The history of thalamic pain syndrome: early descriptions and the seminal contributions of Jules Dejerine, Gustave Roussy, and André Pierre-Marie

(History of Neurology)

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Abstract

It was not until the nineteenth century that the high number of tabes dorsalis cases enabled a better understanding of the physiology of sensitivity, which linked nociceptors, already known at that time, to the peripheral, medullary, and central conduction pathways. The thalamus had initially been considered a structure involved in motor control, based on vivisection experiments, but when its vascularisation from the posterior cerebral artery was identified, Jules Dejerine and Gustave Roussy were able to describe the clinical symptoms of thalamic stroke. The eponymous Dejerine-Roussy syndrome (1903–1905) bears witness to their contribution. Gustave Roussy's 1907 thesis on thalamic syndrome is the first actual work to highlight pain of central origin. In 1924, Roussy assisted André Pierre-Marie, the son of Pierre-Marie, in writing his thesis, which laid down the semiology distinguishing thalamic central pain from potentially painful sensory deficits of cortical origin, based on the treatment of numerous patients with parietal injuries during World War I. This article discusses the many studies and controversies, which, during the last decade of the nineteenth century and the first quarter of the twentieth century, made it possible to understand the central physiology of sensitivity, its pathological deficits, the excruciating experience of hyperpathia, and the associated abnormal movements in some cases.

Keywords: History of Neurology, Thalamic Syndrome, Dejerine-Roussy syndrome, Central Pain syndrome, Pierre-Marie, Gustave Roussy, André Pierre-Marie.

Introduction

For centuries, philosophers alone dealt with pain while religious precepts exclusively ingrained their conceptional domination. It was not until the nineteenth century that the high number of tabes dorsalis cases enabled a better understanding of the physiology of sensitivity that linked nociceptors, already known at that time, to the peripheral, spinal, and encephalic conduction pathways. Anatomoclinical observations established that any lesions in pain pathways logically determined the suppression of pain, but also, paradoxically, they could in some cases cause a considerable amplification of painful sensations: hyperpathia (*Canavero and Bonicalzi 2011*). Thus, until around the 1890s, pain was studied as a global entity without any explicit distinction being made between peripheral and central conduction. From 1890 to approximately 1925, the concept of centrally caused pain was connected exclusively with thalamic lesions. But from that point forward and up till now, centrally caused pain was and is no longer considered thalamic alone but also spinal, subcortical, and cortical, incorporating the role of the sympathetic system, the bulbo-protuberantial region (trigeminal involvement), and the parietal cortex (*De Smet 1986*).

In this study, we aim to elucidate the history of the description of thalamic syndrome between 1890 and 1925, based on clinical data along with pathological anatomy. This syndrome explains disturbances of sensitivity, previously undifferentiated from cortical deficits. One man played an eminent role in conducting this research, Gustave Roussy (1874–1948). He credited his contributions to his first teacher, Jules Dejerine (1849–1917), as the eponymous syndrome of Dejerine-Roussy attests, and to the successor to the Chair of Nervous System Diseases, Pierre-Marie (1853–1940), even though the hostility between these two great neurologists was well and widely known. Something that is not well known is the fact that Roussy specifically helped André Pierre-Marie (1891–1929), son of the famous third successor to Jean-Martin Charcot (1825–1893), in the writing of his doctoral thesis, focused on the distinction between thalamic syndrome and other types of central pain. We will examine the studies conducted during this period, as André Pierre-Marie's thesis remains a little-known summary of them (*de Ajuriaguerra, 1939*).

Seminal description of thalamic syndrome

Inaugurating the anatomoclinical method, Nicolas Saucerotte (1741–1814) was the first to attribute, in 1768, a function of motor control of the limbs to a structure he called the "optic layers" (optic tract or optic nerve colliculus ["thalami nervorum opticorum"]), based on his examination of soldiers with cranial trauma (*Saucerotte 1801*). This name resulted from an error attributing, since antiquity, a relationship between the thalamus and the optic nerves, whereas we now know that the optic nerves are linked to the lateral geniculate nucleus. Indeed, this structure had been considered since Galen (129–ca 216) and up to the eighteenth century as the originating point of the optic nerves, according to François Chaussier (1746–1828) (*Chaussier 1807*). "Layers" meant in this case the "place of origin". Galen used the term "thalamus" ("θάλαμος"), which means, in Greek, literally "bed" (couche in French) and referred metaphorically to the "place of origin" (*Garcia-Cabezas et al. 2021*). This term 'thalamus' remained in international use whereas Franz Joseph Gall (1758–1828) proposed in 1810 the term "large inferior cerebral ganglia", which fell by the wayside (*Gall and Spurzheim 1810*).

Étienne Serres (1768–1868) (Serres 1824), François Magendie (1783–1855) (Magendie 1828), and François-Achille Longet (1811–1871) (Longet 1842) conducted vivisection experiments in

dogs, unilaterally damaging the thalamus and triggering whirling movements. But both the size of the lesions produced and their localisation were imprecise. The brief time these animals survived made detailed observations of any behavioural modifications impossible. In 1866, in one of his lessons, Alfred Vulpian (1826-1887) wrote, "These bulges are thus intermediate parts of the brain strictly speaking and of the isthmus of the encephalon, and their functions are undoubtedly different from those of the brain. But it must be admitted that we have only very limited and incomplete data on the nature of their functions" (*Vulpian 1866*).

In 1873, Édouard Fournié (1833–1886) improved the experimental technique, using a fine needle to inject a caustic solution, such as zinc chloride coloured blue by aniline. After initial convulsions, he thus demonstrated complete abolition of sensitivity contralateral to the injected side: "The centre of simple perceptions, where all conductive fibres end, and its destruction, which coincided with the loss of all senses and the conservation of movement, enables us to assert that the point where the driven movement is transformed into perception or into something felt is found in the optic tract" (*Fournié 1873*). Once again, the brief time these animals survived did not formally validate his conclusions, which resemble extrapolated reasoning rather than scientific proof. Starting in 1859, however, the Austrian Ludwig Türck (1810–1868) published four observations of hemianesthesia, the autopsy revealing lesions of the "optostriated centres" ("centres opto-striés"); that is, the optic tract (thalamus) and striated bodies (striatum) (*Türck 1859*).

Raphael Veyssière (1844–1885), a student of Louis Ranvier (1835–1922), explained in his thesis on August 6, 1874, with Charcot presiding over the jury (*Veyssière 1874*), that Jean-Baptiste Demeaux (1815–1886) was the first, in 1843, to propose an observation of hemianesthesia (*Demeaux 1843*). Veyssière had heard the 1873 lessons of Charcot, in which he examined the famous hysteric Justine Etchevery (in reality Etchegoyhen, 1828–1905, who became a nurse at La Salpêtrière Hospital after Charcot's death), who had hemianesthesia as designated by the master of hysteria (*Charcot 1872-1873*). Veyssière then decided to focus his thesis on this subject, being a former resident under Vulpian. Choosing to use human pathology as a guide to experimental pathology, Veyssière, with the help of Henri-Camille Carville (1828–1885), head of Vulpian's laboratory, added sixteen experiments on rabbits and dogs to the twenty observations compiled by his teachers. After partial ablations of the cortex and localised caustic injections, he concluded: "To cause hemianesthesia by a cerebral lesion, this lesion must act on the cerebral peduncle". For him, the lesion in the "optic layers" did not produce hemianesthesia but rather involuntary movements (shaking, hemichorea).

On March 6, 1874, Charcot presided over the thesis of Louis-Alexis Virenque on hemianesthesia. Virenque tried to distinguish between hemianesthesia of hysterical origin and that of organic origin. The latter was persistent, always meeting the same deficit criteria and accompanied by hemiparesis or hemiplegia on the same side. Virenque reported that for Charcot, the origin was a peduncular lesion (*Virenque 1874*).

According to the Scottish David Ferrier (1843–1928), basal ganglia formed a link between order and action for unconscious autonomic functions whereas complex actions required hemispheric involvement (*Ferrier et Duret 1873 1874*). Henri Duret (1849–1921) translated several articles by Ferrier (*Ferrier 1876; Ferrier 1874*) and established, with Carville, a summary in *Archives de Physiologie* in 1875 (*Carville and Duret 1875*), which showed that for Ferrier, "the optic tract was a sensory centre" (*Ferrier and Duret 1873*).

In 1876 Fulgence Raymond (1844–1910), in his thesis on hemichorea and hemianesthesia, mentioned, in only two observations of forty-four, damage to the optic tract, without noting the

existence of pain accompanying the hemianesthesia, of which he identified the cause as a lesion of the corona radiata. He assumed that the occipital cortex was the seat of sensitivity (*Raymond 1876*).

In 1877, the German Herman Nothnagel (1841–1905) replicated Veyssière's experiments and concluded that the thalamus was "a centre where the sensory perceptions transform into movements (*Nothnagel 1877*). Jean-Abel Lafforge (1851–?), opposing the results of the 1865 studies of Jules Luys (1828–1897) (Luys 1865), affirmed in his 1877 thesis that the thalamus was exclusively dedicated to motor responses and had no sensory function (*Lafforgue 1877*). He indicated, and rightly so, the difficulty of finding lesions only affecting the thalamus without involvement of the internal capsule. As for Albert Pitres (1848–1928), who also contested the proposals of Luys as a spokesperson of Charcot, he admitted with regard to the "optostriated bodies": "We do not yet know the functions" (*Pitres 1877*).

In 1887, Vladimir von Bechterev (Владимир Михайлович Бехтерев, 1857–1927) stimulated the thalamus of diverse animals by pricks and electrification. He concluded that "the optic tract is in relation to the function of innate involuntary movements and complicated reflexive movements. It is the centre of reflex movements of expression" (*Bechterew 1887*), that is to say, facial expressions. For him, it also acted on the digestive motor function, the bladder, and other autonomic functions.

The German Ludwig Edinger (1855–1918) was the first to introduce in 1891 the concept of central neuropathic pain in describing the case of a 48-year-old woman with "heftige Schmerzen und deutliche Hyperaesthesie in den gelaehmten Glieden" (violent pains and clear-cut hyperesthesia in the paretic limbs: the right arm and leg). Her terrible pain, unalleviated by morphine, led her to commit suicide (*Edinger 1891*). During the autopsy Edinger discovered an infarct at the external nucleus of the thalamus and a part of the pulvinar contralateral to the pain. The pain of this patient was associated with hemiathetosis and hemianopsia contralateral to the infarct.

Frederick Greiff (?–?) of Heidelberg published in 1893 an observation of hemiathetosis accompanied by hyperaesthesia and extreme pain. The autopsy revealed an infarct of the posterolateral ventral nucleus of the thalamus (*Greiff 1893*).

In 1898, Jean Sellier (1862–1935) and Henri Verger (1873–1930) precisely destroyed some parts of the thalamus by electrolysis: "The lesions, even small lesions, in the thalamus lead to the typical syndrome of hemianesthesia, said to be organic" (*Sellier and Verger 1898*).

In 1900, Adolf Wallenberg (1862–1939) added a case of pain in the face in the trigeminal territory after infarct of the ventro-posterio-median nucleus of the thalamus (*Wallenberg 1900*).

In 1902, Joseph Babiński (1857–1932) and Jean Nageotte (1866-1948) published a case of syphiloma that had infiltrated the medulla oblongata and was responsible for a complex clinical picture with hemiparesis and hemianesthesia affecting touch and temperature and with disorders of ocular and cerebellar motor control. Their conclusion was modest; they were content to merely add their contribution to understanding conduction pathways (*Babiński and Nageotte 1902*).

While Augusta Klumpke-Dejerine (1859–1927) and Jules Dejerine did not clearly describe the spinothalamic tract in their *Anatomie des centres nerveux* (anatomy of nervous centres) (1895–1901) (*Dejerine and Dejerine-Klumpke 1895-1901*), their student Édouard Long (1868–1929) analysed on it in his 1899 thesis (*Long 1899*), but Dejerine recognised this sensory pathway in

his *Sémiologie des affections du système nerveux* (Semiology of Nervous System Diseases) in 1914 (*Dejerine 1914*).

Contribution of Dejerine and Roussy

Observing in 1903, at the Bicêtre hospital, two patients with hemiparesis but especially with hemianesthesia affecting touch and proprioceptive sensitivity ("very weak hemiplegia; excessive hemianesthesia"), Dejerine and the Swiss Léon Charles Egger (1866-1962) localised the origin of these disorders in the optic tract: "The lesion [is located] in the region where the sensory tract and the motor tract are separated from each other" (*Dejerine and Egger 1903*). They indicated the associated pain, but briefly, as if this was not important, in order to dwell on hemiataxia. André-Thomas (1867–1963) and Maurice Chiray (1877–1954), both students of Dejerine (Figure 1), published in 1904 an autopsied case explicitly named as thalamic syndrome: mild hemiplegia, choreoataxia, and athetosis, which appeared with the disappearance of paralysis in the same territory, and marked deficits in sensitivity, mainly deep sensitivity, but without indication of pain (*Thomas and Chiray 1904*). After these first two cases, Maurice Dide (1873–1944) and Albert Durocher (1879–1906) added in 1904 a case of mild hemiparesis associated with a significant global sensory deficit. The autopsy found a stroke affecting the thalamus and the internal capsule, which limited the demonstrative relevance (*Dide and Durocher 1904*). Be that as it may, organic hemianesthesia was nonetheless affirmed.

Figure 1. From left to right: André-Thomas, Jules Dejerine, and Maurice Chiray in 1904.

On December 7, 1905, Roussy presented at the Society of Neurology of Paris the observation of limited cerebral haemorrhage "at the optic tract without participation of neighbouring encephalic parts" (*Roussy 1905*). He was careful to note that this first case he was reporting was incomplete. Despite survival for twenty days, mental confusion made it impossible for him to identify sensory deficits or evaluate any pain felt.

The following year, on June 7, 1906, he presented with his teacher Dejerine two new clinical and anatomicopathological cases, which, along with three other recently published cases by other authors, made it possible to establish the complete clinical semiology of thalamic syndrome: "A lesion localised at exactly the same point of the optic tract corresponds to an

identical symptomatic clinical picture" (*Dejerine and Roussy 1906*). This thalamic syndrome was characterised by:

"(1) mild hemiparesis usually without contraction and rapidly regressive".

(2) persistent superficial hemianesthesia organic in nature which can in some cases be replaced by cutaneous hyperaesthesia, but is always accompanied by marked and persistent disturbances of deep sensitivity;

(3) mild hemiataxia and more or less complete astereognosis. The following are ordinarily added to these principal and constant symptoms:

(4) sharp, severe, persistent, paroxysmal, and often intolerable pain on the hemiplegic side that does not respond to any analgesic treatment;

(5) choreoathetotic movements in the limbs on the paralysed side".

They added possible sphincter disturbances and hemianopsia to their list. This clinical picture revealed its full purity a few months after the stroke. They did not note the deficit of mimicry seen by Bechterev. Hemichorea, not significant, and hemiathetosis were frequent, especially in the fingers of the paretic hand. Shaking was absent. Hemiataxia was constant, mild, and restricted, quite different than the tabetic ataxia that was at that time so widespread. The disorders of sensitivity "took on major importance". The three sensations—touch, pain, temperature—were objectively impaired but never totally lost. Proprioceptive sensitivity was much more deficient.

But subjective disturbances especially were foremost, with early installation pain affecting not only the paralysed limbs but also the face and trunk. Patients had much difficulty in accurately locating it, but its continuous nature with paroxysmal exacerbations after simple grazing was typical. Patients cried out in pain, which left them unable to sleep or woke them with a jolt. "Pain impotence" as Dejerine said, the incapacity to made anything, was the result.

Dejerine and Roussy distinguished this clinical picture from that recently described by Fulgence Raymond (1844–1910) and Raymond Cestan (1872–1933); that is, upper pontine syndrome, during which ocular paralysis and shaking were foremost, while they were absent during thalamic syndrome (*Raymond and Cestan 1903*).

They concluded: "A lesion in the optic tract, involving the posteroexternal part of the external nucleus and covering part of the median and internal nuclei as well as the corresponding fragment of the internal capsule, results in the same clinical picture in all cases. This collection of symptoms constitutes a new syndrome that must be included in the nosology: the thalamic syndrome". They ended by referring to a later study that would turn out to be Gustave Roussy's thesis.

Gustave Roussy's thesis

In 1907, Gustave Roussy defended his thesis, entitled: *La couche optique (étude anatomique, physiologique & clinique), Le syndrome thalamique* (the optic tract [anatomical, physiological, and clinical study], thalamic syndrome), a monumental, 371-page work (*Roussy 1907*). After a long historical chapter, Roussy described the anatomy of the thalamus, notably its arteries, then its various nuclei. He examined the connections, mainly citing the studies of Augusta Dejerine-Klumpke published in 1901 in *L'Anatomie des centres nerveux* (*Dejerine and Dejerine-*

Klumpke 1895-1901). Roussy discussed his own anatomicopathological experiments at length. After localised destructions of the thalamus in cats and dogs, he sacrificed eight monkeys, only one of which lived long enough, to his great disappointment, to be studied first clinically and then using pathological anatomy. He illustrated his dense text with numerous drawings and photographs (Figure 2).

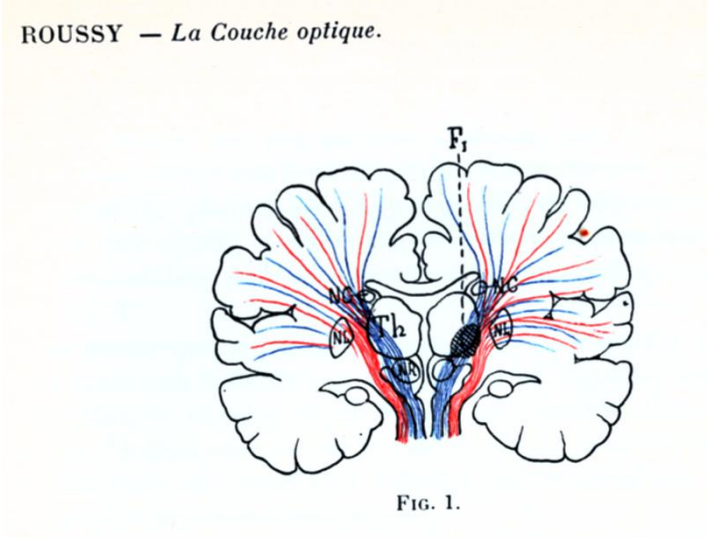

Figure 2. Illustration from Roussy's thesis localising thalamic syndrome, F1 = blue fibres.

The second part of the thesis focused on clinical aspects. Roussy explained how he drew inspiration from the thesis that André-Thomas defended in 1897 and that established the semiology of cerebellar syndrome (*Thomas 1897*); this led Roussy to establish in his thesis a precise semiology of the thalamic syndrome. Bringing together the observations published by Dejerine (*Dejerine and Egger 1903*), André-Thomas, Chiray, and other observations, ten cases in all, he used Dejerine's 1903 description nearly word for word to construct "an identical clinical picture corresponding to a localised lesion at exactly the same point of the optic tract", which proved "the existence of a new clinical syndrome determined by lesions in the optic tract, or the thalamic syndrome".

Roussy established the thalamic relay of sensory pathways: "To produce this kind of syndrome, it is not enough that the thalamus is damaged; this lesion must be located in the special region of this ganglion; that is, in front of the pulvinar, in the posterior and lower part of the external nucleus of the thalamus, which is the region of the Reil ribbon", i.e. also known as Reil's band or the medial lemniscus. This pathway in the brainstem carries sensory information from the gracile and cuneate nuclei of the medulla oblongata to the thalamus, and as internal arcuate fibers they cross to the opposite side in the sensory decussation.

Roussy added to the symptoms previously noted the rarity of the Babiński sign and epileptoid shaking, and noted the possibility of associated hemianopsia. He stressed the distinction between the most frequent forms of hemiplegia where motor systems are foremost and sensory disorders minor and rapidly resolved, and thalamic syndrome, which could be seen as the mirror image, with the foremost symptoms being transient paresis, hemianesthesia, and excruciating

pain. Roussy ended his thesis with a discussion, partly hypothetical, of the pathophysiology of the syndrome and notably its painful phenomena.

Since his thesis, posterity interchangeably uses the name "thalamic syndrome" and the eponym "Dejerine-Roussy syndrome" (*Cambier 1982*). The Czech physician Ladislas Haskovec (1866–1944) reported a case of thalamic syndrome in 1904 and was the first to name it "Dejerine syndrome". Roberto Malassongo (1857–1919) of Padua, a pupil of Charcot, was the first to use the full term "Dejerine-Roussy thalamic syndrome," as reported by Philippe Pagniez in the French journal "La Presse Médicale" in 1907, in a review of an article published in "Il Policlinco sezione medica" (Roma).

Roussy, a former resident under Dejerine and Pierre-Marie, was appointed, one year after his thesis, to head the pathological anatomy studies at the Paris Medical School, when Pierre-Marie held the Chair of Pathological Anatomy. He succeeded in 1925 Maurice Letulle (1853-1929), who had himself succeeded Marie.

It was then in 1924, that André Pierre-Marie (1891-1929) focused his thesis on distinguishing sensory deficits of cortical origin from those secondary to a thalamic lesion.

Brief biography of André Pierre-Marie

André Henri Pierre-Marie (Figure 3), born on 28 March 1891 in Paris, was the son of Pierre-Marie. Pierre Marie, born with a single first name, has a widely used surname that is also a first name. The brevity of his first and last names led to him being referred to as Professor Pierre Marie far more often than Professor Marie. Over time, Pierre became part of the family name, becoming Pierre-Marie (*Poirier and Poirier 2023*). His son André took advantage of this evolution, calling himself André Pierre-Marie. He was a resident under his father at La Salpêtrière for two years (*Poirier and Poirier 2023*). He defended his thesis in 1924, with Georges Guillain (1876–1961) presiding over the jury; Guillain had succeeded his father the previous year as the holder of the Chair of Nervous System Diseases.

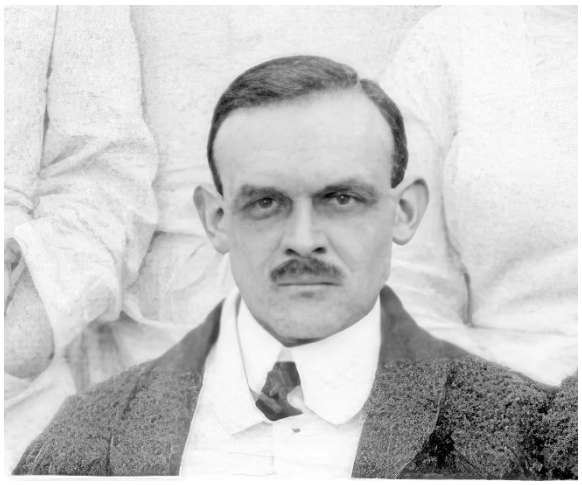

Figure 3. André Pierre-Marie, resident in 1922 at La Salpêtrière Hospital.

He pursued his career at the Pasteur Institute where he focused on digestive infections and intoxications. It is likely that André Pierre-Marie chose to focus on bacteriology research to avoid any comparison with his father. During an experiment, botulinum toxin entered his eye, and he died of botulism a few days later, on 11 July 1929 (*Anonymous 1929*). The release of classified documents has shown that experiments conducted at that time were part of military research involving bacteriological warfare (*Aucouturier 2011*). André Pierre-Marie's main publication remained his thesis (*Pierre-Marie 1924*).

On André Pierre-Marie's thesis

Gilbert Ballet (1853–1916), Charcot's resident in 1880 and his first senior resident in 1882, defended an innovative thesis on 29 March 1881 entitled: *Recherches anatomiques et cliniques sur le faisceau sensitif et les troubles de la sensibilité dans les lésions du cerveau* (anatomical and clinical research on the sensory tract and sensory disturbances in brain lesions) (*Ballet 1881*). At a time when the motor tracts started to be well identified, Ballet observed: "Obscurity still reigns on more than one point of the pathway of centripetal fibres, which pathology has insufficiently elucidated". In 1865, Jules Luys had asserted that all sensory fibres, grouped in the peduncle, terminated in the thalamus. In addition, some of them appeared to have an unknown cortical projection. In 1872, Theodor Meynert (1833–1892) had identified the anterior parietal cortex (primary somatosensory areas) as the recipient of sensitivity (*Meynert 1872*). In 1875, Charcot noted this in one of his lessons on cerebral localisations that examined the trajectories of sensory pathways to the thalamus and its cortical projections by citing the numerous authors who had participated in the slow progress toward this understanding (*Charcot 1875*). At no point in his thesis did Ballet refer to thalamic damage as the cause of hemianesthesia, only describing damage to the internal capsule, without indicating whether it also affected the thalamus, associated or disassociated with a retro-Rolandic cortical lesion.

André Pierre-Marie was assisted by Henri Bouttier (1888–1923), his senior resident, in carrying out his work. He noted as an introduction that opinions on central sensory deficits were controversial. The examination of numerous wounded soldiers in World War I, especially from 1914 to 1915, before the Adrian helmet was adopted, called into question what seemed established before the war. The cruel war brain injuries of these soldiers, equivalent to vivisection, previously unimaginable, led to more in-depth knowledge.

For example, at the 07 January 1915 session of the Society of Neurology of Paris, Dejerine and Jean Mouzon (1892–1964) presented two patients trepanned at the parietal level. Sometime after the initial accident, there was paresis of the limbs accompanying hemianesthesia, which affected sensitivity to touch, temperature, and pain, causing astereognosis by anaesthesia, which they named "cortical sensitivity syndrome", now referred to by the eponym Dejerine-Mouzon syndrome (*Dejerine and Mouzon 1915*).

Chiriachitza Athanassio-Bénisty (1885–1938), born in Brăila, was the first Romanian woman to receive a literature-philosophy degree from the Academy of Paris in 1906 (*Anonymous 1906*). She was a resident under Pierre-Marie throughout the war and defended a remarkable thesis in 1918, focused on lesions in the Rolandic region (*Athanassio-Benisty 1918*). She described cases of apparently radicular anaesthesia caused by cortical lesion, affecting touch and tactile discrimination but with the stereognostic sense intact. Dejerine and Mouzon had already described, at the 04 November 1915 session of the Society of Neurology, the sequela "of a slight bone indentation by projectile weapons", causing anaesthesia, pseudo-radicular in type, which

they compared to symptoms secondary to those of a cortical tumour in a gyrus of the anterior parietal cortex (*Dejerine and Mouzon 1915*).

On 01 December 1921, Jean Lhermitte (1877–1959) and Paulin Fumet (1891–1931) presented to the Society of Neurology of Paris the case of a woman with regressive hemiplegia accompanied by a major, persistent sensory deficit, affecting all modes, without pain, with only paraesthesia in the limbs. They suspected cortical damage and were surprised with the autopsy, which only revealed localised thalamic haemorrhage (*Lhermitte and Fumet 1921*).

At the 07 December 1922 session of the Society, André Pierre-Marie and Henri Boutiter, with the help of the anatomicopathologist Ivan Bertrand (1893–1965), presented the case of "dissociated thalamic syndrome" (*Bouttier et al 1922*), which André Pierre-Marie used in his thesis with photographs.

For around fifteen years, the patient had complained of gradually worsening weakness in his upper left limb, headaches, and paraesthesia, with sharp pain in this arm. This was followed by sudden worsening with spastic contractions and increased, intolerable, grinding pain throughout his arm. The examination found tendon hyperreflexia in the left arm, but the Babiński sign would never return in this man. There was very little involvement in the lower left limb. "The degree of pain is the best sign of damage to the optic tract". During the autopsy, they observed "a whitish, fibro-cretaceous nodule that crunched when cut; it is found at the posteroexternal part of the thalamus and runs along the inner face of the pyramidal tract"; this nodule also extended along the subthalamus. There is a contrast between the degree of the contraction and the absence of damage to the pyramidal system.

At the beginning of 1922, Pierre-Marie and Bouttier had already mentioned "the dissociations of sensitivity in encephalic lesions; that is, all the possible associations or lack of associations of deficits in touch, temperature, pain, or deep sensitivity, to various degrees for each modality" (*Pierre-Marie and Bouttier 1922*). And for thalamic pain, they stressed the dissociations in pain phenomena. They considered hyperalgesia in response to cold as pathognomonic.

André Pierre-Marie discussed at length and in great detail the clinical methods he used to test sensitivity. For example, he used a compass developed by Ernst Heinrich Weber (1795–1878) in 1834 (*Weber 1834; Nicolas and Vobořil 2019*), to test the stereognostic sense, etc. To distinguish between parietal and thalamic syndromes, he first provided two observations of sensory disturbances of cortical origin to exemplify dissociations between the types of sensitivity; some could be abolished while others remained normal. Then he discussed three clinical cases to describe the various sensory disturbances of thalamic origin. At this point, he referred to the works of Henry Head (1861–1940) and the distinction Head made between cortical, elaborate, fine, and precise perceptions and basic perceptions, "which can only assess so-called protopathic sensations, in a way unconscious" (*Pierre-Marie 1924*). André Pierre-Marie illustrated Head's theory (*Head 1918*) by a clinical case with major disturbances in superficial sensitivity but with the integrity of deep sensitivity intact. He named this clinical picture "dissociated thalamic syndrome". When both types of sensitivity were abolished, he spoke of "global thalamic syndrome".

André Pierre-Marie illustrated a case presented to the Society of Neurology by Bouttier; it was one the clinical varieties Marie wanted to distinguish. He spoke of subthalamic syndrome when hemiparesis, hemianesthesia, hemiasynergia, hemianopsia, and athetotic movement are associated, but without spontaneous pain.

André Pierre-Marie completed his clinical descriptions with a reference to nociceptive hemiagnosia; that is, "the incapacity to discern the quality and the point of application of painful stimuli" (*Pierre-Marie 1924*), which is to say the tactile or pain asymbolia, an expression recommended in 1906 by the Swiss Édouard Claparède (1873–1940) (*Claparède 1906*). When the patient has no elementary sensory anomaly and perceives pain stimulation, but cannot identify by palpation a familiar object, the syndrome is designated by the eponym "Verger-Dejerine syndrome", in memory of the 1900 study of Henri Verger (1873–1930) of Bordeaux (*Verger 1900*). In such cases, the patient with damage to the insular cortex will say, for example, he can feel the pain of a pinprick but that it no longer hurts.

After having written the "disturbances of sensitivity" chapter published in the voluminous *Pratique neurologique* (neurological practice), directed by Pierre-Marie in 1911 (*Pierre-Marie 1911*), in 1921 Roussy held a large conference at the Medical School, presenting the latest knowledge accumulated since the start of the century and greatly enriched by the treatment of wounded soldiers. He brought together the anatomy of sensory pathways and centres, the physiology of various types of sensitivity, and all the enumerated pathological disorders (*Roussy 1922*). Roussy demonstrated his adherence to Head's conceptions: "The thalamus is the centre of the development of some sensory impulsions, and secondary and elementary impulsions, controlled by higher centres". It is likely that André Pierre-Marie drew from this the essential elements necessary for developing his thesis.

In 1928, Pierre-Marie published two volumes of "travaux et mémoires" (works and recollections), bringing together his essential works in the form of a testament for the history of neurology, and as a kind of illustration of his personality. In the section on nociceptive hemiagnosia in recent hemiplegia, he wrote: "I pointed out the singular phenomenon of nociceptive hemiagnosia in 1903 or 1904, I demonstrated it before numerous series of students, and I mentioned it in my *Traité de Médecine sur la Pathologie du Cerveau* (medical treatise on brain pathology) (cf. *Pratique neurologique*, probably). Drawing on this teaching, my son André Pierre-Marie, in his doctoral thesis, gave a methodical description of this phenomenon in a special chapter, page 75. This is the description we use here". This text suggests the father contributed to writing his son's thesis!

In his conclusion, André Pierre-Marie asserted that it was dangerous to localise cerebral infarct based on symptoms. He considered cortical damage to exist in the presence of Jacksonian epilepsy, and damage to the thalamus to exist in the presence of choreoathetotic movements with special deformations of the fingers and hands. For him, "nociceptive hemiagnosia is a phenomenon observed frequently in recent hemiplegia . . . This very special sensory disturbance in recent hemiplegia with ictus seems to be due in large part to oedema in the cerebral hemisphere in which the lesion is produced". This conclusion is curious. He focused all his work on confirming that the pain in Dejerine-Roussy syndrome is a sign of thalamic damage, and that in contrast, cortical sensitivity syndrome is essentially due to a deficit and without pain. But he does not express this in his closing remarks.

Thereafter

After the works of Charles Foix (1882–1927) on cerebral vascularisation, particularly that of the thalamus (*Foix and Masson 1923*), Pierre Hillemand (1895–1979) defended his thesis in 1925, with Foix presiding over the jury. He indicated the vascular localisations responsible for stroke affecting the thalamus: "Each arterial pedicule corresponds to a specific clinical

syndrome" (*Hillemand 1925*). For Hillemand and Foix, thalamic syndromes were almost always secondary to damage to the posterior cerebral artery and one of its five pedicules, as confirmed by Orthello Richardson Langworthy (1897–1996) in 1937 (*Langworthy 1937*). As a result, there are as many types of thalamic syndrome as there are pedicules. The two most frequent are the syndrome secondary to a lesion in the thalamogeniculate (motor and sensory) pedicule and the syndrome secondary to a lesion in the retromammary pedicule (shaking, choreoathetosis, and sensitivity syndrome). In all cases, the intensity of clinical signs contrasts with the small size of the causal thalamic lesions. When the cause is a haemorrhage or tumour, there is no longer any systematisation. Hillemand's work opened the way to the Romanian Oscar Sager (1894–1981), who in 1933 established thalamic somatotopy (*Sager 1933*).

In conclusion and discussion

The Czech physician Ladislas Haskovec (1866–1944) reported a case of thalamic syndrome in 1904 and was the first to name it "Dejerine syndrome". That was before Roussy's contribution (Haskovec 1904). In 1907, Roberto Massalongo (1857–1919) of Padua, a pupil of Charcot (*Brigo 2025*), was the first to use the full term "la sindrome talamica di Dejerine-Roussy (Dejerine-Roussy thalamic syndrome)" in the Italian medical journal "Il Policlinco sezione medica" as reported by Philippe Pagniez (1873-1947) in the French journal "La Presse Médicale" in 1907. The eponym is since in common use in France and beyond (*Massalongo, 1907; Pagniez 1907*).

The slow genesis of the physiology of the thalamus has known numerous controversies in the past 150 years. In the 1920s, the work of Gustave Roussy mainly, and that of André Pierre-Marie, reached a decisive milestone in affirming the place of Dejerine-Roussy syndrome within neurological semiology. But the saga was not finished. Thalamic lesions are not alone in being associated with a clinical picture of pain because any structural damage to nociceptive pathways at any level may bring it about. It is now known that thalamus is the relay center of the somatosensory pathway, but any lesion that disrupts the spinothalamic tract through its course, including subcortical, capsular, lower brain stem, and lateral medulla can simulate the symptoms of Dejerine-Roussy syndrome. The specific roles of these various regions of the brain in central pain and the specific pathophysiological processes implicated remain only partially elucidated. Nonetheless, it has been demonstrated clinically and by brain imaging that damage to the ventroposterolateral thalamus cause the thalamic syndrome as Roussy and André Pierre-Marie conceived of it. The cortical regions that can also be physiologically damaged with regard to central pain are the parietal cortex but also the frontal, cingulate, and insular cortices, as well as the basal ganglia in general. The role of each of these structures in the development and persistence of central pain remains to be accurately established as well as the pathophysiology of the chronological progression of lesions and the variable change over time of the pain syndrome for similar lesions that remain unexplained.

To diagnose the Dejerine-Roussy syndrome at the present time, it is necessary that pain, in an area of the body corresponding to the lesion of the central nervous system, is associated with an history suggestive of a stroke and onset of pain at or after stroke onset. A confirmation of a central nervous system lesion through imaging or negative or positive sensory signs confined to the area of the body corresponding to the lesion is necessary, while other causes of pain, such as nociceptive or peripheral neuropathic pain, are excluded or considered highly unlikely (*Jahngir and Qureshi 2023*). The term central post-stroke pain is now preferred to describe the

neuropathic pain after stroke, i.e. thalamic syndrome, or Dejerine-Roussy syndrome, cannot be considered synonymous with all central pains.

As to the behavioural modifications secondary to thalamic lesion, which in reality are rare, they are distinguished, as set forth by André Pierre-Marie in 1924, according to the four arterial thalamic territories. Finally, we turn for elucidation to contemporary data, collected by Julien Bogousslavsky (*Bogousslavsky et al. 1988*).

The clinical picture of an anterior lesion consists in perseverations and in superpositions of unrelated information, with apathy and amnesia. After a paramedian infarct englobing the dorsomedian and intralaminar nuclei, the following occur after disturbances in consciousness: paralysis of vertical gaze and cognitive disturbances accompanying a syndrome of disinhibition, with personality changes, loss of psychic self-activation, amnesia, and, in the case of widespread lesions, true thalamic dementia, quite difficult to distinguish from psychiatric disturbances.

After an infarct of the inferolateral territory (ventrolateral nucleus, ventroposterior and ventromedian nucleus), which is the most frequent form of thalamic damage, what occurs is ataxia with hypoesthesia, aphasia, and executive dysfunction, which is difficult to demonstrate as it mimics the behavioural disorders of cerebellar lesion.

After lesion in the posterior territory of which the pulvinar is the main element, what can be observed is hypoesthesia, horizontal homonymous anopsia, sometimes the clinical picture of negligence, without a specific behavioural syndrome (*Carrera and Bogousslavsky 2006*).

Only around 7% of thalamic infarcts correspond to the clinical picture of Dejerine-Roussy syndrome; these are essentially lateroposterior, which is how Gustave Roussy and André Pierre-Marie conceived of them. A question is as yet unanswered: why do some patients develop persistent, cruel pain exacerbated by the slightest tactile stimulus while others do not, even though the thalamic lesions appear to be similar? (*Guédon et al. 2019*).

Acknowledgements

Many thanks to Julien Bogousslavsky, Francesco Brigo, Hubert Déchy and Jacques Poirier for suggestions, review, and corrections, to the reviewer for his attentive readings and suggestions and to Anna Fitzgerald for her translation.

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Specific Statements

Funding: The author received no specific funding for this work.

Conflicts of Interest: The authors declare no conflicts of interest.

Provenance and peer review: Not commissioned; externally peer reviewed. Submitted: August 30, 206. Received in revised form: September 22, 2026. Accepted: September 25, 2026.

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