Jon de Recondo, a great Basque neurologist in exile, and a digression on a case from his doctoral thesis

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ABSTRACT

Introduction. Jon (Juan José, Jean) de Recondo was born in Irún in 1929; in 1936, his family took exile in France after the outbreak of the Spanish Civil War. With great effort, he completed a degree in medicine and a residency in neurology, and eventually became head of neurology at the Sainte-Anne hospital (1982) and professor at the Cochin Port-Royal medical school at Université Paris-Descartes (1995).
Material and method. The main source of biographical data was the history book written by Jon de Recondo and his wife Anne Marie. De Recondo prepared his doctoral thesis in 1964, directed by Raymond Garcin. All neuropathological studies were conducted under the expert supervision of Jean Laprèsle, who was my own mentor. As a result of this, I was able to access a copy of Dr de Recondo's thesis and the original histological preparations; the preparations from case 2 are reviewed in this study.
Results. During his youth, de Recondo and his family were very active in clandestine organisations seeking to maintain the cohesion and the institutions of the Basque diaspora, particularly during the Nazi occupation of France. Disappointed with the course of events in the Basque nationalist movement and the birth of the terrorist organisation ETA, from 1958 he fully dedicated his efforts to his career in neurology. De Recondo maintained good relations with Basque neurologists and with the Neurology Society of the Basque Country. He published on diverse subjects in the area of clinical neurology, and edited several successful monographs. Case 2 from his doctoral thesis was that of a 56-year-old man with initial symptoms of vertigo and falls, who progressively developed a parkinsonian syndrome with associated pyramidal signs and vertical gaze palsy, with preserved automatic/reflexive eye movements. Analysis of the histological preparations suggests that the patient presented progressive supranuclear palsy, which was described around the same time by Steele, Richardson, and Olszewski.
Discussion. Jon de Recondo's personal and professional trajectory merit recognition by younger neurologists as an example of dedication to work and perseverance in the face of adversity. The review of case 2 from his thesis shows how excessive adherence to canonical nosological classifications can hinder the recognition of new clinical-pathological entities.

KEYWORDS

Recondo, Garcin, Laprèsle, spinocerebellar degeneration, progressive supranuclear palsy

Introduction

Prof. de Recondo (born in Irún, 1929) was christened as Juan José, the name appearing on letters and documents he signed in his youth. He changed his name to Jean when he became a member of French society, and finally adopted the Basque version of his name, Jon, under which he published his most important historical work (Figure 1) in the final years of his life.[1] In this study, we will refer to him by his Basque name. This evolution of his name is somewhat symbolic of the vicissitudes of de Recondo's life. Jon died in Saint-Rémy-lès-Chevreuse on 15 May 2017 at the age of 88, surrounded by his wife, three daughters, two sons-in-law, and five grandchildren. He was buried in Hendaye.

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Figure 1. Front page of the extensive history book written by Jon de Recondo and his wife Anne Marie during their retirement.

Jon de Recondo's personality and life are addressed elsewhere.[2] He was forced into exile with the rest of his family after Franco's troops arrived in Irún during the Spanish Civil War. Two of his uncles, Pepe and Juan José de Mitxelena, were among the leading organisers of the Information and Propaganda Service of the Lehendakari (president) of the exiled Basque government. Jon's life was intensely influenced by this environment of clandestine resistance, in which he actively participated as a youth. He even became president of the Association of Basque Students and was foreign representative of Euzko Gaztedi, the youth wing of the Basque Nationalist Party. He was very close to the leaders of the party, particularly the Lehendakari, Aguirre, who was a guest at de Recondo's wedding. As a result of various conflicts within the nationalist movement, and particularly the birth of the terrorist organisation ETA, he became disillusioned with politics and, from 1958, focused only on his career as a neurologist.

Rather than focusing on his political development, this study aims to raise awareness among neurologists of the figure of Dr de Recondo in this discipline and his relationships with Basque neurologists, with a digression on his doctoral thesis.

Material and methods

Biographical data were gathered from the extensive history book (Figure 1) and personal memoirs published by Jon de Recondo and his wife Anne Marie, a highly distinguished cellular biologist, during their retirement.[1] Other details are from my own recollections of meetings with Prof. de Recondo and his visits to Bilbao. De Recondo's scientific publications were reviewed on PubMed and BIU Santé Médecine.

Jon de Recondo's doctoral thesis was based on four neuropathological cases from the laboratory of Prof. Jean Laprèsle, where the author of the present article also worked; as a result of this experience, I was able to access both the text and the histological preparations. This study presents a basic review of the thesis and the histological preparations from case 2.

Results

After completing his medical degree and residency, Dr de Recondo specialised in neurology at the department run by Prof. Raymond Garcin at the Salpêtrière. It was there that he met two *professeurs agrégés* who would decisively influence his career. The first was Jean Laprèsle, who introduced him to neuropathology, particularly in diseases affecting muscles, and directed his doctoral thesis. The other was Pierre Rondot, with whom he was associated for a significant part of his career, following him to the Sainte-Anne hospital in 1974, when the neurology department was created; he later succeeded Rondot as head of the department, a role he held from 1982 to 2000. He was also professor of neurology at the Cochin-Port Royal medical school at Université Paris-Descartes from 1995.

Jon de Recondo maintained close relations with numerous Basque neurologists, whom he visited on several occasions, including at such significant events as the inauguration in 1984 of the chair of neurology of

Figure 2. Part of the presidential board at the academic ceremony held in 1984 in the great hall of Universidad del País Vasco to mark the inauguration of the chair of neurology. From left to right: Pedro Salisachs, Jean Laprèsle, Jon de Recondo, Maurice Goulon. Laprèsle and Goulon were my *patrons* during my training in Paris (1971-1973).

Universidad del País Vasco, in Leioa (Figure 2).[3] He was a particularly close friend of Javier Urkola, of San Sebastián, whom he thanked for helping secure his mother's admission to Fundación Matia when she developed dementia with severe psychiatric symptoms.[1] Dr de Recondo's last official visit with Basque neurologists was in 2004, at the annual meeting of the Neurology Society of the Basque Country in Biarritz, which he attended with his wife Anne Marie (Figure 3).

Following in his masters' footsteps, Jon de Recondo published numerous clinical works on a variety of neurological subjects, ranging from ataxias, paraplegias, and hereditary polyneuropathies to optic ataxia syndrome, myopathies, adrenomyeloneuropathy, and CADASIL. In the purest tradition of Garcin, he became interested in the neurological complications of many general diseases, such as vasculitis, lupus erythematosus, intravascular lymphoma, monoclonal gammopathies, and sarcoidosis.

Among his scientific publications, several monographs stand out. The first addresses one of his favourite semiological subjects, alterations of conjugate eye movement.[4] His interest in general clinical neurology is demonstrated in two monographs[5,6]: one on semiology and the other addressing the clinical diagnosis of neurological syndromes. Both works were very successful, with several reprints. Another two works, written in collaboration with Prof. Rondot, were dedicated to Parkinson's disease and parkinsonian syndromes.[7,8] With his wife's inestimable help, he published another monograph on muscular disease, at the time of the emergence of molecular genetics and cellular biology.[9]

Under the direction of Garcin, he presented his doctoral thesis in 1964[10]; its long title "Cerebellar atrophy complicated by atrophy of other systems, and a digression on alterations of conjugate eye movement" inspired that of the present study. The thesis gathers four cases studied at the neuropathology laboratory of Prof. Laprèsle (two

Figure 3. Jon and Anne Marie de Recondo at the 2004 Annual Meeting of the Neurology Society of the Basque Country in Biarritz (photograph courtesy of Prof. J.A. Berciano).

of which, cases 3 and 4, were previously published with Sigwald[11]). Case 1 corresponds to a patient with complex neurodegeneration (dorsal columns, corticospinal tract, dentate nucleus, and upper cerebellar peduncle), with retinal (macular) degeneration and cognitive decline (without a clear neuropathological origin). As was traditional, this case had been included in two previous theses, one on oculomotor palsy (B. Lumbroso) and one on gas encephalography (J. Lebourges), although it does not appear to have been published independently. In this study, we analyse case 2.

Summary of case 2

The patient Monsieur Q. was admitted in October 1953 and died in November 1955 (Figure 4). He had no relevant personal or family history.

Symptoms had begun two years earlier, with *vertiges* (with no cochlear symptoms) and falls, which had become increasingly frequent. He had since developed fatigue and cramps in the legs, difficulty walking, and poor hand coordination, frequently dropping objects and losing the ability to write. In the three months prior to admission, he had presented dysarthria and intermittent diplopia. Positive findings from the examination were pronounced muscular reflexes with doubtful extensor plantar reflex, diffuse rigidity with onset of "cogwheel" rigidity, intermittent horizontal diplopia, and upward gaze palsy.

Pyramidal signs continued, showing no clear progression during hospitalisation, although strong corticobulbar reflexes appeared. The parkinsonian syndrome progressed with generalised rigidity and "cogwheel" and Froment signs. Caloric and rotary chair stimulation tests indicated central vestibular dysfunction. Ocular motility worsened, with slow, irregular horizontal eye movements, vertical gaze palsy in both directions (with preserved automatic movement), and retraction of the upper eyelid. The patient died two years after admission, in 1955, in a chronic care ward at Hospice de la Salpêtrière; these wards were typical at the time at important Parisian hospitals that largely served as asylums.

In his summary of the neuropathological findings (Figure 5), de Recondo describes pronounced nigral degeneration without Lewy bodies, degeneration of the dentatorubral system (considered asymptomatic), degeneration of the oculomotor nuclei and association pathways, and degeneration/gliosis of the dorsal columns of the spinal cord.

If we review the histological preparations in the light of today's knowledge, we may highlight several details in de Recondo's findings and draw conclusions. The nigral degeneration is truly massive, with significant neuronal loss and intense gliosis. Detailed observation reveals that the surviving neurons have a rounded shape and contain an amorphous basophilic material that, in all likelihood, corresponds to globose neurofibrillary tangles (Figure 6). The red nucleus also presents near-complete neuronal loss, with the consequent glial reaction, whereas the subthalamic nucleus is less damaged (Figure 7). Neuronal loss in the globus pallidus is mild, and the putamen appears normal. The olivopontocerebellar system is normal, but the dentate nucleus presents significant neuronal loss with the typical signs of grumose degeneration (Figure 8), which is translated into demyelination and gliosis of the hilus (Figure 9) and clear atrophy of the upper cerebellar peduncle (Figure 10).

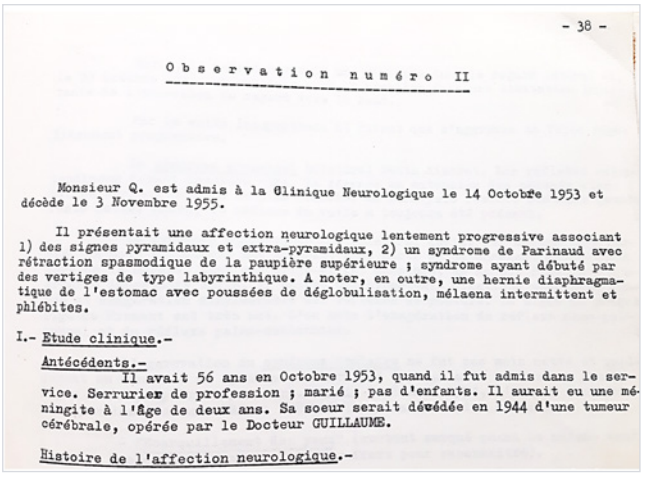
Discussion

Jon de Recondo overcame all the adversity of his difficult childhood as a refugee of the Spanish Civil War in France, and eventually enjoyed a happy family life and a brilliant hospital and university career in the field of neurology.

After his residency, Prof. de Recondo joined the service of Prof. Raymond Garcin, who had earned due recognition as one of the best semiologists in neurology at the time.[12-15] Garcin had been personally assigned a chair (*Clinique Neurologique*), from which he and his disciples rivalled the official chair, Charcot's chair of *Clinique des Maladies du Système Nerveux*, held by Alajouanine. At the time, Hospice de la Salpêtrière had lost none of its international prestige in the field of neurology. Garcin's team was dissolved following his retirement in 1968, but some members, including Laprèsle and Rondot, began working under the other chair, held by the mighty Prof. Paul Castaigne. It was at this service, which I occasionally visited, that I first met Jon de Recondo, in 1971; Prof. de Recondo was always very kind and forgave my inexperience.

The Sainte-Anne hospital had a long history in psychiatric (neuropsychiatric) care, but lacked a neurology department until 1974, when the department was inaugurated with Pierre Rondot as director and Jon de Recondo as his right hand, taking charge of the department when Rondot retired.

Garcin was the antithesis of the modern neurologist, super-specialised in a single area and, sometimes, almost in a single disease. He published over 300 articles, encompassing all areas of the discipline.[14] Rather than super-specialisation, he encouraged his disciples to take an interest in different areas ("*changer de sujet*"), frequently shifting their focus. Clearly, this is not the best method of achieving significant advances in clinical research; however, it can sustain an infinite curiosity, keeping the mind open to links between neurological disease and other disciplines, enabling neurologists to be useful and competent in a broader range of diseases and to make new, previously unnoticed clinical or clinical-pathological observations. This is a highly enriching environment for the training of a young neurologist. Jon de Recondo also followed in this tradition. While his closest mentor, Pierre Rondot, had a special interest (shared by de Recondo) in Parkinson's disease and other diseases manifesting with movement disorders,[7,8] both men published on a broad range of subjects, from neurodegenerative diseases to neurological manifestations of systemic diseases.



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Observation numéro II

Monsieur Q. est admis à la Clinique Neurologique le 14 Octobre 1953 et décède le 3 Novembre 1955.

Il présentait une affection neurologique lentement progressive associant 1) des signes pyramidaux et extra-pyramidaux, 2) un syndrome de Parinaud avec rétraction spasmodique de la paupière supérieure ; syndrome ayant débuté par des vertiges de type labyrinthique. A noter, en outre, une hernie diaphragmatique de l'estomac avec poussées de déglobulisation, mélaena intermittent et phlébites.

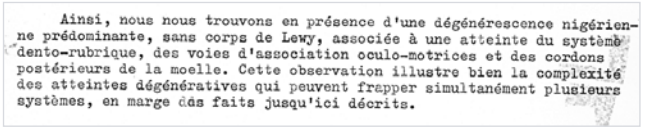
I.- Etude clinique.-

Antécédents.-

Il avait 56 ans en Octobre 1953, quand il fut admis dans le service. Serrurier de profession ; marié ; pas d'enfants. Il aurait eu une méningite à l'âge de deux ans. Sa soeur serait décédée en 1944 d'une tumeur cérébrale, opérée par le Docteur GUILLAUME.

Histoire de l'affection neurologique.-

Figure 4. The first lines of case 2 from Jon de Recondo's doctoral thesis.



Ainsi, nous nous trouvons en présence d'une dégénérescence nigérienne prédominante, sans corps de Lewy, associée à une atteinte du système dento-rubrique, des voies d'association oculo-motrices et des cordons postérieurs de la moelle. Cette observation illustre bien la complexité des atteintes dégénératives qui peuvent frapper simultanément plusieurs systèmes, en marge des faits jusqu'ici décrits.

Figure 5. The synthesis and final conclusion regarding case 2 from Jon de Recondo's doctoral thesis.

Prof. de Recondo was particularly devoted to neurological semiology, as reflected in his more personal monographs, dedicated to disorders of conjugate eye movement,[4] signs and symptoms of neurological disease,[6] and neurological syndromes.[5] These texts are highly recommended for today's young neurologists, as they remain relevant and encapsulate the unchanging basis of clinical neurology.

Prof. de Recondo was a firm believer in the instructional value, during the medical degree, of classical (and not only neurological) semiology. He told me how he had fought bitterly against the threatened discontinuation of the third-year subject general pathology in the medical degree at his faculty. Probably correctly, he considered first a theoretical understanding of the major syndromes, and subsequently their observation in practice, to be the fundamental platform for nosological analysis. As head of the neurology department, named after Raymond Garcin, at Sainte-Anne (1982-2000) and as a professor

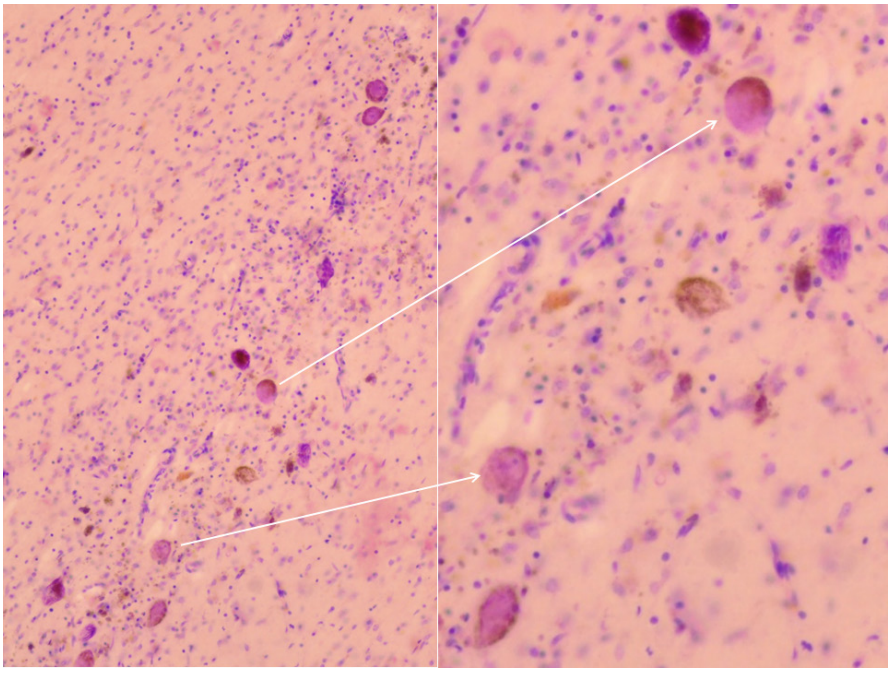


Figure 6. Case 2. Haematoxylin-eosin stain. Substantia nigra. Significant neuronal loss with replacement by intense glial proliferation. The remaining neurons are rounded in shape and contain an amorphous, slightly basophilic material compatible with globose neurofibrillary tangles (arrows). Melanin remnants and macrophages are also observed.

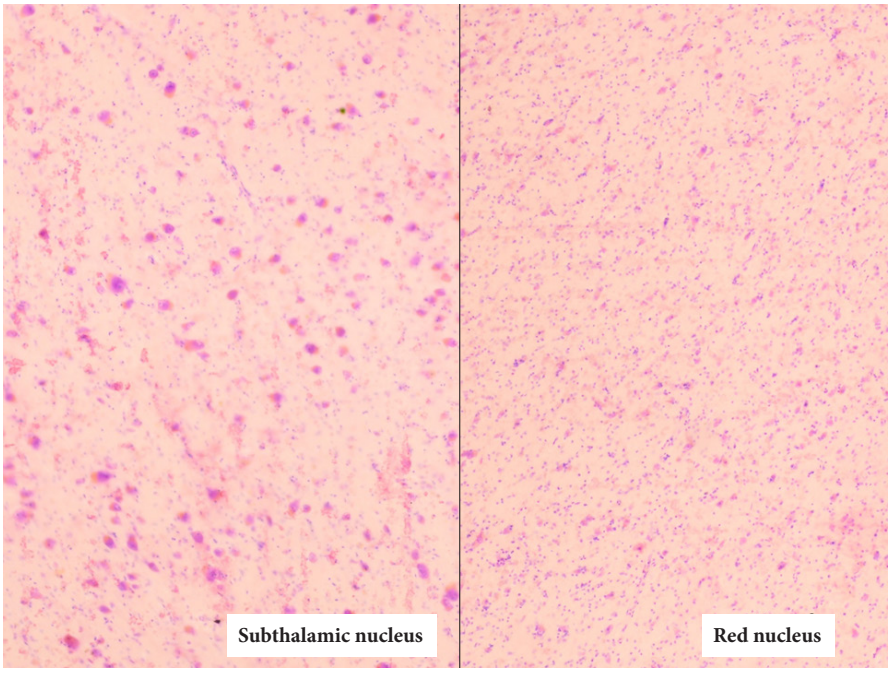


Figure 7. Case 2. Nissl stain. The red nucleus presents severe neuronal depopulation and gliosis, whereas the subthalamic nucleus is less damaged.

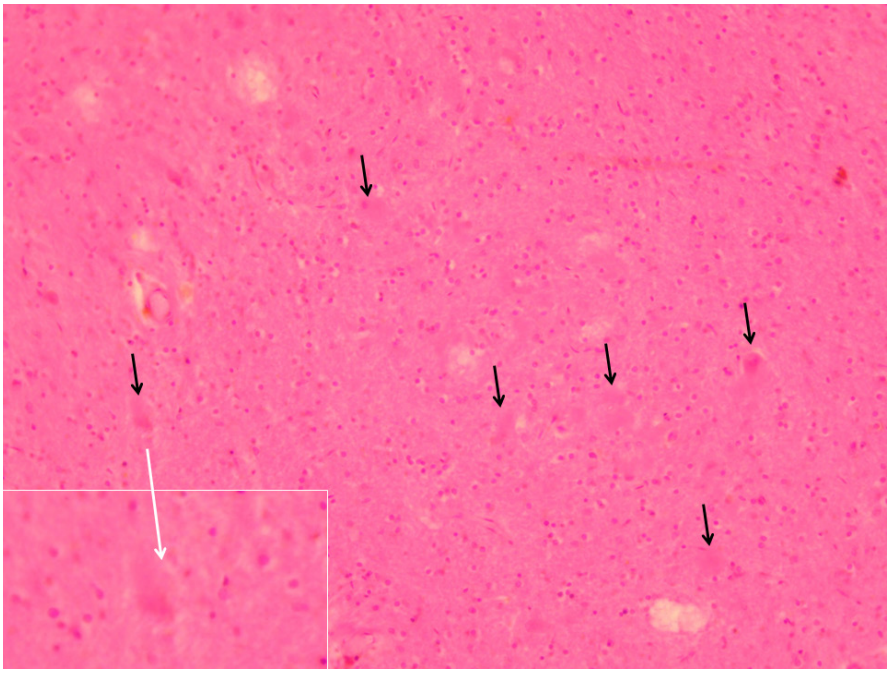


Figure 8. Case 2. Celloidin. Haematoxylin-eosin stain. Dentate nucleus. Pronounced neuronal loss. The remaining neurons (black arrows) are difficult to identify, and consist in poorly defined grumose remnants (the neuron at the left [white arrow] is shown in greater magnification in the inset box).

at the Cochin-Port Royal medical school of Université Paris-Descartes, he was able to develop all his ideas about teaching and clinical practice, with great success.

After this brief overview of Prof. de Recondo's career, let us digress briefly on his doctoral thesis, with a particular focus on one of the cases presented.

The lengthy title of the thesis announces its main conclusion: some atrophies of cerebellar systems are associated with other, systemic atrophy; this fact was widely acknowledged in the previous literature. The thesis follows the format that was traditional in France at the time. Based on clinical cases (clinical-pathological studies, on this occasion), the author presents an exhaustive review, an update on the existing literature. This was the format of the best doctoral theses, which eventually became reference works, treated as a starting point for anybody interested in the subject in future; being cited frequently was a mark of pride. In the light of this, Prof. de Recondo conducted an extraordinary review of previous publications on cerebellar atrophies, maintaining the traditional nosological classification, with the following concepts:

1. Spinocerebellar atrophy (Friedreich ataxia)
2. Pierre Marie hereditary ataxia
3. Cerebellar atrophy (Foix-Alajouanine late cerebellar atrophy, cerebello-olivary atrophy of Holmes, Ramsay-Hunt syndrome, etc)
4. Olivopontocerebellar atrophy (Dejerine, Menzel)
5. Dorsal column atrophy (Biemond), etc.

However, in his thesis, de Recondo acknowledges that, according to the neuropathological evidence, degeneration of the cerebellum and its pathways is often accompanied by degeneration of other systems. This is clearly exemplified in case 2, which was reviewed in detail for the present study.

The clinical history of case 2, "Monsieur Q.," begins with *vertiges* and recurrent falls. The French term *vertige* is less specific than the Spanish vertigo, and, in addition to rotatory vertigo due to labyrinthine dysfunction, also

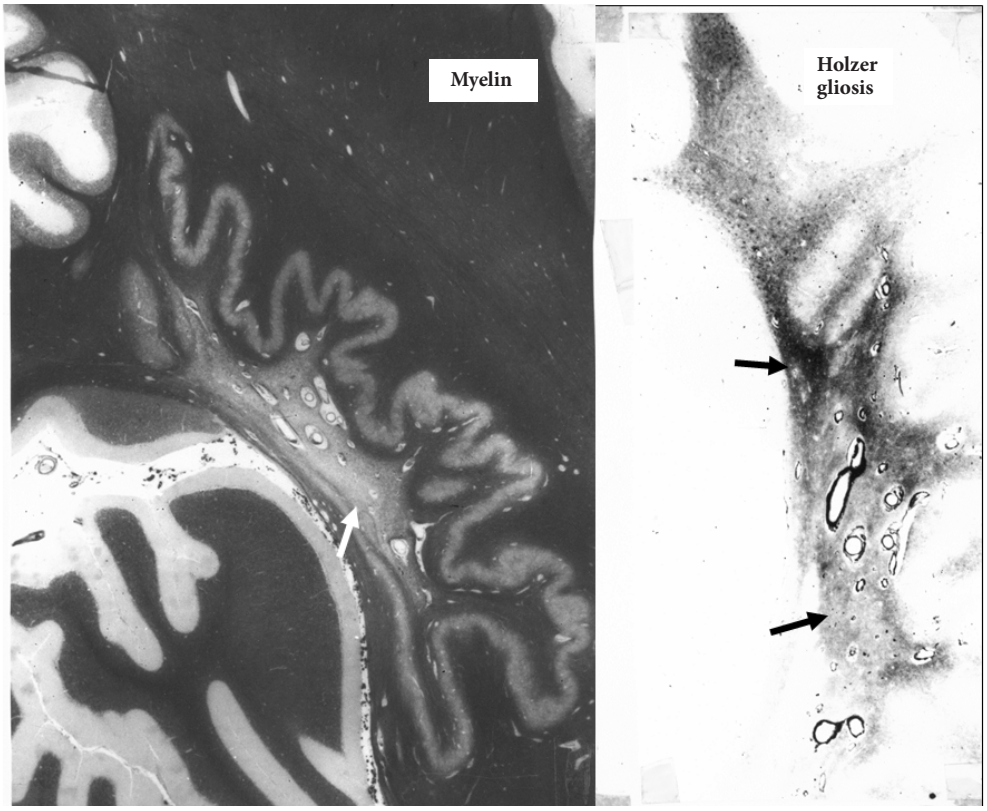


Figure 9. Case 2. Dentate nucleus. With myelin staining, the tissue of the hilus appears pallid due to demyelination, whereas Holzer staining for glial fibres is intensely positive in the same tissue.

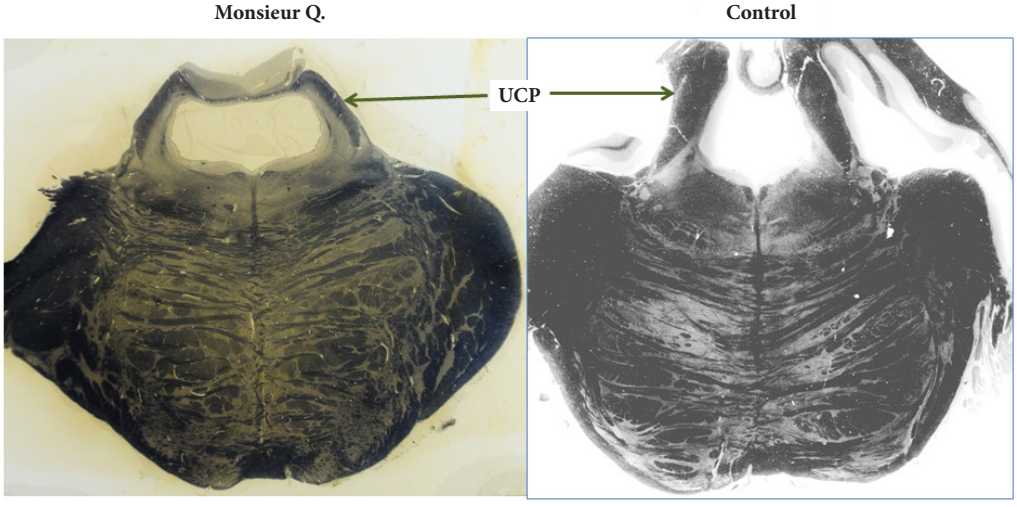


Figure 10. Macroscopic atrophy of the upper cerebellar peduncle from case 2 (left) and a healthy control (right). The dorsal corticospinal tracts are also somewhat pallid. UCP: upper cerebellar peduncle.

includes other sensations with more resemblance to dizziness/lightheadedness or imbalance than the illusion of movement. As the disease progressed, he developed vertical supranuclear ophthalmoplegia with retraction of the eyelid (causing the characteristic expression of fear or surprise), horizontal and vertical gaze palsy, dysarthria, pronounced corticobulbar reflexes, and parkinsonism. In retrospect, the patient meets all the main criteria for clinical diagnosis of progressive supranuclear palsy (PSP), as established by experts in the modern literature.[16]

This clinical diagnosis is confirmed by the neuropathology findings. The histological findings are the same as those that Dr Olszewski described as characteristic, from his earliest articles.[17,18] A noteworthy observation is the amorphous, basophilic intraneuronal deposits in the remaining neurons of the substantia nigra, giving them a globose appearance, as well as the grumose degeneration of neurons in the dentate nuclei of the cerebellum, so typical of this entity. However, in the absence of modern immunohistochemistry techniques to detect the 4R isoform of tau protein, the diagnosis of PSP is not fully confirmed.

Dr de Recondo highlighted in his conclusions that this degeneration of the dentate nucleus had no clinical repercussions. This is one of several frequent clinical-pathological inconsistencies in PSP,[19] of which we may mention three key examples. Firstly, the association between cortical neuronal lesions and cognitive decline is not constant. Secondly, we lack an explanation for early falls in patients who have not yet developed parkinsonian or cerebellar symptoms of any kind; it is not clear why this imbalance, with loss of postural reflexes of the trunk, presents so early. One possibility is that the fastigial nuclei may present similar degeneration to that observed in the dentate nucleus, which appears early but has been poorly studied; these nuclei present close anatomical links with the vestibular nuclei, potentially explaining the falls. The third inconsistency is twofold: such massive degeneration of the dentate nucleus, sufficient to cause macroscopic atrophy of the upper cerebellar peduncle, does not always present with *1)* the florid syndrome of dysmetria and intentional tremor that is nearly always observed in cases of neurodegenerative or non-neurodegenerative involvement of the dentate nucleus; or *2)* hypertrophic trans-synaptic degeneration of the olivary nucleus (which is only observed on occasion).[20]

Finally, as is typical in neurodegenerative syndromes, lesions can either affect all the structures we would expect based on the original description or the accepted patterns, or some structure may be spared. The latter circumstance would apply in this case to the subthalamic nucleus, which was near- normal in Monsieur Q., but nearly always presents lesions in PSP. However, the dorsal columns presented gliosis; this is not typically observed in the literature on neuropathology in PSP (in many cases, the spinal cord is not analysed in autopsy studies).

We should consider Dr de Recondo's final conclusion on this observation (Figure 5): "This case clearly illustrates the complexity of degenerative processes that can simultaneously affect several systems, *contrary to the facts reported to date* [emphasis added]." In other words, both de Recondo and his doctoral supervisors, Profs. Garcin and Laprèsle, were aware that this case represented a departure from previous descriptions. The thesis was published in 1964, but the autopsy study of case 2 was performed nine years earlier. Why would Garcin and Laprèsle not select this case earlier and publish it as a new type of complex neurodegenerative syndrome or disease, rather than letting it hibernate for so many years within the category of cerebellar degeneration? It is worth noting that cases 3 and 4 reported in the thesis, which were examples of complex familial multisystem degeneration, were published separately, as they were studied in the service of Prof. Sigwald, rather than in Garcin's department. The only reason that comes to mind is the excessive adherence of Garcin and his disciples to the traditional nosological classification, and a difficulty accepting changes in the established canon. An example of this resistance to change that I witnessed personally was Prof. Laprèsle's difficulty considering Roussy-Lévy syndrome as a possible familial polyneuropathy rather than as a form of spinocerebellar degeneration, the idea accepted in traditional classifications. It was very difficult for him to indicate a biopsy of the sural nerve in a patient from the family originally reported by Roussy and Lévy,[21] whom he had himself attended and followed up for years and in whom, with Pedro Salisachs,[22] he found the morphological changes typical of hypertrophic demyelinating polyneuropathy.

Clearly, Jon de Recondo was not in the position to make any decision about these observations in his thesis, but

it is a shame that he, alongside his masters Garcin and Laprèsle, should have missed the opportunity to describe PSP before Steele, Richardson, and Olszewski.

Conflicts of interest

The author has no conflicts of interest to declare. This study has received no public or private funding.

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