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CLINICAL LECTURES

ON

DISEASES OF THE NERVOUS SYSTEM

CLINICAL LECTURES

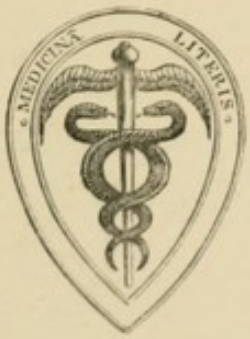
ON

DISEASES OF THE NERVOUS SYSTEM

BY

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PREFACE

Some of the lectures in this volume were delivered at the National Hospital for the Paralysed and Epileptic, and abstracts of them have appeared in the medical journals. Others have not been delivered to a class, although many of the opinions expressed in them have been communicated to medical men and students who have from time to time attended the practice of the hospital. Besides these a certain number of clinical contributions to medical societies and journals have been reproduced, with such modifications and additions as were necessary, not only to preserve some continuity in the style of composition, but with the more important object of securing an harmonious treatment of the several leading topics. So extensive, in many instances, have been the revisions and additions that the lectures, as they now appear, are essentially new.

The series, as will be seen, does not represent a systematic course on even a large division of diseases of the nervous system. Although the choice of the subjects here discussed has to a certain extent depended upon the cases of disease which were under observation at the

time of lecturing, these are always so diverse in character that the actual selection has been suggested by special circumstances appearing to claim for a particular topic a great and preponderating attention. It is hoped that the various morbid conditions have been considered from no narrow stand-point, but with a constant endeavour to employ them as steps in the investigation of diseases of the nervous system as a whole.

Whatever value the lectures may possess depends in a great measure upon the extensive field of experience which the author has enjoyed. It will be readily understood that each of them represents the study of a far larger number of clinical cases than is indicated directly in the text.

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DISEASES OF THE NERVOUS SYSTEM.

LECTURE I

TENDON-REFLEX*

Two cases are under observation, which differ in their nature, but combine to illustrate incidentally certain points in the diagnosis of posterior spinal sclerosis (progressive locomotor ataxy). They have a special interest in connection with the diagnostic symptom which we owe to Westphal, and which may be thus described. If a healthy man sits with one knee-joint resting upon the other (the very common attitude), and the ligamentum patellæ of the supported leg be smartly struck just below the knee-cap with the side of the hand, a sudden contraction takes place of the quadriceps femoris muscle (of which the ligamentum patellæ represents the tendon), and the foot is consequently jerked upwards in a degree which varies in different individuals. Now, in confirmed examples of locomotor ataxy this reaction does not take place. No matter on what part of the ligament below the knee-cap, or with what force the blow is struck, the foot hangs motionless. In order to establish with accuracy the absence of the phenomenon certain precautions ought to be taken. The leg should be bare; the patient must not offer voluntary resistance to the movement of his leg, and the ligament should be struck with some hard implement

* From the 'Lancet,' July 27th and August 4th, 1878.

which can be swung like a hammer. I find an ordinary wooden stethoscope answer very well if it is held loosely by the small end, and the blow given with the edge of the ear-piece. But, however administered, several blows should be struck on the ligament, slightly changing the position each time, as there is generally one spot from

Fig. 1.



which the reaction is peculiarly energetic. This is usually a little below, but very near to, the patella. Deferring for after-consideration any attempt to explain the phenomenon, I will describe how we found the test of importance in the diagnosis of a case now in the hospital. Careful and complete notes of the two patients referred to have

been taken by Mr A. E. Broster, from whose account I will, however, only glean sufficient to illustrate the character of the affection from which each is suffering.

M—, a hairdresser, aged fifty, has been in the hospital since April. He complains of numbness, which is spread generally over the whole of his body and extremities, including his mouth and tongue. A day never passes without pains "like electricity," as he says, occurring in his left side and lower extremities, especially in the shins. There are, besides, sensory troubles of various kinds, burning sensations, aching of epigastrium, "cold water running down the legs," a feeling of tightness over the lower part of the belly as if his intestines were pushed out against the abdominal wall; this tightness is greatest when he stands up to walk, and it then seems to him to "force his motions and urine." There is great loss of muscular sense in the arms and legs. In picking up a pencil from the table with his eyes open much irregular movement is shown, no matter which arm is employed; and when his eyes are shut, and his hands placed upon the object, he clutches it repeatedly, but is unable to take it up, and says that he cannot feel it. The soles of his feet are so anæsthetic that he feels neither the carpet, the strongest faradaic currents, nor the prick of a pin; and this condition is observed, though to a much less extent, over the body generally, the deficiency, however, being but slightly, if at all, marked in the face. There is occasional diplopia. The right optic disc is atrophied, the left unduly pale. In sitting, if his eyes are closed, he tends to sway about on his seat. In rising from a chair he is obliged to help himself up by resting his hands upon his thighs, and even with his eyes open he cannot stand steadily. When they are shut, however, he reels and pitches, so that he would fall helplessly if not caught. His gait is extremely ataxic, the steps short, the legs far apart, and kept stiff at the knee-joints. The muscular power is very considerable. His sphincters are but slightly under control. I have said enough probably to

show that this is a case of sclerosis of the posterior columns. There is, indeed, no symptom wanting to form the picture of a typical case of locomotor ataxy, which the history (omitted for the sake of brevity) shows to be progressive in character. Not only are there the electric-shocklike pains and the difficulty of gait intensified by closing the eyes, but there are also the cephalic symptoms in the form of atrophy of the optic disc, and occasional diplopia. I insist rather upon these latter symptoms, because, as regards the former (pains and gait disturbed by closure of the eye) they are liable to exist alone, and, if not very carefully examined, to lead to error. Indeed, in the case which follows an erroneous diagnosis was actually made from a superficial examination having led undue prominence to be given to these symptoms.

The patient B—, aged forty-three, came to us at the hospital last December on account of "paralysis." He walked very unsteadily, with his legs wide apart, and could scarcely walk at all with his eyes closed. He stood upright with difficulty and uncertainty, and the effect produced by closing his eyes in increasing his insecurity was very marked. There was considerable loss of sensibility in the soles of his feet and legs, a sensation of a tight band around his waist, and a constant feeling of weight in his belly. He complained, too, of pains in his legs, and said that sometimes when walking sudden pains would shoot through his knee-joints and nearly throw him down. In the pressure of work, the case, from these data, was hastily assumed to be one of posterior sclerosis, and a note to that effect was recorded on the patient's paper. It was not till some little time afterwards that the diagnosis was amended, and it is to the circumstances under which the correction was made that I wish to draw particular attention. Looking to the brief notes of the case we found that they contained no reference to the condition of the ligamentum patellæ as regards response to blows, and it was thought well to compare the two patients, M— and B—, who were supposed to be suffering from the same disease.

On the same occasion both these men were tested in the following manner:—The patient being seated, and his legs having been bared, the ham of one leg was made to rest easily upon the knee of its fellow. The ligamentum patellæ was then smartly struck at several points near to the knee-cap with the ear-piece of a stethoscope. In the case of M— there was no response, at whatever part the ligament was struck—the foot never moved; and this failure occurred equally when the opposite leg was submitted to a similar examination. When B—, on the other hand, was tested in a like manner a comparatively slight tap on the ligamentum patellæ—a more gentle blow, indeed, thau is ordinarily required in health—caused the foot to be jerked some four or five inches upwards. A similar effect was produced when the other leg was tested. The result caused surprise and interest, as it was to all appearance a startling exception to that we had got to look upon as the rule—viz. that in confirmed cases of locomotor ataxy a blow on the ligamentum patellæ causes no jerk upwards of the foot. Amongst the cases of confirmed locomotor ataxy in which I had applied the procedure, I had not found one in which examination tended to disprove the correctness of Westphal's observation. The result in the present instance, therefore, of course led us to inquire into the patient's symptoms with more accuracy than had yet been done, and this is what we found. B—'s illness had commenced *suddenly* four years previously. He had gone to bed quite well, and when he got up next morning he felt as if his feet were asleep, and was unable to feel the ground. The numbness crept up his legs and trunk, reaching to the navel in the course of a month, and his walking power failed. A few months later pains began in his legs, but these he described, when closely questioned, as *dull*, *heavy* and *incessant*. The pains in the knee-joint already described had occurred a few times nearly a year previously, but only when he was walking, and they had not been repeated. There was aching pain, too, in the left arm, which was some-

what wasted, and had lost power. The strength of his legs was so diminished that, as he sat and endeavoured to lift either knee, pressure with one finger of the observer sufficed to prevent any upward movement. He was liable to jumpings of his legs when in bed. The bladder was completely paralysed. Tickling the soles of his feet caused a feeling of shocks running up the legs, and there were exaggerated reflex contractions. But here we may stop. In order to make a diagnosis of the cause of this man's paralysis we should need to go at still greater length into the history and symptoms, and the study would be wide of our present purpose. It is only necessary to say that his symptoms point to an affection of the cord which is not posterior sclerosis. The case is not one of progressive locomotor ataxy, and the preservation in this instance of that which Westphal calls the "knee phenomenon" or "leg phenomenon" is therefore in no respect to be taken as an exception to what has been found to be the rule.

It need not be said that this test, to be valuable, must be shown to be fairly constant in its results. If we were to find in many cases of well-marked and characteristic posterior sclerosis the knee phenomenon retained, the value of the suggestion would immediately sink considerably. Up to the present time I have heard of no such exception having been discovered. In 1875 Professor Westphal wrote:* "The leg phenomenon was constantly absent in all the undoubted cases of tabes dorsalis submitted to trial." An extended experience of three years now enables him to confirm his report, and in a paper published last January he advances a stage further, and raises the interesting question whether *before* the development of the characteristic symptoms of tabes dorsalis (locomotor ataxy) the leg phenomenon is wanting, and whether, therefore, the fact of this reaction being absent can be utilised thus early as a diagnostic sign. He has seen, he says, cases in which there were the characteristic pains without ataxy or any

* 'Archiv. für Psychiatrie.' Berlin, 1875.

sensory troubles, but with commencing atrophy of the optic disc, and in these the knee phenomenon was absent.

A case has been recorded by me * in which lightning pains of a very characteristic kind, and continued during many years, were accompanied by atrophy of the optic discs, but were not associated with any ataxy or anæsthesia. In this case the knee phenomenon was "conspicuous by its absence." I have had under occasional observation for some years a gentleman arduously engaged in his profession, who suffers from time to time paroxysmal attacks of shooting pains, which he attributes to gout, but which have all the characters of those belonging to posterior spinal sclerosis. His gait is quite unaffected, there are no symptoms referable to the cerebral nerves, and the only deviation from health, besides the pains, of which he is conscious (and this is trifling, although significant enough), is a tendency to imperfect control of his sphincters, if the opportunity of obtaining relief is long delayed. In this case careful percussion (the skin being bare) failed to produce any evidence of patellar tendon-reflex. I have lately been consulted by a gentleman on account of pains, which had been in his case likewise attributed to gout, but which were characteristically "lightning-like" in character. This person not only exhibits no sign of muscular incoordination, but his delight is in mountain-climbing. He presents, indeed, none of the symptoms ordinarily attributable to posterior spinal sclerosis, except the tell-tale pains. In his case, again, examination shows a total absence of Westphal's knee phenomenon. This is the only example I have at present met with of the test being applied where the indications of tabes dorsalis were absolutely limited to the occurrence of pains. In another case under my observation, in the person of a gentleman over seventy years of age, the pains are precisely of similar character; the gait is, besides, somewhat ataxic. Here equally it is to be noted there is no response to blows upon the patellar tendon.

* 'Brain,' July, 1878.

Two questions naturally arise. What is the cause of the knee phenomenon? Why is it absent in posterior spinal sclerosis? I do not think we are as yet in a position to give a positive answer to either, and the subject is still under investigation. The first idea is that the contraction of the muscle must be brought about by a reflex from the skin at the part which is struck. But Westphal has practically disposed of this explanation. He pinched and pricked and irritated the integument in various ways without effect, even submitting a fold of the skin, lifted away from the tendon, to smart blows with a hammer. On the other hand, when the skin lying over the ligamentum patellæ was frozen by Richardson's process the effect of blows upon this spot in determining the contraction was in no way lessened. So, also, in cases (not being examples of locomotor ataxy) where there was complete cutaneous anæsthesia, the "phenomenon" has been found present. The influence of the skin must therefore be excluded.

Erb has suggested that the blow upon the patellar tendon, by suddenly stretching it, irritates some nervous fibres belonging to the tendon, the impression thus produced being conveyed to the cord, and there exciting the motor nerves to the muscles. On the other hand, the experiments of Tschirjew, of St. Petersburg, seem to show that the reflex is to be ascribed to irritation of sensory nerves distributed to the aponeurosis of muscle. He found that section of the cord in guinea-pigs above the place of entrance of the sixth lumbar nerve roots caused the phenomenon to disappear. In addition to this, when the posterior root of one of the sixth lumbar nerves was divided the phenomenon failed on the corresponding side. If these experiments were free from fallacy, the reflex character would seem to be placed beyond doubt. Degeneration of the cord at the point where the nerves enter may easily be supposed to be capable of interfering with the orderly reflex which thus occurs in health, and in this way the effect of posterior spinal

sclerosis in preventing the exhibition of patellar tendon-reflex may possibly be explained. The very rare circumstance of the lumbar portion of the cord escaping the degenerative changes in tabes dorsalis, would thus sufficiently account for the certainty with which, apparently, this diagnostic sign may be looked for.

There is an exception to this rule of the absence of knee phenomenon in tabes dorsalis, which is pointed out by Westphal, and is very necessary to be borne in mind. In a case in which the degeneration of the posterior columns does not extend to the lower dorsal and lumbar portions of the cord, and in which, at the same time, there is a continuous descending sclerosis of the lateral columns of the cord from the cervical to the lumbar region, the knee phenomenon will be preserved.

We may take it, then, that the presence of patellar tendon-reflex is the rule in health; and that its absence frequently points, as we have seen, to sclerosis of the posterior columns of the cord. This negative symptom may evidently, therefore, prove to be of no small value where there is occasion for doubt, and especially when it is a question of the cause of atrophy of the optic discs in certain cases. It is necessary, however, to remember that the mere absence of this reaction is not of itself necessarily diagnostic of tabes dorsalis. If the cord be greatly disorganised in its lumbar portion—as *e.g.* by acute myelitis or softening—blows upon the ligamentum patellæ will fail to excite contraction of the quadriceps extensor muscle. In such a case the paralysis of the lower extremities will be so pronounced as of itself to exclude the diagnosis of locomotor ataxy. Moreover, there is always the possibility to be considered of the phenomenon being naturally absent in some healthy persons. Westphal says he has seen no instance of this. I certainly failed to produce the reflex not long since in a member of our profession, whose health, happily, leaves nothing to be desired. The experiment was, however, inconclusive, as the skin was not bared.

There is one source of error against which it is necessary to guard, as tending to a false conclusion, and which I can illustrate by the patient whom I now show to you, who represents a typical case of progressive locomotor ataxy. Incidentally, besides indicating an error which is to be avoided, the application of the process in this instance shows, I think, the *modus operandi* of the test.

This patient, then, has the staggering gait so well marked that the nature of his disease is suggested at the first glance, and is confirmed by the account which he gives us of lightning pains and transient diplopia. When his legs are crossed, and one foot is pendulous, if we strike upon any part of the ligamentum patellæ there is no response whatever, the foot remains perfectly still. So, also, if we strike in the situation of the *tendinous* structure above the knee-cap, there is an equal absence of any response. When, however, the blow is allowed to fall where the plump of *muscle* somewhat projects above and to the inner side of the patella (the vastus internus), there is a very slight upward movement of the foot. It is important to distinguish this action from the patellar tendon-reflex proper, which is present in health but is lost in locomotor ataxy. Examining the movement, we see that it is slow as well as slight, and is occasioned by the contraction of a very limited number of the muscular fibres of the vastus internus. That this contraction is confined to the muscular bundles of the part struck is shown by the very palpable wave which appears at the spot. The wave is deep, slow in movement, and its direction is obliquely downwards and outwards, corresponding with the direction of the bundles of muscular fibres. It contrasts as strongly as may well be with the sudden and rapid thrill of contraction which may be felt or seen to occur in various and distant parts of the quadriceps in response to a blow upon the patellar tendon in a healthy person. Just such a wave of contraction usually (but not always) occurs, if you strike the vastus internus of a healthy person, and it appears to show (what is

now generally accepted) that muscular structure may be *directly* excited by a mechanical stimulus—*i.e.* without the intervention of the intra-muscular nerves. That the contraction following upon such a stimulus is really independent of the influence of the motor nerves, and proceeds from the direct action of the stimulus upon the muscular structure, seems to me practically established for the following reasons:—The contraction in question bears the closest possible resemblance to the sluggish wave which may, in certain circumstances, be seen to pass through a muscle which is completely cut off by disease or injury from the influence of a motor nerve, or of its centre of nutrition in the cord, when you excite this muscular structure by opening and closing the circuit of a weak voltaic current. On the other hand, it is in striking contrast with the short, quick, and much more general contraction which responds to stimulation of the motor nerve, where this is healthy and its communications with muscle and a normal spinal cord uninterrupted.

Considering, then, that in this man we have a muscle which is manifestly quite ready to be excited by a mechanical stimulus, and that blows upon the tendon altogether fail to excite it, I think we may take it that the "knee phenomenon" of Westphal is not produced simply by direct and mechanical influence upon the muscular structure of the quadriceps. The muscle is here, indeed, more than normally ready to contract upon direct local stimulation, yet no amount of pulling upon it by means of blows upon the tendon—and the mechanical effect of these blows must be to pull upon the muscle, tendinous structure being practically inextensible—produces the desired effect. The conclusion seems inevitable that the sudden and general contraction of the quadriceps seen to occur in health in response to blows upon the ligamentum patellæ is brought about in a reflex manner through the medium of centripetal nerves, their centre in the cord, and the motor nerves to the muscles. The degeneration of the cord in posterior spinal sclerosis appears, by inter-

rupting this nervous arc, to prevent the accomplishment of the normal reflex. The slight movement of the foot, then, which we have just seen in a typical case of ataxy in response to a blow upon the *muscular* structure, is something quite apart from the patellar tendon-reflex and must not be confounded with it.

Other tendons in various situations, when they are suddenly pulled upon, bring about reflex contraction of the muscular structure continuous with them. It seems probable, indeed, that this is constantly the case in health, but by reason of the size of the structures engaged, and their mechanical disposition, the phenomenon is shown by the ligamentum patellæ better than in other places.

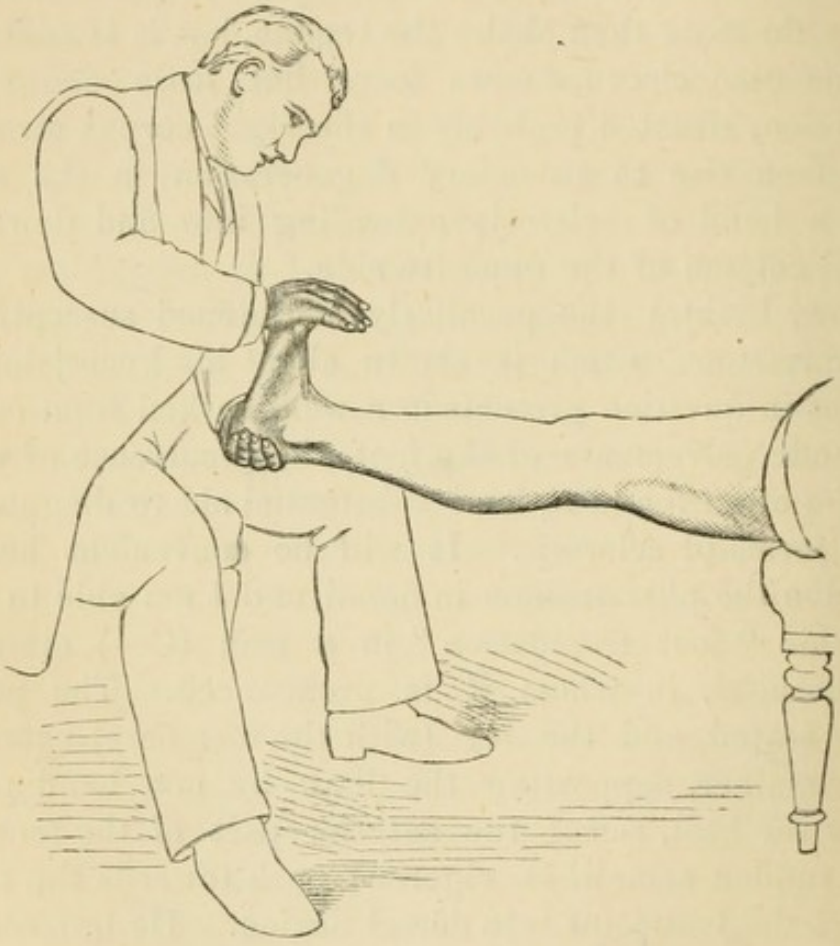
But just as under certain circumstances the patellar tendon-reflex may be absent, it may also under other conditions be greatly intensified. A blow of the same strength as produces a very slight movement of the foot in health, may, in certain circumstances, cause a jerk upwards measuring many inches; or a much lighter blow than will cause any response in health may be found sufficient to produce a very decided kick. This we have seen to be the case in the patient B—. In another patient under observation at the present time this exaggeration of susceptibility is particularly well shown. The man has recovered to a very great extent from an attack of left hemiplegia, diagnosed as dependent upon softening from arterial thrombosis. He is able to be on his feet all day without inconvenience, and shows nothing to the eye but a little stiffness in his gait, and a very trifling awkwardness in the more delicate movements of his left hand. If he sits with the left leg crossing the right, and with the foot pendulous, and one gives a slight fillip with the thumb and forefinger upon any part of the ligamentum patellæ, down to its insertion, there is a jerk upwards of the foot to the extent of from eight to twelve inches. But this is not all; a similar fillip a little *above* the patella produces a like though somewhat smaller effect. It is to be noted, however, that the tap must be on the *tendon* of

the rectus femoris or of the vastus externus to produce this jerk. If it falls upon the *muscular* structure itself there is no kick, but only a slight and slow movement upward of the foot similar to that which we have seen to occur in the ataxic patient. In addition to this, a slight tap upon the centre of the knee-cap itself produces the characteristic contraction of the quadriceps. This can hardly do more than shake the tendon, but it is sufficient. In this case circumstances leave but little doubt that the lesion, situated probably in the right corpus striatum, has given rise to secondary degeneration in the spinal cord, a band of sclerosis extending into and down the lateral column of the opposite side.

Now, besides the peculiarly heightened susceptibility to impressions which is shown about his knee-joint, the patient in question presents in a well-marked form certain rhythmic movements of the foot, the occurrence of which will be often found to lend substantial aid to diagnosis in some forms of sclerosis. It will be convenient here to describe the phenomenon in detail, and I am able to show you this "foot trepidation" in a man (C—) attending the hospital, in whom it is pronounced. The patient being seated and the leg (with the foot bare) extended, the examiner, supporting the limb by one hand placed below the heel, seizes the anterior part of the foot, and by a sudden somewhat vigorous push towards the trunk, brings the foot-joint into dorsal flexion. He immediately feels a downward pressure of the foot—a strong tendency for it to be pointed—which he resists by continuing his pressure. Then comes a series of rapid and rhythmical movements (flexion and extension) of the foot, which continue as long as the pressure is kept up, or until the muscles are tired. (Occasionally if the movements do not appear when the foot is dorsal-flexed, a slight blow upon the tendo Achillis will have the effect of starting them.) Now, this patient tells us that the trepidation is also apt to occur if he puts his foot into a certain position —if, *e.g.* he rests the anterior part of the sole upon the

edge of a chair whilst he laces his boot. In doing this he evidently puts the tendo Achillis on the stretch. That it is to the strain upon the muscle through the medium of the inextensible tendon that we must refer the initiation

FIG. 2.*



of the movements, and not to any influence of the skin of the foot-sole, is proved, I think, very simply and completely by a case in which I first noticed this phenomenon six years ago. The patient, a man aged thirty, was paraplegic, and could not stand without support. Seated, with the foot upon the ground and somewhat advanced, so that the leg formed a slightly obtuse angle with the thigh, he remained still enough; but if with the aid of his hand he

* It is better that the knee-joint should be less fully extended than is shown in the sketch.

dragged the foot backwards towards himself, on arriving at a certain point the knee would be jerked vigorously up and down by the rising and falling of his heel, so violently indeed that the room would shake again with the rhythmic movements. It reminded one so much of the action of a man who works at a lathe that I have often since pointed out a similar condition in other patients under the name of the "turning-lathe movement." In the case described it will be seen that, as the foot touched the floor all along, the skin was necessarily subjected to the same impression throughout the proceedings; but it was only when by drawing the foot under him, the tendo-Achillis was pulled upon, that the movements were started. The possibility of an action upon the skin, therefore, being the exciting cause must be eliminated from consideration. (I may say that when I last saw this patient, two years ago, the foot trepidation continued to occur in similar circumstances.)

This phenomenon is so remarkable that it must have been observed by many besides those who have referred to it in writing. It bears a close resemblance to the symptom which Brown-Séquard called by the name of "spinal epilepsy,* but I have not seen the movements stopped, as he describes, by suddenly and forcibly bending the big toe. Nor has it escaped the ever-watchful eye of Charcot.† Recently increased attention has been brought to bear upon it, and it has been minutely described by Westphal (in the paper already referred to), and also by Erb, who has bestowed upon it the convenient term "reflex clonus."‡ In all likelihood the process belongs to the same category as the patellar tendon-reflex, although at the first glance it does not appear easy to reconcile with this view the apparently automatic *recurrence* of move-

* 'Archives de Physiologie,' vol. i, p. 158.

† 'Leçons sur les Maladies du Système Nerveux.' Paris, 1873, 3me fasc., p. 218.

‡ "Ueber Sehnenreflexe bei Gesunden und bei Rückenmarks-kranken." 'Archiv für Psychiatrie.' Berlin, 1875.

ments. What happens is probably this: The sudden dorsal flexion of the foot, by its strain upon the tendo Achillis, causes reflex contraction of the muscles of the calf, and the tendon is consequently pulled up. But the pressure of the operator's hand, although it has been overpowered for an instant by the contraction of these powerful muscles, is still continuing to act against the anterior part of the foot with the effect of again pulling the tendo Achillis, and this in its turn again excites contraction. The anatomical arrangement is such that the hand works a lever of the first class, the lower extremity of the leg-bones being the fulcrum, and the tuberosity of the os calcis marking the position of the weight. In consequence of the leverage the operator is able to exert a very considerable power of traction upon the tendon which has been pulled up by the contracting sural muscles. The movements may be immediately stopped by bringing the foot into plantar flexion.

The patellar tendon-reflex, as we have seen, is a phenomenon which occurs in health; the reflex clonus of the foot, on the other hand, cannot be produced, so far as I am aware, by dorsal flexion of the foot of a healthy person, although an approach to it will sometimes occur spontaneously in persons who are quite well. The occurrence of the foot-trepidation along with greatly heightened patellar tendon-reflex, as in the case of the hemiplegic patient, is evidence, so far as it goes, of a direct association in the cause of the two phenomena. In his case, as in numerous others in which reflex clonus of the foot has been seen to occur, the existence of lateral sclerosis cannot be doubted. How far the occurrence of these movements is pathognomonic of particular situations of sclerosis in the spinal cord, and the diagnostic value of the symptom in the case of the patient C—, in whose person I have shown them, must be left for future consideration.

LECTURE II

TENDON-REFLEX* (*continued*)

In the remarks which I have to offer on the subject of what is called "tendon-reflex" it is provisionally assumed that the contraction of a muscle which follows a blow upon its tendon is of the nature of a reflex action. The adequacy of such an explanation is, I am well aware, contested by high authorities in physiology. Measurements of the interval of time which elapses between the blow and the contraction are thought to prove that this is too short for the phenomenon to be reflex. I cannot as yet consider this to be a conclusive objection, for whilst, of course, acknowledging the respect which is due to attempts at accurate measurements, experience shows that, even in very careful hands, where the question is one of delicacy—in the present instance it is a matter of a very few hundredths of a second—sources of fallacy are apt to occur in the experiments which vitiate the conclusion. It is quite conceivable, moreover, that a shorter period of time than is commonly needed for a reflex event is requisite for conduction through the centre, where, as in the present instance, the centripetal stimulation is in all probability unusually strong. Nor can we properly compare it with the time required for reflex from the skin, since the fact that in tabes (locomotor ataxy), for example, whilst the skin-reflex is usually preserved intact, the tendon-reflex is almost always lost, shows that the conduction must, at least in some part of the course, take place through a different path.† At any

* From the 'Lancet,' November 27th, 1880.

† The literature of the subject has rapidly become extensive, and includes,

rate, it seems advisable that at the present juncture the clinical facts should have due prominence, and there can be no question, I think, that they make out a strong *primâ facie* case in favour of the reflex character of the muscular spasm. With the view of aiding in the consideration of the question from this point of view some rough diagrams are reproduced, which I have been long in the habit of using in order to illustrate what is possibly the mode of connection between certain pathological changes in the spinal cord and the absence or exaggeration of "tendon-reflex." For the sake of simplicity, the remarks will be confined as far as possible to "patellar tendon-reflex" and "ankle clonus" as representatives of phenomena which may, as is well known, occur in connection with other tendons and muscles.

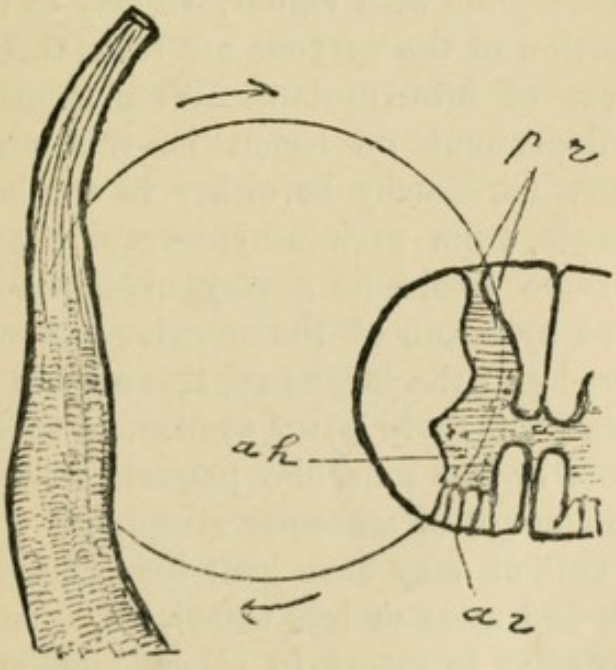
The patellar tendon-reflex may be conveniently tested, in most instances, by the following procedure, which differs from that usually employed:—The patient, who is seated, plants his foot firmly down at such a distance that the leg forms a little more than a right angle with the thigh. Whilst the observer rests the palm of his left hand upon the patient's thigh, he strikes with some implement held in the right hand several blows upon the ligamentum patellæ at about an eighth of an inch or so below the knee-cap. I nearly always employ for this purpose an ebony stethoscope, with a very heavy ear-piece and an india-rubber cord-like ring sunk into a groove on its margin. The quadriceps muscle can be felt, and (if the patient is in male attire) can be seen to contract more or less vigorously in response. If it be desired to ascertain the mere presence of the "reflex," this is sufficient. If, however, the response is very small, or is doubtful, or does not occur at all, the thigh as well

besides the work of Westphal and Erb, who first called attention to these phenomena, most important contributions by Tschirjew, Grainger Stewart, Gowers, Hughlings Jackson, Bramwell, Charcot, Brissaud, and Berger, amongst many others. There is an able article by Dr Augustus Waller in 'Brain,' part x, in which the physiological side of the question is discussed, and the objections to the theory of reflex action are very forcibly put.

as the leg must be bared, and other measures adopted, before a conclusion is arrived at. The leg should be crossed over the other, or the patient should be made to sit upon a table with his leg dangling. In either of these positions the presence of the "reflex" will be shown by the foot being jerked more or less vigorously forwards when the ligamentum patellæ is struck. In the case of a patient undressed, and lying on a bed, the knee should be flexed, and the heel held firmly down upon the bedding whilst the blow is given on the ligament. The contraction of the quadriceps is readily seen.

Supposing that careful examination has established the absence of the knee-phenomenon, we should next endeavour to ascertain at what point in the arc of nervous communication the break occurs by which the process is interrupted. Fig. 3 shows the normal condition as it

FIG. 3.



may be supposed to be if the phenomenon be reflex. Impulses started by a blow on the tendon after traversing the afferent nerve, enter the cord in the posterior root *p r*, and reach the anterior horn, *a h*, where they give rise to efferent impulses, which arrive by the anterior root, *a r*, at the muscle, and cause it to contract. From the tendon

to the anterior horn is the sensory portion of the arc, whilst the motor part includes the anterior horn, anterior root, and intra-muscular nerve. The integrity of this nervous arc ensures the response of the muscle (provided the muscle itself be healthy) to a blow on its tendon. So also the fact that the quadriceps is found to contract freely when the ligamentum patellæ has been struck, *almost always* signifies that the nervous arc is nowhere seriously interrupted in that part of the spinal cord which gives origin to the lumbar plexus. It is not, however, quite conclusive on this score, for a reason to which I shall refer later. If, on the other hand, careful testing shows the phenomenon to be absent, it is evident that there is a "fault" (as the miners say) or break at some point or other in the continuity of the nervous arc pictured in the diagram. Should the patient who presents this abnormality be walking freely about, and able to flex and extend his knee-joint with vigour, we may at once absolve the *motor* portion of the nervous arc from the suspicion of being the seat of interruption. Were it otherwise, and the break dependent on lesion in this situation, the "fault" must necessarily be either in the muscle itself (as, for example, from pseudo-hypertrophic paralysis), in which case there would be a very evident enfeeblement, or else caused by lesion of the anterior root of the spinal nerve or atrophy of the large ganglion cells in the anterior horn. In each of these latter conditions there would be not only more or less muscular powerlessness, but also a considerable amount of muscular atrophy.

But the patient may have kept his bed for some time, and be weak and more or less emaciated. In this case it becomes necessary to resort to other expedients to ascertain the integrity of the motor portion of the arc. If the knee-joint be capable of voluntary flexion and extension with a fair amount of force, it will be sufficient to percuss with some instrument the muscular substance of the vastus internus where it projects just above and to the inner side of the knee-joint. In the absence of a percussion-hammer

this may be done with the thin ear-piece of an ordinary stethoscope.* If this blow, in these circumstances, excites a wave of muscular contraction, it may be taken as corroborative evidence that the muscle, motor nerve, and anterior horn are free from lesion. Should, however, there be a very powerless state of the limb, the occurrence of a wave of contraction in response to a blow upon the muscular tissue of the vastus internus will not be sufficient to prove the integrity of these structures. I have found—and I do not think this fact is generally known—that a muscle which is cut off from its trophic centre, either by lesion of a spinal nerve or atrophy of ganglion cells in the anterior horn of the cord, and fails to contract to the strongest induced currents, will yet (provided it has not lost also the power of contracting to a slowly interrupted voltaic current—the "reaction of degeneration") respond by a very palpable wave of contraction to blows upon it. So that if you strike a muscle and get a wave of contraction, all that you can predicate of it is this, that the muscle will respond to *some form* of electric excitation, not that it will necessarily respond to faradism.† A muscle which thus responds to a blow may be perfectly normal, or, on the other hand, it may be far advanced towards a state of irreparable degeneration. In the latter case, however, the necessarily powerless condition of the muscle secures you from error.

It must be remembered that the patellar tendon-reflex is lost somewhat easily, and as the result of a small amount of structural change. It will often be found wanting when there is but a slight lowering of faradaic excitability. It is best, therefore, whenever there is the slightest doubt about the matter, to test very carefully by

* Hawksley has made for me an ebony stethoscope, provided with a thick and also a thin ear-piece, which are interchangeable. The former is for percussing the tendon, the latter being used for the muscular structure.

† Since this paper was published I have been informed by Professor Erb that he has recorded a similar observation some time ago. The fact was new to me.

induced currents, or the slow interruption of a voltaic current, the reaction of the vastus internus, the rheophore being applied to its motor point, and also to measure carefully the circumference of the thigh and compare it with that of its fellow. The importance of this is evident. If, by any want of care in examination, you jump hastily and erroneously to the conclusion that the motor side of the nervous arc is not in fault, you are driven to the necessity of ascribing the break to a lesion on the sensory side, and consequently err in your diagnosis.

We will consider that no such want of care has been shown, and that the examination excludes the muscle, motor nerve, and anterior horn of grey matter from being the cause of the break in communication. We have now to fall back upon the grey matter of the posterior horn, the posterior root zone, and the posterior root. In one or other of these structures the "fault" must lie. Cases in which the patellar tendon-reflex is absent in health are so extremely rare that this possibility may practically be disregarded—at least until every other explanation has been exhausted.

Through the kindness of my colleague, Dr Ramskill, I am able to refer to a case of extreme interest bearing upon this point. The patient is a young man who is affected with loss of power in the left lower extremity. There is no loss of sensibility in the limb except over a space of about eight inches by four in the neighbourhood of the great trochanter, but there is very extensive wasting of the muscles of the thigh, and their excitability by induced currents is lowered. The right lower extremity is free from paralysis or wasting; the patient can flex and extend it vigorously, and the muscles respond normally to faradaic currents. In this limb, however, there is cutaneous anæsthesia to a very marked extent. He presents, therefore, a typical example of Brown-Séquard's hemi-paraplegia. There would seem to be a lesion, which circumstances show to be probably syphilitic, occupying more or less definitely the left half of the spinal cord in

the lumbar region, and giving rise to motor paralysis on the side of the lesion, and anæsthesia of the opposite limb. The occurrence of anæsthesia, in these circumstances, in the limb opposite to the lesion was explained by Dr Brown-Séquard, it will be remembered, by the fact that the conductors of sensory impressions from the trunk and limbs decussate in the spinal cord, so that an injury or disease of the *left* side of the cord—*e.g.* affects the conductors from the *right* side of the body below. Now, in this patient the patellar tendon-reflex is absent in each leg, not only in the left, which is paralysed and greatly wasted, but in the right also, in which the motor power is intact, but the skin anæsthetic. To explain its absence from the left (paralysed) limb it is evident that the "fault" must be in the anterior horn of the left side of the cord, in the motor portion, that is, of the nervous arc. In the right limb, on the other hand, it is lost in connection with lesion of some part of the sensory tract, the exact situation of which it is not so easy to indicate. It seems probable that the lesion of the cord (myelitis), although greatly confined to the left half, is not absolutely so, but, whilst sparing the right anterior horn, encroaches to some extent upon the right posterior horn, and cuts the communication at this point. The existence of the limited patch of anæsthesia near the *left* great trochanter lends important evidence in favour of this view. The case is a most interesting example of the fact that the absence of the knee-phenomenon may be due to lesion in either the motor or the sensory portion of the nervous arc, both modes being exhibited in one individual.* In certain cases—as, *e.g.* where there is a diffuse myelitis involving the whole transverse section of the cord—it may be due to both.

The most common and conspicuous illustration of a

* On any hypothesis but that of a reflex origin of the contractions in question, it is difficult to explain this equal loss of the phenomenon in two limbs, which are under entirely different conditions—one being affected as to its motility, the other as to the sensibility of the skin covering it.

flaw on the *sensory* side of the nervous arc destroying this reflex, is in the case of tabes dorsalis (locomotor ataxy).

FIG. 4.

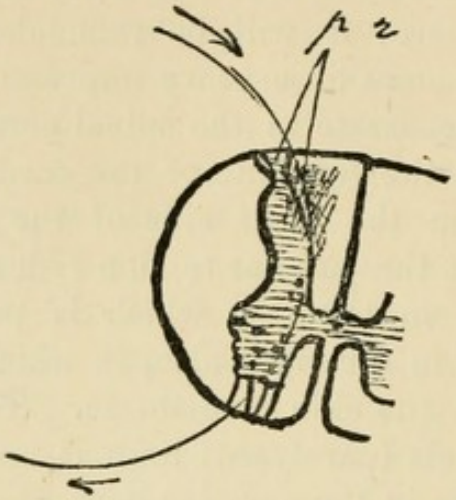


Fig. 4 shows the position of the essential lesion in tabes—sclerosis in the posterior root zone—in that portion of the posterior column adjacent to the posterior horn, which is largely composed of posterior (sensory) root-fibres making their way vertically to the grey matter above. Limitation of the lesion to this spot explains at once the almost constant connection of lost reflex with the occurrence of lightning pains, whilst the integrity of the anterior horn and anterior root accounts for the fact that the quadriceps muscle is neither diminished in bulk nor materially weakened, as well as that it responds perfectly to faradaic currents, and also to blows upon its structure. In my earliest published reference to the subject of tendon-reflex, I remarked of a case of tabes:* "The muscle is here, indeed, more than normally ready to contract upon direct local stimulation." Greatly-extended experience since then has shown me that this condition of exceptional irritability to percussion is very common, perhaps indeed more frequent than not in tabes. Erb has since recorded a similar observation.† He

* The 'Lancet,' July and August, 1878.

† "Zur Pathologie der Tabes dorsalis," 'Deutsch. Arch. f. klin. Medicin,' Marz, 1879.

writes: "In striking contrast to the loss of tendon-reflex, the mechanical irritability of the quadriceps is always completely preserved, and is frequently even very pronounced and lively." I showed you the other day that in an elderly man with typical tabes, in whom there remained not a vestige of tendon-reflex, the irritability to direct percussion of the vastus internus far exceeded that exhibited in the ward attendant, a young and healthy man with a powerful muscular system. An explanation of this increased excitability is probably to be found, it seems to me, in the irritative lesion of the posterior root-fibres, the cause of the lightning pains. The symptom must be classed indeed, I submit, in the same category with the tendency to cramp in sciatica, and the clonic spasm of facial muscles which is so apt to occur in severe trigeminal neuralgia.

It occurred to me a few days ago to examine in reference to this subject a patient whose supra- and infra-orbital nerves have recently been stretched for severe neuralgia of the two upper divisions of the trigeminus on the right side. The patient was for a time greatly relieved, but is subject to occasional paroxysms of pain, and there yet remains a considerable amount of clonic spasm of the muscles on that side of the face. There is manifestly an abnormally irritable condition of the muscular tissue. This irritability, however, does not extend to the tendons. Repeated testing by percussion showed that the contraction of the zygomaticus major of the *left* (sound) side of the face, which followed a blow upon its tendinous origin on the malar bone, was distinctly much more energetic than that of the corresponding muscle on the *right* (affected) side. My reason for selecting this case for observation on this point was as follows:—As is well known, the sensory supply to the face is derived from the trigeminus, the motor influence from the portio dura. Now, the sensory portion of the fifth is acknowledged to represent the posterior root of a spinal nerve. There had therefore been in the operation

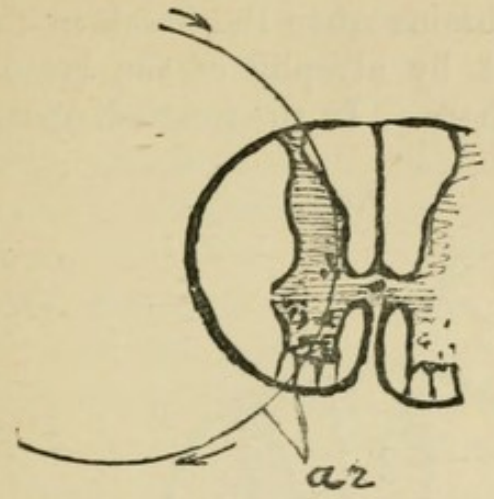
of stretching a kind of vivisection (with a therapeutical, not a physiological object, as it happened) performed upon what is to all intents and purposes the posterior root of the nerve supply to the muscles of the face. In no other part of the body could the operation have been so limited to this effect; in the spinal nerves the posterior and anterior roots are united in one trunk. Now, supposing that aponeurotic nerve-fibres take their course to the nerve-centre in the posterior root, as Tschirjew has stated, I ought to find, it appeared to me, the response to stimulation of fibres on the side on which the "posterior root" had been stretched, and therefore presumably injured, less active than on the sound side. I had some difficulty in selecting a muscle upon which the experiment could be tried, but the zygomaticus major showed it very well, although on a necessarily small scale. It is of no slight interest to observe that although the muscular tissue itself was generally over-irritable (like the muscle in tabes) the response to a tap on the tendon was distinctly less vigorous than that on the sound side. I do not wish, however, to draw positive conclusions from a single observation of this kind.* It would be well to repeat the examination in a patient affected with a destructive lesion confined to the fifth nerve, or in whom the infra-orbital trunk had been actually divided—not simply stretched—for neuralgia.

Let us now suppose the case of the patellar tendon-reflex being absent in a patient who is either entirely confined to his bed or only able to walk with some difficulty. The quadriceps extensor is found more or less wasted, we will say, its response to induced currents being either below the normal standard of excitability or lost altogether. In the latter case there will be more or less complete inability to contract the muscle by voluntary effort. Such a condition necessarily indicates

* I have since had the opportunity of repeating this observation in another case of nerve stretching of the superior maxillary nerve, and with a similar result.

that the muscle is cut off to a great extent, by some means or other, from the trophic influence of the spinal cord. The seat of this influence is known to be in the large ganglion cells which lie in the anterior horn. Now, it will be seen, if we look at the diagram (Fig. 5), that the

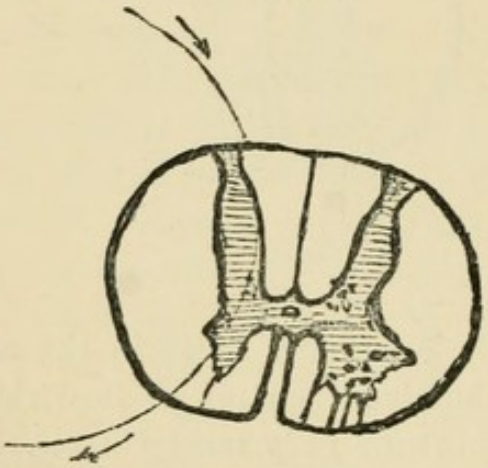
FIG. 5.



interruption in this case may exist in the anterior root outside the cord, the short portion of it within the cord, or in the anterior horn of grey matter itself. As regards the second of these possibilities, nothing is known for certain that will enable us to localise lesion in that particular portion of the anterior root. For the rest, absence or marked decrease of reflex, along with muscular wasting and loss of faradaic excitability, may be caused by neuritis of a mixed spinal nerve, or it may result from spinal meningitis, involving the anterior roots either alone or in conjunction with the posterior roots. The precise cause for the interruption in such a case can only be determined by the consideration of collateral circumstances, into which I need not enter at length. It may be remarked, however, that if the symptoms described are accompanied by pain and tenderness in the nerve trunk, and some cutaneous insensibility, this sensory disturbance makes it evident that the cause of interruption is in the mixed nerve (in which the motor and sensory roots are

combined), and not in the anterior (motor) root alone. I have recorded cases of neuritis in which marked decrease of tendon-reflex occurred in these circumstances.* All that is sure from percussion of the tendon and examination of the muscle is that a break exists somewhere on the *motor* side of the nervous arc. Nor may there be anything in the condition of the muscle in such a case to distinguish it from that which obtains when the break in the nervous arc is brought about by atrophy of the large ganglion cells in the anterior horn. In the rough diagram (Fig. 6)

FIG. 6.



it is seen that the line indicating the nervous arc passes through an anterior horn, which is shrunk and atrophied, and in which there is but little trace of ganglion cells to be observed, in striking contrast to the number and size of those in the anterior horn of the other side. In this shrunken anterior horn, then, the "fault" occurs which causes absence of tendon-reflex. Now, atrophy of the ganglion cells of the anterior horn occurs in various conditions. It often happens as part of a diffuse myelitis, involving not only the anterior horn, but more or less completely the whole transverse section of the cord. In the acute atrophic paralysis of infants and adults (polio-myelitis anterior acuta, commonly called infantile paralysis and acute spinal paralysis of adults) there is acute myelitis,

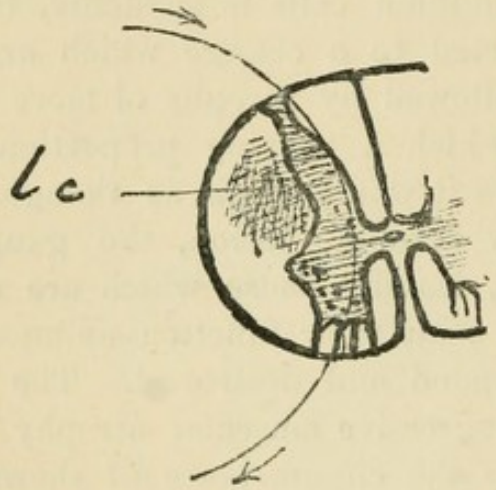
* The 'Lancet,' April, 1879.

which is practically confined to the anterior horn of grey matter. In this disease the loss of faradaic excitability of the muscles is very constant and marked. In the chronic malady, however, in which the same structure is affected (progressive muscular atrophy, protopathic amyotrophy—Charcot), it is somewhat remarkable that, as Duchenne has shown, no decrease of faradaic excitability is to be noticed in the muscle. (This is no doubt the rule, but I have seen exceptions to it). In the former case apparently a mass of ganglion cells is suddenly, or at least very rapidly, subjected to a change which arrests their function, and is followed by atrophy of more or less of their number, of which a varying proportion recovers. In the latter disease it would seem as though, in consequence of a slowly creeping lesion, the ganglion cells atrophied gradually, leaving those which are spared unaltered, or at least with their function unimpaired, until they in turn are invaded and destroyed. The relation of tendon-reflex to progressive muscular atrophy is a good deal complicated by the circumstance of there being at least two classes of this affection—the protopathic amyotrophy and the deuteropathic amyotrophy of Charcot. In typical cases the former is a very slowly progressive muscular atrophy, the gradual loss of power of the patient being in proportion to the extent to which the anterior horns of the cord have been invaded and the muscular system correspondingly atrophied. The latter begins with loss of power (paralysis) independent of muscular atrophy, and is succeeded by a rapid muscular wasting of some or all of the muscles previously paralysed. It is this last form which tends to extend into the medulla oblongata and to occasion death by destruction of the ganglion cells essential to life, which form the continuation upwards of those of the anterior horns of the spinal cord. The duration of this form is much shorter than that of the other. In the first of these—the protopathic form, the tendon-reflex may exist, to some extent at least, for a certain time, to be lost, however, when the atrophy has

advanced, so that it is impossible to say that the behaviour of the tendon-reflex is at all characteristic. The break which causes its absence in such a case is in the anterior horn, as coarsely pictured in Fig. 6.

The condition of the tendon-reflex in the deuteropathic form is extremely interesting. Before considering it, however, I should like to draw attention to Fig. 7. In

Fig. 7.



this diagram there is a rude representation of sclerosis of a lateral column of the spinal cord. In a previous diagram (Fig. 2) the position of the sclerosis of the posterior root-zone was indicated. It was shown that if the afferent fibres from the tendon go, as we believe they do, with the sensory roots, they pass through the outer part of the posterior column, and consequently are there exposed to a lesion which we know produces atrophic changes in them. The nervous arc is therefore "cut" at this point, and that absence of tendon-reflex results which is so conspicuous a feature of tabes dorsalis. The position of sclerosis of the *lateral* column, which we are now considering, is, it will be observed, altogether outside the nervous arc by which we suppose the stimulus from the tendon to the muscle to be transmitted. We should expect, therefore, to find that in sclerosis of a lateral column the tendon-reflex would not be absent. And that

is the case. The tendon-reflex is never lost as a result of uncomplicated sclerosis of a lateral column.

So far from being lost, the tendon-reflex is indeed increased, frequently to a very surprising extent. Ankle clonus (also knee clonus—Erb, Gowers, Waller) may be, in these circumstances, produced not only (in the case of ankle clonus) by sudden dorsal flexion of the foot, but by the patient's own movements, as well as by light blows on various structures (bones, muscles, tendons), and sometimes even by the slight vibration produced by an accidental jar of his bedstead. This excitability to various kinds of stimulation is, however, by no means constant. One sees in certain cases of lateral sclerosis something like the converse of what I have described as occurring in tabes. There is excessive tendon-reflex, knee clonus, as well as ankle clonus, yet the muscles concerned will not respond to a blow, and need a distinctly stronger faradaic current than in health to cause their contraction. This exaggeration of tendon-reflex observed sometimes in cases in which the muscle responds in lively fashion to a blow, and at other times in cases in which the muscle fails to contract to a blow, suggests to my mind very strongly that it is not the condition of the *muscle* which is the cause of the phenomenon.

Here, again, is another example well worth studying in reference to this point. This man, who has all the symptoms of tabes dorsalis except that his ataxy is very slightly pronounced indeed (he finds only a little difficulty in going downstairs), has the patellar tendon-reflex preserved in the right leg, whilst, as you see, it is completely absent in the left. A blow, however, with a percussion hammer causes an equal and exactly similar contraction in the vastus internus muscle of each thigh. Now, we will apply electrical currents, and you see that an equal strength of induced current effects contraction of the muscular fibres in each limb. So also if we place a rheophore on the motor point of this muscle (*i.e.* in close relation with the branch of nerve going to it) and make

this part of a voltaic circuit, we find the normal relation to closure of the circuit with the negative pole, according to Brenner's formula, in either limb. Is it probable, that the absence or presence of knee-phenomenon can depend simply upon the condition of muscular tonicity when we find, as we do here, its absence in one limb and its presence in the other, associated with a state of muscular fibre which all our tests—voluntary action, direct percussion, electrical reaction—show to be in a similar and normal state in each limb?

The question of prognosis is so mixed up with that of diagnosis in regard to the occurrence of ankle clonus that the two may be considered together. The facts appear to show that in all cases the occurrence of increased tendon-reflex in anything like a marked form signifies that from some cause the inhibitory influence of the higher centres is no longer being normally excited. It means apparently that something has come in the way (at some point or other) of the natural propagation of nervous impulses from above. It is generally accepted that impulses from the highest centres (so-called volitional impulses) travel to the ganglion cells of the anterior horns by the antero-lateral columns of the cord (pyramidal tracts). The fact that by what is commonly called "an effort of the will" we can control to a certain varying extent the reflex actions which are subserved in different parts of the cord, would seem to show that in all probability the restraining influence which is continually and unconsciously exerted over certain spinal reflexes in a state of health is conveyed by this same channel.* Now, it is when these (antero-lateral) columns are most extensively invaded by sclerosis, that is to say, when the resistance to the propaga-

* Dr Hughlings Jackson believes that "the rigidity in hemiplegia results because by atrophy of the fibres of the lateral column of the cord "cerebral influence" is taken off from the anterior horns, and thus the "cerebellar influence" upon them is no longer antagonised. On this principle he would explain the increased tendon-reflex in cases of hemiplegia; *mutatis mutandis* also, as regards lesions taking off the influence of the cerebellum."

tion downwards of nervous impulses from above is greatest, that we obtain the most marked ankle clonus. When there is a history pointing to myelitis at some portion of the cord, followed by a more or less pronounced rigidity of the limbs, with excess of tendon-reflex which has existed for some considerable time, say upwards of a year or two, we shall hardly be wrong in assuming that a change has taken place in the lateral columns which is likely to be permanent. And so also in reference to the like symptoms usually consequent upon an attack of hemiplegia. An excess of reflex, combined with more or less rigidity of the limbs, if it has lasted for very many months, may be considered as indicating a structural change in the lateral column which is unalterable. But this is by no means the case when the symptoms have not been of such long standing. There is at the present time in hospital a young woman who came in last June on account of paraplegia of ten months' duration, referable, as it seems, to a subacute myelitis in the dorsal region of the cord. On admission there was such a spastic condition of the legs that they contracted when handled, or upon any attempt being made to flex the joints, which were stiff. There was great excess of patellar tendon-reflex, and ankle clonus was readily produced in a marked degree iu each foot. The patient has gradually improved. She can stand and walk with very little help. There is no longer any marked spastic rigidity, and ankle clonus cannot now be obtained in either foot.

In hysteria the tendon-reflex is, so far as I have yet seen, usually increased, and in several cases I have witnessed the occurrence of ankle clonus, the possibility of producing which has entirely, and in one case suddenly, ceased on the patient's recovery. I submit that this fact is a very important one in reference to the causation of the phenomenon. There would seem to be but little doubt that in hysterical paraplegia the cerebral influence, which is propagated downwards in health by the antero-lateral columns of the cord, is more or less in abeyance.

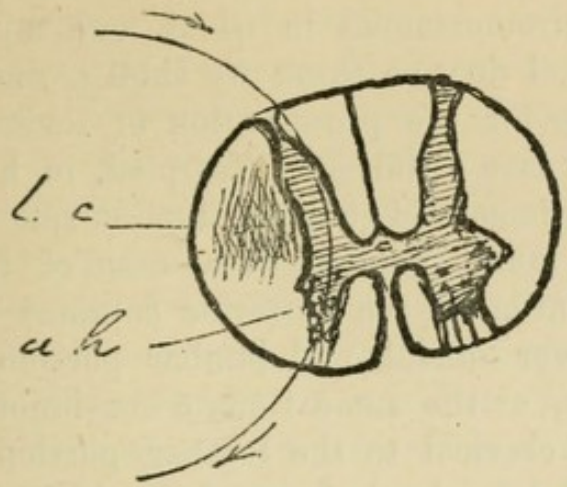
It may be suggested that the reason why an hysterical girl does not walk is because the nervous impulses from above, which ordinarily act upon the various spinal centres for progression, do not arrive there. The absence of impulses downwards through the lateral columns seems to result in an effect upon the tendon-reflex, equivalent to that occasioned by the resistance to the propagation of impulses through the columns, which is caused by the structural change occurring in sclerosis.

It is necessary to remark, then, that the existence of ankle clonus cannot be considered conclusive evidence of a structural lesion of the spinal cord. It may be, to use a term which is generally understood, although scarcely capable of exact definition, "functional." I have never seen, however, in a case of hysteria an excessive degree of ankle clonus. The automatic flexion and extension movements of the foot will go on for perhaps a dozen or more times, becoming gradually weaker and ceasing spontaneously.

The behaviour of the tendon-reflex in deuteropathic amyotrophy (lateral amyotrophic sclerosis, spinal paralysis with rigidity and muscular atrophy) is important. Even when the disease is tolerably far advanced, and a good deal of atrophy of the muscles has taken place, it will be often found that the tendon-reflex not only remains, but may be in some excess. I have a patient now under observation who presents a typical example of the disease. He cannot stand without help, and the muscles of his thighs are not only much wasted, but their faradaic excitability is diminished, and they hang flabbily from his bones. Not only is the patellar tendon-reflex, however, in excess, but there is some ankle clonus spontaneously occurring when he plants his foot in certain positions. I would suggest the following as an explanation of such cases as these (Fig. 8). So long as a certain number of ganglion-cells remain intact in the anterior horn the nervous arc (for tendon-reflex) is complete. Now, if the lateral columns were sound, the inhibitory influence from above trans-

mitted by them would be potent enough to hold the reflex, in its necessarily diminished strength, in complete restraint

FIG. 8.



(as occurs, for example, in infantile paralysis), and no response would therefore follow a blow on the tendon. But the lateral column is diseased, the check is consequently removed, and the reflex manifests itself often with great energy. As a matter of course, however, it is not so strong as in that form of natural sclerosis which is unattended by muscular atrophy.

In an earlier part of this lecture I have remarked that the response of the quadriceps to a blow on the patellar tendon "*almost always* signifies that the nervous arc is not seriously interrupted in that part of the spinal cord which gives origin to the lumbar plexus." In such a case as we are now considering (lateral sclerosis with muscular atrophy) it is evident that now and then the tendon-reflex may be apparently normal, and yet the spinal cord at the part which is concerned be affected with atrophy of some of the ganglion-cells in the anterior horn. The defect in the reflex mechanism is, as it were, just balanced by the defect in the mechanism which restrains reflex. It is necessary to bear this possibility in mind, in order to avoid an error, which, however, a little inquiry into the collateral circumstances will easily prevent.

Here it will be convenient for me to say a few words

on those exceptional cases of tabes dorsalis in which the tendon-reflex is not only retained, but may be exaggerated. Bearing in mind what I have just said in regard to the effect of lateral sclerosis in causing the retention of tendon-reflex in circumstances in which one might expect it to be lost, I do not think we shall see any difficulty in accounting for the preservation of tendon-reflex now and then in a case of tabes. Westphal, in his paper published in 1875, remarked that the tendon-reflex would exceptionally be found present in a case of tabes in which the degeneration of the posterior columns did not extend to the lower dorsal and lumbar portion of the cord, and in which, at the same time, a continuous degeneration from the cervical to the lumbar portion of the cord existed in the *lateral* columns. I should be inclined to go further even than this, and suggest (in view of what we have just seen may happen in lateral sclerosis with muscular atrophy) that there may be degeneration of the posterior columns to a certain extent in the lumbar portion of the cord, and yet the tendon-reflex be retained if, as sometimes happens in tabes, there be sclerosis of the lateral columns. The removal of the check to reflex action thus occasioned permits of the action taking place, even though the mechanism for its production be somewhat impaired.*

In the present unsettled state of the question as to the physiological nature of the so-called "tendon-reflex" I have thought it well to reproduce the clinical observations which tend to support the view that it is of the character of a reflex phenomenon. It is right, however, to say that the numerous measurements which have been made of the period of latency which intervenes between the blow on

* In subsequent Lectures frequent references are made to the behaviour of the tendon-reflex in other conditions. These are specified in the Index.

the tendon and resulting muscular contraction appear to throw very great doubt upon the phenomenon being appropriately described as "reflex" in the ordinary acceptation of the term, unless (as I have before suggested and still think worthy of consideration) we suppose it possible that the unusually vigorous mode of excitation may materially diminish the amount of time ordinarily thought to be required for the process of reflex action. In any case the value of the clinical facts is not affected, whatever be the source to which the phenomenon may owe its origin, and the muscular contraction, if not directly due to reflex action, is at any rate dependent upon conditions which demand the integrity of a reflex spinal arc. In order that the evidence on the physiological side of the question may be fairly presented, I append the following references to observations by various investigators, for which I am indebted to M. de Watteville :—

"Erb (1874)* who first described it took it to be a simple sensori-reflex phenomenon. Westphal,† on the other hand, from the first spoke of it as due to the direct excitation of the muscular fibres, through the sudden stretching which they undergo when the tendon is struck.

Tschirjew (1879)‡ showed that section of the posterior as well as of the anterior lumbar roots abolished the knee-jerk, and gave this fact as a proof of its reflex nature. He further showed that the sentient fibres of muscle are distributed, not to the muscular fibres themselves, but to the aponeurotic tissues, and that they alone form part of the reflex arc. Burckhardt (1877)§ had previously measured the time which elapses between the instant of stimulation and that of contraction, and found it to be ·039 of a second. Tschirjew repeated these measurements by more accurate methods, and stated it to be shorter, viz. ·033 sec. Waller‖ (1880) stated the average latent time

* "Ueber Sehnen Reflexe bei Gesunden und bei Rückenmarks-kranken," 'Archiv f. Psych.,' v, p. 792, 1875.

† "Ueber einige Bewegungs Erscheinungen an den gelähnten Gliedern," Ibid., p. 803.

‡ "Sur la terminaison des nerfs des muscles striés," 'Arch. de Phys.," 1879, vi.

§ 'Ueber Sehnen Reflexe,' Bern, 1877.

‖ "On Muscular Spasms known as 'Tendon-reflex,'" 'Brain,' Part X.

in every case to be ·04 sec. Brissaud* (1880) thinks that all conditions which increase the knee-jerk also shorten its latency (·035 sec. in spastic paraplegia to ·05 sec. in the healthy state). More recently Eulenburg† (1881) has published his results, which give a shorter latent period than hitherto obtained (viz. ·016 to ·032 sec., for healthy individuals). Now, it is obvious that on the accepted views about the rate of travelling of nervous impulses and of conveyance of these impulses across the grey matter of the cord, a period of 3 or 4 hundredths of a second would not be sufficient for a sensory stimulus to travel from the ankle or knee to the cord, and there to be converted into a motor stimulus going down to the triceps or gastrocnemius. Gowers‡ believes that the phenomenon is indirectly reflex only, that is to say, that the contraction following the tap is due to the direct stimulation of muscular fibres, but that this excitability depends upon a reflex action from the cord originating in the tension of the fibres. Waller§ also urges on several grounds that the phenomenon is not reflex, though it depends evidently upon the reflex tonicity from the cord; and in a joint series of experiments with Prevost (1881) shows that the objection raised by the latter against his view from the alleged occurrence of "cross-reflexes" was groundless. Westphal‖ (1881) has restated his view of the peripheral origin of the phenomenon, holding that it is not a simple reflex, but a complex manifestation intimately related to the muscular tonus, which is possibly reflex. Eulenburg¶ concurs with him on the evidence of the measurements just stated. On the other hand, it is argued that though the lost time of the knee- or ankle-jerk is very short for a reflex act, yet it is too long for that of a direct muscular excitation; and that the rate of travelling of nerve influx may be greater than what our measurements lead us to believe. Curare poisoning (Schultze and Fürbinger**), anæmia of the cord (Prevost††), and other conditions abolish the knee-jerk.

I have been able, quite recently, however, to observe a case in which

* 'Recherches sur la contracture, &c.' Paris, 1880.

† 'Zeitsch. f. Klin. Med., vol. iv, p. 179.

‡ 'Diagnosis of Diseases of the Spinal Cord,' 2nd edition, 1881.

§ Loc. cit.

‖ "Ueber das Verschwinden des Kniephänonens," 'Berl. Klin. Woch.,' 1881.

¶ "Ueber die Latenzdauer der Sehnen Phänomene, &c.," 'Neurolog. Centrbl.,' 1882, No. 1.

** 'Centralbl. d. Med. Wiss.,' 1875.

†† "Contribution à l'étude de Reflexes Tendineux," 'Rev. Méd. Suisse Romande,' 1881, 1—3.

both skin and "tendon" reflexes were well marked, and found that the lost time of the plantar reflex was in the gastrocnemius and extensors of the leg about three times as long as the patellar tendon-jerk latency. An interesting tracing shows two contractions following a blow on the ligament; one with the usual "tendon-reflex" latency of ·03 sec., the second with the plantar-reflex latency (minus the time required for the stimulus to travel from the foot to the knee), viz., ·08 sec. I feel, therefore, compelled to agree with those who hold the view that the phenomenon is not a spinal reflex, though dependent upon the integrity of the reflex arc."

LECTURE III

INFANTILE PARALYSIS, AND ACUTE ANTÊRIOR POLIO-MYELITIS IN ADULTS

I PROPOSE in my lecture to-day to bring under your notice some cases of acute atrophic paralysis occurring in children. Up to a comparatively recent period the disease, of which you will see some examples, was called "infantile paralysis," or "essential paralysis of children," in consequence of its being supposed to be peculiar to that time of life. Later observations have, however, shown that it is by no means infrequent in adults, although probably occurring in much less proportion in them than in children, and especially in infants under two years of age.

Pathological investigations have shown that the disease is an acute form of systematic myelitis affecting the anterior portion of the grey matter of the spinal cord, and hence the name *polio-myelitis anterior acuta* (πολιός, grey; μυελός, marrow; ιτις, inflammation). The term systematic myelitis has been applied by M. Vulpian to inflammation which is restricted to certain *systems* of fibres or cells in place of being diffused over various parts of the section of the cord indefinitely. It is especially to the work of M. Charcot that we owe our knowledge of the existence of these systems of fibres. The accompanying scheme, which is very slightly modified from one given by Dr. Grasset, of Montpellier, in his admirable 'Maladies du Système Nerveux,' will be found useful in showing the position of acute anterior polio-myelitis among the other forms of systematic myelitis. It is indicated in italics.

- **Of the white columns.**
  - Of the posterior columns.
    - 1. Of the outer part of the posterior columns (posterior root zones).
      - *a.* Primary. (Progressive locomotor ataxy.)
      - *b.* Secondary.
    - 2. Of the inner part of the posterior columns (columns of Gall).
      - *a.* Primary. (Sclerosis of the columns of Gall.)
      - *b.* Secondary to a lesion of the cord. (Ascending secondary sclerosis.)
  - Of the lateral columns and columns of Turck.
    - 1. Primary (lateral symmetrical sclerosis).
      - *a.* Without muscular atrophy. (Spastic spinal paralysis.)
      - *b.* With muscular atrophy. (Lateral amyotrophic sclerosis.)
    - 2. Secondary to a lesion of the brain or cord (descending secondary sclerosis).
- ***Of the grey matter.***
  - *Of the anterior horn.*
    - *Primary.*
      - 1. *Acute.* (*Acute anterior poliomyelitis.*)
        - *a. In the infant.* (*Infantile paralysis*).
        - *b. In the adult.* (*Acute atrophic paralysis of adults.*)
      - 2. Chronic. (Progressive muscular atrophy.)
    - Secondary to another form of myelitis.
  - Of the nuclei in the medulla oblongata.
    - Primary (labio-glosso-laryngeal paralysis.
      - 1. Simple.
      - 2. With muscular atrophy.
    - Secondary to other forms of myelitis.

It should be remembered that, although a scheme of this kind indicates in a general and convenient fashion the names applied to disease attacking various systems of fibres or cells in the cord, it is not strictly accurate in detail. The pathological changes are not often confined absolutely to the localities indicated, although in these they will certainly be most strongly marked.

Before presenting to you some examples of acute atrophic paralysis which are at present under my care, I will give you a sketch of the symptoms of the disease.

As it occurs in infancy (and it is peculiarly apt to attack children under two years of age) the essence of the disease is this, that after a varying amount of febrile movement, one or more, or all, of the limbs are observed to be paralysed. You cannot always obtain a history of fever in these cases, but experience seems to show that absence of a certain amount of febrile disturbance is at all events exceptional. In a case lately in the hospital, and now attending as an out-patient, Mr Broster, our then resident medical officer, made the following note of the mode of onset:—"A strong healthy child when six months old used to pull herself up from the floor and walk by aid of the furniture. When she was fifteen months old her mother, who had to go out to work, left her in the morning quite well. When she returned at night the child seemed feverish and fretful. She was poorly for several days, and her mother kept her in bed. After this she got up again, but now she did not attempt to walk or climb upon chairs as she had previously done, and her mother noticed that the right leg felt quite soft and seemed quite useless." That seems to me an excellent description of a very common mode of onset. In other cases the febrile movement is much more marked, and excites suspicion of the onset of some zymotic disorder. In others, again, the first symptom to attract attention is the loss of power. In a case which I saw a few days since, a child, eleven years of age, had romped with other children in the afternoon and sat up to her parents' dessert. She went to bed

and only woke once. Her bed companion noticed that she got out of bed and appeared to have some difficulty in getting in again. She was not feverish. Next morning she could not stand nor raise her body in bed.

There is a curious difference in the duration of the febrile disturbance. Duchenne fils found, in examining records of seventy cases, periods of fever lasting from one hour to fifteen days. The duration and intensity of the fever were thought to be, as a rule, less long the younger the child. The paralysis most often occurs with great rapidity, but occasionally, although rarely, there is a gradual loss of power dating from the initial fever. The disease sometimes occurs at the close of a specific fever—as typhoid. I have seen it follow measles.

Out of sixty-two cases, Duchenne fils noted five in which the paralysis was general; nine, paraplegia; one, hemiplegia; two, crossed paralysis (the right upper and left lower limb); twenty-five in which the right lower limb was affected; seven of the left lower limb; ten of the right or left upper limb; two, lateral paralysis of the upper limb; and one case in which the muscles of the trunk and abdomen were paralysed. My own observation would lead me to think that the muscles of the trunk are not unfrequently involved.

I saw a male child, 2¼ years old, lying in bed with the right cheek on the pillow, whence he could not stir it. Nor could he move a muscle of the trunk, arms or legs. His respirations were 52, his pulse 120. He was conscious, but when spoken to answered with a wailing voice. This boy was a very fine grown child, and I think that it is more common than not for the disease to attack fine grown hearty children. He had never had any illness. This was how his attack began.

On July 21st he dined well, but afterwards complained of headache. This did not prevent his playing about.

Next day, 22nd, he would not get up and complained of pain in his forehead.

On the 23rd he was much convulsed, especially in the

face and hands, opened and shut his eyes very much, and muttered a good deal.

24th.—He was crying and asking for his mother, whom he did not, however, recognise.

There was never any vomiting or squinting.

On the 26th his parents observed that he was powerless throughout his body and limbs, in which state I found him on the 27th.

Dr B. Lees exhibited not long since, at the Clinical Society, two cases in which the serrati magni had remained paralysed after an attack of infantile paralysis. Occasionally, though I think very exceptionally, there is paralysis of muscles supplied by the medulla oblongata. I shall be able to show you in the next lecture an example of the kind.

In addition to the fever there may have been convulsion, coma, some transient loss of cutaneous sensibility, and a temporary trouble with the bladder or rectum, but to a great extent (though not exclusively) the brunt of the disease falls suddenly, or at least very rapidly, upon the motor power of the limb or limbs. Now and then there are pains in the limbs, with tenderness to the touch. There is no tendency to formation of bed-sores in typical cases. After the first day or so, any change which takes place in the power of movement is a change for the better. The limbs do not become *more* paralysed. On the contrary, after a few weeks, or sometimes days, there is a gradual clearing off of the difficulty as regards some of the limbs, one or more perhaps remaining unimproved. Or the paralysis may remain limited to a few muscles in one limb. There are rarely or never relapses, so that the return of power in a limb may be regarded with some confidence as of permanent, and not temporary, character.

Let me now draw your attention to the most remarkable feature in this form of paralysis. Many of the muscles paralysed lose their faradaic excitability entirely within a week, and rapidly waste. But although they fail to respond to the strongest induced currents they react to

slow interruptions of the constant current (reaction of degeneration). The *nerves* to the muscles, on the other hand, lose their excitability to both forms of electrical excitation. Some of the muscles, again, whose faradaic excitability has been lowered, but not lost, are not long in regaining the power of contraction to voluntary impulses. This rapid loss of faradaic excitability is peculiar to the disease. In no other form of more or less generalised paralysis (unaccompanied by marked sensory disturbance) do you find within a few days that the muscles fail to contract to induced currents.

The earliest opportunity which Duchenne had of examining into the electrical condition of the muscles in a case of this kind was the third day. In that instance the right arm was paralysed. It was not until the fifth day that the electrical excitability of the deltoid was notably enfeebled. On the seventh day, however, it was completely abolished. His experience was, that if the muscles have retained, after the seventh or eighth day, some amount of contractility to the induced current they always recover their motility, and the more rapidly the less the excitability has been enfeebled. F. Müller has found the excitability of the nerves lost on the fourth day, and that of the muscles on the fifth and sixth days.

The muscles paralysed are flaccid. The reflex from the sole of the foot is usually absent in cases of paralysis of the lower limb, and so also is the patellar tendon-reflex where the muscles on the front of the thigh are involved.

The second period of the disease begins a few weeks, or may be delayed for several months, after the attack. It is called the period of regression, for in it there is a gradual return of power in more or less of the muscles, and their excitability to faradaism again appears. The amount of recovery differs extraordinarily in cases, so that it is impossible to say more than this, that when several limbs are attacked at first it is much more common than not for some of them to recover but rare for all to do so. The

mode in which recovery takes place is also very uncertain, in some muscles proceeding rapidly, in others very slowly. It occasionally happens that muscles which have remained for some time irresponsive to electrical stimuli (either induced or slowly interrupted voltaic currents) will suddenly show reaction to the latter, and increasing amendment follow. More often than not, perhaps, where the paralysis is extensively distributed, the improvement in the upper extremities precedes that which takes place in the lower limbs. With return of voluntary power and faradaic excitability, the muscles which have wasted gradually regain their volume. Those which remain paralysed, on the contrary, take no part in this amendment, but are more and more marked out by contrast with the others. I have found great differences in regard to the excitability by electrical currents in the muscles. In some cases muscles have refused to show response to either form of current after a few months, in others, even after several years, though the limb continued helpless, the application of a slowly interrupted voltaic current would bring about a distinct though feeble contraction. Voluntary power usually returns long before faradaic excitability.

In the sequel of the disease atrophic changes are marked in the muscles, which may be so wasted as to leave the limb in a skeleton-like state, or fatty substitution may mask the real loss of muscular substance and give a false air of plumpness to the limb. The development of the osseous system is more or less arrested, so that a bone may, in course of years, be some inches shorter, and considerably thinner than its fellow. There is diminution in the calibre of blood-vessels leading to comparative coldness and blueness of the limb, which also often shows unusual liability to chilblains. And most important of all, perhaps, the tonicity of such muscles as remain sound or comparatively little injured causes them gradually to overpower those whose function is destroyed, giving rise to deformities which often tend to persist and increase in spite of all efforts to reduce them. In this manner are pro-

duced the greatest number of those forms of club-foot which are not congenital. The relaxed state of the ligaments of the joints in these cases is a noteworthy and characteristic sign of the disease, the shrunk and useless member hanging like a flail. At a comparatively early period of the disease there will often be found a tendency to deformity, though at that time reduction by the hand (which later becomes impossible) is not difficult.

Duchenne says* that, by electric exploration, he has always found a greater quantity of healthy fibres in the contracted or retracted muscles than in the others. And this is what might be expected. In the commencement all the muscles of the limb affected are paralysed; later on certain muscles recover their contractility after having been more or less atrophied, and these draw the limb in their direction when their contractility appears. Their continued shortening determines, in the end, their retraction, the shortening of certain ligaments, the deformity of limbs and articular surfaces. Other muscles remain paralysed, and very probably undergo textural changes. It is in the antagonists of the retracted muscles that the greatest fatty changes are found, but not exclusively in them.

I show you a boy who was attacked five months ago with this disease, and notes of whose case have been taken by Dr Beevor, Resident Medical Officer.

Ernest R—, æt. 4, the youngest of ten children, of whom two died at birth, was admitted into the hospital on October 6th, 1880, on account of loss of power in the *right leg*. It seemed that three months previously to his admission, the child had a febrile attack, which lasted a few days, and on its recovery from this it was observed that it could do nothing with its right leg. The other limbs appear from the history to have been unaffected. Since then it has slightly improved. When admitted, the child could not stand, and could only crawl upon the ground. It could not extend the knee, or flex the hip-

* 'L'Electrisation Localisée.' Third Edition. Page 412.

joint, or move the ankle. The right leg was rather smaller than the left.

The boy has improved, as you see, since he came here. He can now stand and walk, though imperfectly. The knee-joint is lax, the internal lateral ligament being weak, and perhaps still more the capsular fibres (forming a lateral ligament), which are prolonged downwards from the insertion of the vastus internus to the inner tuberosity of the tibia. The leg, therefore, forms an obtuse angle with the thigh, the apex inwards. When seated he cannot extend or flex the leg, or lift the knee or the foot. Standing he can lift the foot off the ground by the iliaco-psoas.

Let me call your attention to certain points in regard to the paralysed limb. In the first place there is no loss of cutaneous sensibility in it. He feels touches and pinches just as well with this leg as with the other, and this has been the case throughout. When he takes a warm bath the water feels just as warm to one leg as the other. He is too young for the muscular sensibility to be tested.

Next, the limb is distinctly smaller than the corresponding one, and Dr Beevor, who has at my request examined into this point, informs me that the diminution in size can be traced especially to the quadriceps (especially the vastus internus), the anterior tibial group, and the peronei, as well as, though to a less extent, to the calf muscles. You will note here, therefore, and this is a point to be remembered, that we have not to do with an affection of muscles all of which are in the district of one particular nerve, or even of a single plexus of nerves. The quadriceps femoris, you will remember, is supplied (along with many of the other muscles in the front of the thigh, and the iliacus) by the anterior crural nerve, which arises mainly from the third and fourth lumbar nerves. The anterior tibial muscles and peronei, on the other hand, derive their nerve supply from the great sciatic, which arises from the lumbo-sacral cord and the four upper sacral nerves. But so also do the muscles of the calf,

which are not so much affected as those in front of the leg. A measurement made on November 9th gave the following:—Right calf 7½ in., the left 8½; right thigh (3 in. above patella) 10, the left 11½ in.

The patellar tendon-reflex, you will observe, is absent on the right side, present on the left. Tickling the sole of the right foot causes a very slight reflex contraction of the muscles. On the left side the contractions are vigorous. There is no loss of cutaneous sensibility. The muscles of the right limb when last examined did not react to faradaism, but contracted to slow interruptions of a constant current from ten cells of a Stöhrer's battery, a weaker current than would cause contractions in health.

If one rheophore from an induced current machine be applied to this child's back and the other placed in succession over the motor points of the muscles of the thigh and leg no contraction is produced by a stronger current than is amply sufficient to cause vivid contractions of the corresponding muscles of the left limb. In order to save the child from pain as much as possible the current is interrupted slowly by pressing the hammer of the machine, instead of allowing this to be done rapidly by the automatic arrangement for the purpose. By degrees a very powerful current can thus be put on, yet no contraction is caused.

If now there be substituted for faradaism the constant current derived from a voltaic battery, one rheophore being placed on some indifferent spot (not on its motor point) (and the other on each muscle in turn), it is found that when the current is gradually strengthened till it comes from ten cells, and this is interrupted by using a commutator, contraction takes place in the paralysed muscle.

I may say that, on October 6th, when the boy was admitted, it required a current coming from twenty cells to produce contractions.

On November 9th, a month later, ten cells were sufficient. Throughout there has been no response at all to any strength of faradaic currents.

This is an example of a very common form of infantile paralysis. Before showing others, I should like to say a few words upon the pathological anatomy of such cases.

I would remind you that motor impulses from the brain travel to the muscles for the most part by the lateral column of the spinal cord opposite to the hemisphere in which they originate. In the anterior horns of grey matter they act upon large ganglionic cells, and it is through the medium of these (and not as a rule directly), that the motor impulses are transmitted to the muscles by the anterior roots of spinal nerves continuous with the cells. The ganglionic cells are also the reflex centres by which afferent impulses conveyed from the periphery by the posterior roots give rise to muscular contractions. There is no reasonable doubt also, that the centres of nutrition for the muscles lie in the cells of the anterior horn of the spinal cord. Destruction of these cells, or such a lesion as without being absolutely destructive suspends their action for a longer or shorter period, betrays itself by paralysis, by such an alteration in the motor nerve going to the muscle as renders it incapable of being stimulated by electric currents, by wasting and degeneration of the muscular structure, and by absence of muscular contraction brought about by reflex action.

The anterior horn of grey matter contains the largest cells that are to be found anywhere in the cord. These are clustered in three groups, of which the most important is usually found towards the outside of the anterior part of the horn. The cells have prolongations which, according to Deiters, receive a coating of myeline and thus become continuous with the nerve fibres of the anterior root. Huguenin describes the anterior root as composed partly of fibres thus connected with the cells of the anterior horn and of others which quit the grey substance and pass into the lateral columns in an upward direction.

It appears to me possible, and I only suggest this as an hypothesis, that the existence of some fibres which run

in the anterior roots and pass into the lateral columns without direct connection with the ganglion cells, may explain the circumstance that a certain amount of voluntary power will often return before the nerve trunk shows excitability to electrical currents, and whilst the muscle still fails to contract to induced currents. The trophic centre for most of the fibres would be the large ganglionic cells, and the fibres would therefore degenerate upon lesion or destruction of these cells. But the fibres to which I have referred above having their trophic centre higher up may be readily supposed to escape all but a temporary obstruction of function.

It is essentially in the anterior horn of grey matter that the pathological change occurs which has been described by Prevost, Vulpian, Lockhart Clarke, Charcot, Joffroy, Roger, Damaschino, amongst others, and last year in admirable detail by Dr Turner, Dr Taylor, and Dr Humphreys at the Pathological Society.

The changes observed by these authorities may be briefly summed up as follows:

Under the microscope, at an early stage, areas of inflammatory softening are found in the grey substance of the anterior horns, especially in the lumbar and cervical enlargements of the cord. In these areas of disintegration the substance is soft, friable, and interspersed with numerous granulation-cells, the blood-vessels overcharged, and there is increase of connective tissue with its nuclei. The large multipolar ganglion cells have in great part disappeared—entire groups having vanished, whilst of those that remain some are degenerated and atrophied, others undamaged. The nerve fibres and axis cylinders within the area of softening have also disappeared. The anterior horn as a whole appears wasted. There is often also some overgrowth of connective tissue with wasting of nerve fibres in the antero-lateral columns.

The anterior roots are diminished in size, atrophied, and under the microscope show signs of degenerative atrophy; those most changed corresponding with those

parts of the anterior horns where the lesion is most pronounced.

Observations made many years after the attack show much firm connective tissue, with thickened blood-vessels in the areas of destruction, together with large numbers of corpora amylacea. The ganglion cells and nerve fibres are more or less extensively destroyed—those remaining being in various stages of degeneration. There may be more or less sclerosis of the antero-lateral columns.

Now, the effect of section of a nerve is to bring about, within a week, structural changes which can be traced throughout its ramifications. And lesion of a ganglionic cell with which the nerve fibre is continuous has a similar effect to section of the nerve. As a result of these changes, not only does the nerve cease to transmit motor impulses, but it is rendered inexcitable by electrical stimuli. When you apply the two rheophores of an induced current machine to the skin covering a muscle, and thereby obtain a contraction of muscular structure, that contraction is brought about by the momentary currents stimulating the intra-muscular nerve, and not by the action of the currents directly on the muscular fibre. So that when, as a result of destruction of ganglion cells, the motor nerve fibres proceeding from them to the muscle undergo a change, the effect necessarily is that you fail to produce contraction of the muscle by applying the induced current. This failure of response then merely shows that there is something wrong with the nerve. If now you apply a voltaic current, you will find that on making and breaking the circuit contractions occur. This is because this form of electrical stimulus acts directly upon the muscular fibre itself. The absence, therefore, of response in a muscle to the induced current is compatible with a considerable degree of integrity of muscular structure. It is probable that muscular structure may retain its electrical contractility for a very long time after it has lost its physiological connection with the trophic centre in the spinal cord.

The principal, as well as the earliest, change which the paralysed muscle undergoes in this disease is one of simple atrophy. Laborde found this as early as fifteen days after the onset.

Portions of muscular tissue have been hooked out by means of a "harpoon" from the living and examined microscopically. It would seem that in the first period of the disease, *i.e.* within the first few months, a great number of fibres are found in a state of *simple* atrophy. They are of very small diameter, but retain the natural striation, and are without traces of fatty granulations. Amongst them are a few presenting different degrees of granulo-fatty degeneration. With the muscular atrophy there is often considerable overgrowth of connective tissue. In the second period fatty substitution is largely added, according to Charcot, to the preceding changes. Masses of granulations and fat globules are substituted in the sheaths of sarcolemma for the primitive fibre which has disappeared. In other parts the adipose cells accumulate outside the sarcolemma in the intervals separating the primitive fasciculi. Hayem describes how in some cases the interstitial tissue is in such excess, that the muscles are converted wholly, or in part, into a hardened fibroid tissue, in which the thin and discoloured fasciculi are distant from one another. The anomalous development of adipose tissue gives a lipomatous consistence and development which may mask the muscular atrophy, and produce a pseudo-hypertrophy.

The patient whom I now show you, and who was attacked with infantile paralysis fourteen weeks ago, illustrates some points of importance connected generally with the diagnosis of the disease.

Daisy A—, æt. 1½, was admitted into the hospital on October 6th, 1880, on account of paralysis of the *right arm*. She had been attending as out-patient since July; notes of her case were taken by Dr Beevor.

The child is said to have had enteric fever when three

weeks old—otherwise it had been fairly healthy. About a year ago, the mother noticed one day that the child seemed sleepy and feverish. Next day when she was moved she "twitched her eyes upwards," and her arms were convulsed; not her legs. Her mother thinks she lost consciousness for three days. During this time she was convulsed, mostly when she was moved. Directly after this her mother observed that she "squinted inwards." This appears to have lasted ten days.

The right arm was not observed to have lost power until ten days after the twitching, when the mother noticed that the arm was always behind the child's back, and could not be brought forwards, and it fell, if lifted, like a dead weight. Neither the left arm nor the legs were affected. For about three days it is said the hands were closed, and any attempt to open them made her scream. She has not been able to move the right elbow or shoulder-joints at all.

The girl is thin and sickly looking. The right arm is smaller than the left, the deltoid and biceps being especially wasted. She can move her fingers and hand, but cannot flex the elbow-joint, though she can extend it. She cannot abduct the right arm from her chest.

When I tap the tendon of the supinator longus of the left (healthy) arm the muscle contracts and causes a sudden flexion of the forearm. (The supinator longus is, as you are aware, in spite of its name, a flexor of the forearm on the arm.) On the right (affected) side a similar manœuvre produces no result. The tendon-reflex is absent, then, at the wrist of the paralysed arm.

In such a case as this, and especially having regard to the unusually severe head symptoms by which it was attended, the suggestion might arise that it was probably a case of hemiplegia of cerebral origin.

Now, if this were a case of cerebral hemiplegia and you applied the test which I have just shown you, one of two results would certainly have followed. Either there would have been no difference between the two arms, or

the reflex on the affected side would have been stronger than that on the sound side.

I will show you a child of about the same age, Florence W—, æt. 16 months, who lost the use of her right arm and leg three months ago. She had been laid up on account of diarrhœa, and it was when she was taken up after this that the paralysis was first observed. You will notice that the right leg, although weak, is not at all wasted, and that the patellar tendon-reflex on that side is somewhat in excess of that of the other side. The limb is not stiff. The upper limb shows some rigidity, and in this the tendon-reflex at the wrist is greatly in excess. The contrast is very striking with what I have just shown you in Daisy A—. There can be no doubt that the case of Florence W— is one of cerebral hemiplegia. Two circumstances, I may add, go to confirm this. We find, on inquiry, that the child's face was at first paralysed, although that has since recovered, and there was also marked cutaneous anæsthesia of the affected limbs for three weeks.

The absence of the tendon-reflex of the wrist has the same significance in the case of Daisy A— as did that of the patellar tendon in the other cases shown. It is evidence of a break in the nervous arc which connects the tendon with its muscle through the mediation of the spinal cord. In order to find out, as far as that is possible, the situation of the break we apply induced currents of electricity to the muscles of the arm. There is no contraction. When, however, we apply a constant current, and slowly interrupt it, we obtain contractions. This is what is called the "reaction of degeneration," a term which must not be taken as necessarily implying the presence of an irreparable lesion. Its presence merely signifies that the muscle is physiologically cut off from the influence of the spinal cord. Now, this influence, as I have said before, can evidently be cut off in two different ways. Either the nutritive centre itself, in the spinal cord, may be diseased, or there may be some solu-

tion of continuity in the spinal nerve passing to the muscles, brought about either by disease or injury.

There is no loss of sensibility in the skin of this child's arm. We may take it, therefore, as certain that the break of continuity, supposing this to be present, does not affect the nerve *trunk*, since this is both motor and sensory in its function. A spinal nerve arises, as we know, by two roots, of which the anterior is concerned in the propagation downwards of motor impulses, the posterior in the conduction of sensory impressions from the periphery to the centre. It is evident, therefore, that the presence of a destructive lesion involving the *motor roots* only of several nerves going to form the brachial plexus might explain the paralysis and wasting of muscles which we see in this case, as well as the absence of tendon-reflex and of reaction to faradaism. But the anterior (or motor root) of a spinal nerve, as I have before remarked, is in intimate association with the large ganglionic cells which are found in the anterior horns of grey matter in the spinal cord. Lesion of this structure, involving atrophy of the cells in question, brings about the same results as a destructive lesion of the anterior roots which are in intimate relation with these ganglionic cells.

As far as regards the particular symptoms which we are considering, we cannot, by the tests applied, say whether the lesion is in the right anterior horn of grey matter, or in certain anterior roots of spinal nerves emanating from the cervical enlargement of the cord. No doubt a spinal meningitis, with resulting effusion, might explain the condition, but we should have to imagine the inflammation to be of so limited and localised a character as to make the explanation an improbable one. It would have to be limited in extent horizontally to one half of the anterior portion of the cord, and vertically to the points of egress of a few of the anterior roots. On the other hand, let me draw your attention to the important fact that, whilst the deltoid (supplied by the circumflex nerve) is most paralysed of all the muscles, the triceps which,

with others, is supplied by the musculo-spiral is one of those least affected. As you see, the elbow-joint can be well extended. Now, it will be remembered that the musculo-spiral and the circumflex are most important divisions of the posterior cord of the brachial plexus, and could hardly fail, therefore, to be equally affected if the lesion were one of anterior nerve roots.

We are driven then, by exclusion, to the certainty that the paralysed and atrophied state of the muscles here seen depends upon lesion of the large motor ganglion cells in the right anterior horn of the cord. Moreover, this brings us to another point of extreme interest in regard to the relation borne by the ganglionic cells to the innervation of the muscles.

I long ago learned from Dr Hughlings Jackson his opinion that the groups of ganglion cells in the anterior horns of the spinal cord represent *movements* not muscles, and Remak has recently published some important observations bearing upon the same point. He has come to the conclusion ('Centralblatt,' 1879, p. 939) that the groups of ganglion cells corresponding to muscles *functionally associated* lie together in the grey substance of the cord, inasmuch as in anterior polio-myelitis and lead palsy those muscles have been found diseased together, which work together synergically, without reference to their innervation by this or that nerve. According to his experience, there are typical combinations of muscles diseased which must correspond to constant and definite territories for movement in the spinal cord. For example, there is an upper arm type, in which of all the muscles the deltoid, biceps, brachialis anticus, and the supinators are alone attacked with degenerative atrophy. One of these muscles will not be attacked severely without the others showing, if it be only trifling, electro-diagnostic signs of degeneration. He remarks upon the escape of the sartorius in affections of the district of the anterior crural nerve and the sparing of the tibialis anticus in affections of the peroneus district.

It was formerly supposed that the hemiplegic form of infantile paralysis never occurred, and that the disease could, in some cases, by this means alone be distinguished from paralysis of cerebral origin. This is, however, not the case. We have here an example of the hemiplegic form :

Arthur P—, æt. 4, was admitted into the hospital on October 27th, 1880, on account of partial paralysis in the *left leg and arm.*

From notes taken by Dr Beevor, it appears that the family history and the previous health of the boy were good. When he was fifteen months old his mother left him one morning quite well, but in the evening found that he had lost power in the left arm and leg. He had no cold upon him at the time. No other limb was affected. For three weeks after this he was quite powerless in the arm and leg. He then gradually improved till he became as he is now. He is a healthy-looking boy ; the face is not affected, and the tongue is protruded straight. There seems to be nothing now wrong with the left arm, which is about equal in size and power of grasp to the right.

The left leg is a little smaller than the right, and he walks and runs rather lame with it. He can flex the hip-joint and extend the knee-joint fairly, but not so well as on the right side.

The patellar tendon-reflex is normal on the right side, absent on the left. When we come to seek an explanation of this absence, we find that the faradaic excitability of the vastus internus is lower in the left than in the right thigh. On the sound side, the rheophore being placed at the motor point, an induced current marked 19 on the scale causes contraction of the muscle. On the left side it needs a current of 17 to produce a like result. (The current scale is graduated from 1 to 30—1 being the strongest power.)

There is no affection of sensibility, nor is the action of the rectum or bladder impaired. The right calf measures 8¼ inches ; the left, 8 inches. The right thigh, 4″ above patella, 10¾ inches ; left, 9½ inches.

The wasting appears to be almost exclusively in the anterior muscles of the thigh, and the feebleness is shown in the power of extending the leg.

The treatment of this case consisted in the administration of cod-liver oil. No electrical treatment was applied.

The amount of recovery in this case is in striking contrast with that observed in the following one, which illustrates one of the most formidable results of the disease that is met with. It is interesting to note that, after so long an interval as thirteen years since the attack, there are still some muscular fibres in one of the paralysed limbs which show a slight reaction to interruption of the constant current, a proof that the contractile element has not entirely disappeared.

Ernest T—, æt. 14, was admitted on January 3rd, 1879, on account of paralysis of all four extremities. It seemed that when he was a year old, and in perfectly good health, symptoms of paralysis were noticed. No cause was known. There had been no fit. The patient had never walked. He is a pale-faced lad who is not able to raise himself from the recumbent to the sitting position. He passes the day "in a heap," as it were, without power to sit upright. Both upper limbs are very small. The left can be raised to the level of the shoulder-joint; the right cannot. The elbows can be flexed. The grasp is very feeble. The patellar tendon-reflex is absent on both sides. He has no power of standing. As he lies on a bed there is just a faint power of moving his lower extremities. Both of these (like his arms) appear to be very small in comparison with the size of his body. There is very marked angular and lateral curvature in the lower dorsal region of his spine, and this it appears has been increasing much during the last twelve months. It would seem to be due partly to weakness of the muscles of the back and partly to disease of the vertebræ.

Cutaneous sensibility is unimpaired in the arms, but is thought to be slightly diminished in the legs. In the

right hip-joint there is marked stiffness, probably due to adhesions.

As regards the electrical condition in the upper limbs, all the muscles react to faradaism in the following order of excitability:—The forearms best; the arms next, the deltoids and pectorals least well; in the lower extremities no muscles of either lower limb (except those of the calf) react to faradaism. In the right thigh there is very slight reaction to the interruption of a voltaic current from forty cells. The same strength of current applied to the anterior tibial group produces no contraction, but being conveyed through the limb causes free contraction of the peronei and calf muscles.

In the back there would seem to be paralysis of both serrati magni, as well as of the mass of the erector spinæ. These last react slightly (at least the superficial ones) to a very strong induced current.

The patient was treated by electric currents, and left the hospital on April 12th unimproved.

In contrast with this preservation of a slight amount of contractility after many years, I would refer to another example, in which at the end of a year no reaction to either current was to be obtained.

Cyril W—, æt. 10, a fair-complexioned, very intelligent lad, was brought here some years ago on account of loss of power in his lower extremities of one year's duration. He could not stand. When seated his legs dangled, the toes pointing downwards. In this position he could not move any part of the right foot or leg. The left leg he could swing forwards and backwards, but could not bring the foot up square with the leg. There was no loss of sensibility, and no affection of the bladder or rectum had existed at any time. The muscles of his legs were flabby. Tickling the soles of his feet was perfectly well perceived, but there was no retraction of the foot; the legs were objectively cold. Neither the strongest induced currents nor interrupted voltaic currents produced any contraction

of the muscles of his lower extremities. There was no loss of cutaneous sensibility. It seemed that one day, about a year previously, whilst practising gymnastics, he struck his back (the lowest part of the dorsal region is indicated), but so slightly that he took no serious notice of it and went on playing. A day or two afterwards he had a very bad headache and felt very sick. At 4 p.m. he went to bed, and slept for a time. In the night, getting up to pass water, he stumbled and found difficulty in walking. Next morning he could walk better, but his legs were still affected. During that day he could walk, but felt very ill and sick. He went to bed at his usual hour, and then found himself very weak in the arms as well as legs. Next day he could not get up, and on being raised in bed he felt great pain about the middle of the back. At this time his legs were quite powerless, and his right arm became gradually paralysed from the shoulder to the elbow, but not, he says, in the forearm; this took upwards of a week to become complete. He could not lift his arm, although he could use his hand very well. In about a month his right arm had quite recovered, and before this the slight weakness of the left arm had entirely disappeared. On admission there was nothing whatever wrong with either arm. In this case I tried the effect of hypodermic injection of strychnia after a plan suggested by Mr Barwell. The solution was of such a strength as that one minim contained gr. $\frac{1}{30}$ of strychnia. Commencing with half this dose I increased the strength every alternate day until he was using gr. $\frac{1}{12}$. These applications produced no physiological effect. The treatment was employed for a month. At the end of that time the condition of the legs remained as before, no contractions occurring to either form of electrical application. The injections were then discontinued, and a daily administration of the constant current was employed. Both feet were placed in a bath with the negative rheophore, and the positive rheophore with a strength of fifteen cells was applied for five minutes to the lower

dorsal region of the spine. This application was continued almost daily for three months, when the condition of the legs remained precisely as on admission.

Another child, æt. 3, whom I lately saw, had been attacked when a year and a half old with feverishness, which lasted three days. At the end of that time it was found that the child, who had been very active in walking and climbing before her illness, could no longer stand. The right leg gradually recovered to a considerable extent. The left leg, notwithstanding electrical treatment which had been continued for a year and a half, showed no improvement, and none of the muscles responded to either current.

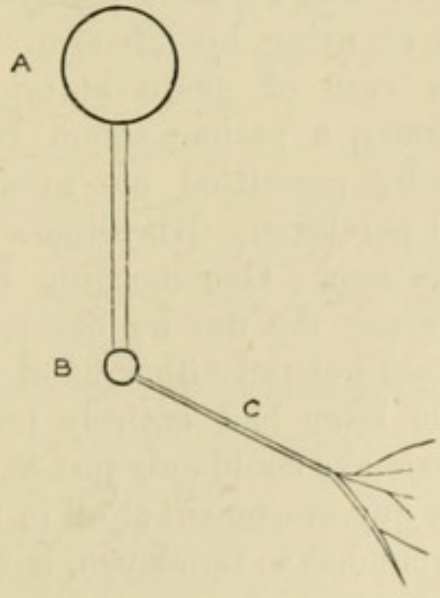
LECTURE IV

INFANTILE PARALYSIS, AND ACUTE ANTERIOR POLIO-MYELITIS IN ADULTS (*continued*)

Some thirty or forty years ago Marshall Hall adopted the term "cerebral paralysis" for cases in which a lesion had severed the cerebral influence from the paralysed limbs; and the term "spinal paralysis" for the condition in which the spinal influence was cut off from the paralysed limbs.

I may very roughly illustrate the two divisions in this way.

Fig. 9.



Let A represent the intra-cranial centres, with a portion of spinal cord below; B anterior horn of grey matter at any part of the cord; C anterior root of spinal nerve branching and distributed to muscle. Now, a lesion located anywhere *above* B would produce "cerebral

paralysis," whether it occurred in the intra-cranial cavity or in the spinal cord below it, whilst "spinal paralysis" would be found, when there was a lesion *at* B or in the nerve issuing from it. As we saw in our last lecture, the seat of the spinal influence is in the anterior horn of the grey matter. A lesion, then, at that point would produce what is called "spinal paralysis." So also would a lesion in the trunk of the nerve, and in its intra-muscular branches.

The difference between these two forms of paralysis is this. In the first case, although paralysis is produced, there is, practically, no impairment of muscular nutrition or alteration of faradaic excitability in the nerves and muscles of the paralysed limb.

In spinal paralysis, on the other hand, besides the loss of power, there is impairment of muscular nutrition, and either complete absence or great reduction of faradaic excitability.

This patient, S—, æt. 39 (a case of ordinary left hemiplegia), is an example of cerebral paralysis. He has but very small power of using his left arm. The other man, W—, æt. 45 (a case of acute anterior polio-myelitis affecting both arms), a patient whom Drs Jackson and Ferrier have kindly permitted me to show you, is an example of spinal paralysis. His illness began suddenly twenty-nine weeks ago. One morning he was unable to raise weights; during the day he became worse, and by the evening he could not put either hand to the top of his head, and a little later had entirely lost power in the hands. On admission he could only just flex his fingers, and there was scarcely any movement at all in his arms. None of the muscles responded to faradaism, but they contracted to the slow interruptions of a constant current. At the present time all the muscles react to faradaism except the deltoids.

I propose to show you very briefly the different electrical conditions of the left deltoid muscle in these two patients.

I have a battery here which enables me to use either induced or constant currents, and I apply one of the rheophores at the back of the neck, the other to the left deltoid muscle of the hemiplegic patient. First, I put on an induced (faradaic) current, and the deltoid muscle, as you see, contracts very freely. When the same strength of current is applied in the same way to the patient W— (the case of spinal paralysis) you observe that there is no contraction of the deltoid muscle. I now apply a stronger and stronger current, and there is still no response in the deltoid muscle. Do not mistake a little contraction which you see in the neighbourhood. It is in the pectoralis major. One rheophore being at the nape of the neck, any muscular fibres which lie in the direct line between that rheophore and the other will necessarily get stimulated, and will contract if they are capable of being excited by faradaic currents. The strongest induced current, as you see, produces no effect upon the deltoid muscle.

I will now employ the constant current (galvanic or voltaic current), and first of all apply it to the patient with left hemiplegia—the case of "cerebral paralysis"—one rheophore being fastened to the back of the neck, and the other, representing the kathode or negative pole, on the deltoid muscle. I suddenly complete the circuit—you see there is a contraction. The current was derived from twenty-eight cells.

Let me here remind you that when the constant current is flowing evenly in circuit through the body, there may be a very considerable power of current, and yet no muscular contraction will occur. It is when the circuit is *suddenly opened* or suddenly *shut* that you get a contraction; and we are indebted to Brenner for the formula which shows the order in which contractions naturally occur in these circumstances.

The positive pole is called the anode (from ἀνά, up, ὁδός, path), and the negative pole is called kathode (from κατά, down, ὁδός path).

Now if we imagine this (a diagram was here drawn) to be the carbon and this the zinc, the current may be supposed to flow up from the carbon or positive pole, and down to the zinc or negative pole. So it is usual to employ the term "anode" for the positive pole, and "kathode" for the negative pole, of a voltaic battery.

To express a sudden closure or shutting of the circuit, the term "Schliessung" (S) is employed by the Germans; "Oeffnung" (O) for opening; "Zuckung" (Z) for contraction. By means, therefore, of these initial letters we can very shortly picture all we desire to record.

With a galvanic current of moderate strength, supposing the kathode to be placed upon the muscle of a healthy subject, and the circuit suddenly completed, you get a contraction—a small contraction, and that is written down thus, K S z (using a small capital z). (I use the German notation because it is convenient that observers of different nationalities should be able to read each other's records without the necessity of translation.) The circuit being opened (K O) there is no contraction. In health the contraction (K S z) is the only one which occurs when a moderate current is employed. When you use a stronger current you get a powerful contraction portrayed by a large Z, *i.e.* K S Z. But besides this you now get a moderate contraction on anodal shutting, and a moderate contraction on anodal opening (A S z, A O z). That is the normal condition.

We will now turn to this case of polio-myelitis, and you see a very remarkable change.

First of all I will show you that that muscle which did not respond to the strongest induced currents does respond to slow interruptions of a constant current. When the negative pole is placed on the deltoid, and the circuit shut, there is a contraction. I am using twenty cells of the battery. This is "kathodal shutting,"—there is a contraction, but not a very large one (K S z).

"Kathodal opening:"—there is no contraction (K O—).

"Anodal shutting:"—there is a larger and stronger contraction than in kathodal shutting (A S Z).

"Anodal opening:"—there is no contraction (A O—).

So that, you see, if we want to record the condition of the muscle we write it down shortly in this way:—Faradaism O; Galvanism A S Z > K S Z, *i.e.* the contraction on shutting the circuit with the anode on the muscle is greater than that which occurs on shutting the circuit with the kathode on the muscle.

This qualitative change is one of the peculiarities of the reaction of degeneration. The test will, I believe, acquire greater importance as we learn to understand better its prognostic significance, of which I cannot say much at present. The process is useful for the purpose of testing and recording very slight changes in muscles where you think there is some temporary or permanent degeneration.

Let me call your attention to another point of very considerable interest. This patient, W—, can move his deltoid to a certain very evident extent by voluntary action.

Duchenne pointed out that after traumatic lesion of a nerve causing loss of faradaic excitability in muscles and the other signs of degenerative reaction, the power of responding to volitional impulses might return long before faradaic excitability. Erb, Bernhardt, and myself have recorded a similar condition in regard to lead palsy.* I have seen, in a case of lead palsy, marked loss of faradaic excitability in muscles belonging to a limb which was supposed by a patient to be perfectly well.

I lately saw a case of infantile paralysis in a little boy five weeks after the attack. His legs were completely paralysed. His arms had been slightly affected, I was told, but had quite recovered. He used them freely in my presence. Yet there was no sign of faradaic excitability in them, even when I used very strong induced currents, far stronger than would be required in health.

I have another case of polio-myelitis in a grown-up patient in the hospital.

* 'Brain,' No. 1.

The person is a young woman sixteen years of age; but as I shall have to show her next Thursday as illustrating another matter, I do not propose to present her to you here to-day.

She was attacked twelve months ago with loss of power in both legs. Four months after this she was admitted here.

At that time there was in the right leg no response in the peroneus longus to the strongest induced current.

The anterior tibial group of muscles responded to strong currents. In the right quadriceps extensor there was no response to either current. But at the present time—and that is the point—that muscle reacts to about twenty cells of constant current, slowly interrupted in the way I have shown.

As regards the diagnosis this is not difficult. An extremely rapid loss of power in limbs after a febrile attack hardly admits of much error, especially if the electrical testing shows that the faradaic excitability of the muscles is absent or notably lowered within a week after the attack. When the upper extremities are affected it is necessary to bear in mind that hypertrophic cervical pachy-meningitis will cause paralysis with wasting of a kind which recalls a good deal the character of that occurring in polio-myelitis. But in pachy-meningitis the course is slow, often occupying weeks, during which the condition gets progressively worse; and in addition to this, owing to the membrane being inflamed in its whole circumference, and posterior roots as well as anterior being thereby involved, strongly marked symptoms will occur on the sensory side. There are pains of extreme severity darting through the limbs, and there is more or less cutaneous anæsthesia. An injury to a mixed nerve will produce paralysis with wasting, and the nerve and muscle when tested will show the reaction of degeneration, just as occurs in polio-myelitis; but the anatomical distribution of the lesion which will be found exactly limited to the district of the nerve, and the affection of cutaneous

sensibility will serve to distinguish. It is much more difficult to differentiate localised lesions of anterior roots of nerves, for the nearer you get to the centre the more apparently irregular becomes the distribution of resulting lesions. The mode of onset and the circumstances will, however, probably indicate whether there is any probability of lesion confined to the anterior roots. One can imagine a tumour pressing upon the anterior surface of the cord and involving the anterior roots, a gumma, for example, causing symptoms indistinguishable from those of myelitis of the anterior cornua.

There is one possible source of error which, so far as I am aware, I am the first to point out. As the affection we are considering is peculiarly apt to attack very young children, it will be useful if I relate an incident which occurred to myself. I saw a year or two ago a female infant, one year old, who was described as having rather suddenly lost the use of the right lower extremity after a few hours' malaise with some little febrile disturbance. The story told was, in fact, exactly the story of infantile paralysis. When she was stripped she appeared a well-nourished, healthy-looking child. She did not move the right lower limb, but looked at it as if in some distress, and appeared to guard it with her hand. I examined it first to ascertain if there were any signs of injury, but found none. It was evident that pressure was painful, but nothing could be found in the limb to account for this symptom. The powerlessness was in striking contrast to the free movements of the left leg.

Now, the occurrence of tenderness, although unusual in infantile paralysis, was not of itself sufficient to remove the case from the category of that disease. It was marked, for example, in the history of a case brought by Dr E. Taylor before the Pathological Society last year. Investigation of the child's general health threw light upon the case. The urine was found to deposit a very large quantity of uric acid, and the explanation of this appeared to be that it was being nourished upon a milk food which contained an

enormous amount of sugar. A few doses of citrate of potash and a purgative completely cured the child, who, in a day or two, was as strong as ever on this limb. What happens in such a case I believe to be this. Minute crystals of uric acid get deposited in the loose connective tissue which intervenes between large muscles, and enables them to glide over each other, especially at the back of the thigh and in the loins. These collections of connective tissue have been shown to be lymphatic spaces, and in these the uric acid sets up some subacute inflammation which not only causes pain and tenderness, but renders the muscles almost powerless for the time being. Such a cause, I think there can be little doubt, frequently obtains in those cases of lumbago, and so-called sciatica, which are rapidly cured by free purging and alkalies with iodide of potassium. I am indebted to Dr. Burdon-Sanderson for originally suggesting this point to me for observation.

The patient whom I now present to you illustrates an important point in the pathology of the disease upon which, as it is novel, I shall have to dwell a little.

Arthur D—, æt. 17, applied here on October, 1879, on account of attacks of numbness. It seemed that for five months past he had been liable to have several times a day a feeling of numbness, which would pass all over him, but affected most the arms and hands. He had never lost consciousness. The attacks, which at first lasted but a few minutes, had become more lengthy, so that the numbness would now continue for an hour or so. He also complained of suffering from dizziness in his head, occurring sometimes along with, at other times irrespective of the numbness, and of occasional twitchings of the legs. I may say here at once that under the influence of bromide and belladonna these symptoms, which are of an epileptic character, have disappeared. It is not my purpose to dwell further upon them.

The point of interest to which I am anxious to draw attention on the present occasion is the condition of his

face and certain of his limbs resulting from a paralytic attack which occurred in infancy. Details of the attack are not now forthcoming, but we learn that he was rapidly paralysed in the left leg, right arm, and right side of face, when he was about one year old.

If you look at his face you will see that the left angle of the mouth is higher than the right. The right cheek looks flatter than the left, and the nasal fold on this side is absent. A like want of symmetry is not to be noted in the upper part of the face. The eyes are closed with equal facility, and he can frown with both brows. In endeavouring to show his teeth there is an absence of the natural upward movement of the right half of the upper lip. He tells us that at times food collects in the right cheek.

There is no impairment of sensation. The pupils are equal, there is no deviation of the tongue, the movements of the eyeballs, vision, taste, smell, articulation and swallowing, are all normal. He hears somewhat better, according to his account, on the right than the left side.

Now, the paralysis of the face which remains in this case looks much more like that which occurs in an ordinary hemiplegic seizure, than that which is produced by a lesion of the trunk or nucleus of the portio dura. It is, as you see, confined to the lower half of the face, and affects especially the buccinator and those muscles of the cheek which pass more or less obliquely to the angle of the mouth. The situation, indeed, is precisely that which is observed in hemiplegia, but at the same time the completeness of the paralysis sixteen years after the attack is greater than that which is commonly seen in the early days of a hemiplegia. When I say commonly, I refer to the hemiplegia dependent upon lesion of the higher portions of the cerebro-spinal axis. You do see this amount of facial paralysis, however, where the lesion is situated in the lowest part of the pons Varolii, and it is then usually accompanied by paralysis of the limbs of the oppo-

site side of the body. To this condition the term "alternate hemiplegia" is applied.

Has this been a case of alternate hemiplegia, then, from lesion of—say hæmorrhage into—the pons Varolii? Let us see what is the state of the limbs. The lad stands and walks without apparent difficulty. If he attempts to run, however, he is lame, and he complains that he is always somewhat unsteady on his legs, and easily knocked over. His lower limbs are of the same length, but there is an important difference in their circumference. The right thigh, seven inches above the patella, measures three inches more in circumference than the left. In the calf the same amount of disparity is not seen, the left being only a quarter of an inch smaller than the right.

Turning now to the upper extremities, we find that the lad's grasp with the right hand is far inferior to that with the left; by the dynamometer, 30° as against 48°. The right arm is three-quarters of an inch smaller in circumference than the left; and the right forearm one inch smaller than the left. The length of the upper extremities is the same. The right deltoid is very much wasted, and in consequence of this the patient has great difficulty in raising his arm to the level of the shoulder unless he brings it in front of him, when the help of the pectoralis major is called in. In this deltoid the amount of contraction to the induced current is small. In the other muscles of the arm, and in the face, it appears to be normal, or at least not materially lessened.

Here we have, then, evidence of paralysis in the *right* side of the face, the *right* arm, and the *left* leg. This is clearly, therefore, not a case simply of alternate hemiplegia; and it is evident that the existence of one lesion will not account for the paralysis observed. On the other hand, it seems certain, from the history, that the paralysis of the various parts took place at the same time. What is the nature of this case?

There are two points which will afford us help in arriving at a conclusion. You have observed that in the

left lower extremity, whereas the thigh measured three inches less than its fellow of the opposite side, the leg is only a quarter of an inch smaller than the other. The wasting of the left thigh is therefore quite out of proportion to the wasting of the left leg. Now, were this a case simply of emaciation of a hemiplegic limb from disuse and impaired nutrition of muscles there would be a strictly proportional diminution in size in the thigh and leg.

In the present case it is evident that certain muscles of the thigh have wasted, whilst those of the leg have almost entirely escaped damage. If now we test the two legs we find that the patellar tendon-reflex is absent in the left but is present in the right limb. This tendon-reflex, I may tell you, is never absent as a result of hemiplegia of cerebral origin. Its absence signifies that there is a break in the arc of nervous communication between the tendon and the muscle belonging to it. That arc includes nerve fibres going from the tendon (along with the posterior roots) into the cord, the grey matter of the cord at this level, with the large motor cells in the anterior cornu, and the anterior roots forming the motor nerve to the muscle.

It remains to be considered at what point there is a break in the arc. In tabes, as you are aware, the patellar tendon-reflex is also absent. In that disease, however, the condition of the quadriceps extensor muscle is normal. It is not (or rather need not be) wasted, and it contracts readily to voluntary impulses, to faradaic currents, and to direct percussion of the muscular substance. The motor part of the arc being thereby proved to be intact, we can readily refer the break to some point in the sensory portion, and in fact it occurs in the posterior root zone, which, as you know, is the seat of sclerosis in tabes dorsalis. But in this lad the muscular atrophy and the defective faradaic excitability show that the lesion is on the motor side of the arc, and a consideration of the symptoms easily relegates it to the anterior cornu. This is doubtlessly wasted, and there is atrophy of large ganglionic cells.

Again, the muscles of the thigh are not atrophied in the district of a particular nerve. As you will observe, the posterior muscles supplied by the sciatic nerve are quite as much involved as those in the district of the anterior crural nerve. On the other hand, the muscles below the knee, which owe their nerve supply entirely to the great sciatic trunk, are scarcely affected. Were there a lesion of the trunk of the sciatic nerve, all the muscles supplied by it would atrophy. We must, therefore, as I have said before, go still further back for the lesion, and we can localise it also from this point of view in the anterior cornu.

The case is evidently, therefore, one of anterior poliomyelitis. But how are we to explain the facial paralysis? According to my experience this is a very rare complication of infantile paralysis. The systematic works treating of the subject which I have consulted either fail to mention it or expressly exclude this symptom from the category of those indicative of infantile paralysis. I have no doubt that in the case before us the facial paralysis has been caused by an extension upwards into the medulla oblongata of the process, which when it affects only the anterior cornua of the spinal cord, occasions paralysis and wasting of the muscles of the extremities. You are aware that the medulla oblongata contains collections of grey matter which give origin to various nerves, and are homologous with the anterior cornua of the cord. In lateral sclerosis with muscular atrophy—a chronic disease—the lesion which at first causes paralysis with wasting of the extremities is apt to invade these nuclei which are contained in the medulla oblongata, and then we see paralysis of the lower part of the face, of the tongue, and the pharynx—the condition known as glosso-labio-pharyngeal paralysis. Sooner or later the adjacent nuclei of the vagus become invaded, and death is thereby caused. Now, in such a case as this before us it is, I think, quite evident that the acute myelitis has invaded, along with the anterior cornua of the cord, the nucleus of the portio-dura, and produced facial paralysis.

The reason why facial paralysis is not more common in infantile paralysis I believe to be this: should the acute affection invade the bulb it is unlikely to spare the nuclei of nerves which are essential to life. It is evident that a disease which produces sudden and complete paralysis of limbs when it affects the anterior grey matter of the cord, would cause rapid death if it attacked in a similar manner the nuclei of the vagus.

And here let me say that I cannot help thinking that many cases of sudden or very rapid death which occur in children, and the cause of which is often left to conjecture, may really depend upon this disease striking the medulla oblongata with the same kind of suddenness with which it ordinarily attacks the anterior grey matter of the spinal cord.*

In infantile paralysis of many years' standing you will sometimes find that, whereas the patient says that he is only defective in one leg, the difference between that limb and the supposed sound one is not nearly so great as you might expect. There was a young man here the other day who said that his left leg had been paralysed since he was a year old. All four limbs, he added, were paralysed at first, but the other three recovered somewhat rapidly, leaving him with this useless limb. He thought it was no shorter than the other, but we found a difference of an inch or more. Considering that the paralytic seizure had occurred when he was a year old, this was little enough—far less, indeed, than might have been anticipated; for we know that the disease often affects the growth of the bones very seriously, and it is not uncommon to find a difference of several inches between the two limbs. On coming to examine him I found the patellar tendon-reflex absent, not alone in the leg complained of,

* Dr Hale White was led by these observations to examine microscopically the medulla oblongata of a child who died suddenly in the Evelina Hospital. He found extreme vascular dilatation over a considerable extent of the bulb, with several hæmorrhages, one of which occurred at the margin of the nucleus of the vagus. Pathological Society, 'Brit. Med. Journal,' Feb. 25, 1882.

but in the other also, and this circumstance leading to a further observation of the supposed sound leg, it was discovered that the latter was far from being healthy. There was but little response to faradaic currents in its muscles, which felt of a flabby and brawny character, and were evidently only less extensively degenerated than those of the left limb, which showed no response at all to either form of electric current. This explained at once the reason why there was so little difference in size between the two limbs. From what I have seen in other cases, I am disposed to think that the condition described is not at all unfrequent, indeed, that it is very common.

There is a young man in attendance, a patient of Dr. Ormerod, who came to the hospital the other day on account of fits, but incidentally mentioned that he had lost the use of the left leg in infancy. I have not yet examined him, but will do so now before you. (The patient was here introduced and the lower extremities stripped.) In answer to my inquiry he says that there has been nothing whatever wrong with any limb except the left leg. You see, however, that the right leg is really not much more developed than the left, and the patellar tendon-reflex is absent on that side as well as on the other. Here, again, there is no doubt the patient is suffering from the remains of infantile paralysis in his right leg as well as the left though in a somewhat less degree. It is the contrast only which has led him to suppose that the right leg was sound. Were we to test the muscles of the right limb electrically, I am sure (from the state of patellar reflex) that we should find more or less loss of faradaic excitability.

A child, Helena M—, now attending the hospital, was brought to us on account of paralysis, which was stated by her mother to affect the right leg exclusively. She has, however no patellar tendon-reflex in either leg, and certain muscles of the left leg will not react to either electric current. It is from applying the test of the patellar tendon-reflex that I have been led to discover how very

generally, in a patient affected with old infantile paralysis, those limbs which were supposed to be sound are not by any means normal. I feel sure that monoplegia in this disease is far less common than it is usually thought to be, and that, as in the case which I have shown you, limbs are very frequently supposed to be sound which are in reality only comparatively so.

I do not feel able to say anything regarding the causation of this disease, except that there does appear to be a certain consensus of evidence to show that exposure to cold is a frequent antecedent, and very possibly, therefore, a cause. There is also, I think, but little doubt that injuries to the back may occasion the disease; but beyond this I have really nothing to say. From the negative side, however, there is a point which is perhaps worth a moment's attention. The disease is commonest in infants, next in children, least common in adults. Children, and especially infants, take no alcohol; the girl F— has had little or no alcohol. A typical case of the disease, B—, whom I shall show you presently, has been a total abstainer for eight years. A single lady, æt. 25, affected with the paraplegic form of the disease, whom I saw lately, in consultation with Dr. Bennett, had taken no alcohol for five years previous to her attack. A rapidly fatal case of acute myelitis which I attended a few years ago, occurred in a man who had been a total abstainer all his life, and was the son of a total abstainer. Now, I need hardly say that I do not refer to this point in order to argue that the disease occurred *in consequence of* abstinence from alcohol; but it is something in these days to be able to absolve alcohol from the charge of being the cause of a very destructive disease. This piece of negative evidence may aid us also in other directions; that is why I refer to it.

A point of great importance in reference to this disease is the prognosis. If we look into the nature of the lesion and reflect that we have to do with the destruction of ganglionic cells, bringing about secondarily atrophy of the nerves rising from the anterior cornua,

and eventually, though it may be long delayed, degeneration, more or less pronounced, in the muscles to which these nerves go, it is difficult to imagine a more hopeless prospect than must appear likely as regards many of the muscles involved. And so in effect we find it. In a large number of cases the future signifies a progressive deformity which does not shorten existence, but is carried through life with scarcely any amelioration. If the lower extremities be concerned the patient may be only able to stand and walk with the aid of apparatus.

In the last lecture I referred to the case of a lad in whose thigh there still remained, thirteen years after the attack, some evidence of the continued existence of muscular fibre in the fact that there was a slight contraction to the interruption of forty cells of a constant current. In a child, seen not long ago in private, I found good contraction in the muscles of the thigh and leg to a constant current from thirty-five cells slowly interrupted, two and a half years after the attack.

These and other examples, however, only illustrate in too many instances the fact that the contractile element is very long lived, even when the muscle is separated from its physiological connection with the spinal cord. But for needs of locomotion we require something more than the existence of an element which is capable of being contracted by an electric or mechanical stimulus. It is necessary that the road by which motor impulses from the brain travel to the muscle should be clear, and if the anterior roots of nerves are atrophied and degenerated the mere persistence of the contractile element of muscle is utterly useless. It is, indeed, the degeneration of the nerve cells and tubules which is the disastrous part of the lesion in this disease. Were it not for the more or less permanent character of these changes, in many cases the wasting of the muscle itself which occurs so early would be unimportant. This is, at least for a long time, a simple atrophy, capable of complete repair should the way down from the nervous centres to the

muscles through the ganglionic cells and anterior roots of nerves be opened up.

Charcot points out, from the experiments of Longet, Schiff, Brown-Séquard, and others, that when a nerve has undergone section or partial excision, its peripheral extremity begins from the fifth to the sixth day after the operation to undergo, even in its finest ramifications, a series of alterations whose ultimate consequence is the disappearance of the medullary cylinder, the axis cylinder persisting according to some but not all observers.

From the fourth day the nerve, M. Vulpian says, is found to have lost the faculty of being excited by the electrical stimulus. As regards the muscular fibre, it does not present at first any modification whatever of the electrical contractility. The decrease and, still more, the utter loss of this property, if they do ensue, are never produced until after the lapse of a considerable time and very slowly.

As to the amount of repair of which a nerve fibre which has suffered degenerative changes is capable we know at present little for certain; but clinical experience would tend to show that after a long while improvement to an unexpected extent may indicate that reparation has been going on. This subject has been admirably discussed by Weir Mitchell in his work on injuries of nerves. The fact is one which should make us hesitate in giving a very unfavourable prognosis even after a great lapse of time.

As a practical contribution to the question of prognosis in the acute anterior polio-myelitis of adult life, I show you a young man who was a patient in this hospital rather more than two years ago. The peculiar interest and importance of his case depend upon the fact that he came under care, and I have notes of his condition taken by Mr Broster, only eleven days after the beginning of his illness. I will read these notes to you now, and then compare them with his present condition.

George B—, æt. 19, who had followed the occupation of a gardener from the age of 13 years, was admitted

into the hospital October 2nd, 1876, on account of loss of power in the left arm and both legs. The patient is a fair, healthy-looking and well built lad. He is quite free from pain as he lies in bed; if he sits up he gets a sharp pain in the lower part of his back, which shoots down his legs. He sleeps well; his general health is good; the temperature normal. He can flex the left thigh on the pelvis completely, and rotate it inwards and outwards, but no other movement is possible in the left lower extremity. The only movement practicable in the right lower limb is a feeble flexion of the toes. The left arm is weak, especially in the deltoid muscle. The spinal column presents no abnormality, nor is it tender on percussion. When his legs get hot in bed there is an aching pain in them. Muscular sense appears defective in his legs. Touching the soles of his feet produces no reflex movement. The patellar tendon-reflex is absent from each leg. There is no rigidity, and he does not suffer from any muscular twitchings. The muscles of the legs are very flaccid and shrunk, and those of the left arm are smaller than those of the right arm. There is no affection of special or common sensation. The heart, lungs, and kidneys show no signs of disease. The bowels are somewhat constipated; the action of the bladder unimpaired.

Electrical examination on the eleventh day of his illness.—There is no reaction to the induced current in any muscles of either of the lower extremities except the vastus internus of the left thigh. The muscles of the left arm react fairly to the same current. To a slowly interrupted voltaic current from fifteen cells of a Stöhrer's battery the muscles of the lower extremities act well.

The patient has been a total abstainer for eight years; he has lived comfortably. There is no history of acute rheumatism or of scarlet fever; and, with the exception of a cold, and an attack of erysipelas in his right leg, he has never had a day's illness until the present attack. There is no history of syphilis.

A grandfather, grandmother, his father, mother, two or

three uncles and aunts, and their cousins, have died of "shortness of breath" and "consumption." His father had heart disease after rheumatic fever. The ages of the above relations at the time of their death varied from twenty to forty-seven. All the family, the patient included, are, or have been, subject to a peculiar attack consisting of giddiness and inability to stand, with mistiness of vision (but without pain in the head, or spectral vision), the whole lasting about five minutes.

A fortnight before admission he had caught a violent cold from getting very hot at work, and then letting himself get cool in a draught. Two days afterwards he complained of very severe pain in the lumbar region, increased by movement. At the same time there was a throbbing pain down the backs of both legs. His legs twitched, he says, and increased the pain. He suffered from retention of urine, for which a catheter was employed on one occasion only. Next morning he found that he could not stand, and was unable to raise his left arm to his head. There was no loss of speech or impairment of consciousness, and he says that he was not feverish.

Three months after the commencement of his illness his condition was as follows :—The right lower extremity is motionless. He can draw up the left leg, but cannot raise the left foot off the bed. The right leg, as he lies, is rotated inwards. There is a wasting of both thighs and legs. Measurement shows that their size is equal. The calves measure $10\frac{3}{4}$ inches each, the thighs (11 inches above the knee) 15 inches each. A month later the right calf measured $12\frac{3}{4}$ inches, the left $12\frac{1}{2}$ inches. The right thigh was $16\frac{5}{8}$ inches, the left 17 inches. There was still no reaction to faradaic currents except in the left quadriceps femoris muscle. In this state he left the hospital. Ten months afterwards, in November, 1879, I saw this lad at his own home. His general health was very good. The left arm was perfectly well, and to all appearance differed in no respect from the right. But he was unable to stand. The right lower limb remained as it was when he left the

hospital, there had been no further wasting. He could flex the leg a little on the thigh, but could not extend it. He could adduct with considerable force, but was unable to abduct at all. The patellar tendon-reflex in the left leg was present, although small. In the right leg it was quite absent.

This was a twelvemonth ago. He has attended here to-day at my request, and we can now compare his present condition with the last report. He can extend the right leg a little, but he can bring the foot back still better than he can throw it forward; and he can also adduct better than abduct the limb. There is still no patellar tendon-reflex in the right thigh. His limbs, as you see, show no wasting; on the contrary, they are large and round; but it is evident that their size greatly depends upon adipose tissue, not muscle. It is sometimes difficult to test the patellar tendon-reflex where there is a very fat knee; but I think we may be sure that the reflex is absent here.

I will apply induced currents to the anterior muscles of the left thigh, and you see that, with a strength greater than is needed for normal response, there is a slight contraction in the vastus internus, and a somewhat better contraction in the rectus femoris.

When, instead of the induced, I apply the constant current, there is a contraction on completing the circuit, but A S z > K S z. The reaction of degeneration is still, therefore, marked.

In the right limb there is no reaction to induced currents, and a very slight contraction when the circuit is closed with either the kathode or the anode on the muscles.

It is to be noted that, although the greatest amount of improvement took place in this patient during the first four or six months of his illness, yet he has not remained absolutely still during the last twelvemonth. He can now abduct the right knee a little, and extend this leg which he could not do a year ago.

F. Müller, who has lately published the result of some

important researches on this subject, says that all muscles remain completely paralysed in which the faradaic excitability descends perpendicularly from its normal level, and disappears entirely in four or five days. In all the cases in which this excitability *diminishes* only, without disappearing before the twelfth day, and in the cases in which it descends only gradually, motility returns, even after several months. From the complete disappearance of faradaic excitability, or from the reaction of degeneration in the first stage, one can conclude nothing in regard to the curability or incurability of the case. When the contraction A S z alone remains, this shows the complete disappearance of the contractile substance.

In infants monoplegia, in adults paralysis of four limbs, or paraplegia, is, according to Müller, the most frequent. But having regard to what I have described, I would suggest that it is probably more common for the lesion to clear off to a large extent in infants than in adults. I feel sure that it is very uncommon for a single limb to be alone struck by the disease. Synergic muscles are frequently attacked, and frequently recover together.

Müller, in a case in which he had the opportunity of testing the muscles from the first day for one and a half years, found that the faradaic muscle excitability still persists when the nerves have lost theirs. On the fourth day faradaic excitability was lost for the nerves; on the fifth for the muscles. Galvanic excitability of the muscles was slightly lowered at first, but in the course of the third week it increased, and AS Z was > K S Z, but there was not such marked increase as we see after traumatic lesion of nerves. The exaggeration, he remarks, does not last long. There is a return to the normal, or else complete disappearance of contraction. Muscular wasting is visible after ten or twelve days. Regression commences in those muscles which have shown only quantitative change. It may begin in four, six, or twelve days, but more often is delayed for some weeks. Motility returns last in muscles

which have shown the gravest signs of reaction of degeneration. Regression is very rarely complete.

According to Duchenne the attitude of a limb depends almost exclusively upon the tonicity of the muscles which move it. The muscles are like so many springs, which, during muscular repose, maintain the limb in its normal attitude. If one of these springs becomes weak the equilibrium of these tonic muscular forces is broken, and the limb is dragged, in a continued manner, into vicious directions. Hence in this disease there is greater risk of serious deformity than in any other form of paralysis. In most paralytic conditions the muscles of a limb are rendered to all intents equally powerless—the interruption of innervation affects them alike—and as a result, though the limb may be useless, its symmetry may remain undisturbed. But in this disease, as I have had the opportunity of showing you in many cases, side by side with a muscle which is absolutely paralysed and wasted, you may find one which acts to voluntary and electrical stimuli. The unopposed tonicity of this muscle will necessarily cause the limb to assume a distorted appearance.

In other cases all the muscles may be wasted, and the joints so loose that the limb hangs like a flail.

Between the disease as it occurs in children and in adults, the main points of difference appear to be as follows:—In the former convulsions are not uncommon at the onset. In the latter these are not seen, but there is usually complaint of pain in the head. It is evident that the deformity will be greater when the patient is attacked during the period of growth than when this has been completed. Hence the results of the disease are not nearly so disfiguring when it takes place in adult life as when children are attacked. In either case the position assumed by the limb, besides being influenced, as I have described, by the want of balance in the muscles, may be also much affected by the presence of more or less sclerosis of the lateral columns of the cord, which is apt

to occur in connection with the affection of the anterior grey matter.

In advanced cases of acute atrophic paralysis in children the affected limb is often much smaller than its fellow, and this not only in circumference, but in length. Duchenne has seen a limb in the course of a few years measure six to eight centimètres less than its fellow. This arrest of development of the osseous system is not necessarily in proportion to the extent of the affection of muscles.

A limb may have lost the greater part of its muscles and yet be only two to three centimètres shorter than that of the opposite side; whilst in another limb shortened by five to six centimètres, the muscular lesion may be localised in one or two muscles. The ligaments become so lax that the head of a bone no longer touches the socket in which it should rest. I would mention that, remarkable as is the effect upon the size of the bones, it is evident that we have not to do here with a rapidly destructive process as in the affection of bones and joints in tabes. The bone ceases, or rather nearly ceases, to grow, or perhaps it would be still more correct to say that its growth is enormously retarded. This arrest is quite in accordance with what might be expected from the interference with the blood supply of the limb. Its temperature is much lower than normal, it looks blue, and the skin is ill nourished.

Yet we never, so far as I have seen, get in any of these cases either spontaneous fractures of a bone, or that active and rapid destruction of a joint with absorption of the articular extremities which is so remarkable a feature of tabes dorsalis. Considering the extensive character of the destruction which overtakes the anterior cornu in many of these cases it seems to me impossible to suppose that if the arthropathy of tabes were really traceable to lesion of the anterior cornu we should not get examples of a similar arthropathy in the course of infantile paralysis.

A very few words as to treatment.

Were I to have the management of a case of this disease from the first hour I should be disposed to adopt some such line of treatment as the following:

The patient should be kept absolutely at rest in bed, a light diet administered, counter-irritation in some form applied to the neighbourhood of the spinal column, and the bowels emptied by a mercurial, followed by a saline purgative. At the same time it would be well to give the liquid extract of ergot in full doses, at intervals of an hour or two at first, and gradually less frequently—the administration of this drug not being continued beyond a week. At the end of eight or ten days the faradaic reaction of the muscles should be carefully tested and recorded, but induced currents should only be applied for the purpose of testing. For treatment I should apply a continuous current from 5—10 cells from the nape of the neck to the bottom of the spine for five minutes daily, taking care to increase the strength of the current from zero upwards gradually, and at the end of the application to lower it to zero before removing the rheophores so as to avoid shock. The constant current might be applied in this way for three weeks at a time, then omitted for a week and reapplied as before for three or four weeks.

It seems to me that there is a positive objection to the use of faradaism to the muscles, especially in an early stage of the disease. Let us be as careful as we may in thoroughly wetting the skin and the rheophores, it is impossible to apply induced currents without, to a certain extent, stimulating the cutaneous nerves. Now impulses started thus in them are conveyed by the posterior roots to the grey matter of the cord, and thence doubtless strike the large ganglionic cells in the anterior horn. In the state of inflammation in which we may suppose them to be, it is evidently desirable to avoid this source of irritation.

Indeed as regards the use of electrical application to

the muscles at any period, it seems to me of doubtful advantage. The difficulty we have to contend with in this disease is, I have said, that motor impulses fail to reach the muscles, and this difficulty is not met by endeavours, more or less successful, to cause contraction in the muscles by electrical stimuli. The best example of recovery which I have shown you occurred in a child who had no electrical treatment at any period. It is notorious indeed that the more or less rapid return to their proper function of many muscles which we so often see occur in the disease, may take place in the complete absence of any electrical applications.

I cannot help thinking that the extremely energetic electrical treatment which it is occasionally the fashion to employ in cases of this disease, has sometimes a mischievous effect. A mild constant current, on the other hand, applied as above described, may not have any powerful effect, but I do not think it does harm. Its use is constantly attended with a sense of increased warmth in the affected limbs. With the same object the limb should be kept well covered, and may be rubbed a little. When signs of contraction and deformity begin to appear it is well to have the limb provided with an apparatus which shall compensate as nearly as possible for the lost muscular power, and endeavour thus to prevent much deformity.

I would not encourage voluntary movements of a limb in which the muscles are unequally strong, and for this reasou : Duchenne has shown that in all muscular movements there are two forces at work—one which produces the movement, the other which moderates it. If the directing muscle overacts it is because the moderating muscle (its antagonist) becomes muscularly insufficient to repress its action. When we flex the hand we do it slowly or quickly, by letting the extensors act very much or very little. Both flexors and extensors act in flexing or in extending the hand. You can easily test this. Lay the left hand on the dorsal surface of the right forearm, and then shut your right hand. You will feel the *ex-*

tensor muscles of the right forearm contract along with the flexors. In lead palsy when the extensors are paralysed, the grip is weak.

If in polio-myelitis, with paralysis of the peroneus longus, you encourage the patient to try and press down the inner border of his foot you excite him really (in the absence of action of the peroneus longus) to throw into contraction its antagonist, the tibialis anticus, which is, we will say, sound, and you thereby only intensify the deformity.

LECTURE V

ON THE DIFFERENTIAL DIAGNOSIS BETWEEN CERTAIN HYSTERICAL CONDITIONS AND MYELITIS

The subject of nervous mimicry of organic disease in general has been, as you are well aware, admirably discussed by Sir James Paget. It is not my intention to take up your time by attempts to add anything of importance on the general question to the account which we owe to one whose unrivalled experience has been recorded with consummate literary skill. But I wish to bring before you to-day some cases of recent occurrence to illustrate the difficulties which often occur in distinguishing between certain forms of acute and chronic myelitis, and hysterical imitations of the symptoms belonging to such conditions. Opportunity will thus be given me for referring to points of diagnosis that may assist in preserving us from errors which are of very frequent occurrence. I may say at once that it is often extremely difficult, and occasionally for a long time impossible, to arrive at a confident opinion in cases of this kind. Yet, unless we can do so, should the case be one of hysteria, we stand little or no chance of curing our patient, and for obvious reasons it is a still more serious matter if we mistake a case of organic disease of the spinal cord for one of hysteria.

But, first, let me mention the forms of disease which, according to my observation, are the most likely to be confounded with hysteria, and I will here refer to Grasset's classification, which has been already introduced to you on a previous occasion.* On the side of *systematic*

* See p. 41.

myelitis there is first spastic spinal paralysis from sclerosis of the lateral columns of the cord, and next acute anterior polio-myelitis, that affection of the anterior cornua upon which I lectured last week. In my experience tabes dorsalis is rarely or never imitated by hysteria. I certainly did see not long ago a lady in whom a complete and temporary loss of the muscular sense in the lower extremities imitated one of the symptoms of locomotor ataxy, and produced a slight incoordination in the gait, but the diagnosis was easy owing to the entire absence of any corroborative symptoms. On the side of *diffuse* myelitis—*i.e.* myelitis invading without distinction any or all of the regions of the cord—two forms alone need special reference. Of the first, a circumscribed and subacute variety (most often, perhaps, when it attacks the dorso-lumbar region); of the second, that special form which we know as insular or disseminated cerebro-spinal sclerosis. Of this last I shall only say now that it is as regards this form of myelitis, and its imitation by hysteria, according to what I have seen, that an insuperable difficulty of diagnosis is very apt to occur. On the present occasion I shall refer to cases in which subacute myelitis, anterior polio-myelitis, and lateral sclerosis come in question.

The first case to which I shall draw your attention is that of a school girl, æt. 14, who was admitted into this hospital early last summer. She had never suffered from scarlet fever or rheumatism, and there had been no phthisis in the family, nor was there any neurotic history. The catamenia had been regular. At twelve years of age she was one day leaning out of an open window, when a child at a window above let some water fall, in play, upon her head, and it struck her behind the right ear. She was much shocked, and all that night suffered from earache. For some weeks after this she continued to go to school, but constantly complained of earache and headache, and it was noticed that she was gradually becoming deaf. About three months after the occurrence she is described as having become childish and delirious, and rapidly losing

her sight, hearing, taste, and smell. The sight was observed to fail first. It is said that she had quite an idiotic laugh. She continued to be "delirious" for a week. Then she suffered from very severe pain all over her, especially in the head. The pain is said to have been very intense in her ankles, and her mother says that "her ankles seemed to be dislocated" from the drawing of her legs. Then she had twitchings of the legs of such violence that they were thrown high in the air. The twitchings were not, it seems, confined to the legs, but the right arm and left shoulder were similarly affected. There was also (from the description) some opisthotonos. The twitchings continued for two or three months.

The sight returned quite suddenly; "a flash seemed to pass before her eyes, and she recovered her sight," and with this improvement the spasms also became less. Then the hearing returned, and the spasms ceased.

Seven or eight months after the onset of her illness, she got into the state which she presented when admitted here, and she had remained in this condition for a year before she came to us. During the whole of her illness her general health, according to her mother's account, had been very good.

On admission here, the following report of her condition was taken by Mr Broster, the then resident medical officer:—She is a well-nourished and healthy-looking girl, who sleeps well, and does not suffer from headache or giddiness. Her hearing, sight, taste, and smell are all normal. There is no want of symmetry in the face. The pupils, which are somewhat large, react to light. The upper limbs are well nourished, and the cutaneous sensibility is normal. There is no pain, stiffness, or tenderness of the spinal column.

As regards the lower limbs, the patient says that she is unable to stand without support. She cannot walk, but with a stick in one hand, and leaning upon some one, she is able to "spring." This, indeed, is the way in which she gets upstairs; and her mother says that she is able to

spring up three stairs at a time. She lies in bed with her legs extended, her ankles and feet *tied together*. In this particular position she avers that she is quite comfortable. She is able to flex the hip and knee-joints with as much force as ever she could in her life, and can also readily flex and extend the ankle-joints, and move her toes freely. The cutaneous sensibility of the legs is perfect.

If one unties the bandage by which the ankles are fastened together, there is sudden and immediate adduction of the knees, with some rotation inwards of each lower extremity. This is accompanied by enormous rigidity of the anterior and internal muscles of the thighs. By the action the ankles are at once thrown apart, and the feet (each pointed strongly inwards) are crossed and locked rigidly one over the other. As all this happens she cries out and complains of great pain. The knees are so firmly locked together that it appears surprising that the portions of skin in apposition are not sore.

The muscles of the lower extremities react normally to the induced current. There is excess of patellar reflex on each side, and ankle clonus.

For five days the muscles of the thighs and legs were faradised with the strongest currents, whilst the feet were untied. A very slight relaxation of the rigidity was produced whilst the current was being applied, but the effect was altogether temporary; the limbs instantly resumed the rigidly locked position when the application ceased. So strongly adducted were the limbs that, with my utmost force, I could not separate them. Placed on her feet, with the ligature removed, she was quite unable to stand, and sunk, "in a heap," to the ground. To judge by the expression of her face and her tears, the rigid position which the legs assumed when unfastened, caused her great pain.

After being six days in the hospital, she was placed under the influence of ether, and as she became insensible, the muscles of the legs were felt to relax. She was then

allowed to recover, but consciousness had not apparently completely returned, when the legs resumed, with extreme suddenness, their rigidly adducted and rotated position. We noticed, however, that they were now not so rigid as before. On regaining a certain amount of consciousness (being still intoxicated with the ether) she was induced to stand with help, and to walk, although feebly and clumsily.

She was now once more placed under the influence of the ether, with the result of producing the same complete relaxation of muscles. This time she was more deeply anæsthetised, and before being permitted to recover the legs were abducted and rotated outwards and held in that position. As she recovered consciousness the legs did not resume their rigidity, and the girl expressed unbounded delight when she knew the position of her limbs. The left foot was perfectly straight, but there was a little inversion of the right foot when left to itself.

Ankle clonus could be obtained easily on the right side, but it was not of quite so prolonged and strongly marked a character as in spastic paraplegia. It was present though to less degree on the left side. This time, whilst recovering from the ether, she vomited profusely. During the first application she was under the influence of ether for two minutes, and for five minutes during the second. The quantity of ether used was one ounce.

Next day we found there had been no return of rigidity, but there was an indisposition to walk. The ether apparatus having been sent for, she improved considerably.

On the following day she walked as well as ever, and two days later walked to church and stood at appropriate times during the service.

A week after the etherisation an endeavour was made to induce ankle clonus, but without success in either foot.

She was discharged quite well within a fortnight of her admission. I learned a few days since that she has since continued in perfect health.

We had the advantage here of a *complete* history, and the diagnosis was easy. But I can quite understand that during the progress of this case there must have been difficulty especially in excluding cerebral and probably spinal meningitis, and that the rigidity with exaggerated reflex would have a *primâ facie* resemblance to that arising from sclerosis of the lateral columns secondary to a lesion of the cord. But the course of the disease—the opisthotonos followed by *sudden* return of sight—was conclusive of hysteria. Moreover, had the rigidity depended on bilateral sclerosis, the jumping feats would have been absolutely impossible. Now, this girl recovered rapidly because we were absolutely sure of our diagnosis. You cannot cure a case of hysteria so long as you have any serious doubts about its nature.

On the other hand, I think that if you are able to be quite certain on this point and are prepared to act with sufficient energy, there are few cases that will not yield to treatment. The kind of treatment does not appear to be of so much consequence as its impressiveness. There can be but little doubt that many of the cases which recover underthe hand of the bone-setter are really cases of hysteria, which are cured by the administration of the anæsthetic, as this girl was cured. The faradaic current is a very potent means of treatment in hysteria. A year or two ago a young lady about the age of eighteen whom I had seen on two or three occasions in the country with her medical attendant was sent up to town by him into lodgings near me in order to be under my observation. She had been for some weeks affected with periodical attacks of dyspnœa, the peculiarity of which was that they were of daily occurrence and commenced precisely at the same hour every morning. The morning after she had taken up her residence in town I was sent for to see her in one of the attacks, and arrived within a few minutes of its commencement. I will read you the notes which I took at the time:—She is sitting up in bed, the eyelids closed, the hands down by her hips, clenched, the thumbs inwards, the muscles of the arms rigid.

There is a noisily drawn inspiration followed by expiration, which is very loud and hoarse, constituting a kind of extremely noisy harsh cough. This is followed by a series of short expiratory catches. The mouth is fixed unduly open, and mucus and saliva flow from it, and sometimes from the nostrils. During inspiration she raises her trunk to the perpendicular. During expiration she bends forward, so that her face nearly touches the bedclothes. If she is touched during the attack, she shows impatience and turns suddenly away. There is general rigidity of the muscles of the trunk and extremities. The larynx is fixed. When I force the eyelids open (for the orbicularis palpebrarum participated in the general rigid contraction of muscles, and the eyes were thus kept closed), I find the eyeballs turned upwards and inwards. The feet are in over-extension.

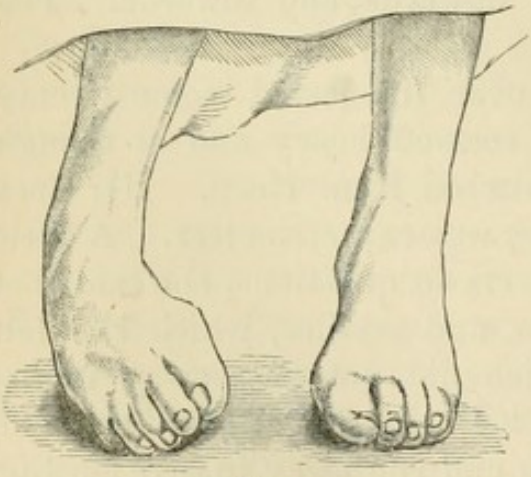
When the attack was over (it lasted some twenty minutes) I noticed that she seemed heavy and as though just awake, and scarcely aroused from sleep. Her face (naturally an intelligent one) was expressionless. A little later, when I obtained answers to questions, she said that there was a feeling of pins and needles, which she felt during the attack, and which did not pass away immediately after its cessation. "During the attack," she said, "she could not have got up and run away to save her life, if for example the house had been on fire."

I sent an induced current machine to the house, and next morning, when the attack commenced at 8.25, the regular hour, I was able to lose no time in applying electricity. The machine was arranged for its full strength. I placed one wetted rheophore on the neck over the larynx, and the other was then swiftly introduced within her open mouth. The shock was very severe, there was a wild shrinking away, and an attempted scream, but it was some few minutes before repetitions of the painful application asserted their power, and the spasms gradually ceased, having, on this occasion occupied less than half their usual time. Next morning I took the precaution of

arriving in her room at 8.20. After five minutes she had raised herself in bed, and the attack was commencing when the electrical application was swiftly called into action, and she recovered in two or three minutes. On the following day I adopted the same plan of visiting her before the time, and it was then sufficient to show her the battery. She never had another attack, and has never suffered from hysterical symptoms since—now more than two years.

In the diagrams before us (drawn from photographs for which I am indebted to Dr Gowers) are two pairs of feet which present a certain superficial resemblance. In

Fig. 10.



F—.

Fig. 11.



B—.

each the inner border is drawn up into the position of a not severe varus. They are the feet of two young women who were in the hospital under my care at the same time, and there is this especial point of interest about them, that F— (Fig. 10), really a case of acute myelitis, had been treated as a case of hysteria; and B— (Fig. 11), really a case of hysteria, came in as a paralytic.

I show you the patient F—. She came into the hospital on April 24th, 1880, on account of loss of power in the lower extremities. The girl is the daughter of a labourer, and has always lived well. Her parents are both alive

and healthy. Two of her brothers have died "in fits," one at four, the other at fifteen years of age. I record this circumstance, but it is really valueless. So-called fits may mean almost any kind of sudden seizure, whether attended by loss of consciousness, or convulsions, or not. The patient herself has never suffered from scarlatina, acute rheumatism, or chorea. The history of her present illness is this:—On December 30th, 1879, she suddenly complained of a feeling of pins and needles in both lower extremities, from the waist downwards, and this continued for a whole day. Next morning she managed to get downstairs, but suffered such great pain in her legs that she returned to bed. In the evening she again got up, and was able at that time to walk with help. The next morning she could not move her legs at all, and could not feel when they were touched. There was no pain or constrictive feeling in the waist. She felt perfectly well the day before the illness began, there had not been any shivering, and she had not, to her knowledge, taken cold. About a week previously she remembers to have had a little pain in the back of her head, and on the morning of the attack she felt giddy. For a week after the attack she was feverish, and very thirsty and restless. She could not sleep at night. It appears, too, that for some time after her attack there was retention of urine, and a catheter was used. Later on there appears to have been some incontinence of urine, as well as loss of control over the bowels, and some not severe bed sores. Two months after the onset she, for the first time, began to move the left leg. Return of power began to show itself in the right leg five weeks later. She recovered sensation in the left leg about six weeks after the onset, and in the right about a week afterwards.

The following note of the patient's state was taken by Mr Broster on admission:—She is a fairly nourished girl, not manifestly anæmic, but her hands are cold and cyanotic, the lips being natural. She feels quite well in herself, has no headache, and sleeps well. There is no paralysis

of the face or upper limbs. The spine presents no curvature, and is free from pain and tenderness. She has no feeling of a tight band about the body. The lower limbs both look blue and are cold, especially about the feet. The left leg in the lower part of the calf measures 10 inches, the right 11 ; the left thigh measures 13 inches, the right 12. She can lift the left heel two feet off the bed, and can bend the left knee, but on the right side she can only just make the faintest attempt at flexing the hip-joint or ankle-joint, and cannot flex the knee-joint at all. The left foot is not distorted, but the right foot is in a position of talipes varus ; it can be everted by external aid, though not by the patient's volition. The foot cannot, however, be extended forcibly. If an effort be made to do this, the tibialis anticus becomes quite tight. There is no rigidity of the joints. The patellar tendon-reflex is absent on the right side, and notably deficient on the left. There is no ankle clonus on either side. The cutaneous reflex from the sole of the right foot is absent altogether, and is only slight in the left.

When admitted, the muscles of the left leg reacted to induced currents of somewhat greater strength than those capable of causing the contraction of healthy muscle. On the right side, however, whilst the anterior tibial group of muscles required considerably stronger currents than in health, the peronei did not react to the greatest strength of Stohrer's battery. The cutaneous sensibility did not appear to be defective in either leg. The action of the bladder as well as that of the rectum was normal. The catamenia had never been established.

After a week the following result was obtained from electrical examination :

Right side (faradaism).—In the vastus internus (motor point) no reaction was obtained. The tibialis anticus muscle responded nearly normally. The peroneus longus did not react to the strongest current which the machine could give.

Left side.—The vastus internus (motor point) required a current of 10° (in the machine employed 20° is the weakest strength, 15° that required normally). The anterior tibial group and the peronei required 12°.

On May 5th the patient found that she could lift her right heel about two inches off the bed, and just flex her right ankle-joint slightly, but she could not bend the right knee. Examined by the voltaic current (galvanism), both left and right antérior tibial group reacted to the interruption of a current from twenty-two cells Stohrer. The left quadriceps extensor responded to thirty cells, but in the right no contraction could be produced by any strength employed.

May 12th.—She can raise the left heel a yard off the bed and the right about six inches. The right ankle-joint can be moved better. She can now just bend the right knee.

November 2nd.—She is able to walk on crutches, the right foot being dragged. As she stands she can move the right lower limb forwards and backwards, and lift the knee from the ground by the iliaco-psoas.

Electrical examination.—Right leg (faradaism). Tibialis anticus is nearly normal. Extensors of the toes and big toe and peroneus longus give no reaction. Quadriceps extensor—no reaction to galvanism. Peroneus longus, twenty to thirty cells slowly interrupted, slight reaction. Vastus externus reacts to thirty cells. Vastus internus shows no reaction.

23rd.—Right leg (galvanism). The vastus internus now reacts to sixteen cells interrupted, the externus to twenty cells, the peroneus longus to twenty-eight cells. There is still no patellar tendon-reflex in the right limb; it is present in the left.

This girl, who was taken ill on December 30th, 1879, went to a hospital on January 9th, having at that time entirely lost the use of both lower extremities. She was at once treated with a daily cold shower bath, and after a week currents from a magneto-electric machine were

applied to her legs daily. The inference is that the condition was referred to hysteria. She says that she could not feel the currents at all for the first three days. They were applied daily for a month. No movements took place in her legs in response to them. Whilst in the hospital she got slight bed-sores on the hips.

At the present time, December 1st, the right foot is in a state of calcaneo-varus, from the paralysed peroneus longus and calf muscles being unable to antagonise the tibialis anticus. The case can hardly be considered a pure example of anterior acute polio-myelitis, as the disorder of sensation, paralysis of bladder and rectum, and formation of bed-sores showed that the mischief was not limited to the anterior cornua, but that the sensory grey matter was also rather extensively involved. But the disorder of sensation and nutrition (as well as the paralysis of bladder and rectum) cleared off, and the condition left is that of a case of infantile paralysis.

In this case the electrical examination alone is sufficient to exclude hysteria. Duchenne wrote that the electro-muscular contractility was normal in hysterical paralysis. I have found exceptions to this, which, however, is doubtless the rule. Occasionally there is a distinct, although slight, lowering of electrical excitability in the muscles in hysterical paralysis, where this has been of long duration. The lowering of excitability applies to both forms of electrical excitation, and in that respect differs from the "reaction of degeneration," where the decrease in faradaic excitability is usually accompanied by increased excitability to the voltaic current. We *never* see in hysteria the various muscles of one limb showing differing degrees of abnormality in their response to faradaism, from a condition of total absence of reaction in some, to nearly a natural state in others. Moreover, in hysteria, according to my observation, applications of electrical stimulus (and especially of the voltaic current) on one or two occasions usually suffice to restore the natural excitability of the muscles

(equally in all) which has simply declined through disuse. A difficulty can only arise where the observer has but one opportunity of testing the electrical condition, and it is then quite possible to occur. It must be remembered that, as a distinct lowering of faradaic excitability almost invariably signifies organic change in a nerve trunk or centre, a diagnosis of hysteria can never safely be arrived at whilst that condition persists. On the other hand, I need scarcely remind you that the preservation of a completely normal faradaic excitability in the muscles of a limb does not show that that limb is not paralysed. It would be hardly necessary to refer to this, but that I once heard it stated in a court of law that the plaintiff was certainly not paralysed, since the muscles of the limbs contracted to electrical currents! In cases of paralysis, it is only when the integrity of the grey matter of the anterior horn is disturbed, or when there is some lesion of the anterior root or trunk of the nerve, that you find decided loss of electric excitability. You frequently meet with complete paraplegia and yet all the muscles will respond normally to electric currents.

As regards the patellar tendon-reflex, I have never seen it lost in a case which proved in the sequel to be hysterical, although I am not prepared to deny the possibility of its absence in such circumstances, if the quadriceps muscle has suffered much from lengthened disuse.

The other patient, B—, æt. 24, came in unable to stand without support. In trying to walk between two persons the left foot was seen to be in a position of varus, as shown in the diagram, and whilst lying in bed it occupied the same position, although this was then not quite so determined.

The following notes of her case were taken by Dr Beevor, Resident Medical Officer of the hospital:

Lydia B—, æt. 24, single, came into the Memorial Wing of the hospital on May 12th, 1880. She had experienced much trouble and anxiety at an early age, having

been forced by circumstances to occupy a position of great responsibility when she was only seventeen. She is an orphan, her father having died from phthisis when she was an infant, and her mother about two years ago, from heart and "brain disease" (hemiplegia). A brother has phthisis. There is no insanity in the family. The patient describes herself as having been quite healthy up to twenty years of age. She has never had scarlet fever or rheumatic fever. Her present illness began two years ago, with pains starting from the nape of the neck and shooting over the top of the head. After this she had pain in the spine and the lower half of the left side of the back, for which she took chloral. She then had pain in the left arm and leg and along the left side. About three years ago she was locked in a room, and, in consequence, had a fit. According to her own account she lost consciousness, struggled violently, but did not bite her tongue. Two attacks occurred within an hour. About three months after this she had another fit, in which she again lost consciousness, but was not convulsed. After being ill for about twelve months, during eight of which she stayed in bed, she got better, but the pain did not leave her entirely. During this time she had about fifteen fits. In April, 1879, she again had pain in the left ovarian region, which shot round her back. This prevented her from walking. Then there was pain, too, in the nape of her neck and back of the head, and in the left arm and leg. In June she was laid up for six weeks, then got about till September, when she became much worse, and in December was again laid up. She then began taking chloral, and this has been continued. Since December she has been getting worse, and has had about three fits each month. The fits are not always preceded by any warning, but sometimes before she is attacked, her tongue feels too large for her mouth. She may have headache too, and pain in the right side of the face and head. There is also sometimes pain in the left ovarian region, shooting up to her head. She began to

menstruate when she was fifteen, and continued regular till she was twenty. Since this time she has been irregular, an interval of two months sometimes occurring between the periods. When the patient was admitted she was in an excited state, praying to have chloral given to her in the afternoon and evening; none was given. On the following morning she was quieter, but said she had not slept until six a.m. Her condition on admission was as follows:—She is rather thin and pale, but not, apparently, very anæmic. She complains of pain in the head and neck and about her body. There is no curvature of the spine. The column is tender on pressure in the upper cervical and lumbar regions. The face is symmetrical. The upper limbs present no difference in size, and with each hand there is a grasp of five kilogrammes. The lower limbs are not wasted. The right thigh measures 13½ inches in circumference; the left 13¼ inches. The right calf measures 10½ inches; the left 10¼ inches. The patient as she lies can lift the right heel about a yard off the bed, and the left about a foot. The patellar tendon-reflex is excessive on the right side, and still more exaggerated on the left; but there is no foot clonus on either side. Patient cannot stand without assistance. When supported on both sides and told to walk she takes a very long time to move the left foot, but brings the right foot forward more precipitately. Urged to move quickly her feet double up under her, and get entangled so that she would fall. The left foot in these circumstances always assumes the position shown in the engraving. The cutaneous sensibility is not at all diminished. There are painful points in the district of the fifth nerve and brachial plexus. The electric irritability of the legs is normal, the muscles contracting to a current which excites healthy muscle. On June 2nd in testing the sole-reflex I found that of the left foot normal. In the right, however, for a minute or two tickling was followed by no response. On its being persistently continued it was evidently felt more and more strongly, and great efforts

were manifestly made to restrain the reflex movements. The muscles of the legs became contracted, and drew up strongly. The face was flushed, and exhibited signs of excitement. The legs were drawn up and separated as though with endeavour to get them out of the way of the tickling process. On the 8th June, after menstruating for two days, patient had a fit in the morning. She had felt faint and complained of pain in the nape of the neck and back of the head. Then her head felt as if it would burst, and she felt "very strong" and "as though she was mad." She screamed and struggled very much, and fought a good deal, but recovered immediately when douched with cold water. After the fit she felt weak and giddy and sleepy. She says that before a fit "her nerves beat" and keep her awake all night. Faradisation of the skin of the legs with the wire brush was begun on June 22nd, and continued daily. On June 25th the patient went for a walk with the nurse, and succeeded in going round Queen Square. Next day she walked to the Foundling Hospital and back. She was shortly afterwards discharged, almost entirely recovered.

About a year ago I admitted a young woman on account of paralysis in all four limbs. She was described by those who sought admission for her as a case of hysterical paralysis. Seven months before she came here the patient had complained of pain in her back, side, and head. The pain was very bad on the vertex. Next she became very weak on her legs, and moved them in an incoordinate manner. At this time there was great weakness of the right hand, and the right side of the face was paralysed. Soon she became unable to walk at all.

A month after she had begun to be ill this patient had a fit, and this had been repeated on five or six occasions before her admission. I have little doubt that it was the occurrence of these fits, associated, as they were, with paralytic symptoms, confined at first to the lower extremities, which gave rise to the diagnosis of hysteria in this

instance. The apparent want of connection between the two symptoms would be suggestive (unless the possible existence of multiple lesion were insisted upon) of the inconsistency of certain forms of hysteria.

When we came to examine this patient with the ophthalmoscope (which we never omit doing), it was found that she had optic neuritis. This fact, of course, immediately carried us a long way on to a diagnosis of serious brain lesion. Moreover, in addition to this, she had a bed-sore, and the presence of this alone was significative of organic disease.

Let me call your attention to the note respecting the state of the muscles of this patient's legs on admission. "Lower limbs powerless; not the slightest power of movement; *no wasting*, no rigidity. No difference between the two limbs. The muscles respond to faradaic currents of somewhat higher strength than is required normally." (As the case advanced there was much wasting, and the limbs were rigidly drawn up). I shall have occasion to refer to the question of wasting of muscles and its diagnostic value. For my present purpose it is only necessary to add that this patient grew worse and worse, that in spite of the most diligent attention her bed-sores extended, and that she died some months after admission.

The autopsy showed basilar meningitis and spinal meningo-myelitis, the cord being disorganised and softened throughout. The patient, it should be said, was the subject of inherited syphilis.

One feature of this case was of especially great importance. As a rule, the pain in the back, which is associated with spinal meningitis, is chiefly felt when the patient turns from one side to the other. But, in this instance, if the patient's account is to be accepted, the description of pain was exceptional. The following note was taken on her admission:—"On inquiry it seems that throughout her illness she had suffered from pains in the back that would come on suddenly and leave her whilst she walked

about the room. At no time during her illness has she had pains in her back which were made worse by sitting up or by movement." Now it is possible that the inflammation of the membranes of the cord occurred as a sequel of the myelitis, and had not begun when the note was taken. I do not know that this was so, but simply suggest the possibility. It is well to remember the point, because, in the case described, I can well understand that the character of the pain was very liable to mislead as to its nature. It would divert the attention from the idea of meningitis. Whilst upon this point I will refer to another case, which may be conveniently contrasted with the last.

In 1872, a married lady, æt. 26, was sent to me by a medical man in the country, on account of loss of use of both lower extremities. She had been married three years, and had two children, the youngest being six weeks old. In early life her back had been "weak," and she was made to lie down a good deal. Her health had generally been very good. There was a history of gout in the family.

The following note was taken :—Fourteen months ago, after getting very wet in a ride, she was seized with rigors and went to bed. A few days afterwards she began to have pain in the lower part of the dorsal region of the spine, and this spread all over her arms and legs. She was not confined to bed. Off and on, ever since, she has had these pains about her. There has not been any swelling of her joints.

Her mother told me that in June last she was complaining exceedingly of her back—always the lower part of the dorsal region. At that time there was nothing wrong with her legs. She had, at times, paroxysms of flying pains about her head, face, and arms. The pains were always most severe at changes of weather. She has never, she says, gone a fortnight, or even a week, without pains since she was attacked fourteen months ago.

Three months ago these pains were especially bad, and it was then that her legs began to feel weak. There was nothing wrong with the sensibility. A month later she was liable to be attacked with great pain from her hips to the feet, accompanied by sudden loss of power, so that she was obliged to lay hold of something. At this time she was approaching her confinement, and one morning, within a month of it, she lost power to such an extent that she had to crawl upstairs on her hands and knees, and three days afterwards became completely powerless in her legs. At this time she could not lie on the left side on account of pressure of the gravid uterus.

Nine days after her confinement, her toes, she says, became perfectly dead numbed, and she could feel nothing with them, not even pinching. At this time she could just slide her feet about, but could not lift them.

When I saw her she lay in bed, and averred that she was unable to move her legs at all. She could move some of her toes a little. Tickling the soles produced no reflex action. There appeared to be almost absolute loss of sensation in the legs; but on inquiry, she said that she was aware of the position of her feet. She had never had any feeling of waist constriction. She could not raise her body in the least without using her arms.

On examination there was tenderness on pressing upon the spines of the dorsal vertebræ from the fifth downwards. There was nothing wrong with the arms, bladder, or bowels, and no tendency to bed-sores. The legs looked thin, but there were no signs of muscular atrophy. I applied electrical tests. There was a little delay in getting the muscles to contract by induced currents, but after application for a few minutes of strong currents, they reacted to a much lower power, to a strength, indeed, which did not at first influence them. The reaction to interrupted voltaism appeared normal. The induced currents were employed vigorously for about five minutes. At first she could not feel them, but after a minute or two she complained of their making her feel ill. The

muscles of the legs generally were well exercised by the currents. Five days afterwards I saw her again. The power of movement had much improved. She could lift either foot from the couch, and could dorsal flex them. I noted now that elaborate and prolonged tickling was felt merely as touch on either sole, although she stated that she was naturally very ticklish. I now faradised all the muscles of the lower extremities energetically. The process gave her evident pain, and there was a great deal of voluntary movement. Two days afterwards she walked across the room, supported by two persons. Faradaism was again applied very freely and sharply. Another two days passed, and the patient was able to walk downstairs by herself. She returned home in a day or two afterwards quite recovered, and has had no recurrence of the paralysis. In this case the exposure to wet and cold, followed by rigors, suggested spinal meningitis, and made the diagnosis somewhat doubtful at first. Where there is a history of injury, the same kind of doubt may easily arise.

Three or four years ago I saw Mrs. B—, a young married lady, æt. 22 (one child), who had just come from abroad. A year previously she had slipped and fallen down twenty-five steps, striking her spine between the middle and lower part. She is said to have been unconscious for ten minutes. She then got up, as she told me, and felt a tingling and pricking in her legs. She walked upstairs, dressed, went out to dinner, and that night slept quite well. Next morning she found herself unable to move her legs, and had not the least feeling in them. She was in great pain in the back, and outer part of her thighs. For forty-eight hours she was powerless in her legs. Then they were rubbed and she recovered power, though whether completely or not she does not know. But she could move her legs about to new postures.

For three months she was kept to her bed, except on one occasion, five weeks after her fall, when she was allowed to get up. But she had so much menorrhagia that she

took to her bed again. Whilst lying there she lost muscular sense downwards from the waist. She had no idea of the position of her body from the waist downwards, but if touched she could feel perfectly well. During the time she lay in bed the catheter had to be employed on one occasion only, otherwise she never suffered from any difficulty with the bladder or the bowels. Nor had there been any feeling of waist constriction. At the end of three months she got about again, and was soon able to walk two miles without fatigue. Six months after the accident she gave birth to an infant, which she nursed perfectly well, and was in excellent health for two months. After this, from time to time, always after fatigue, she got pain in the back where she had been hurt. On the voyage to England she had been quite well, except a little of this pain, until a week before I saw her. She had been up all one night with her baby, who was fretful, walking up and down, and carrying it in her arms. This brought on great pain in her back and pain all down the legs. She lay down all day, and it seemed to pass off. Next day she found her legs would not go where she wanted them to go. They kept crossing one another. This was on a Sunday. She went to church, but coming out found she could not command her legs. Two hours later, when trying to rise from her chair, she found she could not move. She was carried upstairs. She could not feel a touch, or pinching, or hot applications. She was very cold in her body, as well as in her legs. There was no starting of the limbs.

I found, on examination, that there was no irregularity in the spinal column, but she complained of tenderness when the eleventh and twelfth dorsal spines were touched. The faradaic excitability of the anterior tibial muscles was normal. Her general health seemed good, but she was of a nervous temperament and of somewhat delicate constitution. It was said, indeed, that for three years at an earlier period she had suffered from disease of the hip-joint, but I had no means of verifying this. She was

advised to lie in bed, and in a few days all pain had gone, and she felt quite well.

A few weeks later she was at the seaside and swam one day for half an hour out to sea, besides going through much fatigue with picnics and walking. For some time she remained perfectly well. Then, after unusual fatigue, she got a return of menorrhagia, but still persisted in going about and swimming, till one day she "collapsed." Then her feet and hands became icy cold, and for hours it was impossible to warm her. At first she could not walk. She did not lose sensation in her legs nor control of the bladder. In a day or two she succeeded in walking again, but could not coordinate her legs, and was unable to go up or downstairs. She soon recovered, and when I saw her a month later there was nothing the matter with her legs. She then complained of difficulty with her breath occurring just before menstruation. She had, whilst talking, a hysterical gasping respiration. I have since lost sight of this patient.

A case which first came under my notice in March, 1879, has presented such difficulties in diagnosis that I think it may be mentioned with advantage.

Miss —— ——, æt. 16, was seen by me in consultation on account of loss of power in the left leg. It seems that she was well until the previous July, when, in walking, she began to trip with the left leg. This gradually increased so that she had come to walk with difficulty. She told me her leg would not move properly, it dragged and did not bend. She sometimes caught the foot and fell.

She had not suffered any pain. There had never been anything wrong with the left arm. She began to menstruate between twelve and thirteen years of age. The catamenia had always been natural and regular. She is naturally, her mother describes, very nervous, and laughs a good deal. In walking I observed that the movement was a good deal like that which is made with a limb when it is "asleep." The leg was swung round in an arc, the

knee not being bent, and the point of the foot being dropped. She had no power, she said, to dorsal flex the foot.

On examination I found the legs well nourished. The skin was cold, and there were chilblains on the left foot. There was nothing wrong with the function of the bladder or rectum, and no constrictive feeling in the waist. In both legs I found the patellar tendon-reflex greatly in excess. In both, but especially in the left, there was well-marked foot clonus. In neither was there any reflex action on tickling the sole. The faradaic excitability of the muscles was normal.

It is only, so she told me, since her illness—*i. e.* since she began to trip in walking, that she has ceased to be ticklish in the soles of the feet. She used to be very ticklish. Before she lost power she used to complain of tenderness in the spinal column. When she bent her head down she felt that something was dragging her back. Sometimes she felt this on going to bed at night, at others in getting up in the morning. It was not constant, and was a sort of aching. Twice it has happened to her when in a warm room to feel giddy, and as if there were a weight in her head, and then she could not walk at all. Her mother told me that since this began, *i. e.* about eight or nine months, she had never on one single occasion walked properly, and that she had remained exactly as I saw her for two months. At the commencement she complained that her leg trembled, and the knee fell backwards. Percussion on the spinal column gives no sign of tenderness.

There has never been anything wrong with the bladder or rectum, and she has never complained of waist constriction. Her medical attendant some months since found some tenderness about the tenth dorsal vertebra, for which he blistered her.

I did not express a positive opinion as to the nature of her case. She was afterwards taken to two distinguished consultants, who both thought the case one of

hysteria. I heard, a twelvemonth later, that she had not improved in her walking, and that her medical adviser (who was not visiting her) sometimes saw her about his neighbourhood clinging to the arm of an attendant and hobbling along.

In March last (a year after my first examination) I again saw her in consultation with a colleague. At this time, whilst the right calf measured $12\frac{7}{8}$ inches, the left was only $11\frac{7}{8}$. With the left leg she could not feel quite so well as with the other. A purple discoloration was noted on both legs near the ankle. The feet were cold, and there were chilblains.

On applying induced currents, we found that the excitability was slightly lessened in the tibialis anticus of the left leg as compared with that on the right side. Examination of the spinal column showed a slightly abrupt change of level (as though from the prominence or sinking of a vertebra) at the lower part of the dorsal region. Below the level of this there was a decline of cutaneous sensibility.

We recommended that a Sayre's jacket should be applied. This was done. I have since heard that she is somewhat improved.

This case may prove to be of an hysterical character, and if it be so, it furnishes a good illustration of my remark that you cannot cure a case of hysteria so long as you have any serious doubts as to its nature. The girl has been ill three years. If the condition be one of hysteria she will be cured suddenly some day, probably by a bone-setter, and my eminent colleagues, as well as myself, will be covered with ignominy!

Such symptoms as this girl presents might be caused by slow compression of the spinal cord in the dorsal region. One would have expected, however, that a cause of compression sufficient to produce the very considerable loss of power seen here would, in accordance with Brown-Séquard's observation, have been attended with considerable anæsthesia of the opposite limb. The anæsthesia, such as it is,

occupying the same side as the motor paralysis, is suspicious, though not of itself conclusive. Suspicious also is the slight lowering of electrical excitability in the muscles on the front of the leg; but I did not have the opportunity of testing this again, as I should have done, on some other occasion. The exaggerated reflex and foot clonus are, as I have already pointed out, quite consistent with the case being purely of an hysterical character. It is, I think, very important to remember this. As regards the slight anatomical peculiarity described, you will find all sorts of peculiarities in the spinal columns of healthy people if you look for them. I should not, therefore, count much upon that alone, but when associated as it is with symptoms which *may be* due to slow compression of the cord one is bound to allow the possibility of some Pott's disease of the vertebræ lying at the bottom of the case. It was for that reason that we recommended Sayre's jacket. I should add that the absence of sole-reflex in the circumstances points strongly in the direction of hysteria.

I lately saw, in consultation, in the course of a week, three ladies, each of whom had been thought to be suffering from hysterical paralysis, although in two of the cases there had been some difference of opinion on this point on the part of those who had seen the patient.

The first case was that of a lady, æt. 42, who had been confined five months, and for two months previous to the birth of her child had complained of pain about one of her shoulder-blades, which had gone on increasing ever since. For the last six weeks she had gradually lost all power in her legs. More than one consultant had expressed the opinion that she was suffering from hysteria, and she had been treated accordingly, being made to get up daily, with help, and receive a cold douche bath. There was no doubt that she had shown symptoms of hysteria, apart from the pain and loss of power which had been ascribed to this cause. I found her lying in bed on her right side, unable

to move her legs, which occasionally were spasmodically contracted. They presented no abnormal appearance, not being flabby, nor at all wasted. Sensibility was not affected to any appreciable extent. When I dorsal-flexed the left foot, there was strongly marked foot clonus, and the tendon-reflex was generally very excessive. Coming now to examine the spinal column I found a sudden angular curvature about the sixth dorsal vertebra, which was tender when touched. Pain was described as being dreadful in this neighbourhood, and extending along the ribs from this point. The bladder was powerless, and the urine ammoniacal. There was a commencing bed-sore on one buttock. The prone position was ordered, and opium administered. Four days afterwards she had rigors, and her temperature was 102°.

Ten days later I saw her again. She had improved somewhat (doubtless from the recumbent position) as regards the paralytic symptoms, and had acquired some command over the bladder, when she was seized again with rigors, fever, and delirium, and died a few days afterwards, apparently from pyæmia. The case was one of slow compression of the cord from disease of the vertebræ, which was probably due to malignant growth. I can quite understand that earlier in the case there would be great difficulty in diagnosis, owing to the presence of strong hysterical symptoms, and the absence of any but the subjective symptom of organic disease, furnished by the patient's complaint of pain. But when I saw the patient, the signs presented left no doubt whatever. The ammoniacal urine and bed-sore (accompanying paraplegia) afforded positive evidence of organic disease affecting the spinal cord.

The second patient was a young lady of about twenty-five years of age, who three months previously had been attacked with feverishness, aching in the legs, and pain in the back. The next morning she was weak on her legs, and complained of malaise. On the following day she was para-

lysed in both legs. There had been nothing wrong with the bladder, no loss of sensibility, and no cramps.

I found the patellar tendon-reflex absent in each limb, and the muscles of both thighs thin and flabby. On percussing the vastus internus of either limb, there was no contraction. On the right side the tibialis anticus and peroneus longus muscles reacted to percussion, but not in the left leg.

Powerfully supported on either side, she could maintain the upright position, and walk after a fashion, there being some power of lifting the knees by the iliaco-psoas muscle. The feet, however, were both dropped. The cutaneous reflex from the soles was good on each side.

Now it happened that this lady was under the care of a gentleman who is well versed in neurological medicine, and in the scientific application of electrical currents. Anything, therefore, that I learned from him as to the electrical reactions could be depended upon.* I heard that when he had last applied electrical tests (some weeks, I think, before I saw the patient), "reaction of degeneration" had been typically marked in the muscles of the legs, which would only show very slight action to strong induced currents. The reaction of degeneration, and the varying degrees of response to mechanical and electrical stimulation, showed conclusively that the case was not one of hysteria. There can be no doubt that it is one of anterior polio-myelitis, in which there may probably be recovery to some, but not to a perfect extent.

The third case was one of an extremely interesting and important character, for the opportunity of examining which I have to thank my friend, Dr Playfair. The lady was twenty-five years of age, and I saw her last October.

Her history, as I learned it at the time, was briefly

* The accurate testing of electrical reactions is a more difficult matter than is commonly supposed. It is not safe to attach importance to alleged alterations in reaction, unless these are reported by one who is known to be a competent observer.

this:—There had been more or less uterine trouble (chronic endometritis) for four or five years. Three and a half years ago, after the death of her husband, she had suffered from pelvic cellulitis, and the formation of an abscess, which discharged through the bladder and vagina. Two years and a half ago she gradually lost the use of her legs, the paralysis being, in a few months, complete. It was preceded by cramps, and some loss of sensation. Nine months ago she began to get a similar loss of power in the left arm. The least attempt to raise her, I was informed, caused faintness and sickness, and bending the thigh upon the trunk occasioned great pain.

The patient was in bed, lying on her back, and unable, according to her account, to move her legs at all. She was asked to try, but failed to produce any movement. The feet were dropped. The legs were very thin, presenting an appearance of general leanness rather than of a specific muscular atrophy. There had never been any tendency to bed-sores. There was no spinal curvature or marked tenderness, but the patient said she had had pain in the lower half of the dorsal region of the spine. A catheter had been employed to empty the bladder for two years. The urine had always been acid. The use of the catheter had, it should be said, arisen in connection with the local bladder troubles from the abscess.

I found the patellar tendon-reflex present, though not strongly marked, in each limb. The reflex from the soles of the feet was absent, and tickling was not felt. The legs were not contracted, but the joints were rather stiff. After wetting the limbs thoroughly with warm water, I applied strong induced currents to the muscles of the thighs and legs. She said that she did not feel them. There was a certain amount of contraction, but not a free response. There was evidently a good deal of resistance to the current in the skin, which was covered by a thick layer of epithelium, and this was rubbed up in flakes when the skin was sponged vigorously with warm water.

A strong constant current, slowly interrupted, was now

applied to the muscles, and this she felt very much. A return was then made to faradaism, and the currents were now felt acutely, and the muscles responded much more actively than they had done. Moreover, now, when very strong and painful currents were employed to the legs and feet, she used the muscles of her thighs freely, in what was evidently a quasi-voluntary effort to escape the pain of the application.

As regards the left arm, this lay motionless by her side, the forearm crossing her chest. The limb was neither rigid nor peculiarly lax. It was like that of a person who neither aids nor resists movement. When I engaged her attention by examining her gums and throat, and, meantime, held up the arm, it did not drop, but subsided slowly and gradually to the bed when I let go of it.

A strong opinion was expressed that the condition was hysterical, chiefly on the following grounds. The gradual loss of power, with loss of sensation, if it were organic, must have been due to more or less diffuse myelitis, involving grey as well as white matter. The situation of this must have been either in the lumbar portion of the cord, whence the nerves proceed to the lower extremities, or at some point above this. Had it been in the former, the patellar tendon-reflex would have been absent, and the muscles would have lost their faradaic excitability. If it had been above there would necessarily have been by this time descending degenerative changes in the lateral columns, which would have shown themselves by more or less rigidity and exaggerated reflex. Had myelitis been so extensive as to involve the whole of the lower half of the cord in softening, there must have been bed-sores and difficulties with the rectum. The result of the case has justified the opinion which was arrived at by those who consulted upon it. Under high feeding, moral treatment, massage, and faradisation of the muscles (the Weir Mitchell method), the patient speedily regained the use of the arm, was shortly afterwards able to walk to a certain extent, and is now, as I learn, quite recovered.

In this case, as I have seen in many belonging to the class of hysteria, the epidermis, which had arrived at extraordinary thickness, apparently from disuse of the limbs, offered great resistance to the passage of electric currents. In these circumstances a more than usual amount of care in thorough soaking and rubbing of the skin, as well as in selecting the motor point, is necessary in order to avoid fallacies.* This woman's legs were very thin. There is, I think, a very common notion that the existence of wasting negatives hysteria. But a disused limb is of course liable to emaciate. The only way to distinguish with certainty between this wasting and that which arises from change in the trophic centre in the spinal cord, or from disease of a nerve-trunk, is by observing the condition of faradaic excitability and seeking for any qualitative changes on interruption of the voltaic current. I have reason to think that not a few cases of anterior acute polio-myelitis occurring in young females pass as examples of hysterical paraplegia, owing to the precaution being omitted of carefully testing the state of the muscles by electric currents.

Absence of reflex from the sole of the foot is a very constant symptom in hysterical paraplegia. Where this coexists with normal electrical contractility of the muscles of the lower extremities and normal tendon-reflex the case may be looked upon with suspicion, although this is not of itself sufficient to exclude organic disease. Persistence

* As the motor point frequently varies as regards its position from that indicated in works on electro-therapeutics, I am in the habit of testing in the following manner:—The limb having been very thoroughly wetted and rubbed with hot water, and the induction machine arranged to give a somewhat stronger current than is necessary in health, one rheophore is applied to an indifferent part of the body (as *e.g.* the sacral region), and the other is moved about the skin of the limb at the part where the motor point is expected to be found. It will soon be perceived, by comparison, at what point of application the most energetic contraction is excited. The skin is then marked at this spot with a wetted aniline pencil, and the operation is repeated on the opposite limb, as well as for other muscles. The motor points having thus been ascertained and marked, it is easy, by graduating the machine, to discover the weakest current which will cause contraction, and compare it with that required in health.

in the application of a stimulus to the sole will in hysterical cases frequently, but not always, succeed in provoking a response after a few minutes.

Preservation of the roundness and firmness of a limb is often thought to be incompatible with paralysis. This is a dangerous error. In the case of Pott's disease, which I have referred to (the patient died a few weeks afterwards), the lower extremities were rounded, well nourished, and firm to the touch. In cases of localised myelitis of the dorsal region of the cord, followed by descending sclerosis of the lateral columns, there is frequently unusual firmness and tonicity of the lower extremities, which may appear indeed to be models of muscular development. This is important to remember, as hysterical imitations of lateral sclerosis are not uncommon. The most frequent, however, and at the same time the most difficult of distinction, is a condition closely resembling disseminated cerebro-spinal sclerosis.

LECTURE VI

TABES DORSALIS

We had lately the opportunity of examining after death the spinal cord of a man who had suffered for the last year of his life from complete paraplegia. His paralysis was due to destruction of the essential elements of the cord in the mid-dorsal region, brought about by a sharp angular curvature of the spinal column. Sections of the hardened medulla, stained with carmine, which I have prepared, exhibit important changes under the microscope, as you will presently see. But before describing these, it may be well to refer to a few points connected with the anatomy of the spinal cord.*

The spinal cord, I may remind you, consists of an irregular cylinder of grey matter (roughly represented on horizontal section by the letter H), embedded in white substance, much as the wick of a candle is enclosed in the composition. The grey matter is composed of ganglionic nerve-cells and nerve-fibres, and the white substance consists almost entirely of nerve-fibres. Not all, but a very large majority of these fibres, take a longitudinal direction, so that when you cut the cord across they are seen under the microscope as circles of varying diameter with a central dot. This dot is the axis-cylinder (the essential element of the fibre), and the circular line defines the

* The sketch, which is elementary and incomplete, was given at the commencement of the lecture as a simple introduction to the clinical portion. For an admirable account of the topography and secondary degenerations of the spinal cord (to which the writer is greatly indebted) see Charcot's "Localisations dans les maladies de la moelle épinière," Paris, 1878—80.

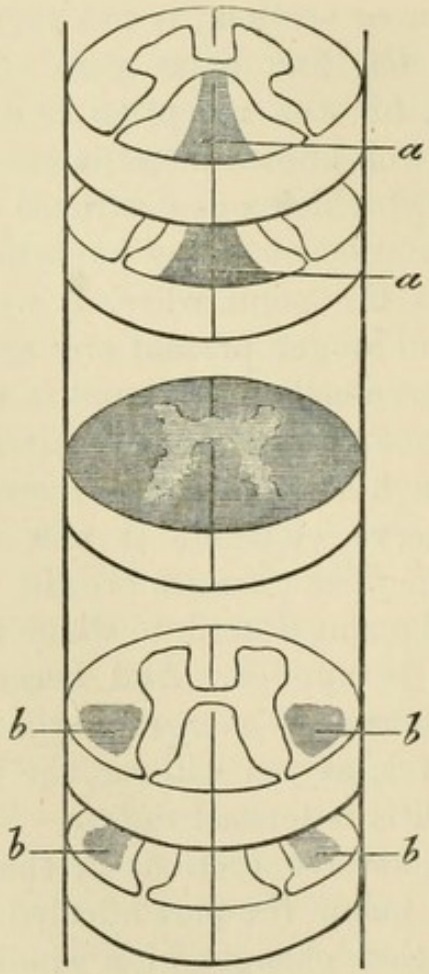
limit of the myeline in which the axis-cylinder is enclosed. But besides the nerve-fibres and cells there is a quantity of interstitial fibrous tissue in the cord forming a network, the use of which appears to be to transmit the capillary blood-vessels to the substance of the cord, and to hold the more delicate nervous elements in their places. This connective tissue surrounds the cord outside beneath the pia mater, and penetrates into its substance at various points, breaking up into meshes of increasing fineness, in which the nerve-fibres and cells are included. Indeed, its office as it appears on cross-sectiom, is not very unlike, in some respects, that of the fine brass wire which forms little *cloisons*, or cells, for the reception and support of the enamel upon the well-known metallic vases of Japan.

Now, in the cord which was destroyed in its mid-dorsal region you will observe some very important changes. In the first place, at the point where it was most damaged the nerve-fibres no longer present any axis-cylinders, and the ganglionic nerve-cells are altered in appearance; and in the second place the connective tissue has increased enormously, so much so, indeed, that instead of being subsidiary to the nerve elements it now tends to monopolise the section. The changes are the result of chronic inflammation of the spinal cord, to which the term myelitis (μυελός, marrow) is applied. And because grey matter and white are indifferently merged in the destructive process, which involves, as you will see, the whole transverse section, this myelitis is termed *diffuse*.

If we make transverse sections of the cord at various points above and below the part affected with this *diffuse myelitis*, we find that changes of a similar kind, as seen under the microscope (especially increased growth of connective tissue and destruction of nerve-fibres), extend to each extremity. In the cervical region, however, as well as in the lumbar region, they are no longer diffused over the whole section, but are concentrated in certain definite tracts. Downwards these changes are seen to occupy, in the lumbar region, the posterior portion of each antero-

lateral column (*b*, *b*); upwards they are limited, in the cervical region, to two bands which border the posterior fissure (posterior median columns or columns of Goll, *a*, *a*). If we make various sections we find similar appearances in each. The term "sclerosis" is applied to this alteration of tissue; and because the changes run in different bundles of fibres taking a longitudinal course, the condition is called "fasciculated sclerosis."

FIG. 12.

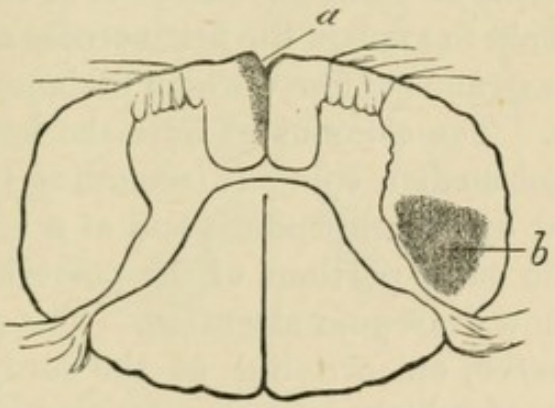


A similar effect is well known to be produced by a destructive lesion of a certain portion of the brain-substance (I need not here describe it in detail), from which degenerative changes of the kind described may be traced downwards through the crus cerebri, pons, and medulla oblongata, into one of the anterior pyramids. The fibres of the

anterior pyramid for the most part, as is well known, cross over to the opposite side of the cord, but it has been shown by Flechsig that a varying proportion of them descend (without crossing) on the same side just at the inner edge of the anterior column bordering the anterior median fissure.

In Fig. 13, which will stand for a section of the cord in its lower cervical region, *a* represents these *direct* pyramidal fibres, and *b* the pyramidal fibres which have crossed over from the left side of the brain to the right side of the cord. Now, it is important to remark that whilst (as shown by

Fig. 13.



this latter instance, as well as by the former (Fig. 12), chronic myelitis or sclerosis follows a *descending* course in a system of fibres in the antero-lateral columns, it takes an *ascending* course in the system of fibres constituting the posterior median columns. Why should this change —systematic myelitis it is sometimes called—travel downwards in the one system and upwards in the other? Türck, who, I believe, first described these changes, explained their occurrence by reference to the law established by Augustus Waller in regard to degenerations of nerve-trunks after section. Waller, it will be remembered, made the extremely important observation that if the anterior root of a spinal nerve were cut through, the nerve-fibres thus separated from the cord degenerated all the way down to their remotest periphery. If, however, the posterior root were cut through between the cord and the ganglion upon

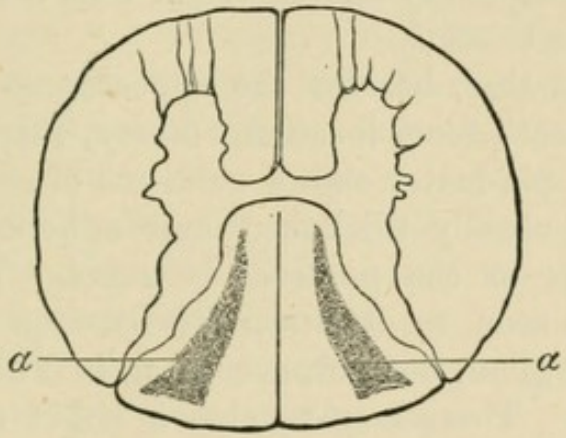
the nerve, it was now not the peripheral but the central portion of the nerve which degenerated—that part which lay between the point of section and the cord. The spinal ganglion thus appears to perform the office of a trophic centre for the sensory nerve, whilst the spinal cord does the same for the motor nerve. According to this view, then, the fibres in the antero-lateral columns, which have been picked out by the degenerative process, would have their trophic centres above, and those of the posterior median columns would have theirs below. You must remember, however, that a centre of this kind is hypothetical, and we cannot indicate its position.

For the rest the mode of development of the spinal cord will probably help to explain the distinctness and independence of various strands of fibres which pathological anatomy thus indicates. The columns of Goll, the lateral columns, and the anterior median columns (according to Pierret) are each developed by separate points and at a somewhat later period than the outer portions of the posterior columns to which I will now draw your attention. The posterior root of a spinal nerve, on arriving at the cord, divides into two parts, one of which immediately enters the posterior horn and the other (the larger) consists of fibres, which take a more or less vertical direction on their way to the grey substance. It would seem, indeed, that these fibres of the posterior roots constitute to a considerable extent the outer portion of the posterior column—the part which lies between the column of Goll and the posterior horn. In consequence of this the part has been called by Charcot and Pierret the "posterior root zone."

Now, it is not only in those columns which you have seen picked out by degeneration (Figs. 12 and 13) that fasciculated sclerosis may occur. It is also found in the posterior root zones which I have just described. The same kind of change, with the same tendency to travel, is found to constitute the essential lesion in locomotor ataxy, and to have its seat in the posterior root zones. In Fig. 14 you will see marked (at *a*) the site of the sclerosis of

the posterior root zones, which is characteristic of locomotor ataxy. But as a matter of fact you must not expect to meet with the change limited, as it is in the drawing, to this situation. You will much more often find in cases of locomotor ataxy that in the upper lumbar and dorsal regions the change involves the whole of the posterior columns from one horn to the other, whilst in the cervical region it may be almost entirely confined to the columns of Goll. It is to Charcot and Pierret, whose investigations

FIG. 14.



into the morbid anatomy of locomotor ataxy are widely known, that we owe the explanation of this apparent anomaly. They have suggested, and the explanation is generally accepted, that the sclerosis of the middle portion (columns of Goll) is really a secondary degeneration consequent upon the systematic myelitis beginning in the posterior root zones of the lumbar region—a sclerosis in fact secondary to a sclerosis. I have called your attention already to the sclerosis of the columns of Goll, which takes an ascending course as a consequence of diffuse myelitis of the dorsal region, and here we have again a degeneration in the same columns, taking a similar ascending course as a result of a destructive change in the outer part of the posterior columns. This change—viz. the sclerosis of the posterior root zones—which is, as I have said, to all intents and purposes the essential lesion in locomotor ataxy, is generally supposed to be a primary one. Whereas, as we

have seen in the two instances adduced, the change in the columns of Goll is evidently apt to occur as a secondary degeneration, the alteration of similar character in the posterior root zones is considered to be independent of a previous lesion—to be, in fact, a primary change. (In both cases, I should say, it is supposed that the change commences in and is propagated by the essential nerve element, not by the connective tissue.) There appears to me, however, to be some clinical evidence to suggest the probability that the change in question is at least not unfrequently a secondary affection, and that it commences in the connective tissue.

Let me add that, besides the appearances described as found after death from locomotor ataxy, there are usually others. The pia mater shows evidence of chronic inflammation; it is usually thickened and adherent to the diseased portions of the posterior columns. The posterior roots also are seen to be atrophied between the cord and the spinal ganglion, but not, according to Vulpian, beyond the ganglion. There may be also a slight and occasionally a considerable amount of sclerosis in the posterior and outer parts of the *lateral* columns. Other changes, too, are found, upon which I need not now dwell.

These, then, are probably the most important anatomical features of a disease for the practical recognition of which we have been first indebted to Romberg, who gave a graphic description of the disease under the name of Tabes dorsalis, and later to Dr. Duchenne (de Boulogne), whose monograph upon it must ever retain its place amongst the classics of medical literature. For the symptomatology of the affection in its typical form, one cannot do better than quote from the definition given by Duchenne in the last edition of his work.* "Progressive locomotor ataxy," he says, "is characterised—1. By an overgrowth of connective tissue (sclerosis) of the posterior columns of the cord to a greater or less extent. 2. During an early period by a paralytic strabismus (paralysis of one or more

* 'L'Electrisation Localisée,' Paris, 1872.

motor nerves of the eyeball) ; by atrophy of the optic disc, and morbid conditions of the pupil ; by special pathognomonic pains, lancinating, lightning-like, tearing, deep-seated, with cutaneous hyperæsthesia of the skin circumscribed to the seat of pain—each pain not lasting more than a few seconds. 3. During a second period by disturbance of co-ordination and equilibration equally characteristic, localised in the lower extremities, extending to the upper limbs during this period, aggravated by darkness, but occurring even when the patient can help himself with his eyes. 4. By a feeling of weakness whilst standing upright and walking, which is manifested at the same time with the troubles of motor co-ordination and equilibration, and which contrasts with the integrity of the force of individual movements. 5. By loss of sensibility in different degrees (analgesia and anæsthesia of the skin and parts beneath it—that is to say, of the nerves, muscles, osseous and articular surfaces), anæsthesia which increases the disorder of co-ordination and equilibration, but is not necessary for its production. 6. By functional troubles of the genital organs, of the bladder and intestine (impotence or satyriasis), micturition and defæcation difficult or impossible. 7. Lastly, by the slow and fatally progressive development of all these morbid phenomena, and by their manifestation usually in the order described."

To these must now be added the symptom which we owe to Westphal—the most frequent and characteristic of all—the absence of the knee-phenomenon, along with preserved muscular integrity of the quadriceps extensor cruris.

Now it is to be remarked that the *αταξια* (*disorderliness*) from which the disease has acquired its most commonly employed title, whilst it may be a very prominent feature, is by no means a constant one. It is often absent for a considerable time, and it may never occur, or it may be so slight as to be recognised with difficulty. This name therefore tends to mislead.

The term Tabes dorsalis, first applied to the disease by

Romberg, seems to me better, because it may represent, whatever the original idea connected with the term may have been, a broad physical feature observed after death —a shrinking or wasting of the cord, especially in its dorso-lumbar portion. So you will understand that when I speak of "tabes dorsalis," or "tabes," I mean to signify the disease more commonly known as locomotor ataxy.*

Of this disease I propose to present several examples. On the present occasion the patient who is shown is a typical iustance. Others, which you will see later, have been selected as representing various phases of the disease, which are liable to give rise to more or less error in diagnosis. Tabes is more widely spread than is commonly supposed, and the mode in which it is often masked constitutes one of the most interesting subjects in pathology.

The patient, C. B—, aged forty-one, came here last December, with symptoms of locomotor ataxy, and although he has in some respects improved, his disease cannot fail to be recognised at a glance. In walking his feet are widely separated and the toes turned out; and it is evident, from the serious attention which he gives to the process, that it is no longer entirely automatic with him. He never takes his eyes off the ground; his steps are uneven in length, and the natural rhythm of walking is wanting, so that, even without seeing him, if you heard him walk along a passage you would recognise the peculiarity. The fore part of the foot is flexed sharply upwards, sometimes even more than once, whilst the leg is making the step, and then as often as not the heel comes upon the ground with a stamp. This gives a look of insecurity to his walk, which is greatly increased when he turns round. If he places the feet close together, thus narrowing the base upon which he stands, and closes his eyes, he totters to and fro, and would fall but for help.

In picking up an object from the table the hand is usually first projected past it, and then you see one or two

* The term "tabes spinalis," which has been suggested, would be still better, but it is not commonly employed.

fingers suddenly over-extended when you would expect them to be flexed. When the hand is closed, as often as not he has failed to grasp the object. There is some cutaneous anæsthesia of his legs and arms.

For several years he has suffered from what he calls "rheumatic" pains—pains in the legs and arms, and occasionally a dart through the head. He describes the pains in his limbs as "excruciating." They are sudden, and last perhaps a second or two. He has them in paroxysms, which have often kept him awake all night, and they are sometimes so bad that he has cried with them. He has lately had a sudden scalding or twitching sensation in the perineum. At times he has had double vision, and on one occasion there was ptosis of the left upper eyelid, which lasted some weeks.

His legs occasionally give way in walking. When he came here there was not much loss of power in his legs, but they are not so strong as they were. His pulse is quick. The pupils of the eyes are small, and do not contract to light. They do contract, however, during accommodation of the eyes.

There is a total absence of the patellar tendon-reflex in each knee.

Amongst the subjective symptoms of tabes, the first place must be given to the pains. It is customary to speak of these as constituting, along with ocular troubles, the first stage of locomotor ataxy. In my own experience it is a very rare exception for pains not to be present at some time or other in the course of tabes, and they much more often than not precede the other symptoms. It is only right, however, to say that more than one observer has mentioned to me that he did not meet with the symptom with anything like this amount of frequency. A highly-qualified friend writes me from the country, "As to the pains, if inquired for they may be remembered as slight and occasional, or may never have occurred. I only remember one or two cases in which they were well-marked." Another told me not long since that he thought

it open to question whether pains occurred in the majority of cases. The symptom, it may be remarked, is so often attributed to rheumatism at first that the difficulty of obtaining exact information about it is great; and this may to some extent account for the difference of opinion.

Our hope of dealing successfully with a disease is greatly dependent, there can be little doubt, upon early recognition of it. It appears, therefore, to be of great consequence that we should recognise the early pains of tabes, and not confound them with other conditions, as I have good reason for thinking is very often done. Descriptions given of them by patients themselves, who at the time of coming under observation were undoubtedly marked examples of locomotor ataxy, are valuable, and I will quote some illustrations of the kind from my notes.

In one case the first onset was marked by "an electric shock-like pain in the right heel." This patient described his pains as being "of a horrible description, generally confined to the lower extremities, but occasionally occurring also in the middle and little fingers of one hand. At times there was great hyperæsthesia at the seat of pain, and then a slight touch on the spot would start a paroxysm." A patient, A— (whom I show to you, and to whose case I shall again have occasion to refer), describes them as "burning pains—as if he had been scalded—in the left arm, and sharp cutting pains at the knee." W— says that "shooting pains catch him under the sole of the foot towards the toes, and dart up the leg to the knee. Occasionally they go up the thigh. They are like a sharp pinch, which is off directly." At another time he spoke of "gnawing pain in the left thigh, especially in the inner part. The pains," he says, "occur daily and have done so for six weeks; sometimes they will go on for the whole day and night." He gets two or three times, in rapid succession, a "drawing" pain, and then none perhaps for half an hour. He sometimes gets a pain "like the stab of a knife" in the right side of his chest. C— is liable to have shooting pains, chiefly in the calves, once

in six or seven days. The attacks formerly lasted from three to four hours, and then gradually extended to ten or twelve hours. "A dart of pain would come perhaps three times in five minutes, and be gone instantaneously like a flash of lightning—a stroke and a quiver." H— for years past has had "the most dreadful pains shooting in the thighs and heels." S— may go two to three weeks without pain, and then it may only last a few hours, in the shins and ankles. On some occasions he has very severe pains of a shooting character in the inner ankle for twenty-four hours. Next day the pain will gradually "work up" the thigh and the left flank. He describes flying pains lasting a few seconds, and like something being "pushed into" the front or the lower part of the tibia, as if into the bone, recurring at intervals of perhaps five seconds. Another man's description was "rats gnawing his shin bones." Another speaks of "pins and needles in his legs, occasionally darting pains like something sharp pushed into his back." The term used by another patient is "spasmodic shooting pains, principally at night, in the heel, calf, or knee." By another, "electric pains in the arms." An educated patient now under my care says that "excruciating paroxysms of pain will occur, especially in the right foot and different parts of the right lower extremity." The pains will come perhaps five or six times in the minute, like "a corkscrew of red-hot iron going into the flesh." "Bouts of pain like this may return for several days together, or their recurrence may be delayed for a week or more." T— had darting, extremely sudden pains in the ankles, calves, backs of thighs, and knees. At the time of the pains the slightest touch on the skin would be agony, but strong pressure could be borne, and was rather comfortable. Damp or draught would be sure to cause the pains. After a time the legs would feel as if they had been beaten, and the pains were accompanied by involuntary movements. F— described a "knife-like spasm" as attacking one or other of his lower extremities in thigh, knee, leg, or ankle, at intervals of a few minutes.

He showed a point two or three inches above the left knee, where, over a space not bigger than a crown-piece, it was as though some one were giving him repeated digs with a knife. M— spoke of "quivering shocks" in either leg. A female patient said her illness began with pains in the knees, which seized her when she walked quickly. K— describes "a grip of pain of momentary duration, followed in a minute or two by another."

Such are a few illustrations, which I could easily amplify, of the character of the pains. As regards the frequency of occurrence of this symptom, I can give some idea of this from a recently published paper of Erb,* and also from my own records. Erb mentions that out of fifty-six cases of tabes which he brings together for comparison the lancinating pains were altogether absent only in five, and in three they were so slightly pronounced as to be not specially characteristic. I have lately analysed a collection of fifty-four cases of tabes in my own experience, of which I possess notes. In only one case was there a complete absence of pains. In one other instance pains had occurred, which were attributed to "rheumatism," and they certainly did not appear to possess those features of suddenness, short duration, and sharpness which ordinarily characterise the pains of tabes. The pain was described as being more or less continuous. The other symptoms of tabes were, however, very well marked, and it is therefore quite possible that the apparent continuousness may really have been owing to an extremely rapid recurrence of momentary pains. I am sure that this occurs in reference to pain, and one is reminded by it of the tetanic contraction of muscular tissue, which, unbroken as it appears to be, is known to consist of vibrations succeeding each other very quickly. It is well to bear this in mind, as a description of continuousness of pain would otherwise be liable to divert the attention from the idea of tabes in a case which was so characterised. But leaving out this case, and

* "Zur Pathologie der Tabes dorsalis," 'Deutsches Archiv für klinische Medicin.' 1879.

adding together the statistics of Erb and myself, we get but seven instances in which pain was absent out of a total of 110 cases, so that as far as the present figures are a guide, this symptom occurs in tabes dorsalis in the proportion of nearly 94 per cent. of the cases.

Now doubtless, as Charcot has pointed out, pains of the lightning or electric shock-like character, may be met with in other conditions. We have seen them occur in a recent case of Pott's disease. They were marked also in a case of almost universal paralysis which recovered under specific treatment—a case the pathology of which is obscure. They occur, too, in spinal pachymeningitis, and in leptomeningitis, sometimes also in disseminated sclerosis and in acute myelitis. What appears to me to be characteristic of the pains of tabes, at least in the earlier stage of the disease, is this—that you may have a person going about his usual occupation with activity, and often presenting no outward sign of ill-health, but who is liable to have from time to time paroxysms of pains, *not usually limited to the district of one nerve, as in neuralgia,* but commonly attacking more than one locality, if not on the same, yet on other occasions—pains which are often horribly severe, and give him sleepless nights, but which may leave him again, for periods of days, weeks or sometimes months, and during the period of immunity, as he tells you, he has nothing to complain of. Now, of course, this does not always happen in tabes dorsalis; the pains may be more continuous or disabling, and other symptoms may be so rapidly developed as to allow no time for the condition described to be noticeable; but it is really a very common story, and sufferers in this way, when their ailment is not ascribed to rheumatism, call it gout or neuralgia, and tell you perhaps that a glass of hot whisky-and-water, some alkaline remedy, a dose of colchicum, or an alterative aperient never fails to disperse these pains. The fact, however, is that it is in the natural history of such pains that they should occur in paroxysms, with intervals of immunity, and, whether

treated medically or not, they usually tend to cease after a few hours or days.

The three patients whom I show you present characteristic pains as part of their symptoms. Of B— I have already spoken. Charles V— says that he became liable to have "pains of a momentary character, like electric shocks, chiefly in the lower limbs, but occasionally also in the shoulders. They would make him jump. The pains would be repeated many times in an hour, and he would have bouts of them lasting for several hours daily for perhaps a week, and then disappearing for a month or so, to recur in a similar manner." For the last year he has been able to carry on his regular employment of a sweep. Charles A— complains of burning pains in the left arm, as if it had been scalded, and sharp cutting pains in both knees.

These patients also present another symptom of great importance, of which I have already spoken some time since. In neither of them does a blow upon the patellar tendon cause any contraction of the quadriceps extensor muscle. The fact that in tabes there is absence of the usual response to a blow on the ligamentum patellæ was first pointed out by Westphal, and I think that clinical medicine owes very much to him for the observation. There are certain misconceptions, I find, in regard to the symptom which it may be as well to guard against by a few hints.

1. If you do not obtain the jerk of the foot upwards on a rough examination you should always lay the leg bare and examine with great care, using a percussion hammer, before concluding that the reflex is absent.

2. Take care that the leg hangs easily, and that the patient is not voluntarily or unintentionally restraining its movement, or, on the other hand, as I have known to happen, voluntarily imitating the reflex jerk. The patient may sit on a chair, and cross one leg over the other, or let his leg dangle from a table;* or, if he is in bed, you

* See also Lecture II, page 18, where another mode of examination is described.

may support the lower part of his thigh with your hand.

3. Remember that absence of the patellar tendon-reflex does not at all *necessarily* point to tabes. Any destructive lesion of the cord at the part from which comes the nerve-supply to the quadriceps extensor will prevent the occurence of the reflex. It is absent, for example, in many cases of acute myelitis, infantile paralysis, and anterior polio-myelitis. Moreover, any coarse affection of the anterior or posterior roots of the lumbar plexus will stop it—anything, indeed, which interferes at any point with the integrity of the nervous arc which extends from the patellar tendon to the grey matter of the cord, and thence to the muscle. It may, therefore be absent in spinal meningitis, in perineuritis, and diphtheritic paralysis, and also where there is change or degeneration of muscular structure, as in myosclerosis (pseudo-hypertrophic muscular paralysis).

4. What you require for this symptom to be characteristic of tabes is this; that along with the absence of reflex there is to a great extent, at least, a normal condition of the quadriceps muscle. In each of these patients the knee can be voluntarily extended with a fair amount of force, and when I strike the vastus internus muscle with the hammer a very evident wave of contraction occurs in the muscular tissue. (A slight upward movement of the foot is caused by this experiment. On a previous occasion* I have called attention to the state of integrity of the muscle in tabes, and cautioned against mistaking this idio-muscular contraction for a reflex.) You will observe that there is no evident muscular atrophy, and I may tell you that induced currents of electricity will provoke the normal response in the muscle.

Now, let me contrast with these conditions that which you will observe in the child brought before you. This little one, of some two years old, who occupies a cot in the hospital, is affected with infantile paralysis limited to the

* 'The Lancet,' August 10th, 1878. See Lecture I, page 11.

left lower extremity. When I strike the right patellar tendon you observe an immediate jerk of the foot; on the left, however, I may tap repeatedly and there is no response. It will be noticed that the left limb is much wasted, that a blow on the vastus internus of that thigh causes no wave, and it is a fact, which I have carefully tested, that the strongest induced currents have no effect in causing contraction of the muscle.

5. The symptom may occur without any peculiarity of gait being present. The case of the patient A— is a very important one in reference to this subject, and I will therefore briefly quote the history from Mr. Broster's notes. Charles A—, aged forty-five, who came to the hospital in April 1879, suffering from paralysis of the right external rectus muscle, told us that he had been affected with drooping of the left eyelid two or three months previously, but this had disappeared. There was anæsthesia of the left side of the face, cheek, tongue, and pharynx (left side), with burning pains in his arms (as previously described), occasional giving way of the leg, and sharp cutting pains of paroxysmal character in both knees. There was no difficulty in standing or walking with the eyes shut; no ataxy. His pupils did not react to light but acted readily during accommodation. His first symptom was diplopia, which occurred in October, 1878. Before that time he had scarcely experienced a day's illness in his life, and certainly (he says) had not had any cutting pains. Now in April, 1879, I found the patellar tendon-reflex quite absent on the left side, and but slightly marked on the right. On August 13th the tendon-reflex was discovered to be absent on both sides.

Passing over a number of other interesting points, let me draw your attention to the important fact that in this patient the patellar tendon-reflex was absent in one leg and but slightly present in the other only six months after the first symptoms of tabes, and that a few months later the phenomenon was not to be observed in either leg. (I shall have occasion later on to speak of another

case in which this early disappearance of the reflex was noted, but it will be more convenient to defer it.) The gradual mode in which the disappearance took place is also very interesting. It does away with the possibility of congenital absence causing a fallacious inference. It is peculiarly important, too, as occurring in a person in whom the disease is to all appearance almost limited to the upper part of the body. The only sign of the lower extremities being involved is found in the man's own description, given in August last: "When I get out of bed in the night I am very unsteady." You have seen him walk and noted that he shows no difficulty of gait whatever. You would never guess, to look at him, that he is affected with tabes dorsalis.

The symptom (absence of patellar tendon-reflex) is of such comparatively recent observation that I do not feel able to say from my own experience whether it is ordinarily to be met with so early as this, but Erb, who began to investigate the matter before I did, has been led to consider it as one of the earliest symptoms. It is certainly one of the most constant. Erb has lately shown that the symptom was found by him in forty-eight out of forty-nine cases of tabes dorsalis in which he sought for it. Out of thirty cases of tabes in my own practice in which I applied the test, I found the tendon-reflex absent in twenty-eight. The two patients in whom it was still retained (for one of whom I am indebted to my colleague Dr. Jackson) presented no ataxy of gait, but I have no doubt that they were cases of tabes. They both suffered from atrophy of the optic nerves, and characteristic lightning pains. One had some tottering on closing the eyes, some bladder and sexual weakness, with anæsthesia of the extremities. The other had some symptoms resembling the *crises gastriques* of Charcot. I do not include here other cases than those which have occurred in my own practice, but it is within my knowledge that the kind of frequency described is met with by others. Now if we add together Erb's cases and my own, we find the

absence of patellar tendon-reflex noted in seventy-six out of seventy-nine cases of tabes dorsalis—*i.e.* in about 96 per cent.* In two out of the three exceptions there was no ataxy.

There can be little doubt, I think, that this symptom holds the same rank as an *objective* sign of tabes as is occupied by the characteristic pains amongst the *subjective* symptoms of the disease. Let me illustrate its utility.

Five or six years ago I attended a gentleman who had hemiplegia and aphasia. He recovered, went to India, and actively engaged in military duties. A few weeks back he came home and called upon me, not for advice, but to tell me how well he had got on. The day after my last lecture he came again, and said he feared he had suffered a relapse the day before, as there had been a peculiar feeling in his right leg, and there had been a "contortion" of it, his head also being drawn to one side. In the course of examination I tested his patellar reflex of the right leg, expecting to find it in some excess, as is most commonly the case in the sequel of hemiplegia. To my surprise it was absent in both legs. Finding this I inquired about pains, and learned that for several years past he has been liable to "rheumatic" or "neuralgic" pains, sudden in character, of very short duration, and coming in almost any part of him. A year or two ago he had such a bad attack of pains of this kind in his foot that, to use his own expression, "it almost made him delirious." The pains would be very sharp indeed, and recur at perhaps minute intervals. He has been kept awake all night by them. He feels lost in the dark, and cannot stand for a moment with his eyes shut. His gait is ataxic. He sometimes gives way at the knee. There is some want of electro-cutaneous sensibility in the legs; the muscles of the thighs respond perfectly to induced currents. The pupils contract, though but sluggishly, to light, and much better during accommodation. His pulse is about 100.

* Subsequent experience of numerous cases confirms this observation.

I have said enough to show that this is a case of tabes. For its recognition, I am indebted in the first instance to the observation that percussion of the ligamentum patellæ produced no upward jerk of the foot. Until this fact was observed the idea that a patient who had previously suffered, to my knowledge, from a sudden attack of right hemiplegia and aphasia, had now come to be an example of tabes dorsalis had not entered my mind. It is of much importance to remember that the two symptoms—on the subjective side pains of the character described, and on the objective side the absence of patellar tendon-reflex (with a fairly normal condition of the quadriceps extensor muscle) —are the most constant, as they are probably the earliest of all. My belief is that if we meet with a patient who exhibits them both we do not need the presence of any other in order to form a diagnosis of tabes dorsalis.

LECTURE VII

TABES DORSALIS (*continued*)

At the last lecture I exhibited three patients affected with tabes dorsalis. B— was a typical case of the disease, with ataxic gait and incoordinate movements of the hands, lightning pains of excruciating character, and diplopia, amongst other symptoms. A— had no ataxy of gait, but all the other characteristics of tabes were well marked. V— was a thoroughly representative case, with no symptom wanting to complete the picture. All three, it will be remembered, showed entire absence of patellar tendon-reflex, the quadriceps extensor muscle retaining at the same time its normal capacity for being excited to contraction by a blow or by induced electrical currents.

Now, in each of these cases there was that condition of pupil to which attention was first called by Argyll Robertson.* The pupils do not contract to light, but they do contract during accommodation. I purposely refer to this peculiar condition of the pupil just now, because it seems to me that it constitutes a further instance of that removal of reflex in tabes of which so remarkable an example is offered by the absence of response to a blow upon the patellar tendon. The symptom is of great importance, for it is extremely frequent. Some highly interesting experience has been published by Vincent on this point.†

* 'Edin. Medical Journal,' December, 1869. See also an interesting article "On the eye-symptoms in locomotor ataxia," by Dr. Grainger Stewart, 'Brain,' July, 1879. Erb, "Zur Pathologie der Tabes dorsalis." Hughlings-Jackson, "Eye symptoms in Locomotor Ataxy," 'Transactions Ophthal. Soc.,' vol. 1.

† 'Des Phénomènes Oculo-pupillaires dans l'Ataxie Locomotrice et la Paralysie Générale des Aliénés.' Par Ch. Vincent. Paris, 1877.

Out of fifty-one tabetic patients which he had the opportunity of observing in Charcot's clinique there were only four whose pupils reacted normally. In seven there was complete immobility of the pupils (usually combined with amaurosis), and in forty the pupils did not react, or reacted imperfectly, to light, but contracted during accommodation.

It occurred to me that the ophthalmoscope would offer a convenient mode of testing this symptom, the examination thus being able to be made at the same time that the state of the fundus oculi is observed. The following is the mode of examination which I have devised, and which answers perfectly. The patient should be directed to look over your shoulder at some object in the distance. With the ophthalmoscopic mirror you then cast a strong light upon his eye, whilst at the same time you watch for any variation in the size of the pupil through a convex lens of about + 8 or + 10 placed behind it. Supposing that the pupil does not contract to light, the patient may next be told to look alternately at the distant object and at one almost in a line with it, but within ten inches (or still nearer, if he be myopic) of his eye. Uniform illumination of the surface of the eye should be maintained during the continuance of the examination, and the diameter of the pupil watched as the patient's gaze is shifted from one object to the other. It is necessary to be careful that no fallacy creeps in from the patient regarding your face (and thereby accommodating), when he should be looking at a distant object.

It appears to me (and I am alone responsible for the suggestion) that a very interesting generalisation can be derived from this symptom. It may be put as follows:— As I have said before, in a large proportion of cases of tabes the natural movement of the iris consequent upon light falling upon the eye does not occur, or occurs only slightly and imperfectly. The iris does move, however (the pupil contracts), when the patient looks at an object placed near to his face. Now, to quote Donders, "the

movements of the iris are of two kinds, reflex and voluntary. Reflex action is exhibited as constriction of the pupil in consequence of the stimulus of incident light upon the retina." It is this reflex movement which is so frequently abolished or impaired in tabes. The contraction of the pupil which accompanies that of accommodation for near objects and, as Donders shows, is to be considered voluntary, remains, in the large majority of cases, unimpaired. We cannot help seeing in this a remarkable analogy with what happens in the leg. The tabetic patient can kick his foot upwards—*i.e.* voluntarily contract his quadriceps extensor muscle—perfectly well. The contraction, however, which in a state of health follows, by reflex action, the stimulus of a blow upon the patellar tendon does not occur, or occurs only to a limited extent. Time will not permit me to go further into this interesting point. I would only remark that the fact described lends additional support, so far as it goes, to Pierret's view, that tabes essentially attacks the *sensory* side of the cerebro-spinal nervous system. Reflex movements, as we know, are excited by impulses which travel along afferent (sensory) nerves.*

In the three patients referred to an important symptom has occured in addition to those which have been already described. B— has suffered occasionally from "darts of pain from one side of his head to the other." A— has anæsthesia of the left half of his face and the interior of his left cheek. "In eating he is apt," he tells us, "to bite the cheek." He can feel but little on the left half of the tongue, and his taste on that side is not so perfect as on the right. He does not feel a touch (I learn from Mr. Broster's notes) on the left half of the palate, and scarcely on the left half of the pharynx. He also suffers from

* Since this lecture appeared in the 'Lancet,' May 1st, 1880, Erb has also made a similar suggestion. "One may speak, therefore (he writes), of abolished pupillary reflexes. Is this not analogous, probably, to that constant and important symptom in tabes, the absence of the tendon-reflex?" 'Archives of Medicine,' New York, October, 1880.

pains in the back of his head. V—, when he came to us, was affected with facial paralysis of the right side, which had immediately followed severe electric shock-like pains of three weeks' duration. I shall have to show you a fourth case, in which one half of the face and tongue is anæsthetic; a fifth case, in which the lightning pains in the head have been so severe as to give rise to the suspicion of intracranial tumour; and a sixth case, which began with sharp shooting pains in the head.

In these three patients we have pains or anæsthesia occurring in some part of the head. How are we to explain this in a disease which affects in so pronounced a fashion the posterior columns of the cord and posterior roots of spinal nerves? As regards the pains which A— experiences at the back of his head, they occur in the district principally innervated on each side by the great occipital nerve, the internal branch, you will remember, of the posterior division of the second cervical nerve. We have, therefore, to do with a spinal nerve after all, although its distribution is to the head. It is evident that such a mode of irritation of its posterior root as causes, when the posterior roots of the nerves supplying the trunk and extremities are in question, the characteristic "lightning pains" in them, is sufficient to explain the occurrence of similar pains at the back of the head.

In the two other cases the pains occupy the district supplied by the fifth nerve.

Now we owe to Pierret* the exceedingly important suggestion that pains in the district of the fifth nerve in tabes dorsalis may be due to sclerosis attacking the descending root of the trigeminus. To show how likely this is I will refer to a few anatomical details which are given by Pierret. The fibres of the trigeminus followed into the medulla oblongata are seen first of all to divide into two bundles, one of which (the motor) root leads to a nucleus common to it and the facial; the other, which is

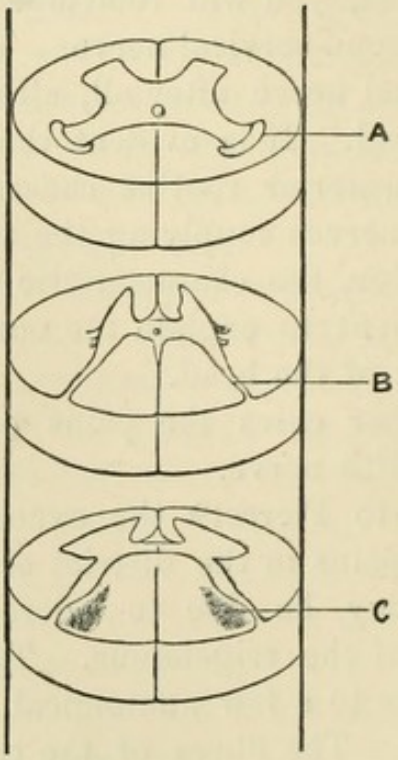
* 'Essai sur les Symptomes Céphaliques de Tabes Dorsalis.' Paris, 1876.

larger (the sensory root), when it reaches the side of the floor of the fourth ventricle, furnishes three branches.

One of these probably mounts to the encephalon; a second is lost in the nucleus known as the locus cœruleus; the third, with which we are particularly concerned, bends from above downwards, and pursuing a course parallel to that of the restiform body, descends to the lower part of the medulla oblongata, being connected throughout its course with the nerve cells which constitute the grey tubercle of Rolando, or inferior nucleus of the trigeminus.

According to Quain, the sensory portion of the fifth nerve arises from a collection of grey substance beneath the outer part of the floor of the fourth ventricle in its middle third. In front of the nucleus a bundle of descending fibres mingled with much grey matter passes down to the lower

FIG. 15.



part of the medulla oblongata. Huguenin describes this descending root as forming at this point a crescent, which encloses the gelatinous substance of the posterior horn. Hence it can be traced as low, it is thought, as the level of the third or fourth cervical vertebra.

In the accompanying diagram the crescent-like cup of the posterior horn (A) represents the descending or spinal root of the trigeminus about where the medulla oblongata ceases and the cord begins. The line drawn from B if prolonged a little inward would indicate the posterior root zone of the second cervical nerve (the sensory origin of the great occipital nerve). The lowest section in the diagram is supposed to be at about the level of the fifth cervical nerve, and C shows sclerosis of the posterior root zone, the seat of the essential change in tabes. It is evident, then, that an extension upwards to B and A of the changes connected with the posterior root zones of the cord (C) must involve in succession the sensory origin of the great occipital nerve and the descending root of the trigeminal, and in this way we have the nerves which supply between them almost all the integument of the head and face in continuous relation with that particular system of fibres, sclerosis of which gives rise to the pains of tabes. For the sake of simplicity the great occipital nerve is alone considered. The remarks apply equally, of course, to the small occipital and great auricular branches of the second and third cervical nerves. Now, it occurred to Pierret to search for changes in the descending root of the fifth in a case of tabes. In a certain case he found that there did exist around this inferior nucleus of the trigeminal nerve a well-defined sclerosis, the form and connections of which recalled exactly what is found in the cord of ataxic patients. With this fact before us, and bearing in mind the relations which I have described, we shall not be surprised to find that symptoms pointing to lesion of the sensory portion of the fifth nerve and the great occipital are really very common in tabes. It is remarkable that this is a point which is frequently to a great extent overlooked, the oculo-motor symptoms and those belonging to the extremities having to a large extent monopolised attention. From a diagnostic point of view its recognition is important. Unless we bear in mind this contingency, a patient's description of intense

pain in the head, so severe as to dwarf other symptoms by contrast, is liable to put us off the scent of tabes dorsalis. May not certain cases of obstinate cranial neuralgia depend upon sclerosis limited to a certain district of the medulla oblongata? It is worth considering whether there are any points to guide us in reference to this possibility.

In an ordinary case of trigeminal neuralgia it is most common to find the pains limited to the district of one or more of the three divisions of the nerve, and it is not often that exploration fails to discover one or more tender spots in situations where experience leads us to expect them to be found. But in the pains (otherwise neuralgic in character) which occur in the region of the fifth nerve in tabes, this accurate mapping out of the district of one or other division of the nerve is not, according to my experience, observed. Moreover, the pains are much more liable than in the case of ordinary tic to affect both sides of the head, either coincidently or in turn. And this is what might be expected, the changes in the posterior columns being disposed to attack more or less symmetrically, though usually not with equal violence, the two sides. The same remarks apply to the occipital nerves. In occipital neuralgia, ordinarily so termed, the district of one or other nerve, not of both, is usually the seat of pain. Flying, so-called "neuralgic" pains in the head, then, *when they attack both sides and do not map out the district of one or other division of the fifth nerve*, should lead to attentive examination for symptoms of tabes. No doubt these may be wanting, as it is quite conceivable, from what we know of the pathology of posterior sclerosis, that the disease may commence in the upper part of the cord, and take time to descend; or even, there is reason to think, may, in some cases, remain limited to the locality in which it originated. But at a later period symptoms may possibly show themselves which will tend to refer the pains in the head to a tabetic origin.

The patient whom I now show to you presents a striking

example of the cephalic symptoms of tabes. J. D— is forty-nine years of age, by occupation a sweep. He had smallpox badly, and ague when he was a lad, and he has also suffered from syphilis. A daughter of his is imbecile, one of his sons has fits, and another, who is now blind, had fits in infancy. There is no history of a fall or mechanical injury. The patient applied here in September last on account of *excruciating pains in his head*, "as though," he said, "it was being scraunched up." He was blind, there being only qualitative perception of light with the left eye.

Now, intense pain in the head, when it is coupled with amaurosis, is very suggestive of the presence of an intracranial tumour, and the idea that the patient was suffering from some encephalic growth crossed my mind at once, as he began to tell his story. When we came to examine him with the ophthalmoscope, we found that both optic discs were atrophied. In the circumstances this was to be expected, and the information which the fact afforded, although important, was not conclusive. If instead of atrophy of the discs we had found optic neuritis, this condition, when taken in connection with the intense severity of the pain in the head, would have gone far towards enabling us to pronounce a somewhat confident diagnosis of intracranial tumour. But the condition was one of atrophy, and atrophy, as is well known, may be (to speak only of two causes) either a consequence of optic neuritis or due to sclerosis of the optic nerve. Now, in most cases in which the atrophy is recent one has not much difficulty in saying to which category a particular example is to be referred. When atrophy succeeds optic neuritis, there is usually to be found for some time afterwards more or less trace of effusion about the disc, the edge of which frequently wears a blurred look. The artery is often diminished in calibre, and the veins may retain more or less of the tortuosity by which they were characterised when the disc was swollen. In sclerosal atrophy, on the other hand, these signs are wanting, and the vessels, undimin-

ished in size, appear as if applied against an opaque white or bluish-green disc, which presents a clear and sharply-cut outline. But these distinctions are not always so pronounced. Gowers, in his able work on medical ophthalmoscopy, remarks of "primary" and "secondary" atrophy, "it is doubtful whether the two forms can be distinguished by the ultimate aspect of the disc." Certainly in this particular instance one would have felt disinclined to say positively whether the observed atrophy had resulted from optic neuritis or not. It is true that there were no signs of antecedent swelling of the disc or effusion, and no tortuosity of the vessels, such as one commonly sees after optic neuritis. But it is surprising how completely the swelling of a disc may after a time disappear, and what little evidence of the nature of past changes is sometimes left. The ophthalmoscopic signs, therefore, although they pointed only to a wasting of nerve-fibres, with overgrowth of connective tissue, were not, properly speaking, conclusive, especially when the condition was taken along with the clinical history of terribly severe pain in the head.

I lately examined after death a man who had incomplete atrophy of the optic disc in one eye and some amblyopia in the other. His symptoms were purely cerebral, and he died of brain softening. My colleagues, Dr Hughlings-Jackson and Dr Gowers, who were kind enough to examine the eye independently during life, were of opinion that the atrophy was not secondary to neuritis, and Mr Brudenell Carter, who also obliged me by examining him, thought that it was of the kind seen in locomotor ataxy—a sclerosal atrophy in fact. I was, for my own part, doubtful, but more disposed to think it secondary to neuritis, for one or two of the veins at some distance from the disc were more tortuous than appeared natural.* However, after death, I

* I should not now attach so much importance to this circumstance, as I have seen similar signs of obstructed circulation in other cases which were doubtless of sclerosal origin.

found a small tumour pressing upon the left optic tract, and flattening it. Such pressure might cause an atrophy which would be indistinguishable from that seen in tabes. Whether or not there had been at any time antecedent optic neuritis in connexion with the presence of the tumour must still remain, I think, doubtful. At any rate, there was cause enough for the atrophy in the pressure of the growth.

I mention the case as an illustration of the doubt which may sometimes exist upon this point, and as a reason why, in a man who came to us with optic atrophy, and intense pain in the head, I did not feel inclined to decide at once whether his case was one of intracranial tumour or not. Our next step was to test his patellar tendon-reflex. It was absent in both legs. Here was evidence, therefore, at once of the existence of a spinal lesion. This reflex is not lost as a result of cerebral lesions, so that, whatever else there might be (and this, of course, did not preclude the existence of an intracranial growth), there was lesion of the spinal cord or spinal nerves. The clue thus given was followed up, and the following history elicited. Two years and a half previously his sight began to fail, the right eye first. At times he saw double. For some years he had been subject to pains in his thighs, on and off "very quick indeed;" "like an electric shock," he said, "sometimes so sharp that he would jump up, or be obliged to get up and straighten his limbs." The pains in the head, upon which I have insisted, did not begin till about two years previous to his coming under observation, when the pains in his limbs had already troubled him for a long while. He worked, it seems, up to Christmas, 1878, but then found that he could not see the numbers on the doors of the houses. He was not aware that he staggered until October, 1878, when, whilst sweeping a chimney in a lady's house, he was ordered out "on account of being drunk," being at the time, he pleads, perfectly sober. Latterly, the pains have affected the arms as well as the legs and

head. The patient's own description of the pains, as taken down by Mr Broster, is very characteristic. "It is like," he says, "a violent shock of rheumatics coming lightning speed up and down your limbs, flying up and down." There is no loss of cutaneous sensibility anywhere, but when he is in bed he does not know where his feet are. The muscular power of his limbs is not very perceptibly impaired, but he says that his right leg seems to be getting weak. His pupils are very small indeed. They do not contract to light, but they contract during accommodation.

There can be no doubt that the patient is suffering from tabes dorsalis, and as little, I think, that the optic atrophy is sclerosal, and the pains in the head due to lesion of the descending root of the fifth, and probably also of the great occipital nerves. The pains, he tells us, affect both sides of the head and the forehead.

I have lately seen in consultation a gentleman, aged 41, who was attacked last year with double vision which lasted a month, and from which he recovered whilst using electricity. Soon after this he began to be annoyed by a constant bad taste in his mouth, which was attributed to indigestion, until a shrewd surgeon discovered an ulcer (evidently syphilitic) at the back of the soft palate. Next, his sight began to fail, and a map of his field of vision, taken by an ophthalmic surgeon, showed that the greater part of the field to the patient's left was in darkness, a small segment in the left eye and a still smaller one in the right alone remaining sound. By the ophthalmoscope I found the discs somewhat atrophied, the right decidedly so. There was no trace of old optic neuritis. There was a distinct history of a chancre twenty-four years previously followed by rupial patches. My diagnosis was a gumma on one of the optic tracts, which had at first probably involved also one of the oculo-motor nerves; this was now producing the hemiopia with partial atrophy of the optic nerves.

The occurrence of a temporary diplopia followed by progressive atrophy of the optic disc is very suggestive of

tabes dorsalis. The question arose whether this case was an example of the kind, and one authority who was consulted took that view. But I could not agree with this opinion. The tendon-reflex of the patient was normal and he had never had anything like lightning pains. Of course the absence of these symptoms is not conclusive against the existence of tabes. The condition may possibly be one of sclerosis attacking exclusively the intracranial portion of the tract. But I think the evidence taken together is far stronger in favour of the idea of coarse disease—a tumour, for example—affecting one of the optic tracts; and this view is the one taken by the large majority of those who have examined the patient.

[The patient died since I wrote the above, and an autopsy, at which I was not present, showed, it is said, softening of one of the optic tracts, and nothing else.]

Whilst upon this subject of optic atrophy, I will refer to the case of a man who has been kindly sent to me by Mr Macnamara. He is a labourer, forty-four years of age, who, besides suffering from aortic regurgitation, is affected with incomplete atrophy of both optic discs, but especially of the left. The disc presents a lustre like that of a tendon. When asked if he had difficulty in walking, he said he "could walk as well as ever he did in his life," and illustrated his assertion by a very fair performance. There is nothing in his gait which would be likely to attract attention. His pupils are small. There is a very slight reaction in the right one, and the left does not contract at all to light; but they both act well enough during accommodation. He tells us, and I have inquired most carefully on this point, that he has never had any pain whatever, nor has he seen double; but on testing his patellar tendon-reflex, we found that there was none in either leg, and when we inquired more particularly about staggering, he allowed that his brother when walking with him had more than once said, "Why, anyone would think you were drunk."

With the exception, then, of this reported stagger, only

occasionally observed, the patient presents no sign of tabes to help us in our diagnosis of the cause of the optic atrophy, except the absence of patellar tendon-reflex, and the existence of the "Argyll-Robertson symptom" (to use Dr Grainger Stewart's designation). Can we doubt that they are amply sufficient?

But where even one of these two symptoms is absent I am disposed to think that in certain cases we may make our diagnosis with a good deal of confidence. I will give you an instance. A gentleman, aged thirty, was lately brought to me by Dr Roose, of Brighton, on account of increasing blindness, supposed to be due to lesion of the central nervous system. The patient had enjoyed excellent health except for an attack of syphilis five years previously. Last May he found that vision with the right eye was failing, and this difficulty has gradually increased, so that there is now only qualitative perception of light with it. It was some time after the right had begun to fail that he found the left eye going also in the same way. On examination with the ophthalmoscope I found what appeared to me to be the signs characteristic of grey degeneration (sclerosal atrophy) of each optic nerve, most advanced in the right. The discs presented no atrophic excavation. They had a tendinous lustre, the veins were rather large in proportion to the arteries, and there was a slight tortuosity in one of them. Patient's perception of colour was imperfect. Scarlet was called "brownish-yellow," green "slaty-brown," a vivid magenta "pale-blue." Blue and yellow were described correctly. Careful inquiry showed that he had never had any pain in the head, vomiting, or any other cerebral symptom, such as might have been expected were the atrophy the result of optic neuritis. But, on the other hand, there had been no flying pains in his limbs or diplopia. Nor was there any ataxy of gait or tottering with the eyes shut. The pupils acted readily to light. There was no anæsthesia, and no trouble with the genito-urinary organs. There was nothing indeed to be noted but the gradually increasing atrophy of both

optic nerves—with one exception I found the patellar tendon-reflex absent in the right leg, and present to a slight degree only in the left. The quadriceps extensor muscle responded readily to blows upon it and to induced currents.*

If I have succeeded in conveying an idea of the importance of this test, as exemplified in many forms of the disease we are considering, you will, I think, be disposed to see in this case, as I do, one of tabes dorsalis, which as yet presents itself in a very restricted form. Dr Hughlings-Jackson pointed out, in the Medical Times and Gazette, September, 1866, that there was at least a clinical gradation in the symptoms from what was called "uncomplicated amaurosis" to locomotor ataxia. He founded this idea upon the occurrence of characteristic shooting-pains in many cases of amaurosis in which the ophthalmoscopic changes in the discs were of the character of those met with in tabes. He believed that amaurosis was sometimes the first symptom of this disease. For some time past this view has been accepted, and cases of optic atrophy, with lightning pains and without other symptoms, have been classed with locomotor ataxy. It is only quite recently that Westphal's observations in reference to absence of patellar tendon-reflex have brought us the most important confirmation in the occurrence of the symptom, as in the last case, where none of the other recognised signs of locomotor ataxy have presented themselves.

There appeared lately in one of the medical Journals† an account of three cases of simple atrophy of the optic nerves occurring in members of one family. Mr Higgens, who had described the cases, was kind enough, at my request, to inquire into the condition of the tendon-reflex and contractility of pupils in one of them. In that instance the patellar tendon-reflex was found wanting on

* Since the above was written the tendon-reflex has disappeared also from the left leg.

† 'Lancet,' November 19th, 1881.

both sides, nor was there any ulnar, tendo achillis, or radial reflex. The patient could stand and walk with the eyes shut. The left pupil was widely dilated, the right moderately contracted. The pupils acted slightly during accommodation, but not at all to light. With the right eye the patient could count fingers; with the left there was only perception of shadows. Both optic discs were bluish white. The blood supply to the retina was good.

LECTURE VIII

TABES DORSALIS (*continued*)

THE patient, Frederick J—, applied here in October, 1878, on account of numbness in the extremities. This was his principal complaint, but he told us that he had been previously affected with difficulty in swallowing, choking in the throat, nasal speech, and regurgitation of fluids. These symptoms had disappeared, but the numbness still remained. The *primâ facie* resemblance to the symptoms of diphtheritic paralysis was very striking, and there was a passing idea that the case might prove to be of this nature. When we came to test for the knee phenomenon we found that it was entirely absent on each side, and this circumstance immediately caused us to examine him in regard to other symptoms of tabes. For at that time I had not had the opportunity of testing the patellar tendon-reflex in a case of diphtheritic paralysis, and did not know (what I have since definitely ascertained) that this reflex is lost for a time in that disease. Any uncertainty, however, in his case rapidly disappeared as we questioned and examined the man.

According to his own account, this man's disease had commenced with numbness in the ring and little fingers of the right hand eight months previously. This had extended up the forearm and still remained. These symptoms were followed by difficulty in swallowing. About a week after he had first remarked that anything was wrong with his throat he became totally unable to swallow solids without taking some fluid to wash them

down. His speech, too, was very indistinct and nasal in character. There was a choking sensation in his throat, and fluids regurgitated through the nostrils. Under treatment in a hospital he lost these symptoms. About four months after the commencement of his illness he was seized with double vision, which lasted three months. At the same time that he began to see double, numbness was felt in the left finger-tips and left side of the face. A few weeks later the soles of his feet and his legs became numbed. Since then, he told us, walking had been getting more difficult, and during the last month he had not been able to stand in the dark.

We found that he had slight double vision, that he complained of a tightness on his hips, "as if there were a weight upon them," of cold feet, of numbness in the feet, legs, thighs, penis, the whole right hand and forearm, as well as the tips of the fingers of the left hand. Cutaneous sensibility was delayed (as regards pain and touch) in the legs and thighs, and cold was thought to be heat in the same quarters. There was complete anæsthesia in the three inner fingers of the right hand. On the trunk, too, there was great diminution of sensibility. He suffered occasional shooting pain in his limbs, with a dull aching in the back. He had also from time to time shooting pains in his head. His gait was ataxic with fair muscular power in the thighs, and he could not stand in the dark. There was some lateral and posterior curvature of the spine.

When his attention was called to the subject he allowed that for ten years past he had been liable to suffer from momentary pains—localised in the hips, thighs, and knees—of a very sharp character. At first there had also been severe but dull pain about the mid-dorsal region of the spine and he had been unable to bend his back. The only ease to be obtained was in lying flat on his back. His knee-joints seemed weak, and he was unable to run. With this exception and the occasional recurrence of the pains, he had noticed nothing especially wrong with his

legs till about three months before he came here. It should be noted that he had a chancre sixteen years ago. followed by sore throat and eruption on the skin. His wife had miscarried several times, and been delivered of one stillborn child.

We took the man into the hospital for a couple of months last year; then he went home and was readmitted a week or two ago. His condition has greatly deteriorated. He now keeps his bed, being quite unable to stand. Indeed as he lies he cannot lift either leg off the bed, though he is able to move the left very slightly. His muscles have wasted very much. He does not know where his arms or legs are in the dark. There is almost complete anæsthesia of his back, left side of the face, tip of the tongue, inside of left cheek, both arms and legs, and there is delay in appreciating heat and cold in the legs. Sensations of temperature are also, though to a less extent, delayed in the arms. He has had double vision for the last seven weeks. He is unable to take a draught of fluid, but is obliged to drink by small mouthfuls. Were he to try a draught, he tells us, he would choke and the fluid would return by his nostrils. His speech is nasal, and when he breathes heavily the breath sounds have a nasal character. He does not feel a touch on either side of the soft palate, nor on the pharynx. If we tickle the back of the throat with the feather of a pen not the smallest trace of reflex action occurs. He cannot taste sweet things. The tip of his tongue feels numbed. Cutaneous sensibility is impaired on the left side of the face. It is especially to these points that I wish to draw attention, and need only therefore rapidly summarise the remaining symptoms which go to make the nature of his disease perfectly certain. The knee phenomenon is absent; he suffers occasionally from shooting pains in the legs; there is cutaneous anæsthesia over the lower extremities, with analgesia of the soles of the feet. He does not know where his legs are. The pupils, which are scarcely larger than a pin-point, do not contract to light, but contract during accommodation.

To observe this they must be looked at through a rather strong convex lens. The patient has occasional diplopia.

In addition to these characteristic symptoms there are others which require to be mentioned. The man is unable to stand, not on account of incoordination, but because his legs are nearly powerless. He seems to have just a little power over the flexors of the hip-joints (not sufficient to raise the knee off the couch), and can just bend the ankles and toes; but with these exceptions he cannot move his legs. There is no skin reflex throughout the lower extremities. His urine is apt to be retained.

The muscular wasting which we observe in this instance is not a rare feature of tabes at a somewhat advanced period. In such cases, after death there is found atrophy of ganglionic cells in the anterior cornua. The large motor cells present great changes. They have sometimes disappeared in great part and given place to a patch of sclerosis.

According to Kölliker, a certain number of the nerve filaments constituting the posterior root of a spinal nerve are directed to the anterior horn, and can be followed in it to the outer group of motor nerve cells. Charcot supposes it is by means of these filaments that the irritative process, first of all developed in the posterior columns, is propagated to the anterior horns, and there determines the changes in the motor cells which result in the muscular atrophy, such as is to be observed in this case. As might be expected the muscles of the lower extremities show a somewhat diminished reaction to the induced current.

Let me now return to the difficulty in swallowing, which is so remarkable a feature in J—'s case. As I have mentioned, this symptom in the first instance suggested the idea that the man was suffering from diphtheritic paralysis. It is evident, if we come to examine closely the source of difficulty, that it depends upon a lesion on the sensory side. When we look into the man's mouth we find nothing remarkable in the appearance of the soft palate; it is symmetrical on the two sides. It is only when we

come to test the sensibility of the mucous membrane covering it and the posterior wall of the pharynx that we come upon signs of a serious change. Touches are not felt in this situation, and they do not provoke reflex contraction. Now we know that swallowing is a reflex act, and that the centre for the process is located in the medulla oblongata, the afferent influence being derived from the branches of the spheno-palatine ganglion, which is situated on the superior maxillary division of the fifth nerve. It cannot be doubted, considering the circumstances, that we have to do here with lesion of the deep origin of the fifth in the medulla oblongata.

Pierret has referred to this symptom. "The anæsthesia," he says, "may extend to the pharynx and produce dysphagia."*

The occurrence of nasal voice is interesting. This peculiarity is known to depend upon the circumstance that the soft palate is not drawn up and applied as it should be so as to shut off the nasal cavities. It would appear then that this action, which takes place in health without our consciousness, is a reflex process, the afferent part of which depends upon branches of the fifth nerve.

Another man, Richard C—, who is evidently affected with tabes, although exceptionally in his case the knee phenomenon is present, has a peculiarity in his voice which cannot well be described. It is distinctly nasal in character, but hollow rather than snuffling. He speaks as if he had enlarged tonsils. His case is briefly this:—He is ataxic; there is reflex pupillary immobility as well as diplopia, but he has had no shooting pains. He can swallow perfectly, but fluids sometimes regurgitate through his nostrils. I sent him to Dr Semon, who was kind enough to examine him and give me the following report:

"The patient presents only one, *possibly* pathological, alteration from the normal conditions, as far as the larynx is concerned, viz. during phonation there remains an elliptic gap between the middle part of the free edges of the

* Loc. cit.

vocal cords, such as is found in paralysis or paresis of the internal tensors of the vocal cords (internal thyro-arytænoid muscles).

"I have seen, however, the phenomenon so frequently in apparently healthy persons that I should be very doubtful whether in this case it ought to be looked upon as a pathological sign of any importance or merely as a slight deviation within physiological borders, the more so because the patient is in the full power of his voice. He can sing and keep high as well as low notes, and I have no doubt in saying that the somewhat altered character of his voice is simply and exclusively due to the changed conditions of the 'sounding-board.' For whilst there is no other motor or sensory paralysis within the borders of the larynx (I have convinced myself as to the latter fact by touching all the sensitive parts with the laryngeal sound), the motor power as well as the reflex irritability of the soft palate are greatly diminished, although not completely destroyed. There also seems to me to be some peculiar want of coordination of the muscles of the tongue during speaking, although this organ is freely movable in all directions."

We have in these two cases, and especially in that of J—, as remarkable examples of *anæsthesia* in the region of the fifth nerve as we had of *pain* in the same district in the case of the man with symptoms resembling those of intra-cranial tumour. We thus see that in the district of the cranial nerve the trouble may be, as it is in the districts of the spinal nerves, either in the direction of pains or of greater or less insensibility, or, as very often happens, both these conditions may be presented. In the case of C—, the remark of Dr. Semon, as to the want of coordination of the muscles of the tongue, the ataxy indeed of that organ, is interesting, especially in relation to Pierret's view, that the motor troubles in tabes are secondary to those on the sensory side. This brings us not inconveniently to the question of the symptom ataxy, about which I have only a very few words to say.

First, let me again refer for a moment to the case of V—, a patient whom I showed in the last lecture—a typical example of tabes dorsalis. This man, when he came to the hospital in April, 1877, was suffering from paralysis of the right side of the face, and it was entirely on account of this condition, which had existed six days, that he applied here. We learned on inquiry (and this appears to me to be a very important fact) that for the three weeks preceding the sudden occurrence of the facial paralysis he had suffered from very severe *electric shock-like pains in the right side of the head.* One morning on awaking he found his face almost completely paralysed on that side. The sudden loss of power was confined to the face; it did not involve the limbs. At the time of his attendance here, six days after the paralysis began, the faradaic excitability of the orbicularis palpebrarum, as well as of the muscles of the chin, was found to be good, whilst that of the muscles about the mouth was somewhat imperfect. This facial paralysis subsided gradually and had disappeared in the course of a month. Now, I think that in this temporary paralysis of the face in a typical case of tabes, following, as it did, upon very characteristic pains in the same side of the head, we must see something more than a coincidence, and allow that the paralysis was in some way or other the result of the lesion which showed itself by pain.

Pierret reminds us that the fifth nerve (its sensory portion) comports itself like a spinal nerve, and that not only its motor portion, but in effect *all* the intracranial motor nerves—the oculo-motor, pathetic, sixth, facial, hypoglossal—must be considered as practically constituting the anterior roots of this nerve. He points out that in man the various functions tend to acquire great independence; but this is not the case in the inferior vertebrates, in whom the cranial nerves retain the characteristics of spinal nerves, and are not dissociated as they are in the human system. According to Huxley, in the lamprey, the external and inferior rectus and inferior oblique are supplied by the ophthalmic division of the fifth, whilst the third and fourth

nerves unite into a common trunk, which gives branches to the superior and internal rectus and superior oblique. In amphibia the third, fourth, and sixth motor nerves are more or less confounded with the ophthalmic division of the fifth, but in all the higher vertebrata the nerves of the muscles of the eye are distinct from the fifth, except when the oculo-motor unites with the ophthalmic into the ciliary ganglion. The facial and trigeminal nerves have common roots in fishes. In amphibia, though the roots are distinct, the facial may be completely united with the ganglion of the trigeminal, as in the frog.

I think we may take it that in V—'s case irritation of the posterior root (fifth nerve) was followed by paralysis in the district of the anterior root (portio dura). This paralysis was of a passing character, and strictly comparable therefore (Pierret would think) with those transitory paralyses of one or other external muscle of the eyeball, giving rise to double vision, which are so frequently met with in tabes. May we not also infer that lesion of the fifth nerve in the case of C— has been the immediate cause, not only of the partial motor paralysis of the soft palate, but of the trouble of coordination of the tongue to which Dr. Semon refers? There are many difficulties in the way of explanation of the symptom ataxy, and I do not for my part feel that we are able to do more at present than suggest hypotheses. It has been supposed by some that the absence of the knee phenomenon is a factor in the production of that marked ataxy in the lower extremities which has given a name to the disease. But I cannot think that the evidence bears this out. We are constantly meeting with cases which are certainly examples of tabes, in which, however, the symptom ataxy has never appeared, and in which there is not a vestige of knee phenomenon. On the other hand, we sometimes (though very rarely) meet with a case of ataxy, and the patient C— is a good example of this, in which the knee phenomenon is well preserved.

It is customary on the part of writers to describe the

muscular power as being unimpaired in tabes, and the disease is forcibly contrasted with paraplegia on this ground. There can be no doubt that before Duchenne's time tabes was constantly confounded with paraplegia, and the credit of differentiation of the two conditions is certainly due to him. But I am inclined to think that Duchenne, in exposing the difference, rather considerably over-estimated the extent to which muscular power is preserved in this disease. For my part I do not very often meet with a case of tabes in which there is not at some time more or less marked loss of power in one or other of the lower extremities, and not unfrequently there is a paraplegic condition of passing character. It is worth considering whether loss of power may not have much to do with the symptom ataxy, *i.e.* an inability to perform complicated movements. Here I must refer to the views of Dr Hughlings Jackson, which were published long since. According to Dr Jackson, the first movement of a complicated kind which fails in ataxy is that in which the peroneus longus muscle takes the lead —the movement by which the body is swung over on to the big toe. The patient, in the earlier stages, has a difficulty in biting the ground, so to speak, with the front part of the foot; at the same time we observe that there is a strong contraction of the tibialis anticus, the antagonist of the peroneus longus muscle. Dr Jackson likens this over-action of the tibialis anticus to the secondary deviation which occurs, for example, in a case of paresis of the right external rectus muscle of the eye. When the patient tries very hard to turn the right eye *out* the left eye turns *in* too much. There is secondary deviation—an over-movement of the associated muscle. "When," he writes, "a centre discharges and one route for the current is stopped, the current, so to speak, overflows in other channels, in those for associated movements. So when the flexors are atrophied an effort to shut the hand is considerable, and then, to use Duchenne's language, a very intense nervous current reaches the extensors, giving rise to exaggerated extension."

These views, which appear to me to be of great importance, fit in well with those which have been since expressed by Pierret, who also founds his hypothesis on the law of association of antagonism enunciated by Duchenne. In all muscular movements, Duchenne has taught us, there are two forces at work: one which produces the movement, the other which moderates it. If the directing muscle over-acts, the moderating muscle (its antagonist) becomes momentarily insufficient to repress its action; then the movement is exaggerated and becomes too brusque; and the same thing happens if, whilst the directing muscle remains normal, the moderating muscle is enfeebled. There is again exaggeration.

Now it has been shown, Pierret reminds us, that section of the posterior roots of spinal nerves occasions loss of irritability of the corresponding anterior roots. Circumstances also seem to make it probable that a centripetal irritation of the sensory nerve may act upon the motor elements and occasion paralysis—reflex paralysis of Brown-Séquard. Anstie has drawn attention to the paralysis of the oculo-motor nerve, which occasionally occurs in neuralgia of the fifth. We had a case of the kind in the hospital the other day. Jane H—, æt. 30, has been subject for years to paroxysms of facial tic, recurring about every fortnight, and concentrated more or less definitely in the ophthalmic division of the right fifth nerve. Within the last year or two each attack has been followed by partial paralysis of the oculo-motor, the right eyeball being turned outwards and the eyelid dropped. This paralytic condition will last for a few days. We had the opportunity of observing one of these attacks during the patient's short stay in the hospital.

Handfield Jones again has suggested that an impression upon a sensory nerve which, if mild and normal, is stimulating, becomes paralysing if unnaturally strong, and that this paresis from shock—depression—can communicate itself to motor and vaso-motor nerves. He has made the important remark that pain ranks with paralysis.

It would seem, then, likely that the exhaustion of the sensory side, produced by the irritation which results in pain, is continued in some way, about which we know nothing, to the motor side of the nerve centre, and occasions paresis of centres for movements. Nervous impulses from the brain, sent down and meeting with obstructions consequent upon this state of centres, overflow into other channels and produce overaction of associated movements. Hence the wild flinging about of the legs in ataxy; the wilder, the more earnestly the patient tries to coordinate them, *i.e.* the more strongly nervous impulses are directed to the part. The transient character of the oculo-motor troubles, and the paroxysmal mode in which the pains occur, tend to give support to this view, which may be taken as a compound of the hypotheses of Hughlings Jackson and Pierret. There are several weak points, it must be owned, in such an hypothesis. The occasional occurrence of ataxy without pains (a rare event, but not unexampled, as the case of C— shows) is difficult of explanation unless we suggest that there may be afferent fibres (which are not sentient although accompanying sensory nerves), and that the paresis of movement is brought about through the exhaustion consequent upon the irritation of these. According to Foster,* an afferent impulse passing along an afferent nerve may in certain cases give rise to reflex movements without causing any change in consciousness.

But it is still more difficult to explain, and I do not pretend to be able to do so, the very numerous cases in which severe and long-continued lightning pains are unaccompanied by ataxy.

The patient, M—, æt. 50, is a typical example of tabes, presenting lightning pains, absent knee phenomenon, ataxy, cutaneous anæsthesia, feeling of constriction around the waist, defective muscular sense, diplopia, partial optic atrophy. He has occasionally also sharp sudden pains over the top of the head and forehead, and there is slight

* 'Text Book of Physiology,' p. 447.

anæsthesia of one side of his face. But it is to the earlier part of his case that I wish now to direct attention. About seven years ago he found that he could not stand so long as usual while carrying on a business which necessitated his keeping upon his feet. A year later pains of a tingling character shot up his legs and he suffered from cramps. Five years ago he found his right leg was weak, and six months later whilst bathing one day he was taken giddy and sick, a pain shot down from the right hip to the foot, and he was only just able to limp home. Pains in this situation continued almost constantly for three days, and then, according to his account, there was *yellow discoloration of the skin* of this limb. A few weeks later he suddenly lost the power of standing, and fell down. After this he found himself staggering about as if drunk. There was great loss of power in the left leg consequent upon this attack, but this incapacity gradually disappeared. The ataxy which has ever since marked his gait was sudden then in its onset, and was accompanied at first by sudden and marked loss of power following severe pains. His muscular power just now seems to be very fair, but he tells us that during the last four years it has varied much, and that at times he is considerably weaker and less able to walk than he is now. He has much ataxy in the trunk, and when seated, if the eyes be shut, he is scarcely able to prevent himself from rolling off his chair. If we look at his back, we see that his spine presents a very notable amount of lateral curvature, an evidence, there can be no doubt, of considerable and unequal muscular weakness. Inequality in the degree to which power is lost on the two sides of the body is conspicuous in tabes. Variation in condition, also, so that such marked improvement as raises strong hopes of recovery in the patient's mind is followed by a great and perhaps sudden change for the worse is equally characteristic. The same thing, I may remark, is seen in the insular form of cerebro-spinal sclerosis.

Now, passing over other points, I would draw attention to this man's legs. On both legs below the knee, and on the anterior aspect, there is a crop of bullæ. At first the skin comes up, to use the patient's own description, in a small blister, just as though he had been scalded. This breaks, and discloses (as we can see) red flesh, which is very tender, and which he says might go into an ulcer if irritated, but if not irritated will scab and dry up. One of these places has a scab on it, and this began, according to his own account, a week ago. The whole duration of a bleb will be about fourteen days. The subcutaneous cellular tissue would appear to be inflamed, as the leg is somewhat swollen, and the skin looks as glossy as a chilblain. The patient began to have these bullæ five or six years ago. They only lasted a few weeks, and then disappeared till three years ago, when he "knocked both his legs (owing to his greatly disordered gait), and then they broke out again." Then nothing more was seen until five months ago, when they appeared again after blows upon the legs, and have been coming out pretty constantly ever since. The blebs smart, burn, and sometimes itch. During this time he has pains like electric shocks in both legs, centring in the shins, "as if in the marrow of the bones." He thinks that he has had as sharp and continuous pains without as with a coincident eruption.

The occurrence of herpetic, bullous, and pemphigoid eruptions in the course of tabes has been long ago noted by Charcot and Vulpian, who point out that their appearance coincides with exacerbation of lightning pains, their situation also corresponding to the district of the affected nerves.

Quite recently Straus* has drawn attention to another form of cutaneous eruption. He describes the appearance of ecchymoses so exactly resembling those produced by violence that he at first attributed them to that cause, until examination showed that they were spontaneous. I cannot help thinking that our patient may have been an

* "Ecchymoses Tabétiques," 'Archives de Neurologie,' No. 4, 1881.

example of this kind. *The yellow discoloration of the skin*, which he describes as having occurred formerly after pains of extreme severity, is very suggestive of a fading ecchymosis. The pemphigoid eruption with which he is now affected occurring in the situation and circumstances described, I have no hesitation in ascribing to disturbed innervation in connection with the irritation which causes his lightning pains. I have had under observation in private practice a case of tabes with outbreaks of herpetic eruption which is so remarkable, both on that account and for other reasons, that I am tempted to give it in some detail.*

It is just eighteen years ago that I first examined A. B—, who was then thirty-six years of age. His complaint was of bad nights, and troubles in his organs of digestion. Incidentally he mentioned to me the state of his sight, which had become greatly impaired. With the right eye he could only discern light from darkness; with his left he could recognise faces, but all outlines were blurred and confused. He had consulted several ophthalmologists on account of his eyes, and they had described his disease, he told me, as atrophy of the optic nerves. For this he had undergone a variety of treatment, including hypodermic injection of strychnia and the application of the constant current, without any effect.

His father, who was gouty, died at sixty-one years of age. His mother, who was then still living, had always enjoyed excellent health. (She has since died, at eighty-four years of age.) There is no history of tubercle or neurosis in the family.

In infancy, so he had been told, he suffered from "inflammation of the bowels" and bronchitis, besides an attack of whooping-cough. All his life he had been subject to take cold easily, and to suffer from bronchial attacks; and there had always been from his childhood a tendency to looseness of the bowels. When twelve years old he had measles badly, from which he recovered after

* This case has been published in 'Brain,' Part 2.

a long illness. After this, for twenty years he enjoyed excellent health. His habits were temperate in all respects; and the mode of his life, a great part of which was spent in the open air, conduced to healthfulness. He had never had syphilis, but had once or twice suffered from gonorrhœa.

In the year 1860 he had an attack of "muscular rheumatism," which lasted about a fortnight, but only kept him in bed a day or two. There was no fever; his joints did not swell; and he was able to do his work except on one or two days. As the pains in his limbs ceased, the attack was immediately followed by jaundice, from which he suffered for three weeks, and during which he lost two stone in weight. He is quite clear that since that attack he has never been "the same man." He never got up his health or strength again. A very notable sequela had been insomnia. He would retire to bed between eleven and twelve o'clock, lie anxiously restless till three or four, and then drop off to sleep for four or five hours. Sometimes, on the other hand, he would sleep on first going to bed, but in that case would wake at three or four o'clock, and sleep no more. Altogether, he had rarely more than four or five hours' sleep at night.

In 1861 he had for the first time an attack of herpes. It was after undergoing great fatigue that he noticed what he took to be a boil on his right buttock, but really consisted of a crop of vesicles, which burst and formed scabs. During the next two years he had, on four or five occasions, a recurrence of the eruptions in the same situation. There was rarely more than a single group. These attacks were not accompanied by any pain, beyond a little burning at the site of the vesicles. The eruption did not always follow the same course. There would be a group which appeared to be going on to maturity, when it suddenly became, as he describes it, stagnant and abortive. The vesicles did not burst, but withered up and disappeared in a few days, without any scab being formed. At another time, after apparently aborting, or at least taking no action for twelve or twenty-four hours,

they would then, as it were, redevelope and go through the ordinary course.

It was not till 1863, two years after the attacks of herpes had commenced, that they began to be associated with pains. These were at first confined to the right lower extremity; and attacks had been since occurring, when I saw him, at intervals of a month or so. Their character will be particularly described later on.

He gave me a graphic account of the onset of his ocular disturbance. It seemed that in October, 1862, after a severe attack of diarrhœa, he suddenly found an imperfection of sight in his right eye. In reading he observed that he failed to see the end of the word at which he was looking; next he found that whilst looking at the top line of a page he could not see the second or third line without lowering the eye, so that ere long he had to put his finger on a line, dropping it as he read, and in this way spelling out the words and lines. This condition, as regards his right eye, stretched over a year. In the autumn following he took a holiday, and amused himself by sketching from nature. He found it necessary to paint by the aid of his left eye, shutting out the right by means of a piece of cardboard suspended from his hat. His health, though then improved, was not up to the old standard, sleep being uncertain and digestion imperfect.

In October, 1863, exactly one year (almost to a day) after the failure of the right eye, the left was similarly attacked. He had been out sketching, the sight of the left eye being perfect; in the night following he was attacked with diarrhœa, which recurred in the morning. Next day he found that he could not see the ends of words with his left eye, and, as with the right eye, so the sight of the left gradually deteriorated, and was diminishing at the time when he first came to me.

With the right eye at the present time he can see two window-lights as two, but there is no evidence of sashes. Very thick black hands on a white-faced watch are quite indistinguishable.

With the left he can see the angle formed by the black hands of his watch, and thus get an approximation to the hour. He cannot in the slightest degree recognise any one by sight, nor can he walk about without a companion to lead him.

In cases of this kind, colour blindness may often be detected before the atrophy of the optic disc is very far advanced. The colours green and red are usually those with which the difficulty is earliest marked.

Another patient of mine,* who is ataxic, and has now advanced atrophy of his optic discs, states that his first difficulty was that he could see no colour in a scarlet geranium. Red gravel looked grey to him. Soon afterwards the grass looked grey and he could not, at a little distance, distinguish it from the gravel. When examined, the only colour to be recognised was violet, which he said looked blue. A medium blue was seen as white.

The patient, A. B—, whose case we are considering, tells me that the colour blue is not seen as such by him, but as bright light. A piece of bright blue ribbon looks like a bit of burning magnesium wire. If he is walking under a bright sun upon yellow gravel, the shadow which he casts (and which under such conditions is of course very blue in tone) is actually perceived by him as something lighter than the sunlit gravel upon which he treads. And so also the shadows of trees, at a little distance, come out quite light in consequence, as he supposes, of the influence of the blue in their composition. In photography, he reminds me, the colour blue comes out white.

A young man, to whose case I have referred in a previous lecture,† described scarlet as "brownish-yellow," green as "slaty-brown," and a vivid magenta as "pale blue." Blue and yellow were named correctly.

To return to the case of A. B—. His sight has continually, but very slowly, deteriorated since his eyes were first affected. The pupils are of moderate size and equal.

* Case quoted in 'Medical Ophthalmoscopy,' Gowers, p. 100.

† Page 152.

There is no manifest affection of the external muscles of the eyes, and there has never been any double vision.

Examined directly by the ophthalmoscope, in the right eye the optic disc presents a marked change in colour and character. Instead of the normal warm white or pinkish tint with fine striation, it is of a cold, slightly bluish-grey tone, which suggests the idea of its being painted in opaque oil colour. Its surface is flat, dense, and uniform, no signs of any central depression or *lamina cribrosa* being perceptible, and the sclerotic ring appears to be merged into the disc, the outline of which comes out sharp against the rich red of the choroid. There is no marked diminution in the size of the retinal vessels, but they appear as though applied to the disc, and subdividing near its upper and lower border leave the large bulk of it free from any trace of a blood-vessel. In the left eye the appearances are practically the same, except that there are a few more minute vessels on the disc than are to be found in the right eye.

A. B— remained under my observation at this period (1864) for three or four months. During this time the symptom of which, next to the insomnia, he most complained was the very frequent occurrence of nocturnal emissions. These would happen sometimes four or five nights together, recurring after an interval of a few nights, and repeated altogether from ten to fifteen times in a month. He had always since puberty been troubled a good deal in this respect, but the discomfort had rather increased than diminished.

Meantime the pains which had begun in 1863 continued to recur at short intervals. In 1867 my attention was particularly directed (as I find by my notes) to the pains from which he suffered, and also to the herpetic symptoms, which he informed me were a repetition of what had occurred on various occasions during the preceding six years. One such attack occurred in December. He woke up in the night with terrible pain in the right sciatic nerve, which he described as "knife-like spasm." It

persisted, occurring at intervals of a few minutes only, all the following day. This kind of pain, he said, had been liable to attack one or other of his lower extremities—and especially the right—in the thigh, knee, leg, or ankle. It would generally last a week, and was always associated more or less with an eruption of herpes. The pain usually came simultaneously with, or preceded, the herpes by a few hours, but on the other hand it sometimes, although less frequently, followed the appearance of the eruption. The pain might be in various parts; the herpes *always came in one place*. It always occupied the upper part of the right buttock. Even when the pain attacked the *left* lower extremity, the herpes came in the right buttock. He calculated that during the preceding four years he must have had at least fifty or sixty of such attacks of herpes. A "good healthy crop," as he described it, would take from ten to fourteen days ere it subsided. The pains generally continued throughout, and ceased about the time that the scabs were scaling off. On a very rough average he would have such attacks once in four or six weeks. It appeared that between twenty-four and thirty years of age he used to be affected very frequently indeed with herpes præputialis, which sometimes occupied the prepuce alone, but on other occasions extended to the base of the penis and scrotum.

For nineteen years this patient has suffered every few weeks (with a certain exception) from attacks of pains which he describes as "forked lightning." The seizures were closer together and more violent in character from the end of 1872 to the middle of 1874. During this period of about eighteen months he would have a bad recurrence every two or three weeks.

I have notes of one such attack which it may be interesting to transcribe.

"1873, Feb. 14.—Pains which were formerly rare now come very frequently, and with tremendous force. The pains always now affect the left leg; formerly the right was alone subject to them. He shows me a point two or

three inches above the left knee, which has been an especially frequent seat of pain. Over a space not bigger than a crown-piece it is as though some one was giving him repeated violent digs with a knife. The present bout came on yesterday at 5 p.m., and has never ceased for more than two minutes at a time. He knows from experience (and the event proved that he was right) that this bout will go on for four or five days, gradually getting less and less acute, and with longer intervals, until he ceases to suffer. And he will be pretty sure to have herpes. Sometimes the herpes appears first. He will have a patch of heat and itching on the buttock, and then he gets a "fine crop" of herpes. Sometimes the vesicles are abortive, and do not come to the breaking stage. Then the pain does not come so rapidly. The herpes is always confined to the *right* gluteal region, and never passes to the left of the spinal column. I can see now some scars of recent attacks . . . .

"The part where the pain first begins always remains as it were the camp or head-quarters of it—pains radiating from it as a sort of focus. So to-day he has had occasionally pains in the left shin and calf, but the left heel, where the pain commenced, has continued to be its principal seat."

From the middle of 1874 till August, 1876, he enjoyed almost complete immunity from these attacks. There would be, it is true, at rare intervals a little herpes, accompanied by a faint reminder of his pains, which he graphically describes as "sheet lightning," in comparison with their ordinary forked-lightning character.

I may say here that I have often known long periods of immunity from pain occur in tabetic patients who were under no medical treatment. The possibility of this is very necessary to be borne in mind ere we jump to conclusions respecting the efficacy of any therapeutical measures. During the period in question this patient was under no treatment.

As regards the relation of the herpes to pain, A. B—

says that perhaps once in six times the eruption will occur without any pain whatever, or at the most, with a very little "sheet lightning." Very rarely indeed has he had pains without any herpes, although the eruption has been sometimes so insignificant that it would have escaped notice but for his long experience of it. As a rule, there would only be one crop of herpes, but occasionally a second crop would appear near to the first. In the years of only moderate severity the intervals of attacks of pain would be from six weeks to two months or so. Since they recurred severely in August, 1876, they have continued to trouble him at about this rate.

Not long ago I had the opportunity of examining carefully an eruption of the kind. About two inches above, and to the left of the sulcus between the nates, very near therefore to the sacro-iliac synchondrosis of the left side, I found a single group of seven or eight vesicles of about the size of a large pin's head—some larger, some smaller. They were collected (almost all touching each other) upon a base the size of a threepenny piece, the skin of which was slightly pink in colour, and when pinched up felt hard, as compared with the adjacent integument. The vesicles contained transparent and colourless fluid. This group had appeared two days previously. At the same time I observed one or two brown scabs (the remains of vesicles) on the upper and inner part of the left thigh, where the scrotum rests upon it. On the following day the base was less hard and paler in colour, the vesicles flattened and shrivelling.

It is only since 1873 that herpes has appeared to the left of the spine. Before that time it always came upon the right buttock. Since then, however, it has been, if anything, a little more frequent on the left buttock than on the right, and most frequent of all, perhaps, about the middle of the sacrum and the sulcus of the nates. Herpes has never shown itself on both sides at once.

It is to be noted that his pains do not appear, at least commonly, to originate in the buttocks, which, however,

are the constant seat of the herpetic eruption. Occasionally, it is true, the pain will seize him in either groin, and then shoot round over the hip to the buttock; but these are much less frequent sites of pain than others. He cannot say which is the most frequent site.

About four or five years ago he began to experience what he calls "sheet-lightning pains," inclined now and then to develop into a passing "forked lightning," in the right side of his chest. On one occasion I saw him when he had suffered for twelve hours, at intervals of only two or three minutes, from attacks of "forked-lightning pains," which invaded the right side of the chest from about the second to the sixth rib, and appeared to go through to the scapula—corresponding apparently with the intercostal nerves. They were not accompanied by any herpes.

A. B— is of healthy aspect and fair complexion, his brown hair being but slightly tinged with grey. His disposition is of the happy kind which is, as some one has remarked, not seldom found in patients suffering from tabes. There is marked arcus senilis in each eye. His arteries show no signs of thickening. The action of the bladder is normal; the bowels regular, and less disposed to diarrhœa than formerly. He does not suffer from any constrictive feeling in the waist. His digestion has improved, and he has increased in weight during the last few years.

Examination shows the nates fleshy and firm, the skin covering them, and the sacral region generally evincing no sign of anæsthesia. There are a few slight scars of recent attacks of herpes. One may see, perhaps, traces of three or four, but all except the most recent are very faint, and in strong contrast with the marked cicatrix which often follows an eruption of ordinary shingles. Of the attacks which took place at periods antecedent to a few months ago, not a vestige remains.

This gentleman, notwithstanding his blindness, walks without the slightest tendency to stagger, and this even

if (as he will sometimes attempt upon a quiet and straight country road) he dispenses with the arm of a companion. His walking power, however, is a good deal diminished; he cannot now manage three or four miles at a stretch without distress. The cutaneous sensibility is absolutely normal in all respects throughout his body. Nor is there any affection of the muscular sense. He knows exactly where his legs are, and can direct his feet readily. So also he can make his two forefingers touch at their points with as great precision as any one in health.

The character of the optic atrophy, and of the pains, together with the absence of the knee phenomenon, all combine to leave no doubt as to the nature of this case. It is evidently one of sclerosis of the posterior columns, although that particular symptom which caused the name of progressive locomotor ataxy to be given to this class of disorder is absent.

The absence of incoordination of movement, whilst other symptoms of the disease—notably the flying pains—are well marked, is, as I have already frequently remarked, not at all uncommon in tabes dorsalis, and it is interesting to note that nineteen years have elapsed in this instance since the commencement of the characteristic pains, and as yet no sign of ataxy has presented itself.

Charcot and Bouchard have published * particulars of a female patient who died of some accidental disorder after having suffered during fifteen years from lightning pains. Up to the time of her death she walked without embarrassment or projection of the legs; she did not strike the ground with the heels, and closure of the eyes did not affect her security in standing or walking. At the autopsy, the only appreciable lesions (except some doubtful traces of meningitis) were found in the posterior columns, and consisted in a multiplication of the nuclei of the neuroglia, with thickening of the meshes of the reticulum, but without, it was thought, concomitant alteration of the nerve-tubes.

For twenty-one years A. B— has suffered from attacks

* 'Comptes Rendus des Séances de la Société de Biologie,' Année 1866.

of herpes, and according to his own computation the eruption has come out upon him not less than from 200 to 250 times. Whilst the pains have been principally referred to the periphery of the sciatic nerves, the accompanying herpes has always, it will be noted, occurred on that part of the skin which is innervated either by the small sciatic or by the posterior branches of those sacral nerves the anterior divisions of which go to form the sciatic plexus. Some of the pains, it is true, have been referred to districts innervated by the anterior crural nerve; but I think I am right in saying that where these have occurred they have not been alone. On the same occasion the district of the sciatic nerves has also participated in the paroxysms. What appears at first sight, therefore, as a great anomaly, viz. the confinement of the herpes to a very small district, the pains meantime ranging over various parts of the lower extremities, is found to be no longer so much so when we remember that the nerves which supply the skin at the part where the herpes was wont to appear have a common origin with those which are distributed throughout the length of the lower extremities.

The case of Charcot's which I have quoted goes far, by exclusion, to prove that we may look for an explanation of the lightning pains of tabes to the irritative alteration of the posterior columns of the spinal cord. The researches of Bärensprung amongst others have shown that inflammation of the spinal ganglia will produce herpes in the district of the corresponding nerve. Weir Mitchell, in his admirable work on 'Injuries of Nerves,'* remarks that some time in the history of a nerve injury, it is common to see certain forms of eruption, which are herpetic, vesicular, or in the shape of bullæ. There is ample evidence also from other sources that irritation of nerves will give rise to eruptions usually of a more or less vesicular character in the district of skin which is innervated by the affected nerve. Whether an irritative process

* Philadelphia, 1872.

attacking the nerve-tubes in their passage *within* the cord would be attended with a like result we are not able to determine, but it seems not improbable that such might be the case. If this be so, the concomitance of herpes, with paroxysms of pain, in my patient, would appear capable of explanation by supposing that such a condition of the cord obtains in this instance as was found in Charcot's case. But it does not appear so easy to explain the occurrence of herpes *alone* for two years before the pains began. So far as it goes, this circumstance would tend to support the hypothesis of a special system of trophic nerves, which it might be imagined were alone influenced until the growth in the neuroglia attained a certain development. Nor does it seem unreasonable to suppose that the herpes præputialis from which this patient began to suffer when he was twenty-four years old may have been really the first outcome of that diseased condition which has since taken so marked a form. It might be well, perhaps, in cases of præputial herpes, to bear in mind the possibility of the local symptom being part of a systemic disease, and to test the patient as to the presence of knee-phenomenon.

LECTURE IX

OPHTHALMOPLEGIA EXTERNA WITH TABES DORSALIS—GASTRIC CRISES

On previous occasions I have insisted upon the circumtance that tabes, as has been shown by Professor Pierret, essentially attacks the sensory side of the nervous system, the motor troubles being usually, at least for some time, secondary to the lesion of the sensory nervous system.

In a typical example of tabes you will have, amongst other symptoms, lightning pains, cutaneous anæsthesia, absence of knee-phenomenon, delay in the transmission of painful impressions, ataxy, absence of pupillary reflex, diplopia (either existing or past), imperfect action of the bladder, together with, perhaps, atrophy of the optic nerve and deafness. These are all symptoms directly or indirectly referable to lesion which is principally situated in the outer part of the posterior columns, along with the analogues of these in the medulla oblongata. In the course of most cases there is evidence of an extension of structural changes to the motor side of the system. The limbs not only fail in their coordination, but become more and more powerless, a condition which points to an invasion of the lateral columns of the spinal cord by sclerosis. Nor is this all. In certain examples, along with this distinct loss of power there is muscular atrophy. This is not a generalised emaciation, but certain groups of muscles which examination shows to be functionally associated are picked out by a wasting process. In such circumstances we know that the disease has attacked the anterior horns of grey matter in the spinal cord, at certain

levels corresponding to the anatomical origin of the nerves supplying the muscles affected, and that in these portions of the anterior horns of grey matter more or less of the large ganglionic cells have been destroyed. Such conditions as these are common enough, especially in examples of tabes of old standing.

The cases upon which I propose to speak to-day, on the other hand, are not very common, and they are remarkable in this, that not only is the invasion of the motor side of the cerebro-spinal system an early symptom, but it is so strongly pronounced, and the lesions occupy so unusual a position, as to give rise to symptoms which dominate the ordinary signs of tabes and create a complex condition which is at first full of obscurity. These are cases in which there is degeneration of the nuclei of certain motor nerves in the medulla oblongata and pons, along with sclerosis, not only of the columns of Burdach (the outer part of the posterior columns), but likewise in parts which anatomically represent the extension of these portions of the sensory nervous system upwards into the medulla oblongata.

I wish to bring under your notice two cases of this anomalous form of tabes, attention to which was first directed by Mr Hutchinson, in a paper upon "Ophthalmoplegia Externa," which he read before the Royal Medical and Chirurgical Society in 1879. One of these, a female, is at the present time an out-patient, having recently been an inmate of the hospital. The other, a male, died in the hospital, and an autopsy was obtained. Portions of the hardened nerve-centres were forwarded to Dr Bevan Lewis, of the West Riding Asylum, Wakefield, who has been kind enough to examine them microscopically.

We will first consider the case of the young female, whose principal symptoms I will indicate as briefly as possible.

C. D—, a female, æt. 25, was admitted into the National Hospital for the Paralysed and Epileptic on

January 16th, 1879. The following account of her case is partly derived from notes which were made by Mr A. E. Broster, who was at that time the Resident Medical officer :

On the right side there is semi-ptosis of the eyelid, together with paralysis of the recti and obliqui muscles, which appears to be absolute, with the exception that the inferior rectus can slightly move the eyeball.

On the left side there is ptosis of the eyelid with paralysis of all the recti and obliqui, with the exception of the external rectus.

The right pupil is 4 mm., the left 4·5 mm. in diameter. There is no action to light in either, but they act a little during attempted accommodation. (I may say, in general terms, that no muscle of either eye acted properly except the left external rectus.) There is no affection of cutaneous sensibility or of motor power in the range of the fifth nerve.

Taste and hearing are good, the tongue is protruded straight, but with some quivering. The right side of the face does not move so perfectly as the left.

The arms are somewhat weak. The grasp of the right hand measures 52 and the left 55 by the dynamometer. There is slight anæsthesia to touch, and pain in the first two fingers of the right hand. When asked to touch the tip of the nose with the right index finger she missed it altogether, but reached it, though with some difficulty, with her left.

As she sits she can raise both knees, but on trying to stand her legs give way, and she needs the help of two persons to walk. Her gait is characteristically ataxic.

She cannot stand by herself with her eyes open ; if they are shut the difficulty is greatly increased.

She feels the ground imperfectly with the soles of the feet, and in these she gets acute "pins and needles," which cause her legs to "jump." The muscular sense of the lower extremities is diminished. The knee-phenomenon is absent in each leg. There is no foot-clonus.

Besides a general emaciation of the arms and legs,

certain muscular groups are picked out by atrophy, especially the right serratus magnus and right interscapular muscles—so that the right scapula tends to drop by its weight—the right pectoralis major, and both sternomastoids. The trapezii and the muscles of the back generally are thin. There is atrophy of the glutæi, especially on the left side.

The patient suffers occasionally from very severe spasms and pain in the epigastric region, together with vomiting (gastric crises).

She complains that she is scarcely ever free from pain. There is a burning pain at the back of the neck and behind the ears, a heavy aching feeling across the forehead, and, in addition, she has very sharp and sudden pains coming and going in the skin at the top of the head. Her arms feel as if they were being gnawed, and there have been sharp shooting pains in the course of the median nerves, leaving an after-feeling of soreness.

She has shooting pains in the lower part of the abdomen, and what she describes as a "dull" pain in the vagina. There is a feeling of fulness in the abdomen and of oppression over the lower half of the chest, which is so severe at times that it is as though suffocation were imminent.

She has a constant sense of starvation in spite of eating largely. Her own words are, "I always feel hungry —always feel starved. When at home I never seemed satisfied, and ate all sorts of things; in fact everything I could get hold of."

The tongue "feels hard," her throat is dry, and she has a sensation of choking. She can swallow without difficulty. There have been at times severe cramps in the left flank. She has numb feelings and creeping sensations down the legs and thighs, with aching of the knees. If her legs are allowed to dangle they feel as though they were being screwed off at the knees.

Seated in a chair she feels giddy and appears to lose all power in the back. A month ago she suffered from

what is described as a "starting" pain (so sharp that she was compelled to cry out) in the front of both thighs, and at times a similar pain has come across the dorsum of either foot, causing the feet to start.

When 17 years of age this girl contracted a chancre, which was followed by sore throat and copper-coloured rashes. At 22, according to her account, she had a bad cold, after which she was always weary, and could not rid herself of a tightness in the chest.

She had severe aching pain in the back and vagina, with frequent desire and imperfect ability to micturate. About the same time there was internal strabismus of a passing character. Next she complained of burning sensations in her loins, and her belly felt as if it were unduly large.

A few months later her right eyelid began to droop, and she had giddiness and pains in the legs, "sharp like needles," causing her legs to jump. The right eyelid recovered its power, and then the left lid drooped. She went into a hospital and her legs began to fail her. She staggered as if drunk, the right shoulder wasted, and the right eyeball became fixed, as it is now, and the left eyeball began to follow its example.

Next the right lid began to droop again and she gradually wasted. The pains have become more frequent and severe.

She was admitted into our hospital in January, and discharged in May.

The treatment consisted in mercurial inunction carried to mild salivation, together with iodide of potassium, in doses of 20 grains, three times daily. Her pains were a good deal relieved by extract of Indian hemp, but beyond this she could not be said to have derived any particular benefit from the treatment, and she quitted the hospital in much the same state as that which she presented on admission.

Before making any comments upon this case it will be convenient if I describe the other one.

X. Y—, æt. 36, served for 16 years in the army (10 years in India), and was invalided on account of heart disease.

He was married and had six children. In his family history there was nothing of note except that he had a sister who was paralysed. For himself he had rheumatic fever ten years before his admission here, and had once suffered from gonorrhœa, but never to his knowledge from syphilis.

He was admitted into the hospital in 1880.*

By his own account his illness only began in the preceding February, but it appeared on inquiry that for five years he had been subject to dragging "rheumatic" pains, which were not sharp or sudden.

Not till six months before he applied here did he begin to have sharp, darting pains in the legs, and also in the fingers. They were very sudden, and described as being like "sparks of electricity" recurring through his limbs. About March he began to stagger in walking. He never lost sensation.

About two years previously he had begun to see double and had "twitching under the eyes." His sight had got bad since Christmas, but he was not aware that his eyes were fixed till his attention was called to it here. He had rapidly become very deaf, and for three months there had been difficulty in swallowing. He had nearly choked on several occasions. He had not suffered from attacks of vomiting.

His state, in September, as taken by Dr Beevor, was as follows:

Patient appears considerably older than his years. He has a dull, heavy, listless look, which seems to be due to his eyeballs being motionless. The eyelids droop, the right more than the left. He can close them apparently with equal ease on either side, but in opening them he is unable to raise the eyelids to a normal extent, and the right still less than the left.

* The substance of this lecture was delivered in 1879. The case of X. Y—, which occurred afterwards, is now added.

The eyeballs are rather prominent. They are completely fixed, the gaze being always directed straight forward, with a slight divergence of the optic axes. The right pupil measures 3·5 mm., the left 3 mm. They are both insensitive to light and do not contract when he tries to look at a near object, but it is to be observed that he is unable to converge the eyes owing to the paralysis of the external muscles of the eyeballs.

The masseter muscles contract firmly and equally. He cannot whistle, and says his food collects between his cheeks and teeth, especially on the left side. The tongue is protruded almost straight, but slightly inclined to the left.

The patient can swallow liquids pretty well, but takes a long time to swallow solids, especially if they are dry, like bread. Smell and taste are not apparently affected. Hearing on both sides is so bad that he needs to be shouted at; the degree of deafness varies. There is subjective sensation of booming of bells, especially in the right ear.

By the ophthalmoscope no change is to be noted in the optic discs. He can just read 8½ Snellen with either eye. Colour vision is good. Distant objects, but not near ones, sometimes appear double.

The skin of the forehead seems rather less sensitive to touch on the left than the right side, but there is no marked impairment of sensibility in the region of the fifth nerve. In the rest of the body it is perfect. The upper limbs are somewhat wasted about the shoulders and forearms, but not to any marked extent. The movements of the arms are free, and the grasp 35 with the right, 30 with the left hand.

The lower limbs are rather more wasted, and the calf muscles are especially flaccid. He has free movements with his legs, and considerable power in them when lying down. The wasting of the limbs, I should say, was of the nature of general emaciation, not of progressive muscular atrophy.

When his eyes are closed he staggers, and would fall. In walking he is very shaky, especially when he turns. He does not bring his feet down with a stamp.

The patellar tendon-reflex is absent on each side. There is no ankle-clonus. Sole-reflex is present, but small on each side.

The cremasteric reflex is absent; the abdominal only imperfectly obtained; the epigastric is marked. The faradaic excitability of the muscles of the thighs and legs is very slightly indeed below the normal.

There is no delay in the transmission of impressions of touch or pain, and he can localise correctly the slightest touch on the arms and legs.

The lungs are free from dulness. A few râles are heard in the interscapular region. At the base of the heart there is a loud double see-saw murmur, the pulse 68, of water-hammer character. The brachial arteries at the elbow are tortuous, and roll under the finger. The urine contains no albumen or sugar.

When this man had been about three weeks in the hospital he was taken one day (September 28th) with a fit of coughing, which was prolonged till he was sick. He was then seized with intense dyspnœa. The face and lips suddenly became of a livid blue colour; the mouth, which was opened wide, gave exit to a moaning cry of very distressing character. He was immediately got into bed and propped up, whilst brandy was administered by enema. After about twenty minutes he was able to speak, having gradually recovered from the dyspnœa. His mind appeared confused for a little time afterwards.

Next day his temperature was 100·6°. There was no dulness in the chest, but râles were heard at the bases of the lungs.

On Sept. 30th he began to have such difficulty in swallowing that food was thenceforth administered by enema. He gradually got lower. On Oct. 2nd the catheter required to be used. The pulse numbered 100, regular, but very compressible. The respirations were 28 in the

minute. There were râles in the chest. On Oct. 3rd his temperature, which in the morning was 102·6°, marked 104° in the afternoon, and in the evening 101·2°. He became excited and delirious. The urine contained a little albumen. Next morning he appeared almost moribund, but answered questions, and was able to swallow egg and milk with brandy. His morning temperature was 102·2°, pulse 104. Respirations 40, very shallow. There was no dulness to be noted in the chest, but râles continued at the bases of the lungs. During Oct. 5th he continued to sink, attacks of dyspnœa occurring two or three times in the day, and on Oct. 6th he died.

An autopsy was made nineteen hours after death. There was nothing noticeable about the dura mater or the general aspect of the brain. On removing the cerebrum some of the cranial nerves could not be found. The right third nerve was seen *in situ*, but the left escaped observation.

On dissecting the outer wall of the cavernous sinus, the third and fourth pair could be made out, but not the sixth. The fifth, seventh, eighth and ninth nerves were seen.

The nerves which could not be discovered had no doubt dwindled so much in size as to escape recognition. Section of the spinal cord disclosed no change to the naked eye.

The right side of the heart was full of dark clotted blood. The left side contained some ante-mortem clots. The mitral valves appeared slightly thickened. The aortic valves were healthy in appearance, but the sinuses of Valsalva were much dilated, and the whole of the ascending aorta was dilated.

The spinal cord with the medulla oblongata and portions of the mesocephale were hardened and submitted to microscopical examination by Dr Bevan Lewis.

The following is an abstract of the account which he has been kind enough to give me of the results of his examination:

In the spinal cord very advanced degenerative changes

were found, and these were invariably limited to the posterior columns. The lesion, which was of the nature of grey degeneration, appeared to have commenced in the lumbar portion, for a later stage of sclerosis was observed there than in the higher portion. The sclerous tissue consisted not only of fibrillated connective, but of numerous multicaudate cells, which differed only in number, size, and readiness of staining from the normal cells of the medullary septa supporting the vascular network of the cord. These modified elements threw out numerous fibrous prolongations, and were generally associated with large, dilated, and tortuous blood-vessels, the walls of which were covered by proliferating nuclei.

The most advanced sclerous change was found in the post-commissural zones throughout all the regions of the cord alike. The change extended in a less intense degree along the posterior root zones, implicating the radicular fibres and causing their destruction to a slight extent in the cervical, to a still less extent in the lumbar, but to a very grave extent in the dorsal region of the cord.

The columns of Goll throughout the cervical region were almost completely sclerosed, but the degree of degenerative change was less than in the regions above mentioned. In the dorsal region a very limited patch of degeneration was found in the posterior median columns, which were otherwise wholly unaffected by the lesion. The median raphé and the posterior border of the same columns in the lumbar region were sclerosed and distorted. In the lumbar region there was a small cyst-like cavity in the gelatinous substance of the posterior cornu on the right side.

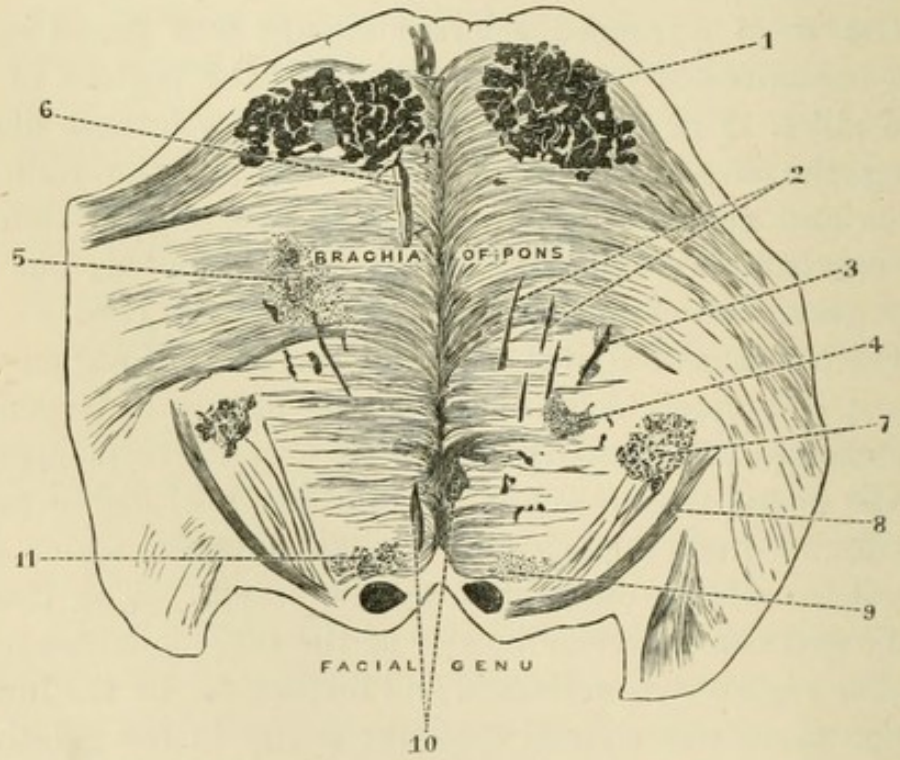
The posterior cornua were invaded both in the cervical and dorsal regions to a slight degree by the same grey degeneration, which extended usually to the point of emergence of the posterior roots. Dr Lewis made a careful examination of the antero-lateral columns in the cervical, dorsal, and lumbar regions, and is able to state positively that the direct and crossed pyramidal tracts, the direct cerebellar tracts, and Gowers' tracts were all free

from any appreciable lesion, the histological elements being well displayed and perfectly normal. Nor was there the least sign of lesion in the anterior cornua at any part of the cord.

Numerous amyloid bodies were scattered throughout the posterior columns of most of the regions of the cord.

Sections were carried through the nuclei of origin of the various bulbar nerves. Of those found along the lower half of the floor of the fourth ventricle—the accessory, hypoglossal, vagus, and glosso-pharyngeal, the

FIG. 16.—REGION OF ORIGIN OF FACIAL AND ABDUCENS IN A CASE OF TABES.



1. Vertical fasciculi from crura.
2. Remains of roots of sixth nerve.
3. Diseased and plugged vessel covered with blood corpuscles.
4. Miliary extravasation.
5. Large focus of extravasation.
6. Blood-vessel in advanced state of disease—plugged.
7. Posterior nucleus and ascending roots of facial.
8. Facial nerve.
9. 11. Abducens-facialis degenerated on both sides.
10. Diseased and plugged vessel.

nuclei, as well as the emergent or radicular fibres were found well displayed and perfectly intact. No diseased tract of tissue was anywhere apparent in the transverse sectional areas of this district, and the restiform columns and olivary nuclei and fibres were throughout free from lesion. Upon reaching the higher level of the origin of the sixth nerve undoubted evidence was obtained of a morbid condition of this tract. The lesion, obviously a vascular one, had secondarily implicated the emergent fibres from the abducens-facialis nucleus—the root fibres of the sixth pair. The nucleus, where it lies in front of the genu of the facial nerve, was represented by mere remnants of degenerated cells and a punctated aspect of tissue from the resulting *débris*. The fibres of origin of the abducens were represented merely by an occasional narrow streak of minute and degenerated fasciculi, taking their usual curved course, but lost to view after a short distance. Along the direction pursued by these diseased fasciculi were several large, swollen, and tortuous blood-vessels, perfectly occluded by dark clots, their coats extensively diseased, and frequently crowded by heaps of red blood globules. Along the course of the same fibres of the sixth pair were several miliary apoplexies, which had probably induced the degenerative changes.*

Unfortunately the pons and medulla oblongata had incurred such damage in the process of preparation as to interfere with an exhaustive examination.

In the case of C. D— we have all the characteristic symptoms of tabes dorsalis. There are pains sharp and sudden in character, an ataxic gait, absence of knee-phenomenon, loss of pupillary reflex, loss of muscular sense, some cutaneous anæsthesia, gastric crises.

In X. Y—, too, although there are differences in details, the clinical evidence of tabes is equally certain, and is confirmed by the microscopical examination. Everything necessary to establish them as examples of locomotor

* The complete report, of which the above is an abstract, will be found in 'Brain,' April, 1882.

ataxy is present, but in addition to these symptoms there is another which it is not customary to find in that disease. I refer to the symmetrical immobility of the eyeballs accompanied by more or less ptosis of the lids. This condition was first described by Graefe under the name of ophthalmoplegia progressiva. An account of it is contained in the 'Lehrbuch der functionellen Nervenkrankheiten,' by Eulenburg (Berlin, 1871). I can remember that, nine or ten years ago, shortly after reading that account, I saw an example of the disease in St Bartholomew's Hospital. The patient was a female, under the care, I think, of Dr Andrew, and she had this symmetrical immobility of the eyeballs typically marked. The disease has more recently received, as I have mentioned, a searching investigation at the hands of Mr Hutchinson, who has applied to it the term ophthalmoplegia externa, in contradistinction to that of ophthalmoplegia interna, which he had given in a previous paper to a state of immobility of the pupil, in which the internal muscles of the eye (the iris and ciliary muscles) are together involved in paralysis. He, moreover, explains that the former term must be understood as including very frequently, if not usually, the immobility of pupil.

Mr Hutchinson's description of the condition is so graphic that I cannot possibly do better than quote it here:—"Drooping of the eyelids," he writes,* "so as to give to the face a half-asleep expression, is usually the first symptom, and it is soon accompanied by weakness of all the muscles attached to the eyeball, so that the movements of the latter become much restricted, or even wholly lost. The condition is usually bilateral, though it is not always exactly the same in degree on the two sides. Its symmetry probably denotes that it is of central origin. It by no means always happens that all the ocular muscles are alike affected, or that they are attacked simultaneously, still it is a very marked feature of the malady that the muscles fail in groups and not singly." Seventeen cases

* 'Transactions Roy. Med. and Chir. Soc.,' 1879.

of the kind are described in the paper. In ten of these it seemed certain that syphilis was the cause, in eight acquired, and in two inherited. Of the remaining seven Mr Hutchinson remarks: "It may be said that a reasonable suspicion of syphilis might be entertained in several." In one case an examination of the brain after death was made by Dr Gowers, who found that degenerative changes precisely similar to those seen in progressive muscular atrophy had implicated the origins of the third, fourth, and sixth nerves, as well as, in that particular instance, the optic nerves also. The large nerve-cells had disappeared from the nuclei of origin of these nerves.

Mr Hutchinson remarks that we have in such cases a very close parallel to the so-called bulbar paralysis—the labio-glosso-laryngeal paralysis of Duchenne—and he thinks it may be plausibly conjectured that the initial lesion is inflammation of the nuclei of the affected nerves, which, in a slowly serpiginous manner, creeps from place to place along certain definite anatomical paths.

In C. D— it will be observed there is a symptom which (as it happens) is not described as occurring in any of those related by Mr Hutchinson. I refer to the atrophy of muscles about the back and shoulders. This association evidently tends to support the view that the lesion of the nuclei of origin of the cranial nerves is of the same nature as that which determines progressive muscular atrophy. In Charcot's lateral amyotrophic sclerosis there is atrophy of the large motor cells in the anterior cornua of the cord, secondary to sclerosis of the lateral columns. The disease tends, as we know, to travel upwards, and in time spreads to the medulla oblongata. Attacking there the nuclei of the hypoglossal, facial, and trigeminal (its motor portion), it causes death by involving also the nucleus of the vagus. Now, it seems probable that, were it not for the fatality necessarily attaching to the destruction of this nucleus, the disease would also, in those cases, be likely to invade the nuclei

of origin of the oculo-motor nerves, and so bring about the condition of the eyes which is to be seen in this girl. Death, in fact, stops the progress of the disease before it invades the region devoted to the innervation of the external and internal muscles of the eye. This would appear to be the explanation of the comparative rarity of the affection.

The lesion of nerve nuclei would appear, in the case of C. D—, to be a more or less continuous one, from the intracranial centres for the higher cranial nerves to the anterior cornua of the cord. The condition of the eyes shows that the nuclei of the third, fourth, and sixth nerves have been invaded by the disease. The pains in the head would imply that there is probably also lesion involving the sensory root of the fifth. On the right side the origin of the facial nerve is involved, though not apparently on the left. The quivering of the tongue shows that the nucleus of the hypoglossal is probably not intact.

This patient suffers at frequently recurring intervals from attacks of violent pain in the stomach accompanied by vomiting, not dependent upon the ingesta, which are exactly of the kind described by Charcot under the name of *crises gastriques*.

In a communication brought before the Pathological Society of London in February, 1880,* I made the suggestion that gastric crises depend upon irritation of the nucleus of the vagus by sclerosis. At that time I had no anatomical evidence to offer in support of the hypothesis, which was based upon the paroxysmal character of the attacks, so completely in accord with that characterising the attacks of lightning pains. It appeared evident to me that if sclerosis, which when it attacked nerves of common sensation produced pain, came to invade the nucleus of the vagus, it might be expected to give rise to symptoms like those of the gastric crises. During the meeting of the International

* 'Transactions of the Pathological Society,' 1880.

Medical Congress in London, 1881, Professor Pierret, of Lyons, showed me sections of the medulla oblongata which he had recently made from a case of tabes with gastric crises. I was greatly interested in his demonstration that the fasciculus gracilis in immediate relation with the nucleus of the vagus exhibited distinct sclerosis. It will be remembered that the funiculus cuneatus and gracilis, which closely adjoin the sensory portion of the pneumogastric nucleus, represent the continuation of the posterior columns of the spinal cord upwards to the cerebellum through the medulla oblongata.*

In the atrophy of the sterno-mastoids and left trapezius there is evidence of invasion of the nuclei of the accessory.

The intense hunger, of which this girl complained, is a symptom that I have frequently observed in connection with the occurrence of gastric crises in tabes. It was marked in a case of tabes with gastric crises and joint affection under the care of Mr Herbert Page. It is present also in the case of a man suffering from tabes with gastric crises, who is now under my care in the hospital.

Only a few weeks ago, whilst looking through 'A System of Clinical Medicine,' by Dr Graves, published in 1843, I chanced upon a description of gastric crises which is so graphic that I cannot refrain from quoting here a rather full abstract of the case narrated. Viewed by the light of our increased knowledge, the case is evidently one of tabes dorsalis, but it occurred long before this

* Whilst this work is going through the press I note a paper by Dr Emile Demange ('Revue de Médecine,' No. 3, Paris, 1882) on the "Chute spontanée des dents, et Crises Gastriques et Laryngées chez les Ataxiques." In a case marked by spontaneous shedding of the teeth (without caries), and also by gastric and laryngeal crises, histological examination of the medulla oblongata showed the greater part of the nuclei of origin of the bulbar nerves plunged in the midst of sclerous tissue. The nucleus of the mixed nerves (glosso-pharyngeal, vagus and spinal accessory), as well as the ascending root of the fifth and other structures, were evidently involved in this sclerosis. The signs of sclerous neuritis were observed in the branches of the fifth nerve.

disease was differentiated from other forms of paraplegia. The autopsy, which included examination of the brain, spinal cord, and nerves, besides the abdominal viscera, naturally failed to throw light upon the cause of the disease, for in those days the methods of hardening preparations of nerve substance, the initiation of which we owe to Lockhart Clarke, had not been devised, and the examination, as far as I can judge, was only made with the naked eye. Dr Graves appears, however, to have been curiously near to a discovery of the truth in a remark which he makes. "Are we to attribute," he writes, "this diseased condition of the stomach and bowels, which, from the remarkable periodicity of its occurrence, was evidently functional, to irritation, congestion, or inflammation of the brain or spinal marrow? From the data we are in possession of it appears that this question must be answered in the negative." He speaks of the case as a very remarkable one, which had made a great impression upon him, and, in his view, was well worthy of the attention of the pathological inquirer.

The patient, Mr B—, æt. 23, was exceedingly strong, and passionately fond of hunting, fishing, and shooting. These habits, however, he laid aside after the occurrence of the first attack of his illness, which happened in 1829. From that time his bowels, previously sluggish, became inclined to looseness, which always increased before the appearance of one of the attacks, accompanied by griping, nausea, and inclination to vomit. Each attack was generally preceded by a copious secretion of insipid watery fluid in the mouth, and then the characteristic symptoms of his disease commenced. These consisted in obstinate and protracted nausea and vomiting. He first threw up whatever happened to be on his stomach at the time, and afterwards everything he swallowed, whether solid or liquid, and the quantity ejected in the course of a day varied from three to four quarts of fluid. He complained also of pain, referred to the stomach or lower part of the chest, which continued throughout the attack,

being most acute at its commencement; for the last year this sensation had passed into a feeling of painful constriction, which he described as a "contracted feeling of his inside," and compared it to something like the effects of a cord drawn tightly so as to compress or strangulate his body exactly along the outline occupied by the insertions of the diaphragm. During the prevalence of the attack he had profuse perspirations, particularly towards the termination of each paroxysm. The duration of the first attack did not exceed four or five days, after which he became quite well, and continued so for six or seven months, when his symptoms suddenly returned. He began to reject everything from his stomach as before, but in the course of a few days the vomiting disappeared, and for a considerable interval he had no return of his complaint. In the year 1830 he had three attacks of a similar description; from these he recovered also completely, and without remarking any diminution of power in his lower extremities. In 1831, however, the disease began to assume a more serious aspect; the paroxysms became much increased in severity, lasted longer, and recurred at shorter intervals. For one of these attacks he took mercury, and was salivated. In 1832 his symptoms became still more violent, and the duration of the paroxysms more protracted. He had one in March, a second in May, and a third in June, each of which was accompanied by some numbness and loss of power in the lower extremities; this, however, was slight, and disappeared altogether as the vomiting subsided. About this time he noticed that his urine was scanty, and deposited more sediment than usual. He also complained of being very apt to catch cold whenever he got out of bed, and stated that he suffered occasionally from *severe twitches and pains in his legs, thighs, arms, and other parts of his body*, which were generally succeeded and carried off by profuse perspirations.

In August, 1832, he had a violent attack, which lasted nearly a month. The vomiting was incessant, continuing

night and day, and he suffered severely from the feeling of painful constriction already described. On getting up after this attack his legs suddenly failed him, and he dropped down on the floor quite powerless. The paralysis did not now disappear during the intervals, although it grew somewhat better after each fit of vomiting had ceased; indeed he used to improve in his walking after the paroxysm had entirely disappeared, and, aided by two sticks, supported himself so as to give some hopes of a recovery, until a recurrence of his attack reduced him again to a state of almost total paraplegia. His legs now began to waste sensibly, and he noticed that they had lost their feeling and were remarkably cold. He also complained of severe twitches of pain in various parts of his body, accompanied by profuse night sweats, and turbid scanty urine.

For some months before his death he was completely paraplegic, and continued to be attacked with violent fits of vomiting. The vomiting went on night and day, and he was unable to retain the mildest and most soothing substances for a moment on his stomach. Everything was tried to allay the irritability of the stomach, but in vain. After continuing to resist obstinately every form of treatment for five or six days and nights the vomiting would suddenly cease, the gentleman would exclaim, "Now I am well," and he could then eat with perfect impunity substances of an indigestible character. The transition from a state of deadly nausea and obstinate retching to a sharp feeling of hunger used to occur quite suddenly. One hour he was a miserable object, rejecting everything, and suffering the most painful constrictions across the epigastrium, the next found him eating with a voracious appetite whatever he could lay hold of, and digesting everything with apparent facility.*

Here again we have the symptom of hunger described very graphically. I would submit that it tends to corro-

* Several cases of typical gastric crises will be found described in Lectures XIII and XIV.

borate the view that I take as to the probable seat of lesion occasioning gastric crises.

It may be asked why, if we have to do here with irritation of the nucleus of the vagus, we should not, in cases of tabes with gastric crises, also meet with symptoms referable to the heart and to the respiratory apparatus. We do, in fact, sometimes meet with laryngeal crises, and rapidity of pulse is of no unfrequent occurrence in the course of tabes dorsalis. It does not seem reasonable, however, to expect that in these circumstances we should necessarily find evidence of impairment in *all* the functions subserved by the nerve.

It cannot be doubted, I think, that there is a very elaborate differentiation of function of the nerve-cells constituting a nucleus. In a paper of great interest and importance* Dr Felix Semon calls attention to the proclivity of the abductor fibres of the recurrent laryngeal nerve to become affected sooner than the adductor fibres, or even exclusively, in cases of undoubted *central* as well as peripheral injury or disease of the roots or trunks of the pneumo-gastric, spinal accessory, or recurrent nerves.

The varying amount of paralysis in the muscles of this girl's eyeballs and lids is also indicative of differentiation of function in various parts of the nuclei of the oculo-motor nerves.†

I do not think, therefore, that we shall see anything remarkable in irritation of the nucleus of the vagus in a particular case being followed by symptoms entirely referable to the stomach or intestines. Whilst upon this point I would remark that, in certain cases of tabes where the more typical form of gastric crises has not been marked, there has been, nevertheless, a peculiar tendency not so much to diarrhœa as to abnormally frequent action of the bowels, the evacuations not being necessarily loose. I

* See 'Archives of Laryngology,' vol. ii, No. 3, July, 1881

† Dr Sturge has published some interesting remarks upon this subject, "Two Cases of simultaneous Paralysis of both Third Nerves," 'Ophthalmological Society's Transactions,' vol. 1.

think it probable that this symptom, like the pain in the stomach and vomiting, may prove to depend upon irritation of the nucleus of the vagus, for it must be remembered that the peristaltic action of the small intestine is largely influenced by nervous impulses passing along the splanchnic and vagus nerves. According to Pflüger, whilst stimulation of the splanchnic nerves has the effect of checking the peristaltic movements that of the vagus tends to excite them.*

In reference to this question of the dependence of gastric crises (of the two kinds which I have mentioned) upon sclerosis in the neighbourhood of the pneumo-gastric nucleus, I would call attention to a point which, although of a negative character, seems to me to possess considerable importance.

The man X. Y— did not suffer at all from gastric crises. It will be observed that Dr Bevan Lewis in *his* case found the nuclei of the vagus, amongst other nerves, well displayed and perfectly intact, and he adds, "no diseased track of tissue was anywhere apparent in the transverse sectional areas of this district." The importance of this remark will be especially evident when it is contrasted with Professor Pierret's observation of sclerosis of the funiculus gracilis in a case of tabes with gastric crises.†

In another respect there was an important difference between these two cases. The muscular atrophy which affects in a marked manner the trunk and extremities of the patient C. D—, was entirely absent in the case of the man X. Y—. The microscopical findings corresponded with the clinical history. (I ought here to say, perhaps, that I kept Dr Bevan Lewis in ignorance of the symptoms noted during life, in order that his observations might be unprejudiced.) Dr Lewis reports that the anterior cornua of the spinal cord were entirely free from any morbid change.

* 'A Text-book of Physiology.' By M. Foster. 3rd edition, p. 267.

† see p. 195.

It is interesting to note that Mr Hutchinson found "in six of his cases the lower extremities more or less weak, and a condition approaching more or less closely to locomotor ataxy," and he makes the important remark, that "there can be no doubt that ophthalmoplegia externa is sometimes a part of the general malady known as progressive locomotor ataxy, especially when that disease is due to syphilis." The two instances which I have described are examples of the first described association. Both patients presented the most characteristic symptoms of tabes dorsalis. In each case there were "lightning" or "electric" pains, absence of knee-phenomenon, and ataxy. No other signs need mention in the presence of these.

As regards the question of syphilis, however, one is not able to speak so positively. In the female patient the history of syphilis was clear and certain. On the other hand, there is no evidence that X. Y— had ever suffered from the infection. The condition of his aorta is a suspicious circumstance no doubt, but the history of rheumatic fever prevents us from laying stress upon the condition as indicative of constitutional syphilis.

In any case the proximate cause of the lesions of nuclei must be sought for in an extensively diffused disease of the vascular system, of which there is abundant evidence in the dilatation of the aorta and the rigidity and tortuosity of the brachial arteries, no less than in the changes which are found in the intrabulbar arterioles, resulting in miliary hæmorrhages.

LECTURE X

TABES DORSALIS AND SYPHILIS

TABES DORSALIS is the most frequent of all the chronic affections of the cerebro-spinal axis, and the prognosis in this disease is practically hopeless. There is increasing evidence, no doubt, to show that cases may go on for a very long time without the symptoms becoming materially intensified, and even with encouraging periods of improvement. But as matters stand we are unable to count upon more than this in any case of a confirmed character. At first sight, therefore, it would seem to be a point of more than ordinary importance to discover whether the disease is so far connected with a syphilitic origin as to encourage us to hope for success by treating the patient energetically with specific remedies.

In many morbid conditions such a discovery, by leading to appropriate treatment, produces the most triumphant results. But, as a matter of fact, in the case of tabes dorsalis expectations that may have been formed of a similar success have not been realised. Now and then, it is true, we meet with cases which improve remarkably for a time whilst iodide of potassium is being administered, but in my experience I have never known a cure to result from specific measures. From mercurial treatment, indeed, which I have had very carefully applied by inunction in a number of cases where the history of syphilis was distinct, I have seen no good whatever, but, on the contrary, as it seemed to me, a tendency to harm.

In 1871, writing upon the subject of syphilitic affec-

tions of the nervous system,* I included progressive locomotor ataxy amongst the nervous affections belonging to the tertiary stage of syphilis. At that time the frequency with which a syphilitic history was to be noted in these cases had long impressed me, and I was in the habit of treating my ataxic patients with iodide. But the remarkable absence of successful results appeared to throw so much doubt upon the matter that I carefully excluded the disease from consideration in my work on syphilitic nervous affections.† The connection between nervous disorders and syphilis was then not generally recognised, and I was anxious to avoid weakening the force of that which was to be said on a very important subject by the introduction of debatable material.

The question of the relation of tabes to syphilis, although it had not escaped the attention of Lancereaux, Duchenne, and other writers, had not been very prominently brought forward before 1875, when M. Fournier, of Paris, expressed a strong opinion on the subject. Out of thirty ataxics he found a history of syphilis in twenty-four. He believed that the tabes in these cases was an outcome of syphilis, although according to his view it does not in these circumstances present any special symptomatology or lesions.

Now, it had been urged by Charcot in 1873 that certain lesions of the spinal cord, primarily developed outside the posterior columns, might at a given moment invade these at a variable height, and produce accidentally, as it were, some symptoms of locomotor ataxy, but this, he went on to say, was not truly progressive locomotor ataxy.

More recently Dr Gowers, in an able paper read before the British Medical Association in 1878,‡ pointed out that it was common for an acute change, as *e.g.* primary myelitis, which had extended widely through the section

* 'Lancet,' March 11th, 1871.

† 'Clinical Aspects of Syphilitic Nervous Affections,' 1873-74.

‡ "Syphilitic Neuroses," 'Brit. Med. Journ.,' March 1st, 1879.

of the cord, to clear off from all except the posterior columns, and to persist in these. In such cases a weakness of the legs of sudden onset would give place to incoordination of movement, and this would increase so as to present the typical features of locomotor ataxy. Eliminating such cases as these, of secondary origin, he thought that nearly one half of ataxics had a history of antecedent syphilis.

I do not know that I am disposed to draw a very hard-and-fast line between tabes supposed to be of primary origin and that which is secondary to acute changes. It is certain that in inquiring into the history of many cases which, when they come before us, present all the characteristics of typical tabes dorsalis, we frequently hear a description of symptoms, often long past, which can only be referred to the occurrence of spinal meningitis, usually of a slight and strictly localised character. The fact that some slight thickening of the soft membranes and adhesion to the posterior surface of the cord is commonly met with in post-mortem examination of tabetic cases is well recognised. Vulpian, who was at first disposed to think that this meningitis might be primary, has rejected the idea, because the change is not proportionate to the amount of lesion in the posterior columns, and he now attributes it to a propagation into the membrane of the irritative action going on in the neuroglia. But it is not disproved that initiatory inflammation of the pia mater might spread itself by continuity to the neuroglia. There may possibly be anatomical reasons connected with the disposition of blood-vessels which would explain the confinement of the inflammation which had thus been started to a certain locality of the cord. I am inclined to think, also, from certain observations, that a limited spinal meningitis may clear off without leaving traces perceptible to the naked eye in the portion of membrane which has been affected.

In the early part of his history Fredk. J—* had very

* See Lecture VIII.

severe pain in the mid-dorsal region. He could not bend his back without severe pain—a symptom strongly suggestive of meningitis. There was a clear account of a chancre six years before the commencement of his symptoms.

In 1870 I saw a gentleman, æt. 58, who was typically ataxic. He had what he described as "a tremendous sensation of a cord around his waist." There had been constant pain in the back and right side of the spine about the level of the lower dorsal vertebræ for two years, and he frequently suffered from what he called "spasms" in the right hypochondrium. There was a distinct history of syphilis.

I examined, in 1880, a gentleman, æt. 33, who had suffered for fifteen months from "a grinding, aching feeling from the left side of the spine to the pit of the stomach." He could walk and had not lost much power in his legs. It was for this strictly localised pain that he sought advice, but examination showed that he was suffering from tabes, and a year later he came to me with paralysis of the left oculo-motor nerve. There was history of syphilis of eleven years' standing.

Such examples could be readily multiplied to almost any extent. They can hardly be explained, I think, except by the existence of a transverse and limited inflammatory lesion about the spot indicated, involving either the membranes and posterior roots or the surface of the cord, or both.

The feeling of "waist constriction" which we frequently meet with in tabes is evidently not a necessary part of those symptoms which are referable to that which is the essential lesion in the disease, viz. systematic sclerosis of the postero-external columns. If it were, we should find it in all cases. It is a symptom which is probably dependent upon a transversely localised lesion (meningitic or myelitic), and hence a common accompaniment of paraplegia. The following case appears to me to have an important bearing on the question:

In February, 1875, I saw in consultation H—, a man,

æt. 45, who was suffering from paralysis of the left oculo-motor nerve, involving the eyelid as well as the recti muscles. Five years previously he had a chancre, which was followed by sore throat. I found somewhat large almond-shaped glands in his groin. Under iodide and mercury the paralysis of the third nerve rapidly cleared up. But in July of the same year his medical attendant asked me to see him again, and I found him in bed almost completely paraplegic. There was very little power indeed of flexing the thighs upon the hips when I pressed lightly upon his knees as he lay in bed. When the spinal column was pressed upon deeply between the fourth and seventh dorsal vertebræ an obscurely painful numbed feeling was complained of. Below this the trunk and extremities showed incomplete cutaneous anæsthesia. Movement of his spine, as, for instance, when he turned in bed, caused pain at the part of the dorsal spine described, and radiating from it. He complained of startings of his legs, his bladder and rectum were weakened, and there was occasional incontinence of urine. Sexual power was in complete abeyance. Under mercurial treatment he gradually recovered the power of his legs to a great extent, and went away to the seaside.

In November, 1877, I saw him again. His *right* oculo-motor nerve had then been paralysed for four days past, and he had very severe neuralgic pain in the forehead for a month. His grasp was equal, and there was fair power in his legs, so that he was able to walk about, but his gait was somewhat tottering. There was some tenderness on pressing upon the seventh dorsal vertebra. The sphincters were weak. Under iodide he lost the paralysis of the right third nerve in a month.

The last time I saw this patient was in October, 1878. He told me that six weeks previously he had been severely attacked with darting pains in his legs, momentary, as if a blunt object had been pushed into him. For some time before this, but how long I could not ascertain, he had suffered occasionally from pains of the character described,

but not so severely. His gait was now distinctly ataxic. There was anæsthesia from the knees downwards. He could not stand with his eyes shut, and in bed did not know where his legs were. There was now also diplopia from weakness of the left external rectus muscle. The pupils were contracted. The ophthalmoscope showed greyness of the left optic disc. The patellar tendon-reflex was absent in both knees.

Such a case as this in its developed form is not distinguishable by any peculiarities from an ordinary example of tabes dorsalis. Yet it had manifestly travelled to this condition through a phase of meningo-myelitis. It is interesting to contrast this patient's almost total inability to use his lower extremities in July, 1875, with the fair power and capacity for walking about which he showed in 1878. There would seem to be little doubt that the lesion (meningitis?) which had caused the earlier symptoms had cleared off, leaving behind a state of spinal cord which gives distinct evidence of the existence of sclerosis in the postero-external columns. Does it not suggest itself as probable that the inflammatory action may have travelled into the connective tissue of the cord through the channel of the inflamed pia mater? I cannot help thinking that in some cases the initial lesion may prove to be a very limited inflammation of the soft membranes of the cord which "lights up," as it were, inflammation in the adjacent connective tissue and leads to sclerosis. It must be allowed, however, that in many cases the most careful questioning elicits no evidence of there having been even a very little spinal meningitis. In such instances the suggested explanation is inadmissible.

It has happened that we have had in the hospital recently a case which bears upon this subject, and may be shortly related.

Mary Anne A—, æt. 44, was admitted into the hospital suffering from paraplegia. She could just move the right foot, but with this exception the limbs

were powerless. There was also complete anæsthesia to touch and pin-pricks throughout the lower limbs, together with great wasting and entire loss of muscular sense in them. She had incontinence of urine and fæces. On the trunk the anæsthesia extended up to the second rib.

Her arms were thin. She was just able to feed herself with a spoon, but not to dress herself. There was great want of coordination in the movements of both arms and hands when she offered to take hold of anything. But beyond this there were fine and somewhat constant flexion and extension movements of the intrinsic muscles of the left hand. The condition was practically one of complete paralysis of the lower extremities with ataxy of the upper limbs. She had acute bedsores which resisted all efforts to restrain their course. Death took place six months after admission.

Now, this patient was married and had given birth to nine children, of whom only three survived, five having died in convulsions and one being stillborn. One child had a rash over its body. The patient had suffered from ulcerated sore throat, and her hair had fallen out. There is no doubt that she suffered from syphilis. The mode in which her illness commenced is important. A year before she came to the hospital she began to have constant aching in her legs like rheumatism, with "dreadful pricking and shooting in them," severe pain between her shoulders, and daily vomiting. After a few months, the pains in the legs continuing to be very bad, she completely and somewhat suddenly lost power, first in one and a week after in the other leg. Numbness now rapidly spread from the right to the left leg, and then to the trunk. At the same time the action of the sphincters could not be restrained, and she lost the power of coordinating the upper extremities.

The autopsy in this case showed no change in the spinal membranes perceptible to the naked eye, but there was distinct grey degeneration of the posterior columns, which examination of sections of hardened cord was found to con-

firm. (Imperfections in the preparation of the sections prevented so minute an examination as would have been desirable. However, Dr Bevan Lewis, who kindly examined the slides at my request, is able to report the following appearances.)

In all regions alike the columns of Türck and the antero-lateral columns were healthy; the posterior columns throughout showed extensive degeneration. In the cervical enlargement the columns of Goll were much degenerated, and the same lesion appeared notably behind the posterior commissure and along both the radicular zones. In the post-commissural zone the medullated fibres were greatly wasted. Along the radicular zones a few very large fibres were seen. The root fibres of the posterior spinal nerves were evidently affected; they could be traced a short distance only, and were encroached upon by sclerosed tracts. The medullated fibres along the posterior margin of the cord were unusually large, the vessels coarse, and in many cases the perivascular channels widely distended and filled with coagulated (albuminous?) contents. In the upper cervical region the posterior cornua were sclerosed, the nuclei numerous, the cells rounded and devoid of processes on one side of the cord. The cells on this side were also much smaller and more attenuated than those of the opposite side. The columns of Goll showed several hypertrophied axis-cylinders. The outer group of cells in the anterior cornu of one side presented decided wasting and attenuation of its cells. In the cervico-dorsal region the anterior cornua showed disease of the outer group of cells; these cells were rounded and devoid of processes. In the lumbar cord there was much atrophy of cells of the anterior cornua, especially on one side, and the anterior nerve-roots appeared diseased.

The hypothesis which is admitted by Vulpian* as the most probable is that in the sclerosis of tabes it is the nervous element proper contained in the nerve-tubes which is first affected by an irritative lesion, and that

* 'Maladies du Système Nerveux,' Paris, 1878, p. 444.

the connective tissue suffers secondarily. The difficulty of accepting this view is to my mind very great. One cannot conceive an atrophy of axis-cylinders without some antecedent changes in the carriers of trophic material for the axis-cylinders, *i.e.* in the blood-vessels and the tissue in which they lie. On the other hand, if a vascular change be the initiatory step, it does not seem difficult to understand that syphilis, which is prone to occasion meningitis, may sometimes lead to sclerosis through inflammation of the soft membranes of the spinal cord.

A few words as to the frequency of association of a syphilitic history in cases of tabes. Erb, in a recent publication,* says that out of forty-four cases he had met with a history of syphilis in twenty-seven. Out of fifty-three cases of my own a probable history of syphilis is noted in twenty-five, but I have reason to think that this figure may not accurately represent the actual proportion, my notes of several of the cases being imperfect. Now, if we add together the statistics of Fournier, Erb, and myself, we shall find that in seventy-six out of 127, or in 59·8 per cent., there was a history of syphilis.†

It is clear that coincidence is not sufficient of itself to

* 'Deutsch. Archiv f. klin. Medicin.,' xxiv Bd.

† Since the substance of this lecture was delivered (December 18th, 1879) a very large amount of attention has been bestowed by numerous writers, both in this country and abroad, upon the statistics of this question. For a very good *resumé* of the varied experiences and opinions upon the subject, the reader is referred to an article by Dr J. L. Prevost, in the 'Revue Médicale de la Suisse Romande,' January 15th, 1882. As regards my own figures up to the present date, I find that out of one hundred cases of tabes there is some history of venereal disorder in forty-five. In many, I need hardly say, the history of constitutional syphilis is perfectly clear. In several, on the other hand, it is so imperfect that were it not for one's experience of the extremely weak history, which is all that can often be obtained in other diseases of the nervous system undoubtedly due to syphilis, I should be disposed to reject them. In some, again, the investigation was either precluded by circumstances, or accidentally omitted. In others the information obtained could not be depended upon. Reserve must be shown, therefore, in accepting my statistics as representing anything more than an approach to probability. I cannot help envying the certainty which has apparently attended the experience of many of those who have published upon this subject.

establish anything like a necessary relation. It might turn out, for example, that a relation of the following kind existed:—Tabes dorsalis was formerly supposed to be due to sexual excesses; the individual addicted to sexual excess would be, *cæteris paribus*, more exposed than another to the chance of syphilitic infection, and the misfortune attributed to the latter might really be due to the former. I do not support this view; on the contrary, I do not think there is any ground for believing that sexual excess is, at all events, a frequent antecedent of tabes, but I mention it as an illustration of one mode in which there might be a coincidence in the occurrence of syphilis and tabes without any real association of cause and effect. There may be others.

It is necessary also to bear in mind another very possible source of fallacy. There is often great difficulty in ascertaining the date of the earliest symptoms of tabes. There may have been some slight flying pains which have left little or no mark in the recollection of the patient, and in nine cases out of ten have been set down by him to rheumatism. Yet these pains, usually the earliest evidences of tabes, may have occurred *before* he became infected with syphilis. In the case of a gentleman whom I have at present under occasional observation there were neuralgic pains in the *head* some years before he acquired syphilis. Who knows whether these did not depend upon sclerosis of the deep root of the fifth nerve, as is probably the case in the patients whom I showed at a recent lecture? On this account I do not think my statistics are to be depended on as showing with any certainty the proportion of cases in which syphilis was acquired *before* the earliest symptoms of ataxy, and it is evident that the same objection may possibly apply to the figures collected by others. The discovery made by Westphal that absence of patellar tendon-reflex is a very early symptom of tabes will help us greatly in the future in regard to this subject, but it necessarily throws no light upon the date of origin of those cases the notes of which were

taken before his all-important observation became common knowledge.

The position of tabes in regard to syphilis is peculiar in another respect. Affections of the nervous system which owe their origin to syphilis are not, as such, distinguishable in any very evident manner from diseases unconnected with such infection. The hemiplegia which results from thrombosis of a cerebral artery affected with syphilitic disease follows the same course as hemiplegia consequent on thrombosis of a cerebral artery thickened by atheroma. Syphilitic new formations in connective tissue bring about symptoms depending on lesion of nervous structure, such as might be referable to growths of any other kind. But there are two points especially which frequently afford strong presumptive evidence as to the syphilitic nature of a certain affection. In the case of lesions referable to thrombosis of a cerebral artery there is the age of the patient. Should this be much below that at which degenerative changes may be reasonably looked for in the arterial system, there being at the same time no evidence of changes in the heart or kidneys, I think we may almost certainly refer the arterial thickening to syphilis. So also in regard to growths. As a matter of experience it is certain that almost all cases of marked paralysis of single cranial nerves (I exclude here the incomplete and transitory paralysis seen in tabes) are due to syphilis. When along with this are conjoined symptoms which can only be referred to the existence of another distinct lesion (one causing, *e.g.* hemiplegia, monoplegia, or paraplegia) the evidence is greatly strengthened. It becomes, indeed, so strong, and the chance of exception due to the presence of tubercle or cancer is practically so small, that, in the absence of the strongest corroborative evidence of the latter diseases, we should be culpable in treating the case otherwise than as one of syphilis.

But in tabes the circumstances are very different. Take, for example, the question of age. If we compare the ages of tabetic patients in whom inquiry elicits a

history of syphilis with those in which this element is wanting, we are not struck by any marked contrast. When we meet with the case of a young man of twenty or thirty years of age who has hemiplegia apparently resulting from cerebral thrombosis, and in whom there is no history of rheumatic fever, heart or kidney disease, we know at once that his disease is almost certainly the result of syphilis. We are struck by the fact that he is suffering from an affection brought about by disease of his cerebral arteries. The more ordinary cause of such disease is the atheromatous thickening which comes in association with other degenerative changes brought about by agedness. Syphilis induces changes in the arterial coats, which lead to a precisely similar result. The indications are simple enough. But in tabes we are left without aid from this question of age. The disease is one commonly of adult life, which most often commences in the period between maturity and middle age, and whether there is a history of syphilis or not, the large majority of patients are about this time of life.

There remains the question of sex, consideration of which may help us a little. My personal experience is that only 10 out of 100 patients suffering from tabes belong to the female sex. Some years ago, in view of a coming discussion on syphilis at the Pathological Society, I tabulated 100 cases of disease of the nervous system dependent, so far as could be determined, upon syphilis. From this list all cases of tabes were excluded. Sixteen out of the 100 patients were females, but I have since had reason to think that the proportion of females affected with disease of the nervous system consequent upon syphilis is far greater than this. Again, the occurrence of tabes in females of good social standing is surely of extraordinary rarity.* Are females of this class equally exempt from

* Out of 100 cases of tabes of which I have notes only three occurred in ladies. It has a significant bearing on the question under discussion that two of these are single women, in whose cases the idea of a specific cause may be confidently dismissed.

syphilitic affections of the nervous system of a kind which is generally recognised? I am sure that they are not, and that in this respect there is a very striking contrast.

If tabes be very frequently of syphilitic origin, how is it that females, who bear their fair share of other diseases of the nervous system of specific origin, furnish only ten per cent. to the ranks of the former disease?

Whilst it appears to me incontestable that there is a remarkable frequency of association between syphilis and tabes dorsalis, I do not think, all things being considered, that the time has yet arrived for us to draw safe inferences as to the precise nature of the relation.

LECTURE XI*

OSSEOUS AND ARTICULAR LESIONS IN TABES DORSALIS: CHARCOT'S JOINT DISEASE

Let me draw your attention to the man who now enters the room walking with the help of a stick. There is a marked peculiarity in his gait, and when we look at his legs for an explanation of it we observe at once that the greater part of the right lower extremity is enormously enlarged. On examining him more closely, we see that this enlargement fails to account for his peculiar walk, for the left leg, which is not notably altered in size, presents an abnormality in movement which resembles that of the right. If we analyse his march we find the following peculiarities equally well marked in the two legs:—In the first stage of progression the foot, which normally should be carried forwards nearly parallel to and at a distance of an inch or so from the ground, is raised some inches higher than this, and in a strongly dorsal flexed position. When it has reached the measure of the step, the heel is put down noisily, with a sort of jerk, the toes being then gradually brought down from their upward flexed posture, and applied to the ground preparatory to a repetition of the step. All this time the patient's feet are turned out like those of the dancing masters of our youth, and his eyes are fixed upon the ground in front of him. When he wishes to turn, he stops short for a second or two, steadies himself, and bring himself round with great

* The substance of this lecture appeared in the 'Lancet' of August 22nd, 1874.

caution. He is not able to move in a small circle. He walks best on an even surface, and goes downstairs easier than up, taking care to plant the entire sole of the foot upon the step. If his feet be placed close together when his eyes are shut, he sways to and fro, and would fall but for help, and he cannot take more than two steps without the aid of sight. His toilette is considerably embarrassed by this difficulty, for he tells us that when he is soaping his face, and consequently shutting his eyes, he is obliged to lean against the wall, or he would fall. With all this, if we try to bend his legs at the knee against his will, or to resist their voluntary extension, we find but little failure of muscular power in either of his lower extremities.

The condition presented by S—, then, it is scarcely necessary to say, is one of ataxy, and not of paraplegia. Moreover, the difficulty of coordination is not confined to the lower extremities. The patient is a carpenter, and he finds it impossible to use a hammer, for in striking at a nail he constantly misses his aim, and goes to one side or the other of the object; and he cannot saw a piece of wood in a straight line.

So much for the disorders of motility. As regards those of sensibility, they are of two kinds—diminution of various kinds of sensibility, and pain. He complains of a feeling of numbness in both feet, extending some inches above the ankles, and also in his hands, especially in the left. The touch of a finger is not felt at all on either sole; in the same situation, however, the contact of ice is immediately recognised, and its coldness appreciated; heat, on the other hand, is more slowly, but still correctly recognised. Electro-cutaneous sensibility is much diminished in the hands, still more so in the legs, and is quite absent in the soles of the feet. The muscular sense is manifestly impaired, for not only can he not tell when in bed in which direction his legs are lying, but he cannot feel the contraction of the muscles of his thighs when they respond to an induced current. This response is normal

in the right thigh, somewhat deficient in the left, and very imperfect (probably for a reason which I will mention presently) in both legs below the knees.

The other disorder of sensibility consists in the liability to "flying pains," and these he has had since 1869, the longest interval of exemption having lasted about three months. His last attack of pains visited him on Sunday, Monday, and part of Tuesday. A pain would last perhaps five seconds, and resemble some sharp instrument suddenly pushed into the lower part of his shin-bone. It would recur every five minutes or so during the daytime, and almost entirely deprive him of sleep during the nights. And this has been the general character of the pains, which, however, were worse formerly than they have been of late. Since the commencement of his illness he has always had a feeling as of a tight band around his waist, and of distension in the stomach.

Although the symptoms described are those common to progressive locomotor ataxy, they do not of themselves suffice to mark the case positively as one of that class, as pictured by Duchenne. To complete the catalogue, there should be some evidence of functional disorder in the district of some cranial nerve. As is well known, diplopia from paresis of one or other of the nerves supplied to the external muscles of the eyeball, and amblyopia from progressive atrophy of the optic disc, are the most common forms which these disorders assume. Now, this man has a well-marked squint; and if we had not inquired particularly about this feature of his case we might easily have jumped to the conclusion that the symptom, as it appears in him, is just that which is wanting to complete the *ensemble* of the requisite conditions. But it seems on inquiry that his strabismus dates from early childhood, and there is little doubt that it is one of those ordinary squints arising from hypermetropia which are so often ascribed (as he, indeed, ascribes it) to a kind of retribution for imitating a school-fellow with the like affliction. We must, therefore, exclude the strabismus

from our calculation. Examination with the ophthalmoscope discloses no change in the optic discs; and the man himself complains of no material weakening of sight. We do find, however, one cranial nerve which shows symptoms of disorder. For the last few months our patient has been growing deaf in the left ear, and now he cannot on that side hear the ticking of a watch, however closely it is applied. Duchenne, Remak, and Topinard have each recorded instances in which the auditory nerve was affected; the latter mentioning seven cases in which he has noted this condition. The feature is a slight one, but in this case so characteristic are the disturbances of motility and sensibility that it suffices to complete the picture. I ought to add, too, that the pupils are minutely contracted. For the rest, we note that at various times he has had great delay in emptying his bladder, and occasional incontinence of urine; and that for a long time past he has been impotent. In the earlier part of his illness he suffered from gastric disturbance of a peculiar kind. He would require to relieve his bowels five or six times in the twenty-four hours, the motions being small in quantity and solid, and he would besides go more than once daily to stool without result. This irritability of the bowels, although not usually included amongst the symptoms of progressive locomotor ataxy, is one which I have occasionally observed in other cases, but never, I think, at so early a stage in the history as in this man.

It seems that our patient first began to stagger in his gait about June, 1868. The "flying pains" commenced early in 1869, and in March of that year he began his attendance at this hospital. Under treatment with arsenic he rapidly improved so much that he was able to return to his occupation, which he had been obliged to quit, and he continued to work more or less for his living until December last. Since that time he has not been able to follow his employment, but has attended here regularly.

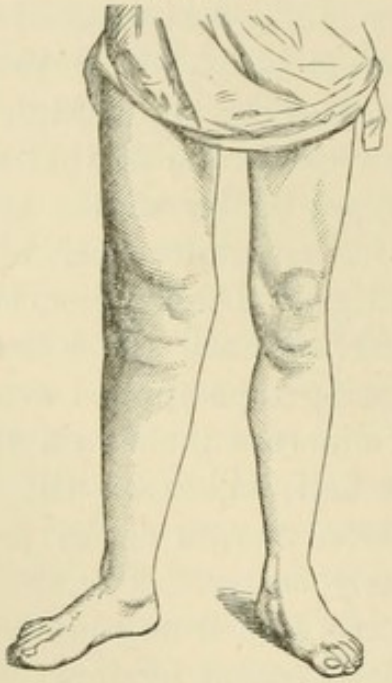
It was in June, 1873, that he called our attention to his

right leg, which he said was enlarged. On examining it, we found the leg swollen and œdematous below the knee. Owing to the fact that he was attending as an out-patient and always with his clothes on, the circumstance after this escaped our attention until the following December, when he again complained very much of this limb. On stripping him, the knee-joint and the thigh for some distance above it were found to be enormously enlarged, evidently with fluid; and this condition has persisted ever since, with but very slight variations of size.

At the present time (July, 1874) the following is the condition of the limb:—The right thigh begins to swell a short distance below the groin, so that at a point eleven inches above the upper border of the patella it measures seventeen inches in circumference, as against sixteen inches in the left thigh. Descending, the enlargement is more and more marked until the knee-joint is reached, and here the measuring tape applied over the patella gives a circumference of nineteen inches on the right side and only thirteen and a half inches on the left. About two inches below the lower margin of the patella the enlargement almost suddenly ceases. The swelling is hard and elastic, the skin being free from redness, so smooth as to look polished, and traversed by large veins. Extension of the leg is perfectly performed, but flexion is somewhat limited. Since this photograph was taken in March last, although the size of the limb has continued the same, you will notice that a curious alteration in its appearance has taken place. The leg now forms an angle of about 45° with the thigh, the apex being inwards—a deformity which is due to subluxation of the joint from the strained and weakened internal lateral ligament gradually refusing its office. That the swelling is not simply œdematous is evident for two reasons:—1st. There is no pitting on pressure. 2nd. When the rheophores carrying an induced current of electricity are placed upon the quadriceps extensor muscle just above the patella, where the enlargement is very great, there is immediate and powerful

muscular contraction. The electric excitability at this spot is, indeed, much more marked than in the corresponding part of the left thigh. The fluid therefore lies under and not superficial to the muscle, as would be the case in œdema. Lower down the leg is certainly somewhat œdematous; and there, as you see, the conduction of the current is interrupted by the presence of fluid in

FIG. 17.



the subcutaneous connective tissue, and the muscles consequently fail to react to the electric stimulus. The apparently increased excitability of the quadriceps extensor in the affected limb is perhaps owing to the thinning and tension of the strained skin favouring conduction to the muscular tissue immediately beneath it. When the leg is rapidly extended, the hand laid upon the knee-joint is conscious of a peculiar scranching thrill. Now it is important to remark that all this accumulation of fluid has taken place without any of the symptoms of ordinary joint inflammation. During the process of enlargement the patient had no pain or heat in the joint; he was conscious only of a gradual increase in its size. At the

present time, if he kneels or walks much, he gets a little aching pain extending down the leg; but so long as he does not bend the knee much he has no pain whatever, and the only inconvenience which he suffers is from the weight of the swollen limb causing him to be readily fatigued. There is no swelling of the ankles or feet. As I pointed out before, the action of the right leg in walking is precisely similar to that of the left, but the movement of the joint is limited in the direction of flexion.

Such is the very anomalous joint affection which this patient presents. It is, you will remark, a condition which asserts itself so very prominently that were this man to present himself for the first time for medical advice it is more than probable that the whole attention of the examiner would be fixed upon the state of the right limb, and the much more serious general disorder with which the patient is affected would be passed over unnoticed.

It so often happens in clinical medicine that we fail to see what we do not specially look for that I am constrained to believe it possible that such a condition as this may have existed (though not, perhaps, to so marked an extent) in others of the numerous instances of progressive locomotor ataxy which have come under my observation; but it is a fact that I have never before noticed this affection, and my colleague, Dr Hughlings Jackson, to whom I showed this patient, tells me that it is likewise the first case of the kind which he has seen. This experience, or lack of experience, contrasts remarkably with that of Dr Charcot, who describes no less than five examples of the kind out of fifty cases of tabes in the Salpêtrière.

It is to Dr Charcot* that we owe, in the first instance, the recognition of this peculiar arthropathy. He looks upon the affection as one of the multiple forms of spinal

* 'Leçons sur les Maladies du Système Nerveux.' Paris, 1873. 2me Série.

arthropathy, by which term he would designate a group of disorders which appear to be directly dependent upon certain lesions of the spinal cord. In his experience the arthropathy in question is always an early phenomenon, occurring between the prodromous period and that of incoordination. If it should happen to be late in appearing, it is then always, he says, in connection with one of the upper extremities, and he attributes this to the circumstance of the sclerosis, which is the cause of progressive locomotor ataxy, frequently invading the upper part of the cord only after the lower part has been for a long time affected. The present case is an exception to this rule, for the symptom in question did not appear until marked incoordination of the lower extremities had existed for many years; and it is in the knee-joint—not a joint of the upper extremities—that it has shown itself. Charcot describes the condition as an extreme tumefaction of the entire limb, composed of (1) a considerable hydrarthrosis; (2) a diffused swelling, for the most part of hard consistence, in which the ordinary symptoms of œdema are not usually apparent. He remarks that this arthropathy is unaccompanied generally by fever or pain. This description precisely applies to the case before us. It will be noted besides that, although the knee-joint is enormously distended with fluid, it does not present the appearance of ordinary chronic synovitis, in which the distended capsule of joint projects with marked distinctness in three places, viz. above the patella and on each side of the ligamentum patellæ. There is no such mapping out of the knee-joint in this case; and this is evidently because, in addition to the fluid in the articular cavity itself, there is effusion in its neighbourhood. This effusion, as it does not occasion the appearances of subcutaneous œdema to present themselves, is probably beneath the muscles. The affection, then, is a peculiar one, and its pathology is by no means clear. Experience of these cases shows that, with a striking similarity in their onset, their progress varies. In some, at the end

of a few weeks or months, the swelling disappears, and the joint apparently returns to its former condition. In others, on the contrary, grave disorders remain—erosions of the osseous surfaces, creaking movements, various luxations, or even total destruction of the joint. As regards the order of frequency, it is first the knee, then the shoulder, the elbow, hip, and wrist which are apt to be affected. Several joints may be coincidently involved.

The affection, which in certain respects is suggestive of dry chronic arthritis (arthritis deformans: rheumatoid arthritis), differs from that disease, as Charcot points out, in several particulars: in the large quantity of fluid by which it is characterised, and the fact of the effusion extending beyond the limits of the joint; in the luxation which is common in the ataxic class; in the fact that the knee is most commonly affected, not the hip, and that the joint affection of the tabetic patient may retrograde or even recover, which is never the case in dry chronic arthritis; in the suddenness of the appearance, and rapidity of progress of the disease. On the other hand, he observes, it is true that in cases of old standing, when the articular surfaces, deprived of cartilage, have continued to rub against each other, some of the characters of dry arthritis may be found; eburnation and deformity of the articular surfaces, with osseous growths on the extremities of the bones.

The possibility of occurrence of slight articular affections dependent apparently upon preceding lesion of the nervous system is generally recognised. Such affections have been observed in connection with lesions of the peripheral nerves, as well as in limbs which are the subject of hemiplegia from hæmorrhage or softening of the brain, in Pott's disease, in acute myelitis, in certain cases of tumours occupying the grey substance of the cord. The articular affections, however, which may occur in these conditions are of a different kind from those met with in the course of tabes dorsalis. The fact that the joint affection peculiar to progressive locomotor ataxy is very

exceptional suffices to show that it is not due to the sclerosis of the posterior columns which constitutes the important pathological change in that disease, but as to the seat of the lesion to which its causation must be referred we are at present without evidence.

LECTURE XII*

OSSEOUS AND ARTICULAR LESIONS IN TABES DORSALIS (*continued*)

THERE is a patient in David Wire Ward whose case illustrates very well one of the comparatively rare features of tabes dorsalis (progressive locomotor ataxy). The study of his condition is important, not only on account of its pathological interest, but also from a diagnostic point of view, because the tendency of certain symptoms which he presents is to mask others which really form the key to the nature of his disorder. Indeed, this has actually happened in his case.

W— is a man aged thirty, who walks down the ward with that kind of distinctly halting gait which shows at a glance, without room for doubt, that one of his legs is considerably shorter than the other. If we let him lie upon his back, and measure the distance from the anterior superior spine of the ilium to the tip of the internal malleolus, the tape shows a length of 33½ inches on the right side, whilst it indicates 35½ inches on the left. When the right foot is grasped, a moderate amount of traction easily reduces this disparity to one inch. On the other hand, when he stands with his bare feet on the floor the difference in length is increased to three inches. As he lies there is no marked inversion or eversion of the right foot, which tends, when it is absolutely left to itself, to roll slightly outwards; but he can easily control this movement, and even invert his foot with the greatest ease. He can

* The substance of this lecture appeared in the 'Lancet,' January 18th, 1879. The patient was exhibited at the Harveian Society in 1879.

extend his right lower extremity perfectly well, but flexion of it upon the trunk is limited; other movements are easily performed. The upper and inner surface of the right thigh—Scarpa's triangle—is distinctly rounded and prominent as compared with the corresponding part in the left limb, which presents the normal hollow. Turning to the hip, the patient being still recumbent, we find that the great trochanter is as nearly as possible on a level horizontally with the ilium, being evidently carried upwards by muscular action. The neighbourhood of the hip is bulged and deformed by what examination with the hand ascertains to be a large collection of fluid in or about the hip-joint. If we lay hold of the man's thigh near the knee we can easily, with the advantage of the leverage thus obtained, move the bone into all sorts of positions, forcing it upwards or downwards and rotating it in or out. These movements, indeed, are very much more easily carried out, and to a greater extent, than in health. But do what we will, the other hand placed upon the swelling can discover no trace of any neck or head to the femur. The bone evidently terminates above with the great trochanter. In some of the movements, especially in rotation outwards, we are conscious of crepitus, which is sometimes much more marked than at others. The patient says that he often himself *feels* this crepitus in the movements of walking. Turning back now to the front of the thigh, and examining the structures below Poupart's ligament, we come upon something which is very singular. We feel a sort of narrow bony splint, not wider than one's little finger, which stretches obliquely downwards and somewhat inwards for about nine inches. Above, it is pretty firmly fastened (not, however, apparently by bone) to the anterior inferior spine of the ilium, but lower down it can be easily grasped, lifted from subjacent textures, and slid loosely to and fro. When the patient extends his leg this process of bone is held tightly, so that it is evidently connected in some way with the quadriceps femoris muscle. It appears, in fact, to occupy

the position of the superior tendon of the rectus. Besides all this, we note that both lower extremities are somewhat wasted, the right especially.

Here, then, is a lame man with such very palpable symptoms of a painless hip-joint disorganisation that there is a temptation, on superficial examination, simply to call the case one of chronic rheumatic arthritis, with a bony growth in the neighbourhood of the joint. And if we ask the man about his past history, he tells us (apparently that no pitfall for an erroneous diagnosis may be wanting) that he has been troubled for years with "rheumatics." But let us find out what he means by this term, which is a fruitful source of error in diagnosis. The pains occurring in syphilis, scurvy, chronic alcoholism, lead-poisoning, neuralgia, myalgia, and some other conditions, are continually styled "rheumatics." Our patient, it seems, gives the name to certain extremely sharp, sudden pains of momentary duration, which have tortured him for the last six years, with uncertain intervals, in the hips, back of the thighs, shins, as well as in the districts of both ulnar nerves. He describes, indeed, with the greatest precision, the characteristic pains of tabes. If we let him walk again we see that, besides the lame man's gait, he staggers now and then, especially when he arrives at the end of the ward and has to make a turn. When asked to stand still he poses himself with evident effort, and the fronts of his feet are continually tilting upwards, as though his soles were so disinclined to keep the ground that without a certain amount of trouble on his part he would be likely to walk only on his heels. In walking, though he gets along at a very good pace, it is only by keeping his eyes steadily fixed on the ground before him. And we find that he cannot stand for more than two or three seconds with his eyes shut, but then staggers, and would fall. There is complete anæsthesia of both feet, and cutaneous sensibility is much blunted up to the groins. His pupils are contracted, and motionless to light.

It is unnecessary to enter into greater detail of symptoms, which combine to make up a typical case of tabes. Here, however, it may be worth while to note that the condition to which Duchenne (de Boulogne) gave the name "Progressive Locomotor Ataxy" was one which was characterised by sensory troubles—electric shock-like pains and blunted cutaneous sensibility—as well as by incoordinate gait, and also, although not so constantly, by affection of some motor or sensory cranial nerve, usually in the orbit. Subsequent investigation has associated cases showing this group of symptoms with sclerosis, affecting especially certain parts of the posterior columns of the cord. It is convenient enough to retain such a standard as Duchenne's for comparison, although it need hardly be said that we are frequently meeting with examples which stray away in some direction from the type—cases in which, for instance, either anæsthesia or lightning pains or cerebral nerve troubles, or even ataxy itself, may be wanting.

The opinion that we are justified in including such anomalous cases in the class was expressed in a report presented by Dr Moxon and myself to the Clinical Society ten years since.* The case reported upon was a patient of Dr Greenhow's, and it was an example of tabes, with all the symptoms except ataxy.

We are all agreed, I suppose, that the mere absence of a symptom in a particular case does not at all necessarily exclude it from the category. But for the purpose of association, as, *e.g.* in the present example of joint affection, and also when inquiring into the value of Westphal's test, it is necessary in the first instance to keep to *typical* cases, as it would be manifestly inconvenient to deal with those which wandered from the type, and thus introduced an element of doubt, however small, as to their nature. This man's ataxic gait, had it existed alone, without the characteristic troubles on the sensory side, might have been open to objection as scarcely affording satisfactory grounds for a

* 'Clinical Society's Transactions,' 1869

diagnosis of locomotor ataxy, considering the mechanical difficulties he is under with such a state of the hip-joint. Troubles in the coordination of movement alone cannot safely be depended upon as evidence of posterior sclerosis. They are seen in other conditions. There is a girl under my care in the hospital now whose gait is very ataxic. In her case there are other symptoms which tend to show that she is suffering from tumour or other gross lesion, which is probably in the pons Varolii. Ataxy may occur also in cerebellar disease and in spinal pachymeningitis. In this man, as in all the typical cases of tabes which I have yet examined, we see that blows upon the ligamentum patellæ below the knee-cap, when one knee rests upon another and the leg swings easily, fail to produce any reflex movement upwards of the foot.

I may say here that amongst the cases which, although they were not *typical* examples of tabes, one had yet but little hesitation in including in the class, I have very rarely, indeed, seen the patellar tendon-reflex preserved where *marked lightning pains in the lower extremities* were amongst the symptoms. In certain cases which I have examined, where these pains had never occurred, although the ataxy itself appeared to be very characteristic, the patellar tendon-reflex remained unaffected. Whether or not this association of the occurrence of characteristic pains with the absence of patellar tendon-reflex is at all constant, further experience is required to show. At present certainly, I think, as I have said in former lectures, we must be content to claim that Westphal's test (the absence of patellar tendon-reflex) is the rule in *typical* cases of tabes.

The man's story is soon told. He is a waiter, whose habits of life have not, by contrast, cast reproach upon other members of a calling which is notoriously exposed to the temptations of the bottle. He has suffered from syphilis. For six years he has been subject to the pains already described, but it was only eighteen months ago that he noticed that the soles of his feet were numb. A few weeks after this observation he was walking in the

street when a sudden pain, described by himself (without any leading question) as being "like a shock from an electric battery," shot upwards from his right heel to his hip, and caused him to drop, it was so terribly and unusually severe. He got up immediately, and walked home, a distance of two miles, without pain, lameness, or other inconvenience. Next day he went on foot to his employment as usual, and felt nothing wrong. A few days later, he was taken, he says, with a "kind of fever," and there was great swelling in the right hip, which extended down the thigh, as far as the knee-joint. It was painful. The patient's own account of this period of his history is not very graphic, but we gather that after keeping his bed for three months he noticed that his right leg was shortened. During his confinement in bed he had suffered extremely from the shooting pains before described in the thighs and hips and right foot.

I need scarcely say that we have no right, because we meet with a disorganised hip-joint in a patient who is suffering from tabes, to jump at once to the conclusion that the joint affection is a consequence of the peculiar disease with which he is manifestly affected. An ataxic patient is not more exempt than others from the chance of being affected with rheumatoid arthritis as a coincident disorder. It is necessary therefore to compare the condition of this man's hip-joint with that which occurs in arthritis deformans, to which it bears a superficial resemblance, and to see besides how far its history is compatible with the probability of its being simply an affection of the latter kind intercurrent with tabes.

We find in this case that, so far as can be made out by careful examination, there is not a vestige remaining of the neck or head of the femur. No doubt, in arthritis deformans of *very long standing*, as a result of a gradual interstitial absorption, there is sometimes but little left of the neck of the femur, and the head may be greatly altered in size and shape; but such a condition as we observe here—the femur literally terminating above with

the great trochanter—is not on record, so far as I know, as a result of the disease named. On the other hand, there is a photograph in Professor Charcot's 'Lectures,' * which precisely pictures the state of the femur as it exists, to all appearance, in this patient. The specimen in question was taken from a female who died of aortic aneurism after presenting during many years the symptoms of progressive locomotor ataxy. On autopsy the characteristic sclerosis of the posterior columns was found. Now in the case of this woman there had been an extraordinary succession of spontaneous luxations and fractures. First the right hip-joint was found dislocated (without any history of violence), and then a few months afterwards the left. Next there came a fracture of the shaft of the left femur, and a short time later of both bones of the left forearm. Three months afterwards the right forearm is found to be fractured in its middle; a month later dislocation of the left shoulder-joint takes place. After death, amidst many other striking changes, it was found that the head and neck and a portion of the great trochanter of the right femur had disappeared. On the left side the head of the femur was wanting, and the neck existed only in a rudimentary form.

It was in 1873 that M. Charcot published particulars of this case. But some years previously to this, in 1868, he had first described the joint affection which was apt to occur in patients affected with tabes. Extended experience showed that the shafts of bone, as well as the articular extremities, were liable to startling defects of nutrition. The arthropathy in question was characterised by a sudden, usually but not always painless, and often enormous swelling, which involved, not only the joint, but also neighbouring structures above and below. Punctures showed the enlargement to be due to the presence of a serous and slightly bloody fluid. There were cracking noises in the joint on movement. Such cases would sometimes terminate in recovery, the fluid becoming

* 'Leçons sur les Maladies du Système Nerveux,' fasc. iv, plate x. 1873.

absorbed, and no apparent change in the joint being left behind. In others, however, there would be a rapid destruction of the portions of bone entering into the formation of the joint.

Four years ago there was an ataxic patient under my care in this hospital, who presented in a very perfect form this sudden painless hydrarthrosis in the right knee-joint.* In his case the mischief appeared to be limited to a considerable amount of interference with the mechanism of the joint by the distension with fluid. In the present instance there is evidence besides of a remarkable and extensive destruction of the articulation.

In contrast with the slowness of the changes in arthritis deformans, I would draw especial attention to the extreme rapidity with which this disorganisation occurred. Within the short period of three months the joint was left apparently in the state in which we now find it. This rapidity is strongly dwelt upon by Charcot,† who expresses the opinion that the defect of nutrition, due to an influence of the nervous system which renders the bones fragile and explains the spontaneous fractures, is also one of the principal elements in the production of these singular arthropathies, and that the very rapid wearing away (*usure*) of the articular extremities (which is carried to an extreme degree) is the principal character which distinguishes ataxic arthropathy from arthritis deformans.

What is the meaning of the splinter of bone which in this man occupies the place of the superior tendon of the rectus femoris muscle? The patient tells us that he never observed it until after the period during which his hip-joint went wrong, and in the nature of things it is very unlikely that he could have overlooked the existence of such an encumbrance.

There would seem to have been an ossifying myositis going on in the immediate neighbourhood of a joint, which was the seat of changes having for their result the rapid

* See the preceding lecture.

† 'Archives de Physiologie.' Paris, 1874.

destruction or wearing away of bone. It is an interesting fact, in relation to this apparent anomaly, that in cases of spontaneous fracture dependent upon tabes a more than usually abundant callus has been observed to be thrown out about the broken extremities. At first sight, the excessive production of bone in such circumstances might appear to militate rather strongly against the view that it was a neurotic cause which occasioned the modification of nutrition leading to fracture. It will be remembered, however, that, after experimental nerve-sections (either imperfect or in some way causing irritation of the nerve ?), Mantegazza observed not only hyperplasia of connective tissue and periosteum, but also formation of osteophytes.

We may take it, I think, that, however strong the superficial resemblance may be, this man's case really differs widely from one of chronic rheumatic arthritis. To some it may appear superfluous to insist at all upon the points of differential diagnosis. But this is not so. The patient, before he came to Queen Square, was for some time in a surgical ward of a metropolitan hospital under treatment for chronic rheumatism ; and I have reason to know that he was not long since submitted to candidates for their diploma at an examining board as an example of rheumatoid arthritis. The fact that he is a typical example of progressive locomotor ataxy had remained hidden behind the very prominent lesion of his hip-joint.

LECTURE XIII

OSSEOUS AND ARTICULAR LESIONS IN TABES DORSALIS: THEIR ASSOCIATION WITH GASTRIC CRISES

The patients whose cases I am about to introduce illustrate very perfectly the trophic changes in bones and joints which are apt to occur in tabes dorsalis, and for our knowledge of which we are indebted to Professor Charcot. It is somewhat remarkable that this subject, although equally interesting and important to the physician and surgeon, should have failed, so far as I am aware, to have engaged the attention of any of the metropolitan societies until 1879, when the man whose case is described in the last lecture was shown by me at the Harveian Society. Some months afterwards, Mr Hutchinson made some important references to the subject in his lectures at the Royal College of Surgeons. In the following February, (1880) I exhibited to the Pathological Society two cases to which I now wish to draw attention, whilst I endeavour to make it clear, in the first place, that the patients are affected with tabes dorsalis; and secondly, that the osseous and articular changes are of the kind peculiar to that disease, and not accidental complications.

I will first describe the two cases mentioned, because a study of them opened up a field of inquiry which has proved of great interest.*

Elizabeth W—, æt. 50, is a widow, who was admitted

* These cases were published in the 'Transactions of the Pathological Society of London' for 1880.

an in-patient of the hospital, under my care, on December 31st, 1879. For the opportunity of observing this patient I am indebted to Dr. Lediard, under whose care she had previously been in the Cleveland Street Sick Asylum, and who kindly permitted me to take her for a time into hospital. The notes of the case were for the most part taken by Mr. A. E. Broster.

Patient has one child living, healthy, and has had, besides, three, who died, one a day or two after its birth, a second when nearly three years old, and a third at twenty-one years. She has never miscarried, and has had no stillborn children. Patient has never suffered from acute rheumatism or scarlet fever, and has never had a sore throat (except from cold) or skin eruption. In the family there is no history of tubercle or nervous disorder. Her husband drank heavily.

For the last eleven years she has been subject to frequent and violent attacks of so-called "indigestion," the most severe of which occurred in July, 1878, and lasted till late in September. It was on account of this attack that she was admitted into the Cleveland Street Sick Asylum, where she had been for upwards of a year a patient when I saw her in October, 1879. She would have attacks of gastric pain and vomiting, lasting ordinarily two or three weeks at a time, and recurring at intervals which varied a good deal in length. She has had as many as four attacks in four months, and the longest period of exemption has been eight months.

In the attacks, she says, there is a "bad sensation" in the stomach, and she can keep nothing down. There is great pain in the chest and stomach, which feel as if they were raw, accompanied by much flatulence and a great deal of retching. She will sometimes bring up greenish stuff and food, but she is sick even if no food has been taken. She has never suffered from diarrhœa, but at times there has been a tendency to tenesmus. During the retching there is a feeling of tightness across the chest, and afterwards a soreness.

Soon after the commencement of these gastric attacks, *i.e.* nearly eleven years ago, she had pains in her limbs, in the legs more than in the arms, very sharp shooting pains, so sharp at times that they would almost make her fall down, and indeed, once or twice, when the pains have seized her in the knee, she has fallen in the street. After the pains she observed a weakness in the extremities. The pains have continued at intervals. Frequently they have kept her awake all night. She may have a "bout" of pains, lasting a day and night, in one situation, and then after a few days', or even weeks' intermission, a recurrence in another place.

For the last four or five years she has had a fear of walking in the dark, and even in the light she would stagger as if drunk. Eight or nine years ago, while walking, a man stopped her and inquired: "Is'nt the pavement wide enough for you, ma'am?" This was the first time her attention was called to the staggering. There was weakness of the right leg, but she could walk without assistance three or four years ago.

In October, 1878, while walking quietly along the ward in Cleveland Street Asylum, "the right leg gave two snaps," and it was found that her thigh was broken.

In July, 1879, whilst retching in bed, the left hip-joint suddenly "bulged out."

Patient has never had double vision. Her bladder has never leaked, and no difficulty with the rectum is complained of.

At the present time the patient is confined to her bed or chair on account of the condition of her lower extremities. At my request Mr William Adams has been good enough to examine the state of the hip-joints, and the following is his report:

"In reference to the condition of both hip-joints, I made a careful examination on the 15th January, 1880.

As the patient is lying in bed on her back the left leg is rotated inwards, and apparently shortened. There is no real or permanent shortening, for upon examination

very little, if any, difference in measurement can be made. On both sides the measurement from the anterior superior spinous process to the upper margin of the patella is twelve inches. When extension is made on the left leg this measurement increases one inch and a quarter, and when on the right one inch.

The right leg lies straight. The movements of both hip-joints are free, but limited in some directions, with crepitation much more marked in the right than the left.

In the right, flexion and extension movements are free; abduction and rotation inwards are limited. The inclination is to rotation outwards. In the left leg, flexion and extension movements are free, but rotation outwards is extremely limited. Rotation inwards is much more than normal. The inclination is to rotation inwards, with a little flexion at the hip and knee.

In neither of the thighs is there any evidence of fracture of the *shaft* of the bones.

On the right side the top of the great trochanter is horizontally nearly on a level with the anterior superior spinous process, and rather depressed than otherwise.

On the left side the top of the great trochanter is not much displaced upwards, but the whole trochanter is extremely prominent and the bone enlarged, at first sight conveying the idea either of dislocation or a large amount of callus thrown out after a fracture. Neither of these conditions, however, can be definitely made out.

There can be no doubt about the existence of structural changes in the head and neck of the bone, as well as great enlargement of the great trochanter and upper portion of the shaft.

There may have been spontaneous fracture of the neck of the thigh-bone in both hip-joints, or the present condition as to shortening (*i.e.* unnatural mobility in this direction, whilst mobility is limited in other directions) may be the result of complete disorganisation of the joint, with atrophic changes and absorption of the head and neck of the bone on the right side, and similar changes

on the left side, associated with enlargement of the great trochanter from throwing out of new bone."

For the rest, she has had during the last three or four years great difficulty in buttoning or doing anything with her hands which she could not see, and this especially with the left hand.

She complains of an aching pain in the right leg. She has not had any sharp pains for a week or two. They come on, she says, in paroxysms, and last twenty-four to forty-eight hours. "It is like electricity," is her description, "shooting from one place to the other, sometimes down one arm and sometimes down a leg." She does not remember the pain ever to have occurred in both legs at the same time, but it will seem, she says, to shoot from one leg or arm to the other, and sometimes from the arm to the leg. The pains are always worse on the right side.

Her pupils are very small, and do not react to light. They both dilate when she looks up. They contract during accommodation. There is apparently some drooping of the eyelids.

No abnormality is to be noticed in the cutaneous sensibility of the face, pharynx, uvula, or palate. The right grasp measures, by the dynamometer, fifteen kilogrammes; the left the same amount.

Cutaneous sensibility.—Upper extremities.—A slight prick with a pin is not recognised in the tips of the left fingers; a sharp pinch is recognised after a delay of two seconds. Touch is felt as "numby." In the right finger tips a slight pinch is not recognised at all, a sharp one only after three seconds.

On both forearms and arms sensibility is not apparently affected.

Muscular sense, as to the position of the arm, is very deficient, but she tells the difference in the weight of variously-loaded balls accurately.

Lower extremities.—The patient feels a needle prick, after marked delay (two or three seconds), in the leg and foot-sole, but not at all in the right. She recognises a

finger-touch, however, on each leg. The muscular sense is very defective. She feels as if her legs were across one another.

The patellar tendon-reflex is absent in each leg. The skin-reflex of the foot-sole is normal.

The other female, Catherine M—, æt. 36, is married, has two children, and has had miscarriages.

The patient is at present an out-patient of the hospital under my care. I am indebted for the opportunity of observing this case to Professor Henry Smith, by whom she was treated in King's College Hospital during the month of January last year. The following are notes which were taken at that time by the surgical registrar and dresser of the case:

"She has had 'rheumatic' pains in all parts of the body at different times.

About six years ago she noticed weakness in her legs, for which she attended at St Mary's Hospital. She describes the weakness as having been chiefly in the left leg, and accompanied by numbness. She could not feel the ground as she walked, and was constantly falling. At this time there was no double vision.

The difficulty in getting about has been gradually increasing until two years ago, when the right leg became red and swollen, without being painful. With rest in bed the swelling subsided, but on getting up and moving about it recurred. About this time, also, pains of a plunging character used to attack her, the shock of them lasting only a second or two.

In June, 1877, she had another attack of swelling and redness in the right leg, which continued until September, when the swelling disappeared, but the joint was affected and useless.

Before admission her left leg began to swell, and has remained more swollen than natural up to the present time.

She also noticed a peculiarity in her vision, a shawl that she knew to be red and black appearing green. At

times she is unable to distinguish colours. She says that when standing she was obliged to keep her eyes open, for on shutting them she felt as though she would fall.

The patient is fairly nourished. The left leg is swollen, painful, and red; the left knee-joint not affected. The muscles of the left thigh and leg are wasted; the same conditions exist on right side.

The right knee-joint is disorganised; the skin over it is natural in colour and to the touch. There is no pain in the joint, even on applying firm pressure. Grating can be felt when the extremities of the bones in the knee-joint are moved upon one another. The leg hangs loosely, and is dislocated backwards and rotated outwards. The internal condyle projects inwards, and is enlarged. The external condyle cannot be felt. The patella rests on the outer surface of the lower end of the femur; the ligamentum patellæ can be felt, but is apparently wasted. Fluctuation can be felt in the joint.

The bowels are torpid; cramping pains are continuous in the belly. There is partial loss of control over the bladder. Micturition is frequent.

Urine.—Reaction acid; no albumen or sugar.

There are occasional sudden starting pains in limbs.

A difficulty is experienced in picking up pins, from inability to feel them.

There is no spinal tenderness.

January 13th.—Since admission the patient has remained at rest in bed. The swelling and redness of the left leg have gone down.

The knee-joint is considerably enlarged, especially the lower end of the femur.

The leg is becoming dislocated backwards and outwards. There is great laxity of the ligaments of the joint.

Other symptoms continue except those of the bladder, micturition being less frequent, and urine scanty, only thirty fluid ounces being passed in twenty-four hours no incontinence.

Both knees are now kept in place by leather cases."

Since her discharge from King's College Hospital she has lived at home, and, thanks to the support given by leather cases for her knees, has been able to employ herself in housework.

At a recent examination of her I made the following observations:

She may go one or two months without pains, and then they attack her for three or four days and nights in succession, keeping her awake. They are plunging and stabbing, "just like being stabbed with a fork." In her gastric attacks she has pain at the pit of the stomach, going through to the back, and there is dreadful griping and belching of wind. For three or four weeks past she has had this every day, but now she is getting better again. The attack usually begins about 4 or 5 a.m. It is not accompanied by diarrhœa, but the abdomen is distended with flatulence.

After a bout of pains of this description she may go one, two, or three months without any, and she feels as though she would never have them again. Then she will be suddenly seized with them.

The stomach pains began more than ten years ago. She has been married ten years, and previous to that had suffered from the stomach pains, though not so severely as she has done since. At that time, too, she used to have stabbing pains in her knees and one of her finger-joints, which she thought were due to rheumatism.

Formerly, when the stomach pains attacked her, "she would vomit all day and all night" for two or three days together. She threw up "enormous quantities." After a few days this gradually ceased.

Between the intervals of the pains she feels quite well. Her appetite is good, and she has nothing (except the crippled state of her legs) to complain of.

The plunging pains described attack her in the joints and limbs, but never in the trunk, except in the stomach. She may have gone three months, but never six months, without the gastric symptoms.

She says that the swelling in the legs extended at first quite down to the feet, so that she could not see her ankles.

Her pupils are very large. They do not contract to light, nor, I think, during accommodation.

She has lately had swelling about the right shoulder-joint, which has almost disappeared. But the joint creaks, and is somewhat painful when moved. The left shoulder-joint has also still more recently begun to "feel queer." She recognises in these symptoms a close resemblance to the manner in which her knees began to get bad. There was no pain in the right knee-joint, she tells me, whilst it swelled.

She is quite lost in the dark. There is difficulty in buttoning things. In sewing she has stuck the needle into her finger and not felt it. She has put her feet into water nearly boiling, and for a few seconds could not tell whether it was hot or cold, but has then had to withdraw them.

The muscular development of her arms is very striking, and resembles that of a labouring man. Owing to the difficulty with her legs a great amount of work is thrown upon the arms in moving about, and hence this muscular hypertrophy.

Mr Broster has been good enough to examine her knee-joints, and the following account is derived from his report:—On removing the leather casing from the right knee-joint, the joint seems to fall abroad. The patient can replace it herself and says that when she puts on the casing she has to take the bones and put them straight in their places, and bind them up with a bandage to keep them together whilst she puts on the casing. The joint can be twisted about and the bones knocked together audibly without causing pain.

When the joint is handled the feeling is as if all ligamentous connections had disappeared, and the bones were simply held together by the muscles and cutaneous tissues. It seems as if the lower end of the femur had

had its condyles bevelled off and the end turned into a rounded stump. It feels smooth, and the posterior fossa can be distinctly made out.

The patella, which is perfect, lies drawn upwards and outwards, about two inches above the end of the femur. Above it is felt what appears to be a synovial sac. The peroneo-tibial articulation (upper) seems perfect.

The upper end of the tibia is bevelled off inwards; the fibula with the outer part of the tibia can be felt projecting along the outer side of the inner hamstring in a firm smooth mass, about one inch long by half an inch in diameter, somewhat rounded.

The ends of the bones are not in apposition, nor can they be got into apposition. The tibia and fibula project upwards on the outer side of the femur. Their upper ends are exceedingly loose, and it seems that when the patient fixes on her casing, instead of putting the bones in normal apposition "end on," she splices them together tightly side by side. She says the lower bone always projects outwards, never inwards. She say that "formerly the joint was enormously distended." There is certainly no distension now.

Left lower limb.—On removing the casing the bones, which had been in apposition "end on," at once separate, the tibia and fibula being dislocated backwards with a tendency outwards. There is marked laxity of the joint. The tibia and fibula can be moved slightly laterally and very freely backwards.

The patella lies over the lower extremity of the anterior surface of the femur. The condyles appear to be rounded off, and the inner one is larger than it should be. There is no pain in examining the joint.

Cutaneous sensibility.—A pin-prick is not felt in either foot-sole. In both legs a severe prick with a pin is felt after three seconds' delay, and then but very indistinctly. In both thighs the same is felt indistinctly after a delay of two seconds. She feels a pin-prick on finger-tips and hands very indistinctly after a delay of two seconds.

In the forearms and arms a pin is felt, but not so well as on the face.

As I have before mentioned, it was in 1868 that Professor Charcot for the first time drew attention, in the 'Archives de Physiologie,' to the arthropathy which was apt to occur in the course of tabes dorsalis. His memoir was speedily followed by that of Professor Ball, a translation of which appeared in the 'Medical Times and Gazette,' 1868-69. Soon afterwards Dr Clifford Allbutt published in the 'St. George's Hospital Reports,' 1869, "A Case of Locomotor Ataxy with Hydrarthrosis," the first example described in England. Since then a considerable amount of attention has been paid to the subject, especially in France, and numerous contributions have appeared at the hands, amongst others, of Richet, Bourneville, Vulpian, Bouchard, Dubois, Voisin, Oulmont, Bourceret, Michel, Forestier, Blum, Damaschino.

Previous to my communication to the Pathological Society the only cases of tabetic arthropathy published in this country, so far as I am aware, since Dr Allbutt's, had been two of my own,* and one by Dr H. Thompson.†

In 1873 Charcot communicated to the Société Anatomique of Paris the case of a woman affected with tabes dorsalis, in whom, concurrently with great disorganisation of certain joints, multiple *spontaneous fractures* of the neck of the left femur, of both bones of the forearms, right as well as left, had occurred. About the same time Weir Mitchell, in America, in an article "On the influence of Rest in Locomotor Ataxy," ('American Journal of Medical Science,' July, 1873), alluded incidentally to the frequency of fractures in tabetic patients. Until then the question had only been one of the influence of tabes in causing a condition of joint which had been previously confounded with arthritis deformans. The relation of a peculiar friability of the bones to this disease of the nervous system now became an important question. In 1874 necropsies

* Described in Lectures XI and XII.

† 'Medical Times and Gazette' for August, 1877.

published by Richet and Voisin, and especially some by Raymond, in 1875 and 1876, threw light upon the subject.

At the present time Charcot considers these osseous lesions as the primordial fact, the joint affection being only, like the fractures, secondary manifestations of this defective nutrition of the osseous tissue.

"Destruction and disappearance, partial or complete, of the epiphyses," writes M. Talamon,* "fragmentation and absorption of the head of the humerus in one case, atrophy of the projecting portions, such are the principal and always identical alterations of the osseous extremities that have been met with in the shoulder, the hip, and the knee. In no case have there been observed the hyperostoses, the epiphysal hypertrophies, which characterise dry arthritis. In cases of spontaneous fracture from ataxy certain bones have been found reduced to a remarkable state of thinness and deformity." In one woman the left femur, which had been fractured, measured nineteen centimètres, the right forty centimètres. The bone was found to be formed of two portions united by an over-abundant callus. And it is noteworthy, in reference to the first of the two cases I have described, that it has been the rule to find in examples of tabetic fracture the bones consolidated with an unusual amount of callus. "In one of the forearms," Talamon writes, "the work of cicatrisation had engulphed in one and the same exuberant mass the fractured extremities of both radius and ulna."

The two patients whose cases are in question present examples of all the conditions which have been described as characterising the osseous and articular lesions of locomotor ataxy. In the one case extensive swelling, usually painless, and never confined to the joint, but extending also down the long axis of the limb, disappearance or disorganisation of the articular ligaments, rapid erosion, and absorption of the ends of the bones entering into the

* 'Revue Mensuelle de Méd.,' Paris, 1878. I have largely availed myself of M. Talamon's references.

joint. The rapidity with which these changes have been brought about is very marked, the period in each instance occupying but a few months. In the other case we have a spontaneous fracture of the neck of the femur, certainly on the right side, and I should have no doubt, from the history, also on the left, although the existing condition is compatible with the alternative of a disorganisation of the joint. I would submit that the evidence of the patients being examples of tabes dorsalis is not open to question.

Before proceeding to describe some further examples of this disease which have since come under my observation it will be convenient if I refer to the question of the probable seat of the lesion which brings about such startling changes in the nutrition of bones and joints in the course of tabes dorsalis. Out of this some points will be found to arise which suggest, as it appears to me, a new direction in which pathological anatomy might turn its inquiries.

I would first remark, that on account of the wasting of muscles which was sometimes observed to be coincident with these osseous changes, it was early suggested by Charcot that the cause of these lesions of bones and joints would probably be discovered in atrophy of the anterior cornua of the spinal cord.

At first the result of observation appeared to be favourable to this view. In three cases, as I gather from Talamon (loc. cit.), atrophy of ganglionic cells in the anterior horns was observed. But in three more recent autopsies, I learn from the same authority, it has been impossible to find the least alteration in these cells, and, moreover, no muscular atrophy existed. This appears to be almost conclusive evidence against the seat of lesion being that which was supposed.

In addition, I would remark that cases of advanced progressive muscular atrophy are of not uncommon occurrence, in which microscopical examination shows extensive destruction of cells in the anterior cornua, whilst there have been during life no changes whatever in the joints

or bones. It is true that this is not quite without exception. I have met with one case, a female, suffering from protopathic amyotrophy, in whom the finger-joints and elbows were the seat of marked changes. But these changes were precisely of the kind which is met with in arthritis deformans, and very distinct from those characteristic of tabes. So, also, it is true that perhaps more often than not, in infantile paralysis, not only are the constituent parts of a joint relaxed so that the limb dangles, but the bones of the affected extremity cease to grow at the normal rate, and more or less deformity occurs. But here the pathological condition is widely different from that which obtains in tabes. Spontaneous fracture, again, forms no part of the history of infantile paralysis, nor of progressive muscular atrophy, diseases in which the lesion consists in atrophy of the anterior cornua of the spinal cord; and in neither of them do we ever see sudden painless and enormous swelling, involving not only the joint, but the whole extremity, followed by a rapid disappearance by absorption of an entire epiphysis and of bone beyond it, such as occurred in two of the cases related. No doubt the partial check to the growth of bones which takes place in infantile paralysis shows that the integrity of the anterior cornua is in some way necessary for the due development of osseous tissue; whether directly or indirectly is another question. But in the arthropathy of tabes there would seem to be a destructive process at work different from any seen in other conditions, and implying, therefore, lesion of some portion of the nervous centres distinct from the anterior cornua of the cord, the effects of lesions in which are well recognised.

In one of the female patients, M—, whom I showed at the Pathological Society, and whose case I have just described, the two knee-joints were so hopelessly disorganised that the patient had to use crutches, and to make great exertions with her arms, as she could not support herself on her legs. As a result, the muscles of

her arms were developed, as I have said, like those of a labourer. Yet it was one of her shoulder-joints, bordered by these large muscles, which had begun to show signs of the same kind of affection as that in her knees. There had been great effusion, both in the joint and under the muscles of the shoulder and arm down to the elbow, and there was crepitation on movement. Such an association of muscular hypertrophy with joint-affection appears to be absolutely conclusive against disease of the anterior cornua being the cause of the latter.

The electrical reaction observed in these cases gives evidence in the same direction.* In three of the cases which will presently be described, I carefully tested electrically the muscles in the neighbourhood of the affected joint. In the case of P—, whose left knee-joint was the seat of disease, the vastus internus muscle of that thigh responded to the lowest strength of induced current which caused contraction in the corresponding muscle in the right limb. In Matilda H—, whose left hip-joint is the seat of change, the muscles of the thigh respond to a normal strength of induced current—to the same strength as causes contraction in those of the right thigh. In the case of Henry H— alone I found a slight reduction of faradaic excitability in the deltoid of the affected shoulder. This muscle was wanting in thickness, and was flabby. It had been exposed to a good deal of pressure from the large quantity of fluid which had recently occupied the joint, and, moreover, it was disused. In the circumstances, I think this was sufficient to explain the slight diminution of excitability. In the case of S—, whose left knee-joint was disorganised, I found the muscles of the affected thigh actually more excitable to faradaism than those of the opposite side.

This preservation of faradaic excitability in muscles in the neighbourhood of disorganised joints affords manifestly a strong presumption against the view that disease of the anterior cornua is the cause of the arthropathy and absorp-

* See Lecture XIV.

tion of bone. The fact, too, that the patient W—, whose right hip-joint was completely disorganised (the head and neck of the femur being absorbed), was still able to walk some miles without crutches bears strongly in the same direction.

With still less probability can we attribute the cause of the affection of bones and joints to the disease of the posterior columns of the cord, which is the essential lesion in tabes. For it is notorious that the most extensive changes in the posterior root-zones are perfectly compatible with complete preservation of the integrity of the joints.

Moreover, although it is certainly common for the patient to have suffered severely from lightning pains in the limb, which afterwards becomes the seat of arthropathy, this is not universally the case, as it would probably be were the joint affection connected with the disease of the posterior root-fibres. I questioned Matilda H— very carefully upon this point, and she told me distinctly that, although she had suffered severely from shooting pains about the malleoli, legs, and popliteal spaces, she had *never* had pains in either of her hip-joints. So, again, P—, whose lightning pains had affected both knees and ankles, had not suffered more in the left than the right knee; and for a year before any signs of mischief presented themselves in the knee-joint he had experienced much less pain than before. On the other hand, it is true, Henry H— told me that the pains had been exceedingly severe in the left shoulder-joint, which was the seat of the changes. Another of my patients (W—) also described the occurrence of an unusually severe shock of pain in the affected limb immediately before the joint changes began.

I have already referred to some of the characters which serve to distinguish these affections of joints from arthritis deformans. Let me add that I have examined a large number of cases of the latter disease, and have only found the knee-phenomenon absent in them when the condition

of the knee-joint showed that the absence was due to purely mechanical causes—osteal growth or contraction of muscles. Additional evidence of the difference between the two conditions may be found in the fact that the arthropathy of tabes often occurs alongside of such a friability of the shafts of the bones as disposes them to spontaneous fracture. This, as is well known, is no part of the pathological history of arthritis deformans. Lionville* in two cases—one of tabetic arthropathy, the other of spontaneous fracture in a tabetic patient, found similar lesions in the respective bones. The Haversian canals were enormously dilated and filled with fleshy papillæ, the osseous substance thinned, and as if eroded by these masses of embryonic cellules. He refers the condition to a rarefying osteitis. Raphael Blancard† has also made anatomical examination of the bones, and finds that the lesion starts by a disappearance of calcareous salts, the erosion of the Haversian system being a secondary phenomenon.

Chemical analysis, according to Regnard‡ shows that in the osseous affection of tabes we have to do with a true trophic lesion of bone, a fatty degeneration with disappearance of the mineral substance. There is an enormous diminution of phosphates (11 parts instead of 48 parts in 100). Fat, of which there is but little in normal bone deprived of its marrow, rises to 37 parts in 100.

It is probable that the condition of osseous tissue described is the real explanation of many of the cases on record of spontaneous fracture which have been referred to various causes. In 1853, for instance,§ Dr Van Oven is described as having brought his own case before the Royal Medical and Chirurgical Society of London. When fifty-six years of age his femur was fractured one night from muscular action, as he lay in bed. He had occasionally

* Quoted by Talamon, loc. cit.

† 'Gaz. des Hôpitaux,' 1881, No. 19.

‡ Soc. de Biologie, 13th January, 1880.

§ See Braithwaite's 'Retrospect of Medicine,' vol. xxvii, p. 131.

suffered from slight *neuralgic pains* in various parts of the body. On the day before the occurrence of the fracture he had felt a slight numbness in the leg and foot. There would seem to be little doubt from these symptoms that he was affected with tabes.

Now, I would direct attention to a very interesting circumstance which has struck me in connection with cases of this class. I find that the gastric symptoms of tabes —the *crises gastriques* of Charcot—are of extraordinarily frequent occurrence in cases of tabetic arthropathy. The frequency of this association is far beyond what might be reasonably explained by an accidental association, as I think will appear evident presently.

The two female patients both present typical examples of *crises gastriques*. The symptoms are very remarkable, and they occur but rarely in tabes. The circumstance therefore of meeting with two such pronounced cases at the same time struck me as being so unusual that I at once referred to published cases of osseous and joint affections in tabes in order to see whether the occurrence of gastric crises had been often noted in the histories of such patients. This was what I found:

Out of the seven cases characterised by osseous or articular lesions which are narrated by Charcot in his lectures, no less than three were marked by the presence of gastric crises. Vulpian* says that he has only seen one case of spontaneous fracture in the tabetic patient. That case was marked by *crises gastriques*.

In looking through French periodicals I came upon nine other cases of osseous lesions in tabetics, reported by different authors, and in two of these there were typical *crises gastriques*.†

In the case of S—, described in a previous lecture,‡ although there were not crises of the remarkable and violent character which occurred in the two females, the

* 'Maladies du Systême Nerveux,' Paris, 1878.

† Heidenbach, Société d'Anatomie, 1874. Voisin, ib., 1874.

‡ Lecture XI. See also p. 199.

patient was troubled with gastric disturbance of a peculiar kind. He would require to relieve his bowels five or six times in the twenty-four hours, the motions being small in quantity and solid. I believe that this symptom may properly be classed with those I have described, but as it is not an example of what may be called typical gastric crises I shall not include it in that category.

In the man W—, on the other hand, there have been no gastric symptoms.

There must have been more cases of tabetic arthropathy in print, but it chanced that these were all that I was able to meet with. If we add together these various observations we find that out of twenty-one cases of osseous lesions of this class no less than eight were characterised also by the occurrence of typical gastric crises. I did not like to include the sixteen cases of arthropathy consecutive to locomotor ataxy published in the mémoire of Prof. Ball,* because many of these occurred in the practice of M. Charcot, and there would be danger, therefore, of counting a case twice over. It is worthy of remark, however, that Prof. Ball had himself been struck by the unusual frequency of visceral symptoms in the cases of arthropathy which he groups together. The following is his reference to this point:

"Il est peut-être interessant de noter ici que dans un quart des cas (4 sur 16) des troubles viscéraux liés à l'ataxie locomotrice progressive, et paraissant dépendre d'une lésion du grand sympathique, se sont développés parallèlement aux accidents articulaires."†

The startling character of the proportion (eight cases of associated gastric crises out of twenty-one cases of tabetic arthropathy) became evident when the comparative infre-

* 'Gaz. des Hôpitaux,' 1868-69.

† I have been much interested, on again referring to Ball's paper, to find that in two of the cases *laryngeal crises*, in two other gastric crises, occurred. The importance of the bearing of this fact on my suggestion that the lesion causing osseous affections is in the neighbourhood of the pneumogastric nucleus is evident.

quency of the special gastric complication in tabes was considered. I had been lately tabulating fifty-six cases of tabes dorsalis, which had occurred in my own practice, for comparison. In only eight of these were there gastric symptoms, and in only four were these so marked and definite as to be properly included in the category of *crises gastriques*.

In the course of the discussion which followed the paper, read before the Pathological Society, in which I pointed out this association, the President (Mr Hutchinson) mentioned the case of a patient of his own, a gentleman of middle age, who, after having been subject to frequent and periodical attacks of severe gastric pain and vomiting, became affected with what was at first supposed to be rheumatic arthritis of the right hip-joint, but which the presence of characteristic lightning pains and the subsequent occurrence of white atrophy of the optic nerves showed to be connected with tabes dorsalis. Dr Allen Sturge also referred to a case which he had under observation, that of a man affected with locomotor ataxy, with marked gastric crises, who had developed hydrarthrosis of the knee-joints. On the other hand, Dr Gowers showed a specimen (the elbow-joint) from a case in which there was no history of gastric crises.

It became of course important to observe whether the proportion of cases in which tabetic arthropathy was associated with gastric crises was preserved over a larger range of numbers than I had been able to quote. But, even as it was, the frequency of this peculiar combination of symptoms appeared to me to point, without doubt, to something more than an accidental coincidence.

Now, the gastric crises, as I have before remarked,* are suggestive of irritation of the roots of the vagus nerve in the medulla oblongata. The character of the retching, vomiting, and pain resembles remarkably that of the symptoms of this class which I have seen occur in hydrophobia, in which disease, as is well known, vascular

* See Lecture IX.

changes are often found in the neighbourhood of the pneumogastric nucleus. The lightning-pains of tabes, which depend upon irritative lesions of the posterior root-zones of the spinal cord, are paroxysmal in character. We cannot explain the reason of this. All we know is, that they tend, after a few hours, days, or weeks, to pass off, and leave the patient absolutely free till another attack occurs. The gastric crises are ordinarily characterised by a precisely similar tendency, and we have only to imagine the inflammatory changes which affect the posterior root-zones of the cord extending upwards into the medulla oblongata, and irritating the roots of the vagi, to see a complete explanation of the peculiarities of the gastric crises.

The probability being allowed that the gastric attacks depend upon sclerosal changes in the neighbourhood of the pneumogastric nucleus, the remarkable frequency with which osseous changes are associated with their occurrence led to that which seemed to be a legitimate inference, that lesion of a structure adjacent to the nucleus of the vagus might be found to explain the former complication. It was only as an hypothesis that I ventured to offer the suggestion. Is there something which serves directly or indirectly as a trophic centre for the osseous and articulatory system in the immediate neighbourhood of the roots of the vagi ?

LECTURE XIV

OSSEOUS AND ARTICULAR LESIONS IN TABES DORSALIS: THEIR ASSOCIATION WITH GASTRIC CRISES (*concluded*).

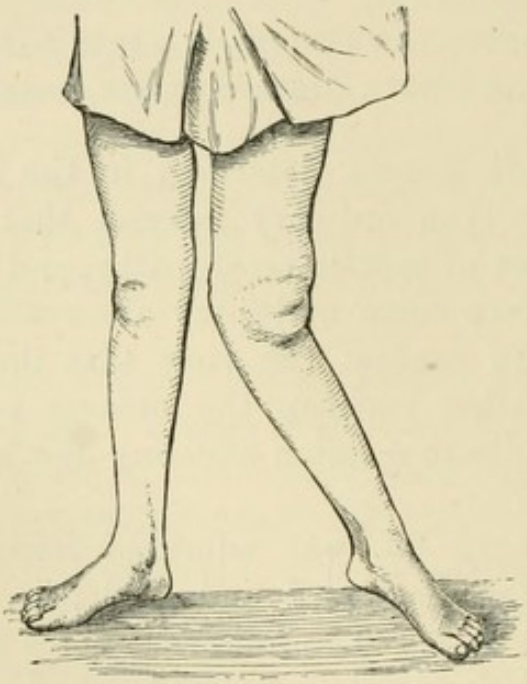
In the circumstances described in the last lecture, it was with more than ordinary interest that I looked out for further cases of tabetic arthropathy, and the following, which have since come under my observation, go far, as will be seen, to confirm the view that there is an important association between the osseous symptoms and those which indicate a lesion affecting the nucleus of the vagus.

J. R. P—, aged 60, was admitted into the National Hospital for the Paralysed and Epileptic on July 2nd, 1880, under my care. The following notes were taken by Dr Beevor. For fourteen years he had suffered from characteristic lightning pains in the legs, and for two years his gait had been ataxic. Cutaneous sensibility was greatly impaired in his feet, and a pin-prick was felt only after a delay of two or three seconds. Patellar tendon-reflex was absent. The pupils were very small: they contracted during accommodation, but not to light. There was delay with the bladder.

Between July and November, 1879, the left lower extremity became greatly swollen from above the knee to the ankle, without any pain. Last February the swelling went down, and the knee-joint was found to be loose, as it is now. The joint remained enlarged; it tapered upwards and was rounded abruptly below. Its circumference was fifteen inches and a half, against thirteen inches and a half, the measurement of the right knee. The joint was semi-elastic on palpation; and the capsule appeared

to be much thickened, especially in front. Grating was felt when the patella, which floated, was rubbed upon the end of the femur, and when the tibia was rubbed against the femur. The joint was very loose, the tibia being

FIG. 18.



capable of abduction so as to form an angle of 150° with the femur. The left thigh lying flat on the bed, the tibia could be over-extended, so that the heel was elevated nine inches; and it could be rotated freely. The left vastus internus muscle contracted at its motor point to the lowest strength of induced current required to cause contraction of the right vastus internus.

Henry H—, æt. 62, is an inmate of the Kensington Workhouse, for the opportunity of examining whom I am indebted to the kindness of Dr Whitmore, resident medical superintendent. It seems that the patient usually had good health till he was about fifty years of age, and then one day he was seized with a dreadful pain in the epigastrium, and a few hours later retching began and continued. He drank cold water, and when that was

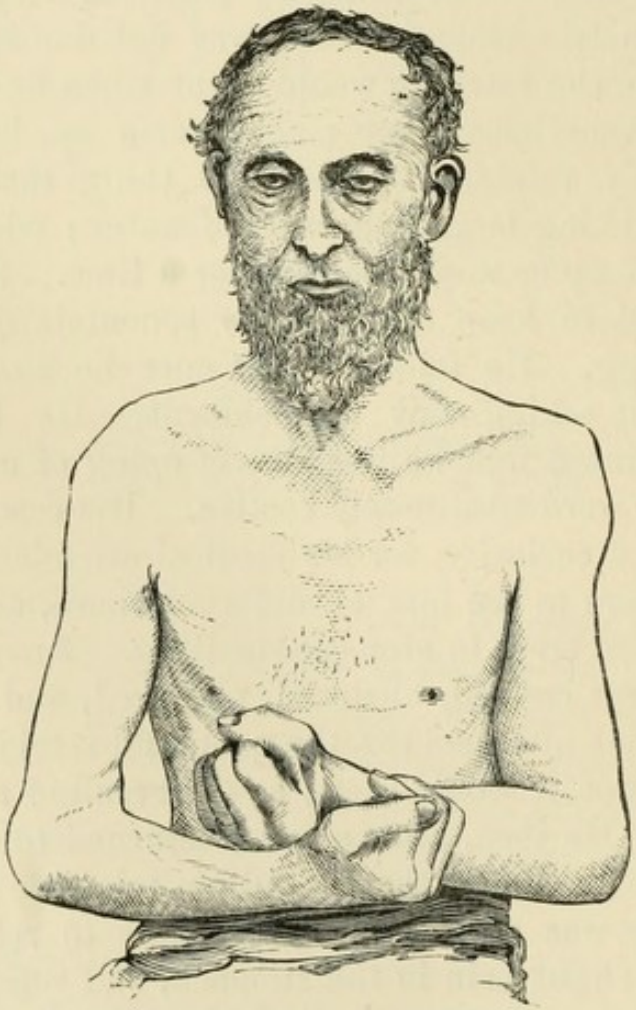
vomited the pain was eased for a time, but then returned. For week after week he went on, with almost constant suffering. There was great aching over the chest generally, but the focus of pain was below the ensiform cartilage.

For nine months, he says, he was never a day without pain and retching; not always equally severe, but so bad that he never left his room, and kept his bed for the greater part of the time. He would roll about with the pain, trying all sorts of different positions to get relief. The pain caused sickness, which was not due to what he swallowed, as the retching would occur when he had taken nothing. Sometimes some mucus came up, but it was chiefly empty retching. The only thing that relieved him was drinking large quantities of water; when he had brought that up he would be easy for a time. Sometimes he contrived to keep down a few spoonfuls of brandy, milk and egg. He tells me that once he was for eight days without taking any nourishment. He became so thin and reduced that he was nearly dying of exhaustion. The bowels were obstinately costive. His case, it would seem, excited curiosity, for his medical attendant brought several doctors to see him at different times, and various remedies were tried to stop the vomiting. Amongst other things, he was cupped, blistered, plastered, and leeched.

After about nine months the pain and retching gradually ceased, not, it would seem, as the result of any special treatment. He then, after a time, returned to work, and remained at his duties, though only in tolerable health, for a year. He was still occasionally liable to relaxation of the bowels, slight pain in the stomach, and retching. At the end of a year he was again laid up for three months, with symptoms like those of the first attack, but on this occasion not quite so severe. Altogether, he has had some half dozen attacks, all of the same character as the first, but none quite so bad. They would last some six or eight weeks, and during this time he was quite incapacitated, and had to take to his bed.

In this case we have, there can be no doubt, an example of the *crises gastriques* of more than ordinary severity. For the time being the gastric symptoms dominated everything, and no suspicion of the general disorder with which this man is affected had ever occurred, until an examination, which I made on account of the joint affection, conclusively showed the nature of his case.

Fig. 19.



For five or six years he has been liable to sharp momentary pains—in the legs at first, and eventually in all parts of the body; an attack of these pains lasting from twelve to twenty-four hours. At the present time he scarcely ever goes a day without them. He is a spare, not unhealthy-looking man. There is no ataxy, but he is

lame in the left leg from localised paralysis of the tibialis anticus group of muscles, which give no response to electrical currents. Electrical currents applied to the skin of his legs cause no sense of pain, though he recognises the very lightest touch. Patellar tendon-reflex is absent. The quadriceps extensor muscles act normally to the induced current. The skin-reflex of his soles is slightly in excess. His pupils do not contract to light, nor during accommodation. The left shoulder-joint is enlarged, and apparently contains fluid. It is abnormally movable, and there is much crepitus when the humerus is rotated, which it can be with quite unnatural freedom. The head of the bone has apparently disappeared, and the limb can be pushed upwards so that the left upper arm becomes more than an inch shorter than the right. The shoulder began to swell nearly three years ago, the swelling extending down the whole upper arm to the elbow, where it pointed and matter was discharged. At the same time, he says, the left side of his chest was also swollen.

Suppuration in a joint affected with this disease is rare, but not unexampled. Charcot has reported having met with three instances out of fifty cases of tabetic joint-affection.*

Matilda H—, æt. 50, was a patient in the National Hospital, under the care of my colleague Dr Radcliffe, to whose courtesy I am indebted for the opportunity of including the case. Notes were taken by Dr Beevor. Ten years ago she had a slight attack of left hemiplegia, from which she recovered entirely in a week. For fifteen years she has suffered from so-called "rheumatic pains" in the arms and legs, and for the last five years she has been ataxic. Eighteen months ago her left hip-joint gave way with a snap as she was stooping down, but without any pain, and she found she could not stand on the left leg. Directly afterwards she noticed a swelling in the left groin, and, a few months later, the left leg

* 'Bulletins de la Société Anatomique,' 1875, p. 334.

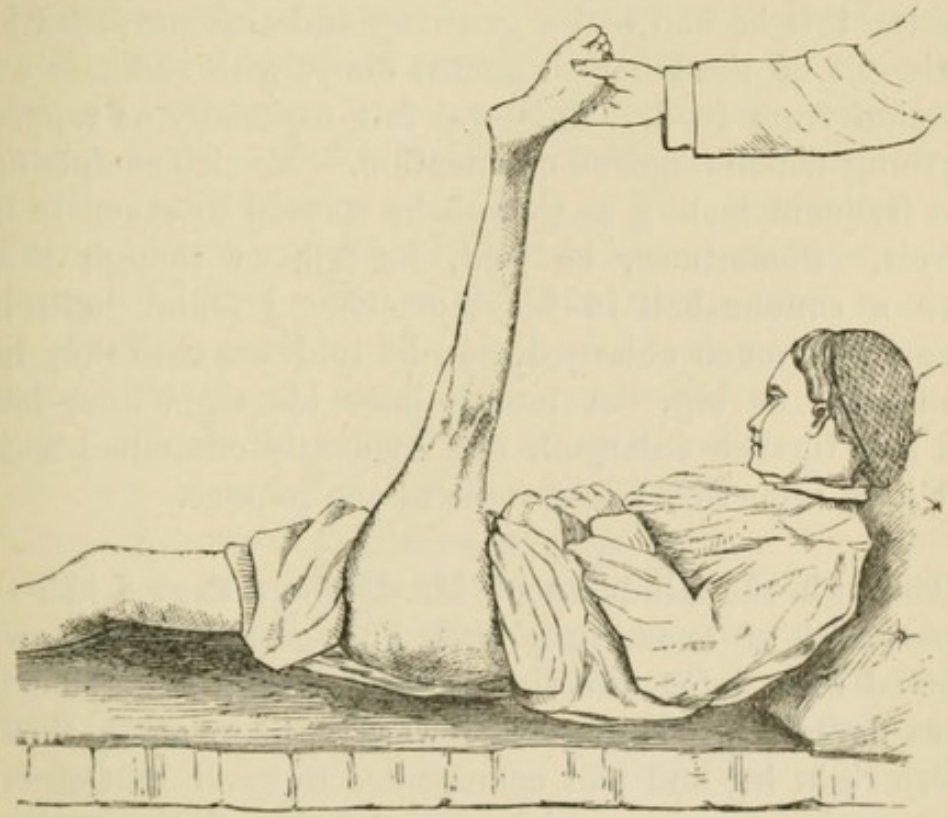
was noticed to be shorter than the right; and it has continued, she says, to grow shorter.

Momentary shooting pains have existed for two years. They come on suddenly, and she feels as though her joints were being "screwed into." The pains have been apt to attack the outer malleoli, the legs and the hams; but they have never occurred in either of her hip-joints. They are very sharp, "like flashes of lightning," and make her cry out. Cutaneous sensibility is somewhat impaired in the legs, and there is a little delay in appreciating pricks with a pin. There is marked hyperalgesia to hot metal in the legs; but there is a delay of three seconds before she can feel the heat, which is then described as intolerable. Patellar tendon-reflex is absent. The vastus internus muscle of each thigh reacts normally to induced currents. The pupils are very small, and equal in size. They contract during accommodation, but not to light.

For seventeen years the patient has been subject to attacks of vomiting, which at first would occur about once in a year, and last from ten to twelve days. For the last two years these attacks have been much more frequent, and she will have one (lasting a week or more) every three or four weeks. There is nausea, vomiting and cramp-like pain in the epigastrium, which makes her "twist about." She brings up a great deal of flatus, and there is much empty retching. The presence or absence of food in the stomach appears to have no influence on the vomiting. Her left lower extremity is shorter than the right, measuring thirty-one inches from the anterior superior iliac spine to the inner malleolus, against thirty-two inches and a half, the measurement on the right side. The left trochanter major is four inches below the anterior superior iliac spine, and half an inch outside a line drawn vertically downwards from that spine. The right trochanter, on the other hand, is four inches outside a corresponding line. The right femur rotates in an arc of a circle of which the head of the bone is the centre, whereas

the left rotates around its own shaft as a centre. The left leg can be drawn down nearly two inches. There is no sign of the head of the left femur being present. The patient can flex, extend, and rotate her left lower extremity fairly well, although with less power than the right. Both thighs, five inches above the patella, measure fifteen inches and a quarter in circumference. The left

FIG. 20.



lower extremity can be rotated and moved into perfectly abnormal positions, as shown in the accompanying wood-cut, taken from a photograph, for which, as for that of Fig. 18, I am indebted to Dr Gowers. She cannot stand without help, and then, on closing her eyes, she reels.

The following case was under my care seven years ago, affected with tabes and exhibiting joint affection with gastric crises :

John C—, æt. 43, a butler, had suffered for five years from shooting pains, chiefly in the calves, occurring at intervals of from six to eight days, and lasting several hours. When he applied at the hospital in February, 1875, the pains had gradually become much less severe and frequent. There was divergence of the optic axes. His pupils were contracted, his gait was ataxic, and he could not stand in the dark with his eyes shut. There was occasional imperfect action of the bladder. He described having had violent attacks of vomiting, occurring perhaps two or three times a year, and lasting a day or two. But besides this he had had a gnawing and sinking sensation at the pit of the stomach almost daily, with sickness and aversion from food. This was felt especially as regards anything which required mastication. He also complained of a frequent feeling as though he wanted to evacuate his bowels. Sometimes, he said, he felt as though there were a cannon-ball in his stomach. I found both his knee-joints much enlarged, and he told me that they had been twice as big. A month later his right knee-joint had still further enlarged, and evidently contained fluid. Not long after this I lost sight of the patient.

Through the kindness of Mr Herbert Page I had the opportunity of examining with him a patient who was affected with joint affection in both feet, associated with tabes dorsalis.* The patient was a man, aged thirty, whose right leg and foot commenced to swell in October, 1880, with some pain which soon passed off, the swelling remaining. When seen the cuboid, scaphoid, three cuneiform, and the metatarsal bones seemed enlarged, and were freely movable on one another in any direction. Whilst under observation the left foot became affected in a similar way to the right, very rapidly and without pain. It seemed, on inquiry, that two years ago this young man had an attack of vomiting, which lasted on and off for

* See "Report on the Congress Museum," 'Transactions of the International Medical Congress,' London, vol. i, p. 124.

three months. In the morning, when he went downstairs, he would begin vomiting before he had taken food. Then he would drink a cup of tea and eat an egg and a slice of bread and butter. He says that he brought up the tea but not the solid food. The vomiting would continue till the middle of the day. He often vomited as much as a dozen times, and did not pass a day without sickness for three months at least. There was pain in the stomach and between the shoulders. Much wind was expelled. He became so weak and bad that he could do no work for six months. The vomiting left off suddenly after remedies had proved unsuccessful. He has never drank spirits. In the beginning of his gastric attack he used to become dreadfully hungry, and would eat much solid meat towards night. But after three or four weeks he lost his appetite and became very thin. The gentleman who attended him during this illness tells me that he had no suspicion of the cause of the vomiting. This man has had shooting pains in his legs; the knee phenomenon is absent; his pupils react in accommodation, but not to light. He has never had any ataxy of gait.

In the museum of the Congress two cases of ataxic arthropathy were exhibited by my friend Mr Charles Macnamara, under whose care they had been admitted into the Westminster Hospital; one as a case of rheumatic arthritis, and the other as an example of probable malignant disease of the right hip or upper part of the femur. These were characteristic cases of the affection, but neither of them suffered from gastric crises.

A male patient, æt. 34, was shown by Mr Keetley at the Clinical Society* on October 14th, 1881, affected with tabetic arthropathy involving both hip-joints. Although he had not suffered from gastric crises, it appeared that he was subject to what might be described as "intestinal crises"—attacks of diarrhœa, occurring fortnightly, for long periods at a time, as well as heartburn.

* 'Lancet,' October 22nd, 1881.

Dr Seeligmüller relates* particulars of a case of arthropathy of the left knee-joint in a woman, æt. 48, affected with all the symptoms of tabes, and especially subject to typical gastric crises. The patient died. There is no record of any microscopical examination of the cord.

I lately saw in private practice a female patient presenting all the symptoms of tabes, who had recently suffered from paroxysms of retching, with pain in the chest, lasting a week or two at a time. Her right ankle-joint was swollen and "gave" under her, the left acromio-clavicular articulation was also swollen, and the corresponding joint on the right side presented a scar, the site of an opening where some fluid had been discharged. This lady's father, I should say, used to have characteristic lightning pains which were called "gout."

A case of great interest in reference to this question recently occurred in the practice of my colleague, Dr Ormerod. The patient was a man, æt. 47, who presented the characteristic symptoms of tabes including gastric crises. Search was made for affection of joints but without success. Two months later the man was attacked with a large painless swelling of the right knee-joint.†

Vulpian has lately‡ reported a case of tabetic arthropathy of the knee-joint and hip, in which there were laryngeal crises and diarrhœa.

A patient of mine, Frederick C—, at present in the hospital, shows symptoms which have an interesting bearing upon the question. He is a typical ataxic, and has besides suffered for a year and a half from attacks of vomiting, recurring every six or eight weeks, and lasting perhaps two or three days. There is great nausea and retching, which do not appear to be influenced by food. Now, this man, although he is free from any notable

* "Ueber Gelenksaffectionen bei Tabes dorsalis," von Dr. Seeligmüller in Halle, 'Neurolog. Centralblatt,' January 19th, 1882.

† 'St. Bartholomew's Hospital Reports,' vol. xvii.

‡ 'Revue de Médecine,' February 10, 1882.

swelling of the joints, complains that his thighs, knees and ankle-joints are all loose. He is very ataxic, and can only attempt to walk between two persons to support him. When he tries, his feet turn under him owing to the lax state of the ligaments of his ankle-joints.

I find, on reference to my notes of a tabetic patient, Wm. W— (taken in 1873), that he complained to me of his hip-joints being so loose that he could scarcely sit. He was also liable to attacks of epigastric pain with such feeling of sickness that for days together he had difficulty in taking any food, but had not had vomiting.

It is right to say that some authorities (Westphal, Althaus, Broadbent, Arnozan) object that the occurrence of gastric crises is so common in tabes as to render the coincidence with joint affection of but little value. This is not, however, the experience of M. Bernhardt,* who describes having met with but three examples of *crises gastriques* out of fifty-eight cases of tabes. In one of these three there was affection of the knee-joint. And my own experience shows also the rarity of this symptom. Out of 100 cases of tabes which I have lately tabulated there are but ten which have suffered from gastric crises of typical character. So also as regards the peculiar affection of the bones, I am disposed to think that this occurs in only a very small proportion of cases of tabes. It is impossible, however, for the following reason, to say what that proportion really is.

In conversation upon the subject with Prof. Charcot, a few years ago, I told him that we rarely met with cases of tabetic arthropathy in England. He said: "You will find them in the workhouse infirmaries and in the surgical wards of hospitals." He was right. Two of my eight cases I discovered in workhouse infirmaries. Another was very nearly having the operation of excision of the knee-joint performed in a surgical ward of a hospital; two others, who had been inmates of surgical

* 'Zur Pathologie der Tabes dorsalis,' Berlin, 1881.

wards, had each been exhibited to candidates for a diploma at one of the examining boards as examples of rheumatoid arthritis. It is not, therefore, in the medical wards of hospitals, nor at a special hospital for paralysis, that one is likely to find many examples of these conditions. In most of those which I have published, the nature of the underlying disease had never been suspected.

Let us see in what proportion of cases of tabetic arthropathy gastric symptoms have been noted. If I include the patient S— and Mr Keetley's case at the Clinical Society, in both of whom the action of the bowels rather than of the stomach was disordered,* I can number twenty-four with this character. When the small proportion of cases showing gastric attacks, out of a range of ordinary cases of tabes, is compared with the frequency of their association with joint affection, the contrast appears very striking. The cases of tabetic arthropathy that I can find recorded (including nine of my own) number forty-eight. In twenty-four of these there were gastric crises. It is possible, indeed probable, that I may be overlooking some examples in which the gastric attacks were absent, and I do not wish, therefore, that these figures should be taken as representing more than an approximation to the facts, but it is evident that there is room for a large amount of error, and the frequency of association would yet remain a very remarkable circumstance. Further observation, it appears to me, has only tended to lend force to my original suggestion that lesion of a structure adjacent to the nuclei of the vagus may be found to explain the osseous affection in tabes.

Whether this may be in the so-called vaso-motor centre, or whether physiology may yet have to discover in the medulla oblongata a centre more directly concerned in the nutrition of the osseous skeleton, it would be entirely premature to discuss. One cannot help feeling, however, that, were the existence of such a centre to

* For reasons why this symptom may be considered as probably due, like the gastric crises, to irritation of the vagus. See Lecture IX.

be rendered probable, we might find in it a valuable clue by which to explain the combination of articular symptoms in acute rheumatism with occasional tendency to high temperature, and still more with the cardiac complications of such frequent occurrence in that disease, an association for which hitherto no hypothesis has reasonably accounted. Light might also be thrown incidentally on the nature of arthritis deformans.

LECTURE XV

ON CERTAIN LITTLE-RECOGNISED PHASES OF TABES DORSALIS

In his description of the symptoms of tabes dorsalis, Romberg gives the greatest prominence to the insecurity of gait. "The patient," he writes, "attempts to improve it by making a greater effort of the will; as he does not feel the tread to be firm, he puts down his heels with greater force. From the commencement of the disease the individual keeps his eyes on his feet to prevent his movements from becoming still more unsteady. If he is ordered to close his eyes, while in the erect position, he at once commences to totter and swing from side to side; the insecurity of his gait also exhibits itself more in the dark. . . . . In no case have I found it (this symptom) wanting." *

So also Duchenne (de Boulogne), in the classical description which he gave of the same disease, under the name of progressive locomotor ataxy, allots the foremost place to this motor trouble. "In the first rank," he writes, "I place the troubles of coordination of movement, contrasting with the, so to speak, latent integrity of the muscular force, because they constitute the fundamental character of progressive locomotor ataxy." †

The picture which these observers, especially Duchenne, have given of a disease in which this symptom holds a prominent place, is widely known, and there is no occasion

* Romberg's 'Diseases of the Nervous System,' Sydenham Society, translation by Dr. Sieveking, 1853, vol. ii, p. 396.

† 'De l'Electrisation localisée,' Paris, 3me édition, 1872, p. 623.

to reproduce it here. I merely wish now to point out in reference to those descriptions that the ataxy of gait has been so strongly insisted upon by these and other writers, that the symptom has of necessity been hitherto regarded as the dominating one of the disease. There can be but little question that the idea never enters into the mind of many, that a patient may be the subject of this disease, unless the incoordination of movement be tolerably well marked.

It appears to me that, in the present condition of the question of tabes dorsalis, we are not quite warranted in saying of a case which does not show any ataxy that it has not yet arrived at a certain "stage." Our grounds for dividing the course of this disease into different stages, appear to become less and less secure as we observe that various parts of the sensory nervous system may be involved in degrees of intensity which bear no fixed relation to any chronological order. Although the point can only be determined after a considerable lapse of time, it seems most probable that ataxy of gait is no more a necessary symptom than optic atrophy, although it is a much more frequent one. Whilst allowing, then, this highly characteristic symptom when present to retain all its former value for diagnostic purposes, it is well to recognise the fact that not only may the incoordination of movement be entirely wanting, but some other symptom which may not ordinarily be at all a striking one, may come to occupy the most prominent place. In these circumstances it is apt to absorb the attention of the observer to such a degree that, without some considerable care, the general disorder, of which it is only a symptom, is masked, and the symptom itself is liable to be referred to some entirely different pathological condition.

Let me repeat, what I have already said in previous lectures, that in my judgment Westphal's test, the absence of the knee-phenomenon, provided that this is associated with fair voluntary power, and idio-muscular contraction of the vastus internus muscle (a provision

which Erb, as well as myself, has insisted upon), is the most constant and important symptom of tabes dorsalis. There is one pathological condition, however, which may possibly give rise to some doubt; indeed, I have met with an instance in point. In *diphtheritic paralysis* there is often a tottering gait, accompanied by numbness in the extremities. If there should be, as sometimes happens, no difficulty in deglutition, and no failure of the power of accommodating the eyes, a strong superficial resemblance to a case of tabes dorsalis is presented. In such a case the patellar tendon-reflex will, in all probability, be absent, and the vastus internus muscle may contract as freely as in health to direct percussion. The acuteness of the symptoms, however, the absence of characteristic lightning-pains, and the history of preceding sore throat, should be sufficient to distinguish this condition from tabes.

When characteristic lightning-pains are present, Westphal's symptom (with the provisions mentioned) gives, I think, evidence of the presence of tabes, which is, as nearly as possible, complete.

I do not propose to dwell here upon the other side of the question—the cases of tabes dorsalis with well-preserved, or even exaggerated knee-phenomenon, of which we occasionally witness examples. The explanation of these presents no difficulty and I have already referred to it. Suffice it to say, that the absence of the knee phenomenon in the circumstances described must either be attributable to the presence of tabes, or be owing to a natural peculiarity, and examples of this are so exceedingly rare that they may be practically disregarded. I am prepared, indeed, from something which I lately saw, to meet with the absence of knee-phenomenon as possibly representing the only symptom of tabes in a certain case. This test of Westphal's is so delicate, that the accompaniment of any one of the ordinary symptoms of tabes along with it, ought, in my opinion, to enable us, with very little hesitation, to relegate the case in which this association is exhibited to the class of tabes dorsalis.

Let me now refer to some examples in illustration of the statement that a comparatively rare symptom of tabes may assume such proportions as to stand prominently forward, and easily give rise to errors of diagnosis. The first to be mentioned are cases in which the *crises gastriques* dominated other symptoms.

A gentleman, aged fifty-two, was brought to me by his medical attendant on June 28th, 1880, suffering from emaciation and neuralgia in the head. It appeared that for fourteen years he had been subject to so-called "rheumatic pains" of a flying character, and besides, had suffered from what was called sciatica five years before, and sudden shooting pains between the ribs the previous year. For about fifteen years he had been liable to occasional attacks of violent vomiting, accompanied by pain in the stomach, lasting from one to four days, and recurring, perhaps, once in six weeks or two months. I examined carefully for any epigastric tumour but could find none. The urine contained no albumen. The patient's breathing was laboured, and there were some not very positively marked signs of a cavity in his right lung. His pupils were contracted to less than the size of a pin's head. They were insensitive to light, but contracted during accommodation. The patellar tendon-reflex was quite absent in both legs. The vastus internus muscle showed excess of irritability to percussion. He had no ataxy of gait, but, in reply to my inquiry, informed me that he could not get on well in the dark.

I lately learned that this patient died two months after I had seen him, and that his case was diagnosed by a very accomplished physician, who examined him subsequently to me, as one of malignant disease of the stomach. No autopsy had been made.

This man was certainly affected with tabes dorsalis, as evidenced by the absence of knee phenomenon, along with well-preserved, and indeed abnormal, contractility of the muscular fibres of the quadriceps extensor, the presence of the Argyll-Robertson pupil, the history of shooting

pains, and of some slight difficulty in the dark. The attacks of vomiting as described were precisely similar to the *crises gastriques* of Charcot, and they had been occurring at intervals of a few weeks for fifteen years. Even without the evidence on the negative side afforded by the fact of my having failed to find any tumour (and, thanks to his emaciation, examination was easy) this history of such long-continued and periodical vomiting is not consistent with malignant disease, but, on the other hand, is precisely what is to be expected in the gastric crises of tabes dorsalis. I have no doubt whatever that this was the explanation of his illness.

In a case which I have already related, that of Henry H—,* where the functional disorder of the stomach was marked to a very extraordinary extent, the gastric symptoms entirely dominated all others, and no suspicion was entertained of the general disorder with which he was affected.

In another of the joint cases it was not till six or seven years after the commencement of the gastric symptoms that the patient began to suffer from incoordination of movement. Another woman, for twelve years before she showed any ataxy of gait, had suffered from attacks of vomiting, which at first would occur about once in a year, and last from ten to twelve days. This patient also is the subject of the joint affection of Charcot, and presents every symptom of tabes.

These cases are sufficient to show that gastric attacks, often of an extremely severe and obstinate character, may occur in persons who, although they show at the time no sign of incoordination of movement, are most certainly examples of tabes dorsalis. There is reason to believe that in none of these cases the nature of the general disorder underlying the gastric attacks had been suspected.

I would suggest that many cases of so-called "gout in the stomach" would be found, if examined by the light of our present knowledge, to be examples of tabes dorsalis

* Lecture XIV.

with gastric crises. The lightning pains, which would probably be associated with the sickness and epigastric pain, would be likely in the minds of many to make the diagnosis of "gout in the stomach" complete.

Looking back into the past, I can call to mind cases in which the diagnosis I had made of malignant disease of the stomach was not confirmed by the sequel—long intervals of exemption from the symptoms taking place—which I now think were probably examples of tabes with the gastric crises predominant.

These symptoms may easily give rise also to suspicion of intestinal obstruction, and may possibly also be mistaken for the attacks of vomiting which are apt to occur in uræmia from Bright's disease.

Duchenne, Topinard, and others have recorded examples of tabes in which deafness occurred, but it is to Pierret that we owe the observation that the auditory nerve is really very often involved. Since his paper appeared ('Sur les Symptômes Céphaliques du Tabes Dorsalis,' Paris, 1876) I have looked more narrowly for the symptom and noted its occurrence in a considerable number of cases. My colleague, Dr Ormerod, has given me notes of thirteen cases of tabes observed by him, in five of which deafness was more or less strongly marked.

I would suggest that in cases of "nervous deafness," as in those of optic atrophy, the examination of the state of the patellar tendon-reflex should not be omitted. It is quite probable that we may discover in certain instances absence of the knee-phenomenon, and thus be led to place the case in the same category with those of amaurosis from sclerosal atrophy.

It has occurred to me to see one patient in whom the presence of a stone in the bladder led me to examine the state of the patellar tendon-reflex. It was found to be absent, and inquiry elicited the fact that the patient suffered from most characteristic lightning pains. I would suggest that tabes may be an occasional source of origin of a calculus, of the kind which Sir Henry Thompson

calls "local," in contradistinction to the calculus (as, *e.g.* uric acid) which is of "constitutional" origin. In many cases of tabes, at an advanced stage, there is actual paralysis of the bladder. But a certain amount of difficulty in expelling urine may be a very early symptom, dependent probably upon vesical anæsthesia and absence of normal reflex. It will often be found, on inquiry, that the patient never has the natural feeling of wanting to pass urine, and does not know when it is being voided, or when he has finished. In such circumstances, unless he contracts the habit of passing it at regular intervals, there is always the possibility of cystitis occurring from urine getting retained; and in the mucus of the bladder, in these circumstances, a phosphatic calculus may easily be formed. It is worth while remembering that a bladder trouble of this kind may be as prominent and absorbing a symptom as either the gastric crises or the optic atrophy; and if ataxy of gait be absent, the real nature of the trouble may easily be overlooked.* I have but little doubt that not a few cases of atony of the bladder for which the surgeon is consulted are examples of tabes with the bladder trouble predominating.

Laryngeal disturbances may be the dominant symptom. In April, 1878, Dr Felix Semon showed a man K—, aged thirty-two, to the Clinical Society of London,† in whom bilateral paralysis of the posterior crico-arytænoid muscles preceded by nearly two years objective signs of tabes dorsalis, although momentary pains were present. Thanks to Dr Semon's courtesy I had the advantage of examining the patient in December last. It is a most interesting circumstance (in reference to what I suppose to be the cause of gastric crises) that this patient had been

* At the International Medical Congress, held in London, 1881, Sir William Gull, President of the Section in which the substance of this lecture was read as a paper, expressed his opinion that the position here taken as regards stone in the bladder was sound, and remarked that the case of the late Emperor of the French was one which, according to his belief, went in support of my view.

† 'Clin. Soc. Trans.,' vol. xi, p. 141.

attacked three or four months previously with fearful pain in the stomach, accompanied by retching and vomiting, which lasted some hours, and had since recurred on four occasions.

In the paper mentioned, Dr Semon referred also to a case of so-called *functional paralysis* of the same muscles lasting for two months only. The patient had previously suffered from transitory paralysis of eye-muscles. Was not this a case of tabes? It would be well to test the knee-phenomenon in all cases of paralysis of laryngeal muscles.

Pains may affect the district of the lower intercostal nerves so severely as to direct the attention exclusively to the abdominal region, and thus lead the observer very wide of the mark indeed.

I was asked by a medical friend a short time since to see a male patient, about forty-six years of age, to whom he had been called the week preceding on account of what was called "colic." After being purged he recovered. A few days afterwards he was attacked with violent pain in the flanks, which was supposed to be due to muscular rheumatism. The pain then got down to the pelvis, and was so bad about the hip-joints that rheumatic fever was apprehended. It was noticed, however, that the pulse and temperature were unaltered.

I found the patient in bed. The skin over the hip-joints was intensely hyperæsthetic, so that he shrank from the slightest touch with the finger. On the other hand, firmer pressure occasioned no distress. The pains in the flanks were described as of a grasping character. On inquiry as to pains in others quarters he informed me that he had suffered for six or seven years from "rheumatics" in the legs. For two years past he had had probably every week a dozen or more attacks of pain in the legs as if the skin were being "gnawed off or gradually torn off." The pain came on suddenly and very sharp, left off as suddenly, and recurred within a couple of minutes as sharp as before. Fifteen years ago he was laid up for seven

months with "rheumatic fever." He had never had vomiting nor diplopia. The bladder was somewhat torpid.

The patellar tendon-reflex was absent on each side, the vastus internus responding readily to percussion. The pupils did not contract to light, but contracted during accommodation. Cremasteric reflex was present and sole-reflex perfect. There was no apparent anæsthesia.

I need not say that the case resolved itself into one of tabes.

Hemiplegia may mask the symptoms of tabes.

The following patient (notes of whose case were taken by Dr Beevor) is now in the hospital:

Henry H—, æt. 48, single, a gilder of picture frames; has drunk a good deal, and had a chancre at twenty-two years old, not followed by rash or sore throat. He had felt some numbness around his waist and in the legs for about a fortnight, when, on April 8th, 1881, whilst out walking, after his work was over, he was seized with a sudden weakness and numbness in the left leg. Then he felt a pain between the thumb and forefinger, and a little numbness and pins and needles in the left hand, with some weakness in the left arm. He continued much the same till the middle of the next day, when his leg got much worse, and in the evening the arm gradually became powerless. By April 10th he had completely lost power in the left arm and leg. The consciousness had never been impaired. On April 12th the left side of his face began to feel stiff, and he could not move it so well as the right. The sensibility of the skin was never lost. On admission the left side of the mouth was observed to be moved less than the right, and the left eye could not be closed so tightly as the right. The tongue was protruded slightly to the left. He was entirely unable to move the left arm, and could not shrug the left shoulder. There was some stiffness in the joints of the left arm, but no pain was caused by moving them, except a very little in the shoulders when the arm was abducted from

the body, and the hand placed on the top of the head. Both legs could be moved freely about, but the right much more strongly than the left, in which there was no power to invert or evert the ankle-joint. There was no stiffness in the left leg.

At the present time—June, 1881—the patient can just stand without help. With the eyes closed he reels and threatens to fall. When he walks with help it is noticed that, whilst the left leg is somewhat dragged, the right is inclined to be thrown about. There is no wasting in his limbs. The knee-phenomenon is absent on both sides. There is no ankle-clonus. The plantar reflex is obtained on either side, on the right rather better than on the left. The cremasteric reflex is not obtained on either side. The abdominal and epigastric reflexes are obtained equally well on either side. No definite loss of sensibility can be made out, but there may be a slight deficiency in the left foot. There is no affection of sight or hearing. No diplopia or hemiopia. The heart's apex cannot be felt; there is occasionally an intermission of a beat. There is no murmur, but the sounds are not clear. The pulse is rather feeble, the brachial artery is not tortuous; urine contains no albumen.

The pupils are of moderate and equal size; they do not contract to light, but contract during accommodation. On inquiry, it seems that the patient has suffered for about ten or twelve years from "rheumatic pains," coming and going in an instant, and gone before he could cry out. Five years ago he had diplopia, which lasted about a fortnight. He has had no difficulty in walking about a room if he knew the locality; but once or twice he has lost confidence in the dark when he did not know his way about. He never had numbness in the legs, nor loss of sensibility; he has never shown any peculiarity of gait which has been remarked, to his knowledge, by others.

There is a most interesting circumstance to be noted in this case; the man's perfectly helpless left arm shows some rigidity and contraction, as is so often seen as a

result of secondary changes in hemiplegia. In such circumstances, as is well known, we confidently expect to find more or less marked exaggeration of the reflex following blows upon the wrist, as well as upon the patellar tendon. In this case there is an entire absence of any reflex at the wrist, as well as at the knee. The condition peculiar to tabes apparently overpowers that which is apt to occur as a result of hemiplegia.

In the history of some other ataxic patients I have come across an account of an hemiplegic attack:

Charles B—, æt. 41, applied at the hospital in December, 1878, with marked ataxia of gait, inability to stand with eyes shut, lightning pains, anæsthesia of legs and arms, diplopia, absence of knee-phenomenon. Sixteen years previously he had suffered from right hemiplegia.

Matilda H—, a typical case of locomotor ataxy, with arthropathy when forty years of age. Five years after she began to get lightning pains she suffered from a slight attack of left hemiplegia, from which she recovered in a week.

S— had a right hemiplegia four years before symptoms of ataxy were noted; although from the history, as I learned subsequently, the hemiplegia occurred whilst the patient was affected with symptoms of tabes.

Let me pass on now to another condition:

A gentleman, æt. 51, whilst standing up in church, felt a sudden tremor in his legs, which gave way, and he dropped into his seat. This sudden giving way of the legs had been repeated on many occasions, and it was for this, and this alone, that I was consulted. There was nothing to be noticed about his gait. Examination showed that he had no patellar tendon-reflex, and inquiry elicited a history of so-called "rheumatic" pains in one of his elbows. He also described a sensation in his legs as though cold water was being gently poured over them from a water-pot, and some numbness of the left hand.

The *sudden giving way of the legs*, so that in many cases the patient actually drops to the ground, is a sym-

ptom which has not, so far as I know, been previously described amongst those characterising tabes. It is, however, one of very common occurrence. Most frequently it happens in connection with a particularly sharp dart of lightning pain in the lower extremity. But it will often take place without the accompaniment of pain. I do not refer to the insecurity and tendency to fall, which is very often seen in an ataxic who is struggling to preserve his balance, amid the wild throwing about of his limbs. The symptom of a perfectly sudden giving way of the legs often occurs in persons who have no ataxy of gait, and quite independent of a dart of pain.

A very intelligent patient, who is blind from sclerosal atrophy, and who has suffered besides, for twenty years, from typical bouts of lightning pains, but is completely free from ataxy, tells me that he has often had this symptom of sudden giving way of the legs. In him it is always associated with pain. He is sure that it is not a spasmodic contraction, but a sudden and temporary failure of muscular power, in fact, a very transitory paralysis.

A letter-carrier, who came to me in 1868, described the first symptoms of his illness thus:—He went for a fortnight's holiday into the country, being then quite well. A week or two after his return he found his knees giving way suddenly under him when running up and down the steps of houses. Soon he found himself tottering, although he could walk fast. He could not run. At that time he had no pains in his legs. He gradually got worse, and walked so much like a drunken person that he feared he should be taken for one. When I saw him he was a typical example of tabes.

A letter-sorter, whom I saw in 1871, told me this:—"One day, whilst walking through London, he was suddenly seized with a kind of loss of power in the legs, and would have fallen but that he laid hold of a doorway. The right leg especially seemed to give way."

This sudden failure or giving way of the knees would appear to belong to the same category as the passing

diplopia from failure of a muscle of the eye. Pierret* refers the transitory paralysis of muscles to irritation of the posterior roots. The suggestion appears to me to be a very valuable one.

On the other hand, paraplegic symptoms may be so strongly marked as to render the diagnosis obscure. A striking example of this has recently occurred in my practice.

X—, æt. 44, tall, and not very muscular, had always been delicate, but fond of walking, and accustomed to walk a great deal. In April, 1881, he had a typical Hunterian chancre, which was open for a month, and was followed by a most profuse roseola, with which he was covered when I saw him on July 1st. It seemed that, although there had been complaints of failing power in walking, sense of weariness, and pain in the sacrum and loins for several months, it was not until the attack of syphilis that the marked loss of power occurred in the lower extremities. From that time, however, there was a rapidly increasing paresis of the lower extremities, and it was for this that I was consulted. I found him sitting up, able to move about on his legs with difficulty if aided by two sticks or friendly arms, but not without. There was no throwing out of the feet, which, on the contrary, were moved in a laboured fashion, suggestive of paraplegia. The power of lifting either knee against the pressure of the hand was small. There was no muscular atrophy or loss of cutaneous sensibility. The action of the bladder was imperfect and delayed, and he did not feel the natural desire to pass urine. His feet were always cold. He had suffered pains in the lower extremities of a neuralgic or rheumatic character.

The superficial reflexes of the foot-sole, cremasteric, abdominal, and epigastric regions were lively on each side. His pupils contracted well to light, and the ophthalmoscope showed no change in the disc. The patellar tendon-reflex was absent on each side. Contraction of

* Loc. cit.

the vastus internus to percussion and faradaic currents was perfect. A fortnight later there was definite diplopia, the false image being above and to the side of the other.

In this case a rapidly progressive paraplegia following syphilis gave a superficial resemblance to acute myelitis, which was, however, negatived by the state of the reflexes. The occurrence of diplopia, along with the absent knee-phenomenon (the normal reaction of the vastus internus being remembered), leaves no doubt, in my judgment, that this case of apparent paraplegia is really one of tabes.

I saw, not long since, by the courtesy of Mr de Watteville, a man, forty years of age, who had suffered for about twelve years from an occasional feeling as though he might fall down; as if, whilst walking, he needed a support. He had no objective giddiness in the ordinary sense. He had a headache, but he could not tell whether his want of power was in the brain or limbs. This feeling recurred many times a day and grew worse. He saw numerous physicians of eminence, by most of whom the condition was pronounced to be cerebral. It was not until Mr de Watteville tested his patellar tendon and found the reflex wanting that the case stood out as one of tabes. Only after these sensations had existed for twelve years did the patient begin to suffer from lightning pains, and it was still later that a slight ataxy of gait betrayed itself. I found that his vastus internus muscle was over-irritable to percussion, and that his pupils scarcely reacted to light, and reacted in accommodation. There was delay in the reception of painful impressions.

LECTURE XVI

CASES OF PROLONGED SOMNOLENCE IN CEREBRAL SYPHILIS

THERE is a kind of stupor which is a noteworthy feature in many cases of intracranial syphilis. It was particularly marked in a man named B—, who is now attending the hospital as an out-patient, and was an inmate last year. The condition is sometimes much prolonged, extending, it may be, over several weeks, during which time it appears as though life were being carried on almost automatically. On recovery, however, should that take place, it is found that the patient has been conscious of his surroundings to an extent which is somewhat surprising. John B—, æt. forty-one, was admitted into the hospital after having attended as an out-patient during two months. His illness had been of this kind: after very bad pain in the head for a week, he had a sudden attack of right hemiplegia, without loss of consciousness. He was gradually recovering from this, when he came to the hospital two months afterwards, and there then remained little to be seen but a slight traction of the mouth towards the left side. There was also paralysis of the right external rectus muscle, of the duration of which we could get no account. His limbs were of equal strength. He had never lost, and still suffered greatly from pain in his head. Whilst attending as an out-patient he took thirty grains of iodide of potassium daily for two months, but at the end of that time, when he was admitted into hospital, his head was as bad as ever, and he looked ill. I should say that not only was there a clear history of

syphilis in his case, but one of his legs presented a scar which was very characteristic of old specific ulceration.

When this man came into the hospital it was noted by Mr Broster, our resident medical officer, that his mental power seemed to be much dulled, his answers slow, and his manner apathetic. He would sleep nearly the whole day, and if he occasionally roused himself, would take a few steps in a dazed condition, and then, as likely as not, try to get into some other patient's bed. He volunteered no remark. Nine days afterwards the story was much the same. "He rarely, if ever, speaks to any one, but sits silent or lies down the whole day. He understands apparently what is said to him, and does what he is told, but any reply which he makes to a question is slow, hesitating, and often not to the purpose. He sleeps very much. He was wandering in a vague way to the door this morning, and on being asked his motive, said, 'I should like to go home, but I don't know where I live.'"

Ten days after this note had been made he had a sudden seizure, in which he is thought to have partly lost consciousness, and became for a second time paralysed in his right limbs, and coincidently the left external rectus muscle ceased to act. After this attack the patient was unable to stand; usually very drowsy, he was sometimes restless, complaining (only when questioned) of pain in the head. He was ordered a grain of blue pill, which he took twice a day for six days, but this peculiar state of stupor growing manifestly worse, I directed that he should be rubbed daily with mercurial ointment. A week later it is noted that his pulse is small and weak, the extremities cold, the respiration slow and stertorous. He is conscious, but has no power to do anything, and will not take nourishment. His urine and fæces are passed under him in the bed. For the next two days his condition appeared to be one of imminent danger, and his friends were summoned. Then some dysenteric symptoms, due, doubtless, to the mercury, showed themselves, and almost simultaneously his mind rather rapidly cleared; he

got up, walked without much difficulty, gradually recovering the lost power in his right limbs. The left external rectus muscle, too, again began to act. Ten days later Mr Broster notes :—"He is up and about, taking a lively interest in everything, and as quick at repartee as any one in the ward. He remembers all that was said to him at a time when he was so restless as to require restraint in bed, and says that he was under the impression that some one was trying to murder him." The state of stupor had lasted upwards of five weeks. When the patient was discharged, some three or four weeks later, he had recovered completely the use of his limbs, was in good general health, with clear intellect, and about a month later the paralysis of the right external rectus muscle cleared up also. He told us the other day that he could walk five miles. He is still kept under observation as an out-patient, and a slight mercurial influence has been continued.

Now, when I said that this kind of stupor was a noteworthy feature in many cases of intracranial syphilis, I did not mean to imply that the condition was of *itself* pathognomonic of syphilis. This is evidently not the case. We often see mental obfuscation of this kind in an old man or woman who has had an apoplectic seizure, especially when this has been due to the thrombosing of atheromatous arteries. But in these latter cases two things must be remembered : in the first place the patient is old, and in the next place more often than not he does not recover. What is peculiar to the syphilitic cases is that the patient is usually young, and that he very frequently recovers.

John B— is only 41. His radial and temporal arteries are elastic, he has no cardiac disease, there is no albumen in his urine. He is not only, therefore, young in years, but in his tissues also, except, however, in one most important quarter—the arteries of his brain. The history goes to show that there is thickening of some of his cerebral arteries, and that this is of syphilitic origin. I

suppose that each of his hemiplegic attacks was the result of a limited brain softening consequent upon thrombosis of an artery, the channel of which had been much narrowed by inflammation and thickening of its walls. Now the question may be asked, How do you know that the hemiplegia was not due to the pressure of a gummatous tumour upon the motor tract or upon the surface of a cerebral hemisphere, instead of to thrombotic softening? Such a question is sometimes difficult, it may be impossible, to be answered. But the present case admits, I think, of but little doubt. The man had a sudden seizure of paralysis—a hemiplegia without loss of consciousness, at a time when he was, so to speak, saturated with iodine.

Whatever may have been the cause of his first attack of hemiplegia, which occurred two months before he came here, it is scarcely conceivable that a gumma would not only grow, but attain a destructive size, in a patient taking thirty grains of iodide daily. Added to which, the sudden mode of seizure is in favour of softening from thrombosis, and opposed to the hypothesis of its being occasioned by a growth. Nor did the ophthalmoscope give evidence of the presence of a tumour. There was no optic neuritis. We may take it, then, as tolerably certain that this man's hemiplegic seizure was the result of arterial disease. The symptoms seemed to show (by exclusion) that the paralysis of the sixth nerve was due to a separate lesion, but upon this we need not now dwell.

It is the occurrence, then, of long-continued stupor with symptoms of thrombosis in a *young* man—in a man who, at least, shows no sign of agedness—and his eventual recovery, which are so significant of syphilis. Moreover, such cases are not at all uncommon. I have reason to think that they very often represent the "brain fever" and the "sunstroke" of the public. The condition becomes even more striking when it occurs in a person of still younger age. Richard S— has come here at my request, a year after his discharge from the hospital, in

order that we may learn how he has gone on. Only thirty-one years of age, he had been brought to the hospital in September, 1877, in a cab, just able to stand. He had left hemiplegia, with partial ptosis and paralysis of the superior rectus muscle of the right eye. This was the manifest condition on his admission, but his mind was so dull that but few particulars could be obtained from him till some time afterwards. He had a dazed look, and could not take in an idea until he had been spoken to many times. He did not initiate any conversation, but in reply to questions said that he had a very bad pain in the head and felt weak and sleepy. The pain was in the back part of the right side of the head and over the left eye. Having gone to bed, he lay there for a month, silent and somnolent. He took thirty, and later sixty grains of iodide daily. About four weeks after his admission he had become somewhat less stupid and heavy, but he still complained of much pain in the head. He was now rubbed regularly with mercurial ointment, the iodide being discontinued. When the gums became tender, which did not take place for a month, it is noted that his mind had cleared up, he had nearly lost the pain, his limbs had become stronger, and in the course of three weeks he left the hospital, perfectly recovered from his hemiplegia. When he came to us at my request the other day, he told us that he had remained perfectly well, was able to walk, and in September last had won the prize in a rowing match on the river.

Let me here refer briefly to the previous history of this patient, which embraced the following important circumstances:—A chancre eleven years previously, which was followed by sore throat and skin eruption. Early in 1876 severe pains in the left lower extremity, styled and treated as "rheumatic," but accompanied by paralysis, cutaneous anæsthesia, and wasting, so that he was left, after six months' illness, with a thigh permanently some three inches and a half less in circumference than the right, and the skin of this limb anæsthetic. He had returned

to his work (with a lame leg) not more than three months when he was taken with pain across the forehead and vomiting, which occurred several times a day, irrespective of food. The vomiting, he says, was ascribed by his medical attendant to "biliousness," but it went on for three or four months, accompanied by the constant pain. It was no doubt of cerebral origin. Then he was seized with ptosis of the right eyelid, and a month later with a gradually increasing left hemiplegia, for which he was admitted here. It should be added that he presented no signs of heart or kidney disease. The ophthalmoscopic evidence was not decisive. There was certainly no optic neuritis at the time of examination, but the appearance of the fundus oculi led me to think that there might have been some at a previous date.

The occurrence of two distinct lesions, as in this case, one evidently in the cauda equina of the spinal cord, the other in the cranial cavity, in a young and otherwise healthy man with a syphilitic history, is conclusive as to the specific character of the hemiplegia. Of the exact mode in which the hemiplegia was brought about we cannot speak with equal certainty. If there were, as in some respects would seem likely, a syphilitic tumour of the right crus cerebri, this, probably, also involved and obstructed a part of the circle of Willis. I mean that the man presented symptoms of obstructed cerebral circulation not to be explained by the simple compression of the crus cerebri by a tumour. But whether an artery had been blocked as a result of inflammatory thickening of its walls, or from its having become involved in a gummatous tumour, must remain doubtful.

I need not say that the mental condition which I have illustrated—a state of obfuscation and somnolence not amounting to coma, but lasting in each case for several weeks—is not an ordinary accompaniment of hemiplegia. It is a condition, however, which one has every now and then seen in cases, also due to syphilis, the result of which has not been so fortunate as those described. In such

cases we have found after death some of the arteries at the base of the brain with remarkably thickened walls, and their channels blocked with thrombi, the brain substance itself in the district of their distribution being more or less extensively softened. Now I suppose that if we could have looked into B—'s cranium shortly after his admission, and explored the base of his brain, we should have found, perhaps one, perhaps several, of the cerebral arteries thickened in their walls by syphilitic new growth. This thickening of the walls—usually of a nodular character—narrows the channel, it may be to a very great extent, so that, whilst being still pervious, the tube is reduced in internal diameter to a fraction of its normal calibre. The elasticity of the vessel is, moreover, almost removed, so that the blood has to find its way, as best it can, through a comparatively unyielding tube of unnaturally small diameter. The resistance thus offered to the current of blood must result in the district to which the vessel is distributed receiving a much smaller quantity in a given time than is natural.* (So far as I can see, the compensatory action of the circle of Willis is brought into play on the occurrence of obstruction in one or other of the arteries below it (internal carotid or vertebral), and is scarcely of avail when obstruction occurs in arteries proceeding from it.) The consequence would appear to be that the cortical substance of the hemisphere must be starved of blood to considerable extent. We can easily conceive that this starvation may stop short of death of the tissue—that is to say, there may not be absolute obstruction and consequent softening, but a state of partial atrophy; and either this or a consequent effusion of liquid to supply the place of shrunken convolutions may possibly explain the peculiar condition of somnolence and half consciousness. As regards the attack of hemiplegia which B—— experienced soon after his admission, I should think that it was the result of a limited softening of the brain consequent

* Heubner has some important observations upon this subject. 'Ziemssen's Handbuch d. Krankh. d. Nervensystems,' vol. i, p. 322.

upon a thrombus absolutely blocking up one of these narrowed vessels. Such an accident would produce an actual necrosis of the nerve-substance, whilst the narrowing of the channels alone (if blocking did not take place) might readily occasion a simple impoverishment.

I will not dwell longer upon this point, because our patient's recovery, happily, leaves the matter of necessity in the domain of hypothesis. But I must call attention to a question in therapeutics with reference to the arterial thickening, which appears to be of no small importance. B—, it will be remembered, had been taking thirty grains of iodide daily for two months, when he broke down in his brain. This drug, then, supposing the view taken of the man's pathological state to be correct, had failed to influence the new formation in the arterial walls, so as to prevent the grave consequences of narrowing and thrombosis. No doubt there is the probability that its administration was commenced too late; that changes had taken place in the growth such as rendered its absorption (which might have been practicable at an earlier stage) no longer likely to happen; and there is the possibility also that the dose was not large enough. But it is a fact, however it is to be explained, that we very often meet with this condition of thickened arteries in the post-mortem examination of persons who have been taking iodide of potassium up to the time of their death. I have long had a rather strong impression that the iodide has comparatively little effect upon this arterial change, and that it is best to employ mercury without delay in most cases of this kind. At all events, under a really effective use of this drug (administered by inunction), the restoration to health which took place in these patients was most striking. It is not unfair to suppose, although it cannot be proved, that under its use a good deal of the new growth in the arterial walls, which had resisted the effects of the iodide, was absorbed, so that the channels of circulation became gradually liberated to a great extent.

It is only, of course, through the medium of the patient's

symptoms that we are able to infer that a cerebral artery which has been the seat of obstruction is again permitting the free circulation of blood through it. The artery itself is far away out of reach of the touch or the sight. But I think it is legitimate to assume that what we see occurring in other parts of the circulatory system may likewise happen in the vessels of the brain. In the case of Caroline D—, a girl of eighteen, who was in the hospital last year,* the brachial artery was blocked by what, as the other symptoms showed, was probably a thrombus in a vessel affected with syphilitic thickening. Under the influence of mercury and iodide (they were both employed) the circulation became restored, so that not only did the radial pulse become perfectly evident to the touch, but sphygmographic tracings of it were at length able to be taken in a situation in which during some weeks no sign of pulsation had been perceptible. The position of the artery in that instance offered a most valuable opportunity for observation, such as is necessarily wanting when the arterial change affects, as it does with marked preference, the vessels at the base of the brain.

* Vide 'Clinical Society's Transactions,' vol. xi, p. 205.

LECTURE XVII

NEURITIS, RHEUMATIC AND SYPHILITIC

It was in December, 1877, that Joseph H—, thirty-one years of age, a carpenter, applied at the hospital, looking haggard and cachectic, and walking with great difficulty, the right leg being dragged. He complained of constant cramp-like pain, extending from behind the right trochanter along the back of the thigh to the knee-cap, and thence down the shin-bone to the middle of the instep. There was a tender point where the sciatic nerve quits the pelvis, and another in the popliteal space. The limb had wasted, so that it measured half an inch less in circumference than the left, and the patient said that he had lost two stone in weight since the beginning of his illness, eighteen months previously.

It appeared that after getting wet daily in the country for ten days, he had been seized with violent diarrhœa which lasted for several days, and left him very prostrate. Very severe pain then attacked him behind the hip-joint, and from that time he had never been free from suffering. The pain did not leave him day or night, but was worst in the middle of the night. His own description was that the pain was so exceedingly severe that he was "like a madman with it," and I was told there was evidence to show that on at least one occasion he had seriously contemplated self-destruction. He could not sit or stand without increase of suffering, and never obtained an hour's sleep at a time. The leg had become so weak that he crawled about with difficulty, and he had been

entirely incapacitated for work during a year and a half. I am informed by the gentleman who treated him when he came to town that his condition was attributed to neuralgia consequent upon the exhaustion of severe and continued diarrhœa. Under treatment the diarrhœa subsided, but the pain remained uninfluenced, and eventually he was sent to a metropolitan hospital, where he remained as an in-patient for three months. Whilst there, he tells us, he was injected every night and morning with morphia, which had the effect of giving him only temporary relief from his suffering, and he left the hospital rather worse than on his admission.

When I came to hear this story, and to note the man's deplorable aspect, my first impression was that he must be suffering from malignant disease, and I therefore a made careful examination of his hip, pelvis and thigh. On the front of the femur, at the part which corresponds as nearly as may be to the level of insertion of the adductor longus muscle, was a hard swelling, measuring three or four inches in its long axis and about one inch and a half in diameter. The tumour was only to be felt on deep palpation, was immovable, and evidently connected with the femur, into the shaft of which it shelved off. It was not tender to the touch. A careful search failed to discover any similar swelling at any other part of the limb, or at such points of the pelvis as could be reached by the hand.

On inquiry we learnt that fifteen years previously the patient had suffered from a bubo, which was lanced. He had no recollection of ever having been affected with "venereal sore," and had never to his knowledge had any skin eruption, sore-throat, or eye trouble.

The opinion was expressed that the swelling was either a syphilitic periosteal node or a cancerous growth of the bone, and iodide of potassium was ordered to be taken in ten-grain doses three times a day.

Within a week the pain had so far subsided that the man was sleeping well. He continued to take the iodide

for five weeks, but long before then he had perfectly recovered. There was no longer any pain whatever, and the swelling had disappeared. He then returned at once to his work, from which he had been absent nearly twenty months. This last Christmas week, at my request, he came here, so that we might have the opportunity of learning how he had been getting on. He told us that there had been no return of the pain, that he had been continually at work since he had recommenced his occupation, and that he had never been better in his life.

We may take it as certain, I suppose, that this man suffered from neuritis—perhaps more properly perineuritis—of the sciatic nerve. The district occupied by his agonising pain, the points where there was marked tenderness on pressure, together with the powerlessness and muscular wasting, bear unmistakable testimony to the existence of this lesion. At the same time he had a large periosteal node on the *front* of his femur, which disappeared under the treatment which cured him of his pain and lameness. This position of the swelling, however, would apparently preclude the probability of its having of itself occasioned the perineuritis. I think, indeed, that we must look upon it as a key to the specific character of the patient's disease rather than as the immediate cause of the symptoms from which he suffered, and suppose that there was another node, out of reach of the hand, which had pressed upon and irritated the sheath of the nerve. The history of a bubo, the occurrence of the node, and the exceedingly rapid and complete cure by iodide of potassium, taken together, leave no doubt as to the syphilitic character of the affection. It is quite possible, therefore, that there may have been a gumma in the sheath of the nerve. Considering that the attack began immediately after long exposure to wet, it is not surprising that the sciatica had been referred, at one period of his illness, to a rheumatic origin, and treated accordingly. But the fact is not the less instructive.

There was another carpenter, Samuel C—, who applied

here in October, 1878. He was sixty-two years old, and had worked extremely hard until the last two or three years. Like H—, this man was worn and haggard with pain—so ill and powerless, indeed, that he was brought to the hospital on a bed, and was wheeled into the consulting room on a chair. As he sat he could not lift the right foot from the ground, and a moderate downward pressure upon his knee prevented movement of the left. He complained of dreadful pain down the inside of his right thigh, which sometimes extended to the inner ankle, and also about the right groin and pubic region. The pain was increased if he attempted to stand on the right leg, and "made him shout out in agony." From notes of his case taken by Mr A. E. Broster it appears that two months previously he had been attacked with severe pain in the lumbo-sacral region, and behind the hip. He took to his bed, and in the course of three days lost the use of his right leg. This failure was accompanied by very distressing pains, "as if you were dragging the nerves out" in the right hip-joint and on the outside of the thigh, as well as by rapid loss of flesh. The left leg was not affected until three days before he came to us, and then pains, but of less severity, attacked that limb, which in its turn began also to get weak. Percussion upon the second and third lumbar spines was found to cause intense pain, whilst there was also evidence of decided tenderness about the first, fourth, and fifth lumbar spines and the sacrum. Over all this part sensibility to cold was exaggerated. The right lower extremity was wasted as to the muscles on the front of the thigh, and in this situation the cutaneous sensibility was blunted. The patellar tendon-reflex was present, but small; there was no foot clonus. When iodide had been taken in ten-grain doses for four days, the pain began to ease in his right thigh, and shortly afterwards the power began to improve. In a fortnight he was able to bear some weight on his right leg, and the left leg, which had previously shaken if he leaned upon it, became much firmer. He

gained flesh. The patient was now admitted into the hospital greatly improved, but still suffering to some extent. He was able to be up and about the ward, but complained of aching pain, if he walked, on the inner side of the right knee. This was removed by a blister. At the end of six weeks he was discharged, feeling nearly as well as usual, and able, as he thought, to walk a mile. Although not free from aching pain, his improvement was most striking. The right thigh, which had measured three-quarters of an inch less than the left, had recovered its size, and the irritability of the muscles to faradaism, which had been greatly diminished, was restored. It was notable that whereas early in the treatment a very moderate strength of the constant current had occasioned exquisite pain at certain spots in his right thigh, at the time of his discharge it was felt less in the right than in the left limb. There was no longer any pain on percussion over the lower part of the spine, nor could any spots tender to pressure be discovered in the thigh.

Here were two carpenters, then, who both, by a strange coincidence, suffered from extremely acute pain, loss of power, and wasting in the right lower extremity, following exposure to chill; who both, according to their own account, lost a couple of stone weight in their illness; and who both got well under similar treatment. In spite, however, of this singular correspondence in fortune, I think we must feel that, although they both suffered from perineuritis, the cause of the inflammation was probably not alike in the two men.

In the case of H— there was a periosteal node. I do not mean to assert that a swelling of this kind could not possibly be occasioned by rheumatism, though I have no experience of such an occurrence. But as a matter of common observation there can be no doubt that if one meets with so well-marked a symptom as this in a case where there is a history of a former bubo, and the node rapidly disappears under iodide of potassium, it is not safe to doubt its syphilitic origin.

In C—'s case, on the other hand, there was no existing sign or past history of syphilis. I should not take much account of this absence of evidence if there were other reasons for strongly suspecting syphilis, for we are continually meeting with cases in which the symptoms caused by lesion of some part of the nervous system constitute of themselves the only testimony to the specific nature of the disorder, and experience shows these to be quite as pathognomonic as affections of the skin. But in this case, the nature of the disorder pointing with at least as great probability to rheumatism as to syphilis for its basis, it becomes necessary to attach very considerable weight to the circumstance that there is an entire absence of all signs of the latter disease. Moreover, it must be noted that, notwithstanding the great improvement in the patient's condition, he is still suffering from more or less pain and weakness, and that, when he came to us last week he complained of pain, evidently of rheumatic character, in the muscles of one arm. That iodide of potassium is often of great service in chronic rheumatism is a well-attested fact, which requires to be remembered when we are inclined, as we sometimes are, to find too readily in the improvement alone produced by its use strong evidence of the syphilitic nature of the lesion for which it is employed. But it has often seemed to me that there is an exceptionally sharp and decided influence exerted by the drug in many syphilitic affections which is only mildly imitated, as a rule, by its effects in chronic rheumatism. In H— this was well seen. In a very short time the whole mischief was swept clean away, and the man remained apparently as strong and well as ever. In C—, although the influence of the iodide was greater than is perhaps usually seen in rheumatic cases, yet the improvement of the first few days was by far the most important, and the cure, though certainly striking enough, cannot be said to be complete. Nor must it be forgotten, in reference to this point, that C—'s illness, when he came under treatment, had lasted

only one ninth part of the time during which H— had been suffering.

The pains in the case last related were referred to the regions innervated by the ilio-hypogastric, ilio-inguinal, genito-crural, external cutaneous, anterior crural, and obturator nerves of the right side. From the fact of the left limb being also, though very partially, involved, and from the intense pain in the lumbar portion of the spine when this was percussed, it seems likely that the actual seat of the lesion was in the nerves going to constitute the lumbar plexus, whilst they still occupied the cavity of the spinal canal. That the lesion did not (as in certain respects the symptoms appeared to suggest) consist of a tumour, pressing especially upon one half of the cord, is shown, I think, by the fact that there was no marked cutaneous anæsthesia of the left lower extremity.

It is very important not to make the mistake of referring syphilitic neuritis to a rheumatic cause, and treating it accordingly. That mistake appears to have been committed by some one in the case of Richard S—, a cab-driver, who was admitted into the hospital on September 5th, 1877, on account of gradually increasing hemiplegia of the left side, with paralysis of the right oculo-motor nerve and great mental obtuseness. He passed into a curious state of prolonged somnolence, and I have had occasion to refer to his progress in hospital whilst dealing with the cerebral symptoms.* It is to what had occurred some time previous to the commencement of his cerebral symptoms that I wish now to draw attention.

This man had suffered from illness with but little intermission since the beginning of 1876, more than a year and a half before he came here. It is necessary to go thus far back in his history.

At first he suffered from what he calls "rheumatic" pains in the left lower extremity. This word "rheumatic" is responsible for a great deal of mischief. Because pain is

* See Lecture XVI.

one of the symptoms in rheumatism almost every pain that we meet with is called rheumatic. The pains of syphilis, of tabes dorsalis, of scurvy, of neuralgia and myalgia are constantly so termed.

The pains that this patient suffered varied in intensity. They were of an aching intermittent character, and affected the thigh and leg of the left side. Sometimes he would be a day or two or even, as on one occasion, a whole week without pain. The pains were more in the front than the back of the limb. They were not increased by pressure, and were worse at night. The leg did not jump. The pains were supposed to be due to rheumatism, and he was treated accordingly. About three months after the commencement of his pains he noticed that his left leg felt weak, and it shortly afterwards grew so powerless that he could only mount the stairs by crawling upon his hands and knees. There was also a much less marked weakness in the right lower extremity at that time. There was nothing wrong with the bladder. There had not been any notable pain in the back (we inquired especially about this), and so far as he knew he had not been feverish.

When these pains had lasted some five months without relief from treatment he went into a metropolitan hospital. A month before taking this course he had observed that the left thigh and leg were smaller than the right. In the hospital he lost the pains, but his limb had not increased in size when he was discharged in the month of August. He then returned to his occupation, at which he had been engaged about three months when he was seized with pain across the forehead and vomiting which continued three or four months, and then he became paralysed on the left side as I have described.

I may here note the result of an examination of this patient's lower extremities which I made early in October, when his mind had become clear and his answers could be depended upon.

There was cutaneous anæsthesia over both lower extre-

mities, but in the left much more than in the right. It extended to the back of the limb, and was more marked on its outside than inside. Above, the impairment of sensibility began on a level with the fourth lumbar vertebra on the left side and the fifth on the right.

A hot spoon was felt as warm only in both lower extremities. A cold spoon was felt as warm in the front of the left thigh.

The induced current was felt as touch only in both lower limbs.

The right thigh, at a point eight inches above the upper border of the patella, measured twenty inches in circumference. The left thigh, at the same point, sixteen inches and a half. The right leg, at a point five inches below the lower border of the patella, measured thirteen inches and seven eighths; the left leg, at the same point, twelve inches and six eighths.

The excitability of the quadriceps femoris muscle by induced electrical currents was equal, and apparently not materially reduced in either limb.

In this case there was a history of a chancre (not followed by sore throat or skin eruption) about ten years before this attack. That his disease was syphilitic there can be no question, the cerebral attack indicative of tumour, from which he entirely recovered under mercurial treatment, leaves the nature of the first affection, in the left lower limb, in no doubt. As regards the situation of the lesion, I think the evidence is rather against the idea of a distinctive lesion of the substance of cord, though this is possible. The loss of power and the muscular atrophy were almost entirely confined to the left lower limb, and it was on this side that the cutaneous anæsthesia was most strongly marked, although it affected both lower extremities. There had probably been a gummatous meningitis over the lowest part of the lumbar region of the cord. The pressure from organised inflammatory product would cause destruction of nerve fibres, and hence permanent muscular atrophy and anæsthesia.

S— came to us again this winter (December, 1881), four years having elapsed since he left the hospital, during which time he has been actively engaged in his occupation. He applied because he had been again attacked with pains in the head. Under a short course of iodide of potassium these disappeared.

We took the opportunity of examining his lower extremities with the following result:

The patellar tendon-reflex is absolutely wanting on the left side; present, but subnormal, on the right.

The vasti interni were very carefully tested with induced currents. At the motor points, currents of one degree greater strength were required on the left than on the right side. To direct percussion the response appeared to be equal and normal. The patient could extend the left leg, *i.e.* voluntarily contract his quadriceps extensor, though not so strongly as on the right side.

LECTURE XVIII

ON CASES OF RAPID AND ALMOST UNIVERSAL PARALYSIS

It is convenient to use some such general title as the above for the designation of certain cases which are of extreme interest and importance, but the true pathology of which we have yet to learn. I have now met with several examples—one has recently quitted the hospital quite recovered. I propose to relate his case together with others which are apparently of very similar nature, but which at the same time present certain differences, the existence of which may perhaps aid in throwing light upon the nature of the disorder.

On April 14th, 1879, at the request of Dr Slight, of Clifford Street, I visited Thomas O—, a tailor's cutter, forty-four years of age, who was lying paralysed in lodgings.

Finding that he was suffering from paralysis of all four extremities and of both sides of the face, which had been rapid in its onset, and was hourly increasing, I ordered him without loss of time to be removed to this hospital, where he was received under my care, and notes of the case were taken by Mr A. E. Broster. The patient was a medium-sized man, sparely built, whose mental condition was perfectly normal. His face was expressionless, being paralysed on both sides, and he was unable to close either eye, a gap between the lids measuring about 5 mm. being left when he tried to do so. He was apt to bite his cheeks when eating, and the food accumulated between the teeth and cheeks on either side, but especially on the left. When he smiled there was just the faintest movement of the right angle of

the mouth, but none of the left. The mouth was very slightly drawn to the right, and opened somewhat wider on that side than on the left. He could not whistle, nor frown, nor move the skin of his forehead in the slightest degree. The saliva ran from his mouth. There was complete paralysis of the external rectus muscle of each eye. The pupils reacted well to light. The tongue was broad, flabby, and somewhat tremulous. It was protruded straight, and was not apparently wasted.

He was unable to swallow solids except with the greatest difficulty; they stuck in his throat, and he was obliged to drink to wash them down. He could take fluids well enough, and they never regurgitated through the nostrils. His voice was normal, the hyoid muscles acted properly, and when asked to swallow, the larynx was felt to rise apparently as freely as in health. The sterno-mastoid muscles seemed exceedingly feeble and flaccid, and so did the platysma on each side.

The respiration was mainly upper thoracic, the lower part of the chest not expanding as it should, and the diaphragm scarcely, if at all, moving. The respiratory movements were not manifestly aided by the sterno-mastoids. There was no marked inequality of expansion on the two sides of the chest. There was some loss of control over the sphincter ani when his bowels were relaxed, and he was obliged to be quick when he wanted to micturate.

As regards the upper extremities, by the dynamometer the right hand gave a grasp of 18 kilogr., the left 14 kilogr. He was just able to feed himself, but could not dress himself. The muscles did not appear to be wasted. Tested with loaded balls it was found that he could not judge of weights with his left arm, though he could do so tolerably well with his right. He was unable to button his clothes, nor could he hold an orange to eat it. "His mouth seemed to run away from it."

The lower extremities were also profoundly affected. The patient was unable to stand. As he lay he could

move one foot across the other, but could not lift either more than three or four inches off the bed. There was great muscular flaccidity. He did not know where his legs were in bed. The patellar tendon-reflex was carefully tested on several occasions, and was found to be absent in both legs. There was no reflex clonus of the feet.

Over the right side of the face there was complete loss of sensibility to touch and pain, with apparently increased (but, at all events, well-retained) sensibility to heat and cold. The loss of sensibility was likewise observed, though to a somewhat less extent, over the left side of the face, and also, though here again in a less complete degree, on his forehead. In these situations the sensibility to heat and cold was perfectly retained, and apparently, indeed, increased. Below the middle of the forearm there was almost entire loss of sensibility to touch and pain, whilst heat and cold were well recognised. Where the alteration in sensibility began there was what the patient described as a "band-like feeling around the arm." In the tips of his fingers there was a constant tingling sensation, and anything which he touched with them felt hot.

He could not pick up a coin. In trying to take a shilling out of his trousers' pocket he brought his hand out several times, thinking he had it in his fingers, which was not the case. In his lower extremities sensibility was also greatly modified. Below the middle of the thigh, on each side, neither a touch nor the prick of a pin could be recognised. Over the whole of both feet, as well as half way up the legs and on the posterior surface of the rest of each lower extremity, sensibility to heat and cold appeared intensified. To the soles of the feet water of the temperature of the air felt "stone cold," as he termed it, causing him to draw his feet away sharply from the discomfort it occasioned. Heat appeared also to be perceived in an exaggerated degree, but not to the same extent as cold. Over the whole of the trunk the cuta-

neous sensibility was normal. He had frequent pains in the calves, sometimes of a very sharp character, and the legs would twitch when they occurred. At other times the pains were of a dull heavy character.

No abnormality could be perceived in the spinal column. Percussion caused no pain or tenderness. He complained of a feeling of stiffness, but this was apparently subjective. On asking him to bend his back it was found that he could not move it, but the attempt to do so caused some pain in the lumbar region.

Assafœtida and sumbul were recognised by the right nostril, but not in the slightest degree by the left.

He complained of occasional muscæ. There was diplopia. A pencil held up was seen as two, about five inches apart, the false image to the left and parallel with the other. There was no hemiopia. The field of vision appeared to be of normal extent. Examination by the ophthalmoscope disclosed no change in the fundus oculi of either side.

Hearing was normal.

He did not recognise salt or sugar with his tongue; his remark was that "they both tasted warm, and hotter on the right than on the left side."

The pulse was quick, temperature normal.

He complained of a good deal of headache in the right frontal region, spreading to the vertex. He had also sharp pains, sudden and of momentary duration, like a knife-stab, in the right thigh and knee. His sleep was not good.

The urine contained no albumen.

The patient showed no disposition to develop bedsores.

Such was his condition when admitted into hospital. His illness had come on in the following manner: The people with whom he lived say that about a month previously they noticed his face to be drawn to the right. He had no idea of this himself, and for his part felt in perfect health till March 31st, just a fortnight before I saw him. He then noticed a sensation of "pins-and-needles" in his

hands and feet. Although this feeling continued he was able to go about and follow his occupation, but with difficulty, till April 5th.

On April 3rd, whilst walking across a street, he tried to hurry out of the way of a passing vehicle, when he found that his legs seemed to stick and refused to move fast—one as bad as the other. The same day he began to see double.

4th.—In the same state. He still continued to go to work.

6th.—He got up in the morning, succeeded in dressing himself, and walked to see his doctor (about two minutes' walk) with much difficulty. He could not work after this date.

8th.—He could not walk. There was a dull aching pain along the hamstrings. He was unable to sleep. He found difficulty in swallowing meat. Very severe right frontal headache was complained of.

11th.—He could not dress himself. The fingers were numb, so that he could not feel the buttons. At first there was no pain whatever in the spine on movement, but between the 9th and 14th he found, in stooping to the ground, an aching pain in the lumbar region.

At no time was there any vomiting.

There was no history of any blow, injury, or exposure to cold. He had not suffered from soreness of the throat, nor had he been brought in contact with any one suffering from sore throat, diphtheritic or otherwise. At the age of twenty-four he had a syphilitic chancre, the scar of which remains visible. Besides this he never had, he says, a day's illness.

There is no family history of gout, phthisis, or nervous disorder.

He was ordered to take ten grains of iodide of potassium every four hours.

On April 15th, the day after his admission into hospital, I found that reaction to the induced current was almost entirely absent in the muscles of the face, and also in the

thenar eminence of each hand; it was lessened, though not to the same extent, in the muscles on the front and back of the forearm.

Next day there was a little more power in moving the external recti muscles. Two days later the sensibility of the face was decidedly improved; a touch could be felt on each cheek. There was still internal strabismus, but the external rectus muscle could now move the eyeball three fourths of its proper distance. He was now able to bend his back, when propped up, with scarcely any discomfort.

On the 22nd he could feel a touch on each forearm as well as ever, but a pin-prick below the middle was as yet only recognised as something hot. He could close his eyes better, though not effectually, and there was scarcely any abnormal deviation of the eyeballs. There were deep excavations in the interosseous spaces from atrophy of the muscles.

On April 29th I found, on examining him electrically, that there was very slight reaction to strong induced currents in all the muscles of the lower extremities, and almost total absence in the interossei and thenar muscles of the right hand. The electrical applications were only slightly felt in all the limbs. He now shut his eyes perfectly, could show his teeth, and could whistle. The diplopia continued, but there was less distance between the images. He was ordered to be rubbed daily with ʒj of Ung. Hydrarg., and to take fifteen grains of iodide every four hours.

During the week ending May 8th he had made rapid progress. There was now no diplopia whatever, and he read with ease. The dynamometer gave with the right hand 20 kilogr., with the left 18. The sensibility of the hands and forearms to touch, pain, and heat had become normal. In the lower limbs, although he felt everything, the sensation was dulled. Of late his hands and fingers had tingled, "as if asleep." He could not stand, but he moved in bed and managed to get on the nightstool by himself, which he could not do a week previously. There

was no delay with the bladder. In the lower dorso-lumbar region there was still some stiffness and uneasiness in bending the back.

On the 17th he stood fairly for the first time, and, with help, walked a few steps. There was still some pain in the lower part of the back on movement.

By May 19th it is noted that "his taste is perfect, and that there is now nothing whatever wrong with the face or eyes. The grasp with the right hand measures 23 kilogr., with the left 22. He is able to button his clothes, feed, and dress himself. He can walk, but says the ground does not feel quite natural to him. His legs seem to spring under him." His gait at that time was ataxic. He complained of a "dull pain" in the lower limbs, which, when he moved them down sharply, seemed to run up to his back "and make his teeth chatter." He continued to take fifteen grains of iodide every four hours, and to be rubbed with ʒj of Ung. Hydrargyri daily.

On May 20th, the gums becoming a little red and tender, the ointment was ordered to be used on alternate days only.

By June 27th the dynamometer gave on the right side 31 kilogr.; left 27 kilogr.

He was discharged on July 24th. The mercurial ointment was ordered to be discontinued; the iodide to be taken in doses of five grains three times a day. It was noted that the patellar tendon-reflex was still altogether absent.

On August 10th it is noted that he could take a walk of three to four miles. The ground felt natural, and the legs did not spring. There was now decided patellar tendon-reflex in the right leg. Three days later it was found also to have returned in the left leg. There remained a little numbness on the front of the left thigh and in the toes. He returned to his work well, and has since continued regularly at his employment.

In 1859 Landry described, under the name of "acute ascending paralysis," a condition characterised by paralysis

mounting from the legs to the arms, and even to the district innervated from the medulla oblongata. I may quote here the definition of this malady which is given by Erb, in his monograph on "Diseases of the Spinal Cord," in 'Ziemssen's Cyclopædia :'—"The disease designated by the name of paralysis ascendens acuta is clinically characterised by motor paralysis, which generally begins in the lower extremities, spreads pretty rapidly over the trunk to the upper extremities, and usually also involves the medulla oblongata, which sometimes runs its course without fever, sometimes with more or less active fever, which but slightly involves the general sensibility and the functions of the bladder and rectum, and which runs its course without any notable atrophy of the muscles and without any diminution or change of their electrical excitability. In the majority of instances the disease terminates fatally, by asphyxia, paralysis of deglutition, and the like; but lighter cases may end in recovery. The anatomical characteristics of the disease are at present purely negative. No pathologico-anatomical alterations are to be found anywhere, and especially not in the spinal cord, which might explain the picture of the disease. In particular there are no signs of hyperæmia within the spinal cord, of myelitis, of acute destruction of the ganglion cells or nerve fibres."

"Exceptionally," Erb remarks, "the disease may be seen to progress downwards within the cord, instead of pursuing the more frequent ascending course. The paralysis then begins in the nerves of the bulb, and successively seizes first the upper and then the lower extremities. So in the case of the distinguished Cuvier, as reported by Pellegrino-Levi."

If we come to examine details it will be seen that the case I have just described although presenting a *primâ facie* resemblance to this disease, differs from it in certain essential particulars.

1. In the extensive and profound affection of sensibility.

2. In the greatly diminished (almost lost in the lower extremities) electrical excitability of the muscular system.

3. In the occurrence of paralysis of the external recti muscles.

Erb, as the result of a summary of the various published cases, remarks :

"1. As a rule the very slight disturbance of sensibility is especially noticeable.

"2. The electrical excitability of the paralysed nerves and muscles remains entirely normal. In all the more recent cases, carefully investigated by skilled hands (Pellegrino-Levi, Bernhardt, Westphal, &c.), no anomaly of electrical excitability worth mentioning has been found, even after the disease had existed for a number of weeks. By this very means," he adds, "the disease seems to be distinguished in a very significant manner from all progressive paralysis caused by gross anatomical lesions within the spinal cord (myelitis centralis, poliomyelitis anterior subacuta, &c.).

"3. Actual paralysis of the muscles of the eye," he says, "has not been observed."

In many respects the case resembles those described by Duchenne under the title of subacute spinal paralysis, but differs in the important circumstance that in Duchenne's cases there is no affection of sensibility. Indeed, the latter appear to be cases of what is now called subacute poliomyelitis.

In the present case it will have been noted that the first symptoms of disease occurred in the face, which was drawn to the right. This was followed a week or two afterwards by "pins and needles" in the patient's hands and feet. A little later and there was loss of power in the lower extremities, followed in a day or so by weakness and numbness of his arms, paralysis of external recti, of deglutition, &c. So that the term "ascending" would not be applicable to the present example.

On the other hand, the resemblance of this case to one which was in the hospital in 1873, and which I brought

before the Clinical Society,* is most striking. So remarkable was it that, struck by the extraordinary likeness, I did not doubt that a similar result would occur; and when this man was admitted into the hospital I ventured to express my conviction to the medical officer, first, that a history of syphilis would be found, and secondly, that the man would recover under appropriate treatment. The patient overheard my remark, and at once said, "I had syphilis many years ago."

The following is an abstract of that case:

William H—, æt. 45, was admitted on January 8th, 1873, unable to stand, with little or no power of moving his arms. He had double facial paralysis, respiration and deglutition were greatly impeded, and there was more or less strongly marked cutaneous anæsthesia throughout the trunk and extremities. On being asked to close his eyes, which were constantly open, the eyeballs were turned upwards, so that the cornea was concealed by the upper lid, but there was complete inability to shut his eyes. He could not wink, frown, or whistle. He could not swallow solids, and fluids frequently returned through his nostrils. There was paresis of the soft palate, especially on the left side. The muscles of mastication were not affected, and taste, hearing, and smell were said to be perfect. There was paresis of the left external rectus muscle. The ophthalmoscope showed no change in the discs. There was but little movement of the diaphragm, and breathing was so impeded by defective action of the intercostals that the patient could not lie down in bed. His sterno-mastoids and trapezii acted freely. The grasp of the hands was almost *nil*; there was but little pain in the arms, and none in the lower extremities. He suffered no pains, but complained for the first two or three weeks of "pins and needles." There was paresis of the bladder.

The muscles about the mouth showed the signs of reaction of degeneration. In those of the arms the reac-

* 'Clinical Society's Transactions,' vol. vii, p. 75.

tion to faradaism was greatly diminished, whilst in those of the legs it was quite absent. In the left thigh it was greatly diminished, and almost entirely absent in the right thigh. But in no part of the upper or lower extremities was there increased action to slow intermissions of the galvanic current. In the face, however, this was marked. The facial muscles reacted to interruptions of a current from six cells (Stöhrer).

There was no headache, intellectual confusion, or vomiting. The heart, lungs, and kidney were healthy. The temperature was 99° Fahr.

A month before he came to the hospital this man felt numbness in his finger-ends, followed on the same day by weakness in the legs, which increased next day, and was then accompanied by numbness about the buttocks extending downwards to the thighs and calves. The weakness increased day by day, and a week after the beginning of his illness he had the sensation of a tight band around the belly. A few days later and he could use neither arms nor legs. The difficulty of swallowing was not observed till a fortnight after the commencement of his illness. There had been no fever.

His general health had been good. He had not suffered from diphtheria. Fourteen years previously he had a chancre, which had not been followed by sore throat or eruption of the skin. Some indurated glands were found in the right groin.

The condition of this man was so grave that I at once admitted him into the hospital, fearing, from the degree to which respiration was involved, that he would die upon the road if I allowed him to return home. He was put upon a water-bed and ordered eggs, wine, and beef tea, with ten-grain doses of iodide of potassium three times a day. Within twenty-four hours he had improved, so that he felt much less distress of breathing, and increased strength. Day by day there was still further improvement. On January 16th, eight days after admission, he could lie down and sleep at night, could close the right

eyelid one fourth of the normal extent, could swallow bread-and-butter in addition to beef tea and wine, but as yet nothing more solid. I now found the muscles on the front of the left leg responding, although sluggishly, to a strong induced current. On January 20th the right eye could be shut completely, and the left to three quarters of its normal extent of closure. He could eat meat, and his features showed a trace of a smile for the first time. His right arm had increased considerably in strength. There were still no reflex contractions on tickling the sole of the foot. He no longer complained of difficulty in breathing. By the end of January both eyes could be closed, and in a few weeks more all the facial muscles had quite recovered. At the end of February his limbs were stronger, but he could not stand. He complained of numbness in the arms and legs. Early in March he could extend the leg against firm pressure, and his other symptoms had improved in a corresponding degree. On May 6th he began walking, leaning on an attendant, and on May 13th with the aid of a stick and crutch. On May 21st he was discharged, able to walk, and use his arms, but still somewhat weak. All symptoms of paralysis had then disappeared.

After that time he presented himself occasionally as an out-patient, rather because I wished to keep him under observation than that he had need of medical advice. On July 23rd he said that he was nearly as well as he had been before the attack, and that he had been able to resume his employment. A little time afterwards I saw him again; he had entirely recovered, and I showed him on March 13th, 1874, to the members of the Clinical Society.

Besides the measures already described, the treatment consisted in the administration of iodide of potassium, at first in ten-grain doses three times a day, increased at intervals to twenty, thirty, forty, fifty, and eventually to sixty grains, at a dose, three times daily. The increase was regulated by his progress. If, after improving for several days whilst taking a certain quantity his progress

appeared to come to a standstill, the dose was increased, and this always appeared to give a fresh impetus to his recovery. About the end of February, when he had been taking sixty-grain doses, and had ceased to show any marked daily improvement, I ordered him to be injected subcutaneously with a solution of mercury.

There is one question which immediately suggests itself in reference to both these patients. Were they cases of diphtheritic paralysis? Apart from the fact, about which both patients were carefully questioned, that there had been no preceding sore throat nor abrasion about the body, the symptoms differed considerably from those of the paralysis which occasionally follows diphtheria. In such circumstances there is usually a striking constancy in the character of the symptoms, and especially in their order. The paralysis nearly always begins in the velum palati, and is marked by a peculiar nasal voice and difficulty of swallowing. Next, there is commonly some trouble in accommodating the eye for near objects. Less frequently some of the external muscles of the eye are attacked, causing diplopia. Then comes incomplete paralysis of the extremities, with more or less anæsthesia. The condition generally is most often one rather of paresis than of paralysis, but it is important to note that Niemeyer reports having seen in one epidemic two cases of total paralysis of all the extremities. I have never met with an instance of complete double paralysis of the facial nerves as a consequence of diphtheria. In both the cases described there was complete paralysis of the lower extremities before any disturbance took place in the act of deglutition, and there was never any difficulty in accommodating the eyes.

In the first case that I have related—that of the tailor's cutter—the superficial resemblance to tabes dorsalis when the patient began to get about a little was very striking. He complained, as I have said, of sharp pains, sudden and of momentary duration, like a knife stab, in the right thigh and knee. His knee-phenomenon was absent.

There was cutaneous anæsthesia. His gait was ataxic, and he said the ground did not feel natural to him; his legs seemed to spring under him. The other symptoms, however, and especially the loss of faradaic excitability in his muscles, were sufficient, I need not say, to prevent an error of diagnosis in this respect. But as between diphtheritic paralysis and this rapid and almost universal paralysis to which I have called attention the differentiation, I must confess, is not so easy.

As I have remarked, difficulties connected with the velum palati and the accommodation of the eyes are usually the earliest symptoms of diphtheritic paralysis. But, on the other hand, it must be allowed that the symptoms which are usually the first and the most constant may not occur at all. Of this I can give three recent examples.

I saw a gentleman on July 4th, 1879, who complained of numbness in his fingers and feet, which had begun three weeks after what had evidently been an attack of diphtheria, under which he nearly sank.

On examination I found no objective signs of disease. The patient looked well, and had no complaint to make of his general health. There was simply, he said, a little feeling of numbness in the ends of the fingers, which made it difficult to pick up small objects and button his clothes, and in the feet a similar sensation. His urine contained no albumen. The ophthalmoscope showed no change in the fundus oculi. His sight was perfectly good, and he had no difficulty in accommodation. There was no history of syphilis. The patellar tendon-reflex was normal in each leg.

On July 7th he complained of some want of power of grasp. The muscles of the forearms showed a normal reaction to induced currents.

A fortnight later (July 23rd) I saw him again. The feet and hands, he said, now felt perfectly dead. In walking he would catch his left foot. The symptoms did not affect any part of the head or trunk, but were entirely

confined to the four extremities, in which there was considerable impairment of cutaneous sensibility and a certain loss of power. As an illustration of the loss of sensibility in his hands, he told me that he had taken his cheroot-box out of his pocket with the full conviction that it was his purse. He had the greatest difficulty in dressing himself.

The sensibility for temperature was to a great extent retained, and he could tell the difference between a linen and a woollen material, not by the texture, but by its power of conducting heat. There was, however, some delay in the appreciation of heat impressions. He told me, for example, that he had to leave his hand in water a few seconds before he could be sure that it was of a proper temperature to use to his face.

He was weak on his legs, so that walking made him feel tired and sick, and he could not go downstairs without keeping hold of the balustrade. I noticed now that he staggered a little in his walk, and when he stood with his feet together, and the eyes were closed, he swayed about. The numbness was described as having now extended up the back of both legs.

I could find no very notable defect of electrical excitability in the muscles of the forearms, but the response in each was alike, and it is not easy therefore to be sure on this point. Examination of the urine, as well as by the ophthalmoscope, continued to give negative results. There was no difficulty in swallowing, the sight was still unimpaired, and the functions of the bladder and rectum remained normal. The patient looked well, and said that he felt perfectly well, except in respect of the symptoms described. The patellar tendon-reflex was now found to be absent from both legs.

After this I lost sight of the patient, who was living at some distance from town, until many months afterwards, when he told me that he quite recovered about the end of August—*i.e.* in about a month after his last visit. Examination now showed the patellar tendon-reflex present and normal in each leg.

The treatment consisted in the use of faradaism, and the administration of iron and arsenic.

There was a complete absence in this case of many symptoms which are so commonly present as to be almost pathognomonic of diphtheritic paralysis. The voice had never acquired any nasal tone, nor was there any difficulty in swallowing, and fluids never regurgitated. The sight remained throughout perfectly good, the smallest type being read at a normal distance, so that the accommodation of the eyes, which rarely escapes in the paralysis following diphtheria, was never in this instance affected. The numbness described was confined to the extremities, no part of the head or trunk being the seat of it at any time.

The disappearance of the patellar tendon-reflex whilst the patient was under observation is interesting. It furnishes proof, if such were wanting, that this peculiar paralysis is dependent, when it affects the extremities, upon a lesion either of the spinal nerves or in the substance of the cord. A cerebral lesion would not account for this symptom. A lesion of the posterior roots of spinal nerves alone would be sufficient to interrupt the reflex; but the fact that there was loss of power as well as anæsthesia shows that, if the lesion were not one of the substance of the cord, it must have involved either the trunks of the mixed nerves or the anterior as well as the posterior roots.

That this case was one of diphtheritic paralysis there could be no doubt.

In August last a man, æt. 57, came to me on account of staggering and uncertainty of gait which had existed for five weeks. Three or four days after the breaking of a "quinsy" in his throat (so he described it) he began to feel numbness in the left foot, and to a slight extent also in the right. It gradually spread to his hips, and later to the fingers of his left hand. Like the last case, he had nothing to complain of with regard to his sight or his power of swallowing, there had been no regurgitation of fluids, and his voice betrayed no nasal accent. His

complaint was chiefly of weakness in his legs. The patellar tendon-reflex was quite absent in each leg.

Now, the symptoms which this man presented might well have led to a diagnosis of tabes dorsalis. I noted in my book at the time: "A very interesting resemblance to tabes; absent reflex; difficulty in turning round." The resemblance was still further marked by the fact that the vastus internus muscle responded to direct percussion, although a blow upon the ligamentum patellæ failed to produce any contraction. I tested the vasti interni muscles very carefully with induced currents, and found so very slight a lowering of excitability as would certainly have passed unobserved except that I had been especially concerned to look for it. The foot-sole and cremasteric reflexes were good on each side.

It may be said with this remarkable resemblance in the condition of the knee-phenomenon: "How do you know that the case was not one of tabes?

Circumstances made this a really important question, for although the symptoms had begun shortly after a bad throat yet it turned out that this had evidently been acute tonsillitis, and, moreover, that he had previously suffered on eight or nine occasions from quinsy. He had been laid up a week with it this time when "it broke," and he felt pretty well again. Immediately after the breaking of the quinsy he went down into a cellar in his house in which there was an untrapped drain, which, it was notorious, stank most offensively. It would seem that with a tonsil presenting an open wound he became readily infected by this exposure.

He had never experienced anything like lightning pains. His pupils responded well to light. If we compare this case with the last, about which there could be no doubt, it stands out evidently as one of diphtheritic paralysis. Under treatment he improved, but I lost sight of him before he was well.

(Let me here make a remark by the way. I feel sure, from observation, that persons subject to quinsy are pecu-

liarly sensitive to poisonous influences from drains and the like. I am also disposed to think that persons whose tonsils have been excised show an equal proclivity to such influences.)

Not long since I saw in consultation a gentleman more than seventy years of age who suffered from symptoms a good deal resembling those of tabes dorsalis, including absence of patellar tendon-reflex. Inquiry in that case elicited a conclusive history of a very bad throat caught in Paris a few weeks previously, which had been diagnosed by medical men as due to sewer emanations.

I have felt it right to consider very carefully the question of diphtheria as a possible explanation of these remarkable cases of almost universal paralysis before concluding that the cause was syphilis, which was successfully treated. Although the cases of diphtheritic paralysis which I have just described are examples which show that there is not the amount of constancy with regard to the early affection of the velum palati, which is usually supposed, yet if we take all the circumstances into consideration I do not think that they will dispose us to adopt diphtheria as an explanation of the first cases narrated. It may be thought, perhaps, unnecessary to discuss this question, inasmuch as neither of these patients had suffered from sore throat, nor had they been, so far as is known, exposed to infection. But a recent communication to a French journal would seem to show that we cannot dismiss the matter so easily.

Dr Boissarie* describes an epidemic of diphtheria without angina in which he was himself a sufferer. It was remarkable, he states, in this—that paralysis in several cases occurred suddenly without angina or any diphtheritic affection of the skin or mucous membrane, and led to death in some hours or days. In other cases the paralysis was *followed by* angina instead of being preceded by it, as ordinarily happens. In the midst of these exceptional cases he observed others of characteristic diphtheritic

* 'Gaz. Hebd.,' 1881, No. 20 and 21.

throat, which were neither preceded nor followed by paralysis.

As regards the question of syphilis, it is to be remarked that both these patients had certainly been infected, and in each case an immediate improvement and rapid recovery took place under the influence of iodide and mercury. It will be interesting to inquire whether in any similar cases which have been recorded there has also been a history of syphilitic infection.

I lately saw a gentleman, æt. 46, who had recovered completely from an attack which was evidently of the same kind as those I have described. It appeared* that the patient one morning felt a slight numbness and loss of power in his right arm, and later on in the day his left arm and legs also below the knees became similarly affected. These symptoms increased, and were accompanied by sleeplessness and pain in the loins and shoulders. On the fifth day from the beginning of his illness he was quite unable to stand. At that time he complained of some slight impairment of vision, his pupils were regular and responded to light. He was unable to close his eyelids, especially on the left side, and the tears overflowed and trickled down his cheeks. The left side of the face was flattened and smooth. He spoke slowly, and was not able to articulate the labial sounds, to whistle, or blow out his cheeks. There was no paralysis of the tongue or palate, no perversion of the taste, and the sensibility of the lips was unimpaired. There was complete paralysis of the arms, and the fingers, which were in a half-flexed position, could not be voluntarily straightened. A tingling sensation was felt in the forearms and hands, but sensibility was only very slightly impaired, so that if one of his fingers were touched he could tell which it was. His legs also were completely paralysed. He could neither raise his feet from the ground, draw them up when in a recumbent position, nor bring his knees together

* My account is derived from an admirably detailed history of the case furnished by the patient's medical adviser. The illness took place abroad.

when they were apart. There was no tingling in them, and sensation was only about as much impaired as in the hands. He complained of difficulty in breathing, and a feeling of tightness across the chest. There was no pain or giddiness in the head, and the intellectual functions were completely unimpaired. The sounds of the heart were healthy although its action was weak, and the pulse was small, compressible, and quick. The hearing was perfect. No tenderness over any part of the spine could be discovered, and the passage of a hot or cold sponge over the spine gave no indication. There was no paralysis of any of the abdominal viscera.

The patient was treated with iodide and bromide of potassium. A month after the commencement of his illness the facial paralysis had nearly disappeared, a slight flattening of the left cheek alone remaining. The power of grasp had returned to a certain extent, so that he could now squeeze a hand, hold a handkerchief in either hand, and wipe his face, touch the back of his head, and put on or take off his spectacles. His fingers, however, were rather cramped and painful at times and he suffered a good deal from a feeling of cramp in his legs, and also from numbness, which was relieved by rubbing. He could not raise his feet from the ground when sitting in a chair, but could bring his knees together and separate them with ease. He could also draw the left foot towards him when lying down, though with great difficulty. He had lost the great pallor which had been marked at first. He was able to eat and sleep well, and was in good spirits.

When I examined this patient, seventeen months after he had been attacked, he was practically well and could walk ten or twelve miles. He had, indeed, nothing to complain of except that the movement of his arms did not feel to him quite so free as they might be. During the first two months of his illness he had emaciated extremely, but had since recovered flesh. He weighed upwards of thirteen stone, but had been at one time reduced to a little over eight stone. The patellar

tendon-reflex was perfect. His present medical attendant informed me that he had found this normal, on his landing in England, four months after the beginning of his illness.

Struck by the similarity of this case to the others, I questioned the patient on the subject of syphilis, with the following result. At eighteen years of age he had a chancre, which remained open three weeks, and was treated with black wash. He does not think he took any mercury. The sore was followed by sore throat and a skin eruption, which came out chiefly on his forehead, and it was about three months before he got rid of his secondary symptoms.

A case which occurred in the practice of Prof. Wagner * presents a remarkable similarity to mine. In this instance, too, more palpable symptoms of constitutional syphilis were present at the time of the patient's being attacked by almost universal paralysis, and afforded the corroborative evidence which is absent in my cases.

The patient, a military man, æt. 35, had contracted a chancre in 1862, which was followed five months afterwards by a throat affection, which subsided, after many weeks' duration, under non-specific treatment. A year later he had a papulo-pustular eruption, for which he was rubbed with mercury. Ere two years had passed he had a recurrence of throat affection, which was treated exclusively by mercury. In August, 1866, he complained of stiffness in the neck, and at the same time there appeared, in the neighbourhood of the spinous processes of the last cervical and first dorsal vertebræ, a swelling, at first the size of a fourpenny-piece, and after a fortnight as big as a florin piece, tolerably flat and compact to the feel, with the skin over it a little reddened. The part was slightly sensitive to movements of the head, and still more so to pressure with the finger. The patient could not explain the origin of the swelling, which had been treated as rheumatic.

* Related by O. Bayer, 'Archiv der Heilkunde,' p. 105. Leipzig, 1869.

About six months after its appearance the swelling began to give way and decrease in size, and in May, 1867, had entirely disappeared.

In the meantime, in December, 1866, an elevation had formed on the left parietal bone, near its anterior angle. It was as big as a groschen at first, somewhat flat and compact, and tender on pressure. It then increased to the size of a thaler, and remained unchanged at the beginning of the severe illness to be described.

On May 5th, 1867, the patient, in mounting his horse, perceived a weight and clumsiness in his legs, although when he left his dwelling-place he had felt nothing of this. During his ride of one hour he felt as usual. In dismounting there was again heaviness and uncertainty in both legs, and now he retained this feeling; nevertheless the patient took a short walk, in which he was aware of more weariness than was common to him. Next morning the condition was somewhat worse; the legs could not be lifted out of bed without trouble, which was still more marked in standing and in walking, but especially in ascending stairs. His steps were taken as though with very tired legs. This weakness increased in the course of the afternoon, so that the patient mostly remained lying on the sofa. In spite of a quiet night, on the next morning the legs could only be lifted with striking difficulty, and it required especial exertion for him to raise himself in bed. To leave the bed was impossible; in other respects the patient had nothing to complain of. During the following day nearly complete paralysis of both legs declared itself. To rise in bed was impossible; active movement of the upper extremities was more and more difficult; till, finally, the arms could only be slightly lifted, and with the hands also light objects could not be laid hold of. Urination was laborious, and was obliged to be aided by pressure upon the hypogastric region. The action of the bowels was retarded, and required purgatives; the appetite vanished. Otherwise nothing abnormal was evident, and neither cramps

nor spontaneous contractions were to be observed. Indifferent treatment only had been employed.

At this stage the patient was transferred to the treatment of Prof. Wagner. The results of the objective examination were as follows :

Body long, of strong bone build ; considerable emaciation ; skin very pale.

Temperature, pulse, and respiration normal.

In different parts of the body the following abnormalities :

The swelling on the head as above described.

Deep clefts in the tonsils, especially the left.

On the chest, belly, and spine small white scars, which the patient referred to the previously existing exanthem. The upper extremities were only moved with much trouble and feebly, at the best, the fingers being scarcely able to grasp anything. The sensibility was not impaired.

The extended legs could not be lifted. In the knees feeble flexion was alone possible, in the left somewhat better than the right; yet the least pressure prevented this. Both feet could be moved a little to and fro. The toes of the right foot were capable of being moved very slightly, those of the left actively. The sensibility in the foot, leg, and thigh was considerably weakened ; in the left somewhat less than in the right.

The patient was not in a condition to raise himself in bed.

All passive movements could be readily accomplished.

The sensibility of the skin in parts of the body not especially named remained normal.

The conditions represented remained stationary under treatment for a week and a half.

From May 22nd the sensibility in both lower extremities improved gradually but evidently, from one day to another ; the power of movement of the toes also increased. At the same time the appetite improved.

In the night of May 30th—31st the patient experienced severe cardialgia, which disappeared on the evening of the 31st; the loss of appetite connected therewith vanished

after some days, whilst the swelling on the head manifestly lessened.

The paralytic symptoms in the legs and arms diminished uninterruptedly; the urine also could be slightly discharged; the sensibility, meanwhile, had become pretty normal. On June 5th numerous petechiæ of the size of lentils showed themselves on the toes of both feet. No trace of salivation. Mercurial eczema was apparent in different places after some days.

On June 10th the hæmorrhages had to a great extent disappeared. The improvement of the paralysis had made such progress up to this day that the patient could lift his arms, and also exercise some pressure with his hands. He could eat without assistance, and could raise himself, although with some trouble, in bed. The knees could be bent with some strength; the bladder and rectum acted spontaneously; nothing more was to be seen of the swelling of the head.

After many fruitless attempts, on June 21st the patient stood freely on the ground; with the aid of an attendant he could also move a little forwards.

A week later the patient walked briskly with the help of a stick.

On July 1st he no longer required the stick, and in the course of this month acquired strength so rapidly that, at the end of it, he had completely recovered.

The treatment consisted in the rubbing in of mercurial ointment. When the hæmorrhages appeared the body was washed, and the inunction was stopped for a few days. Iodide of potassium was likewise given in the beginning, and stopped on account of disorder of the stomach, which was treated with bismuth, soda, and morphia. In the first two and a half weeks in June short applications of faradisation were also made daily to the muscles of both lower extremities.

Strong, but easily digested, nourishment was allowed from the commencement. Careful rinsing of the mouth was especially observed during the inunction.

In this case the affection did not involve so high a portion of the cerebro-spinal axis as in mine; but if we imagine an extension upwards of the lesion to the medulla oblongata and pons Varolii, the resemblance would be perfect.

A case is related by M. Taffe* which bears some resemblance to these. A workman, æt. 25, who had been affected with pronounced syphilis six months previously, fell ill with diarrhœa, and in two days his lower extremities could not be voluntarily moved. On the third day his arms also were completely paralysed, and the bladder required the use of a catheter. In spite of mercurial inunction the attack ended fatally on the tenth day through paralysis of respiration. There was no autopsy. In this case, as in ours, the electric irritability was much lowered on the sixth day, in this respect differing from what has been observed by Erb and others in the acute ascending paralysis of Landry. I should add that there were no bedsores, or vaso-motor disturbances, nor was there any atrophy of muscles. It is remarkable that not only the skin reflexes, but the tendon reflexes are said to have been unaffected, and the mechanical excitability of the muscles was preserved. Only on two evenings did the temperature run as high as 100·4° and 100·8°

I may also refer to an interesting case of "acute ascending paralysis" recorded by Dr Arthur Fox, of Bath.† The patient was a prostitute, and there was a history of sore, followed by eruption and sore throat. In her case the symptoms, which began with pains in the legs and loins, included incontinence of urine, followed by loss of power in the legs, extending shortly to the arms, and causing death in about a fortnight. In several essential points that case differed from mine, especially in the presence of hyperalgesia and delirium, and the absence of symptoms referable to the nerves emanating from the pons

* 'Centralbl. f. d. med. Wissensch.,' 1879, p. 303.

† Vide 'Brain,' part vii.

and medulla oblongata. Very slight changes, if any, were found in the nervous structures post-mortem by Dr Shingleton Smith. "Sections of the cord," he reports, "in the cervical region seem to have some increase in the connective tissue elements. The grey matter took the staining fluid more readily than usual, and some of the cells have a contracted appearance. . . . In the absence of any other morbid change the intense coloration (by the staining fluid) is an indication of some minute and diffuse protoplasmic change such as would exist in the early stage of diffuse myelitis."

In my previous case (W. H—) I was inclined to suppose that a spinal pachymeningitis—an inflammation of the dura mater—lay at the bottom of the mischief, but from the symptoms presented by a patient who died of pachymeningitis some little time since (confirmed by autopsy) I am not now disposed towards that view.

I think there is a fair amount of evidence to show that cases of almost universal paralysis may prove to be dependent upon syphilis. As regards the nature and exact localisation of the lesion I do not feel able to speak with equal confidence. If we take the case of Thomas O— (which I have given in detail) it will be noted that the lesion, whatever it was, affected as well the sensory as the motor side of the cerebro-spinal nervous system, and that not only was the electrical reaction of the muscular system greatly diminished, but there was muscular atrophy. On the sensory side the symptoms were threefold: cutaneous anæsthesia and shooting pains, which invaded the head as well as the limbs, and loss of muscular sense. The reflex activity of the spinal cord both as regards the skin and the patellar tendon was suspended. On the motor side there was partial paralysis of all four extremities, of the muscles of the back, of the diaphragm, and lower intercostal muscles, of both seventh and both sixth nerves, and of the sphincters.

It is evident, from a consideration of this wide extent of disorder, that there must have been a lesion either in-

volving generally the grey matter of the cord, medulla, and pons, or else a meningeal change sufficient to exert compression simultaneously upon both the anterior and posterior roots of the nerves belonging to those portions of the cerebro-spinal system.

Now, we know that in acute myelitis of the anterior cornua the lesion is confined to a remarkable extent to the anterior horns, and it is usually rare, therefore, for any alteration of sensibility to occur in that disease. But here the affection of sensibility was quite as strongly marked as that of motility; and if we would ascribe the condition to a myelitis at all it must be to an acute central myelitis, a poliomyelitis involving both anterior and posterior cornua, and not only the horns of grey matter in the cord, but also their analogues in the bulb and pons, which represent physiologically the prolongation of the spinal cord into the cranial cavity. To say nothing of the fact that pathological anatomy, so far as I am aware, gives us no account of such a condition, it is to my mind improbable that such a complete recovery could take place as occurred in these cases from a lesion of this extent and gravity. It is even a question whether acute anterior poliomyelitis ever clears off without leaving paralysis in at least one limb.

There is only one alternative, and that is some change in the pia mater—a meningitis, or, as I think, perhaps more probable, a state of obstructed circulation in the vessels of the membrane, dependent possibly upon some syphilitic thickening of the walls of the vessels, accompanied by some exudation involving the roots of the nerves, irritating some of them so as to cause pain, and obstructing others by compression. But all this is purely hypothetical.*

* In a case recorded by Schulz and Schultze ('Archiv f. Psych.,' Bd. xii, p. 457, "Brain," April, 1882) ascending paralysis rapidly developed after a lengthened prodromal stage, and terminated fatally in four weeks. There was a syphilitic history. Diminution of electrical excitability and the reaction of degeneration were only observed towards the end of the illness.

The complete disappearance of the patellar tendon-reflex, and its return during the patient's convalescence, are very interesting points in the case, and, so far as I know, this is the first recorded instance of the disappearance and return of the phenomenon in a case of this kind. The circumstance lends us important aid in diagnosis in regard to localisation. We can confidently say from this, as well as from the muscular atrophy and the electrical reaction, that there must have been lesion within the spinal canal. A lesion of the pons Varolii would not explain these symptoms. It does not, however, enable us to clear up the difficulty as between acute diffuse myelitis and some changes in the pia mater—meningitis or otherwise. In the latter case, supposing that there were, as is possible, I think, a lesion of the roots of nerves sufficient to cause cutaneous anæsthesia, shooting pains, paralysis, muscular atrophy, and almost total absence of electrical reaction, it would be quite competent to obstruct also the reflex from the tendinous nerves. Nor, I think, does the interesting fact of the retention of sensibility—a hyperæsthesia, indeed—to temperature whilst touch, pain, and muscular sense were greatly in abeyance, help us to differentiate. The retention or exaltation of the sense of temperature alone, accompanied by some cutaneous anæsthesia as regards touch, is frequently seen in rheumatic perineuritis, and it is also present in two cases of hemiplegia under my care at the present time.

There were also slight paræsthesiæ and transitory vesical weakness. The post-mortem showed acute myelitis of the anterior cornua, of the lateral columns (especially of the pyramidal tracts), and in some places, but to a less extent, of the anterior columns. The morbid appearances were found through the entire length of the spinal cord, and in the lower part of the medulla oblongata. Though the evidences of disease were most marked in the localities named the whole cord showed signs of a degree of inflammation.

LECTURE XIX

PARALYSIS AGITANS: SHAKING PALSY

The disease "shaking palsy," or "paralysis agitans," some examples of which I shall have the opportunity of showing, was first regularly described by our countryman Parkinson in 1817. Parkinson was a member of the Royal College of Surgeons, and his 'Essay on the Shaking Palsy' presents so graphic and admirable a description of the disease that comparatively little has been left for subsequent observers to add to his account. In our time Charcot has also made the disease the subject of clinical investigation. The influence of these writers cannot fail to be felt at every turn by any one desirous of demonstrating the features of this remarkable affection. Parkinson's definition of paralysis agitans is as follows:

"Involuntary tremulous motion, with lessened muscular power, in parts not in action and even when supported; with a propensity to bend the trunk forwards and to pass from a walking to a running pace; the senses and intellect being uninjured."

He quotes the distinction between the kinds of tremor which was drawn by Sylvius de la Boe, who contrasts tremors produced by attempts at voluntary motion with those which occur whilst the body is at rest. Sauvages, in 1763, had distinguished the latter species (tremor coactus) by observing that "the tremulous parts leap and as it were vibrate even when supported;" whilst every other tremor, he observes, ceases when the voluntary exertion for moving the limb stops or the part is supported, but returns when we will the limb to move.

We still depend very much upon this point—the difference of circumstances in which the tremor shows itself, for our diagnosis of paralysis agitans.

The female patient, Mrs G—, is a widow, æt. 62. She sits with her chin resting on her chest, the lower lip and the neighbouring muscular structures about the chin being constantly in tremulous movement. There is a frown upon her brows. Her hands lie in her lap, the fingers being somewhat interlaced, apparently for the comfort of mutual support. When separated, the hands chiefly, and the forearms to a less extent, are in a state of constant tremor. If we count the to-and-fro movements, we find them to number from 160—170 in the minute. The tremor seems to be about equal in each hand.

When I speak to her and she answers, the shaking of the hand becomes more marked. It is not that the rapidity is increased, but the movements occupy a larger space. My hand placed upon her knees cannot distinguish any trembling of the legs.

She can stand and walk without help, but with short, "toddling" steps, the head being carried low and the body stooping forward. The face shows little or no expression, and wears a kind of fixed look. This attitude and general bearing it is very important to note carefully; they are characteristic of the disease. As she stands with her back towards me, if I pull her by the dress, with even the slightest force, she tends to fall backwards. When asked to lay hold of a cup which is offered to her, she puts out her right hand to take it, and whilst doing so the movements entirely cease. When, however, she has got hold of it and is told to squeeze the handle the movements return.

In another form of shaking palsy, that dependent upon insular, multilocular, or disseminated sclerosis, as it is variously called, "the tremor," as Charcot has pointed out, "only manifests itself on the occasion of intentional movements of some extent; it ceases to exist when the muscles are abandoned to complete repose." The cessa-

tion of tremor in this woman when she stretches out her hand to take the cup is not an exceptional circumstance. In many cases of paralysis agitans, it is true, the tremor will persist throughout the voluntary movement. But the present feature also is common enough, and has not escaped the observation of Parkinson, who contrasts it with what happens in that which he styles "tremor." "It is necessary to bear in mind," he writes, "that this affection (shaking palsy) is distinguishable from tremor by the agitation in the former occurring whilst the affected part is supported and unemployed, *and being even checked by the adoption of voluntary motion;* whilst in the latter the tremor is induced immediately on bringing the parts into action. Thus an artist afflicted with the malady here treated of, whilst his hand and arm are palpitating strongly, will seize his pencil, and the motions will be suspended, allowing him to use it for a short period; but in tremor, if the hand be quite free from the affection, should the pen or pencil be taken up the trembling immediately commences."

Charcot, also, when referring to cases of shaking palsy in which the tremor only occurs intermittently, says: "Singularly enough, it is just in such cases that the tremor shows itself rather when the limbs are at rest, and ceases when they are set in motion by the will."

There can be no doubt that in general the persistence of the movements of the hands when the muscles are abandoned to repose is sufficient to differentiate paralysis agitans from insular sclerosis. This is a rule, however, which is by no means without exceptions. In one of my cases, which an autopsy showed was an example of insular sclerosis, the tremor occurred as well in a condition of repose as on the occasion of intentional movement; on the other hand, I think I can call to mind more than one case belonging probably to the class of paralysis agitans in which the tremor was disposed to cease during muscular repose, and evince itself during voluntary movement of the limb.

The attitude of this patient's hands is another very noteworthy feature, and I would ask you to remember it particularly in reference to an examination we shall presently make of the other patient. It is, as Charcot has shown, very much the attitude of the writing hand, the fingers being inclined to an angle of about 45° with the metacarpal portion of the hand.

It is to be noted, however, that the fingers do not present the easy curve seen in the writing hand. Their position, it seems to me, is precisely that which is obtained by faradising the dorsal interossei—the first phalanx is flexed, the second and third being extended upon the first. The tremulous movements of the thumb laterally, as well as backwards and forwards, are marked more strongly than those of the fingers, and convey the idea of the patient rolling some object between it and the fingers. The fingers, *en masse*, tend to deviate towards the ulnar side. The extended position of the fingers would seem to indicate a tendency to muscular rigidity as well as convulsion, and this is indeed what we find when we come to examine other parts of the body. It is especially well seen in the fixed position of the head and neck; the chin can be lifted, but it soon resumes its position. We also learn from the patient that she has a great tendency to become cramped in her limbs. This rigidity, as we shall see in the other patient, may become the most dominant feature of the case. When we take an aggregation of symptoms—*Symptomen-complex*, I think the Germans call it—and give a name to the disease which is characterised by the association of these symptoms, we must take care to remember that in various examples individual symptoms will certainly vary in their prominence. It will often happen that a symptom which perhaps we have thought of but lightly—have "thrown in" among the rest, as it were—will in a certain case assert itself so strongly as to dwarf those others upon the presence of which we are accustomed to rely for making a diagnosis. On the other hand, a symptom which we have come to look upon

as essential may be actually absent. In this woman the presence of the peculiar tremor of the hands must be allowed to be, as indeed happens in the majority of cases of paralysis agitans, the most striking feature. I have mentioned the slight indication there is of a tendency to rigidity of the muscular system. In her case this is a symptom pronounced only in the most trifling degree. We can readily imagine, however, that this symptom might be the most dominant one in a certain case, and that the tremor in its turn might be absent or only slightly marked. There is a man in hospital who, I think, exhibits this kind of anomaly. But for the present let us return to the consideration of the disease of which we have a typical example in the person of this female.

More often than not the muscles of the face fail to show, to any great extent at least, the tremor which is so marked in the hands, but in this patient there is very distinct tremulous movement of the lips, especially the under lip. And in consequence of this inability to keep the mouth sufficiently closed, the saliva is almost constantly dribbling, much as we see it in cases of glosso-labio-pharyngeal paralysis. There is no difficulty, according to her account, in swallowing.

Mrs G— suffers much from flushes of heat, and her face is ever and anon covered with a deep blush. In many cases these symptoms are accompanied by profuse general perspiration, but this is not observed in the present instance. An "habitual sensation of excessive heat" was described, for the first time, by Charcot as characterising paralysis agitans.

There is a symptom in this patient which, according to my experience is a very frequent one, but which has not hitherto, so far as I know, received attention at the hands of any observer. Charcot, it is true has described the affection of speech, which is often observed, in these words: "There is no real difficulty of speech, but the utterance is slow, jerky, and short of phrase; the pronunciation of each word appears to cost a considerable

effort of the will. If the tremor of the body be intense, it may happen that the utterance is tremulous, broken, jolted out, as it were, like that of an inexperienced rider on horseback when the animal is trotting. However, in both cases we should recognise in this a phenomenon of transmitted tremor. Finally, the patients seem to speak between their teeth."

We observe something of the kind in this patient, but the symptom upon which I now wish to speak concerns not the articulation but the voice. The voice has a peculiar "piping" character. It is the conventional voice of the old man upon the stage. We all know that on the stage peculiarities of every kind are obliged to be accentuated for the sake of strong light and shade. When a very advanced period of old age has to be represented, the tone of voice adopted is exactly of the shrill piping character which we note in this woman, who, however, is only sixty-two years of age.

Shakespeare thus refers to this character of voice in connection with the sixth of his 'Seven Ages of Man':

> "His big manly voice,
> Turning again towards childish treble, *pipes*
> And whistles in his sound."

My interest in the study of this voice dates from the days when I was a medical pupil with the run of a large infirmary. I used to be frequently called upon to pass a catheter for a man of about sixty years of age who had an enlarged prostate. He was so severely affected with paralysis agitans that the movements of his arms were very embarrassing to me during the operation, and he had a typical senile piping voice which I have never failed since to remember in connection with the tremulous movements, and have often found associated in other cases of shaking palsy.

Since I first noted this piping note in cases of shaking palsy I have been interested in observing whether it was common in the exceptionally aged persons, not

afflicted with paralysis agitans, whom from time to time I have met. I have not found it to be so. Now, paralysis agitans, although, as Charcot has pointed out, it especially assails persons who have passed their fortieth or fiftieth year, has a slow march and little or no tendency to curtail longevity. Hence there is always to be found a certain number of persons afflicted with paralysis agitans who have arrived at a ripe old age, and who present this peculiarity of voice. I cannot help thinking that the conventional voice of age on the stage has originally been derived from the study of some old person affected with this disease, and thence handed down, as we know is the stage custom, to successive generations of performers.

A slight tremulousness shows itself in the patient's tongue. There is no affection of the cutaneous sensibility, nor of the special senses. The bowels are very obstinate, so that she is habitually obliged to take aperient medicine. She can retain the fæces, but has to be very quick in relieving her bladder, and requires to get up five or six times every night for the purpose. She suffers pain of an aching character in her limbs, but this is not in her case a prominent symptom. It is usually in a later stage than this, when the patient lies helplessly on a couch, unable to help himself in any respect, that pains in the limbs and joints are apt to be very distressing.

We do not observe in this patient a symptom which is very common in this disease, and often enables us to recognise a case at a glance. Parkinson describes it as "a propensity to bend the trunk forwards and to pass from a walking to a running pace." Although the patient presents the fixed head and stooping body, she does not hurry along as we so often see in such cases. There is no "festination," to use the term employed to designate this hurried march.

The amount of muscular power varies much in individual instances, and also in different stages of the disease. At first there may be but little notable weakness; but as time goes on a very distinct loss of power shows itself in the

limbs, and especially in those affected by tremor. In this woman the power of grasp is only feeble.

Before proceeding to consider other points connected with the symptoms of this peculiarly distressing disease, let me refer to the other patient. I am indebted to Dr Beevor's notes for many particulars of his case.

James H— is sixty-four years of age. He has been an upholsterer's foreman, has worked hard, and, according to his own account, has always been steady. He has been married thirty years, has had four children, and his wife has never miscarried. There is no history of phthisis, insanity, or fits in his family, but a doubtful account of hemiplegia in his mother. He says that he never had scarlet fever, rheumatic fever, or syphilis. He had "typhus" fever when young, and has suffered a little from gravel. His present illness dates from only two years.

At first he found difficulty in using his knife and fork and in putting on his clothes. About four months later he began to drag his feet in walking, and gradually power was lost more and more completely in all his limbs. Six months ago he began to "speak thick," according to his own description, and this has since become worse. He has not had trouble in masticating, and could always protrude his tongue. He says that he has sometimes choked in swallowing, but no difficulty has been observed in this respect since his admission into hospital. For six months past he has noticed that when his arms were raised they remained in the acquired position longer than was natural. He has not been able to walk for a year past, or even to get out of bed. His legs and arms have been stiff for the last nine months.

The patient is a very stout old man, who constantly lies in bed without occupying himself in any way. He cannot help himself at all. There is an aspect of marked mental hebetude, or at all events an extreme slowness of expression, so that it is difficult to elicit answers to questions about his history. The difficulty is partly also

attendant upon his mumbling speech, which is sometimes quite unintelligible. Yet we contrive to get, in process of time, though the task is laborious, a fair amount of information from him. The face wears a peculiarly stolid expression. When told to show his teeth he makes only a slow and feeble attempt; he can close his eyes, but cannot screw his eyelids up tight. The tongue is protruded straight although slowly, and it does not show any tremor. Cutaneous sensibility is nowhere affected. The patient has not suffered from pains in his head or body. The movements of the eye-muscles are natural. There is no nystagmus.

The upper extremities are thin in proportion to the lower, but the emaciation, if so it can be called, is symmetrical on the two sides, as is shown by the following measurements. Right forearm eight inches and three quarters; left forearm eight inches and a half. Right arm nine inches and a half; left arm nine inches and a quarter. The hands remain in a position of semiflexion, the fourth and fifth fingers more flexed than the second and third. The finger portion of the hand is much drawn over to the ulnar side—the thumb is applied against the forefinger. The attitude of the hands is exactly that of paralysis agitans, but there is no tremor whatever in the fingers. The two distal phalanges appear somewhat over-extended upon the proximal phalanx. The intrinsic muscles of the thumb and the interossei appear to be thin.

This man can flex and extend his fingers, but the movements are exceedingly slow. He can, in the same deliberate way, flex and extend (though not to the full extent) both elbow-joints, but there is very little capacity for pronation or supination. He can raise his arms straight up into the air and abduct them from the trunk, the shoulder-joints being less affected than the elbows.

There is a very curious circumstance to be noted in connection with the movements of his limbs. In response to my request he raises his hand or foot to some distance above his couch, and there lets it remain for

several seconds until, indeed, he is told to drop it; and if I lift one of his limbs, and place it any position, so it remains in a cataleptic fashion. When told to replace it, it is long before he makes any movement, and then the limb descends slowly. The muscles of the trunk appear to be weak; he cannot raise himself in bed, and, indeed, does not move at all—lying, if not disturbed, for hours together in the position in which he is placed.

As regards the lower limbs, they are not so much emaciated as the upper extremities, but the muscles are flabby. He can lift either heel very slowly off the couch, can bend either knee, but can only move the ankle-joints a very little. All the joints appear very stiff, and require considerable force to move them.

The patellar tendon-reflex is only just obtained. There is no ankle-clonus. The cutaneous reflex of the foot-sole is obtained on each side.

The right pupil is smaller than the left. They both react to light.

The heart-sounds appear distant; there is no murmur. As regards his mental state, in reply to inquiry he says that "he feels lost." He says "he wants to give his address," and gives 139, Bow Road, which is not altogether correct. He seems very obtuse, and scarcely speaks at all, though he is always apparently conscious.

The faradaic excitability of the muscles of the forearms is normal, that of the arms and lower extremities exhibits a very slight and unimportant lowering.

The action of the rectum and bladder is preserved. He has no difficulty in retaining his urine or fæces, but sometimes a little delay occurs in expelling the contents of the bladder.

There is no disposition towards the formation of bed-sores.

The ophthalmoscope shows no change in the fundus oculi.

These represent the principal symptoms of a condition which is manifestly of an unusual and obscure character. I have been led to conclude that the patient represents an

anomalous form of paralysis agitans, partly by the necessity of excluding other explanations, and partly by the resemblance of the patient's state to that which would be produced by an intensification of certain symptoms of shaking palsy. Let us first see how far his condition can be made to tally with that produced by certain other conditions.

The preserved electric irritability of the muscles enables us at once to exclude, what otherwise might be a not unlikely supposition, that the patient is suffering from a severe form of lead-poisoning. In such a condition the faradaic excitability would be very much reduced, or, still more probably, absent. Progressive muscular atrophy is excluded by the universal character of the lesion. As you are aware, that disease picks out groups of muscles physiologically associated in their action, leaving others intact. Here the affection is a general one of the whole muscular system, and, in addition to this, the mental condition, or, at least, the laborious slowness of movement in response to order which we observe in this man, forms no part of the history of progressive muscular atrophy. Bilateral sclerosis, which at first suggests itself, on account of the rigidity of the limbs, is excluded by the condition of the tendon-reflexes, which in that disease would certainly be exaggerated. So also would they be, though not to an equal extent, in Charcot's amyotrophic lateral sclerosis. In both these conditions, the one dependent on sclerosis of the lateral columns alone, and the other upon sclerosis of those columns along with lesion of the large ganglionic cells in the anterior cornua, the tendon-reflexes are intensified. For the same reason, cerebro-spinal insular sclerosis must also be excluded.

On the other hand, let us see what symptoms are to be found which accord with the view that the patient is affected with paralysis agitans. We have not here, it must be remembered, the opportunity of seeing him walk. That is a great loss, for the attitude of a patient with shaking palsy is, as we have seen, exceedingly

characteristic. "The patient," as Charcot says, "loses the faculty of preserving equilibrium whilst walking. In some we notice a tendency to propulsion or to retropulsion (in the case of Mrs G—, the tendency to retropulsion when I pull the skirt of her dress is well marked); without feeling any giddiness, the patient is in the first case propelled forward and, as it were, compelled to adopt a quick pace; he is unable without extreme difficulty to stop—being apparently forced to follow a flying centre of gravity." He adds: "A peculiar attitude of the body and its members, a fixed look, and immobile features should also be enumerated among the more important symptoms of this disease." Again: "The muscles of the face are motionless, there is even a remarkable fixity of look, and the features present a permanent expression of mournfulness, sometimes of stolidness or stupidity."

Although we cannot see this patient stand or walk, we have the opportunity of noting the fixed look and immobile features which singularly recall this description, and resemble what we observe in the other patient. Might not a widespread softening of the brain be attended with a similar physiognomy? I think it might. But it must be remembered that the mode of progress of the disease in this man does not accord with such a supposition. There is a gradually progressive loss of power and increasing rigidity in his members. When you have widespread atheromatous changes in cerebral arteries leading to thrombosis and softening, the paralysis which ensues, although it may easily involve all the limbs, does so not by slow and imperceptible degrees, but by leaps. A more or less sudden loss of power occurs in one limb, and after an interval of varying duration the same thing happens in regard to another. Moreover, in such a case again we should certainly expect the tendon-reflexes to be exaggerated—not lowered in activity as is the case in this man's knees. The same argument applies against the suggestion of the growth of a cerebral tumour, which

besides is additionally rendered improbable, though not, it must be allowed, impossible, by the absence at any time of pain in the head and vomiting, and the fact that no changes are discernible with the ophthalmoscope. But there are still two other circumstances which, although not absolutely conclusive, speak most strongly against the view that the condition described depends on a destructive intracranial lesion. The patient is able to retain his urine and fæces. If his helplessness were dependent upon extensive brain softening, it is in the highest degree improbable that the power over the bladder and rectum would have been maintained. I mention this as a result of clinical experience without being able to say what portion of the encephalic centres must be involved to induce this particular loss of power. It is a fact that in more or less generalised paralysis from cerebral softening we do constantly find that the evacuations cannot be restrained.

The second circumstance is that the patient, though he lies in the position in which he is placed, shows no disposition to the formation of bed-sores. It is true that he was placed on a water-bed as soon as he arrived, but the helplessness which we see had existed for months before his admission into hospital, and during that time he did not enjoy this advantage. But besides the expressionless features and fixity of looks, we have here two other symptoms of paralysis agitans of great importance. The first is the attitude of the fingers which, as we have seen, precisely resembles that which is to be observed in the female patient. Now this attitude is very peculiar. It does not occur in ordinary cases of hemiplegia or generalised paralysis from extensive cerebral softening. In such cases it is true the hand tends to assume a position of more or less strongly marked flexion, but *the fingers are also and indeed especially flexed.* Here, on the contrary, the forefinger and middle finger at least tend rather towards an attitude of over-extension. The phalangeal portion of the hand is besides inclined

towards the ulnar side. It was the look of these hands which first suggested to me the idea that the patient might be suffering from paralysis agitans.

Another symptom is the rigidity. A patient of Charcot's said that his joints appeared "soldered together." Benedikt suggests that the habitual rigidity of a certain number of the muscles undoubtedly contributes towards rendering movement laborious.

The muscular rigidity which occurs in paralysis agitans seems to me peculiar. It is not apparently a spastic condition like that, *e.g.* which we see in the late rigidity of hemiplegia, or in the sequel of myelitis with secondary degeneration of the lateral columns of the cord. It resembles to a remarkable extent the stiffness which results from disuse. You see this condition well marked in the muscles of a forearm which has been for some weeks confined to a splint on account of fracture. The muscles are feeble and stiff, but the stiffness can be overcome with tolerable ease by passive movements. There is no tension of the muscles in this case any more than there is in the case of the fractured arm, and here, as you see, they are actually flabby. Along with his rigidity, if such it can be called, is a condition which Charcot has called attention to—a retardation in the execution of movements. The patient performs movements with extreme slowness. "In relation to the faculty of speech," Charcot writes, "there is a comparatively considerable lapse of time between the thought and the act. One might suppose that the nervous influx cannot be set to work until after extraordinary efforts." Probably, also, I would suggest, the reception of impressions is likewise delayed.

The muscular stiffness is commonly most marked in the advanced stages of the disease. Charcot writes: "There are cases, though these are rare indeed, in which muscular rigidity is a symptom of the early stage of the disease, and a really prominent one. I have recently observed an example which belongs to this category. The patient had scarcely noticed the tremor, which, in fact, showed little

intensity in his case, and was confined to one hand. He already displayed, however, in a high degree, the peculiar attitude of the body and its members, the difficulty of movement and the characteristic gait." In a case under the care of Dr Gowers in this hospital, the patient, a female, exhibited all the symptoms of paralysis agitans except the trembling, which was barely perceptible. In this man there is an entire absence of tremor. But we do not know whether the symptom has always been absent. It it quite possible that there may have been a certain amount, perhaps too small to attract the patient's attention. However that may be, I do not consider that the total absence of trembling is any bar to our reception of this case as one of paralysis agitans. The remaining symptoms appear to me to be quite sufficient to enable us to relegate the case to that category. I may remind you of the analogy in this respect with what obtains in another disease of the cerebro-spinal nervous system—tabes dorsalis. The cases are numerous in which the symptom ataxy of gait, which gives the name progressive locomotor ataxy to the disease, is entirely absent.

It is very probable that such cases as this man's are not so rare as might be thought. Looking back to past experience, I am disposed to think that I have seen several such in workhouses and hospitals, where they have been classed with cases of softening of the brain.

The case of a man, æt. 67, who is now attending the hospital as an out-patient, is full of interest as affording a kind of connecting link between such a condition as this and the more typical form of paralysis agitans. The patient's appearance as regards his attitude and walk is very characteristic. The head is thrust stiffly forwards, the face expressionless, the eyes scarcely ever looking otherwise than straight in front. He walks with very short steps, holding the arm of a companion. His complaint is of "stiffness in all his limbs" especially in his left shoulder, and he says that there is also pain about that joint. If we lift one of his arms and place it in a certain

position, it will remain there for a considerable time, very much as is the case with the other man. The fingers of his left hand lie in the characteristic position which I have described, and there are very slight tremulous movements, especially of the thumb and forefinger. His aspect is one of pronounced mental hebetude, and he tells us that he finds himself, when awake, continuing the dreams which he has had when asleep. With all this it is to be remarked that his brother describes his mind as clear and intelligent. He will discuss questions, we are told, connected with electrotyping and other matters, with which his occupation as a jeweller has made him familiar, with intelligence and judgment.

But the converse to total absence of tremulousness may be observed. The movements may be so intensified as to occupy almost the whole attention. I lately saw the following case :

Mrs M—, æt. 64, a farmer's widow (April 26th, 1881). Twenty-seven years ago she began to get weak in the right hand, and thirteen years since her head began to shake.

Now as she sits she is constantly moving all over; her head is jerked, the arms, legs, and body writhing. She is constantly gasping in her breath, her face twitching—in an incessant state of distress. The larynx is prominent, owing to great action of the sterno-mastoids and trapezii. There is, she says, a "distressing feeling in her inside."

She is constantly obliged to be occupying herself. Whilst I examined her she was knitting.

Her daughter says she is better in herself than she was six years ago, but the movements are about the same.

She sleeps about four hours at night.

The general health is good.

She is "never ill." She always feels better when by herself. Any nervous excitement increases her distress.

There is constant snorting and sniffing.

Here is an example of another variety :

William W—, æt. 64, was admitted into the hospital

on May 20th, 1878, with paralysis agitans, which had begun five years previously.

He was an engine-driver who had never had a day's illness in his life. In the course of his work he had to crawl into very hot boilers to clean them out, and to this he attributed his illness.

His symptoms began with subjective coldness of the knees, shortly followed by shaking of the left hand, left leg, and then of the right hand.

There was almost constant shaking of the left hand. If he took hold of an object and held it firmly, the shaking stopped for a few seconds, but then recommenced worse than ever.

In this man there was a very peculiar condition. Whilst he was at rest there was no shaking of his legs. but if he stood up and attempted to walk, he remained for a time unable to start, his feet, however, beating the ground rapidly. All of a sudden this would cease, and he would start off at a fair pace, though he required some one to hold him up. This "marking time" action when he tried to walk had been observed about eight months. The patient, who was an engine-driver, appeared much struck with Mr Broster's suggestion, that it reminded one of the wheels of a locomotive failing to bite the rails when they are slippery with frost, and making, in consequence, ineffective revolutions.

Paralysis agitans is sometimes acute in its progress, and this, I think, is more often the case when the patient is comparatively young. I lately saw a man, æt. 38, whose symptoms dated from one year only. They had commenced with hesitation of speech, which was followed by shaking, first of the left then of the right arm, and when he came to me he presented all the symptoms in a marked degree.

There was a patient in the hospital, a year or two ago, who was still younger. I gather the following account from Mr Broster's notes, taken on his admission:

Benjamin L—, æt. 22, was admitted into hospital on

April 30th, 1879. His occupation is that of a butcher, and for five years past he has been in London, having previously lived in the country. He looks about his age. Somewhat apathetic in manner, he yet complained of irritability of temper, especially when exposed to noise. His memory is good. He has no headache nor giddiness. The speech is thick, and somewhat indistinct, the articulation being hurried, and the words run together. The tongue showed marked tremor. Saliva dribbled from his mouth at times. There is difficulty in swallowing, and he is obliged to have his food chopped up fine.

His face wears a fixed look. He cannot frown, and can only show his teeth feebly by voluntary movement. During involuntary movement, on the other hand, as when he laughs, he shows them very well. He has no emotional instability. As he sits, there is marked shaking of the head and upper limbs. The arms—especially the right arm—are in a continual state of rather violent agitation, which is increased by excitement, and is least when he is left quietly to himself. There are also involuntary uniform movements of the legs and feet when he is seated. If he lifts his heel off the ground, so as to rest solely on the ball of the foot, there is a rapid movement similar to foot-clonus. No foot-clonus can be elicited by sudden passive dorsal flexion of the foot. The patellar tendon-reflex is normal—free from exaggeration. In his walk he inclines forward, and "scuffles" along, but not so hurriedly as is often seen in these cases. He complains of stiffness in his arms and legs.

When he picks up anything with either hand there is a momentary lull in the movement, and he takes hold of it quietly and steadily. Immediately afterwards the movements recommence. He is very easily pushed forwards or backwards, however much he may try to resist this.

His breathing is of a half-sobbing character.

There is slight lateral curvature. No spinal tenderness.

Now, in this case, it was only eleven months before these notes were taken that the patient had first noticed a slight shaking of the right hand. This very gradually increased, and in three months he was forced to give up his occupation. Two months later he noticed that his left hand was beginning to shake like the right, and after two or three months more his legs also became involved. They shook whilst he sat, and all the more, he says, if he bore his weight upon his toes. A month or two after this, his swallowing became affected, and his mouth, according to his account, was always full of saliva. About the same time his articulation became embarrassed.

This man acknowledged a course of life of the most dissipated character. He had lived freely and drunk heavily. From the age of sixteen to twenty-one, he had masturbated two or three times a day; and from twenty-one to twenty-two, about the time of commencement of his symptoms, had indulged to the greatest excess in sexual intercourse. He had never had syphilis, but had once suffered from gonorrhœa. The family history was a healthy one.

It is worth noting that, in the other case—patient æt. 38—there was a history of excessive sexual intercourse immediately preceding the commencement of the symptoms.

The patient, William V—, who is now attending the hospital, is affected with shaking palsy, which commenced in his left arm. By the dynamometer, the grasp of the right hand measures 50, that of the left 40.

The movements in this case are of a somewhat unusual character. They are much larger in extent than is commonly seen, so that as the patient sits he flaps his knees with both hands noisily and roughly. His legs also are affected with coarse movements. His walk is characteristic, the body being bent forward, and the head maintaining a "set" position.

If we ask him to pick up a pen off the table, his arm is still whilst he is stretching it out to grasp the object;

but when the hand closes upon it the movements begin to recur with great energy.

Another of our out-patients, a female, presents symptoms which are at first strongly suggestive of this disease, but I do not think she is an example of it.

Elizabeth B— is a widow, 66 years of age, who was strong and well, and had nothing to complain of till ten weeks ago, when she woke up one morning retching and vomiting, and found that she had lost power to a certain extent in the left arm and leg. She could not keep her arm still, and her leg also was constantly shaking. A very aged neighbour who came to her, said, "Don't touch me, or you'll pull me down," the shaking was so violent. She knocked the left knee against the right so violently that it was quite bruised. She remained in bed three weeks, during which the movements were very violent, but afterwards they became less so.

Now, we observe that as she rests her hand on her knee it is steady, but when she tries to pick up things from the table there are rhythmical movements—flexion and extension movements of the hand numbering about 180 in the minute. The attitude of the hand is not that of paralysis agitans. A friend who comes with her says that she is sometimes obliged to throw the bed-clothes off, she complains so much of heat. Since she has been in the room her face has come over in a red flush several times.

The wrist-reflex is exaggerated on both sides, but especially on the left, whilst the patellar tendon-reflex approaches to a clonus on the left, and is in some excess also on the right side. She walks with short toddling steps, and is compelled to use a stick, otherwise she falls. The left knee never shakes now, but sometimes in walking it "catches up." The dynamometer shows a grasp of 40° with the right, 22° with the left hand.

She has never had any numbness in the left hand, but there is often aching pain in the forearm, especially towards night.

We do now and then observe a state of tremor in the paralysed limbs of a hemiplegic patient; but so far as I have seen, it is very rare to find such an amount of tremulous movement as we see in this woman. Here the tremors closely resemble those of paralysis agitans, and the case might easily be mistaken for one of that disease.

According to Nothnagel, the condition has been observed in connection with lesion in the following localities:—1. The internal capsule in its posterior segment. 2. Optic thalamus. 3. The foot of the corona radiata. In these situations it is, he points out, that lesions are most apt to be accompanied by hemichorea and athetosis, with which it is supposed these tremors are nearly allied. It is not certain whether the condition occurs as a result of cortical lesions.

This patient evidently had a sudden cerebral attack, accompanied by vomiting and loss of power, in the arm and leg of the left side. In the suddenness of its origin the disease contrasts as strongly as possible with shaking palsy.

The exaggeration of reflex, amounting to foot-clonus on the affected side, is also opposed to the diagnosis of that disorder. The attack has, probably, been of an apoplectic character, the exact situation of which, however, I am not prepared to indicate with any confidence.

Passing over the morbid anatomy of paralysis agitans, about which nothing conclusive is as yet established, I would refer to one or two points in regard to the symptoms.

If we examine the upper extremity in a case of true shaking palsy, we are struck at once by the circumstance that apparently the small muscles, those concerned in the most delicate movements, are those which suffer most. The attitude of the fingers is that which would be produced by alternate contraction and relaxation of the fibres of the interosseous muscles. It is unusual in other conditions to find predominance of affection of such muscles over

the larger ones. In the climax of an epileptic seizure the hand is clenched. The contraction of the large and powerful flexor muscles lying on the anterior surface of the forearm overpowers completely that of the small interossei. So also in the converse of convulsion—in hemiplegia, when the paralysis is complete the fingers lie passively in an attitude of semiflexion. There is only one condition, I think, in which the position of the fingers nearly resembles that which they occupy in shaking palsy, and that is the singular disorder called tetany. It is not easy to see any kind of association between these disorders, but the point is perhaps worth noting.

As regards the piping senile voice, to which I have already referred, I have lately met with an instance in private practice which has afforded me an opportunity of investigation. The patient, William B—, æt. 57, has the characteristic fixed look and the piping voice. His right hand, ever and anon, but not constantly, is shaken to and fro. The left also is shaken, but much less. He feels very weak in the right arm, and says that he has scarcely any use at all in it.

He does not come over in flushes of heat or perspiration. Sometimes, he says, he can scarcely talk. This, he thinks, is owing to something in the throat. His wife has noticed the alteration in the voice.

About two years ago he began to feel loss of power in the right arm—that hand would drop occasionally from the wrist, so that he could not depend upon holding things. The loss of power has gradually increased, especially of late. Within the last few weeks he has been inclined to trip with the right leg, which feels, as it were, shorter than the other.

Lately, for a fortnight past he has been liable to giddiness, and on one occasion nearly fell down.

He does not suffer from sickness. He has usually had good health. His appetite is good; he sleeps well; the bowels act.

He has a difficulty in holding his urine, and would wet

himself if he were not very quick. He gets up two or three times in the night to pass water.

Memory is good. He would rather be by himself, and avoids noise and company. There is a fixed look of the face with a half-frown upon it, and the head is held stiffly, the eyes usually looking straight in front.

The tendon-reflex at the wrist is equal on both sides, and not abnormal.

At my request Dr Felix Semon was good enough to examine this patient with the laryngoscope. His report is that there is neither motor nor sensory paralysis nor loss of reflex sensibility to be detected in either pharynx or larynx, and that both these parts are quite free from traces of previous specific or other disease.

It is possible that the piping voice, which I have likened to that adopted by an actor who represents advanced age upon the stage, may be due to some functional peculiarity in the action of muscles influencing the vocal cords. Its study might possibly help to throw light upon the cause of the "falsetto" voice on which physiologists are not, I believe, agreed.

LECTURE XX

SPASTIC PARAPLEGIA FROM MYELITIS

Before presenting to you a patient who is now in hospital suffering from loss of power in the lower extremities, with a spastic condition of the muscles, I wish to call attention to a few points bearing generally upon this pathological condition. A form of spastic paraplegia has been described by Seguin, Erb, and Charcot, which comes on gradually, and is apparently, though this is not by any means certain, protopathic in its origin.* It is usually associated with systematic sclerosis of the lateral columns of the cord, and this sclerosis, so far as can be judged, is not a secondary degeneration due to a lesion higher up. Or there may be insular sclerosis of the lateral columns. It is apt to attack persons between the ages of thirty and fifty years, and is attended by loss of power in the extremities, accompanied by stiffness, and by exaggeration of the tendon-reflexes. There are no disorders of sensibility, and the functions of the bladder and rectum are commonly, though not invariably, unimpaired. This is not the form of which I shall show you an example, and I need do no more therefore than mention it. A much more common form is that in which, as a result of a lesion of the cord from compression, occurring either in the course of Pott's disease, or from a tumour, or from diffuse myelitis with or without meningitis, there is a secondary

* A case recorded by Dr Dreschfeld, of Manchester, in the Pathological Section at the International Medical Congress, London, 1881, is the only one, I think, in which microscopical examination has tended to confirm the protopathic origin.

degeneration of both lateral columns below the point of lesion.

You will remember that Waller found that whilst section of the anterior root of a spinal nerve was followed by degeneration which extended to the periphery, division of the posterior root between its ganglion and the spinal cord was attended by degeneration of that portion of the root only which remained attached to the cord. It appeared to him that whilst in the first case the spinal cord acted as a trophic centre of the nerve, in the second case it was the ganglion, not the cord, which played this part. We owe to Türck the discovery that in the spinal cord itself something occurs which appears to be analogous to Waller's observation. A destructive lesion of the cord involving both grey and white matter causes degeneration, which is almost exclusively limited to the nerve fibres of the lateral columns below the point of lesion, and of the median portions of the posterior columns above the point of lesion. Now, we know practically nothing as regards any symptoms dependent upon change in the median portion of the posterior columns. We cannot doubt that there are symptoms, although at present they have not been definitely associated with the lesion; but as regards the degeneration or sclerosis (for that is the form which the degeneration takes) of the lateral columns, experience shows that this condition is attended with loss of voluntary power, a spastic state of the muscles, and excess of tendon-reflexes. You will see presently why I consider the patient who is now before you to be an example of this form of spastic paraplegia, and not of the primary form described by Seguin, Erb, and Charcot. Louisa P— is twelve years old, and she was at school when her illness began in February, 1880. Her father is dead; her mother, I find from Dr Beevor's notes, is said to be affected with shaking palsy. There is no phthisis in the family, and the girl has never had either scarlet or rheumatic fever. Without any evident cause, she found her legs giving way under her, and suffered from severe

pains in her knees and legs, "like a needle running into them." Her legs would draw up spontaneously. She lost power so rapidly that in a fortnight she could not move her legs at all, and could not feel in them, or in her body below the waist. At first she could not turn in bed, and, there was pain in her back when turned by others. A month after the onset the urine and fæces were passed involuntarily and without her knowledge. This trouble lasted three months, when she gradually began to regain consciousness of their passage, and later was able to restrain their discharge. The legs could not be moved by her till three months from the time of onset, and she has never been able to stand since the attack. The sensibility of the skin began to return more rapidly than motor power, within a month of the onset.

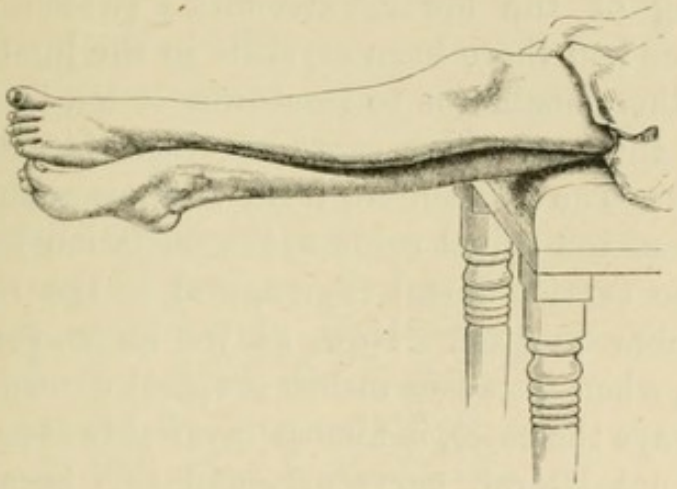
Within a week or two of the time of attack she suffered from bedsores, of which the scars are still plainly visible. There is one at the bottom of the spine, just above the cleft of the buttocks; and another, a puckered cicatrix, near the left trochanter.

Pausing here for a moment, let me note that this history is a perfectly different one from that to which I have just referred as belonging to idiopathic spastic paraplegia. The radiating pains in the lower extremities, the inability to be turned over in bed without pain in the back, point especially to spinal meningitis; whilst the total loss of power and absolute anæsthesia, with paralysis of the sphincters and occurrence of bedsores, leave us in no doubt that there was also myelitis. She has suffered, indeed, evidently from meningo-myelitis of a somewhat acute form. Her present condition is a consequence of this lesion of the cord.

Her legs, as you see, are not much wasted. They are very stiff, and resist movement. The feet are pointed in a position of rigid plantar flexion from permanent contraction of the sural muscles. On the other hand, the last phalangeal joints of the toes are strongly dorsal-flexed. The legs, when left to themselves, tend to cross

each other. She can lift either heel off the bed, and can flex and extend the knee- and hip-joints. If, when the knee is flexed, I take hold of the leg and gradually bring it to a position of extension, it moves smoothly and easily

FIG. 21.



till that position is nearly reached, when it suddenly becomes rigidly extended, and a good deal of force is requisite to bend the knee again. This phenomenon, which calls to mind the action of the blade of a penknife when you open it, has been styled by German authors "clasp-knife rigidity." The result of this spastic condition is that the patient cannot stand alone. You will note that slight taps upon the patellar tendon, or even on the knee-cap itself, cause excessive knee-clonus. On the other hand, if we try to obtain ankle clonus we fail. This failure is simply owing to the permanent contraction of the sural muscles, which renders the movement impossible. For the same reason tickling the sole of the foot is not attended by dorsal flexion of the foot. There is a slight loss of cutaneous sensibility in the right leg. The action of the sphincters is normal.

We have here, then, at the present time, the loss of power accompanied by a spastic state of muscles and exaggeration of tendon-reflexes, almost entire absence of sensory disturbance, and functional integrity of the sphincters, which form the essential symptoms of what is

commonly called Erb's spastic paraplegia. The case is, however, removed from that category, because the condition is manifestly a consequence of myelitis. It depends upon a degeneration of the lateral columns of the cords starting from the point at which the cord was inflamed. The complete immunity of the arms on the one hand, and the absence of wasting of the lower extremities (which must have taken place had there been myelitis in the lumbar region) on the other, enable us to locate the lesion in the dorsal region of the cord. Its cause of origin is not, however, so certain. The girl suffers from very pronounced lateral curvature of the spinal column, the deviation being to the left in the cervico-dorsal region, and to the right in the dorso-lumbar portion. Now, as we know, caries of the vertebræ, when it causes manifest spinal deformity (which is not always the case), is almost invariably associated with angular, not lateral, curvature, and this because of the bodies of the diseased vertebræ collapsing. But it is not impossible that in this case there may be caries (but slightly advanced), which has started the inflammation of the spinal membranes and cord, especially as I find that the girl complains of pain on percussion over the lower portion of the dorsal region. I do not feel at all confident that the cause is what I have suggested, for her early symptoms were more acute and rapid than is ordinarily seen in Pott's disease, but as in any case support to this curved spinal column can only be beneficial, I purpose to have her swung and encased in a Sayre's jacket.

Let me now give you some details of another case of spastic paraplegia which we have lately had in the hospital.

A. B—, a young married woman, of fair complexion and not unhealthy aspect, was brought to the hospital on June 15th, 1880, unable to stand, or, indeed, to move her legs. The following notes of her case at the time of her admission were taken by the resident medical officer. She is twenty-seven years of age, and has been married for nine years; has never had children,

but about three years ago miscarried at two months. There is no family history of any nerve disorder. She thinks she had rheumatic fever four years ago. There is no history of syphilis. Two years ago she fell down a flight of stone steps and knocked the back of her head. She was stunned at the time, but felt no pain in the back after the accident. Her present illness came on gradually, beginning about ten months ago. She first began to stagger in walking, and her legs would give way under her. She suffered very great pain in the back for two or three months. It was so bad that she could not rest at night. It hurt her very much to turn upon her side. She does not remember that touches upon the spine were painful. The pain in the back was between the shoulders for about a month, and then gradually extended all down the back. At the same time she had shooting pains down the legs, the right especially. The legs would jump up during the pains. Two months later she felt numbness and coldness in her right leg, and could not tell whether the foot was on the ground or not. For about six months she could not feel at all when it was pinched. At the same time there was numbness in the lower part of the back, and in the hypogastric region. A little later than the right, the left leg also gradually failed and became numb and cold, but was not so bad as the other, and she never completely lost sensation in it. She has not been able to walk, or even to stand, for three months past. About three months ago she complained of a tightness around her body, but this has improved. Five months ago she found that she could not hold her urine more than three hours, and she was obliged to obey immediately the call to micturate. About two months ago the urine began to come away from her involuntarily. For about two weeks before it began to dribble she had lost power to pass water, and required hot baths to assist her. The bowels are always constipated. On one occasion they were moved without her knowledge.

Such was the history obtained at the time of her admis-

sion, when her condition was noted as follows :—The head and upper limbs are normal. The spine presents a very slight lateral curvature in the lower dorsal region to the right. There is no angular curvature. There is tenderness, on percussion, over the third lumbar vertebra, and to a less degree over the second and fourth. The application of a hot sponge in this region causes her to wince more than when it is applied to other parts of the column, At the lowest part of the spine there is a commencing bedsore with spots around it. The legs are rather thin, but the thighs appear fairly well nourished, and there is no difference, in this respect, between the two limbs. She cannot move her legs or toes at all.

As soon as the legs are passively moved, or an attempt is made to flex the joints, the muscles of the legs contract strongly. Much force is required to flex the knee-joints, which present clasp-knife rigidity. The patellar tendon-reflex is much increased on both sides. There is ankle-clonus on each side. The cutaneous reflex in the soles is present, but sluggish. The cutaneous reflex of the hypogastric region is not obtained ; that of the epigastrium is obtained but faintly. The cutaneous sensibility does not appear to be much altered, and she indicates correctly which toe is touched. The reaction of the muscles of the lower extremities to induced currents is practically normal. The patient can hold her urine for an hour, and then if she does not answer the call at once it is passed involuntarily. Her urine contains a small quantity of albumen. The bowels are constipated. The heart sounds are somewhat distant ; there is no murmur. The ophthalmoscope shows no alteration in the fundus of either eye.

She was placed on a water bed and treated with iodide of potassium in twenty-grain doses.

A week after admission the following note was made :—Patient to-day held her urine for three hours and a half, the longest time during which she has been able to retain it since the beginning of her illness. She complains of pain in the lower part of her back and aching pains in the left

knee. On June 29th she moved her right leg for the first time for three months.

On July 4th it is noted that the patient has moved the little toe of the left foot. She says that her legs do not draw up so much, and are not so stiff as they were, but they still jump a good deal at night. She retains urine now for three or four hours, and when she feels the desire to pass it she can control it for a longer time than had lately been possible. In the middle of July the patient was able to cross her right foot over the left voluntarily, to flex the toes a little, and to invert the left foot, but she could not extend or abduct the left ankle-joint. About this time she had a febrile attack, with pain in the chest, furred tongue, pains in the lumbar region and abdomen, extending to the legs and knees. There was tenderness on deep pressure over the abdomen. The attack passed off, and at the beginning of September it is noted that the patient can lift both heels off the bed; the right better than the left. She can flex and extend the left ankle pretty freely, and can invert and evert it, though to a less extent. The right foot cannot be moved so freely as the left. She can flex the right knee readily, and if it is supported she can extend the leg upon the thigh. She cannot always flex the left knee, but when it is passively flexed and the thigh supported she can raise the heel off the bed. The limbs are much less liable to spasm. There is still much increase of the patellar tendon-reflex, but the ankle-clonus, which is still readily obtained in the right foot, is with difficulty induced in the left. The cutaneous reflex is obtained in both soles. The patient can stand with assistance, and even walk a little between two supports. A burning pain is complained of in the lumbar region of the spine, and percussion there is painful.

November 2nd.—She can walk with the help of one person. The feet are put down with caution, and there is a great tendency in the toes to curl upwards in over-extension. The patellar reflex is excessive in each leg.

Ankle-clonus persists in each foot, most marked in the right. There is no trouble whatever with the bladder. Little or no pain in the back. The dose of iodide on June 29th was increased to thirty grains, which she has taken, with an interruption of a fortnight, till the present time. The water bed was used for rather more than two months.

On November 24th ankle-clonus ceased to be obtainable.

I hear from Dr Beevor that the patient, who left the hospital a week or two ago, is now able to walk up and downstairs.*

The resemblance of this case to the preceding one is very striking. In each there was pain in the back on turning over from side to side—a symptom which is peculiarly characteristic of spinal meningitis, as also were the shooting pains and spasmodic contractions of the legs with which each patient was affected. In this patient the bladder alone was paralysed, the rectum being almost spared. She presented a commencing bedsore in the same situation which was occupied by the deep scar of a healed sore in the other patient, its position being at the bottom of the spine, just above the cleft of the buttocks. It is in the sacral region, as Charcot has shown, that the bedsore of spinal origin is peculiarly apt to occur, that which is of cerebral origin (as happens occasionally, for example, after apoplectic seizures), being located on the buttock of the same side as the paralysed limbs. These symptoms, along with the complete paralysis of motion and sensation, with subsequent rigidity, leave us in no doubt that here, as in the other case, there was inflammation of the substance of the cord as well as of its membranes.

You will have recognised, in the description of the

* The patient attended at the hospital on January 25th, 1882. She told us that she was on her feet all day long, and walked three or four miles daily without a stick or support of any kind. The tendon-reflex was still excessive.

spastic condition of her lower extremities, the close resemblance which the case bears in its advanced stage to that of the young girl who has been before you. But there are two very interesting points of difference on which I will say a few words. In the first place this young woman began almost immediately to improve when she came under treatment by iodide of potassium, and a continuance of the same drug in increased doses has resulted in what is practically a cure. The other patient, who has been but a short time in hospital, has as yet manifested no signs of improvement under similar treatment. In the second of the cases related I am disposed to refer the cause of the meningo-myelitis to syphilis, although there is little or nothing in the general history to confirm the diagnosis, the only circumstances which tell—and these but faintly—in this direction being the unproductiveness of marriage and the miscarriage at two months. But I have long expressed the opinion that certain lesions of the nervous system, when taken along with their behaviour under specific treatment, afford evidence of syphilis which is as strong as the coppery hue of one skin eruption or the serpiginous character of another. The symptoms point to myelitis by compression, a lesion which, as far as my experience goes, when it is not due to Pott's disease or injury, is much more often consequent upon syphilitic disease of the membranes of the cord than anything else.

There is another point of great interest in this case. You will have observed that under treatment in the hospital the patient has to a very large extent recovered from the spastic condition of muscles which was so strongly marked at first. *Pari passu* with this the exaggeration of tendon-reflexes has been very greatly reduced, so that at the present time ankle-clonus is no longer to be obtained. Now, this exaggeration of tendon-reflexes we know to be associated with sclerosis of the lateral columns of the cord. Have we, then, by treatment succeeded in removing the overgrowth of connective tissue,

which, with accompanying atrophy of nerve fibres, essentially constitutes sclerosis? I do not think that this is at all probable, nor indeed is such a supposition necessary. I have in a former lecture described a case of hysterical rigidity of the lower extremities, in which, when the patient was suddenly cured by the administration of ether, ankle-clonus, which had been readily obtainable, could no longer be brought about. I pointed out also that exaggeration of tendon-reflexes was a very frequent condition in hysteria. Only a day or two ago I had to see a young lady suffering from hysterical mania, in whom I found the reflexes not only in the ligamenta patellæ, but also in the wrists, as strongly marked as they are seen to occur in spastic paralysis. It appears to me, as I have before remarked,* that anything which interferes with the transmission of motor impulses from the cerebrum down the lateral columns of the cord will have the effect of exaggerating the tendon-reflexes.† The explanation of this appears to be that in this way the balance which normally exists between the reflex function of the cord and the controlling influence exerted from above (which is transmitted downwards by the lateral columns) is disturbed, and the reflex function, therefore, is exaggerated. Suspended transmission of motor impulses appears capable of causing results similar to those which arise from their mechanical interruption.

Now, sclerosis cannot occur suddenly. You cannot have a mass of fibrous tissue formed without some preceding change in the blood-vessels of the part. There is probably a hyperæmic condition, with effusion. One can readily imagine that such a state, by causing compression of nerve fibres, may interfere with their normal function,

* Page 32.

† Dr Hughlings Jackson has reported a case in which he obtained ankle-clonus in a patient immediately after the occurrence of an epileptic fit, and Dr Beevor informs me that he has since had a similar experience in the case of an epileptic patient of mine, now in the hospital. In both cases the phenomenon did not persist.

so that the propagation of motor impulses is interfered with. Exaggeration of the reflex function of the cord would thus be brought about by interference with the arrival of controlling motor impulses. At the same time there is no reason to doubt that such an effusion might disappear under appropriate treatment. And this is what, I suppose, has happened in the case of this young woman.

Richard W—, a big burly man, who lay in the Extension Ward, and was discharged last July, presented a good example of this spastic paraplegia which is so apt to follow myelitis. The notes are by Dr Beevor. He was forty-three years of age, by occupation a country butcher, and was admitted into the hospital on April 7th, 1880, with paraplegia.

He had always lived well and been well clothed. He is married; his wife has had no children and no miscarriages. His father died of old age, his mother of consumption. There is no history of any nervous disorder or insanity in the family. Patient had never suffered from rheumatic or scarlet fever, nor from syphilis or gonorrhœa. When an infant he had smallpox.

About May, 1878, five months before his present illness began, he fell one day from the top of a hay-cart, about fifteen feet, to the ground. He was hurt across the back, and felt much shaken, but was not disabled. Next day he felt stiff but had no pain. About that time he was suffering from sciatica, from which he had not altogether recovered when he was taken ill. At the time of the fall he weighed about fifteen stone.

Three days before the onset of his present illness he got wet through and felt very shivering and cold. He began to be ill whilst hanging up meat. He felt numbness in the right arm and leg, and then in the left leg. There was a sharp pain also in his back, which ran up to the back of his head. He sat down and found that he could not move his arms or legs. In this helpless state he was put to bed.

He did not lose consciousness. There was no loss of power in the face.

For three months he could not pass urine, and it was drawn off by catheter. After that his urine used to dribble from him without his being able to restrain its passage, and this continued till a month before admission. His bowels were much constipated and have been so ever since. At first he lost sensation in both upper as well as in both lower extremities, and in the trunk as high as the waist, but he says that the remainder of the trunk was not affected, nor was his face.

He began to move his arms three weeks after the attack, the fingers first, and in five weeks' time they had become as well as they are now. He did not move his lower limbs till October, 1879. The toes of the left foot were moved first. The thighs were the last to be moved. The left limbs began to improve a month before the right. Sensation returned in the arms at the same time as the movement (in about three weeks after the attack), and first returned in the palm of the hand, and the whole hand regained sensibility in about five weeks' time. The lower limbs recovered sensibility only in October, 1879. Previous to this he had felt tenderness and shooting pains in his limbs.

From the waist downwards there had been a numbed feeling, as though a tight broad bandage were applied to the front of the abdomen, and a string went from the navel to the bladder. On the left side the feeling had been worse than on the right.

A month after his attack, a number of small spots came on the skin of his right hip and ran together, forming a large sore. Four months after, the bone was exposed, and there was a great discharge. The wound began to improve in November, 1879.

On admission the patient's general health was pretty good. He slept well, did not suffer from headache, and had a good appetite. With the right hand he could grasp with a force of 22 kilo., with the left 11 kilo. There

was no sign of paralysis about the arms. The lower limbs did not appear to be at all wasted. The muscles were very firm. No difference was perceptible in the aspect of the two limbs.

He could stand with help and just managed to walk, but whilst doing so the legs were thrown into clonic contractions. When lying on his back he had some difficulty in raising the right heel from the bed with the leg extended—the left heel he could lift fairly well. He could flex both his knees very well—the left better than the right. We found it impossible to flex the left knee-joint against his resistance.

The patellar tendon-reflex was excessive on both sides, but especially on the right, when a reflex clonus of the limb was produced. Ankle-clonus could be readily induced in both limbs, but especially in the right.

The skin reflex of the soles of the feet was excessive; so also was the abdominal reflex, especially on the right side.

There did not appear to be any marked loss of cutaneous sensibility in the lower limbs. The upper limbs and face appeared normal. The sounds of the heart were healthy. The urine contained no albumen, but was ammoniacal. When the bladder was full the urine ran away. He had no power to pass it by voluntary action. The bowels were always constipated.

The excitability to faradaism of the muscles of the thighs and legs was normal.

The sore on the right hip existed on admission as a red, rough patch, about six inches by five, discharging a thin serum, which formed a crust upon the surface.

He was treated with large doses of iodide, and was discharged from the hospital on June 21st, in much the same condition as on his entry.

The nature of the lesion in this case is not so clear as its probable situation. The fact that at first the arms were involved as well as the legs shows that either the lesion was a localised one above the origin of the brachial

plexus, or a diffused one involving not only that part of the cord but also the dorso-lumbar portion. Now, the fact that when the original disorder cleared off the arms perfectly recovered, whilst the leg remained paralysed and in a spastic condition, is sufficient to negative the first of these alternations. The lesion could not have been a localised one above the origin of the brachial plexus. It must, therefore, have been a diffused lesion, and whilst that part of it which occupied the cervico-dorsal region of the cord entirely disappeared, repair was incomplete in a lower portion, and the resulting destruction of grey and white matter was followed by descending degeneration in the lateral columns, which gave rise to the spastic symptoms. There had probably been a diffuse myelitis of acute character involving more or less generally the substance of the spinal cord. It had not been confined to the grey matter of the anterior cornua, for sensibility had been profoundly affected, but it had passed away very much as the lesion clears off in anterior poliomyelitis (infantile paralysis) from some portions, whilst a permanent destructive change was left in others. It seems to have involved more or less seriously the whole section of the cord, for the descending sclerosis in the lateral columns apparently requires that both grey and white matter above the point of commencement of the degeneration shall have suffered a destructive change. Whilst the upper part of the cord recovered from this myelitis a permanent change was left, a destruction of tissue in the dorsal portion of the spinal cord, the part where it is thinnest and perhaps most liable to become disorganised.

LECTURE XXI

CERVICAL PARAPLEGIA

Some cases have been lately under observation illustrating the condition to which the term "cervical paraplegia" has been (first of all, I think, by Sir William Gull) applied. In these cases paralysis, usually both of motion and sensation, has affected more or less completely all four extremities, the upper in excess, and the circumstances by which the paralysis has been characterised have enabled us to localise the lesion without difficulty in the upper part of the spinal cord.

I need not say that the occurrence of paralysis in all four extremities does not necessarily point to a lesion of the spinal cord. We occasionally see cases of this kind which have been caused by lesions (hæmorrhage, thrombosis, or tumour) affecting the cerebral hemispheres, or higher ganglia, either in succession, which is by far the most common mode, or coincidently on the two sides, of which examples now and then, though but rarely, present themselves. There is usually a great difference, in such circumstances, in the degree to which the limbs are paralysed on the two sides of the body. It is in these cases that we observe an interference with the action of swallowing and a tendency to slobbering, which do not form part of the symptoms of ordinary hemiplegia, and often give rise to the erroneous idea that we have to do with a lesion of the medulla oblongata. The symptoms displayed by cases of this kind go far to establish, as Hughlings Jackson has pointed out, the probable correctness of

Broadbent's hypothesis as to the association of certain functionally related nuclei.*

There may also be paralysis of all four extremities as a result of lesion of extensive character in the pons Varolii, or in the medulla oblongata. It is unlikely that a lesion in these situations, which is extensive enough to interfere with the motor tracts to the extremities on both sides of the body, will escape involving also some one or other of the various cranial nerves connected with these centres. The specially localised paralysis thus occasioned lends important aid to the diagnosis.

In the cases with which we have now to do the lesion is still lower down. In each instance it is situated within the spinal, not the cranial cavity, and affects the cord chiefly, if not exclusively, below the decussation of the anterior pyramids. This much we are able to say with confidence. As regards the exact nature of the lesion, however, I am not prepared to speak so positively, except in the case of one of the patients who died, when the result gave us the opportunity of confirming the diagnosis by a post-mortem examination. The symptoms during life, and the pathological appearances after death in this instance, will serve to throw light upon many points in the other cases described, which have terminated in a greater or less amount of recovery.

* "When the muscles of the corresponding parts on opposite sides of the body constantly act in concert, and act independently either not at all or with difficulty, the nerve-nuclei of these muscles are so connected by commissural fibres as to be *pro tanto* a single nucleus. This combined nucleus will have a set of fibres from each corpus striatum, and will usually be called into action by both, but it will be capable of being excited by either singly, more or less completely according as the commissural connection between the two halves is more or less perfect. According to this hypothesis, then, if the centre of volitional action on one side is destroyed, or one channel of motor power is cut across, the other will transmit an impulse to the common centre, and this will be communicated to the nerves of the two sides equally if the fusion of the two nuclei is complete, and there will be no paralysis; more or less imperfectly to the nerve of the affected side if the transverse communication between it and its fellow is not so perfect, in which case there will be a corresponding degree of paralysis" ('Brit. and Foreign Med.-Chir. Review,' April, 1866).

The first case is that of a young man in the hospital, who is able to walk about with tolerable facility, although the movements of his legs are notably stiff. The following is the history of his case :

George H—, æt. 18, by occupation a gardener, whilst bathing on July 18th, 1878, dived and struck his head against the bottom of the river in six feet of water. He was unconscious for a few minutes, and on coming to himself felt great pain in the back of the neck (level of third and fourth cervical spines), and found that there was total loss of power in all four limbs. Both hands were clenched after the accident, he says, and for a week he could not open them, nor could he move his head "any way." He says that he could feel "when they washed him."

About a week from the time of the accident he began to recover power gradually, in the right leg and arm first. In January, 1879, he could just manage to stand with a chair; in March he could walk a few yards by himself; and in April could dress himself. Since May he has been able to walk by himself with the aid of a stick.

When he applied at the hospital as an out-patient, on July 9th, 1879, I noted that the electrical reaction of the left deltoid muscle was greater than that of the right to faradaism, and less to the interrupted galvanic current. The electrical reaction of the muscles of the legs appeared to be normal.

The patient was admitted into the hospital in October last, when the following note of his state was taken by Mr Broster :

The spinal column shows nothing abnormal. The head can be moved perfectly in all directions. His breathing is natural, the diaphragm acting normally.

Upper limbs.—There is no muscular wasting. The grasp of the right hand is 35 (dynamometer), of the left 20. There is some rigidity about the wrists, especially the left, and the hands have a tendency to drop. During

the movements of the arms there is some shaking. The muscular sense is normal.

Lower limbs.—The legs present some "clasp-knife" rigidity; the thighs resist abduction. His walk is stiff and slow, the left foot apparently dragging more than the right. Tickling the soles of the feet causes excessive reflex action. The patellar tendon-reflex is exaggerated in each knee. There is foot-clonus in both extremities, but on the right side more than the left. He is able to walk about a quarter of a mile.

The functions of the bladder are not impaired. There is no loss of cutaneous sensibility anywhere. The patient says that he has had no pains at any time in his arms or legs, except a little in the stiff shoulder-joints, and this only on movement.

In all probability in this case there was rupture of vertebral ligaments, and possibly also of intervertebral substance, with hæmorrhage. There may have been displacement also or fracture of a vertebra, but the result leaves this uncertain. It is clear that in some way there was a sudden compression of the cord; and it is probable that, although the source of this compression has gradually been removed, the cord itself has sustained about the point indicated by the patient some permanent damage, involving slightly the anterior horn of grey matter, which has been followed by secondary degeneration of the lateral columns.

I would point out that whilst the electrical reaction of the muscles of the lower extremities is normal, that of the right deltoid muscle is not quite natural. There had been, however, complete loss of power equally in all four limbs. The difference of electrical irritability depends, I believe, upon some injury either to the circumflex nerve, the upper and posterior part of the brachial plexus from which this nerve is given off, or to the anterior horn of grey matter. Lesion of a nerve-trunk, like lesion of the anterior horn or grey matter in the spinal cord, gives rise to loss of power in the muscles which thence obtain

their nerve supply, and to reaction of degeneration. As I have explained on previous occasions, this reaction is characterised by diminution or loss of irritability in the muscle to induced currents, whilst contraction takes place on the interruption of a galvanic current, which is often less strong than is required to bring about contraction in a healthy muscle. In the case of the lower extremities neither the anterior cornua of the dorso-lumbar portion of the cord, nor the nerves distributed to the limbs, were damaged. The loss of power arose simply from interruption to the flow of nervous impulses downwards through their ordinary channel, the antero-lateral columns of the cord. The muscles therefore of the lower extremities exhibit no change in their electric excitability.

I have seen a case in private practice which resembles this one a good deal, with the exception that there is no history of any violence.

A lady, æt. 26, married two years, never pregnant, was sent to me in July, 1879, on account of weakness and numbness in her limbs. She complained that her right arm was stiff and ached, the hand being numbed and tingling as though it had "gone to sleep." This had been the case since the preceding March. Her attention had first been attracted by a change in her handwriting. There was nothing the matter with the right leg. About a week before I saw her the left arm, which had been previously unaffected, had also lost power, and continued feeble, without any such tingling or numbness as was experienced on the right side. At the same time, with this accession of weakness in the left arm, she found herself swerving to the left from weakness of the left leg. The left leg, like the arm, continued weak. If she held anything heavy in her left hand it trembled. The aspect of the limbs was unaltered, and there was no apparent wasting.

In the arms the reaction of the muscles to faradaism was normal. In the lower extremities there appeared to be some diminution of excitability in the tibialis anticus group of each leg, but this I think is very doubtful, as it

was equal on the two sides. In the absence of any satisfactory standard it is very difficult to say, in cases where the reaction is the same on each side of the body, whether an apparent diminution in the faradaic excitability is real. The electric current was felt much more strongly in the right than in the left leg.

The patient complained of the lower part of her back aching in the morning. She had never suffered from bad pain in the head, but at times had been troubled with a little neuralgia. She was thin and strumous-looking. On her father's side there is much consumption, six aunts and one uncle (paternal) having died of phthisis before they were thirty years of age. There was no history of any injury. The catamenia were regular.

A month after her visit to me on getting out of bed one morning she fell down, owing to a rather sudden increase of weakness in the left limbs. I saw her a day or two afterwards, when the left arm was very powerless, especially about the scapulo-humeral muscles. In walking she swerved always to the left. There was a difference in the sensibility of the two arms, a touch being felt more on the left than the right—the sensibility of the right being evidently dulled. If she attempted to hold a book in the left hand, it slipped through her fingers.

I found that deep pressure on the spine caused pain over the fourth cervical vertebra. In no other part of the spine was tenderness complained of when pressure was applied, and repeated examination always caused it at this point. Light touches on the surface occasioned no feeling of soreness.

The recumbent posture and general attention to nutrition by food and drugs was the treatment adopted.

In the following October her condition was noted as follows :

The right arm is somewhat stiff, and there is want of perceptive power in the fingers and thumb. The grasp of this hand is appreciable, but weak.

In the left hand there is some slight power of grasp,

though less than in the right, and no apparent affection of cutaneous sensibility is to be observed. In using the arm, as in attempting to hold a fork, there is trembling of the limb. There is some atrophy of the dorsal interossei in the left hand, with over-extension of the first phalanges, and some atrophy also of the thenar eminence. The over-extension of the first phalanges causes the metacarpo-phalangeal joints to project in the palm of the hand. The patient can touch the back of the head with the left hand, the movement being accompanied by tremor, and causing some aching in the arm. The right leg has continued to be unaffected. The left leg is still weak. In walking, this leg is stiff, and the foot is carried in a semicircle. She tends to walk on the inside of the foot. There is difficulty in dorsal flexion, the foot trembling when she attempts it. There is some increase of patellar tendon-reflex in this leg. The general health is good.

On March 5th, 1880, I received the following account of her condition:—The left hand, it was said, could be used perfectly well for all practical purposes, although it felt rather weak and inclined to tremble. The middle finger was still *en griffe*, but she thought there had always been a tendency to that position. During the past winter she suffered much from chilblains, and the fingers had not yet recovered their proper shape. The muscles of the arm ached if she extended it from the shoulder, and if she attempted to do her hair. There was still a little lameness in walking, and the sole of the left foot had a tendency to scrape the ground. The condition has since, I understand, not materially altered.

At no time has there been any affection of the bladder or rectum.

In this case we may at least localise the lesion in the cervical region. The circumstance of the left leg as well as the arm being affected would imply that it is the substance of the cord (and not merely the membranes) in this region which is in some way involved. An affection of the anterior roots of spinal nerves alone might explain the

powerlessness, and also the muscular atrophy in the left hand, but the affection of the left leg, and, still more, the rigidity in these limbs, pointing to secondary degeneration in the lateral column, appears to show that we must seek the cause of the paralysis and atrophy in lesion of the anterior grey matter in the cervical region.

It will be observed that whilst the left arm was most affected in regard to motility the right alone displayed loss of sensibility. This suggests that the lesion was probably more or less confined to the left half of the cord, the decussation of conductors of sensory impressions taking place, by Brown-Séquard's showing, immediately on entering the cord.

There may be a tumour, or, what would be much more probable, considering the family history and the pain experienced when the fourth cervical spine is pressed, some vertebral caries with pachymeningitis, causing compression and some resulting myelitis.

I would here call attention to a point of great importance. The age, sex, and general condition of this patient were such that the case was one which was extremely likely to be misinterpreted and set down to hysteria, although she had none of the emotional characteristics. Dr Todd in his clinical lectures relates the particulars of a case which suggested similar considerations. The patient was a young woman in whom there was complete paralysis of the left arm with flaccidity and some wasting of the muscles. In walking she dragged her left leg with a sweeping movement. "The case had at first very much the appearance of hysterical paralysis; and this view of its nature was favoured by the absence of all palsy of the face and tongue." Like the patient whose case I have just described, she was of delicate frame, and in her case complaint was made of a pain at the vertex, which was just that to which hysterical women are peculiarly subject. A very important remark here occurs, which I can remember Dr Todd was fond of making. It contains a hint which I always endeavour to remember. "In organic diseases

in women," he says, "symptoms purely hysterical are often coincident even in a prominent form, and embarrass the practitioner not a little." So in my patient pressure upon the cervical spine at a certain point was complained of as causing pain. This spinal tenderness is a constant feature of that hysterical condition to which the name of "spinal irritation" is given, so that had the symptom occurred alone I should not have attached much importance to it. But the situation of the tenderness corresponded with the commencement of origin of the brachial plexus, and thus lent a certain amount of support to the view that there was at this point a coarse lesion of some kind affecting the cord—an opinion which was strictly verified by the progress of the case.

We have lately had in the hospital (she has since gone to the convalescent branch at Finchley) a young patient who has recovered in a surprising manner from a condition of the most extreme danger. It is, like the last, a case of slow compression of the upper part of the cervical portion of the cord, but with much more serious complications.

On May 14th, 1879, Mary Ann M—, æt. 13, was brought to the hospital, with gradually increasing weakness of the four extremities, and was admitted at once as in-patient.

Notes of her case were taken by Mr Broster, from which it appears that she came from a miserable hovel, where she was living in great poverty, and had been ill-fed. For a few months in the winter she had been in a situation as general servant, where the work was very hard, and she had to lift heavy weights. It was in December 1878, whilst thus employed, that she complained of sore throat, for which she applied a mustard-plaster, and then bathed her neck in cold water to remove the stinging which this occasioned. Next day she had a stiff neck. Two or three days later, rain came through the ceiling on to her head and body whilst she was asleep in bed. This was followed by pains in the neck and shoulders and the right knee-joint. About the end of

January, 1879, the right hand is described as having been swollen and blue. The thumb is said to have had little blisters on it. About a week later the right arm lost strength, and then her shoulder became powerless. A few days afterwards the right leg began to fail, and at the end of March she was forced to leave her place. A fortnight before her admission into the hospital the right arm had got, she says, to its worst, but the leg was still becoming more feeble.

For a week or ten days before her admission here the left arm failed, and the left leg had been getting numb and weak. For three weeks past she had been unable to walk by herself.

The following note was taken of her state on admission:

The head is rotated to the right, and immovably fixed in this position, the long cranial axis being at an angle of 45° with the inter-scapular axis, on a plane parallel with and above the latter. The third cervical spine is thought to be somewhat unduly prominent, and slightly displaced to the left. Around and above it there is some hard thickening, apparently beneath the muscles. No tenderness is experienced except at a spot in a line with and below the right mastoid process.

The mental condition is unimpaired. Of the special senses, whilst hearing is normal, smell is more acute on the left than the right side, when tested by sumbul and assafœtida. As regards sight she reads 2½ Snellen left eye, 5½ with the right eye. The ophthalmoscope shows no change in the fundus oculi. Taste is said to be somewhat deficient on both sides, but especially on the left. (In the case of a child who is very ill observations respecting the condition of the special senses are seldom trustworthy, and I do not think these are to be depended upon.)

The upper lip has a somewhat expressionless look. When she smiles, the two angles of the mouth act evenly and fairly, but in voluntary effort, as *e.g.* in showing her teeth, there is decided weakness on both sides, most

marked on the left. She cannot frown. She says she bites her right lower lip. Asked to shut her eyes, the lids fail to meet by $\frac{1}{8}$ inch, but by a strong effort and the aid of other facial muscles she can approximate them. The reaction to faradaism of the facial muscles is normal.

The right masseter is thought to be somewhat stronger than the left, the horizontal movement of the lower jaw is good.

The movements of the eyeballs are perfect. There has been no diplopia. The pupils are equal, moderate in size, and react well to light.

She speaks clearly, though there is slight tremor of the tongue.

Swallowing is somewhat imperfect, but liquids have never returned through the nose. The palate goes up more on the left than the right side. The uvula deviates to the left.

The tonsils are large and somewhat reddened. A small ulcer is seen on the left. The laryngeal movements are normal. She is unable to move her head in any direction whatever.

As regards the respiratory movements, she says, "I cannot take my breath so quick as I used. Sometimes I cannot draw a long breath for two or three minutes, although I feel I want to do so." She takes a number of short inspirations.

When the hand is placed on the epigastric region, and the patient takes as deep an inspiration as possible, the abdominal wall is felt to fall in instead of rising with the inspiration. There is very little movement of the ribs. No incontinence of urine or fæces has occurred.

The right upper extremity is very stiff, but no pain is caused by passive extension of it. There is "clasp-knife" rigidity at the elbow-joint. She can just slightly flex this joint, and there is a faint power of grasp with the right hand. She can place her left hand on the top of her head, but the grasp of this hand is not sufficient to affect the dynamometer. The forearms, which are equal in

size, measure 7½ inches at their greatest circumference. In the hands there is hyper-extension of the metacarpo-phalangeal joints, owing to weakness of the interossei, which is especially marked on the right side. There is no diminution of excitability to faradaism in the muscles of either arm. From the drooping position of the wrists (especially the right) there is evidently weakness of the extensors.

The patient can just stand; she cannot walk. There is marked "clasp-knife" rigidity in the right much more than in the left lower extremity. As she lies she can lift the left leg off the bed, but not the right, and can draw up both legs, the left most strongly. The calves measure 10½ inches each. There is no foot-clonus on either side. The patellar tendon-reflex is exaggerated, and is more marked in the right than the left limb.

Cutaneous sensibility is lost in the tips of the right fingers, front of the right thigh, dorsum of the right foot, and the right shin. It is diminished over the trunk and extremities generally, from the neck downwards behind, and from two inches below the level of the clavicles in front; with the following exceptions:—Along the ulnar border of the anterior aspect of the right forearm there is scarcely any diminution. Here there is distinct hyper-algesia to the induced current. The left upper arm is nearly normal as to its sensibility.

There is no alteration of sensibility in the face, or in the soles of the feet.

No complaint is made of a pain anywhere, but she describes a sensation of pins and needles in the forearms.

The patient's mother has twice miscarried at the third month. The father has had no signs of syphilis, but suffered from gonorrhœa in youth. The other children show no signs of congenital syphilis. A brother died, four days before this girl came to the hospital, of rheumatism and bronchitis. Neither he nor any other member of the family had suffered from a bad throat. There was no illness at the place where the girl lived as servant.

The patient had scarlet fever in infancy. She never had acute rheumatism, nor glandular swellings, nor injury by accident.

The viscera were healthy, the temperature normal. The treatment was rest in bed and iodide of potassium. In the course of four days there was a good deal of improvement, both legs could be lifted off the bed, and there was some increase of power in the arms. The head, too, could be turned a little to the right and left, and bent forward a very little. The breathing remained as before, and Mr Broster remarks: "The pectorals and sterno-mastoids seem to do the work."

A gutta-percha support was fixed to the head and neck.

On June 12th it is noted that "during the last two days the patient has complained of pain in the right leg, shooting down from the groin to the knee, and there has been more difficulty in swallowing. There is wasting of the interossei of the hands."

Next day there was orthopnœa, the respirations very rapid, and entirely upper thoracic. There was some cyanosis. These symptoms increasing on the following day, the headgear was removed, as it was thought it might be interfering with the action of the sterno-mastoids. Auscultation showed tubular breathing and crepitant râles along the base of the left lung, with harsh respiration in the right lung. There was very slight cough. The alæ nasi flapped. Her temperature was 101·4°.

On the evening of the 15th her temperature was 103°, the pulse 130, dicrotous. Respirations 36. She was very cyanotic. There were crepitant rhonchi throughout the left lung behind, and along the axillary border of the right. There was some delirium. Deglutition was much impaired, so that she could only swallow very small quantities of fluid. With great difficulty and gasping she let us know that there was some pain in her head. At this time death appeared inevitable, but in a short time gradual improvement took place in the respiratory system. After the amendment the loss of power in the

extremities was at first more pronounced than it had been before the commencement of the pneumonic attack.

On the 18th I observed that the left side of the chest moved a little more than the right; the movement being mainly an elevation of the upper ribs; there was but very little lateral expansion. The epigastrium sank during inspiration.

On the 27th it is noted that the temperature is 100°; the respiration mainly upper thoracic, but with a little unilateral expansion. Over the base of the left lung the percussion note was dull, and there was increased vocal fremitus. Moist bubbling and crackling râles could be heard. On the right side there were a few small mucous râles along the posterior border.

Both legs could now be moved freely; each could be lifted off the bed and held extended for about thirty seconds.

By the middle of July all traces of the pneumonic attack had subsided. There was gradual improvement in the paralysed limbs. In the middle of September she had so far recovered that she was able to be sent to the Convalescent Establishment at Finchley, and the following note of her condition was then taken:—She carries her head in the same "side way" as on admission, and has gained no power as yet over its movements. She cannot look over her shoulder. The thickening previously described is very distinct around and over the third cervical spine. This spine can be felt distinctly prominent beyond the other cervical spines. The weakness described in the facial muscles remains *in statu quo*, except that she can approximate her eyelids a little more than she could formerly. There is some deviation of the uvula to the left. The respiration is entirely thoracic, no movement of the diaphragm being perceptible.

On the right side she can flex her elbow; the limb is slightly stiff. There is a perceptible, but not measurable, grasp of the hand. No distinct wasting of muscles of the arms is to be noted. She can raise the right arm to the level of the shoulder. On the left side too she can hold up

the arm fairly. The grasp of this hand is likewise not measurable. She can feed herself and cut up meat, but cannot dress herself.

She can stand and walk; the calves each measure ten inches in circumference. The walking is performed in a shaky manner, the trunk held stiffly, and the knees bent. She does not drag her feet. In rising from a chair she appears to have much difficulty, and requires a little impetus before she can get on her legs. The patellar tendon-reflex is exaggerated on each side. Foot-clonus is present, though not to a very marked extent, in both limbs—in the right more than in the left.

In the left hand and forearm (except in the tips of the fingers, which are still numbed) sensation is normal, whilst in the left leg it is deficient. In the right upper limb sensibility is still defective, though less so than formerly. It is as good in the right leg as in the face, where it is apparently unimpaired.

For her own part, the patient says that the sensation in the left lower limb is better than it was; and that in the right leg, which used to be the worst, she feels better than in the left. The right leg, which was the weaker, is now, she says, the stronger. She says that a touch upon her back is felt quite right, but not so on the front of the body. She has no pains whatever, and no sensations of "pins and needles."

The appetite is good. She has control over the sphincters. Two months and a half later, whilst at Finchley, she was well able to get about by herself. The movement of the diaphragm had returned.

My first impression, on seeing this patient in the out-patient room, was that we had to do with one of those acute cases of nearly universal paralysis, the pathology of which remains obscure, and of which I have brought two marked examples before the Clinical Society of London.* In view of the history of "sore throat," one had to consider the possibility also that the affection might be

* See Lecture XVIII.

diphtheritic. Both these contingencies were disposed of by the observation of the fixed position of the head, and the discovery of the thickening about the third cervical spine.

The mode of onset is well worth remembering. It was in the winter, whilst working very hard and lifting heavy weights, that this poor drudge of a servant began to complain of sore throat, followed by stiffness of the neck, pains in the neck, shoulders, and right knee-joint. In a remarkably similar case, related by Dr Todd,* there was a similar stiffness of the neck, which had begun six months before the patient's admission, and was, as in this case, attributed by her to cold. In that instance also the head is described as being "drawn to the right side." The face looked forward and rather to the left. There was a very rigid state of the right sterno-mastoid muscle, and on the left side the neck presented considerable deep-seated swelling in the region of the uppermost cervical vertebræ. The rotatory and other movements of the head were much impeded, those to the right existing only to a slight degree, and those to the left being likewise much limited. In our patient the head was also turned to the right, and at first was immovably fixed in that position. When the girl had been kept a few days in bed absolutely at rest, a certain amount of power of moving the head returned, and in addition, there was distinct improvement in the power of the extremities. Although we gave the girl iodide of potassium, I do not think the amelioration, which was associated in point of time with its administration, is to be attributed to the action of the drug. It was probably the rest in bed which produced the good result. We see every day in cases of Pott's disease of the spinal column a remarkable amount of repair following simple rest in bed. Here let me say that I have often been struck with the comparative facility for repair exhibited by the spinal cord, a recuperative faculty, it has seemed to me, undoubtedly in excess of

* 'Clinical Lectures on Paralysis,' 2nd edit., p. 330, London, 1856.

that possessed by the cerebral hemispheres. If I am right in this observation, we should probably have to seek for an immediate explanation of it in the anatomical disposition of blood-vessels, in regard to which I am not able to speak. But it does appear to me that we might naturally expect the spinal cord to possess greater stability and power of repair than those nervous centres which from the point of view of evolution are infinitely younger. Herbert Spencer tells us that, according to Leuret, the average ratio of the brain to the body is, in fishes, 1 to 5688; in reptiles, 1 to 1321; in birds, 1 to 212; and in mammals, 1 to 186—figures which sufficiently though roughly indicate the comparative youth of the highest nervous centres in man.

Leaving transcendentalism aside, there are points of great importance in this case. First, what was the nature of the lesion?

In reference to this I will revert for a moment to the case described by Dr Todd, from which I have already quoted, and which presents a remarkable similarity to the one just related. The girl when admitted into King's College Hospital complained of pains in the head, not fixed, and of some stiffness in the neck. There was complete paralysis of the left arm, with flaccidity, and some wasting of the muscles. She dragged her left leg, but could walk. Later on, she became one day completely powerless in the left leg, and the next morning the right arm was found to be partially paralysed, whilst the action of the diaphragm was "so feeble that it could scarcely be felt in its descent." No contraction could be detected in the intercostal muscles of the left side, and the very slight action of that half of the chest seemed to depend on the movements of the right side. The paralytic symptoms increased rapidly, involving the right leg as well as the arm. On the day of her death it is noted that she was found by the house-physician, who was called to her, speechless with livid face and purple lips, breathing in gasps at intervals of twenty seconds. "The only muscle

which could be observed acting was the sterno-mastoid of the right side; there was no perceptible motion of the ribs on either side, and no abdominal movement." And so she died.

Before describing the result of the autopsy which was made in Dr Todd's case, let me mention what is noted about the condition of the neck, which may well be compared with that which obtained in our patient. "Our attention, he writes, was arrested by a marked stiffness of her neck, in consequence of which the head was drawn to the right side, and the face looked forward and rather to the left. On the left side the neck presented considerable deep-seated swelling in the region of the uppermost cervical vertebræ. It was evident to the touch that this swelling was not due to any accumulation of fluid, nor were the integuments and muscles in any way diseased. They were stretched over the swelling, and a thickened state of bone and ligamentous tissue could be felt through them."

I should think that there could not be a more remarkable similarity in the description of two cases.

Mr Broster writes of ours: "The third cervical spine is thought to be somewhat unduly prominent, and slightly displaced to the left. Around and above it there is some hard thickening, apparently beneath the muscles." Again, as regards the mechanism of respiration he notes, "The pectorals and sterno-mastoids seem to do the work." It will be noted that there was a feature in our case which did not occur in Dr Todd's. Some of the lower cranial nerves, certainly the portio dura, and possibly, though I am very doubtful on this point, the motor portion of the fifth and the glosso-pharyngeal, were involved, as well as the phrenic and nerves to the trunk and extremities, which were affected in both instances.

Now, let us return to the account of the autopsy in Dr Todd's case. "The disease consisted chiefly in an enlargement of the odontoid process of the second vertebra. This extended backwards, wearing through the dura mater, and was covered at its upper part and on the left side by a

fibro-cartilaginous growth, which compressed and flattened the spinal cord on the left of the median fissure. The compression of the cord was so great that it seemed as if a large portion of the nervous matter had been pushed from the left to the right side and partly upwards; and the cord was swollen both above and to the right of the compressed part. The nervous matter on the left side was soft and slightly discoloured as from small ecchymoses; that on the right of the fissure was very soft and diffluent. The pia mater of this portion of the cord was red and congested. No other disease was found in any part of the body."

The more fortunate issue of our case leaves us necessarily in doubt as to the exact nature of the lesion. That there was, as in the fatal one described, slow compression of the cord cannot be doubted. I should be disposed to think that there was caries of one or more of the upper cervical vertebræ giving rise, as we know this will do, to pachymeningitis and consequent pressure upon the cord. It would seem that this inflammation of the dura mater had extended a short distance into the cranium through the foramen magnum and that the thickening of the membrane thus occasioned had exercised a certain amount of pressure upon some of the cranial nerves as they prepared to pass through their respective cranial foramina. This seems to have been especially the case with regard to the portio dura of both sides. It is remarkable that, if this were so, the reaction of the facial muscles to faradaism should have been, as it is represented to be, normal. I think it possible that there may have been some lowering of excitability to induced currents, but owing to the fact of both sides being affected, and a standard of comparison therefore not being available, the abnormality may have passed unobserved.

The pains were not strongly marked. Such as were present affected the head, doubtless through irritation of the occipital nerve-roots.

The most interesting point in this remarkable case is

the long-continued paralysis of the diaphragm. This paralysis was doubtless only partial, like that of the extremities and of the intercostals; but it was pronounced enough to give rise to the abdominal depression during inspiration mentioned by Duchenne as characteristic of paralysis of this muscle. Duchenne thus describes the condition :—"At the moment of inspiration the epigastrium and the hypochondria are depressed, whilst, on the other hand, the chest dilates; the movements of these parts are reversed during expiration." Recovery from such an attack of pulmonary inflammation as this girl suffered must, in the circumstances of her case, be an exceedingly rare event. I have never, certainly, seen any one go so near to death, and yet recover.

It will be observed that the local symptoms pointing to Pott's disease in this patient were not strongly expressed. The note says: "The third cervical spine *is thought to be* somewhat unduly prominent and slightly displaced to the left. Around and above it there is some hard thickening, apparently beneath the muscles." Now, in this instance, there was abundant evidence in the paralytic symptoms of a most serious organic lesion of the cord. But it sometimes happens that we see a good deal more deformity than was to be found in this girl's neck *without* symptoms of compression of the cord, and a very delicate question arises in such a case as to the nature of the startling deformity—whether it depends upon falling in of the bodies of certain vertebræ, or is simply an angular curvature from weakness of the cervico-dorsal muscles.

I saw a case not long ago in which this difficulty had occurred. The patient, a female, twenty-three years of age, had so great and abrupt a prominence of the sixth and seventh cervical vertebræ as to simulate very closely posterior curvature of the spine from Pott's disease. She complained of pain in the situation of the swelling and tenderness when it was pressed upon, and she also said that she had numbness in her arms.

Now these, and especially the last, were very suspicious

symptoms, and more than one of the many medical men who had examined her had pronounced an opinion that the condition depended upon vertebral caries. The girl had a very weak nervous system, and her frame, though large, was fat rather than muscular. It seemed, when I came to inquire, that there was no notable loss of power in her limbs, that the numbness described as affecting her arms was not constant, and that it also affected her trunk and *head*. The prominence of the spine had been coming gradually for upwards of three years. If it depended upon breaking down of the bodies of vertebræ from caries it would necessarily be attended, I thought, by some pachymeningitis, which would form a swelling in the spinal canal sufficient to cause at least some pressure upon the cord, and even if this were not enough to produce any remarkable loss of power in the extremities, it would certainly, from all that I have seen, in cases of Pott's disease above the lumbar portion of the spine, occasion an increase of tendon-reflex in the extremities. On testing the reflexes at the wrist and knee I found that there was little or none to be found at the wrist and a perfectly normal condition at the knee. There was, indeed, a total absence of any exaggeration of tendon-reflex. This fact seems to me sufficient, especially considering the length of time during which the deformity had existed, to negative entirely the idea of caries of the vertebræ. The case is, I believe, one of weakness of extensors of the cervical vertebræ, giving rise to a cervico-dorsal cyphosis. The amount of subcutaneous adipose tissue is so great that it is impossible to judge whether the spinal muscles are atrophied, but it is probable from the circumstances that they are.

Let me now refer to a case which I not long since followed to its termination. It is one which I watched with the greatest interest, and at one time with considerable hope of the patient's recovery.

The patient, S—, was a clerk, about thirty-five years of age, living a few miles from town, whom I visited, at

the request of his employer, on July 14th, 1877. He was below the middle stature, with a squint, which I was told was of many years' standing. He presented a peculiarly muddled look, and a manner so confused that I had difficulty in obtaining information from him. His memory appeared to be very bad. From what I could gather then, supplemented by later information, his family history was as follows:—His father died at thirty-eight years of age in a lunatic asylum; his mother, aged fifty, of a "natural death;" she drank. He had never had any brothers or sisters.

The patient suffered from fits in his infancy until he was about six or seven years old. The next thing one learnt was that when he was twenty-one years of age he lost the sight of each eye in succession, that of the left eventually recovering. At about twenty-six he had a venereal ulcer, which was accompanied by two suppurating buboes, for which he took medicine to make his gums sore. His employer told me that five or six years previously S—'s eyes were "bad," so that he had to be off work for six months. During the last year he had had five or six fits, of which two occurred in the week preceding my visit. On the occasion of the first of these he felt dizziness, and was obliged to lay hold of his desk. He thought he was going to fall, and shortly afterwards he did fall, unconscious. He was not aware of having bitten his tongue.

The sight of the right eye was almost *nil*, on account of a large white opacity of the cornea, concealing nearly the entire pupil. The squint, which was said to be of old standing, was not accompanied by double vision. He walked feebly and with a tottering gait, and there was no power of grasping with either hand. This failure of strength, he said, had only taken place during the last four days, but for the last week he had been feeling increasingly feeble. There was no marked want of symmetry in the face. His tongue was protruded straight. The patient cried out every now and then with aching

pains in the arms, sometimes in one and sometimes in the other. Frequently there was much pain over the brows and across the back. At times, he said, he suffered from pain all round the lower part of the head behind.

For some months past he had been liable to what he called "rheumatism" in his limbs, and aching pains across the head and loins. There was what he described as "painful numbness" in both arms.

The heart and lungs were healthy. The appetite and digestion were bad.

By the ophthalmoscope (under great difficulty, owing to unsteadiness, very small pupil, and some considerable haziness of the media) the retinal vessels in the left eye appeared tortuous, and the outline of the disc indistinct. The large opacity precluded observation of the right fundus oculi.

His landlady told me that six or seven weeks before this his manner had appeared very strange, and his memory had failed him to a large extent. It seemed that for a week past he had been taking small doses of bromide. I ordered perchloride of mercury and iodide of potassium.

A week later he came to my house, distinctly improved in intelligence. I noted that he held his head as though he had a stiff neck, and complained of pains at the back of the head and across the right ear.

There was a peculiarly helpless look about his hands. The excitability of the muscles of his arms was diminished in a marked degree to induced currents.

Whilst examining him with the ophthalmoscope, I noticed that he was constantly swaying about, and frequently exclaiming suddenly as from the effects of pain. His arms would jerk and he complained of sharp pains in them.

His temperature was normal. The urine contained no albumen or sugar.

After another fortnight's treatment with mercury and iodide, he was manifestly much improved. There had

been no repetition of a fit. He had lost the pains in his arms, and there was but little pain in the head. The right leg was stronger. He had experienced for two or three days a feeling of tightness around the waist, which was relieved by the action of the bowels. The mind appeared to be quite clear. By the ophthalmoscope the left disc appeared to be more distinctly defined, but the vessels remained tortuous. He was ordered one sixteenth of a grain of perchloride of mercury with twenty grains of iodide of potassium three times a day, and twenty grains of bromide of potassium at bedtime.

On August 3rd the following note was made: "Three days ago he had a repetition of a feeling he had not experienced for a year previously, which then ushered in a fit. The feeling was of a whirling round in the centre of his head. He had a trifling amount of pain at the back of the head.

"His walking power improves daily. He walked today the best part of a mile, although there is still weakness, especially in the right leg.

"There is wasting of the dorsal interossei in each hand, but the fingers have regained warmth and colour (they had been cold and dead-looking). Their movements are, however, still very clumsy. This morning he lifted the ewer, which he could not have done three days ago.

"The electrical reaction in the muscles of the forearm is somewhat improved."

The following note of diagnosis was also made at this time: "One would think, from the powerlessness and wasting of the muscles, together with loss of electric irritability, that here was a case of pressure upon the anterior roots of nerves, probably from pachymeningitis."

Three weeks later the patient had gained strength, and was able to walk as much as three miles. The arms, too, had greatly improved. He had resumed his usual handwriting with a pencil, but there was too much clumsiness in the fingers for the successful use of a pen;

nor could he pick up coins or pins. "It was only the fingers," he said, "which were still obstinate and had not regained their pliancy." The hands felt generally warm in the evening. The warmth of the bed seemed to soothe them. Contact with metal appeared to send them "all ajar." There was no pain in the head; his appetite was good and he slept well. He had been taking the iodide with a little iron.

On September 12th it was noted that the patient could walk three to four miles without fatigue. The grasp of the left hand was very imperfect, and there was still wasting of the interossei, as well as an "electrical sensation" in that hand. His gait was rather jerky and disorderly. He was somewhat restless at night. There was a sense of fulness, but no pain, at the back of the head. A few days after this he began to get more pain in the right arm, especially from the middle of the upper arm down to the tip of the ring-finger. On examination, the ring and little fingers of the right hand were found to be clumsy and unnatural in their movements.

During the following week there was only a little pain, "almost what he might describe as a sympathetic feeling," in the left arm. But the fingers of the left hand were much more helpless and clumsy than those of the right.

"At this moment (I note on the 20th September) there is no pain in the right arm, but he has had several paroxysms this morning and many in the night. The pain is almost unbearable. It comes on very suddenly—a burning aching pain, 'as if the vein were filled with molten lead.' The day before yesterday it was so intense that he 'started up, and broke into a perspiration all over.' A paroxysm will last about half a minute. It seems to begin at the top of the arm, and it 'must go its course.' After the pain has left, a feeling of relief alone remains. He can lift his arm during the pain; in fact the exertion of the muscles seems to give relief. It is usually when the pain is going on in the right arm that

he gets a little also in the left. But he thinks (questioned) that he has sometimes a little pain in the left arm when there is none in the right."

Examination showed that the excitability of the muscles of both arms to induced currents was much diminished, but not removed. There was still wasting of the interossei, but not, it was thought, so great as had been. He was unsteady on his legs, especially on the left. A week before this he had felt pretty well, and gone by rail to the Crystal Palace. Three days later he went and dined with a friend in the City (a distance of some six or seven miles), The following day was spent indoors. He undressed in my room for examination, and appeared to be very helpless; I had to button his clothes. By the ophthalmoscope, I found the retinal vessels of the left fundus very tortuous, but the outline of the disc distinct enough. I dilated the right pupil with atropine, but the opacity prevented any observation of the fundus.

The patient was now placed under the care of Mr Harrison, of Streatham, whom I met in consultation on November 1st. S— was up and dressed, seated in a chair. The arms were much atrophied, and the legs also, but to a less extent. He did not complain of pain in the arms, but occasionally I observed them to be jerked, and then he would cry out. When asked to explain the cause, he said he had the cramp in them.

The left forearm was hyperæsthetic to cold, but there was delay in receiving impressions of pain.

There was paralysis of the sphincters, with ammoniacal urine.

The patient was put on a water-bed and ordered one sixteenth of a grain of perchloride of mercury with fifteen grains of iodide of potassium three times daily; also some lactophosphate of iron.

November 17th.—I heard that there was complete paralysis of the intercostal muscles, and the breathing was entirely diaphragmatic. The patient was having epileptiform attacks every day. He was thought to be dying.

20th.—As the gums were beginning to be sore, the mercury was stopped and the iodide continued.

December 10th.—Mr. Harrison wrote that there had been a slight improvement. The intercostal muscles were acting slightly, and there was more power in the arms and legs.

About a week before his death, which took place on December 24th, he was removed into other lodgings. At this time he could just slowly and tremulously lift a handkerchief and give a very slight squeeze with either hand. The arms appeared to be equally weak.

For many weeks before the end he could not stand, and was indeed unable to lift his foot from the floor; but during the last few days of his life he could do this. Towards the close he became exceedingly irritable in temper.

On the 23rd he was taken with insensibility and stertorous breathing, and died the following day, having never recovered consciousness.

The autopsy took place fifty-two hours after death, in the presence of Mr Harrison and myself. Weather very cold. The body was extremely exsanguineous; there was a small bedsore on the sacrum. Much wasting of muscles was noted in the forearms and hands, not so much in the upper arms. The legs were thin, but did not appear so much atrophied as the upper extremities.

Head.—There was some slight opacity of the arachnoid membrane. The brain-substance generally was soft, putty-like in consistence, wanting in elasticity. The left crus cerebri was almost in a fluid state of softening. The surface of the pons Varolii, and also of the medulla oblongata, was softened. The basilar artery was thickened in its walls, and contained a thrombus. There was a large quantity of fluid in the ventricles.

Spinal cord; anterior aspect.—On ripping open the dura mater from below upwards no change was noticeable until the upper dorsal region was reached, when this membrane was found glued together with the soft mem-

branes, and the whole mass firmly adherent to the cord. With the finger one felt a hardness about the size of a bean at the left side of the cord at the lower part of the cervical enlargement. Here it was found that for a length of about 12 millimètres there was complete adhesion together of both hard and soft membranes; thence upwards for nearly 4 centimètres the pia mater was much thickened and adherent to the cord. At this part the cord, on section, was found to be of the consistence of rotten cheese. The whole extent of this softening was about 5 centimètres longitudinally. Above this again, and for the remaining 12 millimètres or so of the separated cord, the substance felt firmer, if not of natural solidity.

Posterior aspect.—The membranes appeared natural, until we came to about 16 centimètres above the pointed extremity of the cord, where the dura mater was found thickened and adherent to the soft membranes. Slitting these up, they could with some little force be separated from the surface of the cord, and then this latter, as well as the exposed surface of soft membrane, wore an aspect of erosion, and was pink in colour. It was evident that one was tearing through recent inflammatory adhesion. This adhesion was very close for about 4 centimètres, and thence upwards, continued, though not so firmly, till we came to the cervical enlargement, where there was (as on the anterior aspect) a matting together of all the membranes. On cutting through the part where the hardness previously described was most pronounced, we found that it was of almost cartilaginous quality in section, and measured ·5 cm. in thickness. The cord was nearly fluid at this point, and scarcely a trace of the pattern of grey matter was to be seen on section.

There was no disease of the vertebræ.

My friend Dr H. R. O. Sankey, of the County Asylum, Prestwich, was kind enough to examine the cord which I forwarded to him, and his report is as follows:

"Transverse sections of the piece of cord which was sent to me were very difficult to cut, owing to the friability

of the cord substance and the toughness of the thickened pia mater.

"*Microscopical appearance of pia mater.*—At the posterior part of the cord this was thicker than at the anterior, and where it was thickest it measured about one tenth of an inch. It consisted of connective tissue, but was not at all uniform in structure. At some places it was highly cellular; at others chiefly fibrous. The arrangement was very irregular, but at one part an appearance was seen in which the fibrous bundles were very regular in arrangement and parallel to the cord. But few vessels existed in this structure; those observed, however, were well developed, especially as to the adventitia, which passed insensibly into the general fibrous matrix in which they lay. In one case a very thin-walled lacuna-like space was seen filled with blood, which was uniformly coagulated, and had not long been in this position.

"The band of connective tissue which passes into the anterior fissure was greatly thickened, and contained vessels which were altered in the same manner as those of the substance of the cord (*vide* infra). The adhesion between the pia mater and the cord substance was broken down in the cutting of the sections, and could not be made out, but it is probable that the entering trabeculæ were not thickened, but rather softened, like the cord substance.

"*White matter of the cord.*—This was throughout greatly altered. The changes were as follows:

"1. Ascending fibres were in many parts only represented by homogeneous globules, staining slightly in carmine and logwood, while in others a few more or less normal fibres were seen; and in others only the shrivelled sheath of Schwann could be seen, devoid of axis-cylinder, and filled with a homogeneous drop of myelin.

"2. Connective tissue was greatly increased. As to its cellular constituents, immense quantities of new cells, darkly stained, covered the field of view at every part.

"3. A granular *débris* filled the spaces left by these two elements. (N.B. Some of these appearances might be due

to post-mortem decomposition, *i.e.* the state of the more healthy nerve tubercles and the granular appearance just described; that of the cells could not be.)

"Vessels were large, tortuous, and the walls were covered by new cells, so that their structure was in places quite obscured. In some places, where it could be seen, it was healthy, and the vessels were all void of blood, or nearly so, and patent.

"*Grey matter.*—Of the two cornua (anterior) the left was the more diseased. In this, two or three nerve cells could be seen which were circular bodies, shorn of all processes, showing a granular nucleus evidently in the last stage of degeneration. In the right cornu a larger number of cells were seen. They were more refractive than usual. They were in many instances devoid of processes, but retained them in other cases for a short distance. The posterior cornua were involved in the disorganisation. It is, however, impossible to say that they had suffered more than other parts, or that the right had suffered more than the left.

"All that could be made of the rest of the grey matter was that it contained too many nuclei, and was granular and opaque, and contained vessels surrounded by cells to a great extent. One vessel near the central canal contained an ante-mortem clot.

"*Summary.*—1. Fibrous thickening of pia mater.

"2. Infiltration of cord by new cells from vessels, and increase of existing connective tissue by migration or otherwise.

"3. Destruction of normal constituents.

"4. Post-mortem decomposition."

I take this to have been a case of inflammation of the internal layer of the spinal dura mater, of the arachnoid, and pia mater, the hard and soft membranes being agglutinated at the point described into a mass which was strongly adherent to the cord. The external layer of dura mater appeared to be unaltered. Dr Sankey speaks of the tough mass as thickened "pia mater," but I

imagine it is difficult to say with which of the membranes the hypertrophy described is to be especially associated, for they are all involved.

Charcot,* writing of hypertrophic cervical pachymeningitis, remarks that it consists of an alteration of membranes affecting more especially the dura mater, and notes that the cervical enlargement of the cord is in some respects a favourite seat of it. The alteration in the dura mater is the primary fact, but later on the cord and peripheral nerves are involved. He thinks that the cases formerly described by Laennec, Andral, and Hutin, under the name of "hypertrophy of the spinal cord," properly belonged to cervical pachymeningitis; the swelling, which was really due to the membranes, being attributed to the cord itself. As a matter of fact, the cord, far from being hypertrophied, is squeezed from before backwards. The pia mater is affected, but much less than the dura mater, which may attain a thickness, he remarks, of 6 or 7 mm. (In the case above described the thickness, when the cord was fresh, was 5 mm.) The dura mater is usually altered in its entire thickness, as is proved by the adhesions which unite it outside to the vertebral ligament, inside to the pia mater. In the present case it will have been observed that the external layer of dura mater was free from change.

With this remarkable thickening and agglutination of membranes the cord at the part most affected by meningitis was broken down and disorganised. The condition was one of meningo-myelitis, which I should think was probably syphilitic in origin, for the following reasons:— 1. The patient had suffered some eight or nine years previously from a venereal ulcer. 2. The autopsy showed the basilar artery thickened, and thrombosed; the crus cerebri, pons Varolii, and medulla oblongata softened. The patient's age was about thirty-five—too early for the arterial change to be reasonably ascribed to senile degeneration. The thickening (endarteritis) was precisely

* 'Leçons sur les Maladies du Système Nerveux.'

of the character which we see so frequently in the cerebral arteries in the sequel of syphilis. 3. During life, as will have been observed, an extraordinary improvement took place in the patient, especially as regards his lower extremities, very shortly after he began to receive specific treatment.

An affection of the eye which attacked the patient at twenty-one years of age and produced loss of sight of each eye in succession, that of the left eventually recovering, could hardly have been anything else, I suppose, but interstitial keratitis. Mr Hutchinson, to whom we are indebted for vast additions to our knowledge on this, as on so many other subjects, has established with certainty that interstitial keratitis is occasioned by congenital syphilis. If we are to trust the accuracy of the employer's recollection, there would seem in this case to have been a relapse of the keratitis after an interval of about seven years. With these facts before us and the history of the occurrence of fits in his childhood, we may take it as certain, I think, that this patient was the subject of congenital syphilis as well as probably of the acquired disorder.

I was anxious to discover whether in this case a microscopical examination would disclose thickening of the walls of the vessels in the substance of the cord. It appears to be very likely that in cases of syphilis there is frequently such a thickening of the walls of the spinal arteries as experience has proved will occur as regards the intracranial arteries. Such a thickening was found in the basilar artery in the present instance, and it appeared, therefore, at the least probable that a similar condition might have existed in the vertebral arteries or their branches. I regret that it did not occur to me at the autopsy (which was made under circumstances of some difficulty) to remove specimens of the vertebral arteries for examination. The hardening of the cord was not accomplished very satisfactorily, and Dr Sankey had some trouble in his examination. He found that the vessels, as

well in the substance of the cord as in the connective tissue dipping into the anterior fissure, were large, tortuous, and their walls covered by new cells, but in only one instance was he able to observe the presence of an ante-mortem clot, which indicated that, as regard that vessel, at least, thrombosis had taken place. This, however, isolated as it is, is of great importance. We are still unable to say anything certain regarding the stage at which this thrombosis of a thickened vessel occurred, or as to the relation in point of time between the inflammation of the membranes and the myelitis. Nor does the clinical history aid us in any attempt at deciding whether the cord or its membranes was first attacked with inflammation.

The paralysis of the arms in this man was throughout much more marked than that of the lower extremities. This was doubtless because in the former case it was due in great part to disorganisation of the centres within the cord, in the latter partly to the result of meningitis, partly also to the interruption of nervous impulses caused by the slow compression in the cervical region. The appearances would lead to the inference that under the specific treatment the recent inflammation of the membranes in the dorsal and lumbar regions had very much cleared up. The treatment came too late to affect to the same extent the more or less organised thickening of the membranes in the cervical region, although it is surprising to note the amount of improvement which did take place in the state of the arms. I do not feel able to explain satisfactorily the reason of this.

The pains, as they were described by this patient, were characteristic of a membranous lesion, causing irritation of the posterior roots of the brachial plexus. As Charcot has pointed out, these pains may be met with in intra-rachidian tumours, in Pott's disease, and in vertebral cancer, as well as in hypertrophic cervical pachy-meningitis.

The contraction of the pupils which was observed is

well known as an accompaniment of lesions of the upper part of the spinal cord.

According to Charcot, epileptic attacks sometimes manifest themselves periodically in cases of compression lesion of the cord. Although this association is comparatively rare, he has been able to collect five cases of this kind. In the present case I believe that there was optic neuritis, although I am unable to speak so positively on this point as would be desirable. The difficulty of using the ophthalmoscope was very great even when the pupil was dilated, on account of the haziness of the media, and also because the patient presented ataxic movements, swaying to and fro on the chair, and jerking his arms occasionally, as he was seized with a dart of pain. I could find no trace of gumma in the membranes of the brain, but it is always possible that a recent formation of this kind may have been absorbed under treatment. In view of the probable existence of optic neuritis and the thrombosis of the basilar artery with cerebral softening, I do not think we can reasonably refer the epileptic seizures in this case to the compression of the cord in the cervical region.

LECTURE XXII

SYPHILITIC PARAPLEGIA

A MAN was brought into the hospital with complete paralysis of the right lower extremity, some weakness in the left, and with little or no power over his bladder. He was a coachman, twenty-five years of age; and nine days before he came here he had been seized with "cramp" in the right ham, the pain being intense and paroxysmal, lasting perhaps two or three minutes, and recurring after a few moments' interval. The pains at first appeared to be caused by movement, but gradually came to occur independently of any such provocation. From the first the limb was weak, but the man persevered in driving his master until two days before his admission, when he had become completely incapacitated. Now, it happened that he was in the service of a surgeon, who, being aware, from personal observation, that a year previously he had suffered from syphilitic ulceration of the leg, referable to an infection six months before, thought that his paraplegia possibly depended on the same cause, and sent the patient to the hospital.

When the Resident Medical Officer came to examine him, he found that the man had very severe pain between the blade-bones, if he tried to sit up, and that his back appeared to be very stiff. He was at once placed upon a water bed, but even as he lay on this he was never free from a dull aching in the dorsal region, which was increased to acute suffering if he tried to turn. Percussion along the spine did not occasion pain, but when it was applied about the second lumbar vertebra, the right leg was seen to twitch, and a cramp-like pain was started in the ham.

There were sharp pains "like knives" around his trunk, from the nipples to the hips, which were increased by movement, a numbed feeling about the belly, and a sensation, he said, "as if he had eaten too much." Sometimes the right great toe twitched, and this was accompanied by pains which shot up the leg and thigh. In the right leg a pin prick was scarcely felt at all, whilst in the thigh it was described, not as pain, but weight. In the left lower extremity there was also a similar modification of sensibility, though to a less extent. In both limbs there was blunted sensibility to touch, which was especially marked in the right, and in both pricks or pinches set up reflex twitchings. On the right side the patient could only slightly move the ankle-joint, no other motion being practicable in the least. The left lower extremity, on the other hand, though not so strong as in health, was not very materially lessened in power. The muscles of both limbs reacted well to faradaism. There was paresis of the bladder. The pulse was 88; temperature 98·4° F. Urine free from albumen.

Without loss of time, iodide of potassium was given to the patient in doses of ten grains (increased in a few days to thirty) three times a day. The spine was painted for a few days with the oleate of mercury (15 per cent. solution), which was exchanged for inunction with mercurial ointment ten days after his admission. When a drachm of this had been rubbed in daily for eleven days the gums became tender, and it was then applied at longer intervals. This, with the addition of a hypodermic injection of morphia occasionally during the first few days, when his pains deprived him of rest, constituted the treatment.

The patient's progress was as follows:—On the sixth day he felt stronger, and had slept fairly without a sedative. He could sit up in bed by himself, and was able to bend his back without pain; there was still, however, decided stiffness in his spine. The action of the bladder had become normal. On the tenth day the

cramp-like pains and twitchings had ceased, and he could bend his back freely. For the first time, too, he could now move his right leg and raise it from the bed. The cutaneous sensibility was also distinctly improved; he could feel a gentle touch, but still could not recognise the prick of a pin. The feeling of distension in the belly had disappeared. Days passed, and he continued to improve steadily in the power of his right lower extremity, the force of the muscles which move the hip-joint returning before that of those belonging to the leg and foot. On the thirty-sixth day it is noted that the patient was up and about and walking very fairly, his only inconvenience being that he sometimes felt a cramp in the back of the right thigh and slight pain in the hypogastric region. The right leg seemed as strong as the left. There was no longer any defect of sensibility. On the fiftieth day he was discharged, and returned almost immediately to his occupation, which he continues to follow.

There is a point about the patient's temperature which I must not omit. Even in the first few days, when the treatment was but just commenced, the thermometer seldom marked so much as one degree above the normal rate, and on only two occasions during his illness (both of which were within the first four days) did it reach so high as 100.°

It is quite evident that in this case there was a more or less circumscribed inflammation of the spinal meninges, which the seat of pain and the distribution of paralysis enable us to locate in the lower dorsal portion of the cord, affecting especially, though not exclusively, its right half. With the clear history of syphilis before us, we can well imagine that the meningitis was gummatous, and that it probably involved especially the internal surface of the dura mater (pachymeningitis interna), but extended also to the contiguous soft membranes. In such a condition (as, indeed, I have confirmed post mortem in another case*) there may be a complete agglutination and thickening of

* See the preceding Lecture.

the membranes, leading, if not arrested, to softening of the cord, with no greater elevation of temperature than was observed here. Note also that there was no tenderness on percussing the vertebral spines. The absence of this symptom is too often regarded as being inconsistent with the existence of serious lesion of the cord or its coverings. Nothing can well be less founded on fact. If we put aside cases in which the vertebral column itself is diseased, we shall find that the existence of very marked spinal tenderness points strongly in the direction of a functional nervous affection of comparatively little importance, and is but rarely associated with a serious organic lesion of the spinal cord. On the other hand, the stiffness of the spine which this man had, and the exquisite pain which attended his efforts to turn in bed, whilst they are highly characteristic of spinal meningitis, form no part of the symptoms of "spinal irritation." Happily, in this instance, we have not had the opportunity of confirming our diagnosis. Fortune favoured the patient in giving him a master, who is a member of our profession, for he would probably, I suppose, have become at the least permanently paraplegic, if even he had escaped with his life, had not the nature of his case been early recognised, and the treatment adopted by which he was rapidly and completely cured.

It will have been noticed that although the loss of power was almost confined to the right lower extremity (where it was complete), there was considerable cutaneous anæsthesia in both limbs; in the right, however—the side most paralysed—more than in the left. Now, we know from Brown-Séquard's discovery that in hemiparaplegia from disease or injury of one lateral half of the cord, whilst the limb of the affected side is paralysed as to motion, loss of cutaneous sensibility is confined to the opposite extremity, the paralysed limb itself being indeed somewhat hyperæsthetic. This is explained, as is well known, by the sensory fibres crossing over at once to ascend in the side of the cord opposite to that at which

they have entered. In the present case the history, and still more the result (for the man could hardly otherwise have made the absolute recovery which we have seen), show that the cord itself could not have been the seat of any destructive lesion. The mischief evidently lay in the membranes, and was more or less diffused. There can be little doubt that the inflammatory products, whilst almost confined to the back part of the left side of the cord, where they pressed upon the posterior roots, and thus caused loss of sensation in this limb, left the anterior roots of this side nearly free. In the right half, however, both posterior and anterior roots must have been involved in the inflammatory changes—hence the radiated pains and cramps, followed by loss of power and anæsthesia. I dwell particularly upon this point because it seems to me to present something more than a physiological interest. In treating a patient with complete paralysis, which is almost entirely confined to one lower extremity, if we find the loss of cutaneous sensibility greatest in the most paralysed limb, we may reasonably infer that the lesion is not situated in the cord itself, but affects its coverings, and through them the spinal nerves, and our prognosis will of course be favourably influenced by this consideration.

There is more difficulty in forming an exact diagnosis as regards the seat of lesion in the case of Margaret J—, aged twenty-nine, a married woman of respectable appearance, who came to the hospital with so much loss of power and numbness in both lower extremities (especially the right) that she could only just manage to walk with help into the consulting-room. It seemed that she had been attacked three months before with feelings of numbness and pins and needles in the feet, and that these symptoms had gradually extended upwards, in spite of medical treatment, so that her arms had become involved, and she could scarcely do anything to help herself. There was also constipation and delay with the bladder, besides a feeling as of a tight band around the waist, and "deadness up to the chest." She had been

married four years, had two living children, and one who had died. She said that a sister, aged thirty-nine, was paralysed in the legs. Her left pupil was observed to be smaller than the right, and of somewhat irregular outline, but she had no complaint to make of her sight; could read No. 1 Snellen easily, and did not remember to have ever had anything wrong with her eyes. On using the ophthalmoscope (which we employ, thanks to the wise teaching of Dr Hughlings Jackson, as a matter of routine) we found in the right eye very evident remains of disseminated choroiditis in the form of numerous atrophic and pigmented spots, whilst in the left there were old adhesions of the iris and opacities of the vitreous body. There was no history of bad sore-throat or eruption on the skin.

Ten grains of iodide were ordered to be taken three times a day. In a week she had almost entirely lost the feeling of tightness round the waist and the "deadness." In another week it is noted that there is now no numbness in her legs, and no longer any delay with the bladder. The hands, however, were still very numb and dead, so that she could not use a needle or pen. The dose of iodide was increased to fifteen grains. A week later and she had managed to do a little work, whilst by the end of a month she reported herself as being perfectly well, and we did not see her again.

From the beginning to the end of this case no direct question was asked of the patient or her husband in reference to infection.

The ophthalmoscopic appearances (which Dr Liebreich was good enough to confirm) pointed to constitutional syphilis with such force that the corroborative evidence afforded by the immediate influence of the iodide was scarcely needed to establish the specific nature of the lesion from which this woman suffered.

This case seems to me particularly instructive because, although, as it is now detailed, and with the results of treatment before us, the matter appears to be simple enough, it had evidently been very obscure to some one

who had treated the patient for three months ineffectually, and into whose mind it is pretty certain the idea of syphilis being at the bottom of this gradual loss of power and sensation had never entered. The fact of the patient's sister being paralysed at an early age would perhaps be likely to turn the attention into a wrong channel, and lead to the inference, either that the patient had congenital proclivities to instability of nervous centres, or that the case was an example of nervous mimicry.

In any case I should have prescribed iodide for this woman at first because the symptoms were suggestive of a specific cause for the disease, but I gave it with a great deal more confidence when the ophthalmoscope had shown such a condition of the eyes as pointed, if not certainly, at least with enormous probability to the presence of constitutional syphilis.

So highly favourable a result as occurred in these instances is unfortunately not the rule in cases of syphilitic paraplegia. Recovery is seldom so complete, and sometimes does not take place. An important example of a case of this kind going on to a fatal termination occurred not long since in the hospital. The following is a brief account of it, derived in great part from Mr. Broster's notes:

Mrs X—, æt. 68, came into the hospital in July, 1878, able to walk a little with the help of a stick but dragging both legs. She was of healthy aspect, and did not look so old as she was represented to be. She complained of an occasional shooting pain down the right leg. The lower extremities were not rigid, and the sensibility of the skin appeared to be unimpaired. Her great trouble was a subjective feeling of icy coldness in the feet and legs. The muscles of the thighs and legs were much atrophied. There was no marked affection of the sphincters. The patellar tendon-reflex was almost entirely absent on the right side, and somewhat diminished on the left. There was no ankle clonus.

Her account was that nine months previously, shortly

after an accident in which she had fallen heavily, she was conscious of a sense of extreme coldness in the sole of the left foot, which extended up the leg to the thigh, and this limb became weak. A month after the beginning of this feeling the right leg in its turn became very cold and weak. Gradually but surely the loss of power increased, and was continuing to become more confirmed, as far as the right leg was concerned, when she was admitted. The left leg had remained stationary at a certain point of feebleness. Three months before she came here she complained of a sharp pain in the back, in the lumbar region, which extended down the right leg.

About six weeks after her admission it is noted that the patient is confined to bed and that the power in the lower extremities is almost *nil*. She had some incontinence of urine, which contained albumen, and there was a tendency to the formation of bedsores. Great pain in the back and down the legs was complained of, with pain and tenderness around the waist and obstinate constipation. Vomiting now took place, with increasing exhaustion, and in a few days she died.

On post-mortem examination the arachnoid over the lumbar enlargement was found distended by clear fluid. On the anterior surface of the spinal cord, involving the right side more than the left, was a projecting enlargement about the length and breadth of a mulberry, extending downwards to nearly the commencement of the cauda equina. On section it was found to be of a yellowish-white colour, of somewhat hard consistence, and it occupied nearly the whole right half of the cord, encroaching also considerably upon the left. The kidneys were small and hard, with the capsules adherent in places. The bladder presented the appearances of chronic cystitis.

My colleague, Dr Ormerod, was kind enough to examine the tumour of the cord microscopically. He found a distinct line of demarcation between the tumour and the essential structure of the cord. Under a low power a thin section of the tumour showed the central part rather

opaque, yellowish in colour, faintly marbled here and there with darker spots, but otherwise structureless; no vessels were seen. Under a higher power a certain amount of connective tissue could be made out about the peripheral zone, but for the most part this appeared to be structureless. The peripheral zone under a low power was granular in appearance, with numerous spots, which looked more dense and were more deeply stained by carmine than the rest of the field. There were numerous vessels which were pervious. Under a high power the granular appearance was seen to be due to the fact that the ground substance consisted of small cells closely massed together with little or no stroma. The dark spots consisted of little masses lying in spaces around which was a ring formed partly of aggregated cells and partly of faintly-marked connective tissue. The masses themselves appeared to be formed either of densely aggregated cells or of structureless matter with indications of cells at the edges. Portions of the tumour teased out in glycerine showed small rounded granular cells without distinct nucleus, having the size and general appearance of leucocytes. There were no spindle-shaped or tailed cells. Its general structure and relations, whilst they served to exclude carcinoma, sarcoma, and tubercle, agreed with the characters of a syphilitic gumma.

Although there was nothing in the past history of this patient, who was a widow, to indicate syphilis, she was treated with iodide of potassium as soon as she came to the hospital. No specific treatment had probably been adopted during the nine months previously, when the growth must have been in process of formation. It remains a nice question whether treatment by mercury would have been more efficacious in this case, but without very clear indications of specific disease one naturally shrinks from applying vigorous mercurial treatment to a female nearly seventy years of age. That large doses of iodide will often effect what smaller doses fail in doing, and that mercury will sometimes influence in circum-

stances where iodide is inefficacious I have no doubt. An instructive example of this came under my observation a year or two ago. The patient was a man upwards of sixty years of age, who had a subcutaneous tumour nearly as large as a fist lying on the sternum. There was a history of syphilis some thirty or forty years previously. He was treated for two months with iodide of potassium, at first taking ten and then twenty grains daily, and a Plummer's pill at night. At the end of this time no alteration had taken place in the size of the tumour. The iodide of mercury in small doses was then added, and in a few weeks the tumour, doubtless a large gumma, disappeared.

LECTURE XXIII

TETANY

An example of the peculiar affection to which the name of Tetany was given by Corvisart, in 1852, has come under our observation. So far as my personal experience goes, this disorder is not common in England, whilst, on the other hand, it would appear to happen more frequently in France than elsewhere, and to French writers, until recently, we were almost exclusively indebted for our knowledge of it. In this country Dr Moxon* and Dr Wilks† have reported characteristic examples; whilst in Germany Riegel‡ and Erb§ have given excellent descriptions of the affection, and still more recently N. Weiss, of Vienna, has published a clinical lecture upon the subject.‖ Its importance rests especially upon the fact that when severe and accompanied by elevation of temperature, it may chance to be mistaken for such differing conditions as spinal meningitis, acute rheumatism, or tetanus, according as the attention of the observer is attracted more especially by one or other of the symptoms.

Susan B—, æt. 10, a scholar in the Hebrew School in Palestine Place, was admitted into hospital suffering from tonic contraction of certain muscles of both upper and lower extremities, of three weeks' duration. The

* 'Guy's Hospital Reports,' 1870.

† Idem., 1872.

‡ 'Arch. f. klin. Med.,' xii, 1873.

§ Ziemssen, 'Cyclopædia of Practical Medicine,' English translation, vol. xi.

‖ Leipzig, 1881.

right hand was somewhat swollen, the skin on the back of it being smooth and polished looking. The thumb was rigidly opposed to the forefinger, the first phalanx of which was flexed, the two last extended. The middle finger was partially, the ring and little fingers completely, flexed. (See Fig. 22.)

Fig. 22.



The left hand presented a precisely similar condition as regards the attitude of the fingers, but the rigidity was not quite so strongly pronounced. Any attempt to alter forcibly the position of the fingers gave rise to great pain, which was referred by the girl to the knuckles. The feet were stiffly extended and inverted, the toes being flexed. There were slow and slight choreiform movements in the hands. Over the knuckles there was a slight flush of redness, which, taken in conjunction with the swelling and pain from which the girl suffered—there being pain even when the fingers were not touched—gave at first no little of the aspect of acute rheumatism to the condition, an idea which was at once dispelled, however, by observation of the rigid condition of the hand. The temperature was 100° Fahr., and the pulse somewhat quick. She complained of pain in the feet and hands, and to a certain extent in the arms, but not in the legs.

It was elicited that the child was one of a large family in which there were no cases of neurosis. Her intelligence appeared to be somewhat below the average.

She had enjoyed good health, and had never suffered from acute rheumatism or any ailment except measles in early life. She was well nourished, but somewhat anæmic in appearance. There was a peculiar expression about the lower part of the face, a tendency towards a constant somewhat grim smile, not amounting to, but suggestive of the sardonic grin observed in tetanus; she could move the muscles of her jaw, however, freely enough.

It seemed that, three weeks before, on waking up in the morning, the girl found her hands in the position which I have described. There had been no previous diarrhœa. She was resident at school at the time, and remained there for a fortnight, when she went home to her parents. During this time her hands only were affected, and she attended her class, but as she was unable to grasp a book, a schoolfellow had to hold it for her. We could not ascertain whether the rigidity during this time was intermittent or continuous. Her schoolmistress had called it "the cramp."

There was rather a vague story of her having been frightened by a man who "poked his head through the window." Her parents said that when she came home her nights were restless, and she bored her head into the pillow.

When undressed and put to bed it was noted that the spinal muscles appeared weak and scarcely able to support her in a sitting posture. She was restless, throwing her head about and rubbing the occiput into the pillow. She had difficulty in holding her head erect, as it tended to fall on one side or the other, and when told to keep it still it was somewhat jerked. There was no tenderness of the spinal column on pressing it at any point, or during movement.

She had no headache, or any intellectual disturbance. The only pains of which she complained were in the hands and feet. No intermission of symptoms was observed, and the muscles did not become relaxed during sleep.

On the day after her admission the following obser-

vations were made. A cord was tied tightly round the right forearm for a few minutes without effect upon the spasm, but with the result of causing increased pain. The right hand was placed for five minutes in cold water without modifying the contractions, but with the effect of causing intense pain in the hand, which extended up the arm. The left hand was then placed in warm water. This caused no pain, and did not influence the contractions. I then tested, electrically, the irritability of the ext. communis digitorum and ext. carp. ulnaris muscles, which responded normally, as far as could be judged, to the induced current; but I could obtain no response whatever in any of the dorsal interossei muscles to a current strengthened so as to be acutely painful. At the same time the abductor minimi digiti answered readily. The interruption of a constant current from sixteen cells (Stöhrer) was equally inert.

The patient was unable, voluntarily, to extend still further a partially extended finger, or to flex more completely one already somewhat flexed.

She had passed a very restless night. The temperature was now normal.

On the following day she had greatly improved. The hands were relaxed to a considerable extent, and there was now some slight power of flexion and extension, but accompanied by pain at the knuckles. On testing her electrically we succeeded in exciting contractions in the interossei, but with a strength of induced current far greater than was required for the ext. dig. communis, or for the corresponding muscles on my own hand. After once getting the muscles to move, however, a smaller strength than was at first necessary succeeded in causing them to contract, but they still needed a much greater strength than did the interossei of my own hand.

Next day there was little or nothing left of contraction or pain, but the hand wore a peculiarly helpless appearance, much as though it had been for a long time confined to a splint. (See Fig. 23.)

The treatment consisted in keeping the patient in bed in a private ward, and giving her light nourishment. Whether the applications of the electric current aided in removing the symptoms remains undecided. No drugs were administered. In a few days the girl was sent to the Convalescent branch at Finchley, and when I saw her, a week since, she had had no return of the affection.

FIG. 23.



Let me note here that in reply to a question the patient said that after her hands had "got bad" one of her school-fellows was affected in a similar manner, but only for one evening.

I call attention to this because we shall see that the disorder may sometimes occur in epidemic form. Trousseau, to whom we owe, not the first account of this disease, because it had been described by Dance, in 1831, in the 'Archives Générales de Médecine' under the name of "intermittent tetanus," as well as by several subsequent writers, but the first description of the affection which attracted any particular attention, relates several cases, all occurring in young adults, although he mentions besides that it is not uncommon to see the complaint in children.

In a case published by Dr Wilks* the patient was a child three years old. Its feet were drawn into the position of talipes varus, the legs flexed on the thighs, and these on the abdomen. The muscles of the arm were less

* Loc. cit.

affected, the fingers being extended and brought together in a conical form. The child was constantly calling out in consequence of the pain caused by the cramp.

He relates, too, the case of a schoolboy, aged sixteen, who was so severely affected by cramps that his medical attendant was much alarmed at his condition, fearing he had inflammation of the spinal cord.

Dr Moxon, writing upon this subject in 1870,* describes the case of a boy, three years and a half old, in whom contraction of the extremities was accompanied by embarrasment of breathing, with croupy sounds. In the paroxysms the arms were half flexed, the hands strongly pronated and partially flexed at the wrists, the thumbs were bent across the palms and adducted, the fingers semiflexed and clustered over the thumbs, so that the backs of the hands only were offered to view. The hands were swollen, and the dorsal veins distended with blood. The great toes were flexed and adducted, and the other toes flexed and gathered towards them.

Dr Moxon makes the interesting suggestion that the paroxysms of tetany are very like those convulsive disorders which have occurred epidemically from the use of ergoted rye. Romberg thus describes the ergot convulsions:—"The hands and feet are attacked by cramp of the flexor muscles; the fingers of both hands are bent like hooks, the thumbs being pushed under the fore and middle fingers in an oblique direction; the wrist is strongly curved inwards so that the hands assume the shape of eagle's beaks; the toes are doubled under the sole of the foot."

In these cases of ergot poisoning the spasms go on to the production of tetanus or trismus.

So frequently did Trousseau find the subjects of tetany to be women who were nursing, that at first he styled the disease "rheumatic contraction occurring in nurses." But in process of time he found that nursing was not the only condition favourable to its development, although, as he thought, the most frequent. Diarrhœa

* Loc. cit.

and the influence of cold seemed to him capable of provoking it. I have recently met with a case in private practice (to which I will refer presently) in which nursing and diarrhœa were concomitant with the affection.

Trousseau describes (and all other observers whose accounts I have seen confirm his observations in this respect) paroxysms of tonic contraction lasting from a few minutes to two or three hours, and recurring in series; and he found that pressure on the arteries of the limb or in the direction of the nerve-trunks would reproduce the contractions when they had ceased. He found also, in many cases, that he could arrest the paroxysms in the upper extremity by making the patient dip his arms and hands into a basin of cold water. What may have happened before the girl came here I do not know, but during the time she was in hospital the contraction was permanent, not intermittent. We had, therefore, no opportunity of testing the effect of pressure upon the arteries. As before noted, the influence of cold water in relaxing the contractions was *nil*.

Dr N. Weiss, of Vienna, in a recent clinical lecture,* remarks that there are two points of especial importance in regard to the diagnosis of the affection. One is the so-called Trousseau phenomenon, to which I have just referred—the reproduction of the paroxysms at will by compressing the affected parts either in the direction of their principal nerve-trunks, or over their blood-vessels, so as to impede the venous or arterial circulation. The other is a peculiar increase of electric irritability in the affected muscles as well as in their nerves.

Several observers, including Kussmaul, Benedikt, Erb, and Chwostek have found, in these cases, that the irritability of the motor nerves is materially increased to both induced and galvanic currents.

Erb, for instance, discovered in two out of three cases of tetany, which he most carefully examined, an extraordinary elevation of electrical excitability. This was

* "Ueber Tetanie," 'Samml. klin. Vorträge,' 189, Leipzig, 1881.

observed in the nerves of the extremities going to the affected muscles, but not in the portio dura or its branches. The elevation of the galvanic irritability was apparent, not only in the disproportion between the strength of motor and sensory reaction, but by the early production of muscular contraction with the negative pole on the nerve (Ka S z), and also by the production of a tetanised condition of the muscle (Ka S Te) by a strength of current ordinarily disproportionate to such a result, and finally by a circumstance which had not previously been noted in man, *i.e.* the occurrence of tetanisation of muscle when the electric circuit was opened with the positive pole on the nerve (An O Te), the current being of moderate strength. The most remarkable increase of electrical excitability corresponded with the crises of paroxysms of contraction, and as the patient recovered a normal state of irritability was found. Chwostek has confirmed these observations in eight cases of his own, in all of which he found great elevation of galvanic and faradaic excitability. He also produced An O Te in some of these cases, and in two of them Ka O Te occurred with what was quite a moderate strength of current. Chwostek found a similar increase of excitability in the portio dura. But this was in cases in which cramp had occurred in the district innervated by this nerve. In Erb's cases this district had been free from cramp. Weiss also lends his testimony to confirm this account of increased excitability, but he was able in one only out of twelve cases to produce An O Te, in spite of the fact that in all the cases there was such an increase of irritability that Ka S Te and An S Te were fully expressed. We had not, unfortunately, the opportunity of testing this point in the case described, owing partly to the short duration of the girl's symptoms after her admission into the hospital. There was not, however, any obvious increase of irritability in the *muscles* of the forearm as shown by their response to the induced current. The complete loss of irritability which was discovered in the dorsal interossei muscles is not described

in any work which I have seen, and at first sight would appear to negative completely the idea that, in this case at least, there was any increase of irritability in the motor nerves supplying them. But I am disposed to think that this loss of irritability was only apparent, not real, and owed its origin to the œdema of the hand, which interfered mechanically with the application to the interosseous muscles of a current of sufficient density to excite their contraction. I have often had occasion to observe that a very moderate amount of œdema will produce this effect.

It is reasonable to refer the œdema in this patient to the effect of blood-stasis from compression of the veins by the tonically contracted muscles. Whether or not there was any increased irritability of the nerves I am unable to say, in the circumstances described, but it is clear that, supposing such did exist, it would not of itself explain the phenomena of contraction. I have often discovered marked excess of electric irritability in the motor nerves of an extremity due to gouty, rheumatic, or traumatic neuritis, without observing anything akin to powerful tonic contraction of the muscles supplied by them. But such a condition of electrical excitability of nerves as is recorded by these observers is not at all common.

I am indebted to Dr Barlow for calling my attention to an account of what appears to have been quite an epidemic of tetany. A report of the occurrence was made to the Société de Biologie in Paris.* The persons attacked were girls in a school at Gentilly. Their age varied from ten to fifteen years, and twenty-four girls were reported as patients. In addition to these children there was one adult—a teacher. No diarrhœa preceded the attacks. Close to the school was an establishment for boys, none of the inmates of which were attacked.

It seems, from the account given by M. Magnan, that the first case was in a girl, æt. 10. On July 15th her right hand was contracted, the fingers flexed upon the hand, the wrist slightly flexed upon the forearm. The

* 'Gazette des Hôpitaux,' November, 1877.

wrist, elbow, and shoulder were painful. The attacks lasted for periods varying from half an hour to an hour, movement being possible in the intervals, but difficult. The disorder continued throughout August, September, and October, but towards the end of November she recovered.

The second case, a girl, æt. 10, was attacked on October 10th in both hands, and the third, a girl of 12, on November 7th in both feet. These two cases got rapidly well. The fourth was a girl, æt. 10, in whom both hands were attacked; and the fifth, a child of 8, who suffered from convulsive seizures of a clonic as well as tonic character. She had been much impressed with the sight of the others.

In this first series the cases had been, so to speak, isolated and scattered. But on November 13th a complete epidemic broke out. On the same day eight cases were observed in one class alone, and on the following day eleven cases. The school was then closed, and of the nineteen cases which developed on the 13th and 14th November all speedily got well. There were no cases in another girls' school, or in the adjoining boys' school. People began to talk of mysterious influences upon the house, and the children were greatly impressed. The credulity of the children, the *bizarre* ideas with which they were seized, the immediate cessation of troubles when the school was closed, suggest at once that what happened was something analogous to the epidemics by imitation observed among the "convulsionnaires" of the seventeenth and eighteenth centuries.

Œdema of the wrists was found in two of the cases only, the first attacked, and it was thought that these only were real, the others being imitative.

It is difficult to associate with such cases, one with the characters presented by a patient whom I have lately seen in private practice, but as will be observed, by reference to Trousseau's account of the disorder, there is a real relation between them.

Mrs ——, a married lady, æt. 44, came to me in

November, 1881, complaining of attacks of cramp or contraction of the hands and feet. It seemed that before her last confinement (the child was ten months old when I saw her) she noticed some numbness in her hands which spread to the rest of the body and face.

In August last she began to get attacks of contraction of the hands, during which, as she described to me, the thumb was adducted and bent into the palm, whilst the fingers were strongly flexed over it at the metacarpo-phalangeal joints. This state of contraction was attended with intense pain. It might last, with remissions, for a whole day, but was sometimes over in an hour. It was apt to occur in the morning on awaking. Her feet were also, she said, frequently contracted at the same time, and in them likewise the pain was most severe and distressing. I did not myself see anything of these attacks, but from the description given of them, both by the patient and her mother, who accompanied her, the pain appears to have been very great indeed. On one occasion during a paroxysm the arms were strongly flexed at the elbows. The jaws did not become fixed (I inquired particularly on this point), but she complained of what she described as a "jumping about" of the nerves of the face. She suffered both during the paroxysms and at other times from dizziness.

There was something very striking and peculiar about the aspect of this patient. Her face was of a dusky hue, and so thin and drawn that it reminded one a good deal of the look of a person in the collapse of cholera. It appeared that she had given birth to eleven children, and on each occasion whilst nursing them had suffered from diarrhœa. The present child she had only nursed ten weeks, but the diarrhœa which had affected her during that period continued afterwards and was still troubling her. She was having seven or eight motions a day, pale and yeast-like according to her description. Her mother told me that she had completely altered in appearance, having formerly been stout and, to use her own expression, a

"jolly-looking person," with a healthy colour and round face. She had lost two or three stones in weight. I could find nothing wrong with her lungs or heart, and was not able to explain the cause of the diarrhœa, which continued to distress her in spite of treatment.

As I have remarked before, Trousseau found this disease so commonly associated with the nursing period, that he at one time called it "rheumatic contraction occurring in nurses," but further experience showed him that nursing was not the only condition favourable to its development, though he believed it to be the most frequent and active cause of these intermittent contractions. The influence of diarrhœa as an exciting cause, which had first been clearly pointed out by Dr Lasègue, was confirmed by Trousseau, who found it almost constantly present. It is interesting to note that in 1854 he met with many cases of tetany in individuals who had suffered from epidemic cholera. My patient, as I have mentioned, was the subject of chronic diarrhœa, and her attacks began when she was nursing her child. Trousseau's description of the attacks accords so well with the symptoms from which she suffers, and is withal so graphic, that I will quote it: —"The patient has a sensation of tingling in the hands and feet, and then feels some hesitation, some impediment in the movements of his fingers and toes, which are not so free as usual. Tonic convulsions then set in, the affected limbs become stiff, and the will cannot completely overcome this stiffness, although it struggles with it, and the patient can still use within certain limits the contracted muscles, can move and even extend his fingers. The involuntary contraction increases, becomes painful, and is exactly like a cramp, to which, indeed, the patient compares it. In the upper limbs the thumb is forcibly and violently adducted; the fingers are pressed close together and semiflexed over the thumb; in consequence of the flexion of the metacarpo-phalangeal articulation and the palm of the hand being made hollow by the approximation of its outer and inner margins, the hand assumes a conical

shape, or better, the shape which the accoucheur gives to it when introducing it into the vagina. This aspect of the hand, which you will most frequently meet with, is so peculiar that it is oftentimes sufficient by itself to characterise this kind of contraction. In some cases the index-finger is more powerfully flexed than the other fingers, and is practically bent under them; in other cases the flexion is more general and complete. The thumb is turned into the palm and hidden by the fingers, which are themselves bent, with such force that the nails leave an imprint on the skin, and they are so squeezed together that in a case recorded by Dr Hérard sloughs actually resulted from the prolonged pressure. The thumb alone may be affected while the fingers are scarcely contracted, but such cases are rare, and it more commonly happens that the contractions spread to other parts, the wrist becoming flexed and the hand turning forcibly inwards, the patient having lost the power of straightening it. In the lower limbs the toes are bent down towards the sole and press against one another, while the big toe turns in under them, and the sole becomes hollowed out in the same manner as the hand." These tonic convulsions, he remarks, may last for several minutes or some hours. Then there is a remission, the parts become movable, and after a variable period of rest fresh paroxysms recur, the series of which constitutes the attack, which may be prolonged for several days, "and even for one, two, or three months."

Although this ailment is in general of no gravity, it would appear to have occasionally terminated fatally. In such cases the contractions have not been confined to the extremities, but have affected also the muscles of the face and jaws, and have apparently brought death about by attacking also the mechanism of respiration. This termination must be exceedingly rare.

It is stated by Aran that the local application of chloroform has been attended with success in alleviating the cramps. In Wilks' case cold-water bandages appeared to afford ease.

A very few words on the subject of the pathology of this disorder, about which at present we are very ignorant. If we examine the position of the hand (Fig. 22) we find that the muscles, which are in a state of contraction, are innervated, some of them by the ulnar, and some by the median nerve. Are we then concerned with some source of peripheral irritation of these nerve-trunks in the arm, as would be implied by the heightened excitability to electric currents which has been described by some observers? This seems to me contradicted by one important fact. It will be noted that the second finger has nearly escaped the contractile influence which causes the third and fourth to be flexed rigidly upon the palm. The same exception existed also symmetrically in the left hand. It is an everyday experience that where an influence is exerted upon a nerve-trunk, all the muscles supplied by that trunk below this point will be included in the effect of that influence, whether it be of an irritative or paralysing character. Unable, therefore, in this case to refer the lesion to the nerve-trunks for the reason stated, we must look to some nervous centre as the source of the influence exerted in such a case as this. The continued muscular contraction—the "tetanised" state of the muscles—is due, no doubt, immediately to a series of nervous stimuli following quickly upon one other; a somewhat less degree of tetanisation is represented by minute fibrillary contractions, such as were observed in the second patient whose case has been described.

Whether these exciting impulses proceed from the central grey matter of the cord, the cerebellum, the ganglia at the base of the brain, or the cortical grey substance, we do not know. The condition is usually referred to an exaggerated reflex excitability of the cord, but this of course is no explanation. My colleague, Dr Hughlings Jackson, has suggested that the phenomena of tetany, like those of certain conditions of contraction in hemiplegic children, of athetosis, and some other kindred states, may perhaps be explained as a result of

defective antagonism of the cerebellar influence. He believes that in health the whole of the muscles of the body are doubly innervated; innervated both by the cerebrum and cerebellum, there being a co-operation of antagonism between the two great centres. Whilst the cerebrum innervates the muscles in the order of their action from the most voluntary movements (limbs) to the most automatic (trunk), the cerebellum innervates them in the opposite order. This also is supposed to be the order of their degree of influence on parts; and is equivalent to saying that the cerebellum is the centre for continuous movements, and the cerebrum for changing movements. His speculation would be that in such a case as I have described the cerebral influence was temporarily removed; hence unimpeded cerebellar influence, and rigidity of muscles which in health the cerebrum chiefly innervates. The fact which I have mentioned, that this little girl was unable, when requested, to execute movements with her fingers, is evidence which to a certain extent goes in favour of Dr Jackson's view. Not only could she not, for example, bring to a flexed position a stiffly-extended finger, but she was unable to flex any further a finger already partially flexed. This inability, it appears to me, is not to be explained by a simple condition of heightened reflex excitability of the cord, but must indicate either that there was some obstruction to the passage of pulses of nervous force from the cerebrum, or that the cerebral functioning was temporarily in abeyance. But whether a negative cause, so to speak, the withdrawal, namely, of one kind of influence, and the consequent permission to another kind to have unlimited sway, is sufficient to account for the symptoms without the hypothesis of such a temporary pathological change as would induce of itself overaction of a particular nerve-centre, must for the present remain doubtful. In any case there is something attractive in Dr Jackson's speculation, and we are not likely to commit the error in logic of supposing that because the assumption of the existence

of a principle of antagonism helps to explain the symptoms of such a disorder as tetany, the consequent explanation proves the existence of such a principle of antagonism!

The general resemblance of tetany to conditions styled hysterical is evident, and it is quite possible that the faradisation of the girl's hands acted curatively in the same manner as though the case had been one of hysterical trismus or aphonia. In this connection, the interesting fact, first noted by Trousseau, that even after the patient has been free from contractions for seventy-two hours or more, these may be reproduced by compressing the affected parts in the direction of their principal nerve-trunks, reminds us of the recent observations of Charcot and Richter* in cases of hypnotism in hysterical subjects. They find that there is such an amount of hyper-excitability of the motor nerves that mere pressure upon the trunk will throw into contraction the muscles to which the nerve is distributed.

* 'Archives de Neurologie,' October, 1881.

LECTURE XXIV

ON PHENOMENA OF TRANSFER PRODUCED IN EPILEPTIC PATIENTS BY THE APPLICATION OF ENCIRCLING BLISTERS

THERE is a young woman in the hospital who has been hemiplegic on the left side for the last ten years. Her paralysis is owing to a depressed fracture of the right side of the skull which she sustained from a fall upon the head. For the last six years she has been subject to fits, which have latterly commenced with a drawing up of the left arm (and sometimes of the left leg), of which she is quite conscious. In her case a method of treatment has been adopted which I have found to be attended with certain remarkable results in causing a transfer of the premonitory aura in several examples of unilateral convulsions. My first observations in this matter were made fifteen years ago, and were embodied in a paper which was published in the 'Practitioner' for October, 1868. Before entering upon the details of the present case it will be convenient if I relate my experience upon the subject as it was contained in that paper, as well as some further examples of a similar character which I brought before the British Medical Association at the annual meeting in 1880.

It is known that where a very marked local sensation precedes a fit of epilepsy, a diminution of the number of attacks, and even sometimes a cure, may result from treatment directed to the apparent source of irritation. In Brown-Séquard's work on 'Epilepsy,' reference is made to cases of epilepsy successfully treated by the application of a ligature around a limb or finger, section of a nerve, amputation of a limb, extirpation of a tumour, foreign

body, or tooth; or by the expulsion of worms or calculi. The use of a ligature especially is very familiar to many epileptics themselves, who are frequently able to check a fit by tightening a strap carried on a limb in which the precursory aura is felt. Cauterisation or blistering of the skin at the starting-point of an aura has also apparently been practised with success, although there is much difference of opinion on the part of authorities as to the value of this treatment.

In the course of some observations upon patients I found some curious results follow the application of blisters, and especially of encircling blisters, to limbs which were the channels of very marked *aura epileptica*.

In January, 1868, Alfred M—, a boy, æt. 15, was brought to me at the hospital. He had received a kick on the head seven years previously, followed in a few months by epileptic fits, which had continued ever since. At first he used only to feel a tickling sensation in the left arm once or twice a day. This went on for three or four months, and then one day the tickling was followed by insensibility and convulsion, and since that occasion the same results had always followed the feeling of tickling. At the time of his application, he had had from five to ten fits daily for the four months preceding, without any intermission whatever. He indicated a spot about the centre of the left biceps muscle as the point whence the fit always started. A strip of blister was at once applied just above this spot, and was repeated once a week. No drug was given. For a month after the rising of the first blister he was absolutely free from fits, until one day he received a blow upon the arm, and a fit took place. Ten days afterwards there was another, and then gradually the frequency of the attacks was increased, so that he had one every two or three days, but they were not now preceded by any aura. At this time I gave him fifteen grains of bromide of potassium every night, and then the same dose twice daily, but without effect; the fits became still more numerous and severe.

On June 1st the aura had returned, and then an encircling blister an inch wide was made around the upper part of the left arm, without, however, producing much effect upon the number of attacks. But he now complained, for the first time, of tickling about the middle of the left *leg* before each fit, the aura in the arm having entirely disappeared.

Susan S—, æt. 10, was admitted on March 17th, 1868, having had fits for eight months. She had sometimes had from thirty to forty attacks daily, and during the last month they had been constant, to the number of four or five in the day, and as many at night. Each fit was preceded, for about five minutes, by a tickling in the middle of the left biceps—the same situation as in the last case. In her case, likewise, the tickling alone had occurred daily for six weeks, and then had merged into an attack of insensibility and convulsion. For a week after her admission nothing was done, and she continued to have fits both by day and night. Then a blister was applied, *half* encircling the upper part of the left arm. No effect followed this application. Bromide of potassium at first in ten, and then in twenty-grain doses, was given to her, but with no diminution in the number or severity of the fits. On April 14th I painted a ring of blistering fluid an inch wide, so as to completely encircle the left arm, above the seat of the tickling. Before the blister rose she had three fits; afterwards none occurred till the 18th, and then she had four very slight seizures. From that time till the date of my report (more than five months) she had not had a single attack, nor had she felt the tickling. No other treatment was adopted.

Harriet M—, æt. 14, applied to me in October, 1867, having had three or four fits daily for two years. In each instance the fit was preceded by a sensation of numbness in the *left* wrist. Under the use of bromide of ammonium the fits became much diminished, but in May, 1868, they again increased in number and severity. At this time a ring of blister was made around the left fore-

arm two inches above the wrist by the application of blistering liquid to the skin. During the next fortnight she had three fits. The seat of the aura was now, however, for the first time changed. It was the *right* wrist which now became numbed before the attack, and not the left.

Jane B—, æt. 27, had been epileptic for thirteen years, the fits occurring two or three times daily or sometimes holding off for a week. The fit invariably began with a cramp in the right hand or right leg, and an attack had frequently been checked by tight pressure upon the right wrist. Under bromide of potassium the fits became greatly diminished, and after some months they ceased altogether for a period of several weeks. During this time, she had frequently had threatenings of attacks, which were always stopped by a friend pinching the right wrist, and forcibly flexing the hand. Occasionally a tape would be tied tightly around the upper arm, and this would suffice to prevent the attack. So she went on for six months. Then a ring, one inch wide, was painted with blistering liquid around the right forearm, two inches above the wrist, and the use of drugs was suspended. The fits shortly afterwards recurred, and continued with varying intervals. A change now took place in the premonitory symptoms. It was not the right hand alone which was the seat of the aura, but *both hands* became cramped before a fit.

William C—, æt. 16, employed in a printing office, applied at the hospital on November 26th, 1879, on account of fits from which he had suffered for two years. The fits were attended with loss of consciousness and convulsion. Recently there had been about two each week. In every instance the fit had been preceded by a warning which was always the same, and was thus described. The great toe of the left foot would become flexed and cramped under the others. Then a "numb" feeling would travel up the leg as far as the heart, and then the leg would begin to jump. If some one now leaned his weight upon the leg the attack would sometimes stop at this point.

At other times, in spite of this, the numbness would travel to the left side of the face, which then felt as though it were being pinched hard. Then "it," according to his expression, "went right up into his head, and he did not know where he was."

The boy was ordered to take fifteen grains of bromide twice a day. When seen a week later it was reported that no fit had occurred, but there had been two warnings of the kind described. The left great toe had become flexed and numbness passed up the leg, but stopped at the knee. The knee then began to shake very much, and had to be held down by some one; and then, after a little while, the patient recovered. The bromide was now stopped, and a little simple camphor mixture given. At the same time a blister one inch in width was made to encircle completely the left leg just above the ankle. When the patient came to the hospital a fortnight afterwards he reported that no fit had occurred, but that on four occasions the *right* leg had been seized with numbness and shaking of the character which had previously in every instance attacked the left leg. The bromide was now again resumed, and the patient did not have any return of attacks whilst he was under observation.

Mary M—, æt. 20, came to the hospital on December 10th, 1879, on account of fits. She complained of what she called "a tingling" from her left shoulder to the end of the middle finger of the left hand which lasted a minute or so, and recurred as often as a dozen times a day. Occasionally there was no sensation of this kind for a few days, but she never had a whole week's immunity from it. Sometimes this tingling was the precursor of a fit, but more frequently she had the tingling without any fit following. Now and then, on the other hand, a fit took place without any tingling preceding it. On December 17th she was ordered a small square blister at the bend of the left elbow. On January 21st, 1880 (one month afterwards), she came and reported that she had not had any repetition of the tingling since the

application of the blister. But she added that now *both hands* would frequently jerk up and the *left foot* also.

Harriet C—, æt. 19, was brought to the hospital on December 10th, 1879, on account of fits from which she had suffered for two years. Each fit had been preceded by a drawing up of the three inner fingers of the left hand, of which she was always conscious. Then the attack followed, in which she became insensible and cyanosed, convulsions taking place, which were confined to the limbs on the left side of the body. During the fit she bit her tongue. The last fit occurred two days before applying at the hospital.

She mentioned on that occasion that she had then little or no power of grasp with the left hand, the paresis dating from the last attack two days previously. By the dynamometer the grasp of the left hand was found to be 0. The ophthalmoscope showed no change in the fundus oculi. There was no family history of nerve disorder or insanity.

A blister one inch wide was placed so as to encircle completely the left wrist. One week later, December 17th, she came again, and stated (without any question being directed to the subject) that the day after the application of the blister the left hand had quite recovered its power of grasp, but that the *right hand* then became weak. The dynamometer now showed the left grasp to be 40° the right 18°. There had been no drawing up of either hand. She was then ordered bromide, but I do not know with what result.

If it were necessary I could multiply such cases as these, but those I have narrated are sufficient to show that a very remarkable influence may often be produced by blisters, and especially by such as encircle the limb in which the aura is experienced. The transfer, which was so marked in all these cases, does not always occur, even when, as sometimes happens, the influence of the treatment in retarding the occurrence of a fit may be evident. There is a child now in the hospital in whose case the treatment has been applied without any very noteworthy result,

except a marked delay in the repetition of the attacks. The patient, Amy D—, is a very intelligent girl, ten years old, who came into the hospital on January 11th, 1882, having been subject to fits for four months. On the first occasion she had two fits in succession. Since then they have recurred about every week or two, and she has not had more than one in a day. In her case the attack begins with numbness, pins and needles, and pain in the little finger of the right hand, and this feeling goes up her arm and spreads to the right side of the head, at the top, and stays there. At the same time the feeling described spreads from the arm down the right side of the chest and abdomen, but does not go below the waist. She does not feel anything in the right leg when a fit is coming on. Following the numbness the right hand is clenched and drawn up; then it shakes, and is so painful that she cries. Usually she does not lose consciousness, and she says that she holds her right arm with the left hand whilst it is being convulsed. After a fit she sometimes feels as though she could not speak, and the right arm, for most of that day, is weak. Immediately after the fit, indeed, she cannot use it at all. So, also, for half an hour after an attack she loses her speech.

On January 21st a blister was made to encircle the right wrist, and this was repeated a week afterwards just above the first.

On the 10th February (twelve days after the application of the second blister) she had a fit in the evening. I learn from Dr Coxwell's notes that the right arm and leg were drawn up, the patient screaming loudly. There was no loss of consciousness.

On February 27th she complained of pain, of aching character, down the right arm. When the arm was rubbed the pain went off. The feeling reminded her of the usual commencement of a fit, except that there was no numbness in the little finger, which she had always been accustomed to have.

On the 31st of March she suddenly felt a sensation like

pins and needles in the right little finger, and the right hand became clenched. A pain then mounted up the arm to the right side of the face. At the same time the arm got stiff and rose at the shoulder, the elbow being flexed. The pain and the elevation of the shoulder lasted three minutes, then all four limbs jerked for about a minute. The nurse thinks she would have fallen if she had not been supported. She did not lose consciousness; the face was unaltered in expression; she did not pass urine or bite her tongue in the attack. She cried on account of the pain in her arm. Douching with cold water had no influence upon the seizure. Since that time (two months have passed) she has had no hint or threatening of an attack.

The result of the application of encircling blisters in this case, although it has been of signal service in preventing the occurrence of fits, has been in contrast with that which has happened in most of those previously described, as far as any kind of transfer is concerned.

On the other hand, in the young woman who is hemiplegic from an injury to the head, a very marked influence of this character was produced, and as the case is especially interesting from other circumstances I will describe it in some detail.

Elizabeth D—, æt. 19, was admitted into the hospital on August 2nd, 1881, suffering from left hemiplegia and fits. When she was nine years old a beam supporting a swing upon which she was amusing herself gave way, and she fell on the right side of the top of her head. She became insensible, and remained so, it is said, for a fortnight, when it was found that there was complete loss of power in the left arm and leg and some paralysis also in the left side of her face. Her leg improved, so that she is able to walk about with a stick, but the arm and face have remained as powerless as they were.

Six years ago (four years after the accident) she began to have fits. At that time she would become unconscious at once, without any warning, but latterly the fits have been preceded by "drawing up of the left arm and left

leg, giddiness (the room revolving, as it appears to her, from left to right), stars twinkling in her eyes, and sometimes 'twittering of the tongue.'" The earlier fits were characterised by loss of consciousness, but now she can hear and understand what goes on around her although she cannot speak. The fits mostly occur at the catamenial periods.

On examining her head we find a somewhat circular depression (of a size that may roughly be described as just capable of receiving a halfpenny) situated about 3·5 centimètres to the right of the middle line, and eight centimètres above the upper margin of the right ear. The depressed spot is felt to be tender on percussion, but not when pressure is made upon it. Pain is often experienced about this spot after a fit. The left arm is almost absolutely powerless. The patient can move the elbow slightly, but not the wrist or fingers. The muscles, which are not wasted, are very rigid, the wrist and fingers being strongly flexed and the forearm pronated. In the left leg the knee-phenomenon is much increased, and there is ankle-clonus. There is no affection of cutaneous sensibility. The seat of the depression in the skull corresponds with the upper extremity of the fissure of Rolando, and in general terms with Hitzig and Ferrier's centres for the arm and leg movements of the opposite side.

On September 29th she had a fit after having complained, on the day preceding, of much pain in the right side of her head about the seat of the depression. The pain seemed to her like a narrow band passing from the depression to the temple, and did not extend outwards to the ear. She described a feeling of giddiness as the pain came on; the room revolved to the left, and she herself appeared also to turn to the left. She saw a number of coloured stars. The fit occurred whilst at breakfast, and the details of it having been noted by an intelligent nurse, can be depended upon. The patient on being spoken to took no notice. No change of colour in the face was observed. With difficulty and much help she walked into the dormitory, her eyes being open. She

then lay down, both eyes twitched, she made a sobbing noise, and the left arm was drawn up so that the hand touched the shoulder. It was not observed that the left leg moved. The left arm became stiff for a minute or less. She did not answer when she was at this time again spoken to, but consciousness was speedily recovered. The patient says that she had excessive pain in the head. She saw nothing but stars; the room appeared to revolve from right to left, and she seemed as though she were also turning from right to left. The following morning she had much tenderness at the seat of the depression in the skull, and from that point to the right temple. She was ordered twenty grains of bromide of potassium to be taken three times a day. The patient says she feels the left arm being drawn up in the slight fits, but not in the severe ones.

On the same day, three hours after the occurrence of the fit just described, the patient was heard by the nurse to "gurgle." She was then found with her head lying to the right, and her limbs convulsed clonically on both sides. When seen by the resident medical officer three minutes afterwards she was unconscious. The pupils, which were somewhat dilated, reacted to light. She spoke about a minute afterwards. It seemed probable that this attack had begun, like the last, in the left side and then spread to the right, but the commencement was not observed.

On September 30th a strip of blister, one inch wide, was applied so as to encircle the left forearm just above the wrist. Another blister was applied in the same manner just above the former one on October 24th. No fit had occurred in the interval.

On the 31st of October she had another fit, the whole of which was witnessed by the resident medical officer, Dr Beevor. There was apparently no warning; the head, eyes, and the whole body were turned over to the right, so that she nearly fell out of bed. The *right* arm was drawn up, and the muscles of the face were very much contracted on the

right side for about ten seconds. She was then convulsed in the muscles of the limbs on the *right* side of the body, not in those of the left.

She had no more fits after this, and was discharged from the hospital on January 20th, 1882.

The circumstances of this case are especially interesting because the depression in the skull on the right side of the vertex, with the hemiplegia of the left half of the body, afford abundant evidence that the convulsive seizures are connected with an organic affection of the brain. And here I would say that in all the instances described, in which the transfer took place, we had to do with an epileptic and not an hysterical condition. In the third case which is related, that of Harriet M—, the subsequent history made this manifest. Four years after the treatment had been applied, which resulted in premonitory numbness of the left wrist being transferred to the right, she was again admitted into the hospital, where she died. An autopsy (which was made by Dr Gowers) disclosed a small, reddish, semi-translucent tumour, the size of a walnut, in the white substance of the left hemisphere of the brain. It was situated above the middle of the lateral ventricle, and contained two calcareous masses.

It is right to mention that every precaution was taken in all these instances to prevent the patient from entertaining the slightest idea of what was expected to happen. No comment was made in the patient's presence upon the unilateral situation of the aura, and the blister was ordered without any mention being made of the results which had occurred in the case of other patients treated in a similar manner.

As will be observed, there was little or no uniformity in the mode of transfer shown in these examples. In three cases there was either tickling or tingling in an arm; in one, under the influence of the blister, this disappeared altogether; in another it was transferred to the leg of the same side; in the third both hands and one leg were convulsed. In one instance a sense of numbness in the

left wrist was transferred to the right wrist. In another numbness and shaking of the left leg were transferred to the right leg, whilst in another cramp in the right hand or right leg went on to affect both hands. A transient hemiplegia of the left arm, following a fit, in one patient became transferred to the right arm. In the case of the young woman with hemiplegia from depressed fracture of the skull, convulsion of the muscles of the limbs and face on the opposite side of the body to that which had formerly been affected, followed the application of two encircling blisters to the forearm, which had been the original seat of convulsion.

I do not feel myself in a position even to attempt an explanation of these phenomena, and can only record them. They appear to me to possess considerable interest in more than one direction. The paper to which I have referred, embodying my earliest experiences, was published some years before those experiments with metals were performed by Charcot and others in France upon cases of hysterical hemianæsthesia which were attended, as is well known, by the removal of the anæsthetic condition and its transfer to the opposite half of the body. As regards the question of hysterical hemianæsthesia, it appears to me that the results which I have described tend in a considerable measure to confirm the reality of the alleged analogous phenomena in the hysterical patients. Indeed, when the first examples of transfer of hysterical hemianæsthesia took place, so far from finding in that phenomenon grounds for doubting the *bona fides* of the patients who described its occurrence, my own experience of several years previous date in the cases of epilepsy which I have related caused me to regard the incident as a very probable one. We are not as yet in a position, as I have remarked, to explain the *modus operandi* in such cases as those which I have described. It may, however, be legitimately inferred from the occurrences that we have the power, by the application of a local irritant to the skin, of occasioning molecular

changes in the cells constituting that nervous centre in the cortex to which impressions are conveyed by centripetal nerves proceeding from the portion of skin influenced. This is about as far as we can safely go.*

I do not know that we have experience of anything in health quite corresponding to the kind of transfer which we are now considering, but there is an approach to it.

If the foot-sole be slightly tickled the intrinsic muscles of the toes alone contract; as the stimulus is continued and its application intensified, the muscles of the foot generally, then those of the leg, thigh, and in time more or less of the whole body, are set in action, evidently in consequence of the spread of some molecular change into centres adjacent to that which was first influenced by the centripetal irritation.

And so, again, if we turn to the motor side, we observe, in the act of tranquil speaking, efferent impulses affecting only the organs of articulation. If energy be developed these impulses "overflow," as it were, into centres adjoining those governing the organs of articulation, so that active movements of facial muscles and gesticulations with the right arm, or even with both arms, display themselves in addition.

Observation of the effects produced upon nerve centres by centripetal stimulation in the case of the epileptic patients suggested to me the idea of endeavouring to influence the centre for speech (in cases of aphasia) by blistering the skin of the right arm. I thought that perhaps a strong impression conveyed to the centre for the arm might "run over," so to speak, into the adjacent speech centre, and stimulate it. In a few instances I applied circular blistering to the right forearm in cases of right hemiplegia with aphasia, but with no notable results. In 1880 Vulpian, in an interesting pamphlet,† following

* Dr Augustus Waller suggests to me that "transfer" is possibly analogous on the sensory side to the phenomenon of "diffusion" on the motor side, the law of which has been formulated by Pflüger.

† 'De l'influence de la faradisation localisée sur l'anæsthésie de causes diverses,' Paris.

one which he had published in 1875, showed that faradisation of the skin with the wire brush, very strong currents being employed, will restore sensibility to a limb over an extent of surface far beyond that immediately submitted to the action of the currents. His plan was to faradise a circumscribed space of skin (about 5 or 6 centimétres square) on the posterior surface of the paralysed forearm. When the cerebral lesions in a case of hemiplegia are not very extensive, it is probable, he thinks, that they leave intact numerous elements which, in favorable circumstances, may replace in time those which have been destroyed. These elements need to be, so to speak, solicited to enter into action. "One may assume," he goes on to say, "that when the peripheral ends of paralysed nerves are exposed to excitations (especially electric) the more or less direct stimulation thereby directed upon the cerebral elements capable of a replacing action rouses them from their physiological torpor." The idea upon which I have acted, although bearing a *primâ facie* resemblance to this, really differs from it, as will be observed, though the mode of treatment, centripetal stimulation, is similar.

I have adopted Vulpian's suggestion of faradisation with the wire brush, and use this in addition to circular blistering in cases of aphasia.

At present I have nothing which I feel justified in considering important to communicate in the way of results. In the nature of things observations must be numerous and diversified as well as, in many instances, long continued, before it will be possible to say with safety whether any advantage is to be gained by these means. One method adopted has been directed, as I have said, towards endeavouring to excite, through the medium of proximity, such part of Broca's convolution as may have escaped serious damage. For this purpose I apply circular blisters to the right forearm, together with faradaism by means of the wire brush to the arm, lips, and tongue, using very strong currents. But it is con-

ceivable that such extensive destruction may have occurred in the centre especially concerned in speech as will have left no elements capable of being roused into activity. Where presumably this is the case another plan is adopted. The aim is then to rouse into activity the posterior portion of the third frontal convolution in the *right* cerebral hemisphere by directing painful impressions upon districts, such as the tongue, lips, and left arm, which are in physiological association with grey matter adjacent to that convolution. For this purpose I have used not only strong induced current by means of the wire-brush, and blisters, but also issues to the skin of the left arm. The idea is, as I have said, in the failure from disease of Broca's convolution, to develop by afferent impulses the corresponding convolution in the *right* cerebral hemisphere. With the same view I make the patient go through gymnastic exercises with the left arm, and delicate movements with the fingers of the left hand, with the aim of producing that *dexterity* (using the term in its etymological sense) with which the faculty of expression by speech and signs is so intimately associated.

LECTURE XXV

SOME POINTS IN THE DIAGNOSIS OF LEAD PALSY

About a year ago* a married lady was brought to me by her medical attendant, complaining of great loss of power in the right hand and left foot, which had been gradually increasing for three or four months; and was then so confirmed that she dragged her foot, and could do nothing with her hand. She was twenty-six years old, healthy looking, and the mother of three children, of whom the youngest was two years of age.

On examination, the right forearm, at a point four inches below the olecranon, was found to measure seven inches and five-eighths, as against eight inches and a quarter, the size of the left at the same level. The plump of the thumb, too, of the right hand appeared somewhat thin, and the interossei depressed. The hand could not be brought to a level with the forearm. Its grasp was feeble, and the patient could not perform delicate movements with the fingers. The left foot dropped when lifted off the ground. There was nothing wrong, so far as could be observed, with the left upper and right lower extremity. This *bizarre* arrangement of the paralysis had suggested to more than one medical observer that the affection was of an hysterical character. The general health was described as fairly good, except that there had been occasional vomiting. The patient was not constipated, nor had she suffered from colic, and careful examination

* This and the following case appeared in 'Brain,' April, 1878.

failed to show the slightest trace of any blue line in the gums. Inquiry had not succeeded in eliciting any history of exposure to lead.

When the excitability of the muscles came to be tested by electrical currents, important changes were observed. On the right side none of the muscles on the back of the forearm would respond to the highest strength of induced currents which could be borne, except the two supinators. The supinator longus contracted freely to a moderate strength, and the supinator brevis, which, owing to the wasting, could be picked out by the rheophore as it lay between the short radial and common extensors, answered readily enough to the stimulus, producing brisk supination of the hand. The intrinsic muscles of the hand likewise failed to respond to faradaism. To a galvanic current from eight cells of a Stöhrer's battery, slowly interrupted, all the muscles which had been unaffected by the induced currents contracted freely; whilst the supinators showed no response to this stimulus, which is not sufficiently strong to affect healthy muscles.

The muscles of the left leg presented similar inexcitability to faradaism, coupled with active response to weak galvanic currents in the tibialis anticus group.

In the left forearm, which presented no obvious lesion, it was found, nevertheless, that the excitability to faradaism was distinctly below the normal in the extensor communis digitorum.

In the muscles of the right leg, too, there was slight diminution of excitability to faradaism, but neither in this limb nor in the left forearm was there exaggeration of irritability to interrupted galvanism. There was slight loss of electro-cutaneous sensibility in the right forearm and left leg.

The symptoms exhibited by this patient appeared to point to paralysis from lead poisoning, and the result of examination with electrical currents so strongly confirmed this view as to leave in my mind no doubt upon the matter, and a confident opinion was accordingly

expressed to that effect. But the possibility of such a cause was at first strongly denied, both by the patient and her husband. It was only after persistent inquiry that the following fact was elicited. About three weeks previously, the zinc cistern supplying the patient with drinking water had been cleaned out, and three or four feet of lead-piping were found lying loose in the bottom of it, having been dropped there by a careless workman. How long it had lain there could not be ascertained, but it must certainly have been many months.

The treatment prescribed was the use of the constant current to the affected limb, with slow interruptions, so as to cause contractions of the muscles. Iodide of potassium also was given internally. A few months afterwards I heard that a considerable improvement had taken place, but the sequel has not been brought under my notice.

A few months after I had seen this case a middle-aged lady was sent to me by a medical friend on account of powerlessness of the hands. The weakness, it seemed had commenced more than five years before, and had gradually increased so much that she had great difficulty in doing anything with her hands. In the course of her long illness she had been seen by several medical men who pronounced her, so I was told, to be suffering from Cruveilhier's atrophy affecting the muscles of the hands. She had not had any electrical examination.

There was a helpless look about her hands, which "dropped" at the wrists, and could not, even by a strong effort, be kept perfectly extended. The forearms were round—somewhat plump-looking, indeed—and in contrast with them the hands appeared to be wasted more than a closer observation showed them really to be. The fingers could be moved freely enough in various directions. The patient had pains in them occasionally. There was no flattening of either forearm, but the flesh when handled felt pulpy and wanting in firmness. Whilst the hand could not be extended against even a very slight opposi-

tion on the part of the examiner, the movement of supination was performed readily and with good force.

Electrical examination showed the excitability to faradaism to be very much diminished in the extensor communis digitorum, extensor indicis, and extensores carpi radialis longior and brevior; not so much, but still appreciably lessened in the extensor carpi ulnaris. The supinator brevis could not be reached—the supinator longus reacted to faradaism. The intrinsic muscles of the hands reacted to induced currents equally well on each side, and if there was any diminution of excitability in them, it was but slight. There was an indefinite history of impairment of general health, and the occurrence of occasional spasms of the bowels. An opportunity was not afforded me of examining the gums.

An opinion was given as the result of this examination that the case was one of lead poisoning. On inquiry as to exposure, it appeared that the patient had been in the habit for many years of applying to her head one of the popular "hair restorers," the active ingredient in which is known to be lead. The quantity used at a time was said to be small (which is a relative term), but its application had been constant.

A history of exposure to lead, the presence of a blue line on the gums, preceding colic, and the peculiar character of the affection, are features commonly adduced as the diagnostic signs of lead palsy. The history of exposure, however, has often to be sought out by pertinacious inquiry after the diagnosis has been formed through other sources. So again the blue line may be wanting, and there may have been no colic. It is, then, to the peculiar character of the palsy, and especially to the effects of electrical currents on the muscles, that we have usually to look for the most valuable—the only reliable evidence, indeed, of the nature of the disease. The cases described seem to me worth recording as illustrations of the value of these signs.

In both, the examination by means of electrical currents gave results in accordance with Duchenne's well-known description of the characteristic signs of lead palsy. The excitability to faradaism was absent or sensibly diminished in all the muscles of the forearm except the supinator longus and brevis. The fact that those muscles were not included in the paralysing influence was shown in two ways:—1st, the patient having semipronated the hand, and then flexed the forearm on the arm, the observer tried to extend the forearm, when the supinator longus could be felt contracting strongly; 2nd, when the muscles were tested with induced currents, it was found that they retained their normal excitability. The opportunity which I have had of observing a considerable number of cases of lead palsy has shown me that the signs noticed are those which are commonly found. Duchenne went so far, I think, as to say that he had met with no exception to the rule that the supinators were exempted, and writers have repeated his description. But the rule is not quite without exception. I have seen some well-marked instances of the supinators taking their share in the common lesion. These have been, however, comparatively very few, and have always occurred in cases where the exposure has been long continued, and when, if I am not mistaken, some muscles of the arms, as well as the forearms, were involved in the atrophy. So that for all practical purposes Duchenne's rule appears to me one to be depended upon—at least in cases which are not of very old standing indeed, or of exceptional severity.

In health, the supinator brevis cannot be directly stimulated by electrical currents on account of its deep position. But in lead palsy it very often happens that the wasting of the extensor communis digitorum has proceeded far enough to uncover the supinator brevis so as to allow a small rheophore to be applied to it. In these circumstances the electrical diagnosis is singularly simple, and can be carried out in about the space of a square inch at the upper and back part of the forearm. If it be found

(both arms being affected) that the common extensor fails to respond to faradaism, whilst the short supinator close by, on a lower plane, is readily excited by it, the case may be almost positively set down as one of lead palsy.

The exaggerated reaction to galvanism in the first case is not at all unfrequent in cases of lead palsy of not long standing.

The crossed arrangement of the paralysis in the first patient is curious; and had the apparent exemption of one leg and one arm on opposite sides been as real as it looked, explanation of the anomaly would probably have been impossible. But the electrical examination showed that the difference was in truth one of degree only. The apparently sound limbs betrayed characteristic signs of lesion in the diminished excitability of their muscles, and it was evident that time only was required for them to share the trouble of their fellows. It is much more common than not in cases of lead palsy for one limb to suffer more than the other. In this instance, however, the contrast was more than usually strong.

It is an interesting fact that, in the apparently sound limbs, there was diminution of electric excitability in muscles which, according to the patient's own feeling, had nothing the matter with them.

The entire absence of the blue line in the gums of the first patient is noteworthy, as it tended to obscure the diagnosis. Although the absence of this sign is not by any means unexampled, it is certainly rare in a case of lead-poisoning so marked as this to find no characteristic discoloration in the gum. I attribute its absence here to the fact of the exceeding care which the patient had evidently taken in brushing her teeth, which were scrupulously white and perfect. As Mr Tomes has described, it seems likely that it is to the decomposition of portions of food left about the margins and interstices of the teeth, and in the tartar often collected there, that the discoloration is due, sulphuretted hydrogen thereby formed acting upon the lead in the neighbouring tissue.

It is worth remembering, as this case well shows, that the blue line is not a necessary feature of lead poisoning in persons of cleanly habits. Probably the converse is still less to be depended upon. The presence of a very well-marked lead line is quite consistent with health. Some time ago I examined a number of men employed in lead-mining and lead-smelting in Derbyshire. They had all more or less of a blue line in the gum, but none had any complaint to make of his health. Lead poisoning, indeed, is as rare amongst the men thus engaged as it is common among those who are employed in the manufacture of white and red lead.

To these cases I will now add another, which first came under my observation about a year ago.

The patient, who was between forty and fifty years of age, was suffering from more or less paralysis of all the extremities. There was difficulty in rising from a chair, and she walked slowly and with labour. She could take up things with her hands, but could not bring either hand to the horizontal position with the palms downwards. Her limbs looked well nourished and round; not a trace of atrophy was to be observed in them. There was no blue line on her gums. When seated she could dorsal-flex the foot, though not strongly. When the hand was turned to a position midway between pronation and supination, and the forearm flexed upon the arm, I found that she could oppose very good resistance to the former being extended, and the supinator longus muscle was then felt to be acting strongly. Circumstances were not favourable to an exhaustive electrical examination, but I ascertained by personal testing the following points.

No reaction to the strongest induced currents was perceptible in any of the extensor muscles on the back of either of the forearms. Nor did interrupted galvanic currents produce any evident contraction. I could not reach the supinator brevis owing to the fat which abounded in its neighbourhood. The intrinsic muscles of the thumbs reacted freely to faradaism.

There was no reaction to either current in the anterior tibial groups of muscles. The patellar tendon-reflex was absent on each side.

It appeared that this patient's symptoms had commenced with weakness in the wrists four years previously. There was no accurate history of the order in which other parts had become involved, but this was certainly the first symptom.

Inquiry as to the possibility of lead poisoning elicited the following facts:—For six years (extending down to a period two years before I first saw her) the patient had been in the constant habit of using a hair wash containing a large quantity of lead. It was whilst employing this lotion that the weakness in the wrists had commenced, and she still continued to apply it for two years after the first symptoms had shown themselves, not being aware of the possible association. She used very large quantities of the wash—larger quantities, she allowed, than is at all customary, to her hair every day. It is an important fact in the history that a relative who employed the same wash, and who indeed had introduced it to my patient, began to suffer also from weakness in the wrists, and found herself letting things drop from her hand. She, however, as I am informed, was prudent enough to discontinue the application, and has quite recovered power.

On a subsequent occasion I again examined the patient when she had been taking iodide of potassium in ten grain doses for some months. She appeared to be a little stronger. She could walk a little more freely, and sit down and rise from her chair, though in this last movement she still required some help. In the standing position she could lean well forward without falling, showing that the muscles of the back were strong. When lying down she could lift her knees with tolerable force and could also straighten the leg upon the thigh fairly well, and dorsal-flex the foot upon the leg. There was no deformity about the position of the scapulæ to indicate weakness of rhomboids or serrati, and no wasting was per-

ceptible anywhere. She could now raise either arm to her head, and could also raise the fingers to the level of the wrist, a movement she had been unable to execute at my first examination.

On this occasion, as on the preceding one, I could obtain no response whatever to the strongest induced currents in the anterior muscles of the thigh and leg and the anterior and posterior muscles of the arm and forearm. I examined repeatedly and with especial care the quadriceps extensor cruris and the anterior tibial group, employing currents which were of very painful strength.

In this case there was the same almost unnatural roundness of limbs, with a pulpy feeling of the flesh when it was handled, which had marked the second patient whose case is described, and I cannot help thinking that, as in that instance, there must have been a large overgrowth of adipose and connective tissue which masked the muscular atrophy.

It is noteworthy that the patient had not only a strong gouty inheritance but had herself suffered from symptoms of gout. So also had the relative who, as I have described, had been threatened with wrist-drop. I have long been disposed to think that the association between lead and gout, first pointed out by Garrod, may explain the apparent anomaly of some persons being affected injuriously by lead applications which can apparently be employed with impunity by many others. It should be added that the urine contained no albumen or sugar, and in other respects appeared to be normal.

The preservation to a considerable extent of voluntary muscular contraction, whilst the application of faradaic currents to the skin failed to elicit the slightest reaction, suggests an apparent contradiction.

I do not think, however, that the fact is difficult to understand when we call to mind the pathological state of the part affected. In this patient, as in the second case, the absence of any flattening of the forearms—their marked roundness, indeed—was likely at first sight to

cast doubt upon the probability of there being any atrophy of the extensor muscles. But although in many cases of lead palsy, especially in those of rapid and recent occurrence, there is an amount of excavation which is quite remarkable in the situation of the extensor group of muscles, yet in those of old standing one often sees that the affected limb wears a false air of nutrition. It is evident that this depends upon a large interstitial development of adipose tissue separating and surrounding the wasted and degenerated muscular fibres. No doubt this had happened in the present instance, where the disease had existed more or less for six years, as well as in the preceding case, where the symptoms had commenced five years before I saw the patient.

Hayem, in his important work on 'Muscular Atrophy,'* reproduces a drawing by Gombault, which clearly illustrates the condition. In this we see a number of fasciculi of muscular fibres only slightly wasted, along with numerous bundles of muscular fibres which are extremely atrophied. The whole is invested by a profuse overgrowth of connective and adipose tissue, which would, it appears to me, not only oppose great resistance to electrical currents in their passage to the muscular fibres, but also, it is probable, conceal to a great extent any muscular contraction which might take place in the scattered fibres which were still able to respond to the test.

The question of the essential seat of lesion in lead palsy is still unsettled. In some instances changes in spinal centres have been observed, in others careful investigation has failed to discover any but peripheral lesions. The very frequent exemption of the supinators, as pointed out by Duchenne, is still difficult to explain, in spite of the important observations of Remak† as to the representation together, in the anterior horns of the cord, of muscles which are functionally associated. In his "upper-arm

* 'Recherches sur l'anatomie pathologique des Atrophies Musculaires,' Paris, 1877.

† 'Archiv f. Psychiatrie,' 1876 and 1879.

type" the supinator longus is associated with the brachialis anticus and biceps, the "forearm type" corresponding with the muscles usually affected in lead palsy. Ferrier, in conjunction with Yeo, has recently carried out a series of important investigations as to the effects of irritation of the brachial and crural plexuses of the monkey,* from which it appears that each motor root represents a distinct functional combination, a relation of function and not of mere contiguity of peripheral nervous supply. From these observations it would appear probable that the movement of supination is subserved by roots coming from a part of the cervical enlargement higher than that which brings about extension of the wrist.

The following case, which occurred some years ago, bears upon this difficulty. My notes of it are unfortunately not so complete as could be desired.

Henry S—, æt. 55, a sign writer, applied at the hospital, on November 29th, 1876, on account of weakness in the left arm and both legs, which had been coming on gradually since the preceding April. Until that time he had been quite well.

He explained that at first he felt something like a sprain in the left wrist, and with the idea that he had injured this he bound it up. The arm gradually got weaker.

In August both his legs began to lose power, and a few weeks before applying at the hospital he observed his right arm become weak.

On admission it was found that there was wasting of the left forearm, especially in the extensors of the fingers and wrist, and also in the deltoid muscle. On the right side there was no perceptible alteration. He had had no pains anywhere. There was no rigidity in his limbs. Marked arcus senilis was noted in each cornea. The ophthalmoscope revealed no change in the fundus oculi.

He could walk about half a furlong with the help of a stick, but was then much fatigued. The bladder was not

* 'Proceedings of the Royal Society,' No. 212, p. 12. 'Brain,' July, 1881.

affected, and the urine contained neither albumen nor sugar.

Examined electrically it was found that there was great loss of excitability to faradaism in the wasted muscles. This defect of excitability was as conspicuous in the supinator longus as in the extensors.

There was a blue line along the margin of the gum of the lower jaw, not very well marked. The patient was admitted in December as in-patient into the hospital, where he became rather rapidly worse.

Shortly after his admission it was noted that there was wasting of the interossei of the left hand. On the right side the interossei reacted well, the extensor communis and the supinators badly.

He died on March 21st of an intercurrent lung complication.

This patient's employment exposed him to the influence of lead, and he had a blue line (not, however, a very characteristic one) at the margin of the gum. The paralysis and wasting of the extensor muscles, and interossei might, in these circumstances, have been confidently attributed to the action of lead. But the electrical test made this improbable. The fact of our finding the electro-contractility of the supinator longus as much affected as that of the extensor communis appeared to me sufficient to enable us to say confidently that we were not concerned with a simple instance of lead palsy.

Sections of the cord showed extensive atrophy of large ganglionic cells in the anterior cornua (more marked on one side than the other) in the cervical and dorso-lumbar regions, together with what was apparently a commencing overgrowth of connective tissue in the lateral columns.

The rapidity of this case, coupled with the fact that the exposure to lead had not been by any means of a severe kind, is sufficient, I think, to exclude it from the category of lead palsy. The contrast in the condition of the supinators with that which is observed in the preceding cases is interesting and, I think, instructive.

The value of the blue line on the gum as an element in diagnosis is unquestionable, and is not diminished by the exceptional circumstance that in two out of the three cases of lead palsy here described it was absent, whilst it was present, though not very strongly marked, in a case of progressive muscular atrophy the origin of which there is not sufficient evidence to refer to the action of lead.

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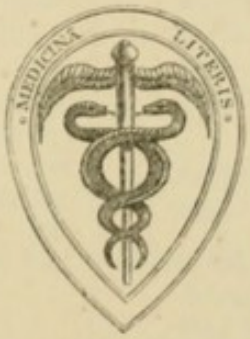
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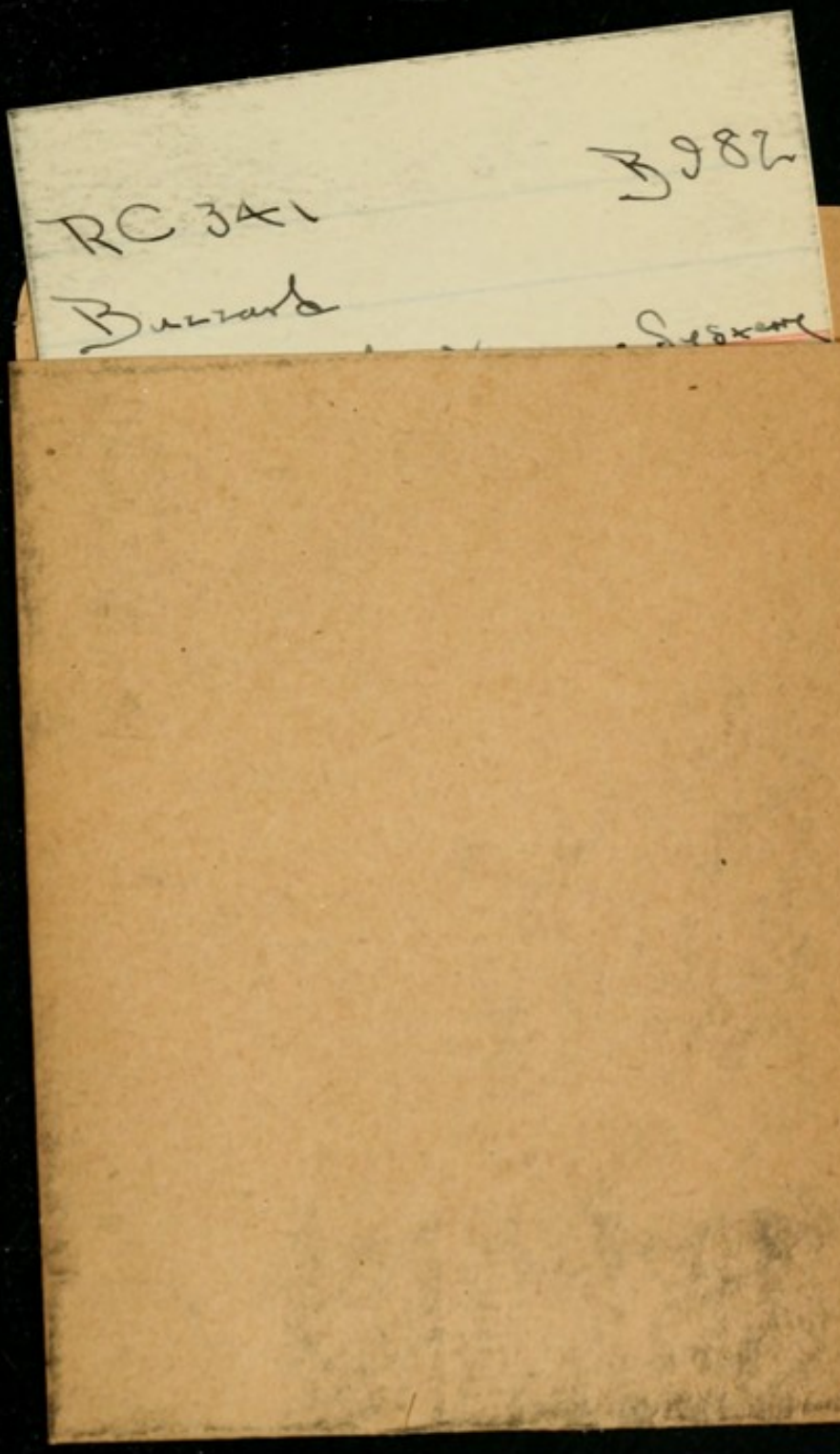
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