of left-sided valve involvement compared to right-sided and detail the tissue destruction caused by the infection, which severs the chordae tendineae and the free edges of the valves. They also describe the varied and multiple localisations of infectious emboli originating in the heart, referred to as a “special morbid poison.” The febrile picture described is of the magnitude of a gravely serious historical case, the likes of which no longer exist.

It is frequently claimed that Charcot had no interest in therapeutics and paid little heed to the suffering of the poor patients who respectfully came to beg his opinion on the origin of their

suffering. Numerous facts contradict these disparaging assertions. His 1864 publication in the *Bulletin général de thérapeutique* provides evidence of this in a medical field unexpected given the image we have of Charcot: sexuality. His article describes the dangers of treatments with Fowler's solution—that is, the use of arsenic, particularly for extensive psoriasis. His introduction reflects his ambivalence regarding the risk of arsenic poisoning: “The unfounded fears that once caused undue apprehension regarding the prolonged use of arsenic preparations in the treatment of certain chronic conditions have now largely dissipated in light of rigorously observed facts, and it even seems that they have given way, in some minds, to an optimism that is undoubtedly not without its dangers” (*Charcot, 1864*). Charcot then recalls the risk of tremors and peripheral neuropathies (*Hall, 2002*), which can lead to paralysis, but adds—a fact that seems to have been overlooked at the time—the onset of impotence, which he euphemistically refers to as anaphrodisia or “inertia of the genital organs.” He does not recommend abandoning this remedy but rather modifying the way it is prescribed, that is, starting with slowly increasing doses and reducing them once the therapeutic goal is achieved.

On April 13, 1865, Charcot informed his colleagues of the nature and progression of an epidemic that had begun in Saint Petersburg (*Charcot, 1865b*). Having ruled out the plague and typhus, Charcot, citing articles published in Germany, settled on the diagnosis of “relapsing fever.” He copied the article published in *Centralblatt für die medicinischen Wissenschaften* by editor-in-chief L. Herrmann on March 25, 1865. Preceded by flu-like symptoms, the acute phase involved fever, prostration, jaundice with hepatosplenomegaly for about ten days, with all symptoms gradually improving over the course of about ten days. Some patients progress to a coma with fatal, uremic, and icteric collapse, while others relapse two or three times over the course of one to two months and then recover. The disease is contagious and was transmitted to many caregivers. Charcot notes, on this occasion, that he had devoted a paragraph to relapsing fever as described by English authors in the medical treatise he completed after the death of its original author, Achilles Requin (1803–1854) (*Requin and Charcot, 1863*). Charcot advises “to gather all documents relating to this singular and formidable condition, so that we are not caught off guard if, by some impossibility, it should reach us in the more or less distant future.”

In 1865, Charcot delivered a presentation to the Medical Society of the Hospitals: “My former mentor and predecessor at the La Salpêtrière Hospital, Dr. Cazalis, used to point out to his students that, in patients who succumb to the disease (cancer), secondary deposits—most often multiple—are very commonly found within the vertebral bodies” (*Charcot, 1865a*). Charcot then refers to what he considers the first case of “painful paraplegia,” that is, vertebral collapse with spinal cord compression. He describes in detail the terrible pain in the spine and limbs, which gradually or suddenly become paralyzed. This was the case for six out of thirty-five women with breast cancer in his department over the past three years. Next, Charcot discusses the cases of four women with uterine cancer who developed arterial thrombosis—in the femoral, humeral, or sylvian arteries—without any atheromatous changes in the arterial walls or evidence that the healthy heart had released emboli. He noted that such thromboses can progress to gangrene in cases of gastric or breast cancer.

In 1866, Charcot presented a study “on rectal temperature in Asian cholera,” conducted during the recent epidemic that had affected Paris. When “the symptoms of coldness and choleraic cyanosis were most pronounced,” temperatures ranged from 37.8°C to 38.5°C. (*Charcot, 1866b*). In a few cases, the temperature exceeded 40°C a few hours before death. He seems to regret that the epidemic ended before he could obtain more data and refine his analysis. While this may seem primitive and of little interest to us today, we must remember that it was innovative and original in 1866.

Conclusion

In 1866, Charcot began his open lectures—that is, lessons given within his department and unrelated to the university curriculum—devoted to diseases of the elderly and recorded by Benjamin Ball. While these lectures were compiled into a book in 1867, the opening of the article published in the *Gazette des Hôpitaux* includes a description of the hospice residents that does not appear in the book: “The large population in our wards (2,500 patients) allows us to examine, from the most diverse perspectives, the main types of a single disease category; but what is even more important, we are able here to follow patients over long periods of their lives, rather than witnessing a mere episode in their history; thus we see the pathological process unfold to its very limits, whereas generally we are familiar only with its initial phase” (Charcot, 1866a). This was the first specialized course in geriatrics in Paris. The only work Charcot recommends to his students dates from 1725, a work by the Englishman John Floyer (1649–1734) (Floyer, 1725). His first lecture is devoted to fever, particularly in cases of “biliary gravel,” a common and fatal condition, pneumonia, etc. All these pathologies are described without reference to microbiology, which had not yet emerged. Many of his lectures are devoted to rheumatism and gout. This book is somewhat like the culmination of Charcot’s geriatric work. After 1868, with the founding of the Archives de physiologie normale et pathologique alongside Vulpian and Charles-Edward Brown, Charcot published exclusively on his research into the nervous system and its disorders.

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