ON

TABES DORSALIS

The Lumleian Lectures

DELIVERED BEFORE THE ROYAL COLLEGE OF PHYSICIANS, LONDON,

MARCH, 1906

BY

DAVID FERRIER, M.D., LL.D., F.R.S.

Fellow of the College; Professor of Neuro-pathology in King's College; Physician to King's College Hospital, and to the National Hospital for the Paralysed and Epileptic

London

JOHN BALE, SONS & DANIELSSON, LTD.
OXFORD HOUSE
83-91, GT. TITCHFIELD STREET. OXFORD STREET, W.

1906
TO

SIR RICHARD DOUGLAS POWELL, BART.
K.C.V.O., M.D.,

PRESIDENT OF THE ROYAL COLLEGE OF PHYSICIANS, LONDON

THESE LECTURES

DELIVERED AT HIS REQUEST

ARE DEDICATED

AS A MARK OF ESTEEM
CONTENTS.

Lecture I.


Lecture II.

Cerebro-Spinal Fluid in Tabes—Tabes a Dystrophy—Relations to General Paralysis of the Insane—Etiology of Tabes—Relation to Syphilis—Accessory Causes—Comparison of Tabetic Degenerations with those caused by other Toxins—Bacillary Origin of Tabes discussed.

Lecture III.

Physiological Pathology of Tabes—Ataxy—Its Sensory Origin—Forms of Sensory Impairment in Tabes—Muscular Hypotonia—Coordination of Movements—The Tabetic Pupil—Theories as to its Pathogeny—Relation to Ciliary Ganglion—Conclusion—Bibliography.
Lecture I.

Mr. President and Gentlemen.—The choice of a subject must always, I imagine, be a matter of difficulty to the one who has the honour of being appointed to deliver the Lumleian Lectures. Having been invited to open a discussion on tabes at the forthcoming International Medical Congress, it appeared to me that I might with advantage to myself, and I hope not without profit to you, take as the basis of my lectures some questions relating to the nature and pathology of this affection.

Tabes seems to be of perennial interest. Nor is this much to be wondered at, considering the vast field which it covers, and the variety of questions which it raises.

The literature of tabes is enormous, and the treatises and memoirs relating to it literally count by thousands. It will be my endeavour in these lectures to present to you as succinctly as I can
the evolution of our knowledge of tabes, its nature and causes, and to indicate the problems as yet unsolved and on which we need further light.

Tabes dorsalis, in the modern acceptation of the term (and in reference to this I would remark at the outset that but for their consecration by custom, I should like to discard the term dorsalis altogether, and the still more barbarous adjective tabetic, in favour of tabes and tabid) is a very different thing from the tabes dorsalis or dorsalis—ἡθίας νοτίας—of the Hippocratic writers. This was essentially a condition of neurasthenia associated with spermatorrhœa, and attributed to sexual vice or excess. It was supposed to be due to wasting of the spinal cord, but on what evidence this was based there is nothing to show. That true tabes ever existed in Europe before the introduction of syphilis in the fifteenth century is more than doubtful. The retention of the name tabes dorsalis (or dorsualis) by the older German writers for the disease as we now understand it, though strictly correct according to modern pathology, tended largely to create confusion and excite prejudice against its unfortunate victims. Though cases of what we now know must have been tabes had been described by various observers, such as Ollivier (1824), Hutin (1827), and Cruveilhier (1835-42), (who figures the post-mortem appearances in his atlas), in France; and by Horn (1827), Jacoby (1842), Steinthal (1844), &c., in Germany; and though in this country Todd (1847) had drawn a clear distinction between paralysis and
inco-ordination,* and was the first to connect inco-ordination with disease of the posterior columns of the spinal cord, yet there is no doubt that the first systematic account of the etiology, symptomatology, diagnosis, prognosis, and treatment of tabes was given by Romberg (1840 to 1857). In his Lehrbuch der Nerven-Krankheiten he describes the characteristic gait; the pathognomonic symptom now called by his name; the increase of the ataxic disorders on shutting the eyes; the shooting pains, anaesthesiæ and parasthesiæ; the bladder troubles; the affections of vision, and the striking myosis and fixity of the pupils.

He mentions the relative infrequency of tabes in women; and as to prognosis, he utters the gloomy verdict: "To none affected by this malady is there any hope of recovery; über alle ist der Stab gebrochen."

Of the morbid anatomy, he says that, notwithstanding considerable differences, the post-mortem examination shows for the most part partial atrophy of the spinal cord, mainly in the lower part of the lumbar enlargement and onwards, and in the nerves issuing from it. The diminution of volume amounts

* Todd (Cyclopædia, vol. iii., p. 621) says two kinds of paralysis may be observed in the lower extremities—"the one consisting simply in the impairment or loss of voluntary motion, the other distinguished by a diminution or total loss of the power of co-ordinating movements." In two cases of the latter affection he predicted disease of the posterior columns of the spinal cord, to which he attributed the faculty of harmonising the movements of the limbs with each other.
to the half or two-thirds of a normal cord, affects the grey and white substance, or only one of these. The atrophy of the nerves in the leash of the cauda equina is often so pronounced that only the empty neurilemma-sheaths remain. Also the roots of higher inserted nerves participate in the atrophy; and what is of particular interest, the posterior roots are sometimes alone affected, at other times along with the posterior columns of the cord, while the anterior roots appear normal.

Romberg, however, did not clearly differentiate the basis of ataxy from that of muscular paresis or paralysis, and he describes as equally belonging to tabes leathery induration of the white substance and, more frequently, softening of the grey matter.

There was no notable addition to the literature of tabes until Duchenne in 1858 published his researches on "ataxie locomotrice." These were the starting-point of the extraordinary amount of attention that the subject has attracted since his time. Duchenne, purposely endeavouring to divest himself of all preconceived notions, and pursuing his investigations in the limbo of so-called general spinal paralysis and unclassified forms of chronic myelitis, discovered what to him was a new disease, which, from its most striking symptom, he termed "ataxie locomotrice," adding the further term "progressive" on account of its habitual tendency to advance rather than recede. He clearly differentiated the affection from muscular paralysis or paresis, and described with a masterly hand its
most characteristic manifestations. Trousseau, in his brilliant clinical lectures, largely spread abroad the knowledge of this affection, but he did scant justice to previous writers in calling it Duchenne's disease. For, though practically a discovery on Duchenne's part, it was no discovery in the true sense of the term, as the malady was undoubtedly the same as had been known under the name of tabes dorsalis. And though Duchenne differentiated the affection more precisely than had been done before him, yet he contributed nothing to the pathological anatomy of the disease, and was inclined to attribute it to some affection of the cerebellum rather than that of the spinal cord.

Trousseau went so far as to look upon the malady as a pure neurosis, and considered such changes as had been observed by others in the spinal cord and posterior roots as probably only the secondary result of the functional disturbances created by some obscure general condition. This view was maintained by others of his school, notably by Isnard who regarded tabes as a neurosis of the muscular sense.

The connection between the symptoms of tabes or locomotor ataxy and degeneration of the posterior columns, first termed grey degeneration by Cruveilhier, had, however, been satisfactorily established by many observers, among others by Todd and Gull in this country.

The minute anatomy of the morbid changes began with the microscopical investigations of Virchow
(1855) and Rokitansky (1857). Rokitansky described it as a proliferation of the structureless, ependyma-like connective tissue and stroma of the nerve centres, which caused destruction of the nerve elements proper, and resulted in induration or sclerosis of the affected parts (schwielige Degeneration).

The process was not inflammatory, however, as there was no exudation; and Rokitansky included in the same term the end-results of various other forms of lesion of the nerve centres.

But the first important microscopical investigations, specially in reference to the now well-defined symptom-complex of locomotor ataxy, were made by Bourdon and Luys (1861).

They defined the condition as sclerosis of the posterior roots, the end-result of a chronic inflammatory process; and similar views as to the inflammatory origin of the degeneration were held by Charcot, Vulpian, Gull, and others. v. Leyden, however, in his celebrated monograph, _Die graue Degeneration der hinteren Rückenmarks-stränge_ (1863), combated the inflammatory origin of the degeneration, and maintained that it had more the characters of a simple atrophy, and that such indications as there might be of chronic inflammation in the membranes and vessels were only secondary or accidental. He regarded the process as a peculiar degeneration of the sensory tracts of the spinal cord, ascending generally from below upwards.