REVUE NEUROLOGIQUE XXX (2024) XXX–XXX

Available online at
ScienceDirect
www.sciencedirect.com

Elsevier Masson France
EM|consulte
www.em-consulte.com

Lessons from the past

The neurologist Henri Hallopeau (1842–1919), a famous dermatologist

O. Walusinski

Lauréat de l'Académie nationale de Médecine

INFO ARTICLE

Historique de l'article :
Reçu le 26 février 2023
Reçu sous la forme révisée le 29 août 2023
Accepté le 10 octobre 2023

The name François Henri Hallopeau remains famous in our day, associated with his numerous novel contributions to dermatology. However, as a student of Alfred Vulpian (1826–1887), he began his career with works in neurology, mostly neglected today. After a brief biographical note, we will review the tribute he paid to neurology.

1. Biographical overview

Henri Hallopeau (Fig. 1–2) was born in Paris on 17th January 1842, the son of François Hallopeau (1811–1879), ''*lieutenant principal des douanes*'' (customs officer), and Adélaïde Rossignol (1810–1886), from the Belgian city of Ostend [1]. After brilliant secondary studies at the prestigious Lycée Bonaparte (later named Lycée Condorcet), Hallopeau, who in 1857 ranked first on the competitive exam marking the end of his secondary education, enrolled in the Paris Medical School in 1858. After passing the Paris Hospitals exam for non-resident students in 1863, he completed several internships in the department of Alfred Louis Philippe Hardy (1811–1893) at Saint-Louis Hospital, then under the surgeon Gabriel Édouard Cusco (1819–1894) at Lariboisière Hospital. He went on to rank second on the competitive residency exam on 28 December 1866. He was successively a resident under Nathan Oulmont (1815–1884), Auguste Millard (1830–1915), Sigismond Jaccoud (1830–1913), and finally Vulpian. He then defended his thesis on 30th June 1871: *Des accidents convulsifs dans les maladies de la moelle épinière* (convulsive seizures in spinal cord diseases) [2]. After failing to pass the *agrégation* exam (which opens the way to academia) in 1875, in 1878 he succeeded with his thesis entitled *Du mercure, action physiologique et thérapeutique* (mercury, physiological and therapeutic action) [3]. Hallopeau was then employed by the Central Office (Paris Hospitals physician) in 1877. Before replacing Vulpian for his experimental pathology class in 1884, from 1883 to 1886 he was the assistant instructor of pathology classes focused on nervous system diseases. He was successively a chief physician at Tenon Hospital in 1880, at Saint-Antoine Hospital from 1881 to 1883, then at Saint-Louis Hospital from 1884 until he retired in 1907.

It was only in 1884, when he was over forty, that he discovered dermatology and became a well-known dermatologist and syphilographer, lending his name to at least four syndromes, leading to his posthumous fame: pyoderma vegetans of Hallopeau (1 to 2% of forms of pemphigus), pemphigus vegetans of Hallopeau, acrodermatitis continua of Hallopeau (pustular psoriasis at the extremities of the fingers and toes), and a genetic disease, dystrophic epidermolysis bullosa (Hallopeau-Siemens syndrome) [4]. According to his biographer, Gaston Milian (1871–1945), writing in 1919, the most important thing to remember about Hallopeau was ''his perseverance and his enthusiasm for therapeutic research on

https://doi.org/10.1016/j.neurol.2023.10.017

Please cite this article in press as: Walusinski O. The neurologist Henri Hallopeau (1842–1919), a famous dermatologist. Revue neurologique (2024), https://doi.org/10.1016/j.neurol.2023.10.017

Fig. 1 – Henri Hallopeau around 1880 (OW Collection).

syphilis''. Atoxyl, arsactin, and hectine are treatments now forgotten, supplanted by penicillin, which is much more effective. Hallopeau can be credited with the first occurrence of the word ''antibiotic'' in 1871, which he used to describe a substance that worked against vital forces [5]. In 1889, he coined the term ''trichotillomanie'', now called ''hair pulling disorder'' in English.

Although Hallopeau was known for his kindness and cheerfulness, his soft voice which did not carry far and his legendary myopia made him the target of mockery by his colleagues and students. He also failed to obtain the Cutaneous and Syphilitic Diseases Chair in 1902, which he lost to Ernest Gaucher (1854–1919), who took over from Jean-Alfred Fournier (1832–1914). Hallopeau became a member of the French Academy of Medicine in 1893 and was also a member of numerous other learned societies. He was a founding member of the French Society of Dermatology and Syphilography. In 1906, he was made an Officer of the French Legion of Honour [6].

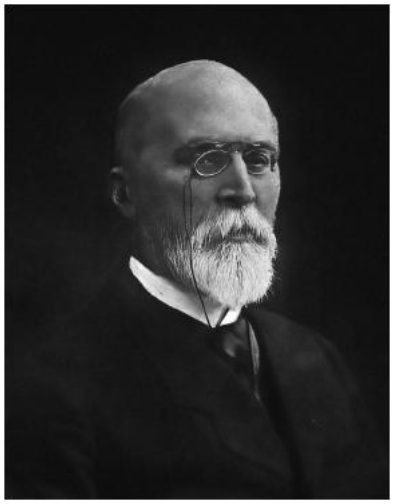

Fig. 2 – Henri Hallopeau around 1900 (OW Collection).

In 1873, Hallopeau married Alice Nicolas de Meissas (1849–1910). They had one daughter and one son; the latter, Paul Hallopeau (1876–1924), would become a famous surgeon in his day in the Paris Hospitals. After going blind, Hallopeau died at his home in Paris on 19th March 1919, shortly after the first session of the French Society of Dermatology after the war, which he insisted on attending [7].

2. First steps at the French Society of Anatomy (1867)

Hallopeau made a presentation in February of 1867, then in August, at the French Society of Anatomy. At the time, he was a resident under Cusco. He described coma with ocular deviation, secondary to skull fracture, complicated by cerebral haemorrhage due to a lesion in a branch of the middle meningeal artery [8]. He also presented the case of a cranial bullet wound that caused no disturbances for more than two months, the patient dying secondarily during febrile coma. During the autopsy, an intracerebral abscess was found, in contact with the corpus callosum [9]. He took the opportunity his presentation afforded him to demonstrate the accuracy of his clinical observations at the bedside of his patients.

3. First steps at the French Society of Biology (1869)

When he was still a resident, on 07 August 1869 Hallopeau published his first presentation to the French Society of Biology, presided by Claude Bernard (1818–1878). ''There is a form of myelitis, not yet described, in which the lesions mainly affect the ependyma and the conjunctive tissue of the commissures'' [10]. It is, in fact, quite complex to understand which pathology he was referring to. His single observation is confusing. The young female patient had action tremor, non-systematised muscular atrophy in the limbs, and neuralgic pain. The autopsy of other patients, not described, supposedly revealed abnormal cavities in the spinal cord. Hallopeau did not indicate symptoms of sensory deficits. Was he describing cases of syringomyelia? It was not until the period between 1883 and 1890 that Augustin Morvan (1819–1897) would establish the precise semiology of this disease [11].

4. Doctoral thesis (1871)

Presided by Vulpian on 30th June 1871, Hallopeau's thesis jury included Alexandre Axenfeld (1825–1876) and the associate professors Auguste Letort (1846–?) and Louis Grimaux (1835–1900). Hallopeau dedicated his work to Guillaume Duchenne de Boulogne (1806–1875) and Jean-Martin Charcot (1825–1893), having attended some of the latter's lessons, proof of his first calling as a neurologist.

Hallopeau started by noting that he had been inspired by the works of Charles-Édouard Brown-Séquard (1817–1894) published in 1856: *Recherches expérimentales sur la production*

Please cite this article in press as: Walusinski O. The neurologist Henri Hallopeau (1842–1919), a famous dermatologist. Revue neurologique (2024), https://doi.org/10.1016/j.neurol.2023.10.017

d'une affection convulsive épileptiforme à la suite de lésions de la moelle épinière (experimental research on the production of an epileptoid convulsive affection following spinal cord lesions) [12]. As Brown-Séquard explained, ''In 1850, I found that some spinal cord lesions in mammals are followed by an epileptoid convulsive affection after a period of a few weeks''. Three or four weeks after spinal hemi-section, he observed seizures in non-paralysed parts of the body, including the face. Sometimes spontaneous, they could also be caused by ipsilateral stimuli applied to the skin, notably on the face, and also by asphyxia: ''The convulsive affection produced by certain injuries to the spinal cord is true epilepsy, or at least an epileptoid affection... The greatest analogy exists between what we know of the aura epileptica in man and what I have found concerning the property that the skin of the face possesses of producing fits in my animals. In them, it seems that the face is the starting point of a true aura epileptica and that, as well as in man, an interruption of nervous transmission between the starting point of the aura and the central nervous system, seems to cure epilepsy'' [13]. The notion of spinal cord epilepsy had, in fact, been introduced in 1824 by Pierre Flourens (1794–1867) who adhered to the theory of non-irritability of the brain and cerebellum [14], which was adopted by Brown-Séquard and would be accepted until the beginning of the 20th century [15]. We should note that the Englishman Marshall Hall (1790–1857) had always opposed this theory [16].

Hallopeau asked the following question: ''In what condition is the excitatory-motor power of the spinal cord heightened? It constantly increases when the spinal cord is separated from the brain by transversal section. Inversely, it decreases when certain regions of the brain are stimulated''. Evaluating the results of vivisection by German authors and Vulpian, he added factors that increased the excitatory-motor power of the spine: ''the modification of the quality of the blood therein or when the blood is prevented from flowing in sufficient quantities; when it is subjected to the influence of certain poisons; when its tissue is directly irritated by a traumatic lesion, by contact with certain substances, or by galvanism; and when it causes, through prolonged irritation of centrifugal or centripetal nerves, a modification that is probably irritative in the constitution of the grey matter''. Hallopeau then transposed these experimental results on human pathology. For example, he considered tetanus to be a traumatic ''centripetal'' irritation of a peripheral nerve responsible for contraction. Then for ''centrifugal irritation'', he used the example of convulsions in the limbs of hemiplegics, which he attributed to ''areas of disintegration and lacunae in the anterior horns'' of the spinal cord. He then referred to one of Charcot's lessons, unpublished and dating from 1870. Charcot, according to Hallopeau, ''categorises these phenomena among the symptoms characteristic of this myelitis, which is limited to a segment of the organ but concerns it in its totality and intercepts all communication between the part of the organ situated below and the brain''. He also referred to compression due to a spinal tumour, an infection, or trauma. His presentation is very confusing. He likens choreic movements, tremor in multiple sclerosis, cramps, and spinal tremor during dorsiflexion in the foot described by Charcot, to spinal epileptic seizures. These assertions in his thesis were accepted by his teacher and jury president, Vulpian, because at that time, all authors accepted the theory of spinal cord epilepsy.

5. Studies on diffuse chronic myelitis (1871–1872)

Inspired by Vulpian, Hallopeau distinguished ''systematic myelitis'' – that is, locomotor ataxia – and ''primitive and secondary sclerosis in the lateral tracts as well as tephromyelitis [from the Greek τεφροζ (tephros), meaning ash-grey and referring to the colour of spinal grey matter] in infantile paralysis and progressive muscular atrophy'', from multiple sclerosis and a new category of lesions affecting the white matter and the grey matter, for which ''it would be pointless to search for a general description in the literature; the author has thus attempted to make up for this shortcoming'' [17]. To support the distinction he proposed, he referred to the book of Rudolf Virchow (1821–1902) [18] who distinguished between damage to nervous components (currently neurons and their axons) and damage to interstitial tissue (currently glia). At a time when the pathogenesis of various pathologies was still poorly understood, this was a complex task. Alongside tabes and multiple sclerosis, Hallopeau attempted ''a special description of peripheral myelitis (meningomyelitis) and myelitis that develops in the connective tissue of the commissures and that we have described under the name of periependymary diffuse sclerosis'' [19]. He endeavoured to describe an initial inflammatory phase progressing to a localised or global sclerous degeneration. Intense spinal pain as well as paralysis and anaesthesia in the limbs and trunk characterised the clinical symptomatology. Progression was variable, either rapidly leading to death, or with remissions without regression but always with a fatal outcome. His descriptions are confusing and it is now quite difficult to recognise the pathologies he refers to. His French is also quite verbose. He probably mixed infectious diseases of the meninges and the spinal cord (tuberculous meningomyelitis, etc.) and inflammatory diseases (sarcoidosis, connectivitis-vascularitis) with forms of myelitis such as multiple sclerosis, tabes, etc.

6. 1875 *agrégation* thesis

At the *agrégation* competitive exam on 15th March 1875, Hallopeau presented a thesis entitled *Des paralysies bulbaires* (bulbar paralysis) [20]. In the absence of documents explaining why the jury did not pass Hallopeau, we do not know the quality of the oral component of this competitive exam, which counted more than the thesis. His thesis outlined the advances in the understanding of brainstem-crossed syndromes over the last quarter of a century preceding the depiction by Adolf Wallenberg (1862–1949) in 1895 [21]. To sum up, Hallopeau did not quote the observation made by Alexandre Marcet (1770–1822) in 1811 [22], but the first thesis mentioning damage to the pons, by Pierre-Henri Josias (1825–1895) in 1851 , followed by the works of Jean-Marie Philippeaux (1809–1892) and Vulpian, describing in 1853 the origin of several cranial nerves [23]; of Adolphe Gubler (1821–1879) reporting on

Please cite this article in press as: Walusinski O. The neurologist Henri Hallopeau (1842–1919), a famous dermatologist. Revue neurologique (2024), https://doi.org/10.1016/j.neurol.2023.10.017

alternating hemiplegia in 1856 [24] and 1858 [25]; of Achille Louis Foville or Defoville (1831–1887) localising lateral gaze paralysis in 1858 [26]; of Duchenne de Boulogne who published his description of labio-glosso-laryngeal paralysis in 1860 [27]; and finally, of Charcot and Alix Joffroy (1844–1908) unifying the description of de Duchenne with their description of progressive muscular atrophy, resulting in a single disease, amyotrophic lateral sclerosis [28]. In addition, Hallopeau did not fail to refer to the works of Henry Duret (1849–1921), which set forth the arterial functional anatomy of the brainstem in 1873 [29].

After noting the medullary origin of the swallowing reflex, Hallopeau reiterated the existence of Flourens's ''vital node''; that is, the presence of medullary structures controlling breathing and regulating heartbeat, whose destruction causes immediate death. He listed the pathologies affecting the medulla and, simultaneously or successively, the spinal cord: multiple sclerosis, amyotrophic lateral sclerosis, and general paralysis. From a pathophysiological point of view, he distinguished primitive damage to medullary nuclei from lesions affecting ''the nervous conductors''; that is, the corticospinal and corticonuclear tracts. He then introduced the concepts of crossed paralysis and alternating paralysis but without using these terms. ''The medullary nuclei are grouped together in a relatively narrow space. In many cases, these organs are simultaneously damaged and as a result, the paralysis is bilateral and multiple. This leads to considerable disorders in the functions where these nerves must be directly active; that is, to cite only the main examples, in facial expressions, the articulation of words, phonation, and mastication and swallowing''.

In the second part of his work, Hallopeau put forward observations illustrating the symptomatology by localisation of the lesions: ''The localisation of bulbar paralysis is closely dependent on the location of the lesions that produce it''. Using three personal observations and following in the footsteps of Duchenne de Boulogne, Hallopeau described in details the symptomatology of isolated labio-glosso-laryngeal paralysis; then, with ten observations from various authors, he showed how this clinical picture was associated with progressive muscular atrophy. Producing the likes of semiology lessons, he described in detail the consequences of the paralysis of the tongue and soft palate and the disturbances of phonation and swallowing, leading in all cases to a fatal outcome. He did not fail to highlight that ''it is Charcot to whom credit is due for having been the first to indicate the anatomical lesion in labio-glosso-laryngeal paralysis; he recognised that it consists essentially in atrophy of the medullary nuclei''. Hallopeau, like all physicians of his day, drew a parallel between this pathology and ''infantile paralysis''; that is, acute anterior poliomyelitis or Heine-Medin disease, named for Jacob von Heine (1800–1879) and Karl-Oscar Medin (1847–1927), which was recognised as a contagious pathology around 1890 [30]. Hallopeau did not fail to pay homage to his three colleagues, Alix Joffroy [31] and Georges Hayem (1841–1933) [32], along with Albert Gombault (1844–1904), who had not yet defended his thesis at that time. Under the supervision of Charcot, they authored microscopic analyses decisive in describing the clinical picture of Charcot disease [33]. Hallopeau emphasised the fact that bulbar paralysis and progressive muscular atrophy were not a single entity, reiterating the obstinacy with which de Duchenne considered them two different diseases. He then reported an observation of ''spinal general paralysis'', which calls to mind a case of amyotrophic lateral sclerosis rather than general paralysis.

Turning to ''bulbar paralysis in multiple sclerosis'', Hallopeau underscored the frequency of damage to the brainstem during the disease. For him, one of the first disturbances to appear is a speech disorder, then tremor in the limbs but also the tongue and lips. He briefly covered sensory disturbances, sometimes mentioning neuralgia in a branch of the trigeminal nerve.

To introduce his section on vascular pathology, Hallopeau reiterated the case he had published in 1876, that of a 36-year-old woman with mitral stenosis complicated by endocarditis causing embolism leading to thrombosis in the brainstem [34]. Hallopeau regretted he only had nine well-documented examples of ''symptomatic paralysis with medullary localisations, haemorrhage, or softening''. In addition to a personal case, he looked for cases observed by Adrien Proust (1834–1903) and his resident Gabriel Luneau (1844–?) that involved vertebral occlusions, but strangely enough, he never cited Gubler.

Hallopeau then reviewed numerous observations of medullary compression, caused by either a tumour or trauma, providing a detailed presentation of the various types of deficits in order to localise the compressive phenomenon. He ended his review of bulbar pathology by mentioning diphtheric paralysis, citing Gubler and Duchenne de Boulogne, among others.

Hallopeau concluded his thesis by noting how difficult it could be to make an aetiological diagnosis while the patient with a pathology clinically localised in the medulla was still alive. Imaging did not exist. In his 1885 *Notice sur les titres et travaux scientifiques* (list of titles and scientific works), Hallopeau underscored, not without pride, that ''this monograph is the only one that exists on this subject'' [17]. For him, ''the medulla is like a prolongation of the spinal cord; there is a close kinship between the affections of these two organs''. ''The lesions that develop there damage parts that are similar in their structure and their functions, and progress in a similar way and provoke the same disorders''. It was in this brief work and not the original thesis that he clearly explained the notion of alternating paralysis, as if he wanted to correct something he had forgotten that was fundamental. Finally, we should note that in this list, Hallopeau wrote that ''the external oculomotor nerve can be, like the facial nerve, paralysed, and this paralysis can alternate with that of the extremities''. And it was here that he added to the name of Foville that of Félix Féréol (1825–1891), whom he had omitted to cite in this thesis [20,26].

7. Pathophysiology of apoplexy (1873)

In November 1873, Hallopeau presented a theory to the French Society of Anatomy concerning the pathophysiology of apoplexy; his aim was to become a member of this body [35]. For him, apoplexy required ''the action of a cause

Please cite this article in press as: Walusinski O. The neurologist Henri Hallopeau (1842–1919), a famous dermatologist. Revue neurologique (2024), https://doi.org/10.1016/j.neurol.2023.10.017

simultaneously affecting the full extent of the central nervous system'' and not correlated to the size of the brain lesion found during autopsy, but rather to the localisation of this lesion. Whether after haemorrhage or softening – and as he said, drawing on a lesson given by Charcot in August 1870 (unpublished) – he rejected ''cerebral anaemia'' or ''an increase in intracranial pressure'' as an explanation for the symptoms of the deficits observed during apoplexy. Cerebral apoplectic lesions ''have a paralysing action on the opposite side of the spinal cord'' and their mechanism is ''an action that stops the excitation originating in the brain''. He did not explain the role of tissular anoxia.

8. Anatomy of the facial nerve (1879)

The complex anatomy of the intracranial pathway of the facial nerve was still poorly defined in 1879. The semiological categorisation of facial paralysis into central or peripheral types had not been established. Based on an observation of hemiplegia, Hallopeau tried to elucidate the innervation of the orbicularis oculi, notably suggesting that a contingent of fibres crossed the lenticular nucleus. On this point, he contradicted the clinical picture, in which paralysis of the eyelid is absent in hemiplegia resulting from Sylvian occlusion. To support his theory, Hallopeau asserted that ''the lenticular nucleus is constantly irrigated by the branches of the anterior cerebral and choroidal arteries'' [36].

9. Pathogenesis of tabes (1879)

At the session of the French Society of Biology on 29th November 1879, Hallopeau responded to a presentation by Charcot's ophthalmologist, Xavier Galezowski (1833–1907), on ''atrophy in optic papillae''. Hallopeau attempted to draw a parallel between this atrophy and the origin of sensory deficits characterising progressive locomotor ataxia (tabes). The causal role of syphilis was not accepted by all at that time, despite the explicit works of Alfred Fournier (1832–1914) [37] in 1876 and the premonitions of Guillaume Duchenne de Boulogne (1806–1875) starting in 1858 [38]. Hallopeau explained that the symptoms of tabes resulted from an initial hyperstimulation of the sensory pathways secondary to ''the suppression, weakening, or perversion of centripetal excitations, be they perceived or not'', which were the origin of ataxia. The cause was ''repeated excitation of the sensory system and notably venereal excesses, muscular fatigue, and frequent chills'' [39]. Hallopeau had no qualms about presenting his theory once again, this time at the International Medical Congress in London in 1881 [17].

10. Cortical localisation (1883)

With the help of his resident Charles Giraudeau (1854–?), Hallopeau wanted to make his contribution to the localisation of cerebral areas, which David Ferrier (1843–1928) had proposed based on experiments involving electrical stimulation of the cortex in dogs [40]. Noting the lack of anatomopathological data that would have enabled the localisation of the ''motor centre of the lower limb'' [41], Hallopeau related the history of a 46-year-old man who presented with convulsive episodes in the lower left limb followed by loss of consciousness. A few months later, headaches set in and worsened relentlessly until the patient's death. During the autopsy, they discovered ''a pinkish tumour protruding from the surface of the cerebral substance of the right hemisphere in the upper third of the parietal region''. The histopathology revealed a glioma. Hallopeau spoke of the difficulty of diagnosing the patient while they were alive at the beginning of the disease, ''in the absence of any other medullary or encephalic symptoms''. He then reviewed the eighteen cases already published by various authors. He concluded that the motricity of the lower limb ''is centered in the upper third of the parietal region as well as in the paracentral lobule, and this centre impinges anteriorly on the upper part of the frontal region and posteriorly on the superior parietal lobule''. These initial observations did not go into detail about the subtlety of motor control in the primary motor area, in the pre-motor cortex, and involving proprioceptive information at the parietal level. The notion of the neuron had not yet been established but a step in the comprehension of cortical activity had been made and roughly localised.

11. Aneurysm of the basilar trunk (1883)

Also in 1883, Hallopeau and Giraudeau reported on the history of a man with ascending quadriplegia with progressive onset, and with the final complication of coma with opisthotonos: ''If he was held up, his head fell forward and breathing immediately stopped during exhalation while the pulse continued to beat regularly for a few seconds then slowed'' [42]. The autopsy revealed an aneurysm of the basilar trunk. Hallopeau suggested that cervical dorsiflexion limited mesencephalic compression by a vascular tumour, while flexion inhibited the activity of the cardiorespiratory centres. He added, ''This fact we have reported is unique in the medical literature'' [17].

12. Localisation of blindness accompanied by hemichorea (1885)

An 83-year-old patient with severe aortic valve stenosis had left ventricular failure when he suddenly and totally lost his sight. His pupils were dilated and unresponsive. The left side of his body was agitated with choreic movements. In a few days, his condition improved, he could once again see shapes, and the involuntary motor agitation had decreased. Hallopeau suggested that the lesion in the brain, probably an infarct caused by embolism, was situated in the quadrigeminal bodies (corpora quadrigemina is the Latin terminology). He referred to the thesis of Fulgence Raymond (1844–1910) [43] to explain the cause of the choreic movements in the left limbs as being in ''the posterior internal part of the thalamus'' [44]. Currently, the diagnosis is acute transient contralateral hemichorea lasting a few hours to a few days, after infarct in the area of the striatum, or contralateral hemiballismus after damage to the subthalamic nucleus (nucleus of Luys).

Please cite this article in press as: Walusinski O. The neurologist Henri Hallopeau (1842–1919), a famous dermatologist. Revue neurologique (2024), https://doi.org/10.1016/j.neurol.2023.10.017

13. Jaccoud's dictionary of medicine and surgery

Sigismond Jaccoud (1830–1913) put Hallopeau in charge of writing the "Encephalon" (1870) and "Spinal Cord" (1876) entries of his dictionary [45]. Hallopeau endeavoured to elucidate the pathological physiology and pathogenesis of various neurological diseases in order to classify them, but he suggested that this way of presenting them also enabled understanding "the production of symptoms". Strangely enough, he discussed the concept of neuralgia and its semiology at length, which he considered to determine treatment to the detriment of other neurological disturbances. In any case, these chapters now have only historical value, attesting to the state of knowledge before Charcot and Vulpian undertook a rigorous study of nervous system diseases.

14. Conclusion

This overview of Hallopeau's neurological publications may explain why he chose to abandon neurology for a career in dermatology. Indeed, none of his works advanced knowledge or contributed new ideas. Only his *agrégation* thesis on bulbar pathology has a few original pedagogical virtues. Undoubtedly aware of the limits of his solitary enterprise, compared with the multiple discoveries that the La Salpêtrière School, directed by Charcot and Vulpian, made during the same period, Hallopeau chose to abandon neurology for dermatology, thereby greatly benefiting the patients at Saint-Louis Hospital.

Disclosure of interest

The author declares that he has no competing interest.

Acknowledgements

Many thanks to Hubert Déchy and Jacques Poirier for their attentive readings and suggestions and to Anna Fitzgerald for her translation.

REFERENCES

[1] Huguet F. Les professeurs de la Faculté de Médecine de Paris, dictionnaire biographique 1794–1939. Paris: Ed du CNRS; 1991.
[2] Hallopeau H. Des accidents convulsifs dans les maladies de la moelle épinière. Paris: A. Parent; 1871 [Thèse Paris no 18].
[3] Hallopeau H. Du mercure, action physiologique et thérapeutique. Paris: JB Baillière; 1878.
[4] Martin PR. Dictionnaire biographique des dermatologues et syphiligraphes français. Angers: Ed. du Martinet; 2003.
[5] de Menten P. Dictionnaire de chimie : une approche étymologique et historique. Bruxelles: De Boeck; 2013.
[6] Archives nationales, base de données Léonore : LH//1260/14.
[7] Milian G. Nécrologie : le Dr H. Hallopeau (1842–1919). Paris Med 1919;32:135.
[8] Hallopeau H. Fracture du crâne et de la mâchoire inférieure. Déchirure de l'artère sphéno-épineuse. Bull Soc Anat Paris 1867;42:121–3.
[9] Hallopeau H. Fracture du crâne par une balle, abcès du cerveau. Bull Soc Anat Paris 1867;42:546–8.
[10] Hallopeau H. Note sur un fait de sclérose diffuse de la moelle avec lacune au centre de cet organe, altération de la substance grise et atrophie musculaire. Comptes rendus des séances et mémoires de la Société de Biologie (année 1869). Paris: Adrien Delahaye; 1870.
[11] Walusinski O, Honnorat J. Augustin Morvan (1819–1897), a little-known rural physician and neurologist. Rev Neurol (Paris) 2013;169(1):2–8.
[12] Brown-Séquard CE. Recherches expérimentales sur la production d'une affection convulsive épileptiforme à la suite de lésions de la moelle épinière. C R Acad Sci 1856;42:86–9.
[13] Brown-Séquard E. Experimental and clinical researches applied to physiology and pathology. Boston Med Surg J 1857;56:54–8.
[14] Flourens MJP. Recherches expérimentales sur les propriétés et fonctions du système nerveux dans les animaux vertébrés. Paris: Crevot; 1824.
[15] Koehler PJ. Brown-Séquard's spinal epilepsy. Med Hist 1994;38:189–203.
[16] Hall M. On the diseases and derangements of the nervous system. London: H. Baillière; 1841.
[17] Hallopeau H. Notice sur les titres et travaux scientifiques. Paris: Alcan-Lévy; 1885.
[18] Virchow R. Die Cellularpathologie in ihrer Begründung auf physiologische und pathologische Gewebelehre. Berlin: A. Hirschwald; 1858.
[19] Hallopeau H. Étude sur les myélites chroniques diffuses. Arch Gen Med 1871;série VI 18 [277–305/435–449/565–579; 172; série VI 19:60–75/191–202].
[20] Hallopeau H. Des paralysies bulbaires. Paris: JB Baillière; 1875.
[21] Wallenberg A. Acute Bulbäraffection (Embolie der Arteria cerebelli posterior inferior sinistra?) Archiv für Psychiatrie und Nervenkrankheiten 1895;27:504–40.
[22] Marcet A. History of a singular Nervous or Paralytic affection, attended with anomalous morbid sensations. Medico-Chirurgical Transactions 1811;2:217–35.
[23] Josias PH. Des hémorrhagies de la protubérance annulaire ou mésocéphale. Paris: impr. Rignoux; 1851 [Thèse Paris no 162].
[24] Philipeaux MM, Vulpian A. Essai sur l'origine de plusieurs paires des nerfs crâniens 3e, 4e, 5e, 6e, 7e, 8e, 9e et 10e. Paris: impr. Rignoux; 1853.
[25] Gubler A. De l'hémiplégie alterne envisagée comme signe de lésion de la protubérance annulaire et comme preuve de la décussation. Gazette Hebd Med Chir 1856;3(43) [749–754/(45): 789–792/(46): 811–816].
[26] Gubler A. Mémoire sur les paralysies alternes en général, et particulièrement sur l'hémiplégie alterne, avec lésion de la protubérance annulaire. Gazette Hebd Med Chir 1858;5(42) [721–723/(45): 765–769/(47): 801–804/(52): 883–886/1859; 6(1): 3–7/(4): 52–55/(6): 86–90].
[27] Foville A. Note sur une paralysie peu connue de certains muscles de l'œil, et sa liaison avec quelques points de l'anatomie et la physiologie de la protubérance annulaire. Bull Soc Anat Paris 1858;33:393–414.
[28] Duchenne de Boulogne G. Paralysie musculaire progressive de la langue, du voile du palais et des lèvres, affection non encore décrite comme espèce distincte. Arch Gen Med 1860;série V 16 [283–296/431–445].

Please cite this article in press as: Walusinski O. The neurologist Henri Hallopeau (1842–1919), a famous dermatologist. Revue neurologique (2024), https://doi.org/10.1016/j.neurol.2023.10.017

[29] Charcot JM, Joffroy A. Deux cas d'atrophie musculaire progressive avec lésions de la substance grise et des faisceaux antéro-latéraux de la moelle épinière. Arch Physiol Norm Pathol 1869;2 [354–367/629–649/744–760].
[30] Walusinski O, Courrivaud P. Henry Duret (1849–1921): a surgeon and forgotten neurologist. Eur Neurol 2014;72(3–4):193–202.
[31] Tinel-Giry L.M. La poliomyélite épidémique (maladie de Heine-Medin).. Paris: Alfred Leclerc; 1911. [Thèse Paris no 5].
[32] Duchenne G, Joffroy A. De l'atrophie aiguë et chronique des cellules nerveuses de la moelle et du bulbe rachidien. À propos d'une observation de paralysie glosso-labio-laryngée. Arch Physiol Norm Pathol 1870;3:499–515.
[33] Hayem G. Note sur un cas d'atrophie musculaire progressive avec lésions de la moelle. Arch Physiol Norm Pathol 1869;2 [263–278/391–406].
[34] Gombault A. Étude sur la sclérose latérale amyotrophique. Versailles: impr. Cerf; 1877 [Thèse Paris n° 353].
[35] Hallopeau H. Note sur un fait de thrombose basilaire. Arch Physiol Norm Pathol 1876;8:794–802.
[36] Hallopeau H. Note pour servir à l'étude physiologique de l'apoplexie. Bull Soc Anat 1873;48:721–4.
[37] Hallopeau H. Note pour servir à déterminer le trajet intracérébral du faisceau supérieur du facial. Rev Mens Med Chir 1879;3:937–44.
[38] Fournier A. De l'ataxie locomotrice syphilitique (tabes spécifique). Paris: G. Masson; 1876.
[39] Duchenne de Bologne G. De l'ataxie locomotrice progressive. Recherches sur une maladie caractérisée spécialement par des troubles généraux de la coordination des mouvements. Arch Gen Med 1858;série V 12 [641–652/1859; série V 13:36–62/158–181/417–451].
[40] Galezowski X, Hallopeau H. Les atrophies traumatiques des papilles optiques. C R Seances Mem Soc Biol 1880;31:330–2.
[41] Young RM, Mind. Brain and adaptation in the nineteenth century: cerebral localization and its biological context from Gall to Ferrier. New York: Oxford University Press; 1970.
[42] Hallopeau H, Giraudeau C. Des paralysies du membre inférieur d'origine corticale. L'Encéphale 1883;3:331–40.
[43] Hallopeau H, Giraudeau C. Note sur un cas de compression de la protubérance par dilatation anévrysmale du tronc basilaire. L'Encéphale 1883;3:657–66.
[44] Raymond F, Étude anatomique. physiologique et clinique sur l'hémianesthésie, l'hémichorée, et les tremblements symptomatiques. Versailles: impr. Cerf; 1876 [Thèse Paris n° 157].
[45] Jaccoud S. Nouveau dictionnaire de médecine et de chirurgie pratiques. Paris: JB Baillière; 1864–1886.

Please cite this article in press as: Walusinski O. The neurologist Henri Hallopeau (1842–1919), a famous dermatologist. Revue neurologique (2024), https://doi.org/10.1016/j.neurol.2023.10.017