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## History of Neurology

# Hippolyte Bourdon (1814–1892), one of neurology's forgotten forerunners<sup>☆</sup>



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The renown achieved by Charcot tended to overshadow many of his French contemporaries, who worked as he did to lay the groundwork of neurology through their clinical and anatomical research. We will focus on one of them, Hippolyte Bourdon (1814–1892), and his innovative contributions, though forgotten over time, which are available to us in his varied and numerous publications.

### 1. Brief biography

Born in Pont à Mousson (northeastern France) on 25 May 1814, Alexis Hippolyte Bourdon was the son of François Bourdon (1772–?), a merchant, and Anne Willaume (1774–?) [1]. He moved to Paris to study medicine, where he became a non-resident student in 1837, before ranking fifth on the competitive residency exam in December 1838, right after François-Amilcar Aran (1817–1861) and Pierre-Louis Gratiolet (1815–1865). His residency was prolonged in 1842, allowing him to defend his thesis on 14 July 1843, before a jury presided by Philibert Roux (1780–1854): *Des paralysies consécutives à l'asphyxie par la vapeur de charbon* (paralysis due to coal vapor

asphyxiation) [2]. He studied anatomy under Philippe Blandin (1798–1849), Constant Duméril (1774–1860), François Magendie (1783–1855), Joseph Récamier (1774–1852), and Roux. Bourdon was senior resident (*chef de clinique*) in 1845 and began to work for the Central Office as a hospital physician in 1849 (*médecin des hôpitaux*). He was successively assigned to the Saint-Antoine, Beaujon and Lariboisière hospitals and to the municipal asylum; he received his private clients at 68, *rue de Seine* (torn down for the construction of Blvd. Saint-Germain) and later at 32, *rue du Bac* (6<sup>e</sup> arrondissement). With the support of his friend Claude Bernard (1813–1878) who introduced him to Charles Robin (1821–1885) and Casimir Davaine (1812–1882), Bourdon was elected to the French Academy of Medicine on 18 June 1872, after having joined the French Society of Anatomy on 15 April 1840, eventually becoming the archivist and later the vice-chairperson. He also went on to become the chairperson of the Medical Society of Hospitals in 1883 [3].

On 12 December 1844, Bourdon married Claire Marie Félicité Raphaëlle Adelon (1822–1898), the daughter of Nicolas Philibert Adelon (1782–1862), a professor of legal medicine, known for having edited the *Dictionnaire encyclopédique des*

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Sciences médicales (encyclopaedic dictionary of medical sciences). Adelon had himself married Adèle Sabatier (1799–1869), the daughter of Raphaël Bienvenu Sabatier (1732–1811), who was a surgeon at the *Hôtel des Invalides* and a professor of anatomy. This type of endogamy was frequent among physicians in the 19th century. Bourdon's brother-in-law, Camille Doucet (1812–1895), was a poet, playwright, and member of the *Académie française* [3]. Bourdon's son, Emmanuel Bourdon (1846–1879), son, grandson, great-grandson of physicians who were professors at the Faculty of Medicine, died at the age of 33 of "exhaustion" as it was often explained at that time, shortly after having been admitted as a hospital

surgeon. He was probably depressed after losing his wife and his son, some time before [4].

## 2. Paralysis following asphyxia induced by coal steam

Bourdon was the first to take an interest in this subject. His thesis examined the neurological consequences of the "inhalation of carbon steam" (Fig. 1) [2]. The physical-chemical phenomena of combustion and respiration had been known since the work of Antoine Lavoisier (1743–1794),

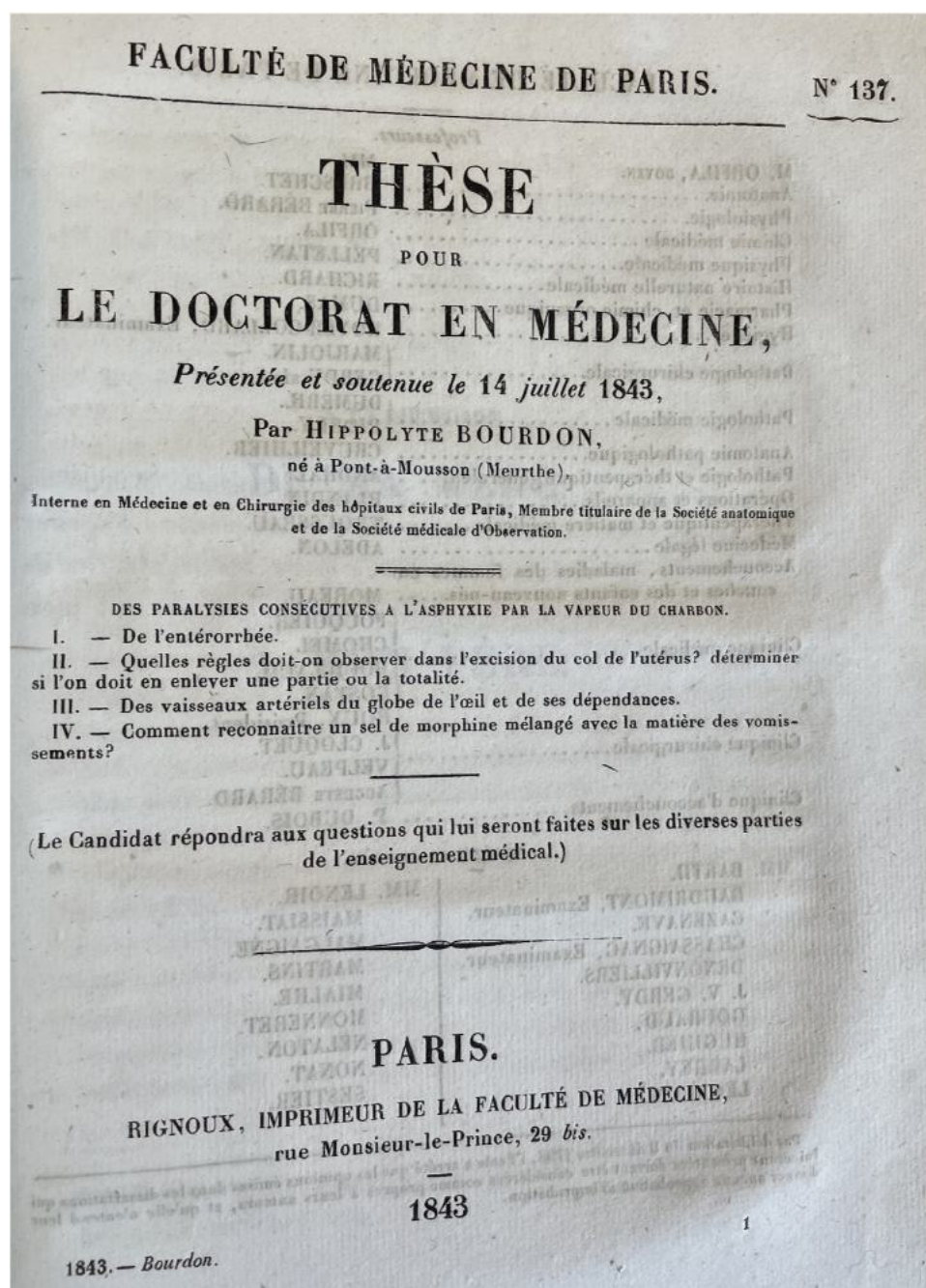


Fig. 1 – Cover of Hippolyte Bourdon's thesis (©BIU santé, Paris).

presented to the Royal Academy of Sciences on 5 September 1777 [5]. Strangely, his discovery of the biological role of oxygen and respiratory function did not cause physicians to immediately change the common view of asphyxia, considered to be merely the “sudden privation of the pulse” and always equated with apoplexy. In his *Avis au peuple sur les asphyxies* (notice to the general public on asphyxia) in 1774 [6], Jacques Joseph de Gardanne (1726–1786) was the first to realize this phenomenon was due to insufficient air renewal in the chest. For Louis Desbois de Rochefort (1750–1786) in 1789, asphyxia is formally “poor respiration accompanied by the simultaneous suspension of all functions of life” [7]. He articulated the cause as follows: “Carbon oxide gas and carbon hydrogen gas are released when carbon combustion begins. The action of these gases on the physical economy, very prompt when they are pure, is rarely sudden when they are mixed with atmospheric air”. Xavier Bichat (1771–1802) confirmed these data in 1800 during his research into life and death [8].

The general effects of inhaling smoke were clearly identified at that time: headache, dizziness, nausea, and muscular weakness followed by loss of consciousness and coma, which could lead to death. In contrast, the long-term secondary effects of asphyxia, notably on the nervous system, had not yet been described. In authoring his inaugural work on “paralysis”, Bourdon was thus a pioneer.

He put forward thirteen clinical observations to which he added his therapeutic advice, also providing the results of pathophysiological experiments conducted on animals. All the victims were young men who slept in a closed room heated by embers in a furnace, without any specific system for evacuating smoke to the outside. Headache and vomiting were followed by significant weakness and coma. When help came for the victims in the morning, they were probably taken out of the smoke-filled room, but this is not indicated. The coma lasted several hours or days, after which the patients often had aphasia or amnesia and presented various forms of paralysis in the limbs, in isolation or as hemiplegia. Recovery was slow and incomplete. In two cases, Bourdon autopsied the deceased victims. He found “black blood” in the vessels of the brain and hemorrhagic dotting in the nervous tissue which he attributed to loss of blood clotting, the blood being poisoned by “carbonated effluvia”. However, he did not mention the lack of oxygen. He referred to brain softening, a term introduced in 1820 by Léon Rostan (1792–1866) to distinguish this condition from cerebral hemorrhage [9]: “In asphyxiated patients, the brain is strongly congested; the blood is black”. Furthermore, Rostan observed the following during his vivisections: “In animals killed by coal gas, the cranial and spinal sinuses are swollen with fluid, dark blood. I not only observed the dotted appearance of the white matter in the brain, but also a red, uniform color of the grey matter in the spinal cord and brain”. Drawing on the observations of François Magendie (1783–1855), he believed poor coagulation of blood was the cause of its extravasation within the nervous tissue [10]. Bourdon attributed paralysis that disappeared secondarily to “congestion”, but for paralysis that definitively persisted, his opinion was that “it is necessary to consider more or less profound damage to the brain or spinal cord”. He pertinently observed that the regressive development of the paralysis was linked to

the duration of toxic exposure. However, his attempts to explain the location of paralysis are unclear and of little interest.

For treatment, he recommended “communicating to the thoracic walls movements to simulate those of normal respiration”, followed by abundant, repeated bleeding, the use of leaches, and “cold affusions” poured on the head. At no point did Bourdon suggest taking the victim out of the area where they had been poisoned. Secondarily, and later on, paralytic sequela were treated with electricity. While Bourdon indicated amnesia when patients came out of their coma, he did not mention long-term amnesic sequelae for any of the reported cases, undoubtedly because the necessary follow-up was not conducted.

Curiously, there was an absence of contributive advances between 1843 and 1925 in the field of delayed posthypoxic encephalopathy, up until the description of posthypoxic myelinopathy by Roy R. Grinker (1900–1993) in 1925 [11]. During the recent pandemic, magnetic resonance imaging of the brain revealed, in some cases, confluent diffuse supratentorial white matter T2 hyperintensity with restricted diffusion, during the sub-acute course of COVID-19, comparable to delayed posthypoxic leukoencephalopathy as previously reported after carbon monoxide poisoning [12].

### 3. Progressive locomotor ataxia

As a preamble, it must be recalled that in 1821, Charles Bell (1774–1842) presented experimental findings on the motor function of the anterior roots of the spinal cord [13]. But it was François Magendie (1783–1855) who added the other half of the picture, demonstrating the sensory function of the posterior roots [14]. Moritz Heinrich Romberg (1795–1873) published his *Lehrbuch der Nervenkrankheiten des Menschen* in sections between 1840 and 1846. The description of the eponym attached to his name is based on his account of sensory ataxia in *tabes dorsalis*, but it must be clearly stated that Romberg did not recognize the cause to be found in the posterior columns of the spinal cord [15]. In 1852, Octave Landry (1826–1865) proposed a new take on the physiology of sensation which laid the ground for the concepts of proprioception and stereognosis, explaining falls when the eyes are closed in *tabes* [16]. Shortly thereafter, Guillaume Duchenne de Boulogne (1806–1875) published a first description of “motor aptitude independent from vision, called by the author muscular conscience” in 1853, and his description of *ataxie locomotrice progressive* (progressive locomotor ataxia) in 1857, which could be seen in *tabes* [17]. He admitted that “the facts this work is based on are all taken from my private practice and my clinical activities; in these conditions, autopsy is difficult if not impossible”. Only one of Duchenne’s patients was autopsied in the department of Auguste Nonat (1804–1887) at La Charité Hospital. No lesions were observed macroscopically, neither in the brain nor in the spinal cord. Duchenne concluded his article by mentioning the observations published by von Romberg (1795–1873) in which he noted, “At the lower part of the spinal cord, where its volume had decreased by more than a third, the spinal substance of the posterior roots (sensory roots) had disappeared almost

completely, giving them a yellow and greyish tint” [18]. Duchenne asserted that Romberg had confused progressive locomotor ataxia with what he called “spinal paralysis” [18]. We should note that before Romberg, Wilhelm von Horn (1803–1871) had been the first to macroscopically describe, in his 1827 thesis, the medullary lesions qualified as *tabes* – i.e., softening and rotting of the spinal cord [19].

At the 28 August 1861 session of the Medical Society of Hospitals, of which Bourdon was the vice-chairperson, he presented the first part of his “clinical and histological studies on locomotor ataxia” [20], continuing the presentation on 22 January 1862 [21]. Bourdon, after reading Duchenne and listening to one of the lessons of Armand Trousseau (1801–1867) [22], observed four cases of progressive locomotor ataxia, in the patients he was treating at the municipal asylum, that matched their descriptions. The death of one of them from an intercurrent condition made it possible for him to publish the autopsy results, including, for the first time, a microscopic examination, the work of Jules Luys (1828–1897) (Fig. 2) [21]. A 28-year-old man presented with “uncertainty in the movement of his legs”, even with the help of visual control; “he [was] not in control of his movements”. Bourdon noted paralysis in the third left pair and recent myopia: “There seems to be poor accommodation [...] considerable dilation of the pupils”. That said, there was not the pain qualified as sharp and searing by Duchenne and developed in greater detail by Charcot in 1866 [23]. Digestive disturbances appeared, causing anorexia and diarrhea with sphincteric incontinence. Sensitivity in the legs decreased, especially in the soles of the feet. Death occurred with intelligence remaining normal to the end. Macroscopic examination of the brain and cerebellum showed no damage.

Examination of the spinal cord revealed degeneration of the posterior tracts, reaching its peak in the lumbar region: “The special coloration of the posterior tracts was due to the transformation of their constituent nervous tubes. Most of these tubes were no longer anatomical elements per se; the only remaining trace of them was the empty sheath, the walls of which were adjacent to each other”. The posterior roots could not be examined. Bourdon thought he would find lesions in the cerebellum based on the work of Pierre Flourens (1794–1867) [24] and Jean-Baptiste Bouillaud (1796–1881) [25]. “We are familiar with the experiments of our excellent friend M. Claude Bernard, experiments that showed the close relationship between sensitivity and voluntary movements”. Claude Bernard had shown that “when the posterior roots that enervate a limb are cut, movement in this limb henceforth appears to be involuntary and uncoordinated” [26]. Bourdon concluded that the integrity of the anterior roots of the spinal cord explained the preservation of “the contractility of the muscles”, but “for the phenomena of locomotion to be exercised regularly and well-coordinated overall, the normal function of all parts of the spinal cord that transmit sensory impressions is necessary, and for excito-motor parts as well”. With Luys’s help, Bourdon confirmed the posterior tracts’ role in the sensory transmission necessary for coordinating movements, i.e., the transmission of the “feeling of muscular activity”, or proprioception, as described by Octave Landry (1826–1865) in 1855 [27]. Charcot and Vulpian did not fail to cite Bourdon in their famous publication on locomotor ataxia in 1862 [28].

Finally, it should be noted that without specifically naming them, Bourdon described digestive disturbances that would become “gastric attacks in locomotor ataxia” or “tabetic

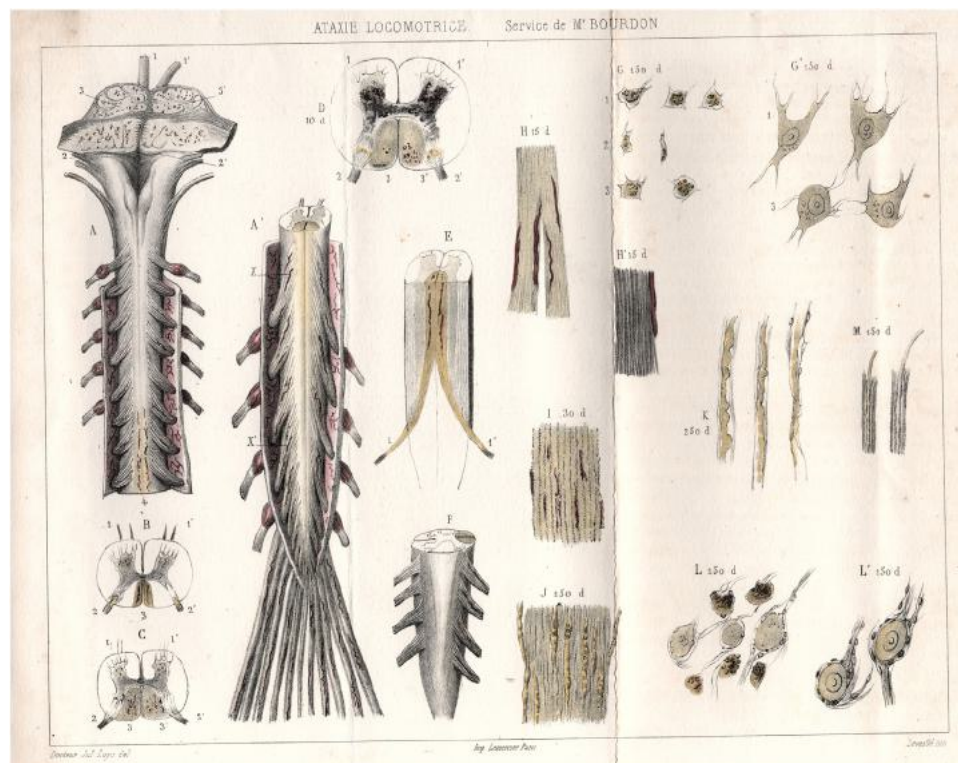


Fig. 2 – Illustration of the hand by Luys for Bourdon’s article on progressive locomotor ataxia (OW Collection).

gastric attacks". Georges Delamarre made this the subject of his thesis in 1866, the first on this topic [29], without mentioning Bourdon. Charcot did not give a lesson on this topic until 1872: "Gastric attacks coinciding or alternating with searing pain in the limbs" [30].

#### 4. Diseases of the medulla oblongata

On 26 March 1872, Bourdon presented a dissertation to the French Academy of Medicine. At the time, he was applying for a vacancy in the pathological anatomy section [31]. Bourdon wanted to fill in the holes in current knowledge that connected clinically discernible deficiencies to lesions in various levels of the brainstem, and mainly the medulla oblongata, given that he had been able to "bring together several unprecedented clinical facts" [32].

If lesions affecting the anterior part of the medulla oblongata were traumatic (vertebral dislocations) or hemorrhagic, death by respiratory arrest was instantaneous. If the lesion was more localized, he observed crossed hemiplegia. Sometimes there were continuous or action tremor and epileptic seizures. Motricity of the face, tongue and soft palate could be affected to varying degrees. Bourdon differentiated between the most anterior parts of the medulla oblongata, in which lesions did not affect motricity, and the more posterior or deeper areas that generated paralysis. He made an attempt at classification, notably for various alternating syndromes, after the publications of Auguste Millard and Adolphe Gubler in 1856 [33,34], Achille-Louis Foville (1799–1858) in 1859 [35], and Hermann Weber (1823–1918) in 1863 [36].

Although the lesions were more central and closed to the floor of the fourth ventricle, "the anatomists have discovered in these regions little masses of cells forming the originating nuclei of the hypoglossal, superior spinal, facial and pneumogastric nerves"; this resulted in deficits in these nerves, such as poor swallowing and phonation. Bourdon credited Charcot with this anatomical-functional research in 1870 [37].

Finally, damage to the posterior parts of the medulla oblongata and disturbances of sensitivity and coordination of movement became apparent. In locomotor ataxia, Bourdon observed hoarse, suffocating coughing fits, which he associated with damage to the medulla oblongata, "in the restiform body", which he considered to proceed from the degeneration in the posterior tracts. He noted that he had based this interpretation on the publications of Félix Féréol (1825–1891) in 1868 [38].

In this field, Bourdon's contribution was mainly to summarize and classify clinical disorders related to each level of the brainstem, which is indicative, for our time, of knowledge that was still very fragmented concerning the physiological functions of this anatomical region.

#### 5. Motor centers of the limbs

On 23 October 1877, Bourdon made a presentation at the French National Academy of Medicine in relation to a presentation by the surgeon Léon-Athanase Gosselin (1815–1887) that focused on choosing a trepanning site based on

clinical symptoms. Bourdon set out to establish the localization of cortical motor centers [39]. Charcot and his intern Pitres had just published their first works on the motor localizations in the first issue of a new journal founded by Charcot, *La Revue mensuelle de Médecine et de Chirurgie* (monthly journal of medicine and surgery) [40].

Bourdon brought together two personal observations of brachial monoplegia, in isolation or accompanied by transient aphasia, together with the anatomical examination, to which he added twelve comparable clinical cases found in the international literature. The etiology of these forms of localized paralysis was varied: ischemic, hemorrhagic, tumoral (tuberculomas), or traumatic with abscesses, setting in progressively or suddenly. Using these data, he deduced the motor localization for the arm: "Our lesions take up the entire height of the *circonvolution frontale ascendante* [ascending frontal convolution i.e. primary motor cortex], several points of the *circonvolution pariétale ascendante* [ascending parietal convolution i.e. somatosensory system], and the portions adjacent to these two gyri, as well as the paracentral lobule". Then he compared his conclusions with those published by

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SÉANCE DU 23 OCTOBRE.

FIG. 1. — Lésions anatomiques dans 16 cas de paralysie et d'amputation du bras (1).

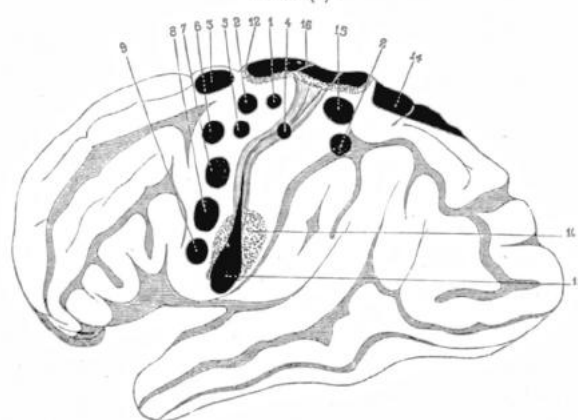


FIG. 2. — Localisations corticales par MM. Charcot et Pitres.



(1) Le numéro de chaque lésion correspond aux numéros des observations

Fig. 3 – Illustration by Bourdon of the motor areas based on his autopsies (©BIU santé, Paris).

Charcot and Pitres but also those of Camille-Henri Carville (1828–1885) and Henri Duret (1849–1921) [41], and those in the thesis of Jules Gromier (1849–19?) concerning localizations in monkeys [42]. For Bourdon, “the damaged zone in pathological cases is much more extensive than in zones that seem to correspond to movements in the arm, according to M. Carville and M. Duret and to M. Charcot and M. Pitres, but the damage in these zones is so grouped together, and they are so close to each other, that we initially believed we had found the actual motor center of the upper limb in man”. In fact, he recognized that many of the collated cases involved hemiplegia and not pure monoplegia, and he considered Charcot and Pitres’s conclusions to be accurate: “In the vast majority of hemiplegia cases, the anatomical damage occupied points in the zone that M. Charcot and M. Pitres had mapped out for hemiplegia, i.e., the upper third of the *circonvolution frontale ascendante* and the upper two thirds of the *circonvolution pariétale ascendante*” (Fig. 3). We can see that in many of his observations, there was damage to white matter associated with grey matter, or even beyond, toward the basal ganglia. To complete his research, Bourdon also studied the brains of deceased adults who had undergone the amputation of a limb in early childhood, leading to partial cortical atrophy. He concluded: “All these clinical facts prove that cortical damage in paralysis and arm amputation, as in hemiplegia, occupies a large zone neighboring the fissure of Rolando in all directions”, whereas Charcot and Pitres had noted, using a microscope, the presence of “giant cells that [were] motor cells”, called Betz cells as described by the Ukrainian Vladimir Alexeïevitch Betz (1834–1894) as recently as 1874 [43].

Bourdon then focused on the motor control of the lower limbs but did not have any cases of isolated paralysis. He studied an observation used in the thesis of Alfred Becquerel (1814–1862) in 1840, involving a child who died of tuberculous peritonitis and was paralyzed in one leg; his autopsy revealed a tuberculous brain lesion [44]. Drawing on the autopsies of amputated adults reported by Luys [45], and citing the experiments conducted by Eduard Hitzig (1838–1907) but without referring to Gustav Fritsch (1837–1927) [46], Bourdon agreed with their conclusions: “All the cortical centers seem to be exclusively situated in the *circonvolution frontale ascendante*, and the following can be found from top to bottom: the lower limb center; the upper limb center; near the middle part, the center of the facial muscles; and in the lower part, the center of mouth, tongue and jaw movements”. David Ferrier (1843–1928) used some of the observations and conclusions made by Bourdon in his own work on brain localizations in 1878 [47], translated into French the following year [48].

## 6. Cerebral rheumatism

The first description of neurological damage during acute rheumatoid arthritis was the work of Nicolas-Joseph Hervez de Chégoïn (1791–1877) from 1845 to 1856, which associated meningitis with paralysis in the limbs [49].

On 11 April 1860, Bourdon presented a dissertation to the Medical Society of Hospitals, describing a case of hemiplegia appearing during or after episodes of acute rheumatoid arthritis. In the autopsy, he only found meningeal congestion

without cortical damage, based solely on macroscopic examination of the brain [50]. He did not indicate the condition of the cerebral vessels, which may have contained detached emboli from valvular endocarditis, as described by Benjamin Ball in his *thèse d’agrégation* (for entering academic process) in 1866 [51].

## 7. Conclusion

While the neurological research of Bourdon was not as significant of that of Charcot, it remains seminal in that it described neurological sequela of carbon monoxide poisoning, a long time before Grinker. Drawing on Luys’s expertise, it added to the work that Duchenne de Boulogne conducted prior to the research of Charcot and Vulpian as well as Duménil in 1862 [52], on damage to the posterior roots in progressive locomotor ataxia.

## Statement of ethics

This work required no approval from an institutional review board and was prepared in accordance with ethical guidelines of the journal.

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The author declares that he has no competing interest.

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