A Clinical Lecture

ON

SOME ANOMALOUS CASES OF TABES DORSALIS.

Delivered at the National Hospital for the Paralysed and Epileptic, Queen Square, on July 8th, 1902.

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THE subject which I have chosen for my lecture to-day is, as you are aware, that of anomalous cases of tabes dorsalis. I have chosen this subject because I have been very much struck recently with the curiously variable character of this disease, and with the cases which have come before me often almost unrecognizable in their symptomatology and variable in their course and in their susceptibility to treatment.

It is difficult to know what one ought to reckon as anomalous cases of tabes, because we have not any definite type from which to start as the normal condition of disease, if one may so call it. But looking upon tabes dorsalis as a disease essentially of the afferent system characterized by wasting of the spinal cord, in the posterior region especially, and having as its chief symptoms difficulty or ataxy in walking or in using the upper limbs, lightning pains, girdle sensation, and interference with the action of the sphincters, I should regard as anomalous, at least for the purposes of this lecture, cases clearly recognized by certain signs as belonging to the same category, but differing in any important respect. I should be inclined to divide such cases into two classes: First, those in which symptoms referable to the eye are the chief manifestation of disease; and, secondly, those characterized by the obtrusiveness of various unusual conditions such as gastric crises, joint troubles, perforating ulcer, or other trophic changes. But I must not be understood to say that cases in this latter group may not also have the characteristic phenomena of the ordinary disease, or may not also have optic atrophy or ocular paralyses superadded to the symptoms I have mentioned.

With reference to the first group of cases, those, namely, in which ocular symptoms or symptoms attributable to disease of the optic nerve are the chief ones, I should mention that many of them are seen in their earlier stage by ophthalmic surgeons, who are naturally consulted on account either of some ocular defect, or of some failure of vision. I have been very much struck with the number of such cases that I have seen during the last few years, while I have had the good fortune to be connected with the Moorfields Eye Hospital, cases which find their way there to consult one of the surgeons, and are then referred to me for a medical opinion. Many of those cases are amongst the most interesting that fall under my observation, and my work at Moorfields has given me the opportunity of seeing cases of this character in considerable numbers, numbers much larger than I before thought possible. Such cases, then, in the most common form, have either paralysis of some ocular muscle, or they have some optic atrophy. The muscle most commonly affected is, I think, the levator palpebrarum; and along with this it is usual to find defect in the other muscles supplied by the third nerve. Ptosis alone, without any other ocular defect, is rare if unilateral; but a slight, though quite definite, degree of double ptosis is by no means uncommon in cases of tabes without any involvement of any other muscle. Besides this affection of the third nerve, and, indeed, sometimes apart from it, we sometimes meet with paralysis of either one or of both sixth nerves, most frequently of one. And it may be said generally of those ocular palsies occurring in cases of tabes, that the paralysis is nearly always of a transient character, clearing up in a few weeks time, usually under the influence of iodide and mercury, although I have sometimes seen such paralyses disappear when neither drug was being taken. It may be asked, Are such cases always definitely tabetic? Occasionally perhaps not, but in the great majority of cases at all events they are, for they have, as a rule, associated with the symptoms I have described

either pupils which are inactive to light, or lightning pains, not uncommonly in the distribution of the fifth cranial nerve, or girdle sensation, or loss of knee-jerk, perhaps all of these. Yet even in such cases the ocular paralysis may be no necessary part of the tabes. In some cases undoubtedly the knee-jerks are still present, and the pain in the head nothing more than might be accounted for without invoking the idea of lightning pains; but even such cases, I believe, are tabetic, and I have known several instances in which true tabetic symptoms, in abeyance at first, were afterwards fully developed.

The same, I think, may be said of cases which seek advice first on account of defective vision. The defect of vision is almost invariably the result of optic atrophy—the grey atrophy which is by far the most common form of atrophy met with in tabes. If we see such a case in the early stage there may be only a slight degree of optic atrophy, with a corresponding defect of vision present. No other symptom, not even loss of knee-jerk, is to be found, although a few months later it is not uncommon to find that the knee-jerk has disappeared, and that the patient is having occasional attacks of shooting pains, not very severe, either in the arms or in the legs, or in both. Not at all infrequently the Argyll-Robertson pupil is present in those cases, but I have been rather struck with the considerable proportion of cases of what afterwards proves to be tabes in which optic atrophy is present, which have no Argyll-Robertson pupil, and in which the pupil remains responsive to light, even when vision is almost destroyed.

I might mention a third class of cases in reference to ocular symptoms, that class, namely, in which the Argyll-Robertson pupil may be the only sign of really definite importance. I have now seen several cases of patients in whom the Argyll-Robertson pupil was present in both eyes, associated usually with a slight inequality in the size of the pupils, in whom there was no other sign of tabes. Such cases, I believe, are nearly always of the tabetic type, and I think that, sooner or later, they do develop some unequivocal sign of degeneration corresponding to the disease.

It is not infrequently stated that the onset of optic atrophy in a tabetic patient is followed by an arrest of the progress of the disease. This is a dictum which should be accepted with a certain amount of reserve. If it is meant that optic atrophy in a tabetic prevents the onset of ataxy, I do not think it is true. I have known a patient who had been blind for some years as a result of tabetic atrophy develop very marked ataxy. Such an experience is undoubtedly rare. On the other hand, it is no doubt the case that in most tabetics who have optic atrophy ataxy is not present, and this is especially true of cases in which failure of vision from optic atrophy is an early symptom. I have, however, seen several cases of tabes, previously quite well marked, in whom the onset of optic atrophy has had no modifying effect upon the symptoms of the disease. Yet it is no doubt true as a general rule that in tabetic patients with optic atrophy, especially if this is very severe and the affection of vision very profound, the other usual symptoms of the disease—especially the ataxy—are either not present or are present in a very mild form.

The second class of anomalous cases of tabes of which I should like to speak are those which are characterized by various curious conditions, such as gastric or other crises, joint troubles, or trophic disturbances. It should be stated, in the first instance, that many of those cases have, in addition to those somewhat unusual symptoms, the ordinary signs of tabes—namely, ataxy, lightning pains, pupillary change. On the other hand, some of them have none of those signs, and apart from the occasional attack of vomiting or other symptom, have no obvious sign of tabes dorsalis. One case of this kind I might mention—that, namely, of a healthy strong-looking young man, aged 35, whom I saw on account of occasional severe “bilious attacks,” as they were called. These attacks of vomiting were characterized by their extremely sudden onset, when the patient was apparently in excellent health, by the fact that they lasted but two or three days, severe vomiting being accompanied by excessive prostration, and that they passed away almost as suddenly as they came. There was nothing in the outward appearance of this patient to excite suspicion that he was suffering from any nervous disease. His pupils reacted perfectly well to

light, but no knee-jerk could be elicited on either side. There was no ataxy, there was no interference with the action of the sphincters, and he was a man who was engaged in very exacting professional work. I believe that there are many cases of this kind which go for a long time without their true cause being discovered; and in every case, in an otherwise apparently healthy man, in which there is a history of recurrent attacks of severe vomiting, I think it extremely desirable not to be merely content with the diagnosis of "bilious attack," but rather to examine the patient in view of the possibility that such a case may be really one of severe nervous disease.

With reference to the after-history of such cases, I have now seen several in which the condition has altered but very little, except in reference to the attacks of vomiting. In one case these gastric crises persisted, but became gradually less and less frequent until they altogether disappeared. In this patient's case there was never any development of ataxy, although he had lost his knee-jerks before he came under observation. He became excessively weak, so that he was almost unable, on account of weakness, to get about; and although he had had no interference with the action of the bladder, he finally died of uraemic coma, accompanied with convulsions. In addition to the changes underlying these symptoms which were found in the kidneys he had the characteristic lesion in the spinal cord.

Another case of a similar character which I have known during the last ten years has remained practically stationary as regards his tabetic symptoms. He had unfortunately acquired the morphine habit on account of the remedy which was resorted to to relieve him during his attacks of sickness; but of this also he got the better, and is now, or was quite recently, although weak and emaciated, yet in fairly good health. So that from my experience I should be inclined to say that the presence of gastric crises, even if there are other undoubted symptoms of the tabetic lesion present, rather has, in regard to prognosis, the effect of encouraging one to say that the patient is not likely to become ataxic, and that, as a matter of fact, the gastric crisis may entirely cease, and the disease otherwise remain, at all events during many years, without any perceptible advance.

I might just say one word here with reference to the treatment of these gastric crises. There is nothing which controls a gastric crisis when it has fairly established itself except the hypodermic use of morphine. I think it is necessary to give this in those cases, but that at the same time it is necessary to have one's eyes open to the effect which the morphine may produce upon the patient. In reference to one of the cases which I have just related, I have said that the morphine habit was actually established on account of this remedy, so that it is essential that the remedy should not be left in the hands of the patient, and should only be given by some one who is capable of preventing the patient from having recourse to it himself. I have sometimes thought that the use of small doses of opium or morphine by the mouth between the attacks had the effect of preventing, and in others of at least delaying, the crises. I have not, I am sorry to say, been encouraged by my experience of antipyrin, antifebrin, and that class of drugs in the treatment of these crises, but I think it quite possible that the energetic use of one or other of them, combined, perhaps, with large doses of bismuth and a small quantity of morphine by the mouth, might have an extremely beneficial effect.

One other class of cases of crises I should like to refer to, that, namely, in which the larynx is affected. These crises are really choking attacks, in which there is extreme inspiratory spasm, the result of the vocal cords being spasmodically adducted; and in nearly all those cases, as Sir F. Semon has pointed out, there is weakness of the abductors of the vocal cords. The immediate effect upon the patient is one of extreme distress, accompanied with cyanosis, not infrequently also with slight twitchings, such as one sees under anaesthesia from nitrous oxide gas, and in some cases apparently with a fit almost indistinguishable from one of epilepsy.

In a good many cases of this character it has been necessary to have recourse to tracheotomy; and I have known one or two instances of patients who were attending the outpatient department of the hospital who have died suddenly, apparently during one of these laryngeal crises. So that this

form of crisis is undoubtedly one of the most serious; and in every case in which there are frequent attacks or in which the attacks are of a very severe character I think it is only right that the question of tracheotomy as a precautionary measure should be considered carefully. In my experience the cases in which laryngeal crises are present in severe degree are cases in which there is usually a very marked degree of ataxy, and this distinguishes them in a very striking way from cases such as those I last spoke of—those, namely, of gastric crises. Of course it may have been a coincidence that such has been the case in the patients I happen to have seen, but there have been a considerable number, and I cannot recall a single case of laryngeal crisis in which ataxy was not a marked symptom.

Another thing which I have noticed, and which is not surprising, is that many of those cases have associated with the laryngeal crisis some affection of the bulbar structures, and not infrequently also of the ocular muscles. The significance of this probably is that such cases have the abductors of the vocal cords weak on account of an affection of their centre in the bulb, and that other neighbouring structures of a similar character—namely, cranial nerve nuclei—are simultaneously affected.

With reference to the cases in which joint troubles are present, the so-called Charcot's joints, probably the most striking thing about them is the extreme deformity of the affected joint, which may be produced without any pain. In fact, it may be taken as almost axiomatic that if you find a case in which there is extreme deformity of the joint, the deformity having arisen without any pain and with undue mobility, it may be concluded that the joint is a so-called Charcot's joint. In those cases also it is remarkable how very little ataxia may be present, although other symptoms of tabes may be quite striking. I have two cases under my observation at the present time, which I am fortunately able to show you, in both of which this is very marked. Curiously enough, both those cases have the hip-joint affected, and they are the only cases of tabes with joint affection that I have seen in which that joint has been the one to suffer. In both of those cases there is a long history of weakness which the joint trouble has produced. You will find, on examining the hip-joint, that the head of the bone has practically gone, and that the shortened femur is working freely in a large cavity on the dorsum of the innominate bone. The remarkable thing is that a patient with this condition is able to get about, and has been able to get about for the last twelve years without a very great deal of difficulty, and without producing any fracture or any other coarse lesion of the leg. It is true there have been lightning pains, there is an absence of knee-jerks, and the Argyll-Robertson pupil is present, but a very remarkable thing is what I have just stated, that there is practically no affection of the power of locomotion and no interference with co-ordination. The same thing has been true of a large proportion of the cases of joint affection which I have known. Of course it is not a very common complication, and I do not suppose that I have seen more than twelve or fifteen cases; but I can only recall one or two in which there was a slight degree of ataxy, and I do not remember any one in which the ataxy was severe.

With reference to cases in which other trophic signs—for we must regard Charcot's joint as trophic in character—cases of course of perforating ulcer are not very uncommon; but I cannot agree to what I at least was taught, that a perforating ulcer never heals. I have known several cases of perforating ulcer in which complete and so far permanent healing has taken place. In those cases also my impression is that the ataxy has not been a striking feature in the clinical picture. In some cases, it is true, there has been some ataxy, but I think it is not the rule that severe ataxy is present. I have known trophic ulcer, associated with a condition very like that occurring in Raynaud's disease, extreme cyanosis of the extremities, but no alternation of pallor, and no loss of substance. One case of this kind I remember very well in which all the symptoms of tabes were present, except that of ataxy, and slight knee-jerks could still be obtained.

Before showing you the patients who have been kind enough to come here to-day, I should like to say a few words about the treatment of these cases of tabes. As to the cases in which ocular muscles—especially those supplied by the

third nerve—are affected, I think mercury and potassium iodide should be freely and energetically administered in all of them. I have thought that such conditions are possibly the result of true syphilitic lesions associated with the post-syphilitic degeneration which tabes probably is, and that the almost invariable improvement which follows the use of anti-syphilitic remedies is evidence in favour of this view. At all events it is, I am sure, in such cases very desirable to push this treatment. In reference to the optic atrophy, that, I fear, if it is tabetic, is a most intractable condition. In the cases in which it is an early symptom, it almost invariably, in spite of all treatment, even the hypodermic use of strychnine, goes on to complete atrophy and consequently total blindness. When, however, it occurs after the disease is well established, especially if ataxy is present, it is nearly always very slow in its advance, and in some cases does not seem to be progressive. I cannot offer any explanation of this—as I believe it to be—fact. Of course, unfortunately the most common tabetic optic atrophy is that which occurs as an early symptom of the disease, or as a rule unassociated with ataxy. In such cases the outlook as regards the patient's vision is extremely bad. And it must also be remembered that such optic atrophy occasionally marks the early stage of general paralysis. I have already indicated the danger in cases of laryngeal crisis and the precautionary operative measures that may be necessary, namely, tracheotomy. In regard to the joint cases, drainage seems to be occasionally beneficial. This is probably more appropriate in cases in which the affected joint is of the upper limb. In cases of affection of the lower limb, while the patient is still able to get about fairly well, one would naturally hesitate to urge any operative measure, as the after-condition of the patient might be considerably worse, and is not likely to be improved.

CASE I.—The first patient I shall speak of is a man of 31, who came complaining of pain in his legs and face, which had been present for some time. Recently, however, he had been alarmed by his gradually diminishing vision, and it was this that finally drove him to seek advice. He has also some difficulty in holding his water. His pupils are inactive to light, and his knee-jerks cannot be elicited. He has marked optic atrophy, with considerably diminished vision, but little or no ataxy. The outlook in his case is, I fear, an extremely bad one as regards his vision. His other symptoms are not serious, and may subside or not become worse.

CASE II.—The next patient whose case I should like to refer to is a man of 37, whose vision began to fail three years ago. He attended Moorfields, but his sight has gradually deteriorated. He suffers also with pains in his head, he has Argyll-Robertson pupil and optic atrophy, and his knee-jerks cannot be obtained. He has no ataxy, and although he has some slight pains (which he attributes to muscular rheumatism in his legs), the severest pains are those in his head. This case is similar to the last in the absence of ataxy and the presence of optic atrophy, but on account of the slower progress of the atrophy, I think there is more hope in his case than that it may stop before sight is completely lost.

CASE III.—The next case is that of a man of 47, who was under the care of my colleague, Mr. Percy Flemming, at Moorfields. He came there in September last with the history of failing vision since the previous October. He had $\frac{1}{2}$ in the right eye, and could not see even fingers with the left. He had Argyll-Robertson pupils and optic atrophy but no pains, and his knee-jerks were active. This is one of those interesting cases in which the diagnosis is one of difficulty between tabes on the one hand and the early stage of general paralysis on the other. It is true no mental symptoms are present and the knee-jerks are likely to disappear, yet at present I am inclined to think the case is more likely to be a commencing one of general paralysis.

CASE IV.—This is a similar case—that of a man of 32, whose sight recently became bad. He was very nervous, his speech a little hesitating. He had unequal pupils, which did not react to light, but his knee-jerks were present. In this case also it is doubtful whether we are not dealing with a tabetic general paralytic in an early stage. I am inclined to think that is the probable diagnosis.

CASE V.—H. R. The next is an interesting example of tabes without ataxy. A man of 35, without pain, with absent knee-jerks, Argyll-Robertson pupils, and optic atrophy. His sight has only now become defective, but the discs are very pale and undoubtedly atrophic.

CASE VI.—F. R. The next is a similar case—a man, aged 57, without knee-jerks, who has a few pains but very atrophic discs. His pupils, however, react normally, and his is a type of the cases I have referred to in which, in spite of the presence of optic atrophy, the pupil reaction is normal.

CASE VII.—G. R. N. The next patient, a man of 38, came first in December, 1900. He then had failing vision, atrophic discs, Argyll-Robertson pupils, but active knee-jerks. At the end of a year, however, the knee-jerks could not be obtained without reinforcement, and even then were exceedingly slight. His sight had still more failed, and he is now blind.

CASE VIII.—E. L. The next case is a very interesting one—a man, aged 51 in 1898, when I first saw him. He came on account of weakness in the legs, and gave a history of dislocation of his leg ten years before. He had remained lame since. There was no accident, nor was the so-called dislocation attended with any pain. On examination it was found that one leg was much shorter than the other, was deformed at the hip, and unduly movable. The head of the femur could be felt moving on the dorsum of the ileum, he had no knee-jerks, and his pupils did not

react to light, the case being undoubtedly one of Charcot's disease of the hip-joint in a case of tabes. The ataxy is very slight indeed.

CASE IX.—E. B. The next case is very similar, only it occurs in a woman. The lameness has been present for only one year. She has ptosis, Argyll-Robertson pupils, no knee-jerks, and no definite ataxy. The deformity at the hip is almost identical with that in the previous patient.

CASE X.—C. I. The last case is that of a man, aged 39, with absent knee-jerks, optic atrophy, and Argyll-Robertson's pupils. He had no ataxy, nor has he now five years after being first seen; but his sight rapidly failed, and he has now been blind for some years. His other symptoms have not become any more marked.

ACUTE NON-SUPPURATIVE PERINEPHRITIS.

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INFLAMMATION of the cellular and adipose tissue surrounding the kidney has been divided into three varieties, the lipomatous, the sclerosing, and the suppurative. The case about to be described does not, however, come under any of these headings, and must be described as one of acute non-suppurative perinephritis, and as it is the first case of the kind which has come under my notice, either in surgical practice, or, at an earlier period, while I was pathologist in the Glasgow Royal Infirmary, I deem it to be one worthy of placing on record.

Acute Pleurisy lasting for over four weeks, followed by severe pain in the Right Lumbar Region, unassociated with Fever or any Symptoms of Renal Disease. Physical Signs showed great Enlargement in the Right Renal Region.

R. A. was admitted to the Glasgow Royal Infirmary on December 20th, 1901. He enjoyed good health up to eight weeks prior to admission, when, according to Dr. Service, of Mossend, he developed a pleurisy. For this he was treated and confined to the house during four weeks. The pleurisy was said to have started on the right side and then extended to the base of the left lung. The symptoms and physical signs of pleurisy having disappeared, the patient was allowed out for the first time about three weeks prior to admission to the infirmary. On this occasion the patient believes that he "caught a chill," and the next day he complained of severe pain in the right lumbar region, and this continued and increased in severity up to the time of admission. When he was brought into the hospital he looked extremely ill. His face was pale with dark rings under the eyes. The temperature was 99°F , respirations were 20 per minute, and the pulse was 88, and of very low tension. He had a slight cough which he tried to restrain, on account of the pain produced by the act of coughing, and there was a little muco-purulent expectoration. There was very slight dullness at the base of the right lung behind, marked diminution in the respiratory murmur, and also of the vocal resonance and fremitus. The heart was normal.

While sitting or standing the body was bent forward and inclined towards the right side. In bed he lay with the spinal column fixed and slightly deviated to the right, so much so that the lower ribs almost touched the crest of the ilium. The right thigh he kept flexed, and he could not extend it beyond 165° degrees. Every movement cost the patient great pain in the loin, but he did not complain of any pain in the thigh or knee. There was no atrophy of the muscular structures, no feverishness and no pain below the level of the crest of the right ilium. He complained of considerable spontaneous pain, of great tenderness on pressure on the right side of the abdomen, and on palpation the abdominal muscles were found to be very rigid, so that it was only when the patient was placed under the influence of an anaesthetic that the swelling in the renal region could be made out.

On examination of the abdomen under chloroform the whole space between the right costal cartilages and the ilium was found to be occupied by a swelling extending forward to within four inches of the middle line. The liver was pressed downwards and to the left, so that the lower margin was three and a-half inches below the ensiform cartilage. The swelling in the renal region was non-fluctuant, firm, and slightly elastic; there was no very distinct bulging, and no unnatural coloration of the skin over the part. The urine was pale amber colour, slightly acid, the specific gravity was 1.015, and on standing a slight mucous deposit was thrown down. There were no tube casts, no albumen, and no pus or blood found in the urine.

December 21st.—During the night the patient suffered great agony, and the pain was only relieved by hypodermic injections of morphine. During the night the temperature never rose above 99.8° . The patient was placed under an anaesthetic and the lumbar region explored. Surrounding the kidney on all sides there was a huge mass of recently formed inflammatory tissue mixed with fat. Before the kidney was reached the incision had to extend through fully $3\frac{1}{2}$ inches of this inflammatory material. On pressing the hand upwards under the diaphragm the inflammatory induration was found to extend almost to the middle line, and downwards it spread well into the false pelvis. Several incisions were made with the expectation of finding pus, but none was discovered. Drainage tubes were inserted and the wound sutured. The kidney appeared to be healthy and did not contain any calculi.

December 30th.—Since the operation the patient was almost entirely relieved of pain, and a considerable quantity of serous fluid and oil escaped from the wound, but no pus. The temperature remained normal, and the urine typically healthy.

January 8th.—Since last note was made the same kind of discharge has continued to escape, but in smaller quantities than during the first week after the operation. Now the swelling is diminished in size by about one-third, and the patient does not now complain of any pain.

January 24th.—The drainage tubes were kept in until January 17th, when they were removed as the discharge had practically stopped. Now