CLINICAL LECTURE

ON

A CASE OF LOCALISED MYELITIS,

AFFECTING THE FIFTH LUMBAR AND THE SACRAL SEGMENTS OF
THE SPINAL CORD, THE RESULT OF A SLIGHT
TRAUMATIC INJURY;

AND

A CASE OF PARALYSIS OF ALL FOUR LIMBS,

DUE TO A SPINAL INJURY.

Delivered at the Edinburgh Royal Infirmary

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THE first case to which I propose to direct your attention to-day is one of great interest and rarity; it presents a number of important diagnostic problems and raises several questions of great physiological interest; it is, therefore, well fitted for the consideration of those of you who are senior students and who are already acquainted with the symptoms which characterise the ordinary forms of disease of the spinal cord. But since it also very clearly demonstrates the manner in which the clinical examination of the spinal cord should be conducted and the methods by which we endeavour to arrive at an exact diagnosis in cases in which the spinal cord is diseased, it will also prove instructive to those of you who are beginners. The notes of the case are as follows:

CASE 1. Complete Motor and Sensory Paralysis of the Parts (Muscular and Skin Areas) supplied by the Fifth Lumbar and the Sacral Segments of the Spinal Cord, of very slow Development, together with Bed Sores, Total Loss of Sexual Power, and Partial Paralysis of the Bladder and Rectum, the result of a Slight Traumatic Injury, and probably due to Sclerosis (Chronic Myelitis) the result of a Minute (Capillary) Hemorrhage into the Grey Matter of the Cord: Partial Recovery under Treatment.—G. A., aged 39, a hotel keeper and book-maker, was admitted to the Edinburgh Royal Infirmary on May 26th, 1897, suffering from paraplegia.

Previous History of Present Illness.—The patient states that on June 17th, 1895, he jumped off the box of a waggone on to the hard road, alighting on his feet. His legs gave way and he fell on his back. He immediately got up, feeling as if he had got a severe shake, and walked into the house, a distance of twenty yards. After sitting down for five or ten minutes he felt all right, except for a slight pain and what he terms a "twittering" sensation in the right ankle. There was no pain in the back; in fact, he felt nothing wrong with his back. He then got into another "machine" and drove to the railway station. The next day the right ankle still felt weak and painful, but it was neither swollen nor discoloured. For the month following the accident he continued to go about his ordinary duties, feeling the right ankle weak and painful. He states that during this period he experienced a feeling of numbness and coldness in the right foot and leg below the knee, but that when he took hold of the foot and leg they were warm and natural. At the end of a month, as the weakness and numbness in the right ankle were increasing, he consulted his ordinary medical attendant, who advised him to give the ankle as much ease as possible. Notwithstanding this advice, he continued to walk about and do his ordinary work. Soon after this (about five weeks after the injury) the pain in the ankle disappeared, while the weakness increased. Four months after the accident the left ankle also became weak. Seven months after the accident he noticed that he was unable to move the toes of the right foot, and that the right ankle was so "wobbly" that it appeared to be out of joint. The calves had now, he states, become considerably smaller (wasted). In February, 1896 (nine months after the accident), he went to an auction sale, and was exposed to cold and wet. On returning home at night the left leg and foot felt cold and numb, and he noticed that he was unable to move the toes of the left foot. He again consulted his medical attendant, who sent him to bed and prescribed for him. He remained in bed for about three weeks. He then, contrary to the doctor's advice, got up and went about his usual work. After this date (March, 1896) the difficulty in walking and loss of power in the legs slowly but gradually increased. For the past ten months (since August, 1896) he has not been able to walk without assistance (the help of a friend, or holding on to furniture, etc., with his arms); nevertheless, he continued to get up every day and sit about the house. In February, 1897, he again took to bed, and has been in bed ever since. During the second week of March, 1897, he burned his right heel with a hot water bottle; although the burn was very severe it caused him no pain. As the ulcer did not heal, he was admitted, by the advice of his doctor, to the Carlisle Infirmary on March 24th. He remained in the Carlisle Infirmary for six weeks, until the ulcer was healed. During his stay in the Carlisle Infirmary the paralysis continued to increase. A week after his admission to the Carlisle Infirmary, he experienced, for the first time, pain in the small of the back. The pain was of a shooting character, and extended round the sides of the abdomen. These back and girdle pains still occasionally occur. About the same time he also experienced for the first time some difficulty

in making water; he states that he had to exert greater force to get the water away, and that there was some dribbling at the end of micturition. For the past year he has completely lost all sexual appetite and power. For the first year after the accident he noticed no difference in his sexual power.

Previous History, Prior to the Present Illness.—Up to the date of the accident the patient was a very muscular and robust man, his weight being 15 st. 8 lbs., and his height 5 ft. 10½ in. He has never had any venereal disease. He had an attack of rheumatic fever at the age of 13, and congestion of the lungs at the age of 21. He has been a very heavy drinker. He states that for the past eight or ten years he has, during the racing season, which lasted six or seven months, drunk on an average over a bottle of whisky a day, besides large quantities of wine and malt liquors.

The Family History is unimportant. The patient does not appear to inherit a tendency to any special form of disease. He has had five healthy children; the youngest is 4 years of age.

State on Admission.—The patient, who is, you will observe, a strongly built and very broad-shouldered, though somewhat flabby looking man, weighs 14 st. 2 lbs., his height being 5 ft. 10½ in. The temperature is sub-normal; the general health is good; and the viscera are all healthy. There is in short no evidence of disease except in the parts supplied by the fourth and fifth lumbar, and the sacral segments of the spinal cord. The other parts of the nervous system appear to be absolutely healthy. The description of the patient's condition on admission, in fact, resolves itself into a description of the motor, reflex, sensory, vasomotor, and trophic conditions of the parts supplied by the fourth and fifth lumbar and the sacral segments of the spinal cord. The spinal column is perfectly normal, straight, and well formed; there is no tenderness on percussion, no hyperæsthesia—nothing, in short, to suggest that the patient has ever received any spinal injury.

The Condition of the Motor Nerve Apparatus.—The patient is unable either to stand or walk, though he states that he thinks he could do so if he were supported on each side. There is complete paralysis of all the muscles of both lower extremities below the knee. Footdrop is very marked in each leg, more particularly in the right. The patient is absolutely unable to move the toes, or the feet on the ankle-joints. The patient can flex and extend the leg on the thigh, but these movements are performed much less forcibly than one would expect in a man of his powerful muscular physique. Flexion, adduction, and abduction of the thigh on the abdomen (hip joint) are normal and vigorously performed; extension of the thigh at the hip joint (gluteus maximus) is feeble.

The Condition of the Paralysed Muscles.—The muscles on both the front and back of the legs are markedly atrophied and flabby. The muscles of the thighs are large and fairly firm. The buttocks (glutei muscles) are soft and very markedly atrophied. The right calf at its thickest measures 12½ and the left 12½ inches; while the right thigh at its middle (¾ inches above the patella) measures 19½ and the left 19½ inches. The muscles both on the front and back of the legs do not respond to the strongest faradic currents; while the muscles of the thighs react normally to faradism. Fibrillary twitchings occasionally occur in the vastus externus and quadriceps extensor femoris on both sides. The calves and paralysed muscles are not, and never have been, tender to pressure (pinching, etc.).

The Condition of the Reflexes.—The plantar reflex is absent on both sides. The cremasteric reflex is very active on both sides. The knee-jerks are present, but markedly diminished in degree, more especially on the left side.

The Vestical Reflex.—The patient knows when he wants to make water, but does not feel the water passing. In order to empty the bladder voluntarily, much forcing and straining (of the abdominal muscles) is required. The water dribbles away; laughing and coughing cause the involuntary expulsion of urine. In short, both the sphincter and the detrusor muscles of the bladder appear to be partially paralysed.

I may take this opportunity of stating that at the date of the patient's admission to hospital the urine was slightly alkaline, but free from any abnormal constituents (mucus, pus, albumen, etc.).

The Rectal Reflex.—The patient is more constipated than he used to be, but, as a rule, the contents of the bowels are evacuated without medicine. He knows when he needs to defæcate, but he has no sensation as the stool passes. When the motions are loose (that is, after a dose of laxative medicine), the fæces come away without his knowledge. The contents of the rectum are often expelled involuntarily and without his knowledge during the straining efforts which are required for micturition. The sphincter, as well as the expulsores muscles of the rectum, appear therefore to be partially paralysed.

The Condition of the Sensory Nerve Apparatus: Tactile Sensibility.—There is complete loss of the tactile sensibility over the toes, the adjacent parts of the feet, both the soles and dorsa, more particularly the outer sides; a small area on the lower and outer sides of the legs, the penis and scrotum, the perineum, and the middle of the back of each thigh. The exact distribution of the anaesthesia which was present at the date of admission is shown in Figs. 1 and 2.

Sensibility to Pain.—This is lost over the same areas as the tactile sensibility, but the area of the analgesia is somewhat more extensive than the area of the anaesthesia, involving as it does the whole of the plantar and dorsal surfaces of the feet, and a larger area of both legs. On the right side of the scrotum the painful sensations are retained in a small area in which tactile sensations cannot be perceived.

Sensibility to Heat and Cold.—This is lost over the same areas as the sensibility to touch and pain, but the area of the thermal anaesthesia is more extensive than the area of the analgesia, and still more extensive than the area of anaesthesia, involving a larger surface both on the front and back of the legs.

Vasomotor and Trophic Condition.—Over the right tendo Achillis there is a purplish cicatrix, measuring 2 inches by 1½ inch, the result of the burn previously described. Over the head of the right metatarsal bone there is a brown discoloration about the size of a sixpenny piece, due to an injury (a horse trod upon the foot) received about some six or seven months after the original accident. On the left buttock there is a superficial ulcer of considerable size (2½ by 2 inches), and on the right buttock a smaller ulcer about the size of a shilling. When the feet are exposed (swung down over the bed for a short time), they become blue, cold, and swollen.

I have now described the condition of the patient when he came under our observation. Before commenting on the case, let me say a word or two as to the treatment which has been employed and the alterations which have taken place up to this date (June 29th, 1897).

Figs. 1 and 2.—Front and back views, showing the area of anesthesia at the date of the patient's admission, in the case of G. A. described in the text. The solid black areas to which the letter A points shows the area of distribution of the tactile anesthesia. The shaded area to which the letter B points indicates the extent of the loss of sensibility to pain (analgesia). The dotted area to which the letter C points indicates the area of thermal anesthesia.

Treatment and Progress of the Case.—The patient was placed on a water bed; special instructions were given to the nurses to see that he was kept scrupulously dry and clean, and a mixture containing boracic acid and strychnine was prescribed. On June 2nd the sole of the right foot was burned by a hot-water bottle. The blister was opened and the wound was washed with carbolic lotion (1 in 40), and dressed with zinc ointment. On June 7th it is noted that the urine is now acid in reaction, and deposits crystals of oxalate of lime. The patient states that he has more control over his urine; he knows when the bladder is full; he can pass urine with a more forcible stream; the dribbling has ceased. June 11th. Says he feels a tingling sensation in the left foot and toes. The burn on the right sole is healing very slowly; there is no change in the ulcers on the buttocks. June 30th. There is little change since the last note. The urine continues to be acid. The patient has more control over both the bladder and rectum. The area of tactile anesthesia is slightly smaller in the feet and legs, unchanged over the buttocks, penis, scrotum, and thighs. The ulcers on the buttocks show no signs of healing. The paralysis, the condition of the paralysed muscles, and the superficial and deep reflexes are unchanged.

Such are the facts of the case. Let us now consider the diagnosis.

DIAGNOSIS.

In all cases of disease in which there is an organic lesion of the nervous system, the object of the physician is not merely to give a name to the disease, but to make an exact anatomical and pathological diagnosis. Both the anatomical and pathological diagnoses are of importance, not merely from a scientific point of view, but for the practical purposes of prognosis and treatment.

The object of the anatomical diagnosis is to determine the exact part of the nervous apparatus which is directly implicated by the lesion. The first point which has to be decided (and this is usually arrived at without difficulty) is, Does the lesion involve the peripheral nerves, the spinal cord, or the brain?

Now, in the particular case which we are studying to-day the symptoms are paraplegic in distribution; they consist in very marked and localised paralysis of motion, of reflex

action and of sensation, together with trophic and vasomotor disturbances in those areas of the body which are supplied by certain nerves coming off from the lower end of the spinal cord. The paraplegic distribution of the symptoms and the complete absence of cerebral symptoms enable us to exclude a cerebral lesion, and show that the lesion is situated either in the spinal cord or the peripheral nerves. A lesion in either of these situations (spinal cord or peripheral nerves) may produce paralysis of motion, loss of reflex action, loss of sensation, trophic and vasomotor disturbances, and in both cases the symptoms may be distributed in a paraplegic manner; nevertheless, careful examination usually shows that the symptoms are grouped and arranged in a different manner. In those cases in which the lesion is situated in the spinal cord and peripheral nerves respectively.

Paraplegically-distributed symptoms (such as are present in this case—paralysis of motion, sensation, reflex action, with trophic and vasomotor disturbances) due to a lesion in the peripheral nerves may be the result either of (1) a peripheral neuritis caused by a poison circulating in the blood which affects a large number of the peripheral nerves throughout the body—a polyneuritis; or (2) to the fact that a gross lesion, such as a tumour, is so placed that it involves the nerve roots or the peripheral nerve trunks, which are distributed to both lower extremities.

In this particular case it is obvious that the symptoms are not due to a peripheral neuritis of toxic origin, a polyneuritis or widespread inflammation of the nerves resulting from the action of a poison circulating in the blood. The symptoms are clearly indicative either of a lesion of the spinal cord or of the cauda equina, though some of you may perhaps have come to the conclusion that the case is one of alcoholic neuritis. This diagnosis was, in fact, suggested by the patient's medical attendant, who sent the case to me.

The points which at first sight appear to be suggestive of alcoholic neuritis are:

That the patient has for many years been very alcoholic; that the muscles below the knee are paralysed, and that there is double foot-drop; that the paralysed muscles are markedly atrophied, and that they present the reaction of degeneration; that the sensory functions of the skin in the paralysed parts of the limbs (legs below the knees) are markedly involved; and that there are vasomotor and trophic derangements in the same parts.

But the following facts are entirely opposed to this view:

(a) *The Gradual Mode of Onset and the somewhat Progressive Course of the Symptoms.*—A paralysis due to alcoholic neuritis is much more rapidly developed, it much more quickly reaches its height; and, after the maximum degree of paralysis has been reached, it tends, almost without exception (provided that the cause—alcohol—is removed, that suitable treatment is adopted, and that there are no visceral complications preventing recovery), to improve, at all events in some, and usually in a very marked, degree. In uncomplicated cases of alcoholic paralysis the progress is in fact (always provided that the conditions for treatment are satisfactory) eminently favourable.

(b) *The Remarkable Limitation of the Paralysis.*—An alcoholic neuritis, sufficiently severe to have produced such complete paralysis and such extreme atrophy as are present in this case in the muscles below the knees and in the glutei muscles, would have been much more widely spread. It would not have been confined merely to the muscles below the knee and the glutei muscles; it would almost certainly have involved other muscles, particularly the extensor muscles of the forearm, producing wrist-drop, the small muscles of the hand, and in some, and probably in a marked degree, the muscles of the thighs, and probably also the muscles of the upper arm. In particular, if the case had been one of alcoholic paralysis, the glutei muscles would not have been picked out, paralysed and atrophied in the extreme degree in which they are in this case.

(c) *The Absence of Pain and Muscular Tenderness.*—In this case there has been absolutely no pain in the paralysed parts, and there is no muscular tenderness. Now, tenderness of the paralysed muscles, particularly on pinching the calf muscles, is one of the most important diagnostic signs of alcoholic peripheral neuritis. It is so constantly present as to constitute a most important diagnostic feature of the disease.

(d) *The Complete Absence of Cerebral Symptoms.*—An alcoholic neuritis sufficiently severe to produce the extreme degree of paralysis and atrophy which are present in the muscles of the leg in this case would have probably been attended at its onset with some disturbance of the cerebral functions. But such was not the case.

(e) *The Presence of Symptoms Indicative of Derangement of the Vesical, Rectal, and Sexual Reflexes.*—In cases of alcoholic neuritis, these important reflexes are not impaired unless the spinal cord should at the same time happen to be implicated. This is an important point of differential diagnosis between a paraplegia due to disease of the peripheral nerves and a paraplegia due to disease of the spinal cord.

(f) *The Peculiar Distribution of the Sensory Paralysis.*—This is a very important point. In this case the loss of sensibility to touch, pain, and temperature is very definitely limited to the areas of distribution of the nerves coming off from the fourth and fifth lumbar and the sacral segments of the spinal cord. In particular, I would beg you to note the presence of the saddle-shaped area of the anæsthesia on the buttocks, and the very localised area of anæsthesia over the penis and scrotum. Now in a case of alcoholic polyneuritis the anæsthesia would not be localised in this way.

(g) *The "Disassociated" Anæsthesia.*—The fact that the area of anæsthesia is less extensive than the area of analgesia, and that the area of thermal anæsthesia is still more extensive than the area of analgesia. This is not what one would expect, though I am not prepared to say it might not occur, in a case of alcoholic neuritis.

These facts clearly show that the case is not one of alcoholic neuritis, nor, I may add, of neuritis due to any other toxic cause such as diphtheria, lead poisoning, beri-beri, etc.

The next question which has to be determined is, Does the paralysis depend upon a lesion of the spinal cord, or upon a gross lesion, situated in the pelvis or in the lower part of the spinal canal, which involves the nerve roots or the peripheral nerve trunks passing to both extremities at a point at which they lie in close contact, in close anatomical juxtaposition?

A lesion in the pelvis may be at once excluded because of the following facts:

(a) The motor paralysis is practically limited to the glutei muscles and the muscles below the knee. Now, a lesion in the pelvis, such as a tumour, which produces paraplegia by pressing upon or causing inflammation of the great nerve trunks passing to both lower extremities, would be very unlikely to produce the extreme degree of paralysis in the glutei muscles and in the muscles below the knees which is present in this case, and the marked muscular atrophy and the reaction of degeneration, etc., which are present in these muscles, without at the same time producing a very marked degree of paralysis of the muscles of the thighs.

(b) The absence of neuralgic or shooting pains in the paralysed parts. A tumour in the pelvis which involves the great nerve trunks, and so produces paralysis of both lower extremities, is almost invariably (? always) attended with more or less pain in the areas of distribution of the nerves, which are irritated and pressed upon. The same statement applies, as we shall presently see, to lesions of the cauda equina. In both of these cases the pain is usually such a marked and striking symptom that they are termed cases of "painful paraplegia." The term "painful paraplegia" may, in fact, in most cases be regarded as practically synonymous with a paraplegia due to the pressure of a gross ("coarse") lesion, such as a tumour, a displaced vertebra, or a mass of inflammatory products, upon the peripheral nerve trunks, or upon the motor and sensory nerve roots within the spinal canal.¹

(c) A pelvic lesion involving the nerve trunks would not be likely to produce the very sharply localised derangements of sensation which are present in this case. The anæsthesia or painful anæsthesia (for in many cases of paraplegia due to pressure on the nerve trunks painful sensations are experienced by the patient in those areas of skin which are completely devoid of sensation when touched, pinched, etc., hence the term "painful anæsthesia" or "anæsthesia dolorosa") would be much more widely distributed; it would involve the thighs as well as the legs, and it would not (or would be most unlikely to) pick out the saddle-shaped area over the buttocks, the scrotum and penis as the anæsthesia in this case does.

(d) There would probably be no derangement of the bladder and rectal reflexes.

But, further, (e) in this case there is absolutely no evidence of any lesion in the pelvis, and (f) there are no symptoms indicative of pressure on the blood vessels (arteries or veins). A pelvic tumour which produces paraplegia in the lower extremities by pressing upon the large nerve trunks very usually at the same time produces engorgement or obstruction of the veins and swelling of the feet and legs.

We may exclude, then, a lesion of the peripheral nerves outside the spinal canal, and narrow the diagnosis down to a lesion of either (a) the spinal cord, or (b) the cauda equina.

It is sometimes extremely difficult to determine whether a paraplegia depends upon a lesion involving the spinal cord or the nerve roots composing the cauda equina; but in this case the nature and distribution of the symptoms, the manner in which they have been developed, and the whole progress and course of the case, seem to me to show that the lesion is probably situated within the spinal cord, and that the symptoms are not due to a lesion of the cauda equina.

But in order that you may clearly understand this point, it will be necessary to discuss the question in more detail:

(a) The first point which I would wish you to note is that the symptoms (paralysis, anæsthesia, disturbances of reflex action, vasomotor and trophic derangements) are distributed in a physiological rather than an anatomical manner. The motor, sensory, reflex, vasomotor and trophic disturbances are sharply limited, or almost entirely limited, to the areas of distribution of the nerve roots coming off from the sacral and perhaps the lowest segment of the lumbar enlargement of the spinal cord (the fifth lumbar, first, second, third and fourth sacral segments). The only symptoms indicative of derangement of the fourth, third, second, or first lumbar segments, or of the nerve roots connected with those segments of the spinal cord, are some slight weakness in the muscles of the thighs and some diminution of the knee-jerks (third and fourth lumbar segments).

Hence, if the motor paralysis and the other symptoms were due to a lesion, such as a tumour or inflammatory mass, involving the cauda equina, it is necessary to suppose that the special nerve roots connected with these segments (fifth lumbar, first, second, third, and fourth sacral segments) of the spinal cord have been picked out and damaged, while the other nerve roots connected with the lumbar enlargement have been spared, or at all events damaged in very slight degree.

The motor paralysis is, practically speaking, limited to the muscles below the knee, to the glutei, and to the muscles of the bladder and rectum. The sensory paralysis is limited to the areas of distribution of the first, second, third and fourth sacral nerves, and perhaps of the fifth lumbar nerve.² The vasomotor and trophic derangements are localised in the same parts. The disturbance of reflex action chiefly involves the reflexes passing through the sacral segments of the cord. The plantar and sexual reflexes, which are connected with the first, second and third sacral segments, are completely lost. The vesical and rectal reflexes connected with the fourth and fifth sacral segments are markedly impaired. The knee-jerks, which are chiefly connected with the third lumbar segment, though markedly diminished in degree, are preserved; and the cremasteric reflex, which passes through the second lumbar segment, is not only preserved but is usually active.

This physiological distribution of the symptoms is suggestive of a lesion of the spinal cord, though it might of course be produced by a lesion of the sacral plexus.

(b) *The "Disassociated" Character, or rather Distribution, of the Sensory Disturbances.*—The anæsthesia is less extensive than the analgesia, and the analgesia than the thermal anæsthesia. This is a strong argument in favour of the view that the lesion involves the cord itself rather than the nerve strands of the cauda equina.

(c) *The Absence of Pain.*—Lesions involving the cauda equina are usually due to disease of the bones of the spinal column, new growths such as syphilitic gummata or other forms of tumour involving the vertebral column, or situated within the lower part of the spinal canal, and inflammatory collections in the lower part of the spinal canal. In all of these cases pain in the back and shooting pains in the lower extremities and pelvis are usually marked symptoms. Now

in this case there was a complete absence of pain until a few months ago, and the pain which has occasionally been experienced since that date has never been severe, and has been more of a "girdle sensation" than the severe neuralgic pains which are characteristic of painful paraplegia. The pain which the patient has experienced may quite well be explained by irritation of sensory nerve fibres within the spinal cord at the junction of the diseased with the healthy cord tissues.

(d) *The absence of spasms and muscular contractions* is, for the same reasons, opposed to a lesion of the cauda equina.

(e) *The Symmetrical Manner in which the Symptoms (motor and sensory paralysis, etc.) are Distributed.*—We have seen that, if the paralysis, etc., were due to a lesion of the cauda equina, in order that the symptoms should be distributed in the exact manner in which they are in this case, the damage would (practically speaking) have to be limited to the roots of the cauda equina connected with the fifth lumbar and the first, second, third and fourth sacral segments of the spinal cord. But further, from the very symmetrical manner in which the symptoms are distributed on the two sides of the body, it would be necessary that each pair of these nerve roots would have to be involved in exactly the same degree. Practically speaking, this seems to me to be very unlikely. Coarse lesions such as tumours or inflammatory collections which involve the nerve strands of the cauda equina, usually involve some of the nerve strands (roots) more than others. Hence the symptoms (paralysis, anaesthesia, etc.) are rarely distributed in the highly symmetrical manner in which they are in this case.

(f) *The Mode of Development (very Slow and Gradual) and the Progressive Course of the Case.*—The history of the case and the mode of development of the symptoms (when taken in conjunction with the nature of the symptoms) are hardly such as one would expect if the paralysis were due to a lesion of the cauda equina.

From the account which I have given you it seems clear that the slight traumatic injury which the patient sustained on June 17th, 1895, was the starting point of the lesion. Immediately after the accident he felt that the right ankle was weak and that the right foot and leg were cold; and he states that these symptoms were not, so far as he can judge, the result of any direct injury to the ankle or foot. Four months after the accident the opposite (left) ankle became weak. After this the paralysis slowly but gradually increased until in the course of some months it reached its present degree of development.

Now, hæmorrhage on the surface of the cord, involving the strands of the cauda equina and sufficiently extensive to produce the symptoms which are present in this case, would in all probability have been attended with more marked symptoms at its commencement; and it is difficult to conceive that if a "coarse" extramedullary lesion (a meningitis, a tumour, or bone disease) had resulted from the injury and had implicated the nerve strands of the cauda equina, it would have developed in this case without producing other and more marked symptoms (pain, spasm, etc.). For these reasons we may, I think, exclude a lesion of the cauda equina, and arrive at the conclusion that the lesion is probably situated within the spinal cord itself.

The diagnostic discussion has occupied some time, and has been a somewhat complicated one, but I have done my best to make the argument clear. It would have been very easy for me to have given you my opinion in a few words, and to have arrived at the diagnosis by making a few dogmatic statements, but such a method of procedure would not have taught you anything. I am always anxious to carry conviction to your minds, to let you see the process of thought and reasoning by which I arrive at the conclusions I form, to explain to you the "whys" and "wherefores" of things, to teach you principles, and not merely to enumerate bare facts. Clinical medicine cannot be crammed up. True clinical knowledge can only be gained slowly and gradually. The student should not attempt to overburden his memory with mere facts. A knowledge of facts will come gradually, and can only come gradually. Take time, be patient, make a careful study of individual cases; train your powers of observation and of reasoning; but above all try to intelligently

understand everything that comes before you. Never be content with merely knowing that such-and-such symptoms characterise such-and-such a disease, or are associated with such-and-such a lesion; but try to intelligently comprehend why they are present in that disease, and the manner in which they are produced by the lesion.

(To be concluded.)

THE BENEFICIAL EFFECTS OF ONE DISEASE AS REGARDS ANOTHER.*

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ONE disease may act beneficially as regards another by preventing or curing it.

I. THE PROPHYLACTIC EFFECTS.

These can be dismissed in a few words, since our knowledge of them is limited. Passive pulmonary congestion and hypertrophic emphysema would appear to diminish the tendency to pulmonary tuberculosis. Certain germ diseases, again, are preventive of others. Thus the bacillus pyocyaneus counteracts the activity of the *B. pneumoniae* and the micrococcus erysipielatis. The various exanthems, on the other hand, do not confer immunity against one another, numerous instances having been recorded, from Murchison downwards, of two or more of them coexisting. One other instance of prophylaxis: Sir Dyce Duckworth¹ refers to the fact (observed by Diday and J. Hutchinson) that typhoid fever and pneumonia cause the postponement of the secondary symptoms of syphilis for from three to five months.

II. THE CURATIVE EFFECTS.

These are much more marked and well defined, and will be considered under five headings:

1. *Metastasis.*—This may be defined as the disappearance of disease from one part concurrently with its appearance in another. Thus, a urethral discharge may suddenly disappear—temporarily, at least—upon the supervention of orchitis, coriza upon the occurrence of pneumonia, quinsy upon the development of gouty arthritis. Dr. Mutch, of Brixton, saw an instance of the latter: Husband and wife were stricken with quinsy; when the former was in the height of the distress caused by the swelling, he developed acute gout in the toe, and forthwith, as if by magic, the throat trouble subsided; in the wife it ran the ordinary course. Now, the important question we have to ask regarding these cases is whether the occurrence of the new disorder acts curatively towards the one which it replaces. Perhaps the best examples of metastasis are afforded by functional nervous disorders, which shift their site or alter their expression from moment to moment, day to day, or even from year to year. Thus, in the course of an hour or two headache may give way to backache, backache to a fit of palpitation, and this again to an attack of gastralgia; and similarly, in the lapse of years, we may find migraine, spasmodic asthma, and angina pectoris successively displacing one another. Here again the question arises whether the supervention of the new disorder does not tend to keep its predecessor at bay, thus acting beneficially towards it. The probability is that it does. The alternation of morbid phenomena may be regarded as a variety of metastasis. As instances, we may mention the alternation of glycosuria and eczema, deafness and vertigo—both of which have been observed by Dr. James Taylor—eczema and asthma. Dr. Denne, of West Kensington, has told me of a woman who is a great sufferer from dyspepsia. A short time ago she developed eczema upon the back of the neck. Since then the dyspepsia has much improved, and each shows a tendency to alternate in intensity with the other. I have recently had under observation a patient of Dr. Chittenden's, who has twice had a severe attack of urticaria, which on each occasion has terminated in a copious

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