The Lumleian Lectures

TABES DORSALIS.

DELIVERED BEFORE THE ROYAL COLLEGE OF PHYSICIANS

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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 tabes which are likely to be debated by the neurologists assembled in Lisbon.

Tabes seems to be of perennial interest at neurological and medical congresses. 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 acceptance of the term,† is a very different thing from the tabes dorsalis or dorsualis—*φθίσις νωρίς*—of the Hippocratic writers. This was essentially a condition of neurasthenia associated with spermatorrhoea, 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), etc., 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, anaesthesiae and parasthesiae; 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

* The lecture was illustrated by lantern slides.

† In reference to the term 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.

‡ Todd (*Cyclopaedia*, 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 harmonizing the movements of the limbs with each other.

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 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 connexion 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 Rückenmarksstränge* (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.

At this time, however, he did not, as he has done in later times, causally connect the degeneration of the posterior columns of the cord with that of the posterior roots or sensory nerves, though he specially noted their almost constant coincident affection. In fact, in commenting on a case reported by Gull, to whom it seemed as if the degeneration of the posterior roots caused by inflammation of the pia mater had propagated itself to the posterior columns, he says explicitly: "But the view that the atrophy of the posterior roots caused by inflammation of the pia mater leads to a similar degeneration of the posterior columns is very unlikely. Moreover, the disease of the posterior columns progresses more from the posterior periphery and middle line, rather than follows the distribution of the roots" (p. 152).

The next great advance in the pathology of tabes was made by Pierret (1871), who in the course of his investigations observed that the primary lesions of tabes consisted, not in a degeneration of the posterior columns as a whole, but "in two symmetrical islets of small extent, situated in a special region of the posterior columns."

"This region," he says, "which I propose to call the

'rubans externes' of the posterior columns, appears on transverse section in the form of two narrow bands skirting the internal aspect of the posterior horns. It appears to contain two orders of fibres—commissural and internal radicular. The sclerosed areas do not actually touch the posterior cornua, but are separated from them by tracts of healthy white substance, though they are connected by fibrous bands. The sclerosis, beginning in the middle of the rubans externes, tends to spread inwards towards the median column, and outwards towards the posterior cornu."

Tabes then came for a time to be considered a system disease or sclerosis of the rubans or bandelettes externes of the columns of Burdach. But at this epoch nothing was known as to the relation of the bandelettes externes to the posterior roots. Vulpian, however (1879), saw the difficulty of connecting the supposed primary disease of the bandelettes externes with the atrophy of the posterior roots. A primary degeneration of the posterior columns could scarcely, he maintained, lead to atrophy of the posterior roots whose trophic centre was in the spinal ganglia. This would be a retrograde degeneration and opposed to the Wallerian law. He therefore regarded the atrophy of the roots as undoubtedly of primary origin. That of the bandelettes externes might perhaps be so

also, but only in so far as they contained fibres not directly continuous with the posterior roots themselves.

He proposed to substitute for the formula "sclerosis of the posterior columns" "sclerosis of the posterior roots" as the primary basis of tabes. He thus inaugurated the radicular or root theory of tabes which is now entertained in one form or another by the majority of neuro-pathologists.

In order to follow the bearing of this theory on the pathological anatomy of tabes it is necessary to make some preliminary remarks on the relation of the posterior roots to the posterior columns of the spinal cord.

The facts on which this is based have been established mainly on the results of degeneration consequent on lesions of the posterior roots by disease in man, and of experimental section of the posterior roots in animals, especially in monkeys. These we owe in part to the labours of Schiefferdecker, Schultze, Kahler and Pick, Singer and Munzer, etc., and notably to the careful experiments and investigations of Tooth, which he made the subject of his Gulstonian Lectures before this College in 1889.

The posterior roots emerging from their respective ganglia penetrate the cord in two divisions—an external, consisting of fibres of small calibre which enter the apex of the posterior cornu, or zone of Lissauer, and distribute themselves here; and secondly, an internal division, formed of fibres of large calibre which alone take part in the formation of the posterior column. This division of the posterior root gives off short, medium, and long fibres. It occupies in the first place a crescentic area on the inner border of the posterior cornu—the *cornu-radicular zone* (Marie)—and gives off short fibres to the

Diagrammatic representation of constitution of posterior columns of spinal cord in relation to tabes.

adjoining cells of the posterior cornu. With the advent of the next and successive roots above, the lower roots are displaced from the cornu-radicular zone inwards towards the middle of Burdach's column, and occupy a space elongated antero-posteriorly, which corresponds to what was termed by Pierret the *bandelette externe*. The *bandelette externe* is only the second stage in the course of the posterior roots. From this are given off the medium fibres, which form two principal groups: first, the sensitive-reflex group or reflex collaterals of Kolliker, which arise mainly from the middle of the *bandelette*, and are distributed among the cells of the anterior cornu; secondly, the fibres to Clarke's column. These emerge principally from the anterior aspect, and if derived from the lowermost roots, ascend a considerable distance before they finally reach their destination, inasmuch as the cells of Clarke's column do not descend below the second lumbar segment. Besides the short and medium fibres above described there is another set of fibres which, instead of ending in the cord, ascend in the column of Goll as far as the nucleus gracilis of the medulla oblongata. These fibres appear to come from the posterior aspect of the *bandelette externe* and postero-lateral zone, and gradually pass inwards, and ultimately reach the mesial septum. The lowest fibres are those which are nearest the middle line.

Only the long fibres from the lumbo-sacral and lower dorsal roots ascend in the column of Goll. Those from the upper dorsal and the cervical roots ascend in the column of Burdach, the lowermost nearest the column of Goll, and end in the nucleus cuneatus.

The columns of Goll, therefore, are formed by the long fibres of the lumbo-sacral and lower dorsal roots, and belong to the root system of the cord—the *exogenous* or *extrinsic* system (Marie). All these degenerate upwards on transverse section of the spinal cord at any part.

But, in addition to the exogenous, or root fibres, the posterior columns of the cord contain tracts which are probably commissural in function between different segments and different levels of the spinal grey matter. These tracts constitute the *endogenous* system of the spinal cord (Marie), and form two divisions, the ascending and descending. They only degenerate on destruction of the grey matter, and remain intact after all the exogenous fibres consequent on destruction of the posterior roots have undergone degeneration.

The ascending fibres occupy the *cornu-commissural* zone or ventral field of the posterior columns, lying posterior to the grey commissure, and extending some distance along the inner aspect of the posterior cornua. These vary in size according to that of the horns of the lumbar, dorsal, and cervical regions, and degenerate *above* lesions of the grey matter.

The descending system of fibres degenerates, on the contrary, *below* a transverse lesion of the grey matter. This system of fibres was first described by Schultze (1883), and is known in the cervical and dorsal regions as the "comma" tract. It occupies a position in the column of Burdach, running obliquely in an antero-posterior direction parallel to, but at some distance from, the internal border of the posterior cornu. Schultze described it as not reaching either the grey commissure or the posterior surface of the cord, but the Marchi method has shown that this is not quite correct.

This comma tract was supposed by him to consist only of descending root fibres, but the researches of various pathologists, especially those of Gombault and Philippe, have shown that this comma tract is continuous from above downwards with other tracts which have been differently named according to their appearance in different parts of the cord—namely, the *septo-marginal* tract of Bruce and Muir in the lower dorsal region, the *oval field* of Flechsig on each side of the mesial septum in the lumbar region, and the *median triangle* of Gombault and Philippe in the sacral region.

These variously-named tracts thus constitute one continuous system of descending commissural fibres, receiving and giving off fibres at different levels of the grey matter, and connecting together the various segments, as well as the upper and lower spinal centres, with each other. The continuity of these systems with each other has been clearly demonstrated by Purves Stewart in a case of crush of the cervical cord.

We may now proceed to consider the degenerations characteristic of tabes, and to inquire how far they correspond with the exogenous and endogenous systems of fibres above described.

In a moderately-advanced case of tabes one observes a notable atrophy of the posterior roots. These are thin, flattened, and greyish in colour, contrasting vividly with the white and plump anterior roots. This appearance is most marked in the roots forming the cauda equina. The posterior columns of the cord are also thinned, and of a greyish, semitransparent aspect, and the posterior cornua are often similarly affected. The pia mater over the posterior columns more especially is frequently thickened and adherent.

The changes are, as a rule, most pronounced in the lumbar and lower dorsal region, but may in certain cases—so-called cervical tabes—be more or less limited to the cervical region. In advanced cases the whole of the posterior roots and whole of the posterior columns may be affected.

If one examines a transverse section of the lower dorsal or upper lumbar cord in an early case, one observes an area of degeneration occupying the middle third of the posterior column, extending from the posterior cornu forwards and inwards towards the posterior commissure. This area constitutes Pierret's *bandelette externe*, and varies in breadth according to the number of posterior roots involved. The two *bandelettes*, not always symmetrical, may meet almost in the middle line. They correspond to the middle stage of the trajet of the posterior roots. From this part of the course, as we have seen, are given off the reflex collaterals to the anterior cornua, and the fibres to the vesicular columns, or columns of Clarke. These are the fibres first, and specially affected in tabes.

For a considerable time after the degeneration has appeared in the *bandelettes externes* comparatively little degeneration may be visible in the cornu radicular or root entry zone, in the long fibres occupying the postero-external zones, and still less in the cornu commissural zone and oval fields bordering the posterior median septum. In more advanced cases, all the posterior columns are degenerated except the cornu commissural zone and the oval fields of Flechsig. These form, as already remarked, the endogenous systems of the cord, and only degenerate in very advanced cases in which the upper regions of the cord are invaded.

The appearance of sections of the cord higher up will vary according as the degeneration of the posterior roots is confined to the lumbo-sacral region, or also implicates the dorsal. In the first case the only degeneration visible is in the posterior median columns, which contain the long fibres from the lumbo-sacral region in their course up to the nuclei graciles of the medulla oblongata, all the short and medium fibres having by this time found their primary termination in the cells of the posterior and anterior cornua and in the columns of Clarke.

The appearance presented will be that seen in the figure which I throw on the screen.

If, on the other hand, the dorsal roots are also affected, we get, in addition, degeneration in the columns of Burdach, varying in extent according to the number of roots involved; and we may have a degenerated zone in Burdach's column separated from that of the column of Goll by healthy tracts corresponding to healthy roots entering at a lower level.

In the cervical cord, if only the lower dorsal and lumbo-sacral roots are affected, the degeneration will be confined to Goll's columns, as these are composed exclusively of the long fibres passing up from these regions. If the upper dorsal is affected, degeneration of Burdach's column will be found immediately adjoining that of the column of Goll; and if the cervical roots are also implicated, the degeneration will occupy the columns of Burdach, varying in extent with the amount of degeneration of the roots. In cases of cervical tabes there is no degeneration in the columns of Goll. The degeneration is confined exclusively to the columns of Burdach, and the long fibres of these roots end in the nuclei cuneati.

It is only in the most advanced stages of tabes that all the posterior columns, exogenous as well as endogenous tracts, are alike degenerated. Of the exogenous system we have seen that they are not all equally affected at the

same time. The first incidence of the degenerative process is on the medium fibres, namely, the reflex collaterals and those to Clarke's columns. The long fibres which, in large measure at least, constitute the postero-external root zones are more resistant, and often exhibit scarcely any degeneration when the *bandelettes externes* are completely sclerosed. There is some doubt, however, whether the postero-external root zones are composed only of long fibres. There are grounds for believing that they contain also endogenous fibres which are relatively more resistant to degenerative change (Schmaus and Sacki).

Similarly, the fine fibres of the posterior root which enter the zone of Lissauer often escape degeneration when it is already well-marked in the medium fibres of the column of Burdach.

The tabetic degeneration would therefore appear to be selective in character, unlike the Wallerian, which affects all indiscriminately, attacking in succession fibres and tracts of perhaps different functional significance. In this aspect it has been attempted to establish a relation between the tabetic degeneration and the fetal differentiation of the various tracts which enter into the composition of the posterior columns as determined by the stage of development at which they respectively receive their myelin sheaths. Flechsig, by the myelinization method, has divided the posterior columns into six main divisions, which receive their myelin sheaths at four successive stages of fetal development. He thus divides the posterior columns into an anterior, middle, and posterior root zone; this, again, into a medial and a lateral. Added to these are Goll's column, and a mesial tract bounding the posterior median fissure. The first tract of fibres to myelinate—his anterior root zone—corresponds with the cornu commissural zone of Marie; the last to myelinate—his postero-lateral zone—corresponds with the zone of Lissauer. These various tracts constitute according to Flechsig elementary systems, and it has been assumed that they are composed of fibres of common functional significance. The researches of Trepinski, however, on the same subject differ in many important respects from those of Flechsig; and there are many discrepancies between both, and the results of secondary degeneration. Nor has it as yet been satisfactorily shown that the tabetic degeneration coincides precisely with any of the fetal systems described by these authors. Further research, however, may clear up many doubtful points, for it is not improbable that the tabetic degeneration follows the lines of functionally different fibres, which myelinate at different stages of fetal development.

The tabetic degeneration, so evident in the posterior columns and posterior roots, appears to stop short at the spinal ganglia. The sensory nerves distal to the ganglia have a normal appearance and normal structure. But not infrequently, as has been shown by Westphal, Pierret, Déjerine and others, the finer peripheral ramifications in the skin, muscle, etc., undergo atrophy similar to that in the intramedullary terminations. Nor is the process limited to the spinal cord and the spinal nerves. The optic nerve is frequently affected, and occasionally, also, other cranial nerves. Degenerative changes have also been demonstrated in the ciliary ganglia and in the sympathetic nervous system, particularly in the cardiac plexus. These will be considered more in detail in connexion with the physiological pathology of some of the more prominent symptoms of tabes.

If we now turn from the more gross anatomy of tabes, and consider the intimate nature of the process, we find, according to the most modern research, that its essential character is a progressive dystrophy, or demyelination and ultimate destruction of the nerve fibres. The myelin sheath, as shown by the Marchi method, breaks up, becomes granular, and finally disappears. The appearances vary somewhat according to the acuteness of the process, but in general a proliferation of the neuroglia network occurs only in proportion to the atrophy of the nerve tissue proper. There is no active hyperplasia of the neuroglia, and no vascular changes such as can be considered as the primary starting point of a sclerosis leading secondarily to atrophy of the nerve fibres. The Marchi method in the early stages of the process reveals the degeneration of the myelin long before proliferation of the neuroglia has occurred, and such sclerosis as ultimately ensues is only what may be

seen in any organ consecutive to degeneration of its true parenchyma.

The obvious relationship of the exogenous tracts of the spinal cord to the spinal ganglia—being, as it were, the central prolongations to the sensory neurons of which they are composed—naturally suggests that the cause of the progressive dystrophy of the posterior roots and their intramedullary terminations may be found in some morbid alteration of the cells of the spinal ganglia, the trophic centres of the fibres in question. For, as Marie remarks, it is difficult to believe that the branches of a neuron, whether central or peripheral, and performing the functions only of conductors, can undergo degeneration apart from that of the cell body which is their trophic centre. Therefore, in every case of degeneration of a nerve fibre, whether central or peripheral, the point first to investigate is the *cellule malade*.

Much research has been spent on this point, but the conclusions reached as to morbid changes in the spinal ganglia, or their constituent cells, have been far from harmonious. Gross changes such as were described by Bourdon and Luys in one case are uniformly absent.

As regards the cells, Marie himself found in one case that they appeared less numerous than normal; that some of them seemed to show signs of degeneration, and that there was distinct atrophy of a considerable number of the nerve fibres traversing the ganglia. Some support has been given to Marie's view by the investigations of Wollenberg, Oppenheim and Siemerling, Stroebe, Redlich, Marinesco, Thomas and Hauser, etc.; but on the other hand, Schaffer, Juliusberger, Meyer, Mott, and many others have found them practically normal.

The alterations which have been described by Wollenberg, Marinesco, and others who have obtained positive results, consist mainly in chromatolysis, pigmentation, or fatty changes in the cells, occasional deformity or diminution in numbers, and increase of the connective-tissue elements. Köster, the most recent investigator on this subject, states that in the early stages of tabes he has observed appearances similar to those described by Wollenberg; but that in more advanced cases there are marked changes, such as chromatolysis, pigmentation, abnormalities in the nucleus, and deformation of the cells. They were similar to those resulting in course of time from experimental section of the posterior roots in animals.

It is therefore doubtful whether such changes as have been discovered in the spinal ganglia afford a sufficient anatomical substratum for the amount of degeneration, more particularly in the posterior roots and in their intramedullary terminals. And it is considered (Schmaus) a powerful argument against the spinal ganglion being regarded as the primary origin of the dystrophy that the peripheral process, as it emerges from the distal pole of the ganglion, practically shows no abnormality (with the exception, perhaps, of some of its cutaneous terminals); while the central process, the posterior root, may be degenerated right into the ganglion itself. Whether these and other arguments are altogether valid against the primary ganglionic origin of the tabetic process will be considered later, but it must be admitted that the anatomical changes hitherto actually demonstrated in the ganglion cells are altogether incommensurate with the degree of degeneration of the processes (central and peripheral) as compared with that observable in other forms of cell degeneration, such, for instance, as in the multipolar cells of the anterior cornua in atrophic spinal paralysis.

Instead of regarding the spinal ganglia as the primary origin of the degeneration of the posterior roots and exogenous medullary tracts, v. Leyden and Goldscheider are inclined to look upon the peripheral nerve endings, which not infrequently exhibit dystrophic or neuritic changes, and are naturally more exposed to traumatic influences, as more probably the starting-point of the process. It is known that after amputations and section of nerves, atrophy occurs in the posterior horns, posterior columns, and also in the anterior horns of the spinal cord. This is the form of degeneration termed retrograde, and is probably due to the cessation of the normal influences which serve to excite and maintain their functional activity. And it has been shown by Lugaro, Cassirer, and more recently by Warrington, that marked chromatolytic changes occur in the cells of the spinal ganglia on section

of the sensory nerves, and of certain cells in particular, according to the kind of afferent nerve—muscular or cutaneous—which is divided. It is assumed, therefore, by the supporters of the peripheral origin of tabes, that changes, organic or functional, in the spinal ganglion cells may be induced by neuritic or other processes in the peripheral nerve endings, and thus secondarily lead to degeneration of the posterior roots and posterior columns. It has not, however, been demonstrated that degeneration of the peripheral nerves always precedes that in the cord, nor has it been shown that such peripheral degeneration is a constant feature of the morbid anatomy of tabes, which it ought to be if the theory is at all well founded. Nor does it seem likely that neuritic or other degeneration, ascending from the periphery, can induce such dynamical or organic impairment of the ganglion cells as the theory postulates, without making itself evident at the point where the sensory nerves enter the ganglia. The peripheral theory of the pathogeny of the tabetic degeneration of the spinal cord has therefore to overcome many serious objections before it can be accepted as at all a satisfactory one.

Other pathologists are of opinion that they have discovered the origin of the tabetic process in inflammatory conditions of the meninges affecting the posterior roots. The theories of Nageotte, and of Redlich and Obersteiner are the most important in this relation. Nageotte, whose views have received a large amount of support, especially in France, has called attention to an anatomical disposition of the posterior roots which renders them peculiarly liable to noxious influences. Nageotte holds that in tabes there is a diffuse chronic meningitis which has all the characters of syphilitic origin—namely, infiltration with lymphocytes and plasma cells especially round the veins, a meningomyelitic affection of the periphery of the cord, medulla, and even the cortex cerebri.

This general meningitis induces the degeneration characteristic of tabes by implicating the spinal roots at the point of their emergence from the dural sac. The anterior and posterior roots after leaving the spinal cord traverse the subarachnoid space, and at the point of exit from the dural sac receive a sheath both from the dura and arachnoid, which constitutes the perivascular sheath of the roots, and beyond the ganglion the two roots unite to form the mixed nerve. The portion which lies between the ganglion and the dural sac, which varies in length in different regions, he terms the radicular nerve. The perivascular sheath, derived from the dura and the arachnoid, divides the radicles into compartments, and the intravascular tissue is continuous with the pia mater. The lymphatic spaces of the radicles communicate with the subarachnoid space, and constitute a narrow and complicated channel between the great lymphatic sac and the efferent vessels. Owing to this disposition the radicles are specially liable to injury by meningeal inflammation and toxins circulating in the cerebro-spinal fluid. Hence is developed a transverse interstitial neuritis, which specially affects the posterior radicles; and the anterior also to some extent, which, however, is only transient.

In consequence of this neuritis, secondary degeneration is set up in the intramedullary continuations of the posterior roots, and spreads back to the spinal ganglia. The process, however, is not a true Wallerian degeneration, but a mere dystrophy, or lowered vitality, which affects more particularly the short and medium fibres of the posterior roots. In support of his views, he adduces the lymphocytosis of the cerebro-spinal fluid which occurs in tabes, to which I will call your attention presently. But in the meantime Nageotte's theory appears to offer such a simple explanation of the tabetic degeneration that it seems almost a pity to subject it to destructive criticism.

But it is open to the same objections as can be urged against the theory of Redlich and Obersteiner, in that the meningitis, which it postulates, is not a constant phenomenon, and is perhaps more frequently absent than present, and when it exists is more often of the character of a secondary thickening than an inflammatory process. And, further, it has been found in the early stages of tabes that the intramedullary degeneration is often well marked when the posterior roots exhibit no appreciable change and no indications of a local neuritis. The practical escape of the anterior roots is, I think, a fatal

objection to Nageotte's theory. That the neuritis which he also finds in the anterior radicle should be evanescent, while that of the posterior radicle leads to such profound intramedullary degeneration is unintelligible; and is, moreover, opposed to the usual relatively greater affection of the motor than of the sensory fibres when neuritis attacks mixed nerves.

Redlich and Obersteiner maintain that at the point where the posterior roots penetrate the pia mater there is a constriction which constitutes a *locus minoris resistentiae*, and that in consequence of meningeal inflammation and the retraction of cicatrization, as well as by thickening of the closely-adherent blood vessels, the posterior roots become, as it were, strangled. Hence ensues degeneration in the intramedullary exogenous tracts, followed at a later stage by that of the posterior roots in a retrograde manner.

Redlich and Obersteiner's view has the merit of harmonizing better with the more common starting-point of the tabetic degeneration than that of Nageotte, but, as has been remarked, the meningitis basis on which it rests is not very substantial. It is not universally present, nor is meningitis, syphilitic or otherwise, when it does occur, a common cause of intraspinal degeneration by implication of the posterior roots. That such a result is possible, and perhaps actually occurs in some cases, may be admitted, but it is extremely rare. The symmetrical and segmental character of the tabetic degenerations, which it would be difficult to correlate with any, so to speak, brutal meningeal inflammation, and the occurrence of degenerations elsewhere, such as in the optic nerve, ciliary ganglion, and sympathetic system, are all formidable arguments against the meningitic origin of tabes, whether at the point maintained by Nageotte or that of Redlich and Obersteiner.

But that the so-called "ring of Obersteiner" is a *locus minoris resistentiae*, or specially vulnerable point, in the course of the posterior root fibres, has been argued with much force by Orr and Rows in their memoir on the system lesions of the posterior columns in General Paralysis.

At this point the extramedullary fibres of the posterior roots lose their neurilemma sheath, and the myelin sheath is also much thinned. That the absence of the neurilemma sheath renders the nerve more vulnerable is probable from the researches of Kennedy, Bethe, Ballance, and Purves Stewart, which have demonstrated the importance of the neurilemma and its cells in the nutrition and regeneration of nerves. These show that even in the distal segment of a divided nerve regeneration of the axis cylinders and medullary sheaths takes place, although the full maturity of the new nerve fibres is not attained until union of the proximal and distal segments occurs, and the fibres become functionally continuous with each other. This regeneration is dependent on the cells of the neurilemma sheath. Where these are absent, as in the intraspinal fibres, regeneration does not occur; and it is probable, therefore, that the absence of the neurilemma cells is the cause of the greater proclivity to degeneration of the intramedullary terminals than of the other branches of the sensory protoneurone in the tabetic dystrophy.

It has been shown, also, that degeneration of the intramedullary root fibres similar to those of tabes may occur in the course of cerebral tumours, diabetes, anaemia, and other cachectic states (Homén, Nageotte, Williamson, Bruce, Erbsloh, etc.). The degeneration extends from Obersteiner's ring into the reflex collaterals and fibres passing into Clarke's columns. Whether it begins first in the terminal fibres, and gradually extends backwards, or vice versa, is the subject of some difference of opinion. Orr and Rows hold that it commences at Obersteiner's ring and extends centrifugally; while Nageotte considers that the terminal fibres of the reflex collaterals and those of Clarke's columns are first affected. The more recent investigations of Spielmeyer, by means of Cajal's new axis cylinder stain, are in favour of the view that the terminal fibres which arboresce around the cells of Clarke's columns as well as those of the posterior column nuclei first shows signs of degeneration. This is followed by glia proliferation in proportion to the destruction of the nerve filaments.

Whether we regard the intramedullary degeneration as centripetal or centrifugal, or more probably simultaneous,

it is clear that this portion of the posterior root is more liable to show signs of impaired nutrition at an early stage than either the extramedullary portion or the cells of the spinal ganglia. Some conditions probably exist favouring the incidence of noxious influences on this portion of the sensory protoneurone. In this connexion Marie and Guillain have laid great stress on the peculiarities of the lymph circulation in the posterior columns of the spinal cord. They maintain that in the pia mater of the posterior columns, between the outer and inner layer, there exists a lymphatic system, the spaces of which communicate with those of the posterior columns, but are independent of those of the antero-lateral columns. In this system the current of lymph is upwards and towards the central canal. In tabes they have found this system dilated, and, by the Marchi method, black granules in the cells of the ependyma in cases where the central canal is permeable. They attribute the condition to a syphilitic affection of the posterior lymphatics, and derive the lymphocytosis of the cerebro-spinal fluid from the elements of this system.

That noxious substances or toxins originating in the periphery ascend to the spinal cord more readily by the posterior than by the anterior roots has been shown by Homén, and also by Orr and Rows; and that in this way, or by peculiarities in the lymphatic circulation in the cord, the posterior roots and posterior columns are specially exposed to injury is extremely probable. But that there is in tabes a posterior meningitis of a syphilitic character, as maintained by Marie and Guillain, is not generally admitted. It is not supported by the results of anti-specific treatment, nor can the lymphocytosis of the cerebro-spinal fluid be adduced as a conclusive argument in its favour.

[Bibliography will be given at end of third lecture.]

Demonstrations

ON

THE CYSTOSCOPE

AND A METHOD OF ILLUSTRATING DISEASES OF THE BLADDER AND THE APPEARANCES IN RENAL DISEASE BY THE OPAQUE PROJECTOR.*

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[WITH SPECIAL PLATE.]

PART II.—CYSTOSCOPIC APPEARANCES IN SOME DISEASES OF THE BLADDER AND KIDNEYS.†

In the cases to be presented to you to-day I have selected those suited to illustrate the conclusions to be submitted.

Fig. I.—Normal ureter opening.

the mouth is easily recognized on the ridge of the trigone.

I have avoided taking what might be regarded as "typical cases," choosing by preference (with one or two exceptions) those most commonly met with in practice.

Fig. I represents the appearance of the normal ureter opening and the mucous membrane of the bladder when the viscus is distended to 6 fluid ounces. The lips of the opening are distinctly seen and slightly parted so that

A. CALCULUS.

1. Stone Lying Free in Bladder.

Probably the most easily-observed morbid object within

* The second of a series of Post-graduate Demonstrations given at the Royal Infirmary, Glasgow.

† The writer has much pleasure in owing his indebtedness to the Editor of the *Practitioner* for the use of the blocks to illustrate Figs. III, X, XII, XVIII, XXIV, and XXVI.

the human bladder is a stone lying on its floor (Fig. II). The presence of such a foreign body can easily be discovered by means of a sound, but its probable composition, its form and size can only be accurately determined by the cystoscope. The spherical, smooth phosphatic calculus caused comparatively little irritation, and its presence did not induce much change in the appearance of the mucous membrane of the bladder.

Fig. II.—Stone loose in bladder.

The patient had complained of symptoms of stone for over two years, the most pronounced being pain at the end of micturition, but the suffering was not sufficient to preclude him from following his occupation (a salesman in a grocer's shop) until within two weeks of the time I saw him. He sought advice on account of a hæmorrhage; when examined the stone was at once seen, and the following day it was removed.

In many cases, however, without the aid of the cystoscope, stone may elude detection, either from being covered by a thick layer of mucus or by being placed in such a position that it cannot be reached with the sound.

2. Stone Obscured by a Thick Layer of Mucus.

When the surface of the stone is regular but finely honeycombed without having prominent projections, thick mucus may become

adherent to it, so that it presents the appearance of being covered with wet cotton-wool (Fig. III), and this mucous layer may form a very efficient protection to the mucous membrane of the bladder. I have seen several instances of this kind, some where a stone has been present in the bladder for a considerable period without inducing signs of irritation, others where the symptoms have abated in a most remarkable way, as a consequence of the protective coating.

Fig. III.—Stone obscured by mucus.

Fig. III is an illustration from a case in point. The history was that of chronic cystitis, frequent micturition, pyuria, but no hæmaturia. The pain was so slight that the presence of a stone was not suspected. The case was rather regarded as one of septic cystitis. I found, however, a white woolly mass occupying the floor of the bladder, the shape and size of a pigeon's egg. From its appearance and form, the presence of a stone was suspected, but on passing a sound into the bladder no hard object could be found. The bladder was washed out regularly twice daily for a week, and a considerable quantity of mucus was in this way removed. Then a second examination was made, when a phosphatic stone was seen lying on the floor of the bladder, only with a light covering of loose flaky mucus.

3. Calculus in Sacculæ.

A calculus may be hidden in a sacculæ or diverticulum in which to find a stone may be extremely difficult (Coloured Plate, Fig. IV). The mucous membrane of the bladder was practically normal. The colour, except immediately around the opening of the sacculæ, was pale, but the lips of the opening were deeply congested, rounded, thickened, and, in marked contrast to the bright red lips of the orifice of the diverticulum, a pale white object was seen.

In such cases the difficulty when first seen is to determine whether such a white object is a stone or a tuberculous ulcer covered with a slough, but by carefully washing out the bladder the mucus covering the stone is removed and by passing the finger into the rectum or the vagina, while at the same time observing through the cystoscope, if the object be a stone, the bladder wall