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## History of Neurology

# François-Amilcar Aran (1817–1861) and the recognition of spinal muscular atrophy

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### ABSTRACT

Jean-Martin Charcot coined the term Duchenne-Aran atrophy. The inversion of names compared to standard practice shows the respect Charcot had for Guillaume Duchenne de Boulogne, who had encouraged him to study nervous disease. Using innovative localised electrification, Duchenne identified various types of muscular atrophy which he distinguished from paralysis. But it was François-Amilcar Aran who, published the observations that he had compiled and studied with Duchennes help first in 1848 and again in 1850. The result was the seminal articles that led to the eponym “Aran-Duchenne hand”. Focusing on the second half of the nineteenth century in Paris, this article will explore how knowledge evolved around the nosography of different types of muscular atrophy, starting with Duchenne and Aran and then with Charcot and his students, notably Albert Gombault, Joseph Babiński, Fulgence Raymond, and Jean-Baptiste Charcot. This historical overview is accompanied by a biographical account aimed at rescuing Aran from the sea of oblivion and covering the other subjects he wrote about, especially in neurology: including cerebral hydatid disease, skull base fractures and “cancer of the dura mater”.

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Twenty-first century French neurologists refer to certain types of muscular atrophy of the hand using the eponym “Aran-Duchenne hand”. Guillaume Benjamin Amand Duchenne (1806–1875) remains well known as one of the pioneers of neurological study, admired and revered by Jean-Martin Charcot (1825–1893). But who nowadays has heard of François-Amilcar Aran?

## 1. A short life devoted to work

Born on 12 July 1817 in Bordeaux, Aran began his studies at the medical school in his native city before completing them in

Paris. He started working in the Paris hospitals as a student in 1836. In 1838, he came third in the competitive exam to become a resident, performing better than Pierre-Louis Gratiolet (1815–1865) [1], among others. In 1842, during his last year of residency at Hôtel-Dieu Hospital in Paris, he published the Manuel pratique des maladies du cœur et des gros vaisseaux (Practical Manual of the Diseases of the Heart and Great Vessels) [2]. Drawing on the work of James Hope (1801–1841) [3] in Manchester, considered the first English cardiologist in the modern sense [4], Aran reviewed the knowledge on valve physiology and compared it with data from auscultatory semiology, still a new and developing field at the time. Without doing so explicitly, he challenged the

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Traité des maladies du cœur (Treatise on Heart Diseases) by Jean-Baptiste Bouillaud (1796–1881) [5], an established Parisian professor whereas Aran was still a young unknown aged twenty-five. Aran did something unusual for his time by making reference to a text in English for his book. Throughout his career, he used his language skills in English and German, translating a book by the English physician James Henry Bennett (1816–1891) in 1850 [6] and a book by the Hungarian Josef Škoda written in German (1805–1881) in 1854. Škoda was known for perfecting and simplifying cardiorespiratory semiology through his interpretation of percussion and auscultation, based on the physics of sound and its transmission [7]. It is worth noting that Aran was listed as a physician who spoke German in the 1854 directory

Adressbuch der Deutschen in Paris [8] for German nationals resident in Paris [9].

On 26 May 1843, one year after publishing his cardiology book, Aran defended his thesis: Des palpitations de cœur considérées principalement dans leurs causes, leur nature et leur traitement (the causes, nature, and treatment of heart palpitations) [10] (Fig. 1). The jury was presided by Léon Rostan (1790–1866). This work bears witness to the state of knowledge at this time, when pulse measurement and auscultation were the only means of evaluating the heart. The same year he became an official hospital physician, in 1853, he passed the agrégation exam, opening the way to a career as a professor. His jury was presided by Gabriel Andral (1797–1876). The thesis he wrote, entitled Des morts subites [11] (cases of sudden

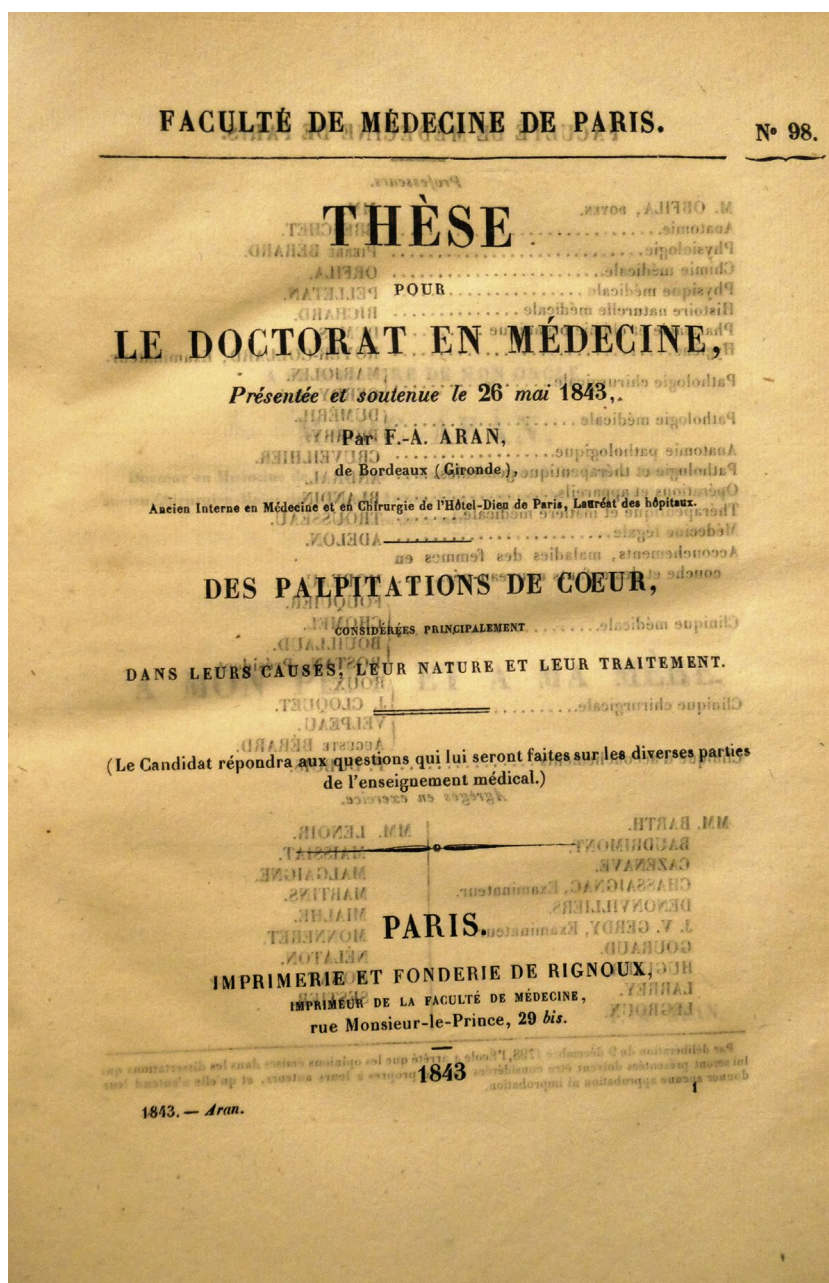


Fig. 1 – Arans 1843 thesis (BIUsanté Paris).

death), compiled from the written findings of coroners. The only neurological cause of sudden death listed by Aran is “apoplexy”.

Working at Saint-Antoine Hospital, Aran gave classes on therapeutics at the *École pratique* (laboratories, classrooms, and practicums at the Paris medical school). Those in attendance were described as “dedicated and enthusiastic” [12]. At the medical school, he deputised for Andral as an associate professor, teaching the class on pathology and general therapeutics in 1856. Then he taught Rostans class on internal clinical medicine from 1859 to 1860 [13]. Aran submitted many articles to the most widely read medical journals of his day: *Archives générales de Médecine*, *Gazette médicale de Paris*, *L’Union médicale*, and *Bulletin de Thérapeutique*. He provided reviews of recently published books, in all areas of medicine, as well as translations and abstracts of English or German publications. After his translation of a book by Bennett, in 1858 he published a collection of *cours libres* on gynaecology that he taught at the *École pratique* and in his department: *Leçons cliniques sur les maladies de l’utérus et de ses annexes* (clinical lessons on the diseases of the uterus and its associated structures) [14]. When he died, he was preparing a *Dictionnaire de thérapeutique*. “Aran was passionate about learning and research. He read voraciously and was familiar with the medical literature in every country” [12].

Very little is known about Arans personal life and no portrait exists. He was thirty-five when the Revolution of 1848 broke out. The right to form associations was a newly gained freedom and Aran participated in the foundation of an association of physicians. He was shocked when the Dean of the Paris medical school, Mathieu Orfila (1787–1853), and Achille-Louis Foville (1799–1878) were ousted under the Second Republic. It was a move worthy of the monarchy during the Restoration [15]. Aran proposed that all public medical positions be subject to election or competitive examination to combat arbitrary decisions concerning the medical staff as well as the administration, namely the minister. The National Assembly rejected his proposal [16].

Aran never married. On 22 February 1861, he died aged forty-four, within a few days, “ripped away from the tenderness of a beloved mother to whom he had resolved to devote his entire existence” [1]. His death was attributed to a “rheumatic phlegmasia of the meninges”, unsuccessfully treated by his renowned colleagues Bouillaud and Armand Trousseau (1801–1867). “We do not doubt that excessive work and a constant over-stimulation of the brain resulted in the mortal severity of the disease that took the life of our unfortunate colleague” [12]. Similar descriptions of his disease can be found in the biographical accounts that his contemporaries wrote, suggesting a death secondary to complications from acute rheumatoid arthritis. Isn’t it more likely that he died of tuberculous meningitis?

## 2. His most well-known publication: *Atrophie musculaire progressive*

In 1897, Pierre Marie (1853–1940) provided a vivid depiction of the 1850s: “If we wish to form a clear idea of the question we are examining, we should return in thought to fifty years ago,

between 1850 and 1853. Neuropathology was at that time in limbo; the various morbid states had not yet been defined. As a result, it was not possible to distinguish functional infirmity from actual paralysis, nor the latter from muscular atrophy, or rather no one thought to do so. In the old literature, muscle atrophy was described in the most fantastical terms: the limbs had dried out, or the nerves or tendons had withdrawn from them” [17].

When Aran looked after three patients suffering from atrophy of the hand and published his observations in 1848 in *L’Union médicale*, he was not yet appointed to a specific department and signed the article using only his initials. In the first patient, he had seen in Professor Rayers department at La Charité hospital he noted the “emaciated appearance of the hand”, in contrast to his general condition. Furthermore, both hands remain continually half-flexed, the palm is deeply hollowed out, and the tendons of the flexors and metacarpals can be felt through the skin. There is complete atrophy of the thenar and hypothenar eminences.” He presciently proposed the following hypothesis: “We are led to conclude that in certain cases, a disturbance may exist in the nervous system in some parts of the body such that the muscular system gradually atrophies and may even completely disappear, despite a perfect state of general health. Tactile sensitivity and irritability of muscular fibres under galvanic stimulation is maintained” [18]. He had asked Duchenne de Boulogne to come and test his patients muscles with faradism.

Two years later, he explained his thinking: “It seemed to me, after some bibliographical research, that this condition completely escaped the attention of the physicians who preceded us. They had confused it with paralysis.” He took this observation further by finding other similar cases, visiting the wards of La Charité Hospital, particularly the department of Jean Cruveilhier (1791–1874), Andral, and Pierre Éloi Fouquier (1776–1850). And he added the following note to his famous article published in September 1850, in *Archives générales de Médecine* [19] (Fig. 2): “Above all, I cannot fully express my gratitude to my friend Dr. Duchenne [de Boulogne], who was kind enough to provide me with all the facts he had gathered, and without whose obliging help I would have been unable to provide as much detail on the state of the muscular system of my patients”. Aran entitled his article: *Recherches sur une maladie non encore décrite du système musculaire (atrophie musculaire progressive [research on an undescribed disease that affects the muscular system [progressive muscular atrophy])*”. This long, seventy-two pages paper, published in two parts, summarises the clinical history of eleven patients. All of the patients were subjected to Duchennes electrodes for a “galvanisation” that tested the contractility of their muscles and made it possible to assess nervous conductivity (Fig 3 and 4). Whereas Aran was unable to precisely distinguish the functional capacity of each muscle using only clinical means, “Duchenne knew how to reveal the impalpable” [20] using faradic stimulation, which is to say that he assessed the contraction of muscle fibres that had remained intact. So one of the major contributions of Arans work was actually based on the data from Duchennes examinations!

Despite his knowledge of English, Aran was not looking for reflexes as Marshall Hall (1790–1857) had described them in



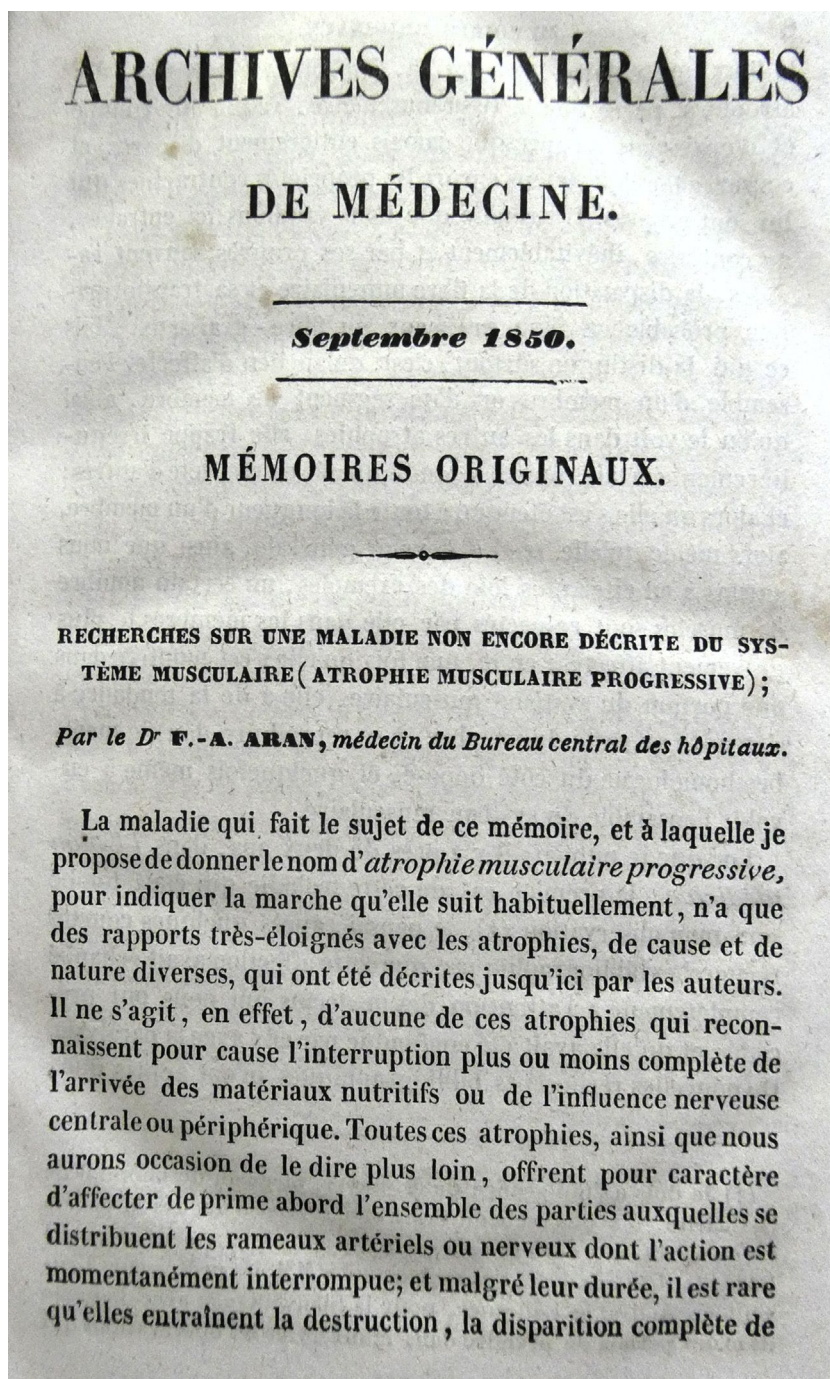


Fig. 2 – Arans 1850 article describing progressive muscular atrophy (OW Collection).

1841 [21]. and notions of an upper motor neurone lesion had not yet emerged.

In an envelope stamped with the number 857 [22] filed at the French Academy of Sciences on 16 October 1848 (not opened until 17 November 1982), Duchenne clearly distinguished progressive muscular atrophy from paralysis, considering it as an organic disorder of the muscles [23]. He interpreted the difference between the muscle contraction he saw and how the patient perceived it. He underscored the

fundamental distinction between the persistence and the decrease of the “electromuscular contractility” and was able to determine whether the damage was cerebral, in the peripheral nerve, or in the muscle. The following year, Duchenne filed a dissertation with the French Academy of Sciences entitled: *Recherches faites à l'aide du galvanisme sur les propriétés vitales du système musculaire dans l'état de santé et de maladie* (using galvanism to conduct research on the vital properties of the muscular system in states of health

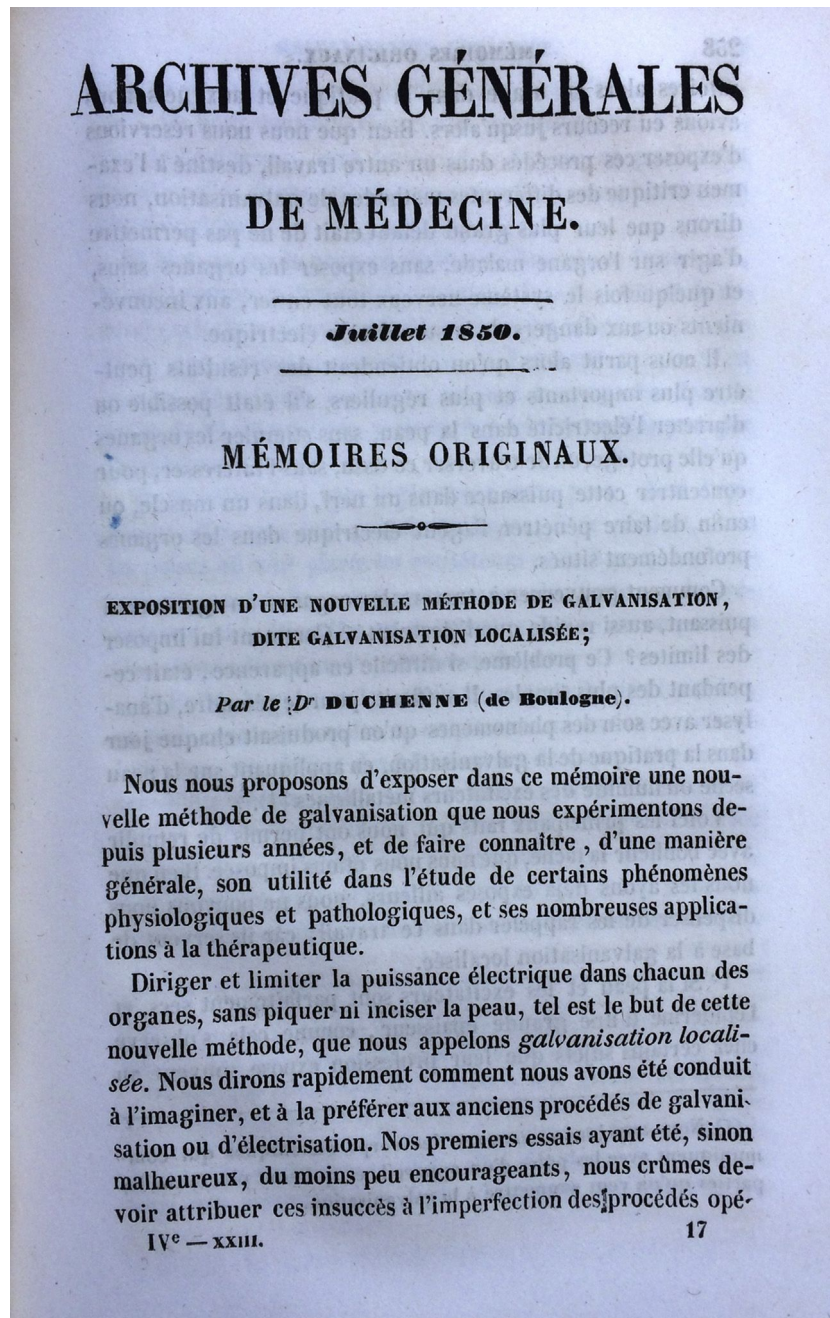


Fig. 3 – Initial 1850 Duchenne article that unveiled his galvanisation procedures (OW Collection).

and sickness) [24]. This put the finishing touches to the methodology he was using. A more detailed version was published in January 1850 in the Archives générales de Médecine [25]. Speaking of Duchenne, Jules Simon (1831–1899) noted in 1866, “I saw him demonstrate over and over at the La Charité Hospital what no one wanted to believe, that atrophy was a condition distinct from paralysis. He summarised his main findings in his first dissertation in 1849.”

Here is how Duchenne described the origins of his work, which began at the end of the 1840s: “For a long time, I carried out anatomico-physiological research based on my private

practice and my work in some of the hospitals. This allowed me to observe several types of muscular atrophy that had, up to that point, been confused with partial or general paralysis, while in fact they were nothing like paralysis. In these various cases, I saw the muscles atrophying in isolation, in the most irregular way, while maintaining their voluntary contractility until the atrophy reached its final stages” [26].

In his famous article, Aran did not neglect to mention that “without localised electrification, the history of progressive fatty muscular atrophy, the most serious and strangest of muscular conditions, all too common and indiscriminately





**Fig. 4 – Duchenne de Boulogne testing a patient. Bas-relief of the monument in his honour at La Salpêtrière Hospital. (Photo OW\_ Hôpital La Salpêtrière).**

affecting all classes of society, would have yet to be written”. The observations he compiled are marked by their heterogeneity. Some of them are cases of progressive muscular atrophy as seen in amyotrophic lateral sclerosis, as evidenced by “muscular twitching”; others are undoubtedly polymyositis, while still others are cases of Duchenne muscular dystrophy, where muscle fibres are replaced by fatty tissue [27]. Various pathologies poorly described at the time account for the cases of “chronic anterior poliomyelitis”.

Duchenne explicitly addressed patients X and XI to Aran, proof of their close collaboration. This is also made evident by Duchenne himself in 1853: “A muscular condition that has long remained misunderstood has entered into the nosological framework thanks to Mr. Aran, who charted its history under the name of progressive muscular atrophy” [28].

Case no. 5, referring to Madame Marguerite Duranger, a 31-year-old seamstress whom Aran examined with Cruveilhier, is the emblematic description of the Aran hand: “The right hand was very emaciated, the outer surface showed deep hollows and grooves, tracing the metacarpal spaces. The thenar and hypothenar eminences were sunken in, especially the latter, and no muscular resistance was detected at these locations. The skin was shrunken. The palm of the hand was somewhat thinned and was a little emaciated. The fingers were spread, especially the last three. They were half-flexed with a slight degree of extension, giving them the look of claws. The thumb was constantly opposed. The fingers could be easily spread, but it was difficult to move them together. The forearm was more emaciated than on the opposite side but not as much as the hand. The arm was also a little emaciated, but much less than the forearm, such that the emaciation of the upper limb seemed to decrease in the direction of hand to shoulder. The galvanism did not result in any contraction of the posterior palmar interosseal muscles, nor in the muscles of the hypothenar and thenar eminences, nor in the long abductor of the thumb” (Fig. 5).



*Fig. 110. — Main d'un sujet chez lequel l'atrophie musculaire progressive a détruit tous les muscles de l'éminence thénar (\*).*

**Fig. 5 – Illustration from Duchennes book on localised electrification (OW Collection).**

In 1854, Aran submitted a dissertation, *Recherches sur l'atrophie musculaire progressive* (research on progressive muscular atrophy) to the French Academy of Sciences to compete for prizes in medicine and surgery [29]. Of the 151 pages of his dissertation, one hundred were the transcription of twenty-two observations. From the anatomico-pathological point of view, he noted the inconsistency of fatty atrophy. He clearly reported on the atrophy of the anterior roots of the spinal cord, but like Duchenne at this time, he considered this as “a purely consecutive effect”; both believed that the disease was muscular in origin: “The correlation I find most difficult to accept consists of classifying this disease among the different kinds of paralysis. This is a disease of the muscular system and its morbid effect is primarily and solely in this system. It is totally unrelated to any lesion in the central nervous system” [19]. This was to be Arans last publication on the pathology.

André Thouvenet (1825–1905), from Limoges, was a resident in the Paris hospitals in 1847. He was a student of Cruveilhier who would preside over his jury and use his 1851 thesis as a means of disseminating his views. This thesis, entitled *De la paralysie musculaire atrophique* (on atrophic muscular paralysis) [30], was a detailed clinical description of amyotrophic lateral sclerosis which Charcot validated in 1874. Thouvenet reports on eight observations, some of which involved damage to the limbs and bulbar musculature. He also

describes “fibrillary contractions”. The various modes of progression are described in great detail. Finally, he confirms that “the electromuscular contractility seems to be maintained until the muscular tissue disappears”, probably referring to an examination performed by Duchenne de Boulogne even though this is not explicitly stated. Thouvenet did not mention the reflexes. He excluded a neurological origin, noting that “the nervous centres must apparently be excluded from consideration”. He affirmed the primary muscular origin, seeming to suggest that Aran had argued in favour of a neurological origin.

In the first edition of his 1855 book *De l'électrisation localisée et de son application à la physiologie, à la pathologie et à la thérapeutique* (on localised electrification and its application to physiology, pathology, and therapeutics) [31], Duchenne mentioned the “beautiful description” given by Aran in 1848, while in the third edition, in 1872, the word “beautiful” disappeared and he asserted that he was the first to describe this type of muscular atrophy: Arans article “was largely based on my findings”. However, we have seen that Aran was always careful to thank Duchenne for his decisive contribution. Arans premature death put an end to this spat, which arose after a long seemingly harmonious collaboration between the two men [32].

Cruevilhier can be credited with having macroscopically recognised in 1853 the atrophy of the anterior roots of the spinal nerves [33], which Louis Duménil (1823–1890), from Rouen [34], confirmed using a microscope in an 1859 case of bulbar and spinal damage. But it was Jules Luys (1828–1897) who clearly demonstrated in 1860 that atrophy of cells in the anterior horn of the spinal cord was the main anatomical lesion of the disease [35]. Shaken by this finding that overturned his initial thinking, Duchenne admitted in the third edition of his 1872 book (*De l'électrisation localisée - localised electrification*), demonstrating ones sense of fair play, that “this examination forced me to abandon the position I had defended up to this point concerning the pathogenesis and especially the peripheral origin of progressive muscular atrophy” [36].

Eugène-Louis Evrard (1843–1912) defended his thesis on 22 August 1867, in Strasbourg, under the direction of Charles Schützenberger (1809–1881) [37]. In it, he put forth the idea that, as a consequence of the controversy surrounding a nervous system origin or a primarily muscular origin, “the study of the symptoms has been brought to a very high degree of perfection”. He concluded, “Progressive muscular atrophy, characterised by a gradual degradation, is due to fatty degeneration or atrophy of the fibres of the sympathetic system that join the motor nerves. This cuts off nutrition to the muscles.” He ruled out myopathy. As to the agrégation thesis of Auguste Ollivier (1833–1894) in 1869 [38], it exemplifies the confusion at that time concerning the understanding and classification of muscular atrophy.

Charcot was able to clarify things by distinguishing between several different origins for progressive muscular atrophy. He notably described what he called “amyotrophic lateral sclerosis” by associating damage to the pyramidal tract with atrophy [39,40]. In his lessons published in 1872, compiled by Désiré-Magloire Bourneville (1840–1909), Charcot, “seeing several different conditions that had previously been confu-

sed” [41], introduced the classification of “protopathic progressive muscular atrophy”, which combined infantile spinal paralysis (i.e. poliomyelitis) with chronic paralysis in adults, and of “deuteropathic progressive muscular atrophy”, i.e. amyotrophic lateral sclerosis [42]. He returned to this distinction, honing his ideas on pathological anatomy, in his lesson on 19 March 1889 [41]. In the protopathic form, to which Charcot added the term of Duchenne-Aran atrophy, chronic poliomyelitis was broken down by the description of forms of peripheral neuritis in 1866 by Duménil [43], then syringomyelitis after 1882 [44]. The forms of amyotrophy with a muscular and hereditary origin became part of the nosography of atrophy after 1875 [45].

Albert Gombault (1844–1904) was a resident under Charcot in 1872. In his thesis, defended on 1 August 1877 with Charcot presiding over the jury, Gombault compiled all the clinical and paraclinical elements that characterised Charcot disease, based on nine cases observed at La Salpêtrière Hospital [46]. Antoine-Louis Florand (1857–1927), a resident under Fulgence Raymond (1844–1910), also centred his thesis on the disease in 1887 [47] tracing the history of the diseases description and citing foreign authors. He summarised all the available knowledge ten years after the work of Gombault. However, this research, completed before the discovery of neurons did not distinguish between central and peripheral motor neurons, nor take account of the diagnostic value of exaggerated tendon reflexes. Florand did his work prior to the description of the plantar reflex by Joseph Babiński (1857–1933) [48], meaning that he had no understanding of pyramidal syndrome.

It is worth also mentioning at this juncture the thesis defended on 5 June 1895 by Jean-Baptiste Charcot (1867–1936), the son of Charcot. The younger Charcot was at the time a resident under Raymond, his fathers successor. His thesis was entitled: *Contribution à l'étude de l'atrophie musculaire progressive de type Duchenne-Aran* (Contribution to the study of Ducenne-Aran type progressive muscular atrophy). As an homage to his father and Duchenne, he kept the name order used by his father during his clinical lessons [49]. He tried to show that atrophy, as described by Aran and Duchenne, was a disease in itself, to counter its reduction to a syndrome having various causes [50]. In 1897, Pierre Marie (1853–1940) opposed this idea: “Aran-Duchenne progressive muscular atrophy has become such a dogma, and its description is so stereotypical in all internal pathology and neuropathological treatises, that it logically seems misplaced to present this disease as a pure phantom, like a term to be deleted from the nosography. However such is the opinion we must adopt. Among the authors who oppose my view and have fought against it most earnestly, I am obliged to cite my friend Jean Charcot who, in his remarkable thesis, defended the integrity of Aran-Duchenne progressive muscular atrophy” [51].

Current neurological semiology still uses the expression “Aran-Duchenne hand”. The seminal description remains that of Aran in 1850, who described Marguerite Durangers hand, among others. Amyotrophic lateral sclerosis (Charcot disease) remains the most frequent cause, ahead of Charcot-Marie-Tooth disease, syringomyelia, and, in rare cases, syphilis [52].

Finally, Aran-Duchenne syndrome is also applied to a lower radicular palsy (C8-T1) such as described by Augusta-Dejerine-Klumpke (1859–1927) in 1885, when she was still a student [53]. This palsy affects the intrinsic hand muscles, the finger flexors, and the flexor carpi ulnaris. The sensory and pain disturbances affect the inner part of the arm, forearm, and hand and are sometimes associated with a Horner's syndrome.

### 3. Dissertation on cerebral hydatid disease

In response to two patients whose autopsies revealed the presence of hydatids in the brain, Aran observed, “I wanted to turn to previous authors to find something, some thread in the middle of this labyrinth, but I only found chaos and obscurity” [54]. The person who had recently reviewed knowledge in this area was none other than René Théophile Laënnec (1781–1826) [55,56], but in reality, the reproductive cycle of the worm, its mechanism of penetrating a mammal, and so forth, were largely unknown. After an attempt to classify the varieties of the worm, Aran noted how complicated it was to give a diagnosis of cerebral hydatid disease during a patient's lifetime. The rest of the text referred confusingly to “cancerous brain tumours” and tubercles. Aran frequently referred to a work by Louis-Florentin Calmeil (1798–1895) in 1828 that described incidental hydatid cysts found during the autopsies of insane patients at the Maison Royale de Charenton who had suffered from psychotic episodes and convulsions leading to their deaths, probably the result of intracranial hypertension [57]. Aran's text retains its historic value emphasising the extreme and prolonged suffering of these patients and their stoic fatalism.

### 4. Skull base fractures

To explain his interest in skull base fractures, Aran advanced two arguments. On the one hand, he blamed his colleagues for not taking a real interest in this area, notably because they considered knowledge on the subject to already be exhaustive. On the other hand, the book recently published (1842) by the surgery professor Édouard Chassaignac (1804–1879) seemed to Aran incoherent and too heavily in favour of trepanning while failing to first establish clear diagnostic criteria [58]. Aran, influenced by his teacher, the surgeon Philippe-Frédéric Blandin (1798–1849), set out to model the location and type of fractures based on how they occurred and their potential cerebral repercussions. His reasoning came to be known as “Aran's laws” [59]: “It seems that, using a more rational approach, we can depend on the anatomical arrangement of the contained and containing parts of the skull and take account of the appearance and sequence of certain symptoms to obtain, in the great majority of cases, accurate and satisfactory results”. For Aran, “the skull base is arranged in levels and the brain can be said to be shaped around it”; this made it possible to use immediate and secondary symptoms following trauma as a diagnostic basis.

He established that continuous bleeding from the external ear was a sign of tympanic perforation from the pressure of

the blood generated by a fractured temporal bone. He also noted that bruising of the eyelids and subconjunctival haemorrhage, when they appeared secondary to trauma, were the sign of a skull base fracture. Aran believed that the transformation of initial nasal bleeding into a clear, non-coagulable flow, was in every case a sign of cerebrospinal fluid leakage. This leak, according to Aran, was “a stimulus that, like a thorn, could cause a burst of phlegmasia in the meninges and brain”; that is to say, an infection would inevitably develop, though he had no knowledge of microbes. “Hernia of cerebral matter by the auditory canal” was in his opinion always fatal.

Aran finished with a chapter that covered the sensitivity and specificity with which physicians could localise the fracture, depending on the occurrence of a neurological deficit of the facial nerve, trigeminal nerve, external oculomotor nerve, and/or auditory nerve. “The concordance of symptoms” between the conditions of the trauma and its effects was important to study. For example, the physicians' ability to establish a prognosis depended on whether the “cerebral commotion” (i.e. the level of vigilance) became less marked or not. The presence of negative neurological signs of a cranial nerve “precluded trepanning”. His work also touched upon hemiplegia but did not mention localised or generalised convulsions secondary to traumatic brain injury, his article is innovative and courageous when one considers he was a young 27-year-old physician contesting the findings in a recent book by an established professor of surgery.

The same year Aran published an addition to his book in the form of an observation of a brain abscess that developed three months after severe traumatic brain injury with perforation of the skull and initial removal of bone shards [60]. His teacher Blandin had attempted a trepanning hole but did not open the dura mater. This was not followed by any purulent drainage. Aran found a vast brain abscess in the right frontal lobe and was surprised the patient had not suffered from a sensory, motor, or intellectual deficit, and in particular, that he could still speak, given that the frontal lobes were at that time considered to be the language centre, without specific lateralisation. He recommended that surgeons perform “the operation of trepanning”, and even incise the dura mater or brain, in certain indications. This was bold advice given the risk of infection which, at a time when the rules of asepsis were not yet followed, was invariably fatal.

### 5. “Cancer of the dura mater”

In 1854, Aran published an observation made in 1851. He compared it to three other similar cases subsequently published in the English literature [61]. A young 17-year-old man had rapidly developed facial palsy with complete homolateral deafness. His general condition was also affected. He was extremely pale, with hepatosplenomegaly and prolonged, repeated nosebleeds. His autopsy revealed regions of bone tumours invading the dura mater at the frontal and parietal levels and in the temporal bone, the macroscopic appearance of which had a strange greenish tint. Other tumours were found in the kidneys. “The results of the



micrographic examination: the presence of particular cells, most of them rounded and regular in shape; cells with nuclei and free nuclei, the interior of which was granular, while some of these nuclei contained nucleoli." Aran did not immediately make a specific diagnosis. Instead, he focused on the extension of the bone tumour into the dura mater. The term "chloroma" is currently used to describe a granulocytic sarcoma or an extramedullary myeloblastoma, which is usually associated with myeloblastic leukaemia. Localisation in bone is rare but all the tissues of an organism can be infiltrated with primitive precursors of myeloblasts and promyelocytes. These immature cells have a greenish tint because of their high levels of myeloperoxidase [62,63].

## 6. In conclusion

Aran, a clinician, was a prolific author, writing about all medical subjects, most often in relation to cardiology, gynaecology, and therapeutics. However, his name is now known to us only through one of his numerous publications, his 1850 dissertation describing "progressive muscular atrophy", which for Bonduelle, "remains one of the foundations of neurology" [32]. Arans close ties with Duchenne de Boulogne, a prodigious researcher, fully justify the fact that their names remain linked through an eponym. The Charcots, father and son, always used the name "Duchenne-Aran type muscular atrophy" to preserve the importance of each mans publications and underscore the homage to Duchenne, whom they both held in great esteem. Standard practice has made the inverse, alphabetic order more widely used. It is true that Duchenne was first in his research and discoveries, which he owed to his accurate functional analysis using the localised electrification he had invented. My purpose here is not to assign credit, as this has already been done; Bonduelle [32] gave precedence to Aran, whereas Delaporte and Pinell [20] and Fardeau [64] saw fit to put Duchennes name first. In any case, Aran transmitted knowledge and leveraged his talent in the same way Trousseau did. His premature death prevented him from enjoying the brilliant university career that in all likelihood would otherwise have awaited him.

## Statement of ethics

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