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THE

PATELLAR TENDON REFLEX

Read before the
Medico-Chirurgical Society of Edinburgh on June 4, 1879

BY

BYROM BRAMWELL, M.D.

LATE PHYSICIAN AND PATHOLOGIST TO THE NEWCASTLE-ON-TYNE INFIRMARY
LATE JOINT LECTURER ON CLINICAL MEDICINE AND PATHOLOGY IN THE UNIVERSITY OF DURHAM COLLEGE OF MEDICINE, NEWCASTLE-ON-TYNE

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THE PATELLAR TENDON REFLEX.

In the year 1875, Professor Erb, of Heidelberg, and Professor Westphal, of Berlin, directed the attention of the profession to the fact that the upward movement of the foot, which follows a sharp blow upon the ligamentum patellæ, when the knee is semiflexed, and the leg at rest, is in certain pathological conditions absent, and in others exaggerated. These observations, together with some illustrative cases, were brought before the notice of this Society by Professor Grainger Stewart at the January meeting of last year, at which I had the good fortune to be present; and more recently Dr. Buzzard has published the results of his experience in the pages of the *Lancet*.*

Dr. Gowers, too, has, since this paper was written, made an able communication on this subject to the Royal Medical and Chirurgical Society of London.†

The phenomenon is one which, as Professor Grainger Stewart tells us, has been long known to schoolboys. It is readily produced by striking the ligamentum patellæ of a healthy individual, who is seated on a table with the feet dangling, a sudden jerk forwards of the foot being the result.

That the movement is not mechanical, but that it is due to the contraction of the quadriceps extensor femoris, is proved by the following facts:—

1. The movement does not accompany the blow, but follows it by an appreciable interval.

2. The fibres of the muscle can be felt to contract just as the movement is about to be produced.

3. When the muscle is paralysed the phenomenon is absent as in the following case.

* *Lancet*, July 27, 1878. † *Lancet*, February 1, 1879, page 158

Case 1.—Case of Traumatic Injury of the Spinal Cord—Paralysis of the Left Quadriceps Extensor Femoris—Absence of the Patellar Tendon Reflex on the Left Side.

A strong, healthy labourer, aged twenty-three, was working at the top of a house in Tynemouth, on August 8, 1877, when the scaffolding on which he was standing suddenly gave way, and he fell to the ground, a distance of at least fifty feet. His fall was fortunately broken by his striking first against some joists, and then against a staging which was raised some ten feet from the ground.

He was insensible for a quarter of an hour. When he came to himself he experienced pain in the small of the back, and found that the lower extremities were completly paralysed. During the first twenty-four hours there was retention of urine; and for several days the bowels were obstinately constipated. The back showed signs of bruising in the lumbar region, and he remembered striking it against the joists as he fell.

On September 6 he was admitted to the Newcastle-on-Tyne Infirmary under my care. His condition was then as follows:—There was slight tenderness over the lumbar region. The right side of the back at the level of the first lumbar vertebra seemed more prominent than the left. There was complete loss of motor power in the lower extremities; the muscles were flaccid and much wasted, the atrophy being chiefly marked in the muscles on the anterior surface of the left thigh and on the anterior surface of the right leg. Fibrillary twitchings were frequently seen in the atrophied muscles. Sensibility was fairly normal in the right leg, considerably impaired in the left. No reflex action could be elicited on tickling the soles of the feet. The bladder and rectum were normal.

Under appropriate treatment the patient made a slow but gradual recovery. On October 30 he was able to sit up for the first time. In December he could manage to drag himself about the ward. In January he could walk fairly well with the help of two sticks. He was discharged on January 31. The left quadriceps extensor femoris was still completely paralysed. The patellar tendon reflex was present to a

normal degree in the right leg, but was completely absent in the left.

In this case, then, in which there was a traumatic injury to the cord, followed by paralysis of the left quadriceps extensor femoris, the patellar tendon reflex was absent on the same side.

That the contraction of the muscle is *reflex* is proved by the following facts:—

1. Destruction of the lower portion of the spinal cord (*i.e.*, the centre through which the reflex travels) prevents the movement. This fact has been experimentally proved in the lower animals,* and is confirmed by cases in the human subject.

2. In many cases of locomotor ataxy the development and excitability (both to electrical and mechanical stimulants) of the quadriceps extensor femoris is quite normal, and yet the patellar tendon reflex cannot be produced.

3. Lesions of the spinal cord above the level of the lumbar region (*i.e.*, the centre for the patellar tendon reflex) which interrupt the reflex inhibitory fibres passing downwards from the brain—such lesions, for instance, as compression of the cord, transverse myelitis, sclerosis of the lateral columns,—are associated with a marked exaggeration of the phenomenon.

4. The free use of strychnine, which increases the reflex excitability of the spinal cord, causes also an increase in the extent of the movement.

5. In some cases in which the reflex functions of the spinal cord are increased, as in two I shall presently relate (Cases 9 and 10), a blow on the ligamentum patellæ of one side is followed by contraction of the muscle of the opposite leg (radiation of the reflex). The same fact has also been observed by Dr. Gowers.

6. The length of the interval (average in health and

* F. Schultze and P. Fuerbringer, *Centralblatt d. Med. Wiss.*, 1875, No. liv., as quoted by Erb in "Ziemssen's Cyclopædia," vol. xiii., page 50. Tschirjew, *Berlin. Klin. Wochenschrift*, April 29, 1878, as quoted by Buzzard in *Lancet, op. cit.*

disease ·10 or ·11 seconds) which occurs between the tap and the muscular contraction has been proved by Dr. Gowers* to correspond with the time necessary for a reflex action· (Conduction ·045 second, latent stimulation ·01 second, reflex process in cord ·05 second.)

The movement of the foot is therefore due to a *reflex* contraction of the quadriceps extensor cruris; and the phenomenon has been called the "*patellar tendon reflex.*"

Now, in every reflex act a nervous arc—consisting of (1) a sensory or centripetal nerve, which receives the impression and transmits it from the periphery; (2) a centre which receives it; and (3) a motor, or centrifugal nerve, which transmits it from the centre to the muscle—is, of course, necessary.

The Centripetal Nerve.

A good deal of discussion has taken place as to the exact point of origin of the afferent impulse in the particular reflex act which we are considering. Nearly all observers are at one in thinking that the sensory filaments which receive the impression are not situated in the skin, and for the following reasons:—

1. The reflex cannot be produced by any slight irritation of the cuticle, such as tickling.

2. Freezing of the skin with ether spray does not prevent the occurrence of the phenomenon.

3. If a portion of skin lying over the tendon be pinched up and forcibly struck, the movement is not produced.

4. In some cases of locomotor ataxy the tendon reflex is destroyed, but the skin reflex remains.

I have carefully repeated these experiments, and have satisfied myself as to their accuracy. I agree, therefore, that, in the normal condition of things, the sensory filaments which receive the impression are not situated in the skin.

I am not quite satisfied, however, that in some pathological conditions the sensory filaments of the skin can be so absolutely excluded.

* *Lancet*, February 1, 1879, page 156.

In one case, which I shall presently relate (see Case 9), a distinct contraction followed a blow upon a pinched-up portion of skin over the ligamentum patellæ, notwithstanding the fact that every care was taken to prevent any dragging of the tendon.

In another case (which I shall also give in detail) the movement was markedly lessened after freezing. Possibly this was owing to the frozen skin acting as a protector to the tendon. Against this view, however, is the fact that a tonic contraction of the muscle took place when the spray first touched the skin over the patella.

The fact that the movement is in all cases *most readily* and in many cases *only*, produced by a blow upon the centre of the patellar tendon below the knee, suggests the idea that the sensory filaments are either situated in the tendon itself or in the fibres of the muscle (quadriceps). According to the latter view, the blow upon the tendon acts by stretching the muscular fibres; and the fact that the movement is most readily produced by a blow upon the tendon is explained by the facility with which the fibres of the muscle can be stretched *all at once* by pulling the tendon. This view necessitates the tension of the muscular fibres through the tendon; but, as Erb points out, the phenomenon can in many cases be produced where no such tension is exercised.

This was remarkably well seen in a case which I lately had an opportunity of examining with my friend and late colleague Dr. Drummond.

Case 2.—Case of Rigid Paralysis in a Child—Tendon Reflex greatly exaggerated, and produced by Blow on Tibia—Ankle Clonus not produced by Simple Pressure on Sole.

The patient, a girl aged four, was admitted to the Children's Hospital, Newcastle-on-Tyne, under Dr. Drummond's care, suffering from rigid paralysis. A slight blow upon the free surface of the tibia, when the heel was supported so as to prevent any dragging on the fibres of the quadriceps, caused marked contraction of the muscle. In this case, too, I may remark, in passing, we had an opportunity of confirming

Dr. Gowers' observation that the foot phenomenon is not produced by simple pressure on the sole (balls of the toes) in children who have never walked. If, however, in this, as in Dr. Gowers' case, a slight tap was given on the front of the tibia while such pressure was being exercised, marked ankle clonus resulted.

Dr. Gowers, in the paper to which I have already referred, leaves the exact position of the afferent fibres doubtful, concluding, however, that the impulse must arise either in the tendon or the muscle. He has very kindly communicated to me the facts which seem to him to indicate the possibility of the origin of the reflex from tension of the fibres of the quadriceps. They are as follows:—

1. When the reflex is excessive, as in "lateral sclerosis," it can be obtained almost as readily by a blow on the top (upper edge) of the patella as by a blow on the ligament below the patella.

2. In such cases it can also be produced by a blow on the tendon above the patella.

3. Also by a blow on the substance of the rectus in the middle of the thigh. In this case the movement is not merely the result of the direct contraction of the fibres struck, but a contraction of the whole muscle similar in degree and in time (rough observation) to that which occurs when the patella tendon is struck.

"These facts," which I can from my own observation confirm, "prove," says Dr. Gowers, "that the afferent impulse may originate elsewhere than in the patella tendon. They also show that the afferent impulse may originate in the muscle. The phenomena of the rare knee clonus indicates the same fact, since in this sudden tension must originate each afferent impulse." He adds—"I do not think, however, that in man, when the patella tendon is struck, the afferent impulse is from the muscle, the sensation at the point struck is so peculiar and sometimes very painful. But I think it probable that the blow upon the tendon above the patella may act on the muscular fibres, and excite the afferent impression in this manner."

The conclusions which I have come to as regards the origin of the afferent impulse are:—

1. That in the normal condition of things it originates in the tendon.

2. That it can also originate in the muscle.

3. That in some cases, where the phenomenon is greatly exaggerated, it follows a blow upon the free surface of the tibia. In such cases it is supposed to arise from the periosteum.

4. That it is doubtful whether it may not in some pathological cases originate from the skin.

The Centre.

The centre which receives the impression is situated in the lumbar region of the spinal cord.

The Centrifugal Nerve.

The centrifugal or efferent nerve is the anterior crural (motor nerve to the quadriceps extensor femoris).

So much then for the character of the phenomenon and its mode of production. I now pass to the alterations which are found in disease.

Before, however, entering upon this part of the subject it is necessary to mention that this reflex, like the ordinary reflex from the skin of the sole, is occasionally absent even in healthy persons. Dr. Gowers examined 300 healthy individuals, and found it absent several times. Other observers have met with the same fact. I have not myself made any systematic observations on the point. In several of my hospital cases no contraction followed a blow with a light instrument, such as the end of a stethoscope; but in all of these a contraction (not a mere mechanical movement) followed a blow with a heavier object, such as a percussion hammer. In all cases, therefore, in which there is any doubt I use the heavier instrument. It is absolutely necessary, too, that the individual who is being experimented upon should be in ignorance of the exact moment when the blow is about to be struck; for this, like other reflex acts, can in many cases be to a great extent controlled by the inhibitory

power of the will. Where there is any doubt as to the presence of the phenomenon, I am in the habit of bandaging the patient's eyes and placing him with both legs dangling in a pendulum-like manner over a high table. By this means it is possible to make quite certain that there is no muscular resistance, and to strike either tendon without the patient being aware that you are going to do so.

Alterations of the Phenomenon in Disease.

The patellar tendon reflex may be either diminished, absent, or exaggerated in disease. Practically, it is best to consider only those cases in which it is absent or decidedly exaggerated; for, owing to the great differences in degree which we find in apparently healthy individuals, it is impossible to fix upon a common normal standard.

Cases in which the Patellar Tendon Reflex is Absent.

Anything which impairs the integrity of the nervous arc will, of course, prevent the due conveyance of the stimulus, and hence the contraction of the muscle.

As yet, so far as I am aware, no case has been recorded in which it has been clearly shown that the arrest has taken place in the sensory or motor nerve trunks which convey the stimulus from the muscle to the cord, and from the cord to the muscle, respectively (sensory fibres of the anterior crural and obturator nerves, motor fibres of the anterior crural). Lesions of the second, third, and fourth lumbar nerve roots, both anterior and posterior, will, of course, arrest the phenomenon. Such lesions not unfrequently result from meningitis.

Disease, however, of the cord itself, lumbar portion, is the chief cause of arrest of the reflex. All diseases of the lumbar cord will not, of course, interfere with the phenomenon. It is only when that particular portion of it through which the impulse has to pass is diseased that an arrest will occur.

Let us stop, then, for a moment to trace the course of the reflex through the cord. "The centripetal paths, which

convey a stimulus inward," says Erb, "lie beyond a doubt in the posterior roots; those which convey forth a stimulus, the centrifugal, or motor, lie in the anterior roots; but of that which lies between these two routes and its histological structure we are not well informed. We may, however, guess that there are branch conductors given off both from the sensory and from the motor paths at various points within the spinal cord, which meet each other at certain ganglia and groups of ganglia (reflex centres), and enter into conductive communication with each other."

Lesions, therefore, of the posterior root fibres of the second, third, and fourth lumbar nerves; of those portions of the grey matter through which the reflex fibres pass; or of the anterior root fibres of the second, third, and fourth lumbar nerves, by interfering with the reflex filaments, will prevent the occurrence of the phenomenon.

I now pass to consider some of the special diseases in which the patellar tendon reflex is absent. The first, and by far the most important, is locomotor ataxy.

Locomotor Ataxy.

Westphal, Grainger Stewart, and Buzzard state that the patellar tendon reflex was absent in all the cases which they have examined. Such, too, was the fact in three cases which I have had an opportunity of examining during the past year.

The leading particulars of these cases are as follows:—

Case 3.—Locomotor Ataxy—Tendon Reflex lost—Skin Reflex retained.

R. C., aged forty-seven, married, a striker, was admitted to the Newcastle-on-Tyne Infirmary, under my care, on April 23, 1878, complaining of difficulty in walking.

Previous History.—He has been ill three years, and has been off work two years. Until the present attack commenced he enjoyed good health. He remembers, however,

that nine years ago his left eye was "bad," it used to lead him wrong, and he thinks he had a squint. He has occasionally had sharp, shooting pains in various parts of the body. The present attack commenced with a feeling of numbness in the fingers of the left hand. He used, too, at the same time, to stamp the left foot while at work. His fellow-workmen used to make remarks about his stamping. He could not help it. Soon after this he first noticed that he had some difficulty in walking. It was worse in going down a bank, and much worse in the dark. He thinks he has lost some flesh, but has always been a thin man. He has never suffered from headache. He has been steady all his life; has not had syphilis; his occupation was a very laborious one.

Family History.—Very good.

Present Condition.—The patient is very thin, and looks ill. The gait is most typically that of locomotor ataxy. Co-ordination in all four extremities is much impaired. The balancing-power, when the eyes are shut, is very bad indeed. Sensibility of all sorts (touch, pain, temperature, power of localising impressions, power of appreciating weights) is very much impaired in the lower extremities, and to some extent in the upper. He complains of a tight feeling round the abdomen. The muscles generally are wasted and flaccid. Fibrillary twitchings are occasionally seen in all four extremities. The muscular power in the left side of the face and left arm is decidedly less than in the right, and there has evidently been some former cerebral lesion. *The patellar tendon reflex is quite absent, but the ordinary reflex on tickling the soles of the feet is well marked.* Sight is good with spectacles; the pupils are equal and contracted, the discs slightly indistinct at their edges. He is very costive, and makes his water with great difficulty; it has occasionally dribbled away from him. The other systems and organs are normal.

Treatment.—He was treated with full doses of iodide of potassium, cod-liver oil, and good food, and on June 19 was, at his own request, made an out-patient, his general health being decidedly improved.

Case 4.—Locomotor Ataxy—Typical Case—Tendon Reflex absent.

C. W., aged forty-five, married, a rivetter, was admitted to the Newcastle-on-Tyne Infirmary, under my care, on October 3, 1878, suffering from locomotor ataxy.

Previous History.—The disease has been characteristically marked for five years. He was under my care four years ago. The first symptoms were "rheumatic" pains and double vision; this was so bad that he had to close the left eye when at work. The rheumatic pains began to trouble him about six years ago. Unsteadiness of gait commenced about a year after the pains, and rapidly got worse. He has been under my care, every now and again, during the past four years. Twenty years ago he had syphilis. He has been a hard drinker, and much given to sexual excess.

Family History.—None of his relatives have suffered from nerve disorders.

Present Condition.—The gait is the most typical, the patient being unable to advance a step when his eyes are raised from the ground; the legs are thrown out wildly, and the heels come down with the characteristic stamp. The muscular force of the lower extremities is very great, the muscles being powerfully developed. There is most marked loss of co-ordination and of the muscular sense. Sensibility of all sorts (tickling, power of localising impressions, pain, temperature) is very much impaired. When the eyes are bandaged he cannot tell the position of his legs, and is unable to place his feet in any given position. He is quite unable to stand with his eyes shut, even when the feet are placed wide apart. The power of distinguishing weights is very defective. *The patellar tendon reflex and skin reflex are quite absent.* There is some loss of tone in the bladder. The bowels are obstinately constipated. Sexual power is much weakened. He frequently suffers from sharp, shooting pains in the lower extremities, and complains of numbness in the soles of the feet. He feels as if he were standing on something soft, but has no tight band round the abdomen. The contractility of the muscles of the lower extremities is

normal to the interrupted, somewhat impaired to the constant current. The pupils are decidedly contracted; sight is good. The other special senses are normal. He is, on the whole, decidedly better than he was four years ago. At that time he could not get up from the sitting position, and could not walk a step without assistance. Now he goes about with the help of two sticks. The upper extremities are normal. There are no complications. The treatment which has seemed to be advantageous in this case is the administration of ergot of rye and iodide of potassium. He himself thinks the battery, which at one time was assiduously employed, was useful; of this I am very doubtful.

Case 5.—Locomotor Ataxy—No Lightning-like Pains during the first five years of the Disease—Absence of the Tendon Reflex.

X. Y., aged forty-eight, was seen by me, in consultation with my father, on March 1, 1879.

Previous History.—The patient had been a dissipated man. His illness commenced five or six years ago with unsteadiness of gait. He never had pains until this last winter. He has never suffered from rheumatism. He has not had syphilis.

Family History.—Another member of the family has suffered for twenty years past from melancholia.

Present Condition.—The gait is very characteristic. The muscular development of his lower extremities is good, the muscular force great. He complains of numbness in the feet and legs, and he cannot feel the ground properly. Tactile sensibility is somewhat impaired. Sensibility to pain, temperature, and the power of localising impressions seem almost normal. The patellar tendon reflex and the reflex actions on tickling the soles of the feet cannot be elicited. Sight is very bad, especially in the left eye; the pupils are contracted. Two years ago he could not see at all, and was said to have optic atrophy. He can now see well enough to read a newspaper with the right eye, and large print with the left. I had not an opportunity of examining the fundus. The patient's general health was good. There were no complications.

Westphal not only states that the patellar tendon reflex is absent in cases of locomotor ataxy, but he has shown that in some cases it is absent at an early stage before the appearance of the inco-ordination. Dr. Buzzard, too, has reported a case in which the early absence of the phenomenon was observed.* As the result of his observation, Westphal came to the conclusion that the absence of the patellar tendon reflex is a most important diagnostic of locomotor ataxy. The value of the phenomenon in this respect, although no doubt great, is probably much less than Westphal at first supposed, for—1. The phenomenon is sometimes absent even in health. 2. It is absent in many other diseases than locomotor ataxy. 3. Undoubted cases of locomotor ataxia do occur, in which it is still preserved. Cases of this sort have been observed in this country by Dr. Gowers, Dr. Clifford Allbutt, and Dr. Sawyer.

My friend Dr. Beatty Smith, of Stockton, has also published a case which presented some of the features of locomotor ataxy, but in which the patellar tendon reflex was exaggerated. Through the kindness of Dr. Smith this patient was admitted to the Newcastle-on-Tyne Infirmary under my care, and remained for some months under observation. The case is one of considerable interest. The gait at first sight closely resembled the gait of locomotor ataxy, and there was a well-marked history of the characteristic pains. In many respects, however, the case did not resemble locomotor ataxy, and after careful consideration I find myself obliged to differ from my friend's view. It is only fair, however, to Dr. Smith to say that after again examining the patient, he adheres to his published opinion. The notes of the case, which are published with Dr. Smith's kind permission, are as follows :—

Case 6.—*Doubtful Case of Locomotor Ataxy—Persistence of the Patellar Tendon Reflex.*

J. D., aged forty-seven, single, an engine-fitter, was admitted to the Newcastle-on-Tyne Infirmary, under my

* *Brain*, vol. i., page 168.

care, on September 30, 1878, complaining of difficulty in walking and of weakness in the lower extremities.

Previous History.—The weakness in the legs commenced at the beginning of last year (January, 1877), but was nothing to speak of until the cold weather set in. The weakness was always worse first thing in the morning. At the beginning of the present year (January, 1878) he became worse, and suffered for the first time from pains in the knees, right elbow, and right shoulder. The pains used to come on quite suddenly, and to last for from two to five minutes at a time. He describes these pains as being like the pain which would be caused by a knife being put into and turned round in the joint. Sometimes the pains were simply sharp shoots, lasting for a moment—"like the pain caused by the electricity which I have been getting to-day," said the patient. The pain was sometimes so severe that it made him cry out. The joints were not swollen, and there was no tenderness on pressure. The pains were generally worse at night. Walking about seemed to relieve rather than to make them worse. He has had frequent attacks of pain since January. The knees have been chiefly affected of late. In May he experienced some difficulty in retaining his water. Calls to urinate had to be obeyed immediately. This symptom gradually got worse, until he was admitted to the Stockton Hospital in July. The bowels, which are naturally costive, have become more so. There is no difference in sexual power or sexual desire. He knows no cause for his illness, unless it be standing all day in one position and in a draught. He has been a steady man both as regards "wine and women." He says he has not had syphilis. Since July he has been under treatment in the Stockton Hospital, and has improved considerably.

The Family History is good. So far as he knows there is no tendency to nerve complaints.

Present Condition.—He is a pale man and has a somewhat worn-out appearance. He walks very badly, and required to be assisted to the ward. At first sight the gait looks as if it were the gait of locomotor ataxy. He takes short steps, throws out his feet a little, and brings down the heels first. On observing him more closely this gait is seen to be due to

the fact that in walking the knees are kept stiff and that the legs are feeble. The right leg is evidently much weaker than the left, and is dragged. In walking, the eyes are never fixed on the ground in front of him nor on the feet. He can walk backwards almost as well as forwards. He can also walk across the room with his eyes shut. He can stand steadily with his eyes shut, the feet being quite close together. There is absolutely no loss of co-ordination in the lower extremities. When lying on his back in bed, with the eyes bandaged, he can place his feet slowly and with exact precision in any given position. The power of appreciating weights is natural. Sensibility of all sorts is perfectly normal. The muscular development is fairly good; the muscular force moderate. There is no tension of the muscles nor resistance to passive flexion of the knee.

The *patellar tendon reflex* and the skin reflexes are exaggerated.

The electrical contractility of the muscles of the lower extremities is decidedly below par.

There is no appreciable loss of power in the upper extremities nor in the muscles of the face. The tip of the tongue is turned to the right side, and the organ is tremulous. The uvula is deflected to the left. Speech is drawling, hesitating, and a little thick, resembling somewhat the speech of a general paralytic. The pupils are equal and considerably dilated. Sight is good; the optic discs are healthy. The other special senses natural. Memory is decidedly impaired. The nervous system is otherwise quite normal. Urination is now natural.

Treatment.—On September 30 he was ordered five grains of iodide of potassium thrice daily. This was gradually increased but without any apparent benefit. On October 19 half a drachm of the liquid extract of ergot was substituted.

On November 5 it was noted "he is very much improved, and can walk infinitely better than on admission. He has had no pains for three weeks, and has gained three pounds and a half in weight." On November 9 he complained of severe pain at the bottom of the spine; the face was flushed, the temperature 101° Fahr. Two days previously he had com-

plained of pain in the right elbow, and had noticed that the slightest touch over the outer aspect of the forearm caused great pain. The skin of the part was quite natural. This hyperæsthesia has now disappeared. On January 5 he was very much better, and could walk for an hour at a time without feeling tired. On February 15 he discharged himself, saying he felt quite well and able to go home. He was accordingly discharged. He had still to some extent the peculiar gait which was present on admission. The weakness in the legs was comparatively trifling. Since November 9 he had been entirely free from pain.

The exact value, therefore, of patellar tendon reflex as a diagnostic of locomotor ataxy is still *sub judice.*

Let us now endeavour to ascertain the particular part of the transverse section of the cord in which the arrest takes place in locomotor ataxia.

Pathological Anatomy of Locomotor Ataxy.

The characteristic lesion of locomotor ataxy is said to consist, when fully developed, of a sclerosis of the posterior columns with degeneration of the posterior root-fibres and of the posterior horns of grey matter.

The arrest must, therefore, take place in one or other of these parts. But, since the patellar tendon reflex is sometimes absent at a very early stage of the affection, it will aid our inquiry to determine in what part of the transverse section of the cord the lesion commences.

Observations in this part are necessarily very few. We all know how seldom cases of locomotor ataxy are followed by a dissection, and it is infinitely rare to have the opportunity of examining cases in the early stage of the disease. The observations which we do possess are somewhat contradictory.

Charcot and Pierret say, "The sclerosis begins in the external bands of the posterior columns, where the inner root-bundles mix with the vertically ascending (probably commissural) fibres of the posterior columns. This sclerosis of the lateral bands or ribbons is, according to these autho-

rities, the only essential anatomical change in locomotor ataxy."*

Lockhart Clarke found the posterior horns so constantly involved, that he raises the question whether they are not the *first* to be diseased, or at all events very early affected in all cases.

A recent observer, Dr. Takács,† says that the sclerosis of the posterior columns is a secondary process, the primary affection, according to him, being an atrophy of the posterior nerve-roots and posterior cornua, or a meningitis posterior.

Part of the Cord in which the Arrest Takes Place.

In the present position of the subject we can hardly, I think, come to a decision as to the exact point at which the arrest occurs.

If we suppose, with Dr. Gowers, that the stoppage takes place in the posterior root-fibres, it is necessary to grant that those fibres of the posterior roots which are concerned in this particular reflex have some special situation which favours their early affection in locomotor ataxy; for we meet with cases of the disease in which the ordinary skin reflexes are preserved, or even exaggerated, but in which the patellar tendon reflex is destroyed. Cases are also seen in which the patellar tendon reflex is absent, with little or no disturbance of sensibility. In such cases, if the arrest takes place in the posterior root-fibres, the lesion of the root-fibres must necessarily be a very limited one—in fact, confined to those fibres concerned in this particular reflex, the fibres for the skin reflexes and for the conveyance of sensations being intact.

If Charcot's observations as to the commencement of the lesion be correct, we can easily understand that those root-fibres which are in closest connexion with the posterior columns will be first diseased. If, therefore, the arrest takes place in the posterior root-fibres, and if Charcot's observa-

* As quoted by Erb, in "Ziemssen's Cyclopædia," vol. xiii., page 38.

† *Centr. Med. Wiss.*, December 14, 1878.

tions as to the position of the lesion be correct, we may theoretically conclude that the innermost fibres of the posterior roots—the fibres which pass through the outermost part of the posterior column (median portion of posterior nerve-roots) in making their way to the posterior cornu—are the fibres concerned in this reflex act.

Leaving it, therefore, undecided whether the arrest takes place in the posterior root-fibres or in the posterior cornua, it only remains to explain those exceptional cases of locomotor ataxy in which the patellar tendon reflex is still present. This we can readily do by supposing that in such cases the particular portion of the lumbar cord through which the reflex travels is as yet unaffected by the lesion, or possibly that, the lumbar cord being affected, the lesion has as yet spared the particular fibres concerned in this reflex act. But we know from actual observation that it is quite exceptional to find the lumbar cord free from disease; hence it is also quite exceptional to find the patellar tendon reflex still present.

Other Diseases in which the Patellar Tendon Reflex is Absent.

Destruction of the lumbar portion of the cord, the centre through which the reflex travels, will of course arrest the phenomenon. This destruction may be the result of traumatic injury, or it may be the result of myelitis, cancerous deposits, etc.

In the following case of cerebro-spinal sclerosis the phenomenon was absent, the ordinary skin reflex being well-marked.

Case 7.—Case of supposed Cerebro-Spinal Sclerosis (chiefly Intra-cranial)—Absence of the Patellar Tendon Reflex.

J. M., aged twenty-seven, single, a soldier, was admitted to the Newcastle-on-Tyne Infirmary, under my care, on January 24, 1878, complaining of shaking of the body, dimness of vision, and deafness.

Previous History.—He enjoyed excellent health until three years ago. He then began to complain of deafness. He was

admitted to the hospital at Malta, where he was stationed, and remained there for six months. He was then sent home to Netley, and was finally discharged as incurable. He has been giddy since his illness commenced. His eyesight has gradually failed. For the past four months he has suffered from "shakings." The tremblings commenced in the legs, and only occurred on movement. He has been a hard drinker and has had syphilis.

The family history is unimportant.

Present Condition.—The facial expression is dull and stupid; his memory seems much impaired. The movements of the eyes are slow and peculiar. He seems to have some difficulty in getting them into the proper axis for vision. The left pupil is small, the right dilated. Sight is considerably impaired, especially in the right eye. Both optic discs are markedly pale, but the vessels seem of normal size. Hearing both to external and skull sounds is very imperfect in the left ear; *nil* in the right. The external ears are normal. Taste and smell seem natural. Speech is thick, and characteristically jerking. He suffers every now and again from headache; the pain is of a dull character. He has never had a fit.

Sensibility of all sorts seems normal.

The *gait* is uncertain; he is unable to regulate his movements, and often sways from side to side like a drunken man. The walk is not at all like that of locomotor ataxy. He can stand as steadily with his eyes shut as when they are open. After walking, the head and neck oscillate in a characteristic quick rhythmical manner. There is at times some nystagmus. No oscillations are seen in the extremities, but he says they used to occur in the legs.

The patellar tendon reflex is quite absent. The ordinary reflex movements from the skin are, on the contrary, well marked.

The diagnosis was disseminated sclerosis (Charcot's disease), as yet mostly intra-cranial and probably chiefly affecting the cerebellum.*

* Possibly the case was one of cerebellar motor tumour, with slight pressure on the strands of medulla.

He remained in the hospital for two months, and was then made an out-patient, being *in statu quo*.

On October 31 he was re-admitted. His walking was much worse, and he complained of numbness in the left thigh and leg. The oscillations of the head and neck were greater.

Remarks.—This may have been, and probably was, a case in which the patellar tendon reflex was naturally absent. If its absence was the result of this disease, it was presumably due to the presence of a patch of the lesion in the reflex tract. Such an occurrence is of course quite accidental. In another and much better marked case of disseminated sclerosis, which I have had under observation for the past five years, the patellar tendon reflex is not only present but exaggerated.

Lesions of the anterior cornua, or of the anterior roots of the second, third, and fourth lumbar nerves, will also arrest the reflex. The following are the brief notes of a case of old paraplegia, resulting from acute myelitis, in which the lesion seems chiefly to have affected the motor portions of the cord. In it the patellar tendon reflex and the ordinary skin reflex were both absent. An interesting point in it was the fact that sensibility to pain was markedly increased.

Case 8.—*Case of Acute Myelitis—Total Loss of Motor Power and of Sensibility—Paralysis of the Bladder—Sensibility beginning to be Restored at the end of First Year, completely Regained at the end of Three Years—Motor Power very slowly Restored—Locomotion possible at the end of Seven Years from the date of the Attack.*

G. K., aged fifty-nine, a publican, married, came under my care on February 16, 1879, suffering from the effects of old paraplegia.

Ten years previously he had suddenly lost the sensibility and the motor power in the lower extremities. The attack, which came on after prolonged exposure to cold and wet while shooting, was not accompanied by pain. The bladder and rectum were both affected. The loss of motion and of sensibility was complete for a year. Sensibility then began to be regained. In two years it was

completely restored. He was then able to move his legs a little. Since that date (seven years ago, three years after the commencement of the attack) he has been gradually regaining motor power, and for the past three years has been able to get about by the help of two sticks. He has been a steady man through life, and has not had syphilis.

Present Condition.—The patient's general health is good. The muscles of the lower extremities are extremely emaciated. Tactile sensibility, the power of localising impressions, and sensibility to temperature are quite natural. Sensibility to pain is markedly in excess.

The patellar tendon reflex and the skin reflex from the soles cannot be elicited. Urination is natural; the bowels are obstinately constipated.

Erb states, as we should theoretically expect, that this patellar tendon reflex is absent in the paralysis of children (acute anterior polio-myelitis). It is lost in the later stages of pseudo-hypertrophic paralysis (Gowers).

Cases in which the Patellar Tendon Reflex is Exaggerated.

In this as in the ordinary reflex contractions which result from tickling the soles of the feet, the reflex movements are exaggerated in those cases in which the particular portion of the spinal cord through which the reflex fibres travel is healthy, but in which there are lesions that sever the connexion between the brain and the cord, and thereby arrest the controlling impulses which are supposed to pass downwards from the brain to the cord.

In cases of destruction (by injury or disease) of the spinal cord above the lumbar region (the centre through which the patellar tendon reflex passes), the phenomenon is exaggerated. Compression of the cord by an abscess will have the same effect. The following case is an example of this condition:—

Case 9.—Rigid Paralysis resulting from Disease of the Dorsal Vertebræ—Enormous Increase of the Patellar Tendon Reflex—Radiation of the Reflex to the Opposite Limb—Contraction of the Quadriceps produced by a Blow on a pinched-up portion of Skin over the Patella—Evacuation of an Abscess—Disappearance of the Paralysis—Diminution of the Patellar Tendon Reflex.

J. P., aged thirty-three, married, a fitter, was admitted to the Newcastle-on-Tyne Infirmary, under my care, on April 18, 1878, suffering from paraplegia.

Previous History.—He has never been a very robust man, but enjoyed fairly good health until two years ago, when he began to suffer from pain in the small of the back. The pain was severe and was increased by movement. The pain has continued more or less constantly until a few weeks ago. He still feels it occasionally. There has never been any swelling or tenderness over the spine. He knows no cause for the complaint. Has been a steady man and has not had syphilis.

Five months ago he began to suffer from weakness and numbness in the legs. The weakness rapidly increased, and for the past three months he has been quite unable to walk. The left leg was at first more affected than the right. Ever since the commencement of his illness he has noticed jumpings in the muscles of the legs. For the past three weeks the legs have been quite rigid and stiff.

Family History.—So far as he knows, his friends are all healthy.

Present Condition.—He is a pale, dark-complexioned man, of spare habit of body. The lower extremities are extended, stiff and rigid; he is unable to move them from this position. On movement (both active and passive) the rigidity is increased, and the tension of the muscles can be felt to become greater.

The muscles are badly developed, but have always been so. There is no great difference, he thinks, in this respect since the attack commenced.

Urination is quite natural, and has been so since the

commencement of the attack. The *bowels* are obstinately constipated.

Sensibility of all sorts in the lower extremities is impaired, though not absolutely lost. He complains of numbness in the legs and lower part of the trunk, and feels as if a cord were tied round his waist.

On tickling the soles of the feet the lower extremities become more stiff and rigid, but there are none of the usual reflex jerks.

When the patient is placed over the bed, and allowed to sit with his feet dangling, the rigidity is at first very marked. After a time, however, it passes off, and the knees become semi-flexed. If the patellar tendon be now struck, the whole extremity is thrown into a convulsion, with inversion of the foot, exactly resembling the tonic and clonic contractions of an epileptic fit. The spasm frequently extends to the other leg, but is never so violent in that leg as in the one struck. The spasm lasts for a considerable time; on several occasions it was timed to last for two or three minutes.

The convulsive spasm can also be produced by striking the quadriceps extensor femoris in the middle of the thigh, but is then less violent than when the patellar tendon is struck. If a portion of skin lying over the patellar tendon be pinched up and struck, a marked contraction of the quadriceps can be produced, notwithstanding that every care be taken to prevent any dragging on the tendon.

The legs frequently jerk of themselves, and are sometimes drawn up when the patient is lying quietly in bed.

Spine.—On the left side of the spine, at the level of the sixth dorsal vertebra, the back looks somewhat swollen and is tender, but no fluctuation can be detected.

The *nervous system* is otherwise normal.

The *tongue* is clear; the appetite good. The pulse numbers from 80 to 90 in the minute, and is visible, tortuous, and jerking. These characters are not increased on raising the arm, and there is no aortic regurgitation. The jerking is evidently nervous, the heart's action being very irritable.

Progress of the Case.—During the first two months after

his admission he continued, as regards the lower extremities, *in statu quo*; but the swelling in the back became more prominent, and at last yielded distinct fluctuation. It was opened antiseptically, and a large quantity of pus was evacuated. The patient was then transferred to the surgical wards, under the care of my colleague, Dr. Arnison.

Shortly after the abscess was evacuated the rigidity and paralysis of the lower extremities began to get less, and at last altogether disappeared. The exaggeration of the patellar tendon reflex also decreased. When I last saw him the abscess was still unhealed; the rigidity and paralysis had quite gone.

Lesions of the particular portions of the spinal cord which contain the controlling fibres, passing downwards from the brain, will, by interfering with the functions of those fibres, lead to an increase in the patellar tendon reflex.

Some of these controlling fibres are supposed to pass downwards in the lateral columns.

In lesions, therefore, of the lateral columns we should expect to find the patellar tendon reflex exaggerated; and such is the fact.

Lesions of the lateral columns may be primary or secondary, the latter being by far the most common.

Secondary or descending degeneration of the lateral columns sometimes follows myelitis.

The following are the notes of a case of paraplegia resulting from acute myelitis. In it the patellar tendon reflex was enormously increased, the increase being partly the result, in all probability, of exalted excitability of the grey matter.

Case 10.—*Acute Myelitis—Paralysis of Lower Extremities and of the Bladder and Rectum—Recovery at the end of five weeks—Relapse—Total Loss of Motion and Sensation—Paralysis of Bladder and Rectum—Flaccidity of Muscles—Some Increase of the Patellar Tendon Reflex—Slow Partial Recovery—Onset of Rigidity of Muscles—Spastic Gait—Enormous Increase of Patellar Tendon Reflex.*

C. J., aged forty, married, a shoemaker, was admitted to the Newcastle-on-Tyne Infirmary, under my care, on June 15, 1878, suffering from paraplegia.

Previous History.—He has been a fairly healthy man through life, but has drunk hard (whisky and beer). Seventeen years ago he had rheumatic fever. Two years ago he contracted syphilis. The present attack commenced eight weeks ago. He used, he says, to have a bad habit of holding his water. One day, after sitting at work for a long time, during which he overcame his desire to urinate, he found, on attempting to get up, that he was unable to do so. His feet were cold and numb, and he felt a pain at the lower part of the abdomen. After a little time he managed to move his legs, and walked home with assistance. The next morning he was very much pained in the region of the bladder, and was unable to make water; the urine had to be drawn off with the catheter. For the next five weeks the paralysis of the bladder continued, and he had very little power in the lower extremities. He then began to improve, and became able to walk about with the help of a stick. A fortnight ago he again suddenly lost the use of his legs, and has been confined to bed ever since.

For two months before the first attack he noticed that his legs were weak and shaky, and that he had some difficulty in making water.

Family History good.

Present Condition.—Is very pale and anæmic. The lower extremities are completely paralysed, the muscles being flaccid and somewhat wasted. The *bladder* is quite paralysed. The *bowels* are very costive.

Sensibility of all sorts is very greatly impaired in the lower extremities. He says he could feel nothing during the first week of the relapse. He feels a tightness round the lower part of the abdomen. At the upper edge of this band there is very distinct hyperæsthesia.

Both the skin reflex and the reflex from the patellar tendon are exaggerated.

There is no irregularity of the spine and no tenderness on pressure.

The temperature of the right foot is 97·2° Fahr., that of the left 96·5° Fahr. The memory is somewhat impaired; the nervous system otherwise natural. The tongue is dry,

and he complains of thirst. The pulse is 60, full, and somewhat irregular.

He was treated with the liquid extract of ergot and with tonics, and improved considerably.

On September 7, being able to walk with a little assistance, he went home of his own accord.

For some time after his discharge he continued *in statu quo*, some days better, and other days worse. One day he was able to walk round his house by the help of two sticks. At the beginning of November he began to feel his legs stiff, and noticed that they trembled whenever he attempted to walk ; this trembling was often so violent as to shake the people who were supporting him.

On December 2 he was re-admitted to the Infirmary under my care. He could still move the legs when lying on his back in bed. If, however, he turned on to his face, he was unable to stir them. Any exertion caused the legs to become rigid. Sensibility was now almost normal.

All the reflexes very greatly exaggerated. On striking the ligamentum patellæ the leg was jerked violently forward, and continued to be so jerked for two or three minutes. A blow on the ligamentum patellæ of one leg was followed by strong jerking movements of both legs: these movements were alternate—that is to say, they did not occur exactly at the same time.

On freezing the skin over the ligamentum patellæ the movement was very greatly reduced in intensity, but was still present. It now resembled, both in its extent and in the fact that it was a single jerk, the normal movement in a healthy person. At the commencement of the freezing—*i.e.*, when the spray first came in contact with the skin over the ligamentum patellæ—a tonic contraction of the quadriceps, resulting in elevation of the foot, took place.

The ankle clonus was very marked. On striking a limited area over the inner edge of the right tibia, a very marked upward movement of the leg and foot resulted, quite different, however, from the jerk due to the contraction of the quadriceps. The thigh was in this movement raised from the

table, the movement being apparently due to a contraction of the psoas and iliacus muscles.

He cannot walk unless supported on each side; his gait is then very peculiar and characteristic. The feet and knees interlock, the feet stick to the ground, and the toes in movement make a scraping noise. As soon as the toes leave the ground, the foot is jerked upwards, and raised very much higher than one would expect. The foot is then brought forward, and tends to cross its fellow of the opposite side. The legs often strike when the toes touch the ground. He complains of feeling the back and hips weak. The urine has still to be drawn off with the catheter.

The electrical condition of the muscles was carefully tested: there was a simple diminution, moderate in extent, to both forms of current.

Progress of the Case.—He remained under treatment for some months, but there was no real improvement. Remarkable changes, however, took place in the rigidity of the limbs and the extent of the tendon reflex. For some weeks the rigidity was much less, and the patellar tendon reflex proportionately diminished; then both returned, and continued to be as great as ever. These alterations could only, I think, be due to some altered condition of the grey substance of the cord, whereby its excitability to reflex stimulation was altered.

Secondary degeneration of the lateral columns also occurs after lesions of the internal capsule and cortical motor centres of the cerebrum.

The following case, which occurred in the practice of my friend and late colleague, Dr. Philipson, to whose kindness I am indebted for the opportunity of examining the patient, and with whose permission I publish these notes, is a good example of hemiplegia presumably associated with secondary lateral sclerosis. In it the patellar tendon reflex was much greater on the paralysed than on the sound side.

Case 11.—*Case of Hemiplegia and Rigidity in a Boy aged nine, following a Wetting—Tendon Reflex Exaggerated on*

the Rigid or Paralysed Side—Great Improvement under Treatment.

D. C., aged eleven, schoolboy, was admitted to the Newcastle-on-Tyne Infirmary, under the care of Dr. Philipson, on September 26, 1878, suffering from left-sided hemiplegia.

Previous History.—He had been a healthy lad until two years previously, when he caught cold after bathing in his clothes and allowing them to dry on. He was confined to bed for some time with fever and headache. The attack was followed by paralysis of the left arm, left leg, and left side of the face.

When admitted to the Newcastle Infirmary, the paralysis of the arm, leg, and face was still very marked; he could walk with great difficulty; the paralysed limbs were very rigid and contracted. He was treated with iodide of potassium and blisters to the right side of the head, Dr. Philipson having diagnosed that the hemiplegia depended on arachnitis and effused lymph. Contrary to the usual experience in old-standing cases of hemiplegia with rigidity, he steadily improved: the rigidity became much less, he regained to a large extent the use of the lower extremity, and to a considerable extent that of the upper.

I examined him a few weeks after his admission, while the rigidity of the leg was still marked; *the patellar tendon reflex on the rigid or paralysed side was very much greater in extent than on the sound side.*

This case is very interesting from the rarity of the lesion (Dr. Philipson diagnosed it as hemiplegia resulting from arachnitis) and from the great improvement under treatment.

Primary sclerosis of the lateral columns is said to be the lesion in the disease which Erb calls spasmodic spinal paralysis. The following is a typical example of that affection:—

Case 12.—*Paraplegia due to Rigidity and Weakness of the Lower Extremities—Sensibility Normal—Bladder and Rectum Normal—Patellar Tendon Reflex Exaggerated.*

Robert S., aged forty-three, widower, a pitman, was admitted to the Newcastle-on-Tyne Infirmary, under

my care, on April 19, 1877, complaining of difficulty in walking.

Previous History —His present illness commenced about three years ago, and was brought on, he thinks, by exposure to cold and wet. The first symptoms were numbness and stiffness in the left leg. The right soon became similarly affected, and he began to have difficulty in walking. For the past year he has been much worse, and for several months has been unable to walk. There has been no pain. He has felt twitchings in the lower part of the abdomen, and has been much troubled with "twitchings" and "jumpings" in the lower extremities.

He never ailed anything until the present attack commenced. He has not had syphilis. Has been somewhat addicted to drink.

Family History good. No tendency to nerve disease.

Present Condition.—He is quite unable to walk unless supported on each side. He then gets along in a very characteristic fashion: the feet are dragged, the toes appearing to be stuck to the ground, and at each step cause a very unpleasant scraping noise; the knees seem stuck together. At each step the foot, which is carried forward, is brought in front of and across its fellow; this evidently adds to the difficulty in getting along. The back is much arched, and he seems to walk from the spine. He can stand with the feet close together; the back is not then arched, and there is no appearance of pseudo-hypertrophic paralysis. The muscular development of the lower extremities is good: there has been no wasting; in fact, he thinks the legs are, if anything, thicker than they used to be. The right calf, four inches below the head of the fibula, measures fourteen inches; the left at same spot, thirteen inches and three-quarters. The right thigh was broken when he was fourteen years of age, and the right leg is consequently a little shorter than the left.

When lying in bed his legs are kept extended; he is unable to raise the heels from the bed. The legs are generally rigid. This is always the case when he makes any attempt at voluntary movement or when passive movements are made.

Very marked tremblings and twitchings are seen in all the muscles of the lower extremities, also in the great pectoral muscles. Some of the twitchings are fibrillary. The twitchings and tremblings are much worse after exposure to cold.

Sensibility.—He has no pain, but complains of numbness in the soles of the feet. The toes sometimes feel swollen, and as if they would burst. The power of localising impressions is almost perfect. Tactile sensibility and sensibility to temperature are normal.

Intellectual faculties natural. Memory good. He sleeps well.

Special Senses.—Sight good; pupils equal and moderately contracted. Slight arcus senilis. Discs natural.

Hearing slightly duller on the left than on the right side.

Taste and *smell* natural.

Reflex from skin natural. Urination natural; bowels very costive.

Alimentary System.—Tongue slightly furred. Appetite poor. Bowels costive.

Circulatory System.—Heart-sounds weak but natural; pulmonary second louder than aortic. Radial pulse 72, regular, visible, and tortuous, but no jerking collapse. Temporal artery also visible and tortuous.

Respiratory System normal.

Urinary System normal. Urine, specific gravity 1023, and quite normal.

Progress of the Case.—He remained under treatment until the beginning of September, and was then discharged much *in statu quo*. While in hospital there were periods of temporary improvement, during which the rigidity became much less, and during which he was able to walk by himself. A variety of drugs were used: strychnine made him worse; ergot of rye seemed to be beneficial.*

In the next case, too, the lateral columns seemed to be those

* In June last the patient was admitted to the Edinburgh Royal Infirmary, where, through the kindness of Professor Sanders, I had an opportunity of seeing him. *The patellar tendon reflex was greatly exaggerated.*

chiefly affected. The other parts of the cord were also probably involved at the earlier stage of the case. The case is interesting from the peculiar "hopping" gait.

Case 13.—*Male, aged twenty-four—Gradual Weakness of Bladder—Exposure to Cold—Weakness, Numbness, and Rigidity in the Lower Extremities, gradually increasing to Absolute Paralysis, with Loss of Sensibility and Affection of the Bladder—Tension of Muscles, with Twitchings—Partial Slow Recovery—Hopping Gait—Great Increase of the Patellar Tendon Reflex and of the Ankle Clonus.*

T. H. M., aged twenty-six, a compositor, single, was admitted to the Newcastle-on-Tyne Infirmary under my care on July 2, 1878, suffering from paraplegia.

Previous History.—Three years ago he contracted syphilis; the primary sore was followed by a skin eruption, sore throat, and loss of hair. With this exception he has enjoyed good health until two years ago, when his present trouble commenced. It was brought on, he thinks, by catching cold. One day, after bathing, he sat for some time on damp grass. He felt chilled, and a week afterwards he noticed that his feet and legs were cold and numb. He felt his legs and knees gradually become weaker and weaker. He became unsteady in his gait, and walked as if he were giddy. His legs were stiff and tense; he frequently felt twitchings and jumpings in the muscles. At the end of two months he had completely lost the use of his legs. There was no feeling in the paralysed parts; his urine dribbled away from him; the bowels were obstinately constipated. He was at this time admitted to the Sunderland Infirmary.

After careful cross-examination, I ascertained that for some months before the supposed commencement of the attack he had experienced some difficulty in making water, and had noticed that he was sexually weaker than formerly. He remained three months in hospital at Sunderland, and was then discharged slightly better, being able to stand and to move his legs up and down in bed. He has slowly improved since, and can now walk by the help of two sticks.

For some years he had been unsteady, but had been

strictly teetotal for three months before the commencement of the attack.

The *Family History* is good ; there is no evidence of any hereditary nerve-disease.

Present Condition.—He is well nourished and looks fairly healthy in the face. He can walk tolerably well with the help of sticks.

Gait.—The gait is "hopping" and very peculiar. Each step is evidently a great effort. He seems to walk entirely from the hips, back, and shoulders ; the knees are kept quite stiff and rigid ; the toes are dragged along the floor with a most unpleasant scrape. At the commencement of each step the toes are flexed, the foot is slightly inverted, and it is the outer aspect which scrapes the floor ; the foot is then suddenly raised with a jerk high off the ground. When the foot comes to the ground the toes touch first, and the heel is afterwards brought down with a "jolt," which, he says, is a very uncomfortable sensation. He thinks the difficulty in walking is due (1) to the toes and feet dragging against the ground, (2) to the stiffness of the knees, and (3) to weakness in the back.

He walks a great deal better in thick boots with high heels. He walks much worse when anyone is looking at him.

Condition of the Muscles.—The muscles of the lower extremities are somewhat flabby, but there is no appreciable wasting. There is, however, marked loss of power in certain muscles. When lying on his face he is unable to raise his legs from the bed. There is, too, some resistance to passive flexion of the knee ; the hamstring tendons are rigid. He himself says he feels a tightness under the knees when the legs are flexed.

When lying on his back he can raise his legs from the bed, but only with difficulty. Flexion of the thigh on the abdomen can be naturally performed ; but if he tries to extend the leg on the thigh there is a great deal of trembling in the limb, and the movement cannot be completed.

The *electrical contractility* of the muscles to both currents is below par, but there are no qualitative changes.

The legs frequently tremble and jump; there are, however, no true fibrillary twitchings. Sometimes, when he is lying in bed, both legs will be suddenly flexed on the abdomen. The trembling of the legs is not increased by exertion, but the slightest excitement brings it on at once. Twitchings of the facial muscles occur at the same time.

All the reflexes (skin, patellar tendon reflex, and the ankle clonus) are very much exaggerated.

There is no impairment of co-ordination. He stands unsteadily with his eyes shut, but this is simply from nervousness and the trembling which is thereby excited.

He feels his legs and back weak. He is always worse first thing in the morning. The *urine* contains phosphates, but is otherwise normal; it is still passed in a slow stream; sometimes it stops; sometimes he cannot hold it. The *bowels* are very costive.

Sensory Functions.—He occasionally feels darting pains in the legs below the knee. These pains are always worse when he is quiet; he never feels them when he is moving about. They appear to be myalgic. He feels his legs and feet numb.

Sensibility.—Tactile sensibility is somewhat impaired on the outer aspect of both legs below the knee; elsewhere it appears to be normal. Sensibility to temperature is natural, except in the outer aspect of the left leg. Sensibility to pain is everywhere normal.

The optic discs are natural, the pupils somewhat widely dilated.

His *mental* functions are normal.

The *special senses* are all normal.

The *heart* is irritable and excitable; he suffers occasionally from palpitation.

All the other organs are normal.

Treatment.—A great many drugs, antisyphilitic and others, together with electricity (constant current), were tried, but without any decided beneficial effect. The patient was discharged on October 10, much *in statu quo*.

Increased excitability of that portion of the grey matter

of the cord with which the reflex fibres are connected will also cause exaggeration of the phenomenon. This is seen in some cases of myelitis; also after the administration of strychnine. I took the opportunity of proving this experimentally in the case of compression of the cord which I have already related (Case 9). To that patient I gave several full doses of the drug, with the result that this phenomenon was much intensified.

To sum up, then, the following conditions tend to increase the patellar tendon reflex:—

1. Increased excitability of the grey matter of that particular portion of the cord through which the reflex travels.
2. Lesions which destroy or arrest the function of those fibres which inhibit this particular reflex act. In these cases the segment of the cord through which the reflex travels must be sound.

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