A history of the description of cerebellar haemorrhage in the 19th century

(History of Neurology)

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Abstract

The history of cerebellar haemorrhage is inseparable from the history of knowledge gradually acquired throughout the 19th century, in both the physiology and semiology of cerebellar pathology. Jean-Baptiste Hillairet (1815–1882) published the first series of cerebellar haemorrhage cases in 1858. At that time, the only diagnostic option was an autopsy. In 1862, Manuel Leven (1831–1912) and Auguste Ollivier (1833–1894) presented the clinical picture of functional cerebellar damage by drawing a parallel between tuberculosis and cerebellar stroke, noting disturbances of balance and gait. In 1864, Alfred Vulpian (1826–1887) and his students explained the inconsistency of hemiplegia, which their predecessors had attributed to cerebellar haemorrhage by the more or less significant compression of the motor pathways within the brainstem; this was caused by the proximity to the cerebellum. It was André Thomas (1867–1963) who, at the turn of the 20th century, established the complete clinical picture of the aetiology and symptomatology of cerebellar diseases. As for, Joseph Babiński (1857–1932) refined the clinical examination, introducing the concepts of asynergy, hypermetria, and adiadocokinesia in 1902. Thus, it took a century of work and hypotheses to understand the physiological functions of the cerebellum.

Keywords

Cerebellum, Cerebellar haemorrhage, Cerebellar syndrome, Jean-Baptiste Hillairet, André Thomas

Introduction

The history of vascular neurology is well documented. However, only the history of haemorrhage and infarct affecting the cerebral lobes is generally studied; cerebellar damage, on the other hand, is only exceptionally mentioned (*Van Gyn, 2023*). We intend to fill in this gap.

From antiquity to the 17th century, the concept of "apoplexy" referred to all aspects of hemiplegia, whether associated with coma, leading in most cases to death.

André Vésale (1514–1564) was the first to describe the anatomy of the cerebellum in 1532 (*Vesalius, 1543*). His work was further developed by Thomas Willis (1621–1675) in 1664 (*Willis, 1664*), Vincenzo Malacarne (1744–1816) in 1776 (*Malacarne, 1776*), then Félix Vicq d'Azyr (1748–1794) in 1786 (*Vicq d'Azyr, 1786*). Vicq d'Azyr suggested that the cerebellum played a functional role in coordinating movements, whereas in 1809 Luigi Rolando (1773–1831) saw in the "little cerebrum" (cerebellum) the origin of all movements (Rolando, 1809). In 1823, Achille-Louis Foville (1799–1878) and Félix Pinel-Grandchamp (1798–1851) concluded their experiments involving limited destruction in animals with the finding that "the cerebellum is the seat of sensation and not the regulator of movement, as M. Flourens has asserted" (*Foville & Pinel-Grandchamp, 1823*). Nonetheless, in 1822, Jean-Pierre Flourens (1794–1867) proved through his vivisection experiments that the cerebellum did not initiate movements or thought; rather, it assured the coordination of motor activities and bodily balance, during rest and in movement, by adjusting posture. In 1825, he collected earlier works presented to various scholarly societies (*Legée, 1974*) in a book (*Flourens, 1824*). In 1826, Étienne Serres (1786–1868) (*Walusinski, 2025*), returning to the theory developed by Franz-Joseph Gall (1758–1828) (*Finger & Eling, 2019*), believed that the cerebellar "hemispheres excite[d] the voluntary movements in the limbs, especially in the pelvic members; the cerebellum stimulates jumping" (*Serres, 1824-1827*). In 1827, Jean-Baptiste Bouillaud (1796–1881) demonstrated the inanity of the propositions of Serres and Gall for whom "the cerebellum is the organ of instinct of propagation (*Bouillaud, 1827*). He concluded: "The lesions of the cerebellum disturb or completely destroy the phenomena of balance or locomotion", then "the cerebellum associates, combines, and coordinates locomotor movements and supplies the animal body with the elements of its balance". In addition to these conclusions, Bouillaud added the hypothesis that the cerebellum possessed a memory of complex sequences of movements. In 1828, François Magendie (1783–1855) (*Magendie, 1828*) and, in 1830, Charles Bell (1774–1842) (*Bell, 1830*) completed the work of Flourens and Bouillaud by showing that the connections between the cerebellum, spinal cord, and muscles are essential to the coordination of movements and balance.

Étienne Serres

Serres considered himself the first, in 1822, to describe intracerebellar haemorrhage (*Serres, 1822*). In reality, Giovanni Battista Morgagni (1662–1771) had already described sudden death revealing bipolar cerebellar haemorrhage in 1765 (*Morgagni, 1765*). Serres analysed around ten observations, some made by the alienist Jean Pierre Falret (1794–1870), establishing deep coma with generalised contraction that could lead to opisthotonos and sometimes hemiplegia. In all cases, he noted a permanent erection persisting even after death (Figure 1). For him, this was a specific symptom of intracerebellar haemorrhage, leading him to tell his students even before opening the skull that it was a case of intracerebellar haematoma. To validate this assertion, he drew on the writings and Parisian lectures of Gall,

who attributed the cause of sexual behaviour to the cerebellum (*Gall, 1823-1825*). In 1827, Bouillaud noticed that apoplexy led to loss of function, meaning it is improbable that it triggers an erection.

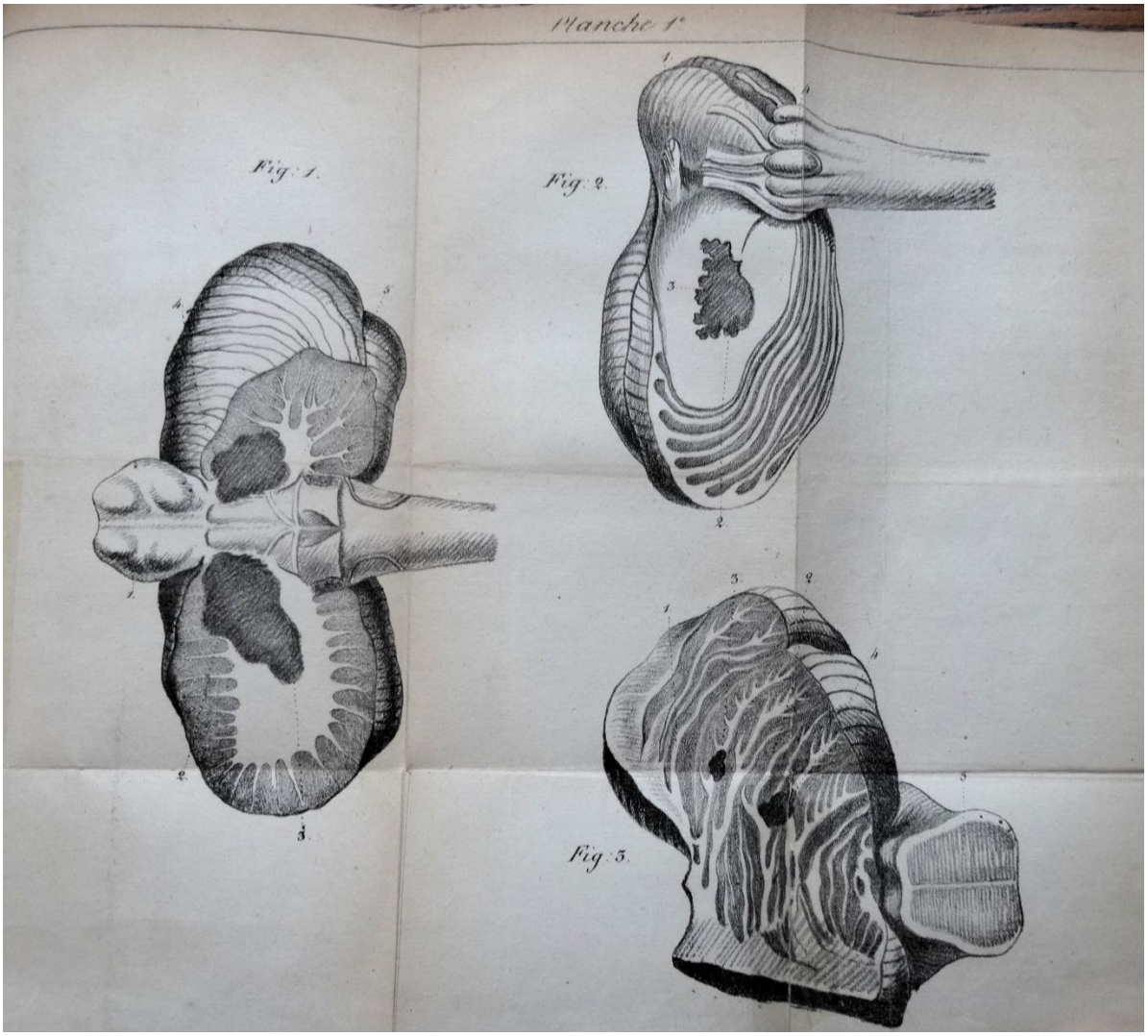

Figure 1. Intracerebellar haematoma observed by Serres in 1822 (*Serres, 1822*) (© BIU Santé).

Following Serres, Félix Cazes (1797–?), in his 1824 thesis, reported a case of mortal stroke in a 74-year-old woman. Her autopsy revealed an isolated lobar haemorrhage in the cerebellum (*Cazes, 1824*). In 1824, Antoine Laurent Jessé Bayle (1799–1858) published an observation of severe dizziness with sudden loss of consciousness, without paralysis or sensory deficit. The autopsy showed haemorrhage had flooded the entire cerebellum. Cazes concluded that this organ did not control motor function or sensory perception, but saw in this an argument in keeping with the theory defended by Flourens (*Bayle, 1824*). In his 1825 thesis, Louis de Droullin (1797–1848) presented the clinical picture of two forms of hemiplegia with contralateral cortical haematoma and ipsilateral lobar haematoma of the cerebellum (de Droulin, 1825). These observations seem similar to those reported by Léon Rostan (1790–1866) in 1823 (*Rostan, 1823*). In 1827, Joseph Théodore Michelet (1801–1869) described in his

thesis, presided by Jean Cruveilhier (1791–1874) (*Michelet, 1827*), the case of a 74-year-old woman with only headaches who suddenly fell victim to "a profound coma" accompanied by "limb palsy". She died in less than two hours. The autopsy revealed that "the left lobe of the cerebellum had become a vast cavity filled with coagulated blood".

Clinical cases in the first half of the 19th century

In 1834, Gabriel Andral (1797–1876) reported six observations of cerebellar haemorrhage (*Andral, 1834*). The episode, which "in no way differed from cerebral haemorrhage in its symptoms", was sudden with the immediate loss of consciousness and deep coma associated with hemiplegia. When he opened the skull, he found a haematoma in the cerebellar lobe contralateral to the hemiplegia. He noted: "We are sure that among the men, none of them had an erection". Among these cases, of note was the observation from the department of Pierre Adolphe Piorry (1794–1879) involving a 75-year-old woman who died following left cerebral softening that took place one year after the onset of incomplete hemiplegia on the right side. The autopsy found recent infarct of the left hemisphere at the level of the centrum semioval of Raymond Vieussens (1641–1715) and the sequelae of a haematoma in the middle of the cerebellum (*Piorry, 1829*). Andral also counted thirteen observations of cerebellar softening which he broke down into uni-lobal, bi-lobal, or medial. Curiously, hemiplegia was the only deficient symptom described. In no case did Andral report movement disorder suggesting cerebellar syndrome. In Germany from 1834 to 1864, work entirely comparable to that of Rudolf Wagner (1805–1864) led to the same finding as that of Andral, associating cerebellar haemorrhage and hemiplegia (*Wagner, 1846*).

In 1836, Auguste Duplay (1805–1872) proposed five observations of hemiplegia where the autopsy showed the absence of hemispheric lesion, but to either cerebellar haematoma contralateral to paralysis or tuberculous abscess lacerating one of the cerebellar hemispheres (*Duplay, 1836*). In 1849, the Englishman Robert Dunn (1799–1877) published an observation of "apoplexy of the cerebellum". It described gait instability that made the patient look like a drunk man; there was also pronounced somnolence accompanied by violent occipital headaches that made him aggressive. During the autopsy, Dunn discovered a haematoma in the right lobe of the cerebellum and signs of left cortical softening with highly calcified vertebral arteries and basilar trunk (*Dunn, 1849, 1850*). It was the first case where the patient was not immediately comatose and presented ataxia.

What is original about the *Traité du système nerveux* (manual on the nervous system) by Jean-Baptiste Sarlandière (1787–1838) (*Walusinski, 2025*), published posthumously in 1840, is that it brings together physiology, still quite rudimentary, along with comparative anatomy and embryology encompassing invertebrates up to mammals. Much inspired by Serres's vivisection experiments, Sarlandière developed a thalamic and cerebellar test of motor function. He proposed an initial conception of cerebellar syndrome in his chapter entitled "The functional coordination and agreement of the nervous system" in the section "interrelation of voluntary and involuntary movement". After a cerebellar lesion, "balance was overthrown or the movements were not correctly balanced [...]. The directions are heeded, intended, and indicated, but the indication is in no way followed by ineptitude; rather, it is through a lack of cohesion in these living instruments used for execution. Some of them were more or less weak or failed entirely in their mission, while others, lacking contrast, are not properly balanced; hence these abrupt, jerky, or overly vigorous movements, lacking in measure and precision" (*Sarlandière, 1840*).

Jean-Baptiste Hillairet and the first comprehensive review in 1852

In 1852, Jean-Baptiste Hillairet (1815–1882) published the first comprehensive review of the knowledge acquired on cerebellar haemorrhages (*Hillairet, 1858*). The son of the chemist Jean Nicolas Hillairet (1787–1868) and Marguerite Gaury (1797–1882), he was born on 14 December 1815 in the city of Angoulême (Charente region). He studied medicine in Paris, becoming an extern in the French hospital system in 1838, and becoming Bouillaud's senior registrar in 1858. He was appointed as a doctor at the Central Office in 1852, and became senior register at the Hospice des Incurables (for men; becoming Laënnec Hospital in 1854). Finally, he was appointed to work at Saint-Louis Hospital in 1859, where he remained until his retirement in 1881 (*Fiche de carrière, 2025*). Georges Gilles de la Tourette (1857–1904) was his extern in 1880. Hillairet was elected to the Academy of Medicine on 13 July 1875 (Figure 2). He died in Paris on 14 September 1882.

Figure 2. Bust of JB. Hillairet by Eugène Lorrain (1857–1923) (Academy of Medicine).

Hillairet started his article published in 1858 with a question: "Can cerebellar apoplexy be cured as cerebral apoplexy can?" By responding without any doubt, he explained the diagnostic difficulty involved. Patients who seemed cured were considered to have cerebral damage

because doctors were unaware of cerebellar damage, which could only be revealed by autopsy. He thus proposed setting up a clinic specifically for previously unknown cerebellar conditions.

For him, "cerebellar haemorrhage is one of the diseases for which the symptomatology is the least understood". He considered that for one case of cerebellar haemorrhage there were thirty cases of cerebral haemorrhage and regretted that the observations collected by Serres "were not complete or detailed enough to meet the scientific demands of our era". He noted that "the works of Rostan, Rochoux (*Rochoux, 1814*), Abercrombie (*Abercrombie, 1835*) and Lallemand (*Lallemand, 1830*) show the facts concerning cerebellar haemorrhage and other conditions of the cerebellum, but without any special description". But how could cerebral haemorrhage be distinguished from cerebellar haemorrhage during the patient's lifetime? Several symptoms were the same: "general headache, dizziness, sensitivity to light, ringing in the ears, hemiplegia, etc.". The "violent" and immediately fatal onset was, for him, specific to cerebellar haemorrhage. It is rare to see a form rapidly leading to death. He considered the spontaneous, uncontrollable vomiting to be specific to cerebellar damage "or to any other lesion that can increase the cerebellum's volume". The concept of intracranial hypertension was unknown at the time. Hillairet attributed this vomiting to compression of the vagus nerve. When the patient did not immediately fall into a coma, "contact not yet lost" and "limb weakness was also a habitual symptom of cerebellar apoplexy". When there was paralysis, it was always cross-sided.

In a single observation, "a patient complained of being pushed by an irresistible force on the left side". He never noted "the tendency to move backward and to rotate" observed by experimenters using animal vivisections, such as Flourens or Bouillaud. Sensation remained intact, sensory organs seemed unaffected, there were no convulsions. Survival was a day and a half on average. Hillairet never proposed a pathophysiology or an aetiology for cerebellar haemorrhage, but he did distinguish it from "softening"; that is, infarct.

Hillairet wrote this work when he applied to join the Medical Society of the Hospitals in Paris. His assessors—Jean-Baptiste Barth (1806–1877), Léon-Julien Gros (1824–1875), and Charles-François Léger (1813–?)—concluded that "this work is worthy of praise because in certain cases it will help elucidate the diagnosis, so often unclear and uncertain, of diseases of the brain" (*Léger, 1858*). This work made Hillairet a member of the learned society.

Filippo Lussana, Thomas Edouard Turner, and Charles Poelman

In 1851 (*Lussana, 1851*) and 1852 (*Lussana, 1852*), the Italian researcher Fillipo Lussana (1820–1897) repeated Flourens's vivisection experiments but with a more precise method of selective excision in order to ground the theory of motor coordination in validated anatomy and ensure a longer lifetime for the animals. He contested the idea of a single "coordination organ" and suggested a role in the regulation of perceived from bodily positions (what would become proprioception) and balance. He published a summary of his research, including the cerebellar activity within cerebral physiology in 1885, noting that the cerebellum did not directly control movements, instead regulating their precision and coordination (*Lussana, 1885*). He identified three major disturbances after partial removal of the cerebellum: weakness which he called asthenia, atonia, or astasia. This triad would become "Luciani's cerebellar syndrome" as noted by Jules Dejerine (1849–1917) in his *Sémiologie des affections du système nerveux* (semiology of conditions of the nervous system), in 1914, when he described the semiology of centrally caused ataxia.

On 05 January 1856, Thomas Edouard Turner (1829–1892) defended his thesis, presided by Cruveilhier and covering the atrophy of the cerebellum (*Turner, 1856*). To prepare his thesis, he assisted by "my learned friend, M. the Dr. Charcot". At that time, Charcot was his chief resident. Turner did not discuss cerebellar haemorrhage per se but confirmed Hillairet's observation of cross-sided damage in the cerebellar and cerebral hemispheres. He added partial and consecutive atrophy "of the anterior pyramid and cerebral peduncle" and of the spinal cord in cases of lesions developing before birth or shortly thereafter, allowing survival by manifestation of neonatal hemiplegia. For this purpose, he drew on the work of Jacques-Étienne Belhomme (1800–1880), notably his 1824 thesis (*Belhomme, 1824*), Jean-Baptiste Cazauvieilh (1802–1849) and his 1827 thesis (*Cazauvieilh, 1827*), Rudolph Virchow (1821–1902) and his thesis which Jean-Martin Charcot translated for him (*Virchow, 1854*). He also had his personal clinical cases.

At the session of the French Academy of Sciences on 30 November 1860, Flourens read a letter sent by Charles Poelman (1815–1874), professor of physiology in Ghent in Belgium. In it, Poelman described a dog with involuntary circular movements. Its autopsy showed calcifications in the cerebellum and the middle cerebellar peduncles. He deduced a diagnostic approach: "The loss of contact indicates the location of the apoplexy in the brain strictly speaking; the loss of balance in locomotion indicates the seat of the apoplexy within the cerebellum; and the sudden death indicates the seat of the vital centre" (*Flourens, 1860*); that is, the medulla oblongata.

Manuel Leven, Auguste Ollivier, Alfred Vulpian, Jules Cotard, Jean-Louis Prévost, and others

In 1862, Manuel Leven (1831–1912) and Auguste Ollivier (1833–1894) published the results of vivisection experiments on guinea pigs. They performed punctures of the cerebellum. They added seventy-six clinical observations including tuberculosis in children and cerebellar haemorrhage in adults. They concluded there was no decline in cognitive function and no sensory deficit, while there were "motor disturbances", which they listed: intermittent hemiplegia, difficult or impossible to maintain upright position, difficulties walking, "wobbling of the head, swaying movements, irresistible tendency to lean to one side, speech difficulties", and convulsions (*Leven, 1862*).

Impressed by the data in Gustave Lanoix's1863 thesis, covering cross-sided hemiplegia during cerebellar haemorrhage (*Lanoix, 1863*), Alfred Vulpian (1826–1887) devoted, in 1864, one of his lessons at the Natural History Museum to the physiology of the cerebellum (*Vulpian, 1864*). After indicating the large number of observations in which a lesion of one lobe of the cerebellum was followed by cross-sided hemiplegia, he dared to say the following: "I have no hesitation in saying that it may seem reckless to dare to challenge such a body of evidence. Indeed! I have no hesitation in saying that the observations cited are far from having the value to which we attribute to them, and I am led to a conclusion very different from that which is accepted in the sciences". He then offered his interpretation: following damage to a cerebellar lobe, there may be ipsilateral hemiplegia, as well as cases without ipsilateral or contralateral paralysis. Intracerebellar haemorrhage or tumours cause an increase in the volume that compresses the corresponding side of the medulla and pons, resulting in crossed-sided hemiplegia. When compression is limited or absent, the hemiplegia is incomplete and direct or absent.

In 1867, the future politician Léon-Théophile Raymond (1844–1931), then a house officer under Jules Baillarger (1809–1890) at La Salpêtrière, presented a paper to the French Anatomical Society. A 73-year-old woman with dementia suddenly lost consciousness and vomited profusely; no sign of hemiplegia. She died after three days in a coma. Upon autopsy, Raymond observed: "In the middle and slightly to the right of the posterior margin of the cerebellum, a jagged opening is visible, leading to a haemorrhagic focus occupying almost the entire portion of the brain, but particularly the right hemisphere […]. A large number of aneurysms were found along the course of the posterior and superior cerebellar arteries". His description showed miliary aneurysms exclusively in the cerebellar arteries and advanced atherosclerosis in all the cerebral arteries (*Raymond, 1867*).

In his 1868 thesis, Jean-Louis Prévost (1838–1927) reported a case of cerebellar haemorrhage (*Prévost, 1868*). After initially losing consciousness, the 70-year-old man complained only of a headache and feeling "dazed" and had no paralysis. But within a few hours, he fell into a coma with ocular deviation to the right, then died. The autopsy revealed a brain with no macroscopic lesions and a haematoma in the left lobe of the cerebellum that extended to the pons and medulla. This case was in keeping with the interpretation developed by his teacher, Vulpian.

Jules Cotard (1840–1877) defended his thesis on 31 July 1868. Charcot was a member of the jury as the assistant professor (*Cotard, 1868*). In his study of congenital or acquired brain atrophy, he reached the same conclusions as Turner. In the majority of cases, atrophy of one cerebral lobe is associated with contralateral lobar atrophy of the cerebellum. This formed the basis of the concept of cross-sided cerebellar diaschisis, introduced by Konstantin von Monakov in 1914 (1914), and now visualised using the modern imaging techniques employed by Marie-Germaine Bousser's team (*Baron, 1981*).

Lockhardt Clarke (1817–1880), a British neuroanatomist and physiologist, reported in 1867 two cases of hemiplegia accompanied by aphasia which had developed in several episodes and involved cortical lesions and sequelae of cerebellar haemorrhage: "We can therefore say, based on current scientific knowledge, that the olives are the centres responsible for coordinating the movements necessary for articulate speech" (*Clarke, 1869*).

In 1878, Edward MacKey of Brighton, referring to John Hughlings Jackson (1835–1911) who said he had never observed this, reported an observation of lobar haemorrhage in the cerebellum. The patient, suddenly struck with headache and vomiting, went blind. When he tried to walk, he circled in place and could not move forward. He fell into a coma the following night in his sleep. KacKey observed hemiplegia. The autopsy revealed only a lobar haematoma in the cerebellum (*Mackey, 1878*). In 1894, James Samuel Risien-Russell (1863-1939) advanced physiological research by replacing invasive surgical procedures with localized electrical stimulation (*Risen-Russell, 1894*). In 1895, he also described the phenomena of postural recovery and compensation, characteristic of cerebellar activity (*Risien-Russell, 1895*).

André Thomas

André Thomas (1867–1963) defended his dissertation which focused on the cerebellum, on 19 November 1897, presided by Charles Richet (1850–1935) (*Thomas, 1897*). It was the most comprehensive work of the late 19th century, covering histology, physiology, and clinical practice alike. It included a lengthy chapter on the history of vivisection experiments carried out since the 18th century by authors from various European countries, to which he added his

own experiments and with which he drew comparisons. In it, he outlined the still incomplete clinical picture of cerebellar syndrome. André Thomas focused his observations on cases of neonatal cerebellar atrophy, given that vascular, infectious, or tumour-related lesions were never confined solely to the cerebellum but also involved the brainstem, thereby preventing the observer from making a specific diagnosis of cerebellar disorders. This view is illustrated by his 1905 publication in the *Revue neurologique* of a case of an old bulbar ischaemic stroke with progressive worsening of hemiplegia, accompanied by a cerebellar syndrome and trigeminal nerve involvement. The autopsy revealed the sequelae of cerebellar stroke and histological lesions characteristic of multiple sclerosis (Figure 3). It should be noted that the patient had been examined by Charcot before being treated by Dejerine and André Thomas at Bicêtre (*Thomas, 1905*).

Figure 3. Illustration of André Thomas's observation, published in 1905 (OW Collection).

André Thomas was interested in the cerebellum throughout his career. In 1911, he published a book aimed at general practitioners, *La fonction cérébelleuse* (cerebellar function) (*Thomas, 1911*). He wrote, "very little is known about the clinical symptomatology of haemorrhagic lesions or areas of softening in the cerebellum". These findings were identified during the autopsy; they had not been recognised during the patient's lifetime due to the peculiar symptomatology. As he pointed out, the case report published by Vasil Negel (1854–1923) and Anibal Théohari (1873–1933) in 1903 (*Negel, 1903*) is one of the earliest examples of cerebellar softening for which a diagnosis was made while she was still alive. The patient suddenly developed isolated cerebellar syndrome, characterised by motor asynergy and adiadokinesia—a condition recently described by Joseph Babiński (1857-1932)—along with intentional tremor and speech articulation disorders.

In 1918, André Thomas published a book on injuries to the cerebellum observed during the war, noting how rare they were. Only three soldiers were treated at the Dejerine hospital for injuries sustained during the hostilities. this rarity was attributed to the concomitant involvement of the brainstem, which was rapidly fatal. He added, "the similarity between the symptoms observed in our wounded patients and in the animals on which we conducted experiments during our studies on the physiology of the cerebellum is striking" (*Thomas, 1918*). In 1925, André Thomas wrote a major chapter on the physiology and pathology of the cerebellum in the *Nouveau Traité de Médecine et de Thérapeutique* (new manual on medicine and therapy) by Henri Roger (1860–1946), Fernand Widal (1862–1929), and Pierre Tessier (1864–1932) (1925). He described the arterial circulation of the cerebellum in detail, after which he devoted a paragraph to cerebellar haemorrhage, "which [was] much rarer than cerebral haemorrhage". The symptoms he associated with this condition were severe occipital headache accompanied by vomiting, dizziness, with nystagmus, and then, in the absence of coma, a more or less complete cerebellar syndrome with preserved cognitive function and no true paralysis of the limbs.

In London, the physiologist and neurosurgeon Victor Alexander Haden Horsley (1857–1916) and the neuroanatomist Robert Henry Clarke (1850–1926) contributed to advancing our understanding of the physiology of the cerebellum by using electrostimulation techniques (*Horsley, 1908*). In a seminal article published in *Brain* in 1908, they described a stereotaxic apparatus capable of locating and stimulating specific areas of the brain and cerebellum in animals using three spatial coordinates. Their instrument was the predecessor of those used in modern stereotactic surgery (*Fodstad, 1991*).

Joseph Babiński

Babiński's research into disorders of the cerebellum and the labyrinth was carried out at the turn of the century: "Cerebellar ataxia, a poorly defined condition whose clinical features, traditionally attributed in standard texts to disorders of the cerebellum, are also characteristic of disorders of the vestibular system" (*Babiński, 1913*). In order to clarify the clinical symptoms, Babiński, between 1899 and 1913, conducted a lengthy and repeated examination of his famous patient Henri Mouninou (1864–1920) (*Babiński, 1909*). From this, he deduced a set of symptoms specific to the cerebellum: asynergy in 1899 (*Babiński, 1899, 1906*), cerebellar catalepsy (or spontaneous postural rigidity) in 1899 (*Babiński, 1899*), adiadocokinesia in 1902 (*Babiński,1902*), and hypothermia in 1913 (*Babiński, 1913*) (Figures 4 and 5).

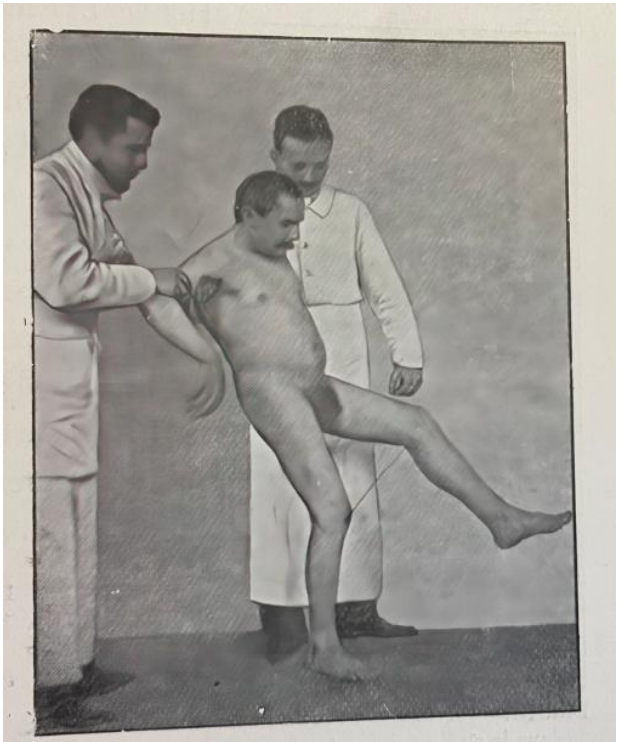

Fig. 15. — Attitude du malade pendant la marche, soutenu par deux aides.

Figure 4. Asynergy and hypermetry by H. Mouninou (OW Collection).

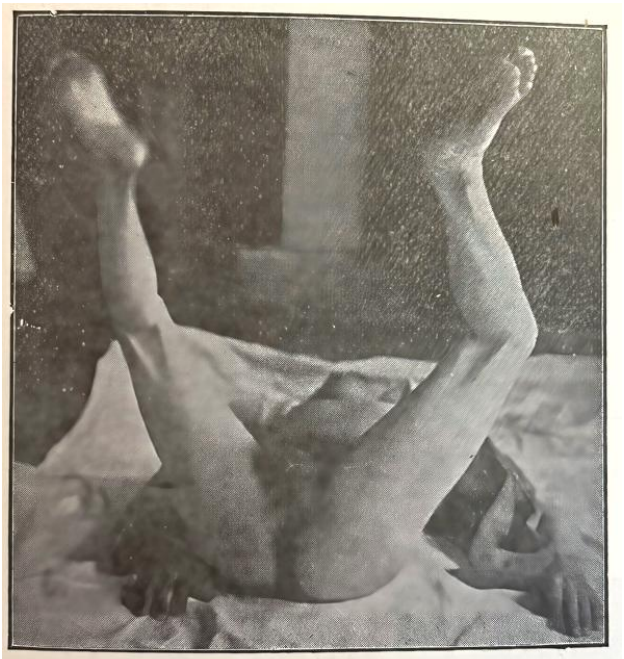

Fig. 19. — Catalepsie cérébelleuse. Pose de 15 secondes.

Figure 5. H. Mouninou's cerebellar catalepsy (OW Collection).

Conclusion

It is impossible to separate the history of cerebellar haemorrhage from the history of our knowledge, gradually acquired in both the physiology and clinical semiology of cerebellar disease. Physiologists first attempted to elucidate the physiological role of the cerebellum by performing partial resections on animals. Until the mid-19th century, a diagnosis of cerebellar haemorrhage was never made while the patient was still alive; rather, it was solely an unexpected finding at autopsy following a brief, sudden coma, attributed to "apoplexy". In 1840, Sarlandière was the first to report certain symptoms of cerebellar syndrome—such as hypermetria and asynergy, though he did not use those terms—which he observed during the brief period of survival following a stroke.

Hillairet remains known for having been a pioneer in 1852 when he published an article devoted entirely to cerebellar haemorrhages. In this series of cases, the diagnosis was, once again, based solely on autopsy findings. The clinical approach he envisaged remained rudimentary, modelled on crude vivisection experiments. For some thirty years, various authors in Europe agreed on a clinical picture characterised by sudden-onset, severe occipital headaches accompanied by vomiting, balance disorders, and movement disorders. Only after 1860 were authors starting to supplement their autopsy descriptions by noting the calcification of the cerebral arteries, and in some cases the presence of microaneurysms in these arteries or cardiac valve disease. The distinction between haemorrhage and cerebellar infarct was therefore never specified.

In the mid–1860s, Vulpian clearly criticised the work of his predecessors, demonstrating that the intermittent hemiplegia previously considered to be cerebellar in origin was merely evidence of localised damage to the descending motor pathways at the level of the brainstem. The related compression was due to the expansion of the cerebellar lobe caused by the haematoma or haemorrhage spreading to the brainstem.

At the turn of the century, André Thomas laid the foundation for the clinical understanding of cerebellar syndrome, which was later expanded upon and refined by Babiński. The misnamed cerebellar ataxia became asynergia accompanied by hypermetria, adiadokokinesia, action tremor, and "stuttered speech". It took a century of vivisection and autopsies to establish the clinical picture of cerebellar syndrome in living patients before modern imaging techniques made it possible to determine whether the cause was haemorrhagic, ischaemic, tumour-related, or infectious (*Wittwer, 2019*).

The distinction between cerebellar haematoma and cerebellar infarct was not recognised until the early 20th century. In general terms, cerebellar haemorrhage presents with a sudden onset and symptoms that are immediately at their peak, characterised by extremely severe, "thunderclap" headaches, early vomiting accompanied by impaired consciousness, and progressing from drowsiness to a coma within a short period of time. A cerebellar infarct usually begins gradually, with little or no headache. Vomiting, which is rare at first, may occur later alongside impaired consciousness. Cerebellar syndrome and dizziness accompanied by nystagmus are clinical indicators of cerebellar dysfunction. Identifying the affected territory remains a clinical challenge. Dysarthria may indicate thrombosis of the superior cerebellar artery, while dizziness and lateral drift may indicate posterior-inferior cerebellar involvement. Audio-vestibular impairment is characteristic of a lesion in the territory supplied by the anterior inferior artery. The main cause of this is heart infarct embolism (*Mano, 2022*).

The first cases of cerebellar haemorrhage of venous origin were identified by Otto Binswanger (1852–1929) in 1902 (*Binswanger, 1902*) and Heinrich Obersteiner (1847–1922) in 1903

(*Obersteiner, 1903*). In 1938, the American neurosurgeon Walter Dandy (1886–1946) identified the causes. Cerebellar lesions are secondary either to thrombophlebitis of the lateral sinus of otogenic origin (otitis, mastoiditis), or to thrombosis of the cerebellar cortical veins, whether or not secondary to thrombosis of the transverse sinus (*Dandy, 1938*).

Even in the 21st century, cerebellar haemorrhages still carry a very poor prognosis.

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