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History of neurology

Augustin Morvan (1819–1897), a little-known rural physician and neurologist

Augustin Morvan (1819–1897), médecin de campagne et neurologue méconnu

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ABSTRACT

Augustin Morvan (1819–1897) was a contemporary of Jean-Martin Charcot who practised medicine in rural Brittany. A perspicacious and astute clinician, he described three clinical pictures not previously isolated: in 1875 the semiology of myxoedema, in 1883 the neurological semiology of syringomyelia which he called “paretic analgesia of the upper extremities”, and finally in 1890 the semiology of “fibrillary chorea”, currently considered a model of synaptic pathology involving immunological damage to potassium channels and causing (as perfectly described by Morvan) myokymia, autonomic nervous system disturbances and agrypnia. “Fibrillary chorea” is today known as Morvan's syndrome and linked to limbic encephalitis.

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R É S U M É

Contemporain de Jean-Martin Charcot, Augustin Morvan (1819–1897) exerça, lui, la médecine dans la campagne bretonne. Perspicace et fin clinicien, il décrit trois tableaux cliniques non individualisés auparavant : en 1875, la sémiologie du myxoédème, en 1883, la sémiologie neurologique de la syringomyélie qu'il baptisa « paréso-analgésie des extrémités supérieures », puis, en 1890, la sémiologie de « la chorée fibrillaire », reconnue actuellement comme un modèle de pathologie synaptique par atteinte immunitaire de l'activité des canaux potassiques, responsable comme l'avait parfaitement décrit Morvan, de myokimies, de troubles neurovégétatifs, d'agrypnie et apparentée à « l'encéphalite limbique ».

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Augustin Morvan, of Lannilis in Brittany (1819–1897), was a rural physician working in his native region in the 19th century. By an exceptional fate, he still has his name associated with a syndrome listed in the PubMed medical database. A keen observer and astute clinician, he described myxoedema in 1875, then the clinical signs of syringomyelia, which he named in 1883 “paretic analgesia of the upper extremities” (known as Morvan’s disease among his contemporaries). In 1890, he described a muscular pathology which he named “fibrillary chorea”, currently referred to as Morvan’s syndrome, or as autoimmune channelopathy, and linked to limbic encephalitis.

1. Morvan, from Brittany to Paris

Morvan died on 20 March 1897 in Douarnenez at the age of 78, after five years of progressive physical and intellectual decline secondary to hemiplegia. His funeral service brought together an immense crowd of peasants, colleagues and important figures in Brittany. All the speeches given that day bear witness to his flawless devotion, his far-reaching reputation and his extensive medical knowledge, gained over 45 years of practice. Morvan was born in a peasant family on 7 February 1819 in the village of Foz-Nevez, which is part of the city of Lannilis (Aberwrach, Brittany). He was the eldest of eight children. After his studies at the military medical school in Brest, he was named a Marine surgeon on 18 June 1839, but resigned a few months later as he was unable to tolerate seasickness! After completing his training in Paris, he obtained a house officership there in 1844, thus becoming an *interne* along with Ludger Lunier (1822–1885) and Paul Broca (1824–1880). On 11 March 1847, he defended his thesis, entitled “Varicose aneurism” (*De l’anévrisme variqueux*), to a jury that included his teacher Auguste Nélaton (1807–1873). Despite the appeal of working in Paris, he planned to set up his practice in Brest. When he informed his mother of his plans, she burst into tears. According to legend, Morvan immediately unhitched his cabriolet and followed the dictates of filial love. He decided to settle definitively in Lannilis, a city of 3000 inhabitants. He quickly established a reputation and his fellow citizens pressed him to become mayor, then a regional administrator in 1857. In 1871, he was elected Deputy for the Finistère region under the new Third Republic, as part of a group known as the “Republican list for order and peace”. Famous for only providing free medical care during his term as Deputy, Morvan voted for the resignation of Adolphe Thiers (1797–1877) and supported the legalisation of civil funerals. But he is most associated with the Morvan-Roussel law, the first concerned with unfortunate children and aimed at protecting those who had been abandoned as well as young mothers. He personally developed this law at a time when public opinion reflected people’s fears about their collective responsibilities. He thereby gave a social orientation to legislative power, and later inspired other progressive physicians such as Désiré-Magloire Bourneville (1840–1909). His legislation also led to the law on obligatory public assistance (Anonymous, 1897; Le Gallo, 1992; Létienne, 1897; Robert and Cougny, 1889). Despite striving only to ease suffering and correct injustice, this distinguished practitioner was

constantly confronted with the superstitions of his patients and pressure from clergy. He was also subject to defamation and hostile acts, some taking place in front of his home. An example of the slogans attacking him: “People of Lannilis, you have elected an unworthy mayor, you have voted for a defender of prostitutes. Doctor Augustin Morvan of Lannilis is the father of prostitutes” (Desse, 1957). Undoubtedly because of this defamation, he was not re-elected and stopped his political activities. Nonetheless until the end of his life, he doctored tirelessly. He was known throughout Brittany—“An aotrou Morvan” or Monsieur Morvan—pronounced with fervour and respect by countless patients.

2. Myxoedema in lower Brittany

After writing to Jean-Martin Charcot (1825–1893) in 1875, Morvan published his correspondence in the *Gazette Hebdomadaire de Médecine et de Chirurgie* in 1881: “I believe I have observed an as-yet undescribed disease in Brittany, but I fear I am lacking in knowledge and thus appeal to your expertise. This disease is specific to women; at least until now I have not encountered it in men. It is characterised by anasarca and incomplete paralysis, but does not entail muscular atrophy or degradation of mental faculties. In other words, it is not explained by any known disease, no more so than oedema is explained by an infection of the heart or kidneys. Facial swelling, which coincides with slow speech and hoarseness, is pathognomonic. [...] There is always pronounced muscular weakness, but never to the point that it prevents walking. [...] All of our patients are very sensitive to cold”. In 1875, Morvan was thus the first to describe the semiology of what would become hypothyroidism. He continued: “If this disease has not been described, I intend to gather my eight observations together into a little study. I would be greatly obliged if you would allow me to call upon your erudition, if necessary. [...] Not obtaining a response as early as we wished, and believing that perhaps Monsieur Charcot, with whom we had no previous contact, reserved his time for more pressing obligations, we called upon our old colleague Verneuil. [...] We received a response from Monsieur Charcot. He apologised for not responding earlier, but he took advantage of the time elapsed to search his memory and the medical publications over and over, where he found nothing similar. He concluded by strongly advising us to publish our observations. We had not yet published anything and would not have at any rate, as we were lacking in material and hoped that time would provide us with further information. Then, in May of this year, we fell upon an article by Dr Merklen published, or rather republished in the *Gazette Hebdomadaire de Médecine et de Chirurgie* and entitled « Pachydermatous cachexia (cretinoid state, myxoedema) ». Out in the countryside where we live, *La Gazette* is our only journal. After noting that William Gull (1816–1890) had presented similar observations in London in 1873, in 1881 Morvan put forward 15 observations of women around 50 years old and exhibiting the same symptoms. He noted sensitivity to cold and measured their temperature at 36 °C; he also made mention of stubborn constipation, pulse rates slowed to 50 and slowness in thinking. He noted that, contrary to his initial finding, men and children can be

affected. He concluded with a pathophysiological discussion: “This appears to be an oedema of neuro-paralytic origin with vasomotor paralysis” [...] “We conclude that myxoedema is a central nervous system disease that only affects the motor nerves (in both the somatic and autonomic systems) which are paralysed. Thus it does not affect the portion of the central nervous system related to mental faculties or the sensory nerves”. He concluded these first observations of myxoedema, which were perfectly relevant from a clinical perspective, by alluding to his disappointment at the failure of all his therapeutic efforts (Morvan, 1881).

3. From paretic analgesia of the upper extremities with panaris inflammations to syringomyelia

With the clinical astuteness and observational precision that established the reputation during the same period of Charcot at La Salpêtrière Hospital, Morvan collected a series of observations of a disease that once again appeared to him as not yet isolated. From his first publication in 1883 in the *Gazette Hebdomadaire de Médecine et de Chirurgie*, he used a picturesque style (one that he maintained from that point forward) to describe his first patient: “The first case we observed dates back 25 or 30 years. It involved a 60-year old man who came to us with a panaris inflammation on one of his fingers. His hand and entire forearm were swollen. We observed necrosis of the finger at the nail and proposed an incision to the patient for its extirpation. Since the patient only accepted with moderate enthusiasm, we added that the incision would be lightening fast, that he would not have time to suffer. We proceeded with the incision, which involved a rather wide cut. How surprised we were to observe the calm demeanor of this good man who, in our view, was not exactly a hero but nonetheless did not bat an eyelid. He did not complain once. We would have believed him made of wood had he not been flesh and blood before us. He affirmed that he did not feel any pain. This was our first encounter with one of these analgesic panaris inflammations of the upper extremities”. In five successive communications from 1883 to 1889 addressed to the Académie de Médecine and published in the same journal, he described a novel clinical picture of a disease characterised by successive panaris inflammations, leading to necrosis and definitive deformation of the fingers, accompanied by muscular atrophy of the hand and upper limb, with disturbances in tactile and thermal sensation. He added to his initial description by noting the progressive appearance of bone fragility, hyperhidrosis, subcutaneous haemorrhage and bone/joint deformations. He highlighted the progressive nature of the deficits: “The disease which we will study involves paresis with analgesia in the upper extremities, initially limited to one side, then moving in most cases to the other side and always resulting in the production of one or more panaris inflammations” (Morvan, 1883, 1890). Well informed of the latest anatomopathological studies by Parisian neurologists, Morvan went so far as to propose a pathophysiological explanation, drawing on the first publication of Augusta Klumpke (1859–1927, the future Madame Dejerine), which described radicular paralysis in the brachial

plexus (Klumpke, 1885). Morvan wrote: “The disease started in the posterior tract and, probably at a later time, moved to the anterior tract. I believe the natural progression of the disease to be the following:

- analgesia; an initial analgesia, because it is incomplete, can be found alone, never paresis;
- when the two orders of nerves are involved, the sensory paralysis is always one degree more advanced than the motor paralysis, the former being complete whereas the latter is not yet complete and may never become so” (Morvan, 1886).

Morvan situated the cause of the disturbances in the spinal cord based on his clinical reasoning alone; he was unaware of the anatomical pathology of the disease he was describing since none of his patients had died and undergone autopsy. As a result of this study, Morvan was elected Corresponding Member of the Académie de Médecine. Matthieu Prouff (1849–1931), an interne in the Paris hospitals and originally from Morlaix, Brittany, published in 1889 with the help of his teacher Albert Gombault (1844–1904) the first anatomopathological observation of Morvan’s disease, but the severity of the scoliosis, recognised as secondary to the muscular atrophy, made it impossible to extract the spinal cord without damaging it. They did, however, note that the “central canal is very voluminous [...]. On several sections, the central region is seen to have collapsed, leaving only detritus; often this central region is occupied by a cavity giving way at the back, on either side, to a prolongation that follows the direction of the posterior horn” (Prouff, 1889). A form of university recognition, two theses defended in Paris were entitled “Contribution to the study of Morvan’s disease”, that of Georges-Charles d’Oger de Spéville (28 July 1888, jury president: Paul-Georges Dieulafoy (1839–1911)) and that of Henri Louazel (11 June 1890, jury president: Charcot) (d’Oger de Spéville, 1888; Louazel, 1890).

In 1882, Otto Kahler (1849–1893) in Prague and Friedrich Schultze (1848–1934) in Dorpat in Germany published observations of patients with amyotrophic paresis associated with thermo-anaesthesia of a limb. Kahler and Schultze explained these symptoms by the discovery during autopsy of an abnormal vertical cavity in the spinal cord. They called this pathology “syringomyelia”, employing the term first used by Charles-Prospér Ollivier d’Angers (1796–1845) in 1827 (von Kahler, 1882; Schultze, 1882). In France at this time, there were debates between those who believed Morvan’s disease and syringomyelia were one and the same, and those who opposed this idea, including Morvan himself. There were also aetiological discussions in which the ideas of Jules Dejerine (1849–1917) evoking a toxic (lead) or infectious (leprosy) peripheral neuritis were juxtaposed with those of Charcot’s internes: Paul Blocq (1860–1896), Adolphe Dutil (1862–1899) and their *chef de clinique* (specialist registrar) Georges Guinon (1859–1932) (Fig. 1). The latter group demonstrated through autopsy the existence of an abnormal spinal cord cavity in 1890 (Dejerine, 1890; Guinon and Dutil, 1890). While in his Tuesday lesson on 28 June 1889, Charcot was “not very favourable to the single doctrine”, in his March 1891 lesson he said, “There are discussions on these two morbid states as to whether they



Fig. 1 – Fingers of a patient with Morvan’s disease. Aspect des doigts dans un cas de maladie de Morvan. Georges Guinon et Adolphe Dutil. Deux cas de maladie de Morvan. Nouvelle Iconographie de La Salpêtrière 1890;3(1): 1–15. Private collection of the corresponding author.

represent independent diseases perfectly separate from each other, despite their exterior similarities, or whether they are simply the same condition. In this case, so-called Morvan’s disease would only be an episode or form or variety of syringomyelia. It became apparent at a certain point that only anatomical pathology could provide a definitive solution to the problem. It has spoken, and in my opinion, has done so peremptorily in favour of the single doctrine” (Charcot, 1889; Achard, 1890; Bruhl, 1890; Walusinski, 2012).

4. From fibrillary chorea to autoimmune channelopathy

On 12 April 1890, Morvan published a novel clinical description, once again in *La Gazette Hebdomadaire de Médecine et de Chirurgie*. With his customary modesty, he noted: “I have once again been favoured by chance which, as always, has sown my path with rare, little known cases. In his observation of the first patient, he wrote: “Paul Ernest de Plabennec, a farmer with a strong constitution, came to my office on 18 July 1885. [...] Eight days prior, he experienced a sort of trembling, specifically fibrillary contractions in his calf muscles. When I examined the patient, this twitching was mainly in the gastrocnemius; however, it also could be seen intermittently in the posterior muscles of the thighs. [...] By 21 July, the twitching had reached other regions of the body, tending to generalise. [...] The fibrillary twitching results in raised points that, in the long muscles of the limbs, only occur at limited locations along the muscle bundles, appearing and disappearing with great irregularity and creating contours similar to those known as “myoïdèmes”. [...] The irregularity of the twitching occurring simultaneously at diverse points of the body is such that it is impossible to determine the frequency at a given time; one would have to be watching all points at once.

[...] These contractions, despite the pronounced raised points they produce under the skin, do not result in any trembling or movement of any part of the body; they disappear where they began, having no useful effect. The patient is still capable of gripping movements and locomotion. Furthermore, the twitching stops or at least significantly decreases during voluntary muscle contraction. [...] There are constant, shooting pains throughout the body, but mainly in the muscles where the twitching occurs. These pains are intense enough to disturb sleep. The patient has slept poorly for three days”. Within a few days, the patient’s condition worsened: “Due to an unexplained agitation, the patient cannot remain in bed for long periods and is constantly getting up. Excessive perspiration. The patient is drenched with sweat; his shirt is as wet as if he had been in water”. In early August, the patient’s condition deteriorated rapidly. He could no longer move about and continued to sweat heavily. He became delirious then went into a coma, dying after less than one month of illness. Morvan described four other, less serious cases in which there was a spontaneously favourable progression. These cases were characterised by “fibrillary muscular contractions” that started in the lower limbs, then “moved upwards, without trembling or movement in any part of the body”. In three of the cases, he noted pains, agitation often associated with insomnia and “generalised excessive perspiration”. Morvan believed that “fibrillary chorea is caused by damage to the anterior horn of the grey matter. [...] Initially limited to the columns of motor cells, fibrillary chorea sometimes extends beyond these; it can be seen to go deeper, reaching the excitation and heart acceleration centres, then arriving at the intermedio-lateral tract, where it affects the vasomotor centre, found at this level according to Pierret” (Antoine-Auguste Pierret [1845–1920], *interne* under Charcot in 1874, spent his career at the Bron asylum). Morvan went on to compare his observations with the symptoms of Sydenham’s chorea, noting that the latter is characterised by uncoordinated movements with an onset that frequently occurs in childhood and is therefore distinct from fibrillary chorea (Coirault, 1946; Morvan, 1890). However, Morvan noted similarities with paramyoclonus multiplex. This term, no longer used today, refers to a pathology described in 1881 in Germany by Nikolaus Friedreich (1825–1882) and analysed by Pierre Marie (1853–1940) in *Progrès Médical* in 1886. When Morvan published his description, a number of similar clinical pictures had been established in Europe: chorea electrica (Henoch-Bergeron or Dubini or Begdie), myoclonus fibrillaris (Kny), myokymia (Schultze) and familial epileptic myoclonus (Unverricht). Edouard Krebs, an *interne* under Joseph Babinski (1857–1932), reviewed and compared these clinical descriptions in his remarkable 1922 thesis, establishing a prescient parallel between Morvan’s description and the agryptic forms of encephalitis lethargica (von Economo, 1931; Krebs, 1922). In 1890, Morvan concluded: “I would be rather inclined to accept that fibrillary chorea, despite certain particularities, is only a variety of Friedreich’s paramyoclonus, the variety without movements, without shifts in any part of the body and with sudatory and vasomotor disturbances in some cases”. Morvan also noted the similarity of these sudatory disturbances with the vasomotor disturbances he described in parietic analgesia, which here he was

willing to assimilate with syringomyelia (Friedreich, 1881; Marie, 1886; Morvan, 1890).

The end of his article was devoted to the pathophysiological cause of the disturbances, but curiously he spent little time on the origin of fibrillary chorea, which he placed in the anterior horn of the spinal cord, instead focusing on the cause of the hyperhidrosis. This led to a discussion on the path of the nerves involved in perspiration and allowed Morvan to cover a cherished theory, on the existence of two sudation tracts, one excitatory and the other inhibitory. Based on his observations of fibrillary chorea, he placed these tracts in the posterior horn of the spinal cord and their efferent pathway in the posterior roots. Morvan's medical training in Paris had placed a strong emphasis on experimental physiology, and it is amusing to note that he wanted to verify his hypothesis. At the very end of his article, he described in rich detail the spinal cord dissection of two 20-year old horses, kept alive under anaesthesia. The first experiment was problematic because the horse was difficult to anaesthetise. Furthermore, as it was winter and Morvan had started late in the day, he didn't have enough light at the end of his procedure. He therefore repeated the experiment with another horse, allowing him to draw conclusions on the sudatory pathways in the spinal cord.. This surprising and somewhat surreal description demonstrates the influence of physiology at the end of the 19th century and its impact on the thinking of the time. Although he worked deep in rural Brittany, Morvan was full of Enlightenment ideas and convinced of the power of experimentation. He persuaded a veterinarian friend named Bergot, who probably shared his ideas, to provide him with horses and experimental materials (Morvan, 1890).

Morvan's publication on fibrillary chorea did not have the same impact as his syringomyelia publication. It was not until 1930 that Pierre Mollaret (1898–1987) and Georges Guillian (1876–1961) presented, for the first time since Morvan, an entirely similar case in *La Revue Neurologique*: "Above all there are fibrillary twitches, some of which are limited, appearing and disappearing in one place and giving the impression, through their incessant combinations, of worm-like writhing". They observed excessive perspiration and mentioned scarlet fever as a possible triggering factor, acting by a mechanism of "toxi-infection" that would be referred to in the years that followed (Mollaret and Guillian, 1930). In 1934, Jean-Albert Chavany (1892–1959) and André Chaignot published an observation of a tuberculous patient who, after having received 1.45 g of gold salts over three months, developed complete insomnia, hyperhidrosis and "numerous muscular twitches" (Chavany and Chaignot, 1934; Dujardin, 1944). Working as a team, Henri Roger and Joseph Alliez in Marseille reviewed 70 cases in 1953, without modifying the clinical picture described by Morvan, and proposed a pathophysiological explanation based on metal intoxication. They drew a parallel with acrodynia from mercury exposure, which had already been described in 1946 by Michel-Pierre Coirault in his thesis (Lambrechts, 1934; Coirault, 1946; Roger et al., 1953; Paris, 1957; Gil et al., 1984; Porot, 1934). However, the pathophysiology remained uncertain until Hyam Isaacs suggested in 1961 that the spontaneous hyperactivity of myofibrils, which he called neuromyotonia (without ever evoking Morvan's syndrome despite its similarity), can be

located at the neuro-muscular junction (Isaacs, 1961). The first complete observation with EMG exploration of sleep and muscular activity was not published until 1974 in Lyon, by Michel Jouvet and his team, who of course were looking for an explanation for agrypnia (Fischer-Perroudon et al., 1974).

Morvan's syndrome, as it is accepted in current international publications, associates all the symptoms presented by the first patient described by Morvan in 1890. The term "fibrillary chorea" has been replaced by "fasciculations" or "myokymia". These phenomena are visible clinically and can be recorded by electromyography. Predominant in the lower limbs, they are associated with cramps, involuntary nervous system disturbances including significant hyperhidrosis of the head and hands, sinus tachycardia, urinary frequency/urgency and impotence. Encephalic symptoms include severe insomnia (agrypnia excitata), hallucinations and disorientation (Cornelius et al., 2011; Lugaresi et al., 2011; Provini et al., 2011). These central signs have common features with limbic encephalitis and call to mind descriptions of von Economo disease. These pathologies are sometimes associated with thymomas (paraneoplastic syndrome) or autoimmune pathologies such as myasthenia (Lüscher and Slesinger, 2010). The pathophysiology, only recently established, seems to be explained by the presence of antibodies that disturb the function of potassium channels (voltage-gated potassium channel antibodies, VGKC-antibodies). They specifically induce prolonged depolarisation, explaining the uncoordinated muscular hypercontractility and the autonomic disturbances. This hypothesis was recently called into question by the demonstration that these antibodies do not bind directly to the ion channel, but to two recently identified proteins that coprecipitate with the ion channel, namely Lgi1 (leucine-rich glioma inactivated 1), a synaptic protein strongly expressed in the hippocampus, and CASPR2 (contactin associated protein 2), which is strongly expressed in the nodes of Ranvier (Irani et al., 2012; Lancaster et al., 2011). It is interesting to note that mutations in the genes coding for these proteins are also found in hereditary forms of epilepsy (Loukaides et al., 2012; Newsom-Davis, 2007; Serratrice et al., 2004; Serratrice and Serratrice, 2011; Vincent et al., 2011). Much work is still needed for an exact understanding of the specific role of these autoantibodies, but there is increased interest in Morvan's syndrome as it is considered a pathological model for neurological diseases involving autoantibodies and provides insight into a number of pathologies affecting the synapse and neuromuscular junction, whether they are caused by genetic, autoimmune or toxic factors. The current developments in molecular neuroscience should not lead us to forget Augustin Morvan, who provided detailed descriptions of the first cases. Not only did he include all clinical signs without omission; he also discussed all physiological and pathophysiological aspects of his observations.

5. Augustin Morvan's other claim to posthumous fame

The hospital in the city of Brest, built between 1937 and 1949, was named Hôpital Augustin Morvan in 1950, in honour of this important clinician. Unfortunately, the name was changed

when the facility became a university hospital centre, and now only one of its units bears Morvan's name.

This discussion of Morvan would not be complete without mention of another recent development. His great granddaughter Colette Destouches, born in Rennes in 1920 and married to Yves Turpin, died on 9 May 2011 at the age of 90. As chance would have it, on 10 August 1919, Louis-Ferdinand Destouches, better known by his pen name Céline, married Edith Follet, the daughter of Marie-Louise Morvan and Anasthase Follet, who was also a physician and would go on to direct the medical school in Rennes. With his father-in-law's help and social aptitude, Céline became a physician by taking an accelerated course of study open to veterans of World War I. He wrote his first book for Colette, his only daughter, entitled *Le petit Mouck* and illustrated by his wife, Morvan's granddaughter, a well-known illustrator for the girl's magazine *La Semaine de Suzette* (Guitton, 2009). Was Céline thinking of Morvan when, in *Voyage au bout de la nuit*, he used the expression "aimable comme un panaris" (as pleasant as a panaris inflammation)?

There is no better tribute to Morvan than the biographical note published anonymously in the *Bulletin de l'Académie de Médecine* on 23 March 1897: "Although he lived in a remote corner of Brittany, outside of all scientific movements, Dr Morvan distinguished himself by his great appreciation for science and associated his name with several discoveries".

Disclosure of interest

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