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History of neurology

Louis Duménil (1823–1890), surgeon and pioneer in neurology



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ABSTRACT

Louis-Stanislas Duménil (1823–1890) was a surgeon from Normandy who was a contemporary of Jean-Martin Charcot (1825–1893). Throughout his career, Duménil published annotated observations of neurological pathologies. One year before Guillaume Duchenne de Boulogne (1806–1875), he reported a case of “progressive muscular paralysis of the tongue, soft palate, and lips”. He added five other cases of progressive muscular atrophy in 1867, together with histological examinations which showed atrophy in the anterior horns of the spinal cord. Charcot, who described amyotrophic lateral sclerosis, did not fail to pay homage to Duménil for his contribution. In 1862, Duménil added clinical observations of progressive locomotor ataxia, one of the first to do so. This included anatomopathological examinations, thus significantly completing the clinical picture presented by Duchenne in 1858. He confirmed the damage to the roots and posterior tracts of the spinal cord. Finally, by providing multiple observations of the syndrome described by Octave Landry (1826–1865) in 1859, he contributed to the clinical picture of “acute ascending paralysis” which has come down to us as Guillain-Barré syndrome, with no mention of the perspicacious physicians of the previous century who had already perfectly recognised this disease. Finally, Augusta Dejerine-Klumpke (1859–1927) paid a warm tribute to Duménil in her 1889 thesis, calling him one of the pioneers in understanding “the individuality and autonomy of the peripheral nervous system.” He was indeed a pioneer, although he has been often overlooked.

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1. Brief biography

Louis-Stanislas Duménil was born on 30 November 1823 in Fontaine le Bourg (northern France). His father was a farmer, Jean-Louis Duménil (1776–1831), and his mother's maiden name was Rose Quéval (1792–1858). After studying the humanities at the secondary school in Rouen, he started his medical studies at the Rouen medical school then left for Paris.

After winning the prize for the best non-resident student in the Paris hospitals, he ranked first in the resident entry exam in 1849; Octave Landry (1826–1865) was sixth. He won the resident's silver medal in 1851.

Duménil (Fig. 1) defended his thesis on 08 March 1854: *Quelques points de l'histoire du lupus* (some aspects of the history of lupus) [1], with Achille Requin (1803–1854) presiding over the jury. This work aimed to differentiate between tuberculous lesions of the skin and atrophying

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Fig. 1 – Portrait of Duménil in *La Revue médicale de Normandie*, 1890 (Collection OW).

erythematous pathologies, the nature of which is currently difficult to interpret.

Following his studies, he returned to Rouen to practice medicine. In 1857, he was appointed deputy chief physician of Hôtel-Dieu hospital. In 1862, he became deputy chief surgeon of the Hospice général, and from 1865 to 1867, he served as head of the surgery and childbirth department. From 1868 to 1883, he was a surgeon at the Hôtel-Dieu hospital in Rouen before becoming a professor of external clinical medicine at the Rouen medical school, a post he held until his death in 1890 [2]. His most famous student was Charles Nicolle (1866–1936), whom Duménil taught in his third year of study. Nicolle went on to win the Nobel Prize in Medicine. In Nicolle's notes, collected after his death, he said this of Duménil: "I carried the mark of this man into my work at the Institut Pasteur. Never have I had a greater teacher" [3].

The first medical conference in France was held in Rouen, starting on 30 September 1863 and lasting for four days. It was initiated by the local medical society in which Duménil played a critical role: "We saw physicians from the farthest cities devote what little leisure time their many duties left them to come to us and contribute their knowledge and experience. The swiftness with which the conference was conceived of and organised proves how much support it garnered and how much success lay in the future for this new institution" [4]. His personal presentations covered surgical fields [5].

After becoming the director of the school of medicine and pharmacology in Rouen, Duménil was elected on 02 June 1885

as a national correspondent of the French Academy of Medicine in the surgical pathology division. He was a Rouen city council member as well as a Knight of the Legion of Honour and an officer of public education. On 04 September 1890, he died suddenly at his home on 45, rue Thiers in Rouen.

2. Neurological publications

Duménil was a contemporary of Jean-Martin Charcot (1825–1893), and although he was a surgeon, throughout his career he published articles not only in his speciality fields, but also in medicine, particularly in neurology, always providing anatomopathological data based on autopsies he performed himself. Before focusing on these articles, a book that played a large role in his fame among his contemporaries should be mentioned. Duménil was a Germanophile and translated the hepatology treatise of Friedrich Theodor Frerichs (1819–1885) in 1862 and the three editions that followed it [6].

3. Syringobulbia or amyotrophic lateral sclerosis?

Duménil's first neurological publication dates from 1859. A 53-year-old man, after pain in the palm of his left hand, noticed a progressive weakening of his strength, from the hand to the shoulder, over the course of a year. Paresis dominated in the territory of the median nerve. Then total paralysis of the patient's tongue set in, making any swallowing impossible. His tongue was insensitive to pricking and his sense of taste had disappeared. The two facial nerves were paretic whereas sight and hearing were totally unimpaired. There is no information on eye movement. The patient had low cervical pain and difficulty turning his head. Intellectual faculties were intact until the patient's sudden unexpected death. Duménil performed his autopsy. The brain and brainstem appeared to have no macroscopically visible lesions. The two hypoglossal nerves were atrophied from their origin outside the medulla and were greyish in appearance. The muscles they enervate were healthy. The facial nerves were healthy at their origin and appeared atrophied where they left the stylomastoid foramina. The trigeminal nerves were intact whereas the spinal nerves and cervical spinal nerves had the same pathological appearance. Duménil wrote, "Despite the multiple points of the peripheral nervous system that have atrophied, it is surprising to see this damage exclusively in the motor nerves" [7]. He wanted to understand the discrepancy between the macroscopic damage to the nerves and the absence of secondary muscular atrophy. He did not write any commentary on the state of the cervical joints and did not suggest any diagnosis. He also did not report any sectioning of the spinal cord and medulla for examination. In 1859, syringomyelia and syringobulbia were still unknown, although Charles-Prospér Ollivier d'Angers (1796–1845) had coined the words in 1837. He had observed the existence of the physiological spinal cord canal and decided it was pathological; for him, the normal spinal cord did not have one [8,9]. Did Duménil's patient have syringobulbia or a bulbar localisation of amyotrophic lateral sclerosis?

In 1860, Guillaume Duchenne de Boulogne (1806–1875) published his famous article, “*Paralysie musculaire progressive de la langue, du voile du palais et des lèvres*” (progressive muscular paralysis of the tongue, soft palate, and lips) [10], which was the first milestone in the description of amyotrophic lateral sclerosis in its bulbar form. Armand Trousseau (1801–1867) would give a name to this clinical picture in his clinical lessons: “Duchenne labio-glosso-laryngeal paralysis”. Duchenne cited the observation written by Duménil and noted several similarities with his own cases. Duchenne asserted: “This is a case of progressive fatty muscular atrophy, limited to the upper limb and associated with progressive paralysis of the tongue, pharynx, and lips”. Duménil’s response appeared in 1861. He contested Duchenne’s analysis which had included nothing of his description of the state of atrophied nerve roots whereas the muscles and notably the tongue were not atrophied [11]. Duchenne also did not include the total anaesthesia of the tongue. Neither of them described muscular fasciculations. Nevertheless Duménil, a skilled clinician, was one of the first to describe the clinical picture of progressive spinal atrophy even though he did not understand the various possible origins [12]. This leads us to some elements of the controversial debate between Edward Meryon (1807–1880) [13] in London and Jean Cruveilhier (1791–1874) [14] in Paris, namely whether paralysis arose from the central nervous system or whether it was an autonomous muscular pathology [15].

In 1867, Duménil published five new clinical cases, four of which were comparable to his first description involving progressive muscular atrophy of the limbs. The associated bulbar damage was either initial or secondary over the subsequent course of the illness, but for the two cases, he wrote, “The tongue is always agitated by partial movements, a sort of fibrillary shaking” [16]. His patients benefited from electrical exploration: “There is no contractility in any [atrophied] muscle”. All patients died in a few months due to their inability to swallow and ingest food. The microscopic examination revealed that “the cells of the anterior horns are generally underdeveloped, without prolongation. The grey matter of this region appears rarefied at some points.” The cause in the fifth case seems to be a lumbar vertical abscess that Duménil did not explicitly differentiate from the others, merely observing and analysing the atrophy in the lower limbs. He did note, however, that “the nervous tubes [were] for the most part normal in volume”. For his first four cases, “The anatomical lesions [were] characterised by atrophy in the motor nerve roots”. He saw this as “glosso-laryngeal paralysis” associated with “progressive fatty muscular atrophy”. He thus used the same terms as Duchenne at this time. In conclusion, Duménil proposed a pathophysiological hypothesis based on a disturbance of the sympathetic nervous system.

In 1869, Charcot and his resident Alix Joffroy (1844–1908) associated primitive sclerosis of the lateral tracts of the spinal cord, responsible for pyramidal syndrome, with a syndrome involving anterior horn lesioning along with muscular atrophy, concomitant paralysis, and fasciculations. This formed the clinical picture of amyotrophic lateral sclerosis or Charcot disease [17] (Fig. 2). Charcot did not fail to pay homage to Duménil from the start of the article: “We will have

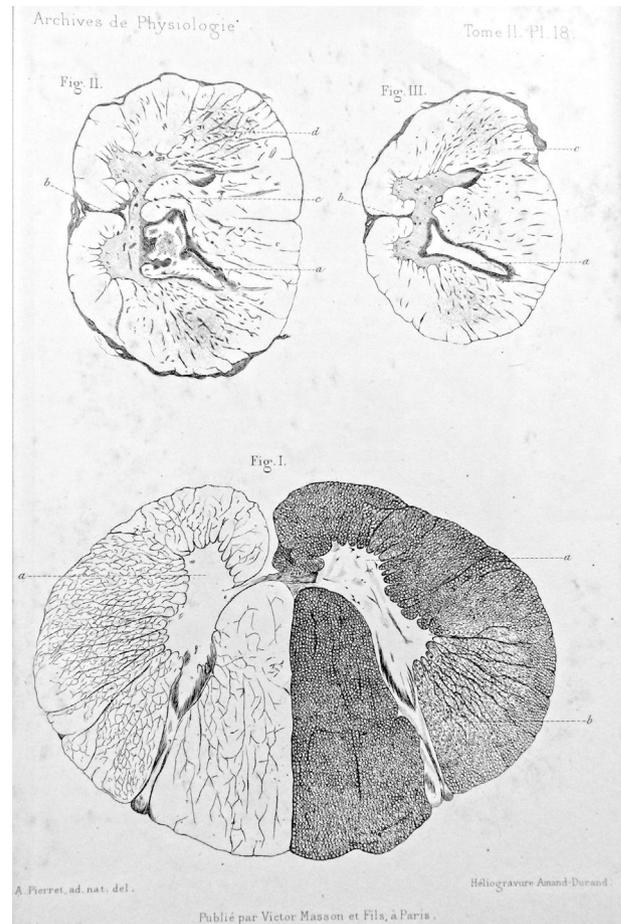


Fig. 2 – Illustration by Auguste Pierret (1845–1920) of the article by Charcot and Joffroy (1869) (Collection OW).

the opportunity to cite several of the highly remarkable observations of the Rouen physician, Duménil”, recognising in this way Duménil’s distinguished observational work.

4. Progressive locomotor ataxia

In 1862, Duménil presented the following note to the Société Médicale des Hôpitaux: “Note sur la dégénérescence, avec atrophie, des cordons postérieurs de la moelle épinière et ses rapports avec l’ataxie locomotrice progressive” (note on the degeneration with atrophy of the posterior tracts of the spinal cord and its relationship with progressive locomotor ataxia) [18]. The debate at the time centred on the anatomical site of the lesions to explain progressive locomotor ataxia, described in 1858 by Duchenne [19]. In 1851, Moritz von Romberg (1795–1873) had described *tabes dorsalis* (from the Latin for “to melt” or “to liquefy”) [20]. Charcot referred to this description as an initial sketch. Based on a detailed clinical case (“The sensitivity of the limbs to a pin is not keen”), Duménil discussed the anatomopathological examination, only macroscopic, of the entire spinal cord and transverse sections. He confirmed damage to the atrophied posterior roots and

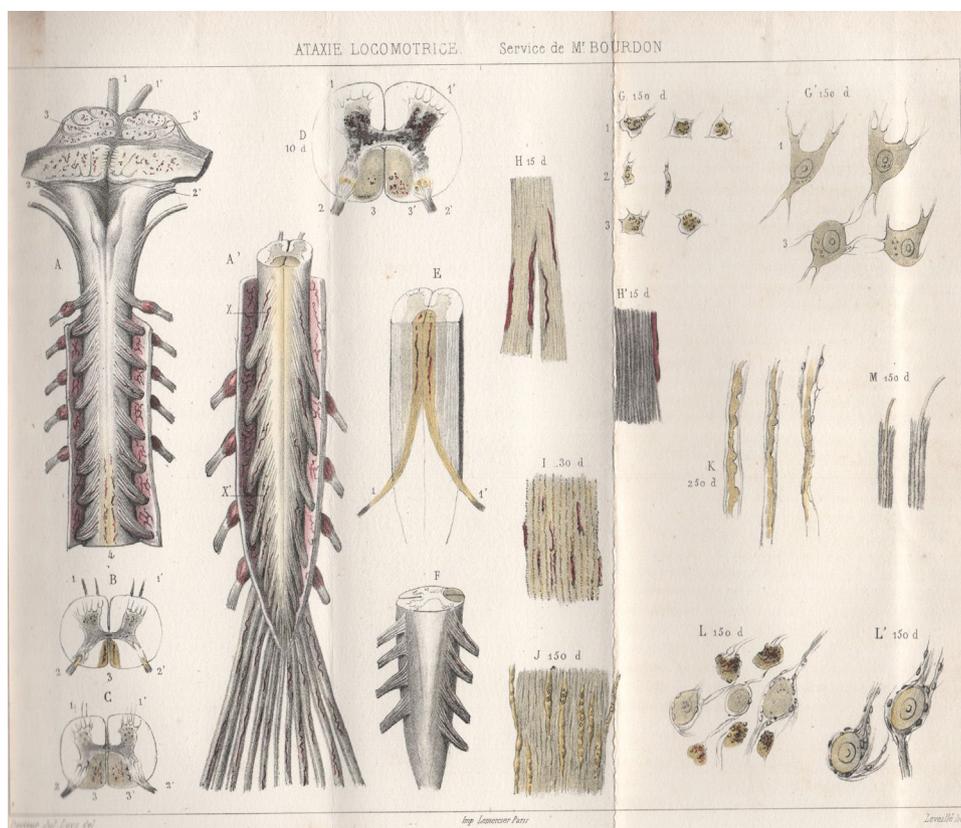


Fig. 3 – Illustration by Jules Luys for the article of Hippolyte Bourdon explaining damage to the posterior horns in Duchenne progressive locomotor ataxia (Collection OW).

tracts, in keeping with the presentation of Hippolyte Bourdon (1814–1892) to the Société Médicale des Hôpitaux on 20 August 1861 based on a microscopic study conducted by Jules Luys (1828–1897) [21] (Fig. 3). Charcot and Alfred Vulpian (1826–1887) made good use of this in their 1862 article published shortly thereafter: “Sur deux cas de sclérose des cordons postérieurs de la moelle avec atrophie des racines postérieures (tabes dorsalis, Romberg, ataxie locomotrice progressive, Duchenne) (on two cases of sclerosis in the posterior tracts of the spinal cord with atrophy of the posterior roots [tabes dorsalis, Romberg, progressive locomotor ataxia, Duchenne]) [22]. Duménil wrote, “We do not hesitate henceforth to consider the lack of motor coordination to be the expression of the lesion in the posterior tracts of the spinal cord”. But a question remained. All of the described cases involved patients who had died of pulmonary tuberculosis: “Is this merely a coincidence or is there a link between the spinal cord damage and that of the lungs?” When he had presented the same observation to the Société de Médecine de Rouen, “several members spoke out against this denomination. When I later presented the result of the autopsy of the subject to the same society in Rouen, everyone thought the facts supported the objections made previously regarding the denomination of locomotor ataxia”.

One can conclude that the notion of ataxia had not yet entered the semiology. However, starting in 1855 Landry attempted to clarify the physiology of sensation which was

still particularly vague at the beginning of the nineteenth century: “There are really only a few cutaneous sensations which may be called primitive or special: temperature, pain and, contact. All others result from them and can be called derived sensations. There is also a primitive or special sensation involving muscular activity that gives rise to secondary or derived sensations. This sensation does in fact reside in the muscular tissue itself, being an actual perception by the brain of the state of nervous and sensitive extremities enervating the muscles” [23]. Landry was the first to distinguish, before Duchenne, “the paralysis of the feeling of muscular activity”; that is, he put forward the idea that anaesthesia of touch differed from the loss of deep elementary and proprioceptive sensitivity [24]. At the same time as Charcot and Vulpian, Duménil supported Landry’s novel idea, which many of their colleagues clearly did not understand.

5. Extenso-progressive ascending paralysis

Before examining Duménil’s writings, some events in Paris occurring more than thirty years before need to be mentioned. In 1828, Auguste-François Chomel (1788–1858) reported an epidemic that was moving through the capital. Those affected complained of “a numbing and dimming of the sense of touch. Several patients are entirely unable to move” [25]. The Irishman Roberts F. Graves (1796–1853), who witnessed these

events, gave a lesson on them in Dublin in 1843, published in 1848. According to him, it was an epidemic of polyneuritis with a mysterious cause [26]. When in 1859, Landry wrote the article that resulted in the eponym, Landry syndrome, a term whose use has waned, he associated the reports left by Chomel and Graves with the clinical cases that he had compiled with his teacher Adolphe Gubler (1821–1879) [27]. His aim was to show the novelty of the clinical picture for this type of paralysis with a progressive and ascending course, which he called “extenso-progressive ascending paralysis” [28] and which was presaged by paraesthesia in the limbs. Only one of his cases involved an anatomopathological examination which revealed that the spinal cord and the paralysed muscles were intact, but it did not cover the examination of the nerves.

In 1864, Duménil published the observation of a 71-year-old man who, within two weeks, developed an ascending paralysis, mostly asymmetrical, from the feet to the thighs, accompanied by insensitivity to pricking and later extending to the hands and up to the shoulders. “Electrical contractility using Duchenne’s device is absolutely zero” [29]. The patient died of tuberculosis. The anatomopathological examination was performed by Georges Pouchet (1833–1894): “We appear to have in this case real atrophy in the spinal substance of the peripheral nervous tubes” forming radicular neuritis. This would become “the sites of segmentary demyelination” in the words of Augusta Dejerine-Klumpke (1859–1927) in her 1889 thesis: *Contribution à l’étude des polynévrites en général et des paralysies et atrophies saturnines en particulier* [30] (contribution to the study of polyneuritis in general and saturnine atrophies and paralysis in particular).

That same year, in 1864, Émile Bablon (1842–1914), a student of Louis-Théodore Laveran (1812–1879) at the Val de Grâce hospital, drew on Landry’s publication publish a typical case of ascending paralysis leading to death. The anatomopathological examination, which was only macroscopic, was considered to show no anomalies [31].

In 1866, Duménil reported on the case of a 36-year-old woman suffering from paraesthesia of the extremities followed by progressive and ascending paralysis affecting successively the right and then the left side of the body before extending to the bulbar cranial nerves. After a progression over eleven months, she died “of slow asphyxiation due to progressive weakening of the respiratory system” (Fig. 4) [32]. Duménil completed his description with two other cases in which the same type of ascending paralytic and sensitive disturbances were followed by a slow improvement, such that the patients were sent home. He described this pathology as “ascending neuritis” and suggested that the atrophy of the nerves should be considered as “the anatomical basis for some types of peripheral paralysis. As to the pathological process that leads to this atrophy, it is indisputably inflammatory in nature.”

One year before Duménil, in 1865, the Italian physician Pellegrino Lévi (1834–?), then a resident under Hermann Pidoux (1808–1882), published an observation of “acute ascending paralysis” leading to the death of a 22-year-old man, a tetraplegic with bulbar damage [33]. The anatomopathological examination was performed by Victor Cornil (1837–1908), Charcot’s resident in 1863. The microscopic examination of sections of the spinal cord did not reveal any

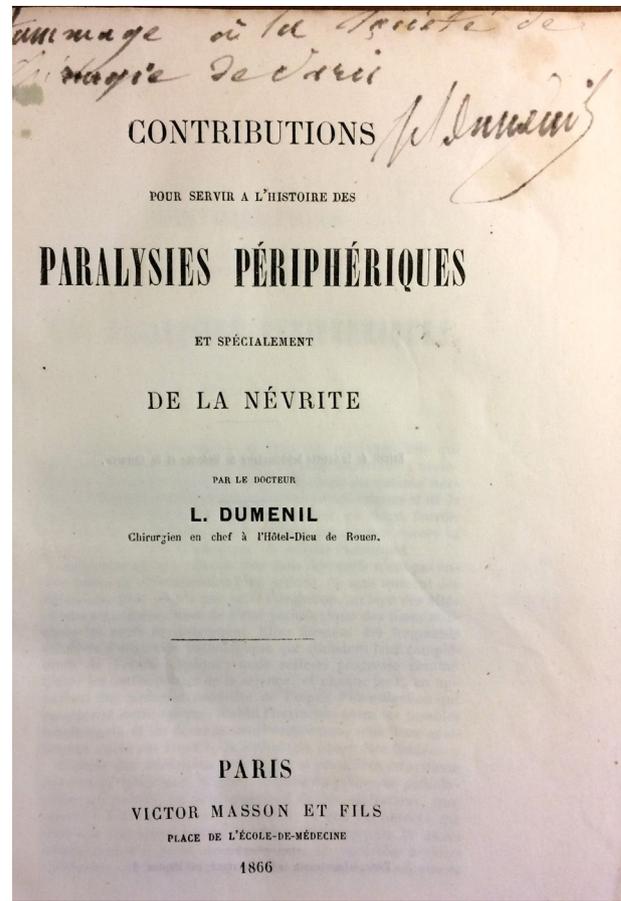


Fig. 4 – Dedication by Duménil of an offprint addressed to the Society of Surgery of Paris (Collection OW).

morphological anomalies. Lévi included the observation of the painful death of the famous naturalist Georges Cuvier (1769–1832), which was similar to that of his young patient. Cuvier thus did not die of cholera, as it is often written, but rather of Landry syndrome [34].

Alfred Petitfils (1845–1915) defended his thesis on 05 June 1873 [35], with Charcot et Charles Lasègue (1816–1883) as jury members. In his thesis, after describing progressive muscular atrophy (Charcot disease), he compared the clinical picture of Landry’s acute ascending paralysis and that of acute spinal paralysis, which would become the disease described by Jakob Heine (1800–1879) and Karl Oscar Medin (1847–1927), or acute viral poliomyelitis. Petitfils referred notably to Duchenne’s book, *De l’électrisation localisée et de son application à la pathologie et à la thérapeutique* (on localised electrification and its application to pathology and treatment) [36]. Petitfils, influenced by Charcot, concluded: “From a pathogenic viewpoint, acute ascending paralysis could be positioned between acute spinal paralysis in children and adults and progressive muscular atrophy”. In his thesis defended on 28 April 1873, with Gubler presiding over the jury, Alexandre Henry (1848–1904) refuted this conclusion and unified these types of paralysis into two progressive forms of the same disease [37]. This reasoning was strictly opposite to that expressed by Carl Westphal (1833–1890) in 1876. Westphal clearly distinguished

acute ascending paralysis from acute anterior poliomyelitis [38].

Thus, there were various explanations why a spinal cord remains ‘normal’ in acute ascending paralysis. For example, Samuel Wilks (1824–1911), working at the Guy’s Hospital in London, suggested that: “there may be a state in which the so-called reflex paralysis has occurred, in which the cord is in no way structurally altered, and therefore may at any time recover its function”. Wilks referred to nine cases of ‘acute ascending paralysis’ which he had seen. All died within four to thirty days. The spinal cord was examined in six. In no case was there any macroscopic change. In four, the microscopic study was non-conclusive. In his book published in 1878, Wilks did not mention Landry or Dumenil [39].

In his thesis defended on 22 February 1879 with Vulpian presiding over the jury, Jules Dejerine (1849–1917) referred to the hypotheses put forward by another of Vulpian’s students, Jean-Urcisse Chalvet (1845–?) in his 1871 thesis [40]. Dejerine, after an anatomopathological study in 1878 of “nervous system lesions in diphtheric paralysis” [41], had focused his thesis on “research into the nervous system lesions in acute ascending paralysis” [42]. Using new techniques, including fixation in osmic acid and staining with picro-carmin or haematoxylin, Dejerine improved the histopathological examination. For him, the damage to the anterior roots was identical to that of the peripheral end of a sectioned nerve; he referred to “parenchymatous neuritis”. Later he added, “We do not believe that this damage of the roots is primitive, that it alone constitutes the lesion of ascending paralysis. Rather, we believe that it is subsequent to damage to the grey matter of the spinal cord. This damage remains inaccessible to our current methods of investigation, but we can view it as probable according to the progression of the illness”. Dejerine had already proposed this interpretation in 1876 [43]. His wife, Augusta Dejerine-Klumpke, would refute this theory in her own thesis, defended ten years after that of her husband.

During these various periods, several causes were postulated: toxic causes (lead, arsenic, alcohol), infection (diphtheria, poliomyelitis, rabies), and idiopathic causes. Gubler referred to “paralysis of convalescence” for paralysis occurring after diphtheria or “eruptive fever”. Duménil referred to the works of Charcot and Vulpian [44] for the description of damage to “the motor nervous element in the paralysis of the soft palate following diphtheric angina” [29].

In 1916, Georges Guillain (1876–1961), Jean-Alexandre Barré (1880–1967), and André Strohl (1887–1977) would compile different variants in terms of aetiology, clinical aspects, and progression. More importantly, they would describe in detail the biological profile of “albumino-cytological dissociation of the cerebrospinal fluid” [45], the basis for the eponymous syndrome.

Marcel Petiot (1897–1946), who owes his infamy to being one of the deadliest serial killers of the 20th century, guillotined in 1946, defended his thesis (did he buy it?) in 1921 after his very brief medical studies. Remarkably, his thesis contains an observation of Landry’s syndrome, involving bulbar damage that was fatal in less than a week. He never cited Guillain and Barré but made a poorly supported attempt to lump the data together, suggesting a common aetiology for “Landry’s disease” and the encephalitis lethargica described

by Constantin von Economo (1876–1931), which had reached epidemic proportions by that time [46].

As noted in 1981 by John D. Spillane (1909–1995) [47], based on Dejerine-Klumpke’s work, Duménil was the first to have provided a histological description of the lesions of the peripheral nerves when the anterior horns of the spinal cord remain normal. He postulated a hypothetical method of inflammation, like the author of the seminal description, Robert Graves, to whom he paid homage. The current SARS-CoV-2 epidemic makes the Guillain-Barré syndrome, the novelty of which Duménil and Landry were the first clinicians to grasp, all the more relevant at this time [48].

6. Unilateral paralysis of the soft palate originating in the central nervous system

In April 1875, Duménil wanted to shine light on a neglected clinical symptom, unilateral paralysis of the soft palate [49]. He noted the initial description by a little-known author, H. Montault, in 1831 [50], but omitted to cite the foundational thesis of François Magendie (1783–1835) in 1808: *Essai sur les usages du voile du palais* (essay on the uses of the soft palate) [51]. Duménil contested the idea that the simple deviation of the uvula sufficed for diagnosis, adding, “We have found a much surer indicator of the lack of symmetry in the two halves of the isthmus of the pharynx, especially during muscular contraction. Contractions are provoked by touch. The paralysed side can be seen to be open whereas the arch on the opposite side diminishes by the coming together of the pillars toward the median line and the lowering of the summit. At the same time, the posterior pillar of the healthy side detaches from the pharyngeal wall, while that of the paralysed side remains attached to it”. He classified these types of paralysis as isolated or associated when paralysis of the facial muscles is present. He gave two examples of the first type in general paralysis. His other examples were alternating syndromes or cases of embolic cerebrovascular pathologies occurring in arrhythmia or mitral lesions. Regarding the unilateral paralysis of the soft palate, disturbing speech and swallowing, Duménil wrote, “It is not encountered more frequently because physicians do not look for it”. This paralysis is most often only part of the clinical picture of cranial nerve paralysis. Duménil attributed this clinical picture to central or peripheral damage to the facial nerve rather than the vagus nerve, and nothing suggests he foresaw Collet-Sicard syndrome (Frédéric Collet 1870–1966–Jean-Athanase Sicard 1872–1929) [52,53] or Villaret syndrome (Maurice Villaret 1877–1946) [54]. He also did not foresee jugular foramen syndrome or Vernet syndrome [55] which refers to paralysis of the IX, X, and XI cranial nerves traversing the jugular foramen and was first described in the *Paris médical: la semaine du clinicien* in 1917 by Maurice Vernet (1887–1974).

7. Cerebral concussion

In 1877, Duménil made a presentation to the Society of Surgery presenting two observations of cranial trauma leading to death after several hours. He tried to determine the cause of

death during the autopsy. With or without fracture of the skull, he only observed “haemorrhagic dots” or congestion of the grey and white matter, which he attributed to a disorder in vasomotor regulation. At that time, intracranial hypertension and post-traumatic cerebral oedema were not yet known [56].

8. Spinal cord concussion

Duménil's last publication appeared in January 1885, co-written with the Rouen surgeon Edmond Petel (1848-?), in the *Archives de Neurologie*: “*Commotion de la moelle épinière, étude clinique et critique*” (spinal cord concussion, clinical and critical study) [57]. The frequency of spinal trauma was increasing at the time and rail accidents were believed to be the cause. During this time, when only clinical approaches were available, the diagnosis of dislocation or vertebral fracture was difficult and inexact. For Duménil, initial paraplegia was a sign of haematomyelia or haemorrhage of the spine. The slow sensory and motor recuperation was most often interrupted by complications from decubitus such as urinary infections, complicated by septicaemia and extensive bedsores. This led to a delayed death after extreme suffering. The physician's role was often reduced to predicting whether the course would lead to a hypothetical recuperation. Finally, the lack of knowledge is illustrated by the origin of the trauma often being attributed to locomotor ataxia. Syphilis was only considered a predisposing factor among a multitude of possible aetiologies. All of this indicates that in the field of post-traumatic neurology, the contributions of Duménil, a surgeon, are not very relevant.

9. Conclusion

Overlooking Landry, Augusta Dejerine-Klumpke provided the most valuable acknowledgement of Duménil's work, citing him several times in her thesis: “Credit lies mostly with Duménil, who worked in Rouen, for having shown, more than twenty years ago, that nerves could be primitively damaged, without earlier modifications to their trophic centres. With admirable clinical skill, this author linked a series of generalised atrophic paralyses to a peripheral lesion and showed the similarity that exists between these paralyses and the condition described by Duchenne under the name of subacute anterior spinal general paralysis. He observed the ascending progression of the lesion and concluded that certain types of generalised paralysis should henceforth be attributed to neuritis; he went on to adopt the name of ascending neuritis. We reprint here the author's conclusions in his remarkable work: “The fact that peripheral paralysis may spread to a large portion of the nervous system is not among the least significant and the least curious points in this illness's history. One could almost speak of its generalisation, to the point of compromising existence, by the invasion of nerves that are most essential to life, such as the vagus nerve. We see that the disease leads to hemiplegic glosso-laryngeal paralysis, preceded and accompanied by impaired sensation, which is clearly the repletion, on more important organs, of a morbid process that has invaded so many different points” [30].

Augusta Dejerine-Klumpke thus highlighted the pioneering role of Duménil in describing the “individuality and autonomy of the peripheral nervous system”, a pioneer who has been forgotten, except in Rouen, where a street bears his name.

Statement of ethics

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

Disclosure of interest

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