THE GULSTONIAN LECTURES, ON SPASM IN CHRONIC NERVE-DISEASE.

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LECTURE II.

In my first lecture, I considered the part played by chronic diseases, which injure the intracranial portion of the pyramidal tract, in the production of spasm. It was pointed out that the results are similar, whether the lesion is situated in the motor area of the cortex, in the central ganglia, in the crus, pons, or medulla, provided that the function of the fibres of the pyramidal tract be interrupted.

A point which is of great importance, and which will be again referred to when we come to a consideration of functional spasm, is this, that congenital defects of portions of the central nervous system are as capable as destructive diseases of producing paralysis of limbs, accompanied by spasm.

Case of Congenital Spastic Hemiplegia.—In the *Medico-Chirurgical Transactions*, vol. lxvi, 1883, I published "a case of Asymmetry of the Brain," and discussed the bearing of the anatomical peculiarities of the specimen on the question of the connection between the optic nerves and certain definite areas of the cerebral cortex. But the case is also a good example of the disease called congenital spastic hemiplegia, and for this reason I refer to it here. E. A. was admitted into St. Thomas's Hospital, under the care of Mr. Mason, owing to injuries received by the fall of a house; and she died of the results of the accident. I never saw her until I performed the *post mortem* examination upon her; and no account was obtained during her lifetime of the abnormality in her right arm and leg, nor of the state of her mind and special senses. After death, her father informed me that his daughter had always been healthy up to the time of the accident, which proved fatal in a few weeks; that she had never suffered from fits or other nervous affections; that from her very birth she had had a small and stiff right arm; and that she had always been left-handed and a little lame. He had never noticed any mental deficiency; and she had never complained of her sight, hearing, or other senses. No peculiarity in her eyes, speech, or aspect was noticed during her stay in the hospital; but the sister of the ward said that she struck her as being intellectually dull, even for a person in her position in life.

At the *post mortem* examination, both the right arm and leg were seen to be smaller than their fellows, though they were otherwise normally formed; and the muscles examined presented the rich brown colour of health. The right wrist measured $4\frac{1}{2}$ inches, the left $5\frac{3}{8}$ inches; the right forearm $6\frac{1}{2}$ inches, the left 8 inches. The circumference of the left leg, round the calf, exceeded that of the right by one inch. The heart, lungs, and other viscera were healthy, with the exception of the kidneys and the bladder, which were in a state of acute inflammation. The skull and the membranes of the brain were normal, as were the intracranial blood-vessels, nor was there any marked inequality of the latter on the two sides.

I need not enter into the minute details which I have given in the paper referred to, but I may simply state that both the history of the patient and the anatomical peculiarities of the brain, showed that it was a case of malformation of one portion of the left hemisphere, and arrest of development of the remainder. The most striking characteristics of the specimen which is here shown (Figs. 14, 15, 16, 17), were: 1, the general arrest of development of the left hemisphere, including the motor convolutions; 2, the small size of the corresponding crus cerebri and anterior pyramid; 3, the absence of the angular gyrus, and superior temporosphenoidal convolutions, together with the fusion of some of the other convolutions of the left temporosphenoidal lobe; 4, the small size of the optic tract, optic thalamus, and corpora geniculata on the same side. The spinal cord was subsequently examined, and, throughout its whole length, it was seen that the right lateral column was smaller than the left, and the left column of Türcck smaller than the right. (Fig. 18.)

These differences were most marked in the upper regions of the cord, and became less and less so towards the lumbar enlargement. The

microscope showed that in those parts where reduction in size was evident, even to the naked eye, the nerve-fibres were fewer in number, and the tissues a little denser than in health; and the general impression left upon the observer's mind was that there was an absence of nerve-fibres, and consequent undue prominence of the connective tissue.

In this case, there was clearly congenital absence of part of the motor convolutions, and of the corresponding pyramidal tract; and, clinically, paralysis with spasm was observed. Such a combination seems to prove that the mere absence of the controlling impulses which in healthy subjects traverse the pyramidal tract, is capable of giving rise to a spastic condition of the limbs.

Congenital defects similar to that which has just been described may be bilateral, and produce a condition about which a good deal has been written, and which is termed "infantile spasmodic paralysis," or "spastic paraplegia of infancy." Dr. Hadden, writing on this disease in *Brain*, vol. vi, 1884, says, "the lower extremities are more or less flexed at the hip- and knee-joints, the thighs are rigid and adducted, the knees in contact, the legs inclined outwards, and the heels often drawn up from the ground by the contraction of the gastrocnemii." Figs. 19 and 20 represent a well marked, but not ex-

FIG. 20.—From a photograph of a slight case of congenital spastic paralysis.

treme, case of the kind, which was under Mr. Clutton's care in St. Thomas's Hospital. The child was an idiot, as many of them are. Dr. Ross has proved, by *post mortem* evidence, that defects in the development of the brain can produce this condition. In *Brain*, vol. i, p. 477, he records a case of the disease in which he found at the *post mortem* examination a congenital defect in the motor convolutions on both sides, and absence of the large pyramidal cells normally existing in them. He found the same condition of the spinal cord as was present in my case of congenital spastic hemiplegia, namely, absence of a certain amount of the lateral columns, but no other abnormality.

Whether all cases of congenital paralysis and spasm of the limbs be due to arrest of development of portions of the brain is at present uncertain, but it is not at all improbable that intra-uterine disease of parts already formed may be the explanation of some.

Contracture, or fixed spasm, is a comparatively frequent result of cerebral lesions which interrupt permanently the voluntary impulses proceeding from the cortex. But, in addition, mobile spasms have been observed in great variety, either after hemiplegia or independently

¹ The numbered figures which are referred to in the text were shown when the lectures were delivered, but only a certain number are reproduced here.

of it. Sometimes they attend voluntary movements only, sometimes they are involuntary and continuous; but, in all of them, the retention of a large amount of voluntary control over the limbs affected is a striking feature.

In addition to the mobile spasm, there is often a certain degree of fixed spasm, though a marked degree of the latter would be incompatible with the development of the former. Continuous movements of the hands and toes have been termed *athetosis*. The following is a case of the kind which I reported in *Brain* for April, 1885. It is a good example of this condition, though rare because of the number of muscles affected, and because the movements appeared after many years on the opposite side of the body.

G., aged 24, came under my care as an out-patient at St. Thomas's Hospital on March 26th, 1884. Until the patient was three years old, he was quite healthy: he was then suddenly attacked with loss of power all down the left side, accompanied by repeated and prolonged convulsions. As far as he knew, the movements of the limbs and face on the left side gradually followed the loss of power, and had gone on increasing ever since; at any rate, they had done so as long as he could remember. When between four and five years of age, he was taken to Great Ormond Street Hospital, where his parents were told that the movements were so slight, that he would get over them. About three years ago, weakness and movements came on in the right leg, and he went to Guy's Hospital, where he was told that nothing could be done for him. He then consulted a homoeopathic chemist, who gave him "strengthening medicine," and his right leg became strong again, and the movements in it ceased in three months. A month ago, the weakness and movements recommenced in the right leg, and they gradually increased. The arm was said to be free from them. The patient had never had any other severe illness, had not suffered from headaches, and had done his work well as a carpenter until the last six months. During this time, the movements of the left hand had been so bad, that he had been unable to hold the nails with accuracy, though he could hammer well enough with his right arm. He said he had never had any defect of sight or hearing.

On rough examination, vision seemed normal, and he heard the tick of a watch a long way off. There were movements of the greater part of the muscles on the left side of the body, and they ceased during sleep; there was no anaesthesia. The tongue moved a little irregularly, as it did in chorea; speech was drawling. The muscles, both of the left and of the right side of the face, contracted too strongly when the patient attempted to speak, so that a kind of grimace resulted, in which the lines at both angles of the mouth and the naso-labial furrows were very deeply marked. There were spasmodic, more or less regular, movements of the neck to the left, and the shoulder was rhythmically raised, and jerked slightly forwards and backwards. One could see the serrations of the left serratus magnus contracting rhythmically. The abdominal muscles were harder on the left side than on the right, but there were no evident movements in them. There were slight spasmodic movements in the left arm, but they were only slight. The hand, on the contrary, was markedly affected. There was constantly more or less rhythmical flexion and extension of the fingers, mainly at the metacarpo-phalangeal joints, with irregular flexion and extension of the phalangeal joints, and occasional separation of the fingers. In the left leg there were similar slight movements of the muscles, with marked movements of the toes, and most marked of the great toe. They were tremulous flexions and extensions, but the flexor longus pollicis was constantly contracted, so that the great toe was always riding over the others, and its proximal phalanx was usually at right angles to the metatarsal bone. In the right foot, the movements were similar, but not so marked. In the right hand only the little finger moved, and that so slightly, that the patient had not noticed it. The grasp of the right hand, though stronger than that of the left, appeared to be deficient in power; the patient said, however, that he had never noticed that it was weak. There was neither atrophy nor hypertrophy of the muscles of the limbs. The patellar reflex was unnaturally brisk on both sides, but there was no ankle-clonus. The patient seemed very intelligent, and said that his memory and mental power were very good. The fundus of the eyes and fields of vision were normal. The urine contained no albumen.

The next case is one of fixed, combined with mobile spasm, and also differs from that which I have just described in this, that there was no history of hemiplegia or monoplegia, preceding the onset of the muscular contraction.

Case of Mobile and Persistent Muscular Spasm of the Upper Extremity.—E. F., aged 11, came to St. Thomas's Hospital as an out-patient, under me, in June, 1884. She was a very well made, intelligent, lively child, and in these respects was said to resemble the rest of her family. When she was 9 months old, the mother noticed that

she held her left arm behind her back, and did not use it properly. Ever since, the arm had been becoming gradually more distorted and useless. On examination, it was found that the left arm was not undersized, or atrophied, but that it was the seat of extreme distortion, which rendered it perfectly useless. The forearm was excessively pronated, so that the palm of the hand looked outwards, and the wrist was flexed; the metacarpo-phalangeal joints were extended, the proximal phalangeal joints hyperextended, and the peripheral flexed. The elbow-joint was flexed, and looked unshapely. The metacarpo-phalangeal joint of the thumb was flexed, and the phalangeal joint hyperextended; and the thumb, as a whole, was turned inwards towards the palm. All the parts described were rigidly fixed in these positions, apparently by the spasmodic action of muscles. But the spasm could, with difficulty, be overcome, and then the limb assumed quite a new appearance. Thus, if pronation were rectified and the elbow bent, the wrist at once became extended, all the fingers flexed, and the thumb was turned into the palm of the hand beneath them. This state of affairs was only retained by keeping the elbow-joint flexed, and forcibly overcoming the tendency to excessive pronation. As soon as the latter was allowed to assert itself, the whole limb at once returned to the original position. Sensation was unaffected. Dr. Kilner, who tested the electric condition of the affected parts, reported that the muscles reacted normally to the induced current, and presented very slight reaction of degeneration when the constant current was used.

On giving chloroform, the distortion almost entirely disappeared, the muscles were flaccid, and the tissues of the elbow-joint appeared very lax. The child had attended the Orthopaedic Hospital for one year without deriving any benefit, but electricity had not been tried. Dr. Kilner was good enough to undertake treatment by this means; but, though he continued it for weeks, the condition underwent no improvement.

FIG. 21A.—Case of spastic paralysis of the left arm and hand, showing the position assumed when the wrist was extended.

Later in the year, on August 24th, 1885, I took her to have the abnormality photographed, and I then found that certain alterations had occurred in the condition of the hand and arm. The most marked change was that they were never still; in fact, the case might

FIG. 21B.—Showing position assumed when the wrist was flexed.

then have been called one of *athetosis* of the left upper extremity. The arm, when left alone, usually occupied a position midway between the perpendicular line of the body and a horizontal line drawn through the left shoulder-joint, and the latter was extended, the forearm pronated, the wrist flexed, and the fingers flexed at the peripheral phalangeal joint, and hyperextended at the proximal. Such a degree of hyperextension now existed in the metacarpo-phalangeal

joint of the thumb, that partial luxation had been produced. The child generally liked to hold the hand across the front of the body, with the fingers a little flexed. Irregular clonic jerks affected the whole arm from the shoulder, and the fingers were in more or less constant movement when left to themselves. There were occasional slight jerks of the left angle of the mouth, but the leg appeared free from any abnormality. The child asserted that she had always had these movements, though I certainly did not notice them when I first saw her.

Figs. 21, A and B, represent the condition of things in January, 1886.

Mobile spasms do not always assume the characters which are described under the term "athetosis." Movements resembling, more or less, those of chorea, disseminated sclerosis, ataxy, and paralysis agitans, likewise occur.

On June 11th, 1885, a woman, aged 60, came to my out-patient room, complaining of loss of power all down her left side; her face, arm, and leg were all weak, and there was a considerable degree of hemianæsthesia. This condition had slowly developed in the course of three months. Though she had had rheumatic fever twice, there was no apparent disease of her heart or other viscera, but her arteries were somewhat hard. By November, moderate hemiplegic rigidity had developed, and, in addition, continuous movements exactly resembling those of paralysis agitans.

It is unnecessary to describe the various forms of mobile spasm which are seen at the bedside. Dr. Gowers has given an account of a considerable number, in a paper which is contained in the fifty-ninth volume of the *Transactions of the Royal Medical and Chirurgical Society*; and Charcot and others have also recorded cases. What concerns us more on the present occasion, is the question of their pathology. Can they be attributed to any lesion constant either in its nature or in its position? The answer to this must be in the negative; but we have to confess at the same time that our knowledge of this class of spasm is very deficient. Gowers, in the paper already referred to, records a case of "posthemiplegic inco-ordination," in which he found, at the *post mortem* examination, one lesion only, and that was a cicatricial induration confined to the optic thalamus, and unaccompanied by any pathological changes in the spinal cord. Weir Mitchell found the seat of the lesion in two cases in the corpora striata. In the *Revue de Médecine* for May, 1883, Emile Demange records the *post mortem* examination in nearly a dozen cases of posthemiplegic mobile spasm; and among these is a case of hemiathetosis, in which there was extensive softening of the convolutions, but no disease of the central ganglia. In most of the cases, the lesion or lesions were in the central ganglia, sometimes involving the internal capsule, and sometimes not. Hence it seems to me that we must agree with Demange, when he says that these movements may occur from disease of any part of the motor tract in the brain; and he adds that the variety of movement does not depend upon the seat of the lesion, but rather upon the condition of the motor tract below the lesion.

All the varieties of mobile spasm seem to result from a mixture, in varying proportions, of paralysis, spasm, and irritation; and their development depends upon lesions which interfere with the perfect functions of the motor centres and fibres, but which do not completely interrupt them. That such a condition of incomplete destruction is competent to give rise to movements of the kind under consideration, is well shown by a case which I published in the *Lancet*, in a paper on Cerebral Localisation already referred to. The patient was a woman, aged 46, who was admitted into St. Thomas's Hospital on July 20th, 1878, and died on July 21st, 1879. She was suffering from chronic renal disease, and on January 18th, 1879, she complained of being unable to keep her left arm still. The movements both of the arm and leg soon became violent and disorderly. When asked to put her left hand to her face, she did so hesitatingly, and with a series of jerks which had no constant rhythm or direction. The same occurred on voluntary movement of the leg, while the right arm and leg were moved slowly and naturally. The jerking movements gradually subsided until, on January 30th, the leg was freely and normally moved, while the left arm was almost completely paralysed; and, although it recovered some degree of power, it remained markedly paralysed to the end of her life. After death, the middle part of the ascending parietal convolution on the right was softened, shrunken, and of an orange colour. It is clear, in this instance, that the involuntary movements of the limb occurred at the time when the cortical grey matter was in the early stages of softening; and that they ceased when paralysis resulted from its complete disorganisation.

In concluding the consideration of spasm in connection with diseases of the intracranial portions of the voluntary motor path, it is necessary to refer to a statement which I made in my first lecture; namely,

that "our present knowledge of anatomy, physiology, and pathology, does not justify us in concluding that there is any efferent motor connection between the brain and the spinal cord, except the pyramidal tract, direct and crossed, disease of which gives rise to chronic muscular spasm. Evidence in support of this view will appear in subsequent portions of these lectures."

In saying this, I wished to exclude the cerebellum and the central ganglia from consideration, since any rigidity or spasmodic contraction of muscles which arises in connection with diseases of the areas they occupy seems to depend upon involvement of the pyramidal tract, which passes close by them. Evidence has already been supplied in support of this view, as far as the cerebellum is concerned. Let me now record a case which shows that destruction of the central ganglia produces no spasm, so long as the neighbouring internal capsule is intact.

C. B., aged 39, had an attack of right hemiplegia ten years before death, and another somewhat later. He died of carcinoma of the cæcum, having long recovered completely from all paralytic symptoms. At the *post mortem* examination, I found that the hemispheres were free from disease. On exposing the ventricles, it was at once seen that the left caudate nucleus was very much shrunken, and of a yellowish hue. Both optic thalami and the right nucleus caudatus looked healthy. The left crus cerebri was small and discoloured along its inner third, and the left side of the pons Varolii was evidently flattened. Nothing abnormal was noticed in the crus cerebelli on either side, and the pyramids in the medulla oblongata were equal and natural. No descending degeneration was to be discovered in the spinal cord with the naked eye.

The brain was then set aside to harden in alcohol, before any further examination was made. A series of horizontal sections through the hemispheres and central ganglia showed that there was hardly anything left of the nucleus caudatus, or of the nucleus lenticularis on the left side, but there were no evident changes in either division of the internal capsule. The optic thalamus was a little smaller than its fellow on the opposite side, while all the central ganglia on the right were healthy. (Fig. 22). A vertical section through the crus cerebri showed very marked atrophy of the left as compared with the right. A similar diminution in size was observable in the left half of the pons (Fig. 23); but the pyramids, and all other divisions of the medulla oblongata, were healthy and symmetrical.

This case shows that disease so severe as to result in almost total obliteration of the corpus striatum, may produce hemiplegia, which passes off without leaving rigidity, paralysis, or any other symptom behind. And, secondly, it proves, as far as any single case can prove, that the efferent fibres of the corpus striatum do not pass down further than the pons Varolii or medulla oblongata.

The continuous degeneration of the lateral column through the whole length of the spinal cord in cerebral lesions shows that the fibres are not interrupted by entering ganglionic centres, but pass straight to their several destinations in the anterior cornua. It will be readily understood that, if disease destroy them at any level in the spinal cord, degeneration will occur in them below, and give rise to symptoms of hyperphysiological activity of the spinal centres with which they are connected, similar to those which occur when the disease is intracranial. In the latter case, all the spinal centres which are under voluntary control are affected; in the former, only a certain number, more or less numerous according as the lesion is situated high up or low down in the cord. There is this difference, too, that, in cerebral disease, the rule is to find the contracture unilateral, in lesions of the cord bilateral. But this is merely because the pyramidal tracts in that part of the brain where they are most liable to be interrupted by disease, lie at some distance from each other; whereas, in the spinal cord they are so close together as usually to be involved in the same pathological changes. The symptoms, however, of disease of the lateral columns of the cord are the same, whether it follow cerebral or spinal lesions. The affections of these columns are either primary or secondary, and the symptoms differ slightly accordingly. Thus, if a primary chronic inflammation attack them, as in primary lateral sclerosis, or disseminated sclerosis, paresis of the corresponding muscles is an early symptom; and tremors of the limbs when moved, increased tendon-reflexes, and clonus soon follow. In addition to these, transitory involuntary contractions, and longer, though still intermitting, spasms occur, and finally culminate in persistent rigidity. When the latter has fairly set in, the muscles can only be loosed from their slavery by a process which condemns them to perpetual inaction. Their masters, the cells in the anterior cornua, may be attacked, and die; but the muscles perish with them, and the tendon-reflexes and other signs of their activity disappear for ever. But the lateral columns are more often affected secondarily than primarily, and then

the symptoms which culminate in spasm follow those which characterise the original disease. Thus we often see cases of myelitis run their course to destruction of a portion of the spinal cord, and rigid contracture of the limbs then supervenes.

Such disease occurs more frequently in parts below the cervical region than in those above it, and consequently the legs are more often the seat of rigidity than the arms.

But matters are not always so simple as this, for a lesion of the lateral columns may be combined with that of some other tract of fibres; and a combination of symptoms then presents itself, some being attributable to disease of one tract, and some to disease of the other. If we are to draw correct conclusions with regard to the localisation of diseases of the spinal cord, and their progress, we must never lose sight of the phenomena which characterise the healthy action of the nervous system. Physiology is the only safe guide for the diagnosis of the positions occupied by disease. Again, although pressure upon the pyramidal fibres interrupts their functional activity, the symptoms are very different according to the manner in which the pressure is applied, whether from without or from within the cord. As time will not permit me to enter into detail upon all those diseases of the spinal cord which give rise to spasm, I shall try and illustrate from my own experience the two points to which I have specially referred; namely, first, the difference in the results of pressure applied upon the cord from without and from within; and secondly, the modification which occurs in the symptoms of disease of the lateral columns, when it is combined with disease of other parts of the cord.

The following case is a good instance of pressure on the spinal cord from without, giving rise to interference with the function of the lateral columns, and to extreme contracture of the lower limbs.

Case of Tumour (Myxoma) on a Spinal Nerve, which pressed upon the Cord, and produced Paralysis and Spasmodic Contraction of the Lower Limbs.—A. J., aged 24, blacksmith, was admitted into St. Thomas's Hospital, under the care of Dr. Bristowe, on April 20th, 1876. His family-history was unimportant, and, until his present illness began, he had always enjoyed good health. He had drunk freely both beer and spirits. He was in the habit of lifting heavy weights, and had occasionally "ricked" his back, but had never suffered from any serious injury. In August, 1875, he began to feel a sensation of weakness in the loins, but he had no actual pain. This was followed in November, 1875, by a certain degree of weakness in walking, which slowly increased, so that he became unable to walk at all two months before admission. During this period, he suffered from cramp, with slight loss of power in his left arm, and from occasional attacks of sharp shooting pains and cramp in his legs. He had had no loss of sensation or numbness anywhere.

On admission, he was found to have marked paraplegia, but no loss of sensation. He suffered from cramps, and his legs at times were flexed involuntarily. He had incomplete control over his evacuations. His left hand and arm were not as powerful as his right. All his organs appeared to be healthy, and he had no albumen or pus in his urine. Somewhat later, painful contraction of the adductor muscles of the thighs used to come on and last for hours; and he had slight numbness as high as the umbilicus. When these attacks of spasm supervened in the muscles of the leg, pain and similar cramps were experienced in the left arm. The remainder of his life was very painful. His legs became gradually more and more permanently and rigidly flexed and adducted, and bed-sores developed over the internal aspects of the knees, where they were forced against each other, as well as over the sacrum and trochanters. Sensation, however, rather improved than deteriorated. Finally, incontinence of the evacuations, cystitis, and hectic fever set in; and the patient died on August 17th, four months after admission, and one year from the date of his first symptoms.

Post Mortem Examination.—Most of the organs were found to be perfectly healthy, the urinary tract and spinal cord alone presenting evidences of disease. The pelves of the kidneys and the ureters were inflamed, and the kidneys were large and succulent. On one of the anterior spinal nerves, which originated just at the termination of the cervical enlargement, was a tumour about as large as a medium-sized marble. It was firm, pale in colour, and covered with a perfectly smooth fibrous sheath. The nerve was lost in its substance, but the tumour was free from adhesions with the spinal cord and all the surrounding structures. It was wedged in between the bones forming the spinal canal on the one hand, and the anterior surface of the spinal cord on the other, so that the latter was flattened by it anteriorly and slightly laterally. The tumour was a myxoma.

The motor phenomena which characterised this patient's history were paralysis, attacks of tetanic spasms, and, finally, persistent rigidity

of the legs. Making allowance for the position at which the pressure was applied to the spinal cord, namely, below the origin of the nerves supplying the arms, the occurrence of tetanic spasms in the legs from time to time reminds us forcibly of similar but more extensive spasms which occur when the disease occupies the base of the brain, or the cerebellum; and, I think, gives support to the conclusions which were drawn as to the part played by the cerebellum in their production. A point which should be specially noted in this case is this, that when the pains and tetanic spasms occurred, pain and spasm were likewise felt in parts supplied by the nerve on which the tumour was situated. These attacks of spasm in the legs, which were intermittent, appear then to have been produced by changes occurring in the tumour, which also showed their presence by increased pressure on the nerves for the arm which it involved. The same intermittence was observed in the spasmodic attacks produced by the tumours which occupied the base of the brain, and the same thing occurs when a tumour presses upon the cerebral cortex. We may conclude, therefore, that in all such cases the convulsions are due primarily to alteration in the condition of the tumour, though we cannot say in what the alteration consists.

The case I shall relate next is a good instance of the results of pressure on the cord applied from within by central tumours. The disease is a very rare one, and the specimen I show is so beautiful an example of it, that I have endeavoured to work out the histological changes which it has produced in the cord as minutely as possible, in order to see how far they explain the clinical phenomena observed during life.

C. W., aged 50, waiter, was admitted into St. Thomas's Hospital, under the care of Dr. Stone, on January 24th, 1885, and died on March 18th, 1885. His family-history was unimportant; he had a wife and five children, all strong and well. He had enjoyed the best of health since he was a child, and had never had syphilis. His illness commenced somewhat suddenly, with pain in the back, eighteen months before admission. Partial loss of power and sensation in both his arms and hands soon followed, together with tremulousness on attempting to use them. It was not until three months before coming to the Hospital that his legs became affected. Since then, he had had numbness and pricking sensations, loss of power, and a sensation of coldness in them. On walking, the ground felt soft. His bowels had been irregular, and he had had some difficulty in passing urine; his appetite had been poor; he slept fairly well.

On admission, he was a healthy looking man, and complained of loss of power, numbness, and formication in his legs, together with incontinence of urine. On examination, no angular curvature or marked rigidity of spine was discovered; but some tenderness, without prominence, was found about the lower cervical and upper dorsal vertebrae. The legs were almost completely paralysed and anæsthetic, but there was no wasting of muscles, no rigidity, and no tremors. The tendon-reflexes were much exaggerated, and there was ankle-clonus in both legs. The patient could use his hands and arms fairly well, but they were numb and tremulous, and the grasp was weak. There was no ocular paralysis. Urine was retained, and the bowels confined. Pulse was 84; temperature normal; tongue slightly furred. The urine was of specific gravity 1030; no albumen, faintly acid. The abdomen was normal; hepatic and splenic dulness was normal. The lungs were resonant, and the breathing healthy, nor was there anything abnormal detected on examination of the heart. He continued in much the same condition for some weeks, complaining a good deal of pain in the back high up, and of occasional pains in the legs. The muscular power and sensation underwent but slight variations in the upper and lower extremities, though he complained sometimes of twitchings. The temperature was generally normal, but now and then somewhat raised. Incontinence of urine set in about February 5th, and it became alkaline and phosphatic, and contained albumen.

On February 24th, it was noted that there was tenderness in the lower dorsal and lumbar regions of the spine, and that complete anæsthesia existed below the level of the middle of the chest.

On March 12th, the legs were completely powerless, and the right leg œdematous. The patellar and plantar reflexes were very brisk; ankle-clonus was marked, but there was no knee-clonus. Sensation of touch and of pain, in lower extremities, was abolished; sensation of heat was retained. There was no control over the bladder or rectum. In the centre of the sacral region was a large horse-shoe shaped bed-sore, with a red granulating surface, and a smaller one was forming over the right hip. The urine was alkaline, and contained pus and mucus.

On March 14th, the patient took his tea at 5 P.M., and seemed to be in his usual condition. Soon after 6 P.M., when the sister came to him, he was found to be unconscious. At 6 P.M., his eyes were shut, and he was breathing heavily, and when asked how he was, he

said he was "as usual." The pupils acted to light. His tongue was protruded to the left, his left cheek was flaccid, and his left arm powerless. His head and eyes turned to the right. The pulse and heart-sounds were very feeble, but there was no murmur. He altered but little after this, and died on March 18th.

The following is an account of the *post mortem* examination, which I made twelve hours after death. Body emaciated. Over the sacral region in the middle line, a large superficial bed-sore was seen, and there were several smaller ones in the neighbourhood. The peritoneum, pericardium, and right pleura, were healthy; the left pleura was everywhere firmly adherent to the thoracic wall. The lungs were intensely congested and oedematous, otherwise healthy. The heart appeared normal in size; the valves on the right side were natural, those on the left a little thickened, but competent. Along the line of attachment of the mitral valve to the wall of the heart, on its auricular aspect, was a series of papilliform elevations, such as are often found in this position in cases of endocarditis. There appeared to be little or no coagulum upon their surface. The spleen was large, weighing 9½ ounces, and contained several large pyramidal infarcts, which were still soft, and had a very clearly defined white margin.

The bladder, ureters, and the pelvis of the kidneys were inflamed, and presented some phosphatic deposits upon their surface. The kidneys were somewhat large, succulent, and showed evidence of early suppurative nephritis. The capsule peeled off easily, and below it were seen, in large numbers, minute white dots, which felt gritty when touched with the point of the knife. In one kidney was a small infarct, opaque yellowish white in colour, with a red margin. The weight of the kidneys was 10½ ounces.

The liver was somewhat large, and very heavy, weighing 70½ ounces. The pyloric end of the stomach was attached by slight adhesions to the gall-bladder. The latter was completely filled by a large gall-stone. Its walls appeared to be fairly healthy, except at the extreme fundus. The latter was thickened by new growth, and projected as a hard nodule in the small indentation, through which the healthy gall-bladder often projects on the upper surface of the liver. From the nodulated fundus, as a centre, grew a large mass of carcinoma into the substance of the right lobe of the liver. The line which limited its growth farthest away from the gall-bladder was curved, and in its neighbourhood the luxuriant new growth bulged upon the surface of the organ. That portion which was nearest to the gall-bladder was depressed and harder, giving one the impression that it was older than the peripheral growth. Large vessels coursed over the surface of the mass. One or two very minute islands of new growth were seen here and there in the liver-substance at a distance.

The stomach and intestines were healthy, and no focus of new growth was found which could have given rise to that in the liver as a secondary product. The spinal column, as seen from the interior of the body, seemed to be perfectly natural; nor, on removing the posterior portion of it, and taking away the spinal cord, did it present any traces of disease.

On removing the brain, the greater bulk of the right hemisphere, as compared with the left, was very striking; to the finger it felt soft, especially in the lower part of the parietal region, and in the temporo-sphenoidal lobe. On making a horizontal section, so as to expose all the central ganglia to view, it was seen that the whole of the corpus striatum—both the nucleus caudatus and nucleus lenticularis—was softened, and broke up under a stream of water. The internal and external capsules, the convolutions of the island of Reil, and other convolutions adjacent to them, were in a similar condition. The right middle cerebral artery was found to be completely blocked, just beyond the spot where the first few vessels are given off to the central ganglia.

On examination of the spinal cord, it was found that the dura mater was healthy. At the junction of the lower dorsal and upper lumbar regions, was a softened area about one inch in length, and involving apparently the whole thickness of the cord. It was rather deep red in colour, with a pale yellowish island at one spot, which, with its deeper coloured periphery, was very suggestive of infarction, as it is seen in other organs. Higher up, about the middle of the dorsal region, the anterior spinal artery was interrupted in its course, and surrounded by a long superficial hæmorrhage of a brownish-red colour. The cervical enlargement, especially in its upper part, seemed to be of enormous dimensions, retaining, nevertheless, the natural shape of the cord. On pressing with the finger, it was found to be very firm over an area one to two inches in length. On making a transverse section through the cord in the upper cervical region, where there was no feeling of increased density, the central canal was found to be much enlarged, and to contain blood, partially coagulated. Where the canal opened out into the fourth ventricle, it appeared to be

healthy. When a section was made through the enlarged and hardened portion of the cord, a most curious appearance presented itself. A very narrow and even rim of apparently healthy white matter surrounded, as with a sheath, a dense reddish-grey mass, of oval outline. In the centre of this mass was a hollow space, which presented a very marked resemblance in shape to the central grey matter of the spinal cord, and which contained blood. On teasing out a portion of this new growth in the fresh state, it was found to consist mainly of spindle-cells, and of cells with many long fine processes, such as one sees in gliomata.

In the first of these two cases of spinal tumour, where the pressure was exerted upon the cord from without, death occurred in twelve months from the appearance of the first symptoms. The latter were, in order of time, weakness of the legs in walking; increasing weakness of legs, accompanied by cramp and shooting pains; paraplegia, cramp, and involuntary flexing of legs; slight numbness and long continued attacks of muscular spasm in the lower extremities; and, finally, persistent rigidity. In the second case, the symptoms, due to pressure alone, which was exerted upon the cord from within in the cervical region, continued for fifteen months uncomplicated by other disease; they were, simply, partial loss of power and sensation in the arms and hands, and some tremor in them when used. This was the condition of the arms when the patient was admitted into the hospital; and it was little altered even at the time of his death, which occurred twenty months after the appearance of the first symptoms. During the last five months of life, fresh symptoms arose in relation with hæmorrhage and softening in the dorsal region of the cord—accidents which are very liable to occur in cases of gliomata; and the accident which finally proved fatal was embolism of the right middle cerebral artery, giving rise to left hemiplegia.

When a tumour grows in the spinal canal outside the cord, it may produce but few symptoms until it presses the cord against the resisting walls of the canal; but, after this has taken place, the course of the disease is naturally very rapid, as the cord is quickly flattened by the constantly increasing demands for growing space which are made by the tumour. When a tumour arises, on the other hand, within the spinal cord, it disturbs its functions even from the very commencement; but, as the nerve-substance appears to be elastic, and to allow a good deal of gradual stretching without serious interference with its functions, a tumour may go on growing for a long time before it produces striking pathological phenomena, either by pushing the cord against the bony walls of the spinal canal, or by exhausting the elasticity of the membranes which envelope it. These remarks only apply to gliomata and such non-malignant tumours as exert pressure on surrounding tissues, but do not invade them. The description which I now give of the microscopic appearances of the spinal cord and of the tumour in the present case, shows that a state of things is produced by the disease which fully explains the chronicity of some of these cases, and the trivial symptoms which present themselves.

The tumour is situated almost entirely in the cervical region, and mainly in the cervical enlargement. In the middle of the latter, the mass assumes its largest proportions, and the enveloping sheath of nerve-tissue is comparatively smaller than in any other region of the cord (Fig. 24).

FIG. 24.—Transverse section (natural size) of spinal cord in a case of central glioma. *a.* Rim representing spinal cord. *b.* Anterior cornu. *c.* Posterior cornu. *d.* Central tumour.

When examined with the microscope, the greater part, both of the white and of the grey matter, can be readily recognised, though pressure has produced some alterations in them. On holding up a stained transverse section to the light, one observes in its anterior third a small oval area which is more transparent than the parts around; it is situated in the white rim, and lies upon the tumour. Under a low power, this proves to be the anterior cornu, containing the usual multipolar cells (Fig. 26). Symmetrically placed upon the opposite side of the tumour is a similar oval mass, which is the other anterior cornu. The cells are not healthy on either side, but are pigmented, and more or less elongated by pressure (Fig. 28); but, considering the pathological conditions to which they have been subjected, they are singu-

larly well preserved. The anterior roots can be traced from the cornua through the white substance to the periphery of the cord, and the posterior cornua extend backwards along the margin of the tumour. The lateral, posterior, and anterior columns of white fibres are all present, and show but slight pathological alterations (Fig. 27); there is no marked sclerosis anywhere. The same may be said of the whole extent of the cord which is occupied by the main bulk of the tumour. The latter becomes smaller and smaller in the lower cervical region, and is no longer evident as a mass in the lower dorsal region. Here, however, considerable hæmorrhage and softening have occurred (Fig. 25); and to these changes the later symptoms in the case may be referred, when the legs became paralysed, and increase of the tendon-reflexes and ankle-clonus appeared.

As the tumour becomes smaller, and finally ceases to be recognised as such in the dorsal region, it is probable that the changes present in the cord there represent the early stages of its growth. They occupy the left half of the posterior columns and the left lateral column, and consist of a peculiar thickening of the neuroglia, which is transformed into a hyaline substance (Fig. 37). The nerve-fibres in its meshes are at first but little altered, then they become swollen; the vessels are much thickened, and their walls assume the same glassy appearance as the neuroglia. In the cervical region, the central canal cannot be recognised; but in the lower parts of the cord, where the growth is early, it can be seen pushed to the right.

As to the tumour itself, it consists of numbers of islands of new growth situated around large blood-vessels as centres, and starting from them. The vessels have the thickened hyaline walls already referred to, and numbers of fibrillæ can be seen issuing from them. The new growth is richly nucleated, and has a very definite radiate arrangement round each centre. On examining very fine sections under high powers, the new tissue is seen to consist of meshworks of very delicate fibrils, and bundles of fibrils, in which are situated the nuclei. It may, therefore, be put down as a glioma. (Figs. 29 and 30.)

In Virchow's *Archives* for 1884 a very similar case is recorded by Dr. Reisinger, and an abstract of all the cases to be found in medical literature is added. They amount to twenty in all. It is very curious that, in Dr. Reisinger's case, the earliest changes were seen, as in mine, in the posterior columns near the central canal.

The minute anatomy of the specimen I show here explains why the symptoms in cases of central tumour of the cord may be so slight and extend over such long periods of time, and why spastic conditions are not readily induced, as they are when pressure is applied from without. For, even when the disease is so extreme that the substance of the cord forms merely a narrow ring round it, the nerve-tissue presents very slight pathological changes.

I shall now relate a case which illustrates the modification which occurs in the symptoms of disease of the lateral columns, when it is combined with disease of other parts of the cord.

Case of Primary Disease of the Lateral Columns, accompanied by similar changes in other parts of the Central Nervous System.—M. H., aged 53, whose occupation was pulling down old houses, was admitted into St. Thomas's Hospital, under the care of Dr. Ord, on November 10th, 1884, and died on December 2nd, 1884. There was no history of nerve-disease in his family. He had always enjoyed fair health, though troubled with indigestion and bilious attacks. The only definite illness he had had was ague at the age of 23. He had never had syphilis. Before his present illness began, he had been in the habit of drinking five or six pints of beer daily, but no spirits; he also smoked a good deal, about half an ounce of tobacco a day. His occupation necessitated pretty constant exposure to cold and wet, and he had had several falls, but had never had any symptoms of importance after them. About seven years before admission to St. Thomas's, he had had great anxiety about money matters, and was up night and day for about a month. He then got out of sorts, and presently noticed that the toes of both feet were becoming numb, and the legs somewhat stiff; this, together with weakness, produced difficulty in walking, and the right leg began to drag. He was quite unable to walk in the dark, or with his eyes shut, or to turn round quickly. The difficulty in walking had steadily increased, and both legs had become equally affected. Six months ago, total inability to get about made him take to his bed, the numbness increased, and he began to be troubled with jerking of the legs; the arms also got somewhat stiff. He noticed that, although he could grasp large objects well, he could not pick up small ones, such as pins. He had burning pains in the legs and shoulders, and shooting pains in the back and abdomen. During his whole illness, he had suffered from indigestion and constipation; his legs had become thinner, and he had lost weight. His sight, which used to be very good, had gradually become dim, so

that he could only read print when it was held very close to him. His hearing had remained good. The right foot and ankle had been swollen for a short time a year previously, but none of his other joints had been affected. He had lost all sexual desire, and for five years had had some trouble with his urine, sometimes having retention, at others continuous dribbling. He had never had any difficulty in speaking or in swallowing.

On admission, the patient was an emaciated man, complaining of inability to walk, dimness of sight, and shooting pains in the legs and thighs. Although unable to stand, he moved his legs freely in bed, and there appeared to be no loss of power in them, but there was some rigidity. The arms showed no loss of power; the grasp was strong with both hands, but there were slight tremors in the right arm when he used it. The muscles of the face were stiff and tremulous when strongly contracted. There was partial loss of control over the bladder, but none over the rectum. Sensation was impaired in the lower extremities, but not markedly; the patellar reflex was absent, and there was no ankle or knee-clonus. Plantar reflex was brisk on both sides, the cremasteric absent. The lower abdominal reflex was slight on both sides, the epigastric absent. The right pupil was larger than the left, and both acted to light and to accommodation. There was no oculo-motor paralysis or colour-blindness; he had slight lateral nystagmus. There was well marked atrophy of both discs, most marked on the inner side.

There was no affection of speech or of hearing, but the sense of smell was much impaired. The radial vessels were somewhat thickened, the bowels constipated. The abdominal and thoracic viscera were free from serious disease. The urine had specific gravity 1025, was alkaline, and contained phosphates, but no albumen.

During the rest of his life, little alteration occurred in the symptoms which have been described, but cystitis with high temperature set in. He was also attacked with swelling, redness, and œdema of the metatarso-phalangeal joints on both sides and of the ankle-joints, and finally got bed-sores, and died on December 2nd.

The following is the account of the *post mortem* examination, which I made on December 3rd. The body was that of a well nourished, grey haired, old man. The legs were thin, and proportionately thinner than the arms. Both heels were black, as if from commencing gangrene. The external malleolus on the left side, as well as the sacrum, was in a similar condition. The ankle and knee-joints were quite healthy. The heart, lungs, liver, and spleen presented no evidences of disease. The kidneys were healthy, but the bladder was evidently inflamed, as it contained three or four ounces of flaky, purulent fluid, and its mucous membrane was of a deep purple colour. The intestines, pancreas, and suprarenals were all free from disease. The sheath of the spinal cord was distended with clear fluid, so that, when the latter escaped, the cord was seen to occupy but a small part of the tube formed by the dura mater. There was no undue vascularity, nor anything remarkable on external examination. On making transverse sections of the cord from below upwards, it was found that in the lower part of the lumbar region there were no abnormal appearances. But in the upper lumbar region three tracts of degeneration were visible. Two of them were in the lateral columns, which were symmetrically affected; the third consisted of the columns of Goll, which together formed a triangular area. The diseased parts were transparent, and had the colour of glue. The degeneration could be followed in all three tracts as far as the medulla oblongata, but there it became less and less evident, and could not be seen at all in the upper part of the medulla and pons Varolii. The disease in the lateral columns appeared to be superficially situated, and was more extensive in the upper than in the lower regions of the cord. It occupied a larger area on the surface of the lateral columns, but penetrated less deeply into them than is usually the case in descending degenerations. It was impossible to say, from a naked-eye inspection, whether the columns of Goll were the only parts of the posterior columns which were diseased, or whether the degeneration affected those of Burdach as well. The cord was firm, and, with the exception of the regions already referred to, it appeared to be healthy.

The only morbid appearances discovered in the brain were found in the optic nerves. They were both pale and flattened, and evidently the seat of degeneration. The corpora geniculata, corpora quadrigemina, optic thalami, and supposed cortical centres of vision, were all normal in appearance. There were symmetrical ivory exostoses on the fourth and fifth ribs on each side in the posterior third of their length, but they were not so situated that they could press upon either nerves or vessels.

The symptoms in this case which point to lateral sclerosis are weakness of the limbs, tremulousness, jerkings, and rigidity; while numbness, pains, loss of sexual desire, difficulty with the evacuations,

dimness of sight, and absence of the patellar reflex, indicate more extensive disease.

On microscopical examination, the spinal cord proves to be the seat of very widespread changes; and yet the parts affected are so constant throughout, that the affection cannot be looked upon as an indiscriminate one, but must be placed among the more complicated combined system-diseases. It will be seen by reference to Fig. 33 that the regions affected are (1) the lateral columns; (2) the posterior columns; (3) the direct pyramidal tracts; (4) the anterior root-zones; and (5) in a minor degree, the grey matter of the anterior cornu.

But it will also be observed that, in the neighbourhood of the grey matter everywhere, a zone remains healthy, and, in addition, a superficial tract of fibres situated between the crossed pyramidal and cerebellar tracts posteriorly, and the anterior root-zones anteriorly, is unaltered. The pathological changes throughout the diseased areas are the same, and must be considered to be those of chronic inflammation affecting mainly the nerve-fibres, and in a far less degree the neuroglia; in other words, they represent a form of chronic myelitis. In the regions referred to are seen increase in the nuclei of the neuroglia, and in the white corpuscles within the vessels, hyaline swelling of the neuroglia, distension of the neurilemma, and disintegration of the white substance of Schwann (Figs. 34, 35, 36).

Motor phenomena were those which were most strikingly affected during life, and sensory changes were far less marked. The pyramidal tracts, direct and crossed, and the anterior root-zones, are undoubtedly motor regions, while the columns of Goll are afferent, and connect centres below with centres higher up; hence the disease is principally an affection of the motor systems of fibres. The escape of the small superficial area in the lateral column from pathological changes is exceedingly interesting, and marks it out as a tract containing some particular set of nerves. Gowers first called attention to this small tract of fibres which appear to be afferent, and to degenerate centripetally. Several other writers have also mentioned it, among them Dr. Hadden, and Dr. Howard Tooth has a paper on it in the *St. Bartholomew's Hospital Reports* for 1885, vol. xxi.

The medulla oblongata, pons Varolii, crura cerebri, motor convolutions, anterior crural and optic nerves, and the triceps femoris muscle, were all examined microscopically. The optic nerves were found to be atrophied, and there was an excess of nuclei in the anterior crural nerve and in the triceps femoris; but with these exceptions, all the parts examined were free from disease.

I have detailed this case, rather than one of the more usual ones, where chronic affections are confined to less extensive systems of nerve-fibres, in order to show that disease is a very complex process, however much we may endeavour to simplify our notions of it, and likewise to point out in how modified a form disease of the pyramidal tracts presents itself, if there be in addition pathological changes in other regions of the cord.

Before leaving the subject which I have thus far been illustrating, namely, the relation between diseases of the cerebral motor system and the production of chronic muscular spasm, a few words must be added in answer to the question, Does spasm ever result from afferent stimuli reflected on to efferent fibres in the cerebral centres, or from diseases of the commissures? If we limit the term cerebral centres to the grey matter of the hemispheres and central ganglia, I know of no undoubted instances of reflex spasm in connection with them. Convulsions, which may be due to such causes, do not come within the scope of the present lectures; and had I the time to treat of them, I should shrink from casting a shadow upon ground which has been so brilliantly illuminated by the genius of Hughlings Jackson. Several cases of tumours of the corpus callosum have come under my notice, and they have been published by Dr. Bristowe in *Brain*, vol. vii. I cannot do better than quote the words of so distinguished a physician and pathologist in describing the symptoms which were observed in them. The chief characteristic features were:

"1. Their invagrescent character, a character which they possessed in common with other cases of cerebral tumour.

"2. The gradual coming on of hemiplegia for the most part resembling in its distribution the paralytic symptoms usually attending hæmorrhage into one of the hemispheres, or softening due to embolism.

"3. The association with the paralysis of one side of vague hemiplegic symptoms of the other.

"4. The supervention of stupidity, associated for the most part with extreme drowsiness, a puzzled inquiring look when awake, a difficulty of getting food down the throat, and cessation of speech.

"5. The absence of implication of the oculo-motor nerves, and of direct implication of other cerebral nerves; and lastly, death by coma."

From beginning to end of these cases, no muscular spasm occurred.

In the few remarks which the time at my disposal has allowed me to make on diseases of the pyramidal tract, and the part played by it in the production of spasm, I have tried to point out how numerous and varied the clinical phenomena may be, according to the level at which the pathological conditions occur, according to the method of attack—whether from within or from without—and according to the presence or absence of disease of other portions of the central nervous system.

I must now leave this division of my subject, and pass on, in the last lecture, to a consideration of the spinal motor system, and to so-called functional spasm.

TWO LECTURES

ON

TUMOURS OF THE LARYNX; THEIR PATHOLOGY, SYMPTOMS, AND TREATMENT, WITH ILLUSTRATIVE CASES.

Selected from a Course of Lectures delivered during the Winter Session of 1885-86 at the Glasgow Royal Infirmary.

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LECTURE I.

GENTLEMEN,—The cases and specimens which form a text to my lecture to-day illustrate the differences between two classes of tumours of the larynx, the benign and the malignant. These may be separated from one another by their histological distinctions, and by their clinical characteristics.

In pre-laryngoscopic times, the diagnosis of a tumour in the larynx was limited to those cases in which the growth occupied the upper part of the laryngeal cavity, so as to be either visible to the eye when the tongue was depressed, or within reach of the finger. Isolated cases of laryngeal polypus were recorded, even as far back as the middle of the eighteenth century; but at that time, and for many years subsequently, the diagnosis of such tumours was most uncertain. It was only towards the middle of the present century that the diagnosis of these diseases became exact, and that attempts were made to extirpate laryngeal growths by the mouth; although one hundred years previously, it is true, Kaderik performed for the first time, successfully, the endolaryngeal operation. I will not attempt even briefly to trace the gradual development which has taken place during the last half century, in the diagnosis and treatment of the diseases we are now considering; but I think I may say, with confidence, that, whereas new formations in the larynx were formerly supposed to be of rare occurrence, and beyond the reach of the surgeon, they are now not only readily recognised during the life of the patient, but they may be successfully removed by surgical appliances. In fact, there is probably no other class of diseases in which the advantages of laryngoscopic examination have been more clearly demonstrated. Not only are tumours now known to occur more frequently than was formerly supposed, but there is even a tendency on the part of practitioners to believe that they prevail more commonly than is, on reference to statistics, actually found to be the case. You will find in practice that they constitute only a small proportion of the chronic maladies of the larynx, certainly not more than two or two and a half per cent.; so that, although the diagnosis of the individual case may be of the utmost importance to the patient, the number of cases which you will meet with in general practice is comparatively small.

Before proceeding further, I must request you to note clearly the characteristics which separate tumours from growths of inflammatory origin. In the former, the neoplasm has a tendency to persist and increase; while the latter tend always to disappear, to develop into a higher state, or to reproduce the tissue of their matrix. Tumours obey the laws which regulate the nutrition of normal tissues; but at the same time they have an independent life, or they may grow at