Pioneering the Concepts of Stereognosis and Polyradiculoneuritis: Octave Landry (1826–1865)

Olivier Walusinski
Family Physician, Private Practice, Brou, France

Abstract
Octave Landry was one of a long list of fine 19th century clinicians who died very young and whose discoveries in physiology and descriptions of new clinical pictures helped found current-day neurology. In 1852, Landry proposed a new take on the physiology of sensation which laid the ground for the concepts of proprioception and stereognosis. He also described the clinical picture of a rapidly progressing ascending paralysis, which in 1859 prefigured Guillain-Barré syndrome. In discussing his very active life, we will mention the hydrotherapies in fashion at the time and the pleasures of Parisian society. Landry’s career was also marked by terrible cholera epidemics, one of which killed him at age 39, in the prime of his working life as a devoted physician.

The use of eponyms is slowly disappearing in medicine [1]. Who uses or is familiar with the term ‘Landry’s paralysis’? Nonetheless, our current knowledge is based on a gradual accumulation of scientific discoveries, over at least two centuries. Octave Landry is among those fine 19th century clinicians who founded neurology and psychiatry. After a biographical sketch, we will present his main contributions, which include his introduction in 1852 of the concept of muscular sensitivity, which led to the concepts of proprioception (the ability to perceive the position and movement of a body segment without any visual control, as conceived by Charles Sherrington (1857–1952) in 1906) and stereognosis (perception of shape and form of objects via tactile sense without the aid of sight, as conceived by Heinrich Hoffmann (1819–1891)). Landry also published a case of acute ascending paralysis in 1859. Later, Georges Guillain (1876–1961), Jean-Alexandre Barré (1880–1967) and André Strohl (1887–1977) would add ‘hyperalbuminosis of the cerebrospinal fluid without cellular reaction and notes on the clinical and graphic characteristics of the tendon reflexes’, referring to a ‘syndrome of radicular neuritis’ [2].

Education
Jean-Baptiste Octave Landry was born on 10 October, 1826, in Limoges, France (fig. 1). Jean-Martin Charcot (1825–1893) had been born just a year earlier. Michel Landry, Octave’s father, was a wealthy bourgeois landowner. His mother, Catherine-Louis de Thézillat, and her family were originally from Catalonia. His true family name is thus simply Landry, as it appeared on his thesis,
and not Landry de Thézillat, as reported by English-language biographers [3–5]. After his studies in Limoges, he went on to the Faculté de Médecine de Paris (1845 to 1850). The cholera epidemic that began in Dunkerque in October 1848 hit Paris in March 1849 when Landry was externe des hôpitaux. Landry volunteered to treat those affected by the epidemic and went to the Oise region, where he worked tirelessly and became quite well-known, which displeased some of his local colleagues. He was awarded a medal for his devoted service. In 1849, he passed the exam to become an interne des hôpitaux de Paris, fellow student of Alexandre Laboulbène (1825–1898). During his first year, he wrote a dissertation on the cholera epidemic he had experienced firsthand. This magnificent manuscript has been conserved at the library of the Faculté de Médecine de Paris [6]. In 220 pages, Landry presents an epidemiological study, describing the epidemic’s development across France, the socioeconomic conditions, the mortality rate by district in Paris, in-depth clinical information, Landry’s autopsies as well as prognostic estimates and treatments used. He also included observations with favourable outcomes. The work can be considered a thesis before his thesis, and is all the more impressive given that Landry was only 23! In it, he describes the collapsus resulting from ‘absolutely serous stools. With their white tongues and pasty mouths, cholera patients suffer from an inextinguishable thirst’. In addition, there are cramps, prostration, ‘a lost look in the eyes’, ‘algidity or cyanosis’.

Landry was convinced of the therapeutic necessity of ‘complete fasting, bed rest, warm wine with brandy and repeated astringent enemas’. He notes that ‘upon detecting any alteration of the blood, some physicians injected various aqueous liquids in the veins, but nearly always without the least success. Wishing to take advantage of the absorptive powers of the skin and of surfaces other than the gastrointestinal mucous membranes, some physicians used baths with cholera patients. M. Piorry went so far as to inject water in the bladder, hoping it would be absorbed; unfortunately these attempts were unsuccessful.’ He submitted this dissertation for the 1850 Academy of Medicine ‘Prix Monthyon’ but was not awarded the prize.

During his internat, Landry alternated between medicine and surgery, training under Alphonse Devergie (1798–1879), Stanislas Laugier (1799–1872), René Marjolin (1812–1895) and Louis Michon (1802–1866). But it was especially Claude-Stanislas Sandras (1802–1856) at Hospital Hôtel Dieu and Adolphe-Michel Gubler (1821–1879) at Hospital Beaujon who influenced his interest in ‘nervous pathology’. He was also encouraged by his uncle Thézillat (physician and director of the Limoges lunatic asylum). He defended his thesis on 29 December 1854 before a jury presided by Armand Trousseau (1801–1867). The thesis was entitled: ‘General considerations on pathogenesis and therapeutic indications of nervous diseases’ [7]. He noted that his thesis was only the ‘rapid summary of a larger work on the causes and therapeutic indications of nervous diseases, currently being published in the Moniteur des Hôpitaux’. In reality, this journal only published extracts (in three parts from March to April 1855) of his work ‘Research into the causes and therapeutic indications of nervous diseases’, published the same year, which represented his thesis supplemented with new observations [8, 9]. His thesis jury questioned him about the ‘pathological anatomy of the apoplexy of the pons and the spinal cord’ and, in forensic medicine, about the ‘mental lesions dependent on one of man’s physical needs, such as hunger, thirst, the genital act, etc. that relate to the non-satisfaction of one of these needs, or consist in an exaltation or a deviation of these needs’. How unfortunate that the records of his responses do not exist!

Landry’s Medical Practice

Shortly after defending his thesis, Landry set up his practice at 5, rue de l’Université in Paris where his many patients were an indication of his talents. From 1852,
he was secretary of the Société Médicale de Paris and member of the Société Anatomique. His mother, widowed in 1854, still had two young children to raise, which obligated Landry, as the eldest, to provide for them. This is undoubtedly why he did not pass the competitive exams for the university and hospital positions he seemed destined for. Trained as a musician by his violinist father, he excelled at the cello. He was also an accomplished sportsman – rare in his day – who practiced mountaineering and horseback riding. Both a singer and an elegant dancer, he was sought after for Parisian parties. One evening, he fell in love with a very beautiful young woman, Claire-Marie Giustiniani, from Zigliara in Corsica, whom he married on 25 July 1857. When their son was born, on 22 April 1858, they moved to 184 rue du Faubourg Saint-Honoré. His busy medical practice with wealthier patients did not deter him from his research objectives, as his publications at that time attest. Through his social contacts, he met Thomas Brière Desilles (or Desilles Brière, 1806–1872) from Martinique, whose *Journal de Rouen* published his first works, in particular the commercial version of his thesis in 1855 [10]. Although the notion of medical specialty did not exist at that time, his practice focused increasingly on ‘nervous diseases’ for which he was a fervent advocate of hydrotherapy. When Charles-Victor Boullay, head physician at the hydrotherapy establishment in the village of Auteuil (west of Paris) died suddenly in August 1859, Landry moved his entire family there on 15 October 1859 [11]. Founded in 1820, this establishment was renowned for the quality of its waters. Landry renovated it, enlarged it and made it the biggest and best equipped establishment at the time. Auteuil became part of Paris in 1860, which no doubt influenced his initiative. Located in a magnificent, 17 acre wood, complete with performance venues and billiard rooms, the establishment (located at 12, rue Boileau) was soon attracting rich French and foreign epileptics, paralytics, hysterics and those suffering from ‘other congenital or acquired neuroses’ (fig. 2) [12]. It was a high-profile place; a celebration including fireworks marked the birth of Landry’s daughter Berthe on 5 August 1860 (1860–1922). Perhaps as a result of overwork, Landry then began suffering from recurrent and violent headaches, for which he took morphine, completely weaning himself off the drug only 5 years later.

On 1 June 1865, the cruise ship Le Stella docked in Marseille, arriving from Alexandria in Egypt. Within a few weeks, the passengers spread a new epidemic of cholera. In October, workers in Boulogne and Auteuil were infected. Landry, with his considerable experience, went to care for them. Almost immediately, he realized that he himself was infected and isolated himself from his wife and children [13]. His colleagues and friends, Charcot and Noël Gueneau de Mussy (1813–1885), rushed to his bedside, but despite their efforts Landry died after two days of agony, at age 39, on 1 November 1865. The death of a physician had become commonplace during the epidemics (4602 Parisians died of cholera in October 1865); only a few paragraphs in *Union Médicale* and *Gazette Médicale de Paris* commemorated him [14, 15]. The painter Gustave Courbet (1819–1877) left us a portrait of Landry, painted in 1863. Its location is currently unknown, though it is believed to be in the

Octave Landry (1826–1865)
In 1852, while an interne under Sandras, Landry published his 'Physiological and pathological research on tactile sensations', in which he noted, 'The infinitely complex questions related to the sense of touch have been analyzed by one or two authors, and I am not afraid to say that these analyses were carried out with data that was either insufficient or incapable of elucidating the subject' [16]. He criticized the 1837 thesis of Henry Belfield-Lefèvre for confused thinking, directly transposed from the treaty on sensation by the philosopher Étienne Bonnot de Condillac (1715–1780): ‘The number of ideas that can come through the sense of touch is infinite, given that it includes all the relations of magnitude’ [17, 18]. He also criticized Pierre-Nicolas Gerdie (1797–1856), who had explained that ‘the sensations of temperature, of dryness and wetness, of weight, and of the body’s consistency and movement enter the mind immediately and provide us with knowledge of the excitatory causes without any detectable thought’ [19]. Landry also contested the proposition of the German physiologist Johannes Müller (1801–1858) ‘that the idea of weight and pressure was not a sensation in the muscle, but a notion of the quantity of nervous action that the brain is excited to apply’ [20], because for Landry, ‘the sensation of muscular activity is in fact produced by special modifications that the various organs of movement reproduce in the nervous extremities located throughout them’. He drew on the work of Erasmus Darwin (1731–1802): ‘The muscular fibres themselves constitute the organ of sense, that feels extension... hence the whole muscular system may be considered as one organ of sense, and the various attitudes of the body, as ideas belonging to this organ, of many of which we have hourly consciousness, while many others, like the irritative ideas of the other senses, are performed without our attention’ [21]. After having discussed and found the research on touch and pain published in 1848 by Joseph-Henri Beau (1806–1865) to be incomplete as well, Landry explicitly proposed that ‘sensations of temperature be considered essentially distinct and independent from touch and pain’ [22]. Aged only 26 at the time, Landry concluded by accurately formulating concepts of the physiology ‘of sensation’: ‘There are only a few cutaneous sensations which may be called primitive or special, and from which result all the other sensations, or derived sensations: temperature, pain and contact. There is also a primitive or special sensation involving muscular activity that gives rise to secondary or derived sensations. This sensation actually resides in the muscular tissue itself, being an actual perception by the brain of the state of nervous and sensitive extremities distributed in the muscles.’ Landry constructed this innovative theory through careful examination of patients with his teacher Sandras, who had recently published his Traité pratique des maladies nerveuses [23]. He cited observations quite similar to those of Guillaume Duchenne de Boulouge (1806–1875) on the ‘motor aptitude independent from vision, called by the author muscular conscience’ in 1853 [24], and ‘progressive locomotor ataxia’ in 1858 [25]: ‘Today, 20 November 1851, during the visit, this patient said to Sandras that when he tried to walk, as soon as he could no longer see his feet, he didn’t know where he was placing them and could not measure their movements, which was clear for all those present. In response to this account, I examined the patient again, and observed the following: without his watching, I lifted one of his lower limbs, inclining it to the right and to the left, lifting it, lowering it, either the entire limb or part of it. He was absolutely unaware of any of these movements. I had him walk, supported by two people; when he watched his feet, he placed them quite easily where he wanted them. I had him lie down, and I showed him points to place his foot on. He was capable of this with great precision as long as he watched; however, if he stopped watching his legs, his movement was in the same direction but was so disproportionate that he greatly exceeded the proposed objective. He noted that he was unaware of the amplitude of his movement.’ Landry concluded: ‘Movement coordination requires the sensation of muscular activity.’ He must have known that Charles Bell (1774–1842), called ‘the Harvey of our century’ and president at that time of the Royal Society, had presented on 25 January 1826 his essay on the physiology of motor control: ‘Between the brain and the muscles there is a circle of nerves; one nerve conveys the influence from the brain to the muscle, another gives the sense of the condition of the muscle to the brain’ [26, 27], which is complemented by his 1833 book on the physiology of the hand [28]: ‘When a blind man, or man with the eyes shut, stands upright... by what means is it that he main-
tains the erect position? He touches nothing, he sees nothing, it can only be by the adjustment of muscles. It must be a property internal to the frame by which we thus know the position of the members of our body.’ However, Landry was probably not aware that in Germany, Friedrich-August Benjamin Puchelt (1784–1856) had published five observations of patients who, with their eyes closed, were incapable of recognizing what they had in their hand even though they had no disturbances in sensation [29]. Nonetheless, he suggested this interpretation: ‘The characteristics of an object can only be apprehended in detail by the hands acting as gripping elements, and only, I repeat, when the hands are activated in conjunction with the attention and other intellectual faculties [...]. It is this intimate association of muscular action and intellectual faculties with the four primitive tactile sensations that must specially designate the sense of touch.’ Landry can thus be credited with the concept of the physiology of stereognosis (fig. 3) [30]. His ideas were confirmed the following year in a thesis defended 20 May 1853 by Julien-Benjamin Bellion, interne under François-Amilcar Aran (1817–1861), with Jean-Baptiste Bouillaud (1796–1881) presiding over the jury [31]. In 1855, Landry added to this work by publishing observations in which ‘the sensation of muscular activity was decreased or lost’ and ‘the motor incitation had lost its dynamometer’ [30]. He gives Duchenne de Boulogne credit for having, as he himself had, identified the essential role of visual substitution; strangely, however, he did not make the link between Duchenne’s ‘muscular consciousness’ and his own ‘sensation of muscular activity’. The etiology for the ‘paralysis of muscular sensation’ remained mysterious but seemed to him unrelated to ‘lunatic paralysis’; later on, Charcot, motivated by ‘national-scientific’ bias, would reject a common origin for locomotor ataxia, tabes and general paralysis. Landry’s friend from his days as an interne, Louis Victor Marcé (1828–1864), praised him in his thesis [32]: ‘I admit that the research of Duchenne, carried out several years after that of Landry, appears to me to reproduce exactly all that the physiologists have written on the sensation of muscular activity; as for progressive locomotor ataxia which the same author presented as a distinct nosology, this for me is nothing other than the morbid state following a loss of muscular feeling, a state which Landry had drawn attention to in 1855.’ Marcé, one of the most productive alienists of his generation, committed suicide in the Au teuil establishment where he was under Landry’s care for melancholia [33].

Fig. 3. Dissertation on Paralysis of the Sensation of Muscular Activity (1855) signed by Landry. Private collection of the author.

Landry’s Thesis: General Considerations on the Pathogenesis and Therapeutic Indications of Nervous Diseases (1854)

‘Despite the philosophical tendencies of certain modern physicians, the treatment of neuroses is almost entirely dominated by a regrettable empiricism; and unfortunately this state of affairs reflects how we understand these conditions’ [7]. To combat this empiricism, Landry explains that his thesis puts forth a classification of nervous system pathologies, inspired by Samuel Tissot (1728–1797), Laurent-Philibert Cerise (1807–1869) and François Broussais (1772–1838). In an approach that appears very confused to us, he believed he could classify this entire category of causes under the following headings: blood conditions, physical exhaustion, ca-

Octave Landry (1826–1865)

Octave Landry (1826–1865)
chexia, acute and chronic diseases, diathesis, action of cold and humidity, intoxications, influence of certain neuroses on other neuroses, sympathetic neuroses, and causes that act directly on the nervous system’. As he indicated in his conclusion, ‘I have hardly gone beyond the role of compiler that I set out for myself’. Landry did not formulate any new ideas or novel descriptions, as Charcot would do soon thereafter. His thesis is quite ordinary and disappointing in comparison with the originality of his cholera dissertation and his publications on tactile sensations and the sensation of muscular activity.

**Use of Chloroform and Narcotic Agents as Therapeutics and Diagnostic Substances in Certain Paralyses (1857)**

Landry related the case of a female patient with aphonia and dyspnea who was unable to move about, though she did not have real paralysis in the limbs. Landry attributed her condition to paralysis of the diaphragm and the abdominal muscles secondary to a gynecological cause, a ‘sympathetic paralysis’. Her condition seemed to disappear during natural sleep or sleep induced by ether or chloroform. Was the patient suffering from myasthenia? In this publication, which does not contain very interesting information, Landry analyzed six other paralysis observations in which the condition is ‘improved by narcotics’. Overall, the work is quite hard to understand by today’s standards [34].

**Acute Ascending Paralysis (1859)**

Even 4 years after finishing his internat, Landry continued to gather observations in Gubler’s department. After the detailed observation he submitted to the *La Gazette hebdomadaire de Médecine et de Chirurgie* of July 1859, he summed up his findings thusly: ‘In the type of paralysis in which I wish to draw attention, sensation and movement can both be compromised; however, in general, the functional disturbances primarily affect movement and are thus characterised by a gradual decrease in muscular force, with flaccidity in the limbs, and without shaking, contractions, partial or general convulsions, or reflexive movements. In almost all cases, micturation and defecation remain normal. No immediate symptoms in the nervous centres are observed, no rachialgia (neither spontaneous nor developed by pressure), no cephalalgia or delirium; until the end, the intellectual faculties are completely conserved [...]. The paralysis moves rapidly from lower to upper areas, with a constant tendency to generalise. The first phenomena always occur at the extremities of the limbs, and most often in the lower limbs [...]. When the paralysis reaches its peak of intensity, death by asphyxiation is always imminent. However, eight times out of ten, this fatal outcome was avoided, either by medical care, or by a spontaneous interruption in the disease’s development. Only in two cases was death observed at this stage of the disease [...]. A disease that is fatal for a fifth of patients is undeniably a serious disease, and despite the relatively favourable results of these basic statistics, it must be understood that in cases of such accidents, the danger is always extreme and the prognosis at least uncertain [...]. When there is a reversal in the paralysis, the recovery period involves phenomena opposite to those indicated in the development period. Upper areas, being the most recently affected, are the first to recover mobility, which then successively returns in top to bottom fashion. Patients then either recover very quickly, or the disease becomes chronic, with slow improvement [...]. The causes, if the circumstances enumerated above can indeed be called causes, appear to be highly variable. In all cases, these influences may only be considered indirect causes, the direct cause of the functional disorders remaining to be determined. The sole two autopsies carried out to date have only provided absolutely negative results from an anatomical-pathological perspective [...]. This morbid form must thus be classified with the numerous so-called ‘essential’ paralyses, i.e. those without evident damage to the nervous system’ [35]. In an additional note, Gubler wondered: ‘Approaching the matter from another perspective, I’m curious as to whether there might be close links between our case of extenso-progressive paralysis and paralyses following diphtheria as described by Bretonneau, Trousseau, Lasègue, Main-gault, etc., of which science is continually recording new examples’ [35].

It is sometimes proposed that the Irishman Robert-James Graves (1796–1853) was the first to describe this clinical picture, although he is better known for his description of exophthalmic goiter [5]. What did he write? ‘One of the most remarkable examples of disease of the nervous system commencing in the extremities, and having no connection with lesions of the brain or spinal marrow, was the curious *épidémie* de Paris, which occurred in the spring of 1828. Chomel has described this epidemic in the 9th number of the *Journal Hebdoma-
d’aire, and having witnessed it myself in the months of July and August of the same year, I can bear testimony to the ability and accuracy of his description’ [36, 37]. Auguste-François Chomel (1788–1858) did in fact describe an epidemic that took place in spring of 1828 in Paris. Patients complained of ‘numbness and a loss of the sense of touch, which in some cases went quite far. Several patients were unable to tactilely distinguish another body, or a key, for example, from a pair of scissors. They were totally mistaken […]. Walking took on a particular characteristic: the foot flat on the ground did not cling to it and instead had to be lifted like an inert mass with its point constantly dragging […]. Several patients were entirely unable to move’ [38]. Among the cases published subsequently, we can cite that of Emile Bablon, a student under Louis-Théodore Lavéran (1812–1879) at Hospital Val de Grâce in Paris. He noted that his observations deserved ‘to be published as much because of the rapid progression of the disease as for the resemblance with Mr. Landry’s disease’. The fatal outcome revealed no pathological lesion of the nervous system upon autopsy [39].

Jules Dejerine (1849–1917) wrote clearly in his thesis entitled ‘Research on nervous system lesions in acute ascending paralysis’ [40]: ‘Landry was the first to draw attention to a specific form of paralysis, very similar to acute myelitis. He called this condition acute ascending paralysis. The credit for this discovery rests entirely with Landry […]. Landry had been perfectly correct in noting that among the paralyses that he called extenso-progressive, there were some variations where the sudden onset and very rapid progression deserved special attention. Based on certain symptomatic particularities, including the fact that sensation was relatively unaffected, he proposed designating these paralyses by the general name of acute centripetal or ascending paralysis. Landry’s main argument for distinguishing this disease from myelitis, as it was conceived of at that time, was the absence of appreciable lesions in the nervous system.’

In 1864, Louis-Stanilas Duménil (1823–1890), surgeon at the Hospital Hôtel-Dieu in Rouen, published the first observation accompanied ‘by an electrical exploration using Duchenne de Boulogne’s machine’ and by an autopsy performed by Georges Pouchet (1833–1894): ‘It appears that we have here true atrophy of the medulla substance of the peripheral nervous tubes resulting in radiculoneuritis [42, 43]. What he described is now referred to as areas of segmental demyelination. Duménil never cited Landry, but instead the work of Charcot and Alfred Vulpian (1826–1887) on diphtheric paralysis of the soft palate [41], and the work of Duchenne de Boulogne. He had presented his findings on 22 January 1862 to the hospital medical society, as reported by Hippolyte Bourdon (1814–1892). Anna Dejerine-Klumpke (1859–1937), in her thesis ‘Contribution of the study of polyneuritis in general and saturnine atrophies and paralyses in particular’, neglected Landry and praised Duménil: ‘Duménil in Rouen deserves credit for having shown, more than 20 years ago, that the nerves could undergo primitive damage, without prior modification to their trophic centres […]’. We reprint here the author’s conclusions from his remarkable work: ‘Not least important or least interesting in the history of these peripheral paralyses is the possibility of their extension to a large part of the nervous system – one could almost speak of generalisation – to the point of compromising life, through the invasion of the most essential nerves, such as the vagus nerve. We see that the disease leads to hemiplegic glossalaryngeal paralysis, preceded and accompanied by impaired sensation, which is merely the morbid process, invader of so many different points, repeating itself on more important organs’ [44, 45].

Alfred Petitfils, in his thesis inspired by Charcot (who was part of his 1873 jury) believed ‘Landry’s disease’ was merely an intermediary form between poliomyelitis and progressive muscular atrophy [46].

Over time, several causes were considered: toxins (lead, arsenic, alcohol), infections (diphtheria, poliomyelitis, rabies) or idiopathic causes. Maurice Briffaut presented his thesis in Lyon in 1906: ‘Contribution to the study of tuberculous polynourites (Landry’s disease)’ [47].

François Muzard argued that the origins were infectious in all cases [48]. In 1916, Guillaum, Barré and Strohl established the different etiological, clinical and progressive variations. In particular, they clarified the biological picture of the ‘albuminoctyologic dissociation in the cerebrospinal fluid’ [2–49].

Marcel Petiot (1897–1946), one of the deadliest serial killers of the 20th century who was guillotined in 1946, defended his thesis (paid for?) in 1921 after his very brief medical studies. Remarkably, his thesis contains an observation of Landry’s disease, involving medullary damage that was fatal in less than a week. He never cites Guillaum and Barré but attempts an amalgamation, poorly supported, to suggest a common aetiology with Landry’s disease and the encephalitis lethargica described by von Economo (1876–1931), an epidemic in full swing at the time [50].
Complete Treaty on Paralyses (1859)

In his preface, Landry speaks of his goal in writing a treaty on paralyses, only the first volume of which was published in 1859 [51]. ‘The insufficient knowledge acquired, the persistent obscurity surrounding this vast subject, the diagnostic uncertainties, despite the progress made, are all very discouraging for physicians.’ I have written my treaty for these reasons and in view of ordinary practice, so that complex knowledge and that part of the medical art too often abandoned to specialists will be made more accessible. My aim is to fill the unfortunate lacunae that exist.’ His treaty only covers the physiology of the spinal cord and brain; pathology was to be covered in the second volume which was never completed on account of his premature death (fig. 4).

He based his work on that of Ollivier d’Angers (1796–1845), Bell, Müller, Julien Legallois (1771–1814), Georges Prochaska (1749–1820), Marshall Hall (1790–1857), Achille Longet (1811–1871), Charles Brown-Sequard (1817–1894), etc. He mentioned the recent discoveries of Jean-Marie Philipeaux (1809–1892) and Vulpian relating to the origin of several cranial nerves and paid homage to Gubler who had confirmed the decussation of cranial nerves (’Alternating hemiplegia considered as a sign of damage to the pons and as proof of decussation of facial nerves’ [52, 53]). Completely dismissing ideas of ‘animal fluid’ and irritability, he proposed that ‘the transmission of excitations does not involve a special agent, but molecule-by-molecule propagation that in turn entails a tissue property: organic conductivity’. He included all his previous work on sensation and added a chapter on motor function, a compilation of what was known at the time.

Note on the Very Common Nervous Condition Wrongly Attributed to Brain Congestion (1861)

Landry reported a few observations collected with his colleague Nicolas Samazeuilh, in his hydrotherapy establishment in Auteuil, detailing the treatments offered there. They refuted diagnoses of ‘brain congestion’ in cases of walking instability, vertigo or hypochondria, which they attributed to poor habits and prescribed a rich diet, cold showers and electrotherapy. ‘Certain phenomena of this morbid condition have distant links with those of a paralysis of muscular feeling [...]. It is our opinion that this form belongs nosographically with the neuroses, and is more closely aligned with the vertiginous diseases than with any other morbid condition’ [54].

Conclusion

Demonstrating a lack of gratitude, Jean Delay (1907–1987) only paid a modest homage to Landry in his monumental thesis on astereognosis, defended in 1935. As for Guillain, he adamantly defended his own primacy, and in 1953 rejected the designation ‘Landry-Guillain-Barré’ syndrome. However, it is time for the history of neurology to honour this outstanding pioneer [55–57].

Webb Edward Haymaker (1902–1984) prepared a book on the history of neurology for the 14th International
Neurological Congress held in Paris in 1949. It was entitled ‘The founders of neurology: one hundred and thirty-three biographical sketches’. In it, Haymaker gives Landry his rightful place by counting him among the 133 founding fathers of neurology. Though his peers are in many cases better known, Landry richly deserves this recognition [3, 4].

Acknowledgments

Prof. Jacques Poirier and Dr. Jean-Pierre Luauté for their critical revisions. Mrs. Bernadette Molitor and Stéphanie Charreaux (Bibliothèque inter-universitaire de Santé à Paris) for their patience and help with our research.

References

42 Bourdon H: Nouvelles recherches cliniques et anatomiques sur l’ataxie locomotrice pro- gressive. Arch Gén Méd 1862;1(5e série);19: 385–406.