TWO TYPES OF DISEASE OF THE SPINAL CORD IN ADULTS.

CLINICAL LECTURE DELIVERED AT THE NEW YORK POLYCLINIC.

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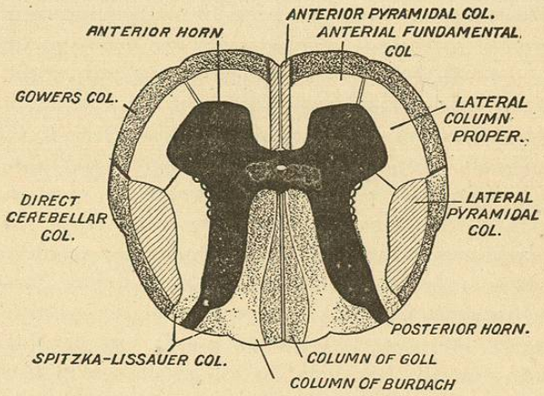
GENTLEMEN,—I have several cases here to illustrate two of the commoner forms of disease of the spinal cord, but before going into the details of them I want to say a few words to you about what we know nowadays of the pathological anatomy of this organ. I presume that most of you are thoroughly acquainted with the elementary matters, and upon them therefore I shall not dwell.

You know that the gray matter of the cord is in the form of horns, so-called *cornua*, anterior and posterior, and that these horns are almost surrounded by gray matter; that the anterior of these is more club-shaped and does not come up entirely to the periphery, whilst the posterior one tapers to a point which reaches the periphery. Our knowledge of the different strands of the white matter has been derived partly from embryological observations, partly from pathological ones, and very little indeed from physiological experimentation. The different strands of the spinal cord in the embryo take on their covering of myelin at various periods of intra-uterine life, so that it is very easy with the naked eye, or at any rate with the microscope, to distinguish those strands which have the myelinic covering from those which have not, because of the great difference in the refraction. In this way Flechsig was able to map out a number of columns, and he published his results some eighteen years ago. I will map them out for you on this blackboard. Dividing the old posterior column into two distinct strands, the one nearest the posterior median fissure is known as the column of Goll, while that which adjoins the posterior gray horn is the column of Burdach. At the side and on each side of the anterior median fissure are found the anterior and lateral pyramids, or, as they are sometimes called, the anterior and lateral pyramidal

columns. On the extreme edge of the posterior lateral column is marked out a little narrow rim of fibres which Flechsig alleged to be in direct connection with the cerebellum, and to which he gave the name of the direct cerebellar column. What was left over of the lateral tract he called the anterior fundamental column. Some of these columns which he outlined had been outlined before, with the exception of the anterior fundamental column, which he never claimed to be one distinct strand, but rather held to be composed of a number of separate strands. Every one of these observations of Flechsig's has been confirmed by pathologists and embryologists in the seventeen or eighteen years that have since elapsed. But we have gone much further than Flechsig went. Part of the column of Burdach has been separated into a column abutting immediately upon the posterior root

FIG. 1.



and described simultaneously by Spitzka of this city and Lissauer of Berlin, to which I have given the name of the Spitzka-Lissauer column. Then it has been demonstrated that there is a distinct column running around from the anterior edge of the posterior cornu to the anterior median fissure, embracing the direct cerebellar column, but elsewhere hugging the periphery, its boundaries being somewhat uncertain, and this has been shown to take on a covering of myelin at a distinct period of fœtal life, and to degenerate separately. It has been named after the gentleman who first called attention to it, Gowers, of London. I have no doubt that this seems to you to be quite a maze, but it is all in reality very distinct and very simple. I will draw a continuous line around all the columns which are sensory,—*i.e.* the columns of Goll,

Burdach, Spitzka-Lissauer, and Gowers. Then I will draw a dotted line around the columns which are motor,—*i.e.*, the lateral and anterior pyramidal, and probably the fundamental lateral column of Flechsig. We know the function of some of these columns, while of others we cannot say as much, and yet we can make a pretty accurate diagnosis and localization of spinal disease; indeed, when you consider that the spinal cord is so very small, not much larger than the piece of chalk which I hold in my hand, it should be a matter of surprise and congratulation that medical science is so exact as to tell us that a disease is located in the centre, or in the periphery, or in some part of the individual columns, as we can do in a large proportion of cases, and as I propose to show you clinically to some extent to-day.

Locomotor ataxia is a disease starting in the central portion of the cord as a subacute inflammation in the connective tissue, and thence extending by means of secondary degeneration through all or nearly all the sensory columns,—*i.e.*, the columns of Goll, Burdach, Spitzka-Lissauer, and Gowers, and the direct cerebellar. We do not know the functions of these sensory columns sufficiently well to say what individual symptoms are due to implication of this column or that, but we can tell you certainly that locomotor ataxia means, in pathological terms, a subacute myelitis starting in the central portion of the cord and thence spreading by secondary degeneration through the sensory strands. This is a man forty-nine years of age. He had a chancre twenty-three years ago. At the outset you should take notice that he had a syphilitic history, because most of these forms of subacute myelitis with secondary degeneration of the sensory tracts, to which we give the name of locomotor ataxia, are syphilitic sequelæ. They do not especially belong to the tertiary state, or to the secondary, or to any stage, because they can follow the syphilitic infection at any time. They are not syphilis, and are not necessarily to be cured as syphilis, but they are in the vast majority of cases true syphilitic sequelæ; and therein lies the importance of the fact that this man had a chancre twenty-three years ago. Five years ago he noticed a numbness in the feet. Two years ago he noticed a girdle or cincture feeling extending two-thirds around his body at the level of the mid-dorsal vertebræ. He has had headaches, insomnia, and boring pains in the legs. The latter came on one year ago, and the sensation was, as he says, as if something were boring into him. He has had incontinence of urine for the last two and a half years. His bowels have been much constipated. The facts that are relevant to the diagnosis are that he has had boring pains in the legs, and obstinate insomnia, and that the

headaches come on generally in the morning and leave him toward the latter part of the day, having a periodicity which is almost as regular as that of malaria. This peculiar headache with a quasi-periodicity, and accompanied by insomnia, is very significant, because every headache, contrary to what you might think if you had had no clinical experience, is not accompanied by insomnia. The headache of Bright's disease is not so attended. The headache of meningitis not only does not produce insomnia, but causes somnolence, and though the individual suffering from either of these two kinds of headache may complain acutely during the daytime, he will generally sleep well at night. So that the fact that the man has a headache which is accompanied by insomnia separates this headache from other forms of headache, and when it is also quasi-periodical let me assure you that you are perfectly safe in assuming in the vast majority of cases that it is due to intracranial syphilis. This headache, therefore, means this: that, in addition to the syphilitic sequelæ affecting the spinal cord and giving rise to the symptoms of locomotor ataxia, he has an intracranial syphilis,—*i.e.*, he has both intracranial and spinal syphilis, which makes a great difference in the therapeutics and in the prognosis, because it is possible for you to relieve an intracranial syphilis, and in relieving this you may possibly check the syphilitic extension in the cord. Bear in mind, however, this possibility is not a certainty, for the damage done to the cord may be irreparable, although you may be able to check the cause of it. In addition to these symptoms he has an ataxia which I want you to study. Let us draw this chalk-line upon the floor and see him attempt to walk along it, putting one foot in front of the other. You see that he fails to do so, that he totters, that his feet make irregular and spasmodic movements, that he cannot bring a foot to the point that he aims at. You might think that this disorder of gait was due to the fact that he had some muscular weakness. When I put his leg out straight, however, and direct him to keep it straight, you see that I am unable to overcome the muscular resistance which he offers in that position, for his leg is like a bar of iron, and if I were to attempt to use my full strength I might actually fracture his patella from muscular action. Now I will give him the aid of the sense of touch, or tact, as it is technically called. I will support his little finger upon my little finger, not lending him enough support to be of any muscular aid to him, but simply putting in contact the surface of his finger with the surface of mine. You see that he now walks better, although he does not lean upon my finger at all. He has got the aid of the sense of touch, and through that he is

getting the benefit of my steady muscles. Let us watch him walk with his eyes shut. You see how immediately his gait becomes impaired, the weak movements of the legs become more marked, and he is actually in danger of falling. Let us observe his walk again when he has his eyes open. You perceive that he is unable to co-ordinate his legs, notwithstanding the strength of the individual muscles which I have shown you. This inability to co-ordinate unless with the aid of something else than the motor and sensory apparatus of the legs, constitutes the characteristic ataxia of that disease which we know especially as locomotor ataxia, and is distinguished from other ataxias or kinds of incoördination arising from many different causes. Ataxia may result from anything that interferes with the proper co-ordination or harmonious action of the muscles. If the muscles themselves are impaired, if the different senses which are necessary to the muscular act are impaired, if the joints which are necessary to the movement of the segments of a limb are impaired, if the spinal cord is diseased in other portions than the site which I have indicated in locomotor ataxia, if there is a lesion of the higher brain-centres, we may have what is in one sense an ataxia, or a disorder or incoördination of movement, but the incoördination of locomotor ataxia which you see in this man is as beautifully shown as I have ever seen it in any case in my life. This characteristic ataxia is one of the most valuable of diagnostic factors. By means of it I will make a diagnosis of locomotor ataxia, even if every other symptom in the case is eliminated. But when in addition you have the history of syphilis, certain severe pains, and atrophy of the optic nerve, which you will have to take my word for his having, you may be perfectly sure of your diagnosis of locomotor ataxia. If, furthermore, you have the peculiar pupil which I demonstrated to you in this man, the so-called Argyll-Robertson pupil, you add to the evidence. This peculiar pupil, when typical, is contracted, does not respond to light, but does respond in movements of accommodation,—*i.e.*, it is small, does not dilate or contract with the light or removal of light, but does dilate and contract sluggishly when looking successively at near or distant objects. With these symptoms in your possession—the ataxia, certain severe pains, atrophy of the optic nerve, and the Argyll-Robertson pupil—you can put everything else out of the case as immaterial. The incontinence of urine, the impairment of sensation around the body (the so-called girdle or cincture feeling), the aching pain, are symptoms which are common to other diseases of the spinal cord and to certain diseases of the peripheral nerves, but their presence would not help

the diagnosis of locomotor ataxia any more than their absence would vitiate it.

Here is another individual, twenty-two years of age. His history was taken about a year ago. Some six months previous to this he had been struck on the back in the lower lumbar region by a gas-generator weighing several hundred pounds. Two ribs were broken. He was told at the hospital that his back was broken, and he was in the hospital seven weeks. He could not walk or sit up when he came out of the hospital. He tells us that he had a large bed-sore on the back. He used the catheter three months, and his bowels moved only by the constant use of cathartics. He lost all sensation in defecation. His sexual power is almost extinct. The pain sense is gone in the perineal region, as well as the tactile sense. He had the so-called girdle-feeling, as of a band encircling the lower part of his abdomen. It was evident to us when he came here a year ago, as it is now, that he had had a severe fracture of the lumbar vertebræ. We tried electrical treatment and internal medication, but he did not improve. We advised him to have the fractured vertebræ removed, but he was very much averse to having this done. Finally he did have it done by a very competent surgeon outside of this clinic, and he comes here to-day, several months after the operation, somewhat improved. It is probable that this man's trauma implicated the spinal cord in the lower dorsal region, and that the cauda equina was also injured. I want to call your attention to a matter of medico-legal interest in this case. The absurd dictum has been laid down by some of the earlier writers upon surgery that a man receiving a blow that fractures his vertebræ will not have his cord injured, and, conversely, when the blow does not fracture the vertebræ, the cord will be injured. This sounds very well in physics, but it is as arrant nonsense as ever was talked in medicine. This man, for instance, received a blow with a heavy gas-generator sufficient to injure both the vertebræ and the cord, as the symptoms indubitably indicate, for we could see that the vertebræ were distorted; he tells us that a portion of two was removed, and his symptoms could not have arisen except from injury of the cord and the cauda equina. The central portion of the cord, which I mark out here, was the portion that was injured. You will perceive that this central portion of the cord which I have indicated is in the gray matter, and is anterior to that portion of the posterior columns in which I have mapped out the subacute myelitis of locomotor ataxia. I make my diagnosis of this central portion of the cord having been injured by means of the paralysis of the bladder and rectum, the loss of sensation in the penis and peri-

neum, the bed-sore, the motor and sensory paralysis of the lower limbs. Some central point must have been affected that would implicate all these nerves, and the only central point is just that portion of the central gray matter which I have mapped out. We might suppose, of course, that myelitis running around the periphery of the cord and catching all these different nerves on their way out to the anterior and posterior roots would give us the same symptoms; but a peripheral myelitis of this kind could not result from a trauma, because it is not conceivable that a trauma would implicate only the periphery of a small body like the spinal cord, and leave untouched the central portions. This man has therefore had a central myelitis which may have extended transversely across the whole cord. We must now ascertain the result of the surgical operation. The incontinence of urine, he tells us, has not been affected. The bowels are no more under his control than they were. The sexual sense is just as much in abeyance as it ever was. The girdle-feeling, however, is gone. He can now walk better. The sensory impairment is otherwise just the same as before the operation. He has the same area of anæsthesia in the sacral region, and the same absence of sensation in defecation. You see that when I tap upon his knee the knee-jerk, or the so-called tendon reflex, is very much exaggerated. You see that when I test his flexor muscles they are weak, but his extensor muscles are fairly strong. You can also see the weakness of his flexor muscles by his manner of walking with a loose flop of the legs, because there is no steady, strong action of the flexor muscles acting antagonistically to the fairly strong extensor ones. This peculiar walk is often mistaken for the gait of locomotor ataxia, but you can easily make the distinction. This case of traumatic myelitis has therefore been somewhat improved by the surgical operation which was done three months ago. I think he will improve still more, but whether he will entirely recover is a matter of grave doubt, because he waited eighteen months after he had the accident before the operation was performed, and during that time a chronic myelitis was extending in the cord, setting up secondary degeneration and inducing irreparable changes of the connective tissues and of the membranes, and probably also extending along the peripheral nerves, so that the only thing the operation could do was to remove the pressure of these fractured vertebræ, and the removal of that pressure could not re-convert these organically changed structures into healthy ones, which might have been done if he had had the operation performed immediately after the accident.

In these two cases you have before you, gentlemen, two types of the

most common diseases of the spinal cord,—viz., locomotor ataxia and central myelitis. In reality, both of them are central myelitis, but locomotor ataxia was diagnosed clinically long before we knew much about the pathology of the cord, and central myelitis came under observation at a much later period, so that time has sanctioned the use of these terms, which, moreover, it is desirable to retain because of the differences in the clinical symptoms. One is a myelitis of very subacute onset and very chronic extension. The other is a myelitis that is usually, even when idiopathic, of acute origin and rapid extension within a few hours or a few days. The locomotor ataxia is a myelitis that is prone to extend throughout the cord, and even travel up into the upper and large portion of the cord which we know as the *medulla oblongata* or oblong spinal cord, and it is also, you should bear in mind, in the vast majority of cases, one of the syphilitic sequelæ. The central myelitis, on the other hand, is much more prone to remain localized in the portion of the cord in which it starts, and is caused either by trauma or by causes whose exact nature we do not know, but not by syphilis, tuberculosis, or other diathetic affections. Locomotor ataxia is much more relievable than is generally supposed; and in some cases, where the syphilitic infection has been very recent and implicated the whole cerebro-spinal tract, there is reason to believe that relative cures have been effected: at least cases have been put on record in which all the symptoms have disappeared. But the disappearance of all the symptoms, unfortunately, does not mean that the pathological alterations of the cord have been repaired, for in a notable case recorded by Erb an autopsy made fifteen years after the disappearance of the symptoms showed the characteristic spinal lesion. Nevertheless, it is practically of little account to a man to know what the pathological alterations may be in his cord, provided that he is able to do his work in life as well and as comfortably as he ever did; and that this result has been attained in several cases where the syphilitic infection has been recent is undoubted, and I have also seen it in some few cases in which the syphilitic infection had dated back some time.

I regret very much, gentlemen, that the time at my disposal will not permit me to go into the treatment of these cases, or to show you other types of spinal disease.