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THREE FRENCH PIONEERS IN RHEUMATOLOGY

BY

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As an old friend and admirer of the Heberden Society, I have enjoyed many times the privilege of hearing the Heberden Oration delivered by some outstanding physician, but it came as a great surprise to be asked to deliver the Oration this year.

I appreciate immensely this mark of friendship, and to-day I propose to record the work of three great French physicians who co-operated in laying the basis of modern rheumatology: Jean-Martin Charcot, Pierre Marie, and Jean Athanase Sicard.

The first step necessary to progress in any field of medicine is the description of the basic clinical syndrome.

Charcot helped to establish the concept of chronic articular rheumatism (which is named rheumatoid arthritis in England and polyarthe chronique évolutif in France).

Marie gave one of the first and certainly the best description of spinal arthritis (ankylosing spondylitis in England and spondylarthrite ankylosante in France).

Sicard, with my father Henri Forestier and Dr. V. Putti, was the first to expound the vertebral origin of sciatica.

J. M. Charcot, 1825-1893

Jean Martin Charcot was born in the centre of Paris on November 29, 1825, where his mother was under 17 years of age.

He studied in the colleges of Paris, and already decided, at the age of 19, to become a physician. He was a young man of cold and very often silent character, but he had a keen sense of observation and humour—so he took pleasure in drawing cartoons and caricatures. Many of these have been preserved in the libraries of the hospitals where he worked. One may remark that the caricaturist’s aptitude to detect at first glance a comical singularity is very close to that of the clinician to detect anomalies and demonstrate them to others.

Charcot’s chief teacher was Rayer, a prominent specialist in internal medicine who became dean of the Faculty of Medicine of Paris.

Charcot performed his medical studies with skill and became a resident (interne des hôpitaux) in 1848. During his stay at the Hospice de la Salpêtrière he prepared his doctoral thesis presented in 1853 under the title:

“Étude pour servir à l’histoire de la maladie décrite sous le nom de goutte asthenique primitive. Nodosités des jointures—rhumatism articulaire chronique (forme primitive).”

La Salpêtrière was at that time and is still the largest building of its type in Paris, covering an area as large as that of a small town, and sheltering over 3,000 female pensionnaires. It is a site of choice for the observation of chronic diseases.

Charcot was enthusiastic for the “modern” medicine, which began to develop all over Europe at the time of the French revolution, and had annexed as auxiliary sciences anatomy and physiology. His whole work is a model of anatomical and clinical method. In his 58-page thesis, Charcot explained that 5 per cent. of the old crippled women who lived at la Salpêtrière presented the clinical picture of arthritis. In addition to six observed cases (five of them females), all of which came to autopsy, he gave a splendid description of the entire course of the disease, and analysed its consequences: joint swelling, or apparent hypertrophy of the joints, flexion-deformity, contractures, deviations, ankylosis, muscular atrophy, irregular evolution with exacerbations and remissions, and the subsiding of the pains in later years.
He pointed out the absence of redness over the joints, and the absence of pus in the articular cavities.

He also depicted accurately the appearance of all the limb joints when attacked by the disease, and discussed the mechanism of the deformities which he believed to be primarily due to muscle spasm and to changes in the ligaments.

His clinical description of the evolution of the disease testifies to his admirable sense of observation, and I cannot resist the temptation to quote the following paragraph:

Marche, enchaînement des symptômes.—Les symptômes arthritiques sont les premiers qui se manifestent; ils se montrent toujours d'abord avec une apparence de bénignité telle qu'on ne leur accorde jamais l'attention qu'ils méritent peut-être. Les malades bravent les premières douleurs, bornées à une ou deux jointures, et accompagnées tout au plus d'un peu de tuméfaction et de rougeur. Elles continuent leurs travaux, souffrent péniblement, malgré l'apparition successive de nouvelles arthrites, et ne s'arrêtent qu'à une époque où la rétraction s'est déjà emparée des muscles, et où la plupart des mouvements sont devenus impossibles.

On remarque généralement durant la période, souvent très longue, qui précède les rétractions, quelques circonstances dont il nous a paru utile de tenir compte; car elles seraient de nature peut-être à faire prévoir dès les premiers temps la marche toujours croissante, et en quelque sorte fatale, que la maladie suivra tôt ou tard.

Ce n'est pas sans un certain ordre que s'opère l'envahissement successif des jointures: ainsi, parmi ces dernières, il en est qui se prennent presque toujours en premier lieu, d'autres qui ne sont affectées que très tardivement, et qui même peuvent ne pas être envahies. En interrogeant à ce sujet 41 infirmes, voici ce que nous avons noté: les articulations des doigts de la main et du pied ont été les premières et les seules malades pendant un temps plus ou moins long dans 25 cas; dans sept autres cas, une ou deux grandes jointures, le poignet ou le coude-pied par exemple, étaient affectées en même temps que les articulations des doigts; neuf fois seulement, l'invasion s'est faite exclusivement par les grandes jointures, telles que le genou et le cou-de-pied; mais, dans ce deroier cas, les petites articulations des doigts n'ont pas tardé à se prendre.

Charcot stated that the involvement of the hip joint never appeared at the beginning, and that cases in which the symptoms began before the age of 30 were much more severe than those which started after the age of 40.

He insisted that it was always chronic, but remarked that patients died at about the same age as healthy persons, and that death was not the direct result of the arthritis.

The masterpiece of this study is Charcot's description of the vicious postures of the joints of the upper extremities, which he classified in two groups according to the position of the terminal phalanges in extension or in flexion. These postures are illustrated by remarkable drawings and sketches, and no better description of them has ever been given (Figs 1 and 2, opposite).

If every student of rheumatology took pains to observe them closely he would have a better understanding of patients so afflicted. Our modern books present a crude and cold description not of the patient but of the signs, and the only symptoms that they describe more accurately is that of morning stiffness.

Finally, Charcot discussed the aetiology of rheumatoid arthritis, which was not much more confused than what it is at present; he admitted that the disease was due to an inflammation of bones and joints, and in describing the morbid changes found at autopsy, pointed out the absence of gouty deposits. Of course, morbid anatomy a century ago was not as accurate as it is to-day, and Charcot was dealing with long-standing cases. He was therefore led to regard as characteristics of the disease process signs which were merely the consequences of it; perhaps that is why he adopted his unitarian view of chronic joint diseases, assembling all forms under the one heading of chronic articular rheumatism (Charcot, 1867a).

Neuro-arthropathy.—His thesis on articular rheumatism is his most remarkable contribution to our knowledge of joint disease, but we must not forget that he was the first to recognize and describe, in 1868, the joint changes which appear in the course of locomotor ataxia (Charcot, 1872-87). He presented this work at an International Congress in London in 1881, and considerably astonished Sir James Paget, who could not believe that the origin of such severe changes could have escaped the observation of such distinguished surgeons as John Hunter, Howship, Langstaff, and Stanley.

The wax model of the female patient who was the subject of the first description is still to be seen in Charcot's Museum at la Salpêtrière, a model such as Madame Tussaud's Museum has never exhibited.

In many countries, neuro-arthropathy is called Charcot's joint (Charcot, 1867b). Anatomical specimens of these lesions were sent not only to the French universities, but also to the Royal College of Surgeons of England, St. Thomas's Hospital, and Owen's College, Manchester.

Charcot's classical description of "rheumatoid arthritis" will never be surpassed, but his concept of the place to be assigned to the disease delayed
the advance of French medicine in the field of the rheumatic diseases. In the lectures delivered at la Salpêtrière, between 1882 and 1889, he defined the disease which he had so splendidly described as part of a fundamental malady:

Le rhumatisme articulaire se présente sous des aspects variés et tellement opposés que plusieurs auteurs ont cru avoir sous leurs yeux plusieurs affections différentes. Nous n’y voyons au contraire que les formes diverses d’une même maladie.

He distinguished three types on clinical and anatomical grounds (Table 1):

1. *Rhumatisme articulaire progressif* (rheumatoid arthritis);
2. *Rhumatisme partiel*, affecting mostly the large joints (morbus coxae senilis);

Because of Charcot’s great reputation as one of the greatest consultants in the world, no French physician was bold enough to propose a different classification for the next 40 years.

For this reason, at the end of my medical studies in 1922, after reading the French textbooks of

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**Table 1**

JOINT DISEASES AS CLASSIFIED BY CHARCOT (1889)

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<td>Chronique</td>
<td>(Rheumatic</td>
<td>(1) Progressif</td>
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<td>fever)</td>
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<td>(2) Partiel (morbus</td>
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<td>coxae senilis)</td>
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<td>(3) Rhumatisme d’Heberden</td>
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medicine, I could not understand how the different cases which came to my observation could be classified under these headings which were conscientiously described as varied types of a single unique disease. It was only when I read the "Treatise on Rheumatism", written by Sir Archibald Garrod in 1890, the year of my birth, and presented by him to my father, Henri Forestier, that my eyes were opened:

Charcot and Trastour (a pathologist) regarded rheumatoid arthritis as merely a form of chronic rheumatism, and their view has always held its own in France, finding expression in the later writings of Professor Charcot, as well in the valuable encyclopaedic articles of MM. Besnier and Homolle.

On the other hand, the majority of English and German physicians have always held to the opinion of Heberden and Haygarth that although rheumatoid arthritis presents certain superficial resemblances to both rheumatism and gout, it is in reality a quite distinct disorder.

In these lines, Sir Archibald Garrod recalled the opinion of his father, Sir Alfred Garrod, who had coined the name rheumatoid arthritis just a little more than a century ago, in 1859. Garrod's classification is shown in Table II.

### Table II

**JOINT DISEASES AS CLASSIFIED BY ARCHIBALD E. GARROD (1890)**

<table>
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<tr>
<th>Gout</th>
<th>Rheumatoid Arthritis</th>
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<td>Acute</td>
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<td>Typical</td>
<td>(Polyparticular</td>
<td>Acute rheumatic arthritis</td>
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<td>Chronic</td>
<td>(rheumatic fever)</td>
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<td>Atypical</td>
<td>Acute</td>
<td>Subacute rheumatic arthritis</td>
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<td>Juvenile</td>
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<td></td>
<td>Heberden's</td>
<td>Subacute rheumatic arthritis</td>
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<tr>
<td></td>
<td>Nodes</td>
<td>Chronic articular rheumatism</td>
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Even then, it was not until 1931 that I dared, with my young colleagues Coste and Lacapère, to propose to the French physicians, in a series of articles in the *Presse Médicale*, a new classification of chronic joint diseases, separating rheumatoid arthritis from rheumatism and from the degenerative forms which Charcot had included in the term "chronic articular rheumatism". We also decided to discard the term "rheumatism" from this system of nomenclature which has now been accepted in France (Table III). We chose the term *polyarthrite chronique évolutive*, to emphasize its characteristic of a self-perpetuating disease.

Nevertheless, in spite of these discrepancies, Charcot had a great regard for Sir Alfred Garrod, whose opinions he frequently referred to in his lectures at *la Salpêtrière*, and he was probably responsible for the translation into French by Ollivier in 1867, of Garrod's book on gout. The translation was annotated by Charcot himself.

**Pierre Marie, 1853-1940**

It is not by chance that the second French physician to be responsible for an outstanding advance in the field of the rheumatic diseases was not only like Charcot, a neurologist, but was also his best pupil.

Pierre Marie was born in the heart of Paris into a bourgeois family in 1853, and he was kept in his youth under strict discipline by his father. When he had graduated from college he told his father that he desired to become a physician, his father replied "No. I have decided that you should study law". For 3 years Pierre Marie studied law and obeyed his father, but he was later able to register as a student at the Faculty of Medicine, and when he was *interne des hôpitaux*, he became, by mere chance, the pupil of Charcot at *la Salpêtrière*.

From amongst all the distinguished young physicians, French or foreign, who attended Charcot's clinic, Marie was selected by his teacher as *chef de clinique*, assistant physician, and later medical secretary, and helped him in a number of neurological publications.

When he became *médecin des hôpitaux*, Marie was assigned to the *Hospice de Bicêtre*, an institution similar to *la Salpêtrière*, but devoted to elderly male patients and invalids. Here he founded a department of neurology, following Charcot's example, and his lectures on the diseases of the central nervous system attracted many students and physicians, including Kinnear Wilson.

In the last years of his academic career, he came back to *la Salpêtrière*, and as the direct successor of Dejerine, held the chair of neurology until he voluntarily and silently retired in 1925, leaving a
magnificent collection of original work and having described six new and important diseases, three being of particular interest to the rheumatologist:

(1) Acromegaly, 1886;
(2) Pulmonary osteo-arthritis, 1890;
(3) Spondylose rhizomélïque (ankylosing spondylitis or Marie-Strümpell disease), 1898.

Acromegaly.—This is not a joint disease but a bone dystrophy. Some isolated cases published before 1886 had been confused mainly with myxœdema and Paget’s osteitis deformans. Marie based his first paper on two cases seen in Charcot’s clinic, describing minutely all the symptoms (particularly the enlargement of the extremities: hands, feet, and face) and giving the disease a name. Soon afterwards several cases previously diagnosed under different names were ascribed to the newly-defined disease.

Marie did not at first discuss the aetiology of this condition, but stated that in the first autopsy by Henrot, the enlargement of the sella turcica had been noted en passant among other peculiarities. Further investigations led him to the opinion that the cause of the disease lay in the pituitary gland, but he did not know what sort of lesion it was. His description certainly led to the research on the secretions of the pituitary gland with which the names of Marinesco, Tamburini, Cushing, Bailey, and Evans and many others are associated.

Pulmonary Osteo-arthritis.—Marie’s original description of this condition was the logical development of his studies on acromegaly. His keen observation perceived that the fingers of some patients who were regarded as acromegals were quite different from the usual form; they were shaped like drum-sticks and the nails curved on to the finger tips. He insisted that all his cases of this type were observed in patients suffering from chronic pulmonary disease. De Sèze pointed out that the condition is due more to bony enlargement than to a joint lesion, but Marie’s description is still valid, and little has been added to it.

Spondylose Rhizomélïque (Ankylosing Spondylitis).—In 1886 Marie had observed with Charcot a patient with a rigid spine; they had been unable to classify this condition in any distinct disease, and made a tentative diagnosis of Paget’s osteitis deformans.

The young assistant, however, had taken accurate notes, and 10 years later he came across a second case; in 1896 he read before the Société médicale des hôpitaux de Paris the first French report on la spondylose rhizomélïque (ankylosing spondylitis), so called because the disease affects the roots of the limbs, especially the hip joints (Marie, 1898). The paper was based on three observations, and it is remarkable that none of the six diseases first described by Pierre Marie required more than eight cases to build up clinical pictures which remain as valuable to-day as they were over 60 years ago.

His description of the three cases of spondylitis is so complete and the symptoms are so similar, especially in two of them, that even to-day there remains very little to add, and no detail can be misinterpreted.

The typical gait with a rectilinear lumbar region, the bending of chest and neck, the flexion of the hips and knees which creates a Z-shaped profile, the flattening of the thorax, the ankylosis of the respiratory movements, all are perfectly described in detail, together with the characteristic complete fusion of the whole spine and both hips, with limitation of the movement in the shoulders.

Marie, who was himself so remarkably observant, always held to the opinion that clinical observation is a fundamental necessity, and this paper proves that morphology is queen, not only in neurology, as he stated, but also in rheumatology. He even described how the patients were obliged to walk with their crutches.

At a time when radiography had not yet come to the rescue of medicine to explain the cause of many symptoms in the living patient, Marie explained how he examined the front part of the cervical vertebrae by digital exploration inside the pharynx. He thus perceived the bony growths which were responsible for the ankylosis of the neck and, in some cases, for dysphagia.

Because of the small number of cases presented, he was careful not to be too precise on the evolution of the disease. A few years later, when he had observed more cases, he stated that the disease was almost exclusively confined to males and usually started before the age of 20 years.

He recognized its inflammatory nature and discussed the possibility of gonococcal infection.

In 1906, in collaboration with Léri, he published the results of the first autopsy of a case of spondylose rhizomélïque, from which he declared:

Spondylose rhizomélïque is primarily an infectious or toxic osteopathy with a rarefying tendency, and secondarily a ligamentous ossification with a compensatory inhibiting and healing tendency.

As a rheumatologist, I prefer a statement from the end of his first paper:
Il semble qu'il est permis aux cliniciens d'opposer à la polyarthrite déformante acromélique (rhumatoid arthritis) le processus ankylosant qui frappe d'emblée le rachis et les articulations de la racine des membres (spondylolyse rhizomélique).

This is the way along which the two principal forms of chronic arthritis are now considered in England and in France.

The question arises to whom should be given the credit of the recognition of inflammatory spinal ankylosis as a specific disease?

Marie (1898) quoted Strümpell (1884) as having reported three cases of a similar malady, and Marie and Léri (1906) included case reports by Strümpell, Koehler, and Beer. In discussing the diagnosis, he eliminated what he called the heredo-traumatic kyphosis of Bechterew, showing that he did not consider Bechterew's cases as similar to his own.

Bechterew (1892) had reported five cases combining spinal stiffness and neurological symptoms; we cannot discuss them in detail, but I share the opinion of O'Connell (1956) and of de Sèze and Phankim-Chapuis (1960), that no more than two of these five patients could possibly be cases of ankylosing spondylitis. German-speaking physicians have named this disease after Bechterew, but of these three authors he is far behind both Strümpell and Marie, and in France we use either spondylolyse rhizomélique or Strümpell-Marie disease, the most modern name being spondylarthrite ankylosante.

Ankylosing Hyperostosis of the Spine.—Following the example of my father who had already published several cases of spondylose in 1901, and being a pupil of Professor J. A. Sicard, I have always been interested in this condition. Struck by the rarity of the anatomical reports in the literature, I decided in 1935 to collect some data of my own. As there was little opportunity for post mortem study of a relatively rare disease at the small Hôpital Reine Hortense at Aix-les-Bains, I ventured to examine systematically all the spines in the anatomy departments of the Paris hospitals and to select those found to be rigid. Eight such spines were chosen, photographed, dissected, and radiographed, but when I compared these records with the radiographs of my spondylitic patients, I had to admit that none was exactly comparable with those from living subjects.

I had indeed discovered some remarkable examples of fusion of the thoracic and lumbar vertebrae, but the sacro-iliac joints and the joints of the facets of the spine were free from synostosis. The condition was not ankylosing spondylitis, but what was it?

The second World War broke out; my patients were dispersed, and with deep disappointment I packed my skeletal radiographs into my cupboard.

But 10 years later, after the end of the war, during the examination of some elderly patients with the clinical symptoms of mild, chronic lumbago, my assistant, Dr. J. Rotes-Querol drew my attention to the fact that they did not show the usual osteophytic spicule formation (beccs de perroquet), but a thick and continuous layer of bone on the anterolateral aspect of the spine from D4 to the lumbar region (Forestier and Rotes-Querol, 1950).

These appearances were similar to the x rays which I had collected from the Paris anatomy schools.

Further studies proved that this condition mostly involved the lower part of the thoracic spine with variations in the lumbar and cervical regions where complete ankylosis is very rare.

Clinically this condition involves mild functional symptoms with moderate girdle pains, but no sciatica. It does not produce severe kyphosis or impair chest movements.

In our first papers, we intentionally provoked discussion by claiming that this spinal ossification represented a new disease; some authors declared that it was merely a form of osteo-arthritis, and showed that the histological changes were of the same type as those of spondylarthritis. However, histology alone is not a sound basis on which to differentiate diseases. Collins (1957) has recently observed that giant cells may be caused by a foreign body as well as by tuberculosis.

We feel certain that this peculiar ossification does not result from the coalescence of large osteophytes. It affects a particular group of people mostly males but also including females of a certain biotype (pyknic, plump with a barrel-like thorax) and is very likely associated with mild endocrine disturbances. It differs both from ankylosing spondylitis and from osteo-arthritis of the spine.

In severe cases, when the peripheral joints (i.e. the hip) are affected, the clinical picture and evolution are different from those of osteo-arthritis; there is moderate pain and stiffness with extensive bony growths in the vicinity of the joint, but no narrowing of the joint space and no cysts. The evolution is slow and far less severe than that of osteo-arthritis.

J. A. Sicard, 1872-1929

Jean Athanase Sicard, unlike Charcot and Marie who were Parisians, was born at Marseilles in 1872. His father was a solicitor and died early. When young Sicard was about to choose a profession, he hesitated between trade and the natural sciences.
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The death of his mother and the onset of an epidemic of cholera which killed several of his relatives showed him the value and the responsibility of the medical profession, and he realized that his vocation was to be a doctor.

Soon afterwards he went to Paris, where he became interne des hôpitaux in 1895 and physician-in-chief in 1903. He was appointed professor at the Faculty of Medicine in 1925.

As a pupil of Widal, he was responsible with his teacher for the introduction of the diagnosis of typhoid fever by sero-agglutination (Widal's test).

Though he remained primarily a specialist in internal medicine, he was attracted by the rapid developments in neurology and worked in close co-operation with Brissaud, whom he considered his most brilliant teacher in this field, and also with Reymond, Charcot's successor at la Salpêtrière.

It is not my purpose to detail Sicard's numerous contributions, but I cannot fail to bear witness to my master's remarkable ingenuity and quickness of imagination, which were immediately apparent to me when I became his interne in 1921, and while I remained his collaborator until his sudden death in 1929.

In several different fields of medicine, he introduced new methods of treatment or diagnosis starting from the observation of a simple fact known to many physicians from which he was able to draw unexpected conclusions.

During the first world war, while serving as a medical officer in a hospital at Marseilles, he noticed that in some patients who had received intravenous injections of an antisyphilitic compound the veins became obstructed so that further treatment was impossible. From that observation he invented a method of sclerosing varicose veins with injections. In 1923 I had the privilege of accompanying him to a discussion of this method at the Royal Society of Medicine; after 40 years the sclerosing of varicose veins remains a basic technique of treatment.

Another outstanding example is his introduction of iodized oil in radio-diagnosis. He had noted since 1914 that injections of this substance into the body (muscles or fasciae) caused the persistence of large opaque blotches on the films for many years, without untoward symptoms in the patients. Several years later he asked me whether I would be interested in testing this substance on animals and human beings as an opaque contrast medium. In this way we examined the epidural space (Sicard and Forestier, 1921), and many other as yet unexplored cavities: the sub-subarachnoid space, the uterus, and especially the bronchial tubes, the investigation of which he left entirely in my hands with the help of Louis Leroux. This is now a routine method in the investigation of lung conditions.

Sicard's contribution to rheumatology sprang from his studies of spinal diseases. He described the various forms of lumbago, and was the first with Henri Forestier in 1911 to support the opinion that common sciatica was a vertebral disease.

At that time the nature of sciatica was still a controversial subject. Certain Scandinavian physicians (Helvig, Folke, Lindstedt) had attempted to prove that it was a form of myalgia, and certain English-speaking surgeons related it to the sacroiliac joints. From clinical and anatomical observations Sicard (1918) built up the theory that peripheral neuralgia was caused by the irritation of the nerves in restricted areas when the nerve trunks were exposed in a narrow passage, e.g. in facial neuralgia (neurodociitis), and still more all along the spine where the root nerves emerge from the dura mater and pass through the intervertebral foramina (funiculitis).

In 1922 I submitted my university thesis on "The Intervertebral Foramen and the Pathology of the Epidural Space".

This theory involved a practical advance, since the logical site of treatment was thereby shifted from the thigh and buttock to the lumbar spine. But we failed to discover the importance of the intervertebral disk before Sicard's sudden death from coronary thrombosis at the age of 54 which came as a great loss to French Medicine.

It remained for the neurosurgeons: Barr, Mixter, and Love in the U.S.A., Glorieux in Belgium, and Petit Dutailis in France, to disclose that the cause of sciatica lay in the herniation of the intervertebral disk, and for de Sèze in France to describe completely and laboriously with his co-workers the morbid anatomy of diskal herniation.

In recalling the original works of my three compatriots, three great physicians who helped to increase our knowledge of joint diseases, my first observation is that all three were neurologists at a time when the science of neurology had just started to come to birth; this is particularly noticeable in the works of Pierre Marie, who brought to light three diseases, the symptoms of which concern principally the locomotor system. Each of these physicians had an outstandingly keen sense of vision, and discerned details on the surface of the body, which had escaped the eyes of ordinary men and from which they were able to build up a synthesis.
Clinical medicine in the 19th century was essentially based on observation, but in the present century progress requires observation to be combined with experimentation.

It is also remarkable that, during this period of great advances in medicine, English and French physicians entertained a friendly co-operation and disregarded political controversies. Nowhere has this been more true than in the field of rheumatology, and today’s meeting proves that this noble tradition and friendship is as lively as ever.

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