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

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LECTURE I.

MR. PRESIDENT AND GENTLEMEN,—When I was informed that I had been nominated to fill the honourable position of Gulstonian Lecturer for 1886, I consulted the records of the College, in order that I might acquaint myself with the conditions attaching to the lectureship. I found that they were few in number, and that the main object which Dr. Gulston had in view was to secure, not only an annual dissertation on certain diseases, but also a practical demonstration of their pathological anatomy. It seemed to be incumbent, therefore, upon the lecturer, to choose a subject which he could illustrate, however imperfectly, from his own experience; and I selected that class of chronic diseases which have this, at least, in common, that they are associated with muscular spasm. To you, who know the wide range of knowledge and the ample experience which would be required to treat such a subject exhaustively, this proposal must appear pretentious to the last degree. I am fully aware, however, that the limited time at my disposal, and still more the limited knowledge I possess, make an exhaustive consideration of it impossible. I merely propose to take a general view of chronic nerve-disease in its association with muscular spasm, and to consider how far our present knowledge of pathology enables us to understand the phenomena which present themselves.¹

By spasm is meant excessive muscular contraction; and, if we confine our attention to those muscles which are under the control of the will, excessive contraction is that which occurs in defiance of the will, or which, although commencing as a voluntary act, transgresses the limits which the will would impose upon it.

There is great variety in the spasmodic affections which have been observed in man, and to which descriptive names have been given. Thus, "tonic" spasm indicates persistent muscular contraction, a tetanic condition of a muscle, or of groups of muscles, in which the individual waves of contraction overtake each other, and are fused into one. "Clonic" spasm is a term applied to conditions in which the individual waves succeed each other less rapidly and often quite irregularly, giving rise to a great variety of intermittent spasms. The "co-ordinated spasms," form another class, in which groups of muscles are the seat of clonic contractions, which are co-ordinated in such a manner as to produce some regularly recurring though involuntary movement. It is unnecessary to proceed further in explanation of the many forms of pathological contraction of muscle, which are embraced under the term spasm; they will be referred to again in connection with the diseases which they accompany.

In health, as a rule, contraction of striated muscles depends on impulses transmitted to them through the nervous system, and travelling down motor nerves; and the same may be said of that which is the product of disease; so that the motor nervous mechanisms afford a suitable basis, in connection with which we may survey our present knowledge of the conditions which give rise to spasm. It is convenient, and I might almost venture to say scientific, to divide the motor mechanisms into a cerebral system and a spinal system; the latter forming a groundwork on which the former is superposed, comparatively late in the history of the development of the individual, and probably of the species. It might be thought that, considering the importance of the cortical centres of the hemispheres, of the central ganglia, and of the cerebellum, the term cerebral systems would be more appropriate than cerebral system. But it is better, at any rate in considering spasmodic diseases, to embrace them all under one head. For 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, diseases of which give rise to

chronic muscular spasm. Evidence in support of this view will appear in subsequent portions of these lectures.

The terms "cerebral system" and "spinal system" are intended to be anatomically exhaustive, and to comprehend all possible nerve-mechanisms by which spasm is produced. There should be no room for any further division of the subject. But our ignorance, especially of the finer molecular changes which accompany not only abnormal but normal neuro-muscular conditions, during action and inaction, necessitates some reference to those spasms which are called functional, and which will, we may hope, with advancing knowledge, become amenable to a more accurate classification. The divisions of the subject, then, as now proposed, are (1) spasm in connection with cerebral motor mechanisms; (2) in connection with spinal mechanisms; (3) functional spasm.

1. SPASM IN CONNECTION WITH CEREBRAL MOTOR MECHANISMS.

The nerve-fibres, which bring the voluntary muscles of the body under the control of the will originate, so far as we know, in the central portions of the cortex. Experiments on living animals first yielded the clue to this discovery in man, and pathological evidence has abundantly corroborated the facts derived from this source. On an occasion like the present, it will not be thought a presumption if I refer to my own observations upon this point. In a paper published in the *Lancet* of September 29th, October 6th and 13th, 1883, I recorded six cases of cortical lesions all occupying the region in question, and all accompanied by motor paralysis, which occupied the face, arm, and leg, according to the localisation of the disease. It is unnecessary at present to enter into much detail on this subject. The two ascending convolutions which form the boundaries of the fissure of Rolando, the superior parietal lobule, the paracentral lobule, and probably some of the parts immediately adjacent to these, constitute the motor area of the cortex (Fig. 1). All we know about it, as regards voluntary

Fig. 1.—Diagram showing the position of the Motor Area of the Cortex on the External Surface of the Brain.

muscular acts is, that the "way out" of the cortex for voluntary impulses passes through it. Some of the motor fibres are connected with processes of large ganglionic cells, which form a very striking feature of this region of the brain, and which resemble the cells of the anterior cornua of the spinal cord. The centres which are in connection with the leg occupy the upper part, those which are in connection with the arm the middle, while the lower portions near the fissure of Sylvius preside over the muscles of the face and tongue. During a part of the very long course which the fibres coming from the motor area pursue, the bundles on their way to the spinal centres for the face, arm, and leg, remain distinct. Thus, passing from the cortex through the white substance of the hemispheres, they converge to the internal capsule, and occupy the knee and anterior two-thirds of its posterior segment, which lie between the optic thalamus and the lenticular nucleus (Fig. 2). In the capsule, the nerves for the face- and tongue-centres occupy the knee, the fibres for the arm come next, and those for the leg lie posteriorly. The knowledge of their relative positions in the internal capsule, which is a somewhat recent acquirement, is of great interest, because it probably affords at least part of the explanation of a long observed clinical fact; namely, that, in hemiplegia in connection with disease of the central ganglia, the arm is usually more paralysed than the leg. For hæmorrhages from the branches of the lenticulo-striate and lenticulo-optic arteries would, from their position, press more upon the anterior, than upon the posterior division of the motor segment of the capsule.

Leaving the internal capsule, the fibres under consideration pass to the superficial part (basis) of the crus (Fig. 3), and there occupy its

¹ I am indebted to my friend, Dr. Theodore Acland, for many of the microscopic preparations from which drawings have been made, and to Mr. Shattock, Mr. Burgess, and Mr. Lapidge, for most of the drawings.

modian portion, and are so placed that the fibres presiding over the face are situated internally, those over the leg externally, and those in connection with the arm centrally.

Arrived at the pyramids, in the medulla oblongata, the fibres going to the spinal motor centres for the body and limbs decussate, those for the face and tongue having already done so a little higher up. It has been estimated that about 91 to 97 per cent. decussate; but the number probably varies within wide limits, so much so, that there is good reason for thinking that in some cases complete decussation, in others none at all, takes place. Flechsig has also shown that, in the same spinal cord, the two

pyramids (the crossed pyramidal tract) occupies the posterior part of the opposite lateral column in the cord, being separated from the surface of the latter by the direct cerebellar tract. The fibres which do not decussate (direct pyramidal tract) pass down the cord on the same side, occupying a narrow strip along the margin of the anterior fissure. (Fig. 4). The fibres of the pyramidal tract finally form connections

Fig. 4.—Diagram of a Section of the Spinal Cord in the Cervical region (Flechsig). *a*, Direct Pyramidal Tract. *b c*, Antero-lateral Column. *d*, Direct Cerebellar Tract. *e*, Crossed Pyramidal Tract. *f*, Columns of Burdach. *g*, Columns of Goll.

with the multipolar cells in the anterior cornua of the cord, either directly or through the intermediation of other nerve-cells.

It is a well known fact that nerve-fibres in general depend for their integrity upon connections with ganglionic cells, and that, when these are severed, the fibres degenerate. Thus, the motor fibres of the cord depend upon the cells in the anterior cornua; the sensory fibres upon cells in the ganglia on the posterior spinal roots; while the healthy nutrition of the fibres of the pyramidal tract is dependent upon the cells in the motor convolutions. If their connection with the latter be anywhere interrupted, or if the cells themselves be destroyed, degeneration occurs in the whole tract below. When this has taken place, tonic spasm supervenes in the muscles, which are, in health, under the control of these fibres. But, between fully developed spasm and the earlier slighter indications of its presence, there are many gradations. Taking, for instance, what is perhaps the commonest condition which gives rise to these events—namely, destruction by a sudden hæmorrhage of the motor portion of the internal capsule—it is often some months before well marked spasm is observed; but, in the meantime, there are phenomena which foretell its coming. The most striking and frequent of these is the increased knee-jerk, which a tap on the patellar tendon elicits. The knee-jerk may be diminished for some time after the apoplectic attack; but, if the fibres of the pyramidal tract have been interrupted, it soon becomes exaggerated. What are termed ankle-clonus and knee-clonus (phenomena which it is unnecessary to describe before an assembly like the present) soon appear, or may do so; for they are not necessarily present where the disease is slight. Charcot reports a case where ankle-clonus appeared eight days after the hemiplegic attack, and late rigidity began within three weeks; and another, in which it set in in one month, and late rigidity in two. Pitres gives cases in which clonus was observed in from eleven to fifteen hours. But, in any case, it may be said that increased tendon-reflexes and clonus precede rigidity; and, in many cases of chronic disease of the lateral columns of the cord, they are to be found a very long time before rigidity, or the latter may not set in at all before death occurs. Sometimes, one tap on the patellar tendon gives rise to a succession of knee-jerks, and like phenomena may appear in the other limb, or in muscles still further removed. Brissaud asserts that even the non-paralysed side is not healthy, but presents in a certain degree these phenomena, which he terms "latent contracture." To what cause are these increased tendon-reflexes to be referred?

Muscular tone is that condition of tension in healthy muscle which is, no doubt, due to nerve-impulses proceeding from the cells in the anterior cornua of the spinal cord. A certain number of observers have come to the conclusion that the knee-jerk and other tendon-contractions are local phenomena, due to direct contraction of the muscle, which is temporarily put upon the stretch by tapping its tendon. Its increase is due to the increased tension which results from severance of the spinal centres from the cerebral motor centres, which exercise a controlling power over those below, this severance producing increased muscular irritability. But for the tendon-phenomena to take place at all, the reflex arc producing tension must be intact.

Other authorities have convinced themselves, by equally careful observations, that the tendon phenomena are true reflex neuro-muscular acts. The objection to this conclusion, which appears insuperable to their opponents, is that the latent period is much too short for a

Fig. 2.—Horizontal Section of Brain (Flechsig). Diagram showing the position of the Motor Strands (*a a* unshaded) in the Internal Capsule.

sides may be unsymmetrical in this respect. Some observers suppose that decussation of fibres of the pyramidal tract occurs in the cord; but nothing is known for certain with reference to this point. That part of the pyramidal tract which crosses at the decussation of the

Fig. 3.—Diagram representing a Vertical Section through the Crura Cerebri: *a a* unshaded area—show the position of the Motor Fibres from the Motor area of the Cortex.

reflex act. But surely it is not legitimate to bind all reflex acts by the exact laws which appear to govern skin-reflexes. It is probable that conduction in nerves does not take place at precisely the same rate in every reflex arc in the body, nor even that the time required to reflect the afferent impulse on to the efferent nerve is uniformly the same. This will depend upon the purpose for which the muscular act is excited, and the speed with which it needs to follow upon the stimulus which evokes it. Now there are, as has been proved, nerve-endings in muscles and tendons; and it is not improbable that the slight stretching of the latter, which is produced by the action of antagonists, gives rise, by means of a reflex act, to the steadying action, which is always aroused by the contraction of opponents. This would require great sensitiveness on the part of the reflex arc; for, considering the suddenness of many muscular acts, the steadying contraction of the muscles not directly producing the movement would require to be almost equally sudden. If the muscular act which follows a tap on the patellar tendon be a purely local phenomenon, due to "myotatic irritability," it is difficult to explain those cases in which contraction of muscles in the opposite leg, or even in the arm, occurs. Gowers states that the latent interval for the knee-jerk is $\frac{1}{2}$ sec. to $\frac{1}{4}$ sec., and that physiological data require that it should be $\frac{1}{4}$ sec. if it be a true reflex. But what will be said of the following equally legitimate argument? The interval for the knee-jerk is $\frac{1}{2}$ sec. Dr. de Watteville and Dr. Waller have found the interval for the jaw-jerk to be $\frac{1}{2}$ sec., which is only half the interval required for the knee-jerk. Therefore, the phenomena are not produced in a similar manner. Dr. de Watteville says that Dr. Waller has determined the latency of the closure of the eyelids to the stimulus of a strong light to be $\frac{1}{10}$ sec., the shortest true reflex known. This differs much less from the interval for the knee-jerk ($\frac{1}{2}$ — $\frac{1}{4}$ sec.) than the latter differs from the jaw-jerk ($\frac{1}{2}$ sec.).

We know too little about the various reflexes which occur in the body to consider it as proved whether tendon-phenomena are reflex or not, but it is at any rate satisfactory to feel that, for all practical purposes at the bedside, either theory is equally applicable. For the key-note to both is integrity of the reflex arc, and changes in the latter are necessarily accompanied by changes in the tendon-phenomena.

Let us assume, provisionally, that the knee-jerk and other allied contractions are reflex. Why are they increased in disease which interrupts the continuity of the pyramidal tract, or destroys the motor cells in which its fibres originate?

Probably the reason is, not that the descending sclerosis produces irritability of the cells in the anterior cornu, but, as Hughlings Jackson says, that the suppression of the functions of the pyramidal tract gives rise to "permitted hyperphysiological activity," the spinal centres being "let go." This view is strongly supported by those cases of congenital absence of the whole or a part of the motor centres in the brain, which are accompanied by contracture of the limbs similar to that which occurs in lesions of the pyramidal tract resulting from disease; for, in these cases, the pyramidal tract has been proved to be deficient. This was the condition found by me in a case which I shall relate presently. The pyramidal tract is not developed until very late, and is not even complete at birth; and, in the cases of congenital disease referred to, it is not developed at all, or only in part. Even in disease which occurs in adult life and produces descending sclerosis, if the cord be examined after many years, the process of atrophy has long been completed, the nerve-fibres have disappeared, and nothing remains but a quiescent cicatrix. This was so in another case which I shall relate. In such conditions, no active process is going on to produce contracture; there is merely destruction of the pyramidal strands. Again, the exhaustion following upon the motor discharge in epilepsy, may give rise to temporary increase in the knee-jerk, or to ankle-clonus; and the same may be said of that suppression of function produced by the slighter degrees of chloroform-narcosis. In short, the excessive tendon-reflexes and contractions which result from descending sclerosis are merely evidences of abolition of the function of the cerebral motor tract, which, in health, controls the action of the spinal ganglia.

Objections have been made to this view on the ground that sudden disease, such as cerebral hæmorrhage, which destroys the fibres of the internal capsule, does not give rise to the phenomena in question immediately, but only after the lapse of a certain time. The cases recorded by Pitres, in which ankle-clonus was observed to supervene in a few hours, prove that this delay is not an invariable rule; and, even if it were, it would not be unreasonable to suppose that cells, which have long been under the control of others, should require a certain time before they learn to exert a power to which they have hitherto been unaccustomed.

We may therefore look upon increased tendon-reflexes, clonus, and

contracture, as degrees of independent neuro-muscular action, originating in the spinal centres, which have run riot owing to the diminution or annihilation of the inhibitory functions of the pyramidal tract.

But, it may be further asked, does the appearance of one or all of these phenomena prove that there is gross and tangible disease of the cerebral motor cells or fibres? The answer is, decidedly not. In the *Lancet* for November 7th, 1885, Dr. Angel Money records his experience of "Reflex Actions, Knee-jerks, and Muscular Irritability in Typhoid Fever, Phthisis, and other continuous Fevers," and he shows that all the phenomena, except tonic spasms, are found in these conditions; but even contracture may occur in fevers. I have myself seen it in typhoid fever, and Dr. Murchison mentions its occurrence in typhus and relapsing fevers as well. Speaking of the former, he remarks: "Contraction and rigidity of certain muscles are observed more rarely, and only in severe cases. The fingers may be tightly clenched, or the forearm flexed, or, in rare cases, there is trismus or strabismus. In twelve cases, I have observed tonic spasms of many different muscles. Twice I have seen the legs and thighs so bent that the knees almost touched the chin." A man, suffering from extreme debility and anæmia, came lately to my out-patient room, and the house-physician called my attention to the fact that he had not only very excessive knee-jerks, but as marked knee- and ankle-clonus as I ever saw. He was sent into the hospital; and, after two days' rest in bed, I was unable to elicit even the slightest clonus. Such cases as these, together with others in which recovery has occurred, justify the verdict which Dr. Donkin gives in *Brain*, October, 1885. "It seems clear," he says, "at present that we must bear in mind that we have not done with spastic paraplegia by calling it lateral sclerosis, remembering the cases which recover, whether they are quite obviously functional or not, but must believe with Friedreich, Wilks, and others, that the symptoms may occur under various forms of disorder or disease of the nervous system."

My own impression is, that the condition which most frequently gives rise to these abnormal muscular conditions in connection with the spinal centres, is partial or complete suppression of nerve-impulses, passing from the cerebral motor centres down the pyramidal tract; and this may occur under a considerable variety of circumstances. But, accepting the fact that contracture of muscles, accompanying or succeeding a phase where tendon-reflexes are exaggerated, is most typically seen and is most characteristic in gross disease of the motor convolutions and pyramidal tract, let us consider some concrete instances of it. Two facts must be remembered, however; and these are, first, that when contracture is well developed, it may prevent us from eliciting ankle- and knee-clonus, or tendon-reflexes; and, secondly, that contracture sometimes passes off, the muscles at the same time undergoing rapid atrophy, and the reflexes being abolished. This occurs when disease of the lateral column extends to the cells of the anterior cornu.

Case of Embolism of the Sylvian Artery, which resulted in Absorption of a large part of the Right Hemisphere of the Brain, and which was followed by Paralysis and Rigidity of Limbs.—The complete history of the patient's illness, and of the post mortem examination, is recorded in the *Transactions of the Royal Medical and Chirurgical Society* for 1884, and I shall not enter into much detail now. The case is an instance of extensive destruction of the cortex and central ganglia on one side, producing descending degeneration in the cord, together with the symptoms which are characteristic of that condition.

E. J., aged 34, was admitted into Guy's Hospital on March 8th, 1876, under the care of Dr. Pavy. She had never had any serious illness, and, though married ten years, had had no children. Suddenly she was attacked with giddiness, faintness, and sickness, and had an epileptiform seizure. After four days, she recovered consciousness, and found she was paralysed on the left side, and had no sensation on that side of her body. On admission, she was found to have ordinary complete hemiplegia and hemianæsthesia on the left side. She had, in addition, a systolic murmur, audible all over the cardiac area. About four months after the commencement of her illness, she left Guy's Hospital with the left arm almost useless, but with very fair power in the leg. She had completely recovered from the hemianæsthesia.

Seven years later, on May 7th, 1883, the patient, aged 41, was admitted into St. Thomas's Hospital, under the care of Dr. John Harley, with aortic disease and bronchitis. Dr. Hadden, the registrar, made the following note. "The fingers of the left hand are partially flexed on the palm, and decidedly stiff. The skin over the backs of the fingers is whiter and more glistening than it is on the right hand. The grasp on the left is feeble, and there is rigidity of the flexors of the forearm and arm. The tendon reflexes are very brisk in the left, but are hardly obtainable in the right arm. The left leg is somewhat

rigid, the patellar tendon-reflex brisk, and there is slight ankle-clonus. The tongue is protruded straight. Slight paresis is observed in the muscles on the left side of the face. There is no loss of sensation. On casual examination at the bedside, the left eye appears to be sound, and there is no hemianopia or colour-blindness. Tested with a watch, hearing is found to be sharp, and equal on the two sides." The patient died somewhat suddenly on May 17th.

The husband informed me that, from the period of her attack, seven years before her death, she had been weak-minded and queer, doing unaccountable things, such as she had never done before, but that she remained a good-tempered, hard-working, and affectionate wife. On *post mortem* examination, marked aortic disease (ulcerative endocarditis) was found, and there was great cardiac hypertrophy, the heart weighing 17½ oz. The lungs were hyperæmic and oedematous at the base, and the lower portion of the right was semi-solid. The liver showed slight congestion in the centres of the lobules ("nutmeg"). The kidneys weighed 11 oz., the capsule was thick and adherent, the substance very firm, and the surface granular. The spleen was hard, and its capsule thick.

In the right hemisphere of the brain, the parts supplied by the middle cerebral artery were extremely wasted, and many of the convolutions had entirely disappeared. The central ganglia, too, were so much affected, that the measurement from their intraventricular aspect to the outer surface of the convolutions of the island of Reil on the right, was not much more than one-third of that on the left. Descending degeneration of the lateral column, and also of the column of Türck, could be seen in the spinal cord; and the alteration in both these strands of fibres was situated on the side opposite the diseased hemisphere.

This case, the brain of which is now shown, together with drawings of microscopic sections of the spinal cord (Figs. 5, 6, 7), is of interest, not only because of the long time the patient lived after such extensive destruction of one hemisphere, but also because it is an instance of total decussation of the pyramidal tract. It will be seen on examination of the sections of the cord, that the left half is very much smaller than the right, and that the white substance bordering on the anterior fissure, the column of Türck, is decidedly smaller on the left than on the right; that is to say, there is no direct pyramidal tract, but the whole of the fibres from the right hemisphere appear to have crossed, and to have gone, some into the left lateral column, some into the left column of Türck.

On microscopic examination of the sclerosed tracts, there is seen simply diminution in the number of white fibres, and increase in the density of the neuroglia in the affected lateral column. Apart from the irregularity which exists in the columns of Türck, there is very little structural difference to be made out in them. The softening, which produced hemiplegia and subsequent rigidity in this instance, involved the cortex, central ganglia, and internal capsule.

Lesions need not, however, be so extensive, in order to give rise to paralysis with rigidity. Destruction of a portion of the motor convolutions, or of the subcortical fibres connected with them, or of that part of the internal capsule through which the motor strands pass, produces the same phenomena. Cases of softening of portions of the cerebral cortex, of long standing, do not often come under the observation of pathologists in the *post mortem* rooms of general hospitals; or, if they do, I have been singularly unfortunate in not meeting with them. A girl about eleven years of age, who was under my care as an out-patient at St. Thomas's Hospital, had had a very severe burn on the right side of the head when she was two years old. The scar occupied the greater part of the parietal region, and it was perfectly destitute of hair. I got no very clear details of the symptoms which succeeded this accident, except that she lost power all down the left side, a condition from which she had never recovered. Since then she had been subject to epileptic fits, in which the left side was convulsed; but she did not lose consciousness. The arm, when I saw her, was rigidly contracted, the leg being much less affected, and the deep reflexes were excessive all down the left side. Now, although the actual state of things was not demonstrated by a *post mortem* examination, it is pretty certain that the burn caused superficial disease of the parts situated immediately below and within the cranium, and destruction of a portion of the motor cortex, with descending degeneration of the pyramidal tract, was the result.

Case of Perforating Tuberculosis of the Skull, with Cerebral Symptoms.

—In *Brain*, April, 1885, Dr. Edmunds describes a case, which affords a good instance of descending degeneration and rigidity from a limited cortical lesion. The patient was a boy of 14, of a delicate family. Eighteen months before admission into St. Thomas's Home, he had suffered from what was supposed to be tubercular peritonitis; and at the same time an abscess, accompanied by much headache, formed

in the region of the left parietal bone. About six months later he complained, occasionally, of numbness in the right arm and hand, and two days before admission he had his first fit. He felt giddy, fell, became unconscious, foamed at the mouth, bit his tongue, and passed urine involuntarily. Both legs were convulsed, the right arm flexed, and the right hand clenched. He was admitted into St. Thomas's Home on September 3rd, 1884. An abscess was found situated over the left parietal bone. The grasp of the right hand was much weaker than that of the left, and there was very slightly deficient power in the right leg and right side of face. Sensation was everywhere normal, but double optic neuritis was present. I saw the patient with Dr. Edmunds at this time, and subsequently the abscess was laid freely open, and a piece of dead bone removed, so that the dura mater was exposed, and the pulsations of the brain could be seen. After this he had two fits, much like the one already mentioned. The discharge from the opening in the skull gradually diminished, and the scalp covered over the edges of the wound, so that when he left the hospital there was a defect in the skull and scalp, through which the dura mater could be seen. A silver plate was made to protect this. On February 4th, 1885, he left the Home, the chief symptom of disease which he presented being partial paralysis of the right arm and hand: when tested with the dynamometer, the right hand only marked 20, the left 70.

In the *Lancet*, for September 27th, 1884, my colleague, Dr. Reid, published his "Observations on the Relation of the Principal Fissures and Convolutions of the Cerebrum to the Outer Surface of the Scalp." Before the patient left the Home, Dr. Edmunds got Dr. Reid's assistance in determining the position of the lesion by surface markings. The boy's head was shaved, and the fissures marked upon the smooth surface, and the opening in the skull was found to be half an inch in front of the middle of the fissure of Rolando. But, as it was known

Fig. 8.—Diagram showing the position of the Cortical Lesion. The lighter shading represents the Area of Adhesion between the Dura Mater and the Arachnoid; the darker area, the Superficial Destruction of the Cortex.

that the disease extended backwards along the dura mater for a short distance behind the opening, the conclusion drawn was that the middle of the ascending frontal and ascending parietal convolutions was the part affected. The account I am about to give of the *post mortem* examination shows how accurate this localisation was. The patient returned to the Home on September 11th, 1885, looking very ill and emaciated, and with a sinus on the right side and back of the neck, connected with an evident angular curvature of the spine. His right arm was flexed at the elbow, and could not be completely extended. He died of exhaustion on November 12th, 1885, the end being accelerated by diarrhoea, which was scarcely controllable. At the *post mortem* examination, relics of past peritonitis were found, and old and recent tubercular affection of the lungs. There was a chronic abscess in relation with caries of the lower cervical vertebrae, the disease which had produced the angular curvature. There was chronic thickening of the membranes of the cord in this region, but no evident disease of the cord itself. Dr. Edmunds was good enough to hand over to me the brain and spinal cord for careful examination. The following is an account of the condition of things which I found, after hardening the specimens in spirit.

The dura mater, which was thickened and adherent over a portion of the left parietal convolutions, was gently peeled off, and that part of the cortex which was so far invaded by disease that it could not be separated from the dura mater, but was torn during its removal, is marked in Fig. 8. The lighter shading represents very slight superficial abrasion, and the darker central part shows where the disease had sunk deeper into the grey matter. The area marked in the figure

measured an inch and a half vertically, and the same horizontally. The position which it occupied was the upper half of the middle third of the ascending frontal and parietal convolutions, together with a small portion of the posterior extremity of the middle and of the superior frontal convolutions. On making a horizontal section through the basal ganglia and the internal capsule, a slight discoloration could be seen in the latter just behind its knee. On making a vertical section through the ascending frontal convolution, and traversing the centre of the diseased area, the destruction of cortex was seen to be quite superficial, and no degeneration was made out in the subcortical white matter. The cord was examined high up in the cervical region, above the seat of the chronic meningitis which resulted from the disease of the cervical vertebrae. The sections show that the right half is much smaller than the left, the diminution in size being apparent, not only in the lateral column, but also in the column of Türek. Under the microscope, the connective tissue in the right lateral column is seen to be denser than that in the left, and the nerve-fibres appear to be here and there less numerous.

Hospitals where old people congregate afford the best opportunities for *post mortem* examination of persons whose brain is the seat of softening originating long before death. Charcot has given his experience at the Salpêtrière, and he says (*Lectures on the Localisation of Cerebral and Spinal Diseases*, translated by Walter Baugh Hadden, M.D. 1883. New Sydenham Society, p. 141): "In these cases, the lesion presented itself under the form of yellow softening (*plaques jaunes*), more or less extensive in area, involving, to a variable depth, the subjacent white matter, and occupying the most diverse regions of the surface of the hemisphere. It is expressly mentioned in all the observations that the softening had not affected the central masses: optic thalami, caudate and lenticular nuclei, and internal capsule. My observations may be divided into two groups.

"The first includes the cases in which permanent hemiplegia had not existed during life, and in which secondary degeneration was found at the necropsy to be absent. In all, the convolutions supplied by the Sylvian artery, and particularly the ascending frontal and parietal, remained intact. The yellow softening was situated in one of the following regions, namely, some part of the sphenoidal lobes, the lobulus quadratus, the cuneus, one or both occipital lobes in their entirety, some part of the anterior two-thirds of the frontal lobes.

"In all cases of the second group, there had been, on the other hand, permanent hemiplegia and well marked secondary sclerosis. The feature common to these cases is that the lesions invariably involved, to a greater or less extent, one or other of the ascending frontal and parietal convolutions, principally in their upper half, and often both at the same time. In addition, the regions nearest to the frontal and parietal convolutions were very frequently involved."

In most instances, destruction of a portion of the pyramidal tract on one side gives rise to paralysis and spasm on the opposite side of the body only; but occasionally, spasm of the muscles on the same side as the lesion follows later in the history of the case. This is probably due to sclerosis appearing in the opposite lateral column, as Pitres has shown that it often does, and Mr. Sherrington has shown that it occurs in dogs which have had one pyramidal tract experimentally injured. But, although this is probably the cause of most cases of bilateral spasm from unilateral lesion, the possibility of the presence of a bilateral lesion must be borne in mind. In the *Revue de Médecine* for May 1883, Emile Demange records a case of the kind where a bilateral lesion was found in the brain *post mortem*.

Leaving affections of the cortical and subcortical portions of the pyramidal tract, let us pass to a consideration of the symptoms which accompany diseases of it as it traverses the basal ganglia, crura cerebri, pons Varolii and medulla oblongata.

It is needless, before an audience like the present, to recount cases of hemiplegia followed by rigidity, which result from destruction of the internal capsule by hemorrhage or softening. Such examples of disease are of common occurrence, and well known to all. I shall rather give an account of a series of cases which are less frequently met with, and which made a considerable impression upon me from two points of view:

1. Because they seem to throw considerable light on the rigidity and tetanic seizures which are observed in tumours of the cerebellum;
2. Because I failed to recognise the position of the disease in the brain during life, though a reconsideration of the symptoms leads me to think that others might have succeeded.

Case in which a Tubercular Mass occupied both Optic Thalami, producing Paralysis, Tumours, and Rigidity of the Limbs.—F. P., aged 4, was admitted into St. Thomas's Hospital under Mr. Sydney Jones, in August, 1878; and, on the 22nd of the following October, excision of the left astragalus was performed. The child did

very well, and was bright and playful until January 28th, 1879, when tremors of the left arm and leg were noticed. They were rhythmical to-and-fro movements, of short excursion, occurring only when the limbs were used voluntarily. There was very little loss of power, no affection of speech, deglutition, or respiration; and the tongue, face, and eyes all moved normally.

On March 5th, the child was still bright and lively, though pale; and it was noticed that the tremors were more marked on some days than on others. The left side of the face and the right hand had also begun to show similar tremors when used. The patient was sent out of the hospital, but was readmitted under Mr. Sydney Jones on July 8th, 1879, because the ankle and general condition were said to be worse. He had gone gradually down-hill, had become very drowsy, passed his evacuations in bed, and was more tremulous. On readmission, his face was pale, and a large dilated vein coursed transversely across the right upper eyelid. He was always asleep unless roused, and would even fall asleep while eating ravenously. His appetite was good, but he never asked for food. He complained of no pain, but appeared to have general hyperæsthesia of the skin. He was very irritable and peevish, crying out loudly when disturbed or contradicted. There were now rhythmical tremors of the right upper extremity, which were excited by voluntary efforts, and resembled those which used to affect the left side; the latter had ceased, and given place to rigid contraction. The elbow was flexed, and the hand clenched so tightly that the finger nails had produced ulcers in the palm. The hand was pronated, and the rigidity was constant. The mouth appeared natural when at rest, but when the child laughed or cried it was drawn up to the right. There was no squint; the pupils were large, and acted to light; the tongue was protruded straight, but was tremulous, and speech was slow. There was no deafness, but the ophthalmoscope revealed double optic neuritis.

There was no vomiting or dysphagia; the bowels were very confined, and the evacuations passed in bed. The pulse was 114, respiration 24, and the temperature varied between 99.4° Fahr., and 101° Fahr. The viscera appeared to be healthy. Until October 8th, the symptoms already described became worse. The right hand, arm, and leg trembled violently when used, and the left leg became perfectly rigid. The patient was very drowsy, and seemed to suffer from some headache; but he was never sick, and his appetite was good. His head was slightly retracted.

On October 8th, he seemed much worse; he vomited three times, took his food badly, and the temperature rose to 103.2° Fahr. Subsequently he became more and more drowsy, his right eyelid drooped, the pupils became unequal, and repeated attacks of general rigidity (sometimes accompanied with foaming at the mouth) supervened, and he died on October 15th.

On *post mortem* examination, I found an acute recent basilar meningitis. Besides this affection, which was, no doubt, the immediate cause of death, a large caseous tubercular mass was seen, involving both optic thalami, and almost completely obliterating the third ventricle (Fig. 9). The right optic thalamus was the more completely disorganised of the two. The specimen which I now show was hardened in alcohol; and, on section, it was then seen that on the left side the internal capsule, though pressed upon by the new growth, was clearly defined, and looked fairly natural, while that on the right was unrecognisable. The tubercular mass was confined to the optic thalami, and did not involve either nucleus of the corpus striatum. A very small caseous tubercle was also found on the internal aspect of the left occipital lobe, but the rest of the organs were healthy.

It is well known that diseases of the optic thalami and corpora striata are not accompanied by any such symptoms as were present in this case, unless the internal capsule is involved, and the paralysis, tremors, and rigidity of limbs, must be attributed to the invasion of this strand of fibres, proceeding from the motor convolutions; while the convulsions of the last week were the result of the recent meningitis. This case has been already recorded by Dr. Bristowe, in *Brain*, vol. vi, p. 167.

Case of Large Caseous Tubercle of the Pons Varolii, producing Persistent Rigidity of the Neck, Back, Arms, and Legs.—C. C., aged 2, was admitted into St. Thomas's Hospital, under the care of Dr. Ord, on October 4th, and died on November 7th, 1881. The mother was attending Brompton Hospital at the time, and she had lost one of her three children from consumption.

The patient was healthy until three months ago, when he began to have diarrhoea, and to lose flesh, but had no vomiting. A week before he came in, he had two convulsive fits. He had had pain in the head, and had been very fretful and drowsy.

On admission, the child lay in a dull and listless condition, taking but little notice of anything, and only crying when disturbed. The

head was drawn strongly backwards, and kept stiffly in that position. The back was likewise rigid. The arms were flexed, and the legs and feet extended, all being quite rigid. Patellar reflex was very brisk. There was no loss of sensation; deglutition appeared to be imperfectly performed. The pupils were dilated, but equal; and Mr. Nettleship pronounced the fundus of both eyes to be natural. Pulse 120. Loud rhonchi were heard over both lungs. The urine was free from albumen.

During the rest of his life, he became more and more drowsy, and had varying degrees of hyperæsthesia, but never loss of sensation. Rigidity of neck, trunk, and limbs continued without intermission, though it varied considerably in degree. Slight left internal strabismus, and partial paralysis of the right facial nerve came on; and deglutition was more and more difficult, the food running out at the corners of the mouth. The fundus of the eyes was repeatedly examined, even as late as November 6th, but no changes were found. The temperature, with very rare exceptions, was normal.

On *post mortem* examination, I found the cerebro-spinal fluid much increased, and the lateral ventricles moderately distended with serum. There was slight basal meningitis, but the hemispheres were free from tubercular disease. In the cerebellum and pons were large greenish tubercular masses of rather irregular outline, that in the latter being in the position marked in the figure. It occupied almost the entire extent of that division of the brain (Fig. 10), having only a thin

Fig. 10.—Diagram showing the position of a Tubercular Mass (c) as seen in a vertical section through the Pons (b). The Rim (e) represents the unaffected Nerve Substance.

sheet of white matter around it. Longitudinally, it extended the whole length of the pons, but seemed to stop short of the medulla oblongata. Unfortunately, no microscopic examination of the spinal cord was made. The lungs were thickly studded with miliary tubercles, but the other organs appeared to be healthy. The masses in the cerebellum were all situated superficially in the cortex, and did not extend deeply into the substance of the organ.

In this instance, again, the muscular rigidity must be referred to pressure exerted by the tumour upon the fibres of the pyramidal tracts in the pons; for tumours situated in the superficial parts of the cerebellar cortex are of pretty frequent occurrence, and are not found to give rise to any such symptoms. I shall presently relate a case in proof of this assertion.

Case in which a Tubercular Mass occupied the Aqueduct of Sylvius, and produced Tremors, Paralysis, and Spasmodic Contraction of Muscles.—E. B., aged 7, was admitted into St. Thomas's Hospital under the care of Dr. Bristowe, on August 2nd, 1882, and died on November 30th in the same year.

His family-history was unimportant, and the child himself had never suffered from fits, or from any serious illness, except from a blow on the head, which he received at the age of four, and which laid him up for a month. From this he perfectly recovered, and it was not until the end of May, 1882, that his present illness began. Having gone to bed quite well one night, he awoke next morning, throwing himself about; and he was found to have lost power in all his limbs, but most markedly in the right arm and leg, and he was unable to walk properly. He was said not to have vomited or lost consciousness. His speech was affected from the beginning, and his eyes about one week after the commencement of his illness. He was becoming weaker and thinner every day; and the tremors in his limbs, which were noticed at first, were becoming steadily worse. His bowels were confined, and he had lost control over his bladder; he was very

drowsy, but his memory was good. He had all along been free from headache, vomiting, and convulsions.

On admission, the child was found to be rather thin and drowsy. He lay apathetically in bed, but could be easily roused if spoken to. He had weakness in the arms and legs, and, when he walked, his legs seemed to drag. The most striking peculiarity in his condition was, however, the tremor with which all his movements were accompanied. The head, neck, jaws, trunk, arms, and legs, were all similarly affected. When he used his hands and arms the tremors in them were marked, of rather short excursion, sometimes in the direction of movement, and sometimes at right angles to it. The jaws trembled when he used them, and speech was slow and drawing. The tongue was protruded straight, and, when he smiled, the mouth was drawn somewhat to the right; hearing was normal. All the muscles of the eyeballs were more or less weakened. There was ptosis on both sides, and the internal rectus of the right eye was markedly affected. The superior and inferior recti on right were likewise weak, as were the muscles of the left eye, though not so weak as those on the opposite side. Both pupils acted well to light and to accommodation, the right being a little larger than the left; no anæsthesia was present. The patellar and plantar reflexes were normal, and there was no ankle-clonus. The temperature was natural, and all the organs, except the brain, appeared to be healthy. During the rest of his life the tremors gradually increased, paralysis became more marked, and attacks of spasmodic contraction of the muscles of the limbs and trunk occurred from time to time. On some occasions, the legs were noticed to remain for a while rigidly extended. The patellar and plantar reflexes became exaggerated. The patient grew more and more drowsy, and passed his evacuations in bed. The temperature presented only occasional slight elevations until November 26th, four days before death, when it rose to 101.2° Fahr.; and it subsequently reached 103.8° Fahr. on the 29th, the day before death.

Throughout the patient's illness there was a singular absence of headache, vomiting, and optic neuritis. Though frequently examined, the fundus of the eye presented no abnormal appearances until November 26th, when there was thought to be neuritis. After death, Dr. Edmunds found distinct microscopic evidences of this condition. Another curious point was, that the tremors were now and then either greatly diminished or absent for a day or two, and then returned with all their former intensity.

Fig. 11.—Drawing of a large Tubercular Mass (c) occupying the Aqueduct of Sylvius, and by its pressure producing a Cup-like depression in the Pons (b). The Section is Vertical and Longitudinal.

Necropsy.—The body was much emaciated, and rigor mortis was present only in the lower extremities. The spleen and lungs contained a few tubercles, and those in the spleen were caseous. All the other organs were healthy, with the exception of the brain. Its surface was sticky, and the veins were gorged with blood. At the base, in the interpeduncular space, the membranes were opaque, and there was fluid in the optic sheath. Miliary tubercles were present in the Sylvian fissures, and the ventricles were moderately distended. There was a large caseous mass in the region of the corpora quadrigemina, which was round in shape, and as large as a medium-sized marble,

and which is well displayed in the preparation before you (Fig. 11). It lay as if wedged in the aqueduct of Sylvius, which was enormously distended. On the upper surface of the tumour were the thinned and flattened corpora quadrigemina, while its lower rounded margin rested upon the upper surface of the pons and crura cerebri. Here it occupied a cup-like cavity, which its downward pressure had produced. No other important structures were injured by the tumour.

In this case, the usual phenomena of pressure upon the fibres of the pyramidal tract are again well represented. Paralysis and tremors occurred first; then attacks of rigidity. Had the child lived longer, and the growth of the tubercular mass progressed, the pressure exerted by it upon the pons and crura cerebri would, no doubt, have produced the final stage of persistent rigidity. The recent tubercular meningitis cannot be said to have produced the tetanic seizures, as the latter occurred months before the patient's death. This case and the next which I shall relate have already been published by Dr. Bristowe in *Brain*, vol. vi, p. 167.

Case of Tubercular Mass occupying the Left Lobe of the Cerebellum, and pressing upon and producing Softening of the Pons and Medulla: Persistent Rigidity of Limbs.—A. G. J., aged 4, was admitted into St. Thomas's Hospital, under the care of Dr. Bristowe, on December 30th, 1878, and died on August 6th, 1879. He had been a healthy child until three months previously, when he was ill with measles and whooping-cough. He began to have fits, which occurred about once a day at first, and then twice a day. He had also been suffering from severe pain in his head, occasional vomiting, and for about two months his mother had noticed that he squinted. The father was said to be asthmatic, and the mother had a weak chest, but their two other children were healthy.

On admission, the patient seemed to be a sensible child, but quite blind. The pupils were widely and equally dilated, and contracted slightly to light. When lying in bed, he could move his arms and legs, but the former were tremulous and ataxic. When placed upon the ground, the patient laughed hysterically, but could not stand, though he could move his legs forwards when supported. The limbs appeared to be equally affected on the two sides. The mouth dropped slightly on the right side, and the tongue was protruded to the right.

The pulse was 140, and regular; respirations were easy. The patient passed all his evacuations in bed. There was well marked optic neuritis in both eyes, the outline of the discs being irregular and blurred, the veins large, the arteries small, and the whole presenting a streaky appearance. Between the time of admission and his death the patient had many fits, in which he was unconscious; the legs remaining rigidly extended, and the arms being flexed, and then extended several times in succession. A certain amount of left facial paralysis developed, so that the patient could not close the eye of that side, and the left external rectus was partially paralysed. His arms became more tremulous, the right being more affected than the left; his speech was very drawing. He remained for long fairly intelligent, had no deafness, and was not much troubled with headache or vomiting. From February onwards the arms and legs became persistently rigid, although the degree of rigidity varied. Finally, the child became emaciated to the last degree, and lay perfectly still, taking food well when it was offered him, but showing very few signs of life. He died on August 6th.

Post Mortem Examination.—There was little evidence of important pathological changes in any of the viscera except the brain. The head was very large, all the sutures were separated, and the bones were movable. There was no subarachnoid fluid, but the ventricles were greatly distended, and the substance of the hemispheres reduced to a thin, soft sheet of nerve-substance. The left lobe of the cerebellum was entirely occupied by a firm tumour, of the size of a tangerine orange, yellowish-green in colour, semi-transparent, and consisting of closely placed, concentric layers. The relics of cerebellar tissue remaining on its surface were soft, as was the neighbouring part of the posterior cerebral lobe. The tumour was adherent to the tentorium cerebelli, and the pons and medulla were pressed upon by it, and somewhat softened. The right lobe of the cerebellum was healthy. Another small tubercular mass was found in the grey matter on the orbital surface of the right frontal lobe.

In this case, again, persistent rigidity was the result of pressure which the tumour exercised on the medulla oblongata and the pyramidal tracts which run in it. For it will be shown presently that, without such pressure, tumours of the cerebellum do not produce such phenomena, and that distension of the ventricles with fluid is not sufficient to explain them.

These four cases appear to me to be of great interest as representing diseases affecting a certain portion of the brain, and capable of being localised during life. The region referred to extends from the basal

ganglia to the medulla oblongata, commencing at that spot where the pyramidal tracts begin to converge as they pass between the central ganglia, and occupying further on the pons and medulla oblongata, where the pyramidal tracts lie in close contact. If the motor tracts be affected simultaneously, or quickly one after the other, in the way about to be mentioned, the disease is likely to be situated between the basal ganglia above and the medulla below, and is, in all probability, a tumour. The earliest symptoms referable to the pyramidal tract are tremors; then come paralysis and rigid contraction, which is finally permanent; and besides these, there may occur from time to time attacks of tetanic spasms, affecting most of the muscles of the body. The phenomena of chronic spasm are due to gradual pressure on the motor tracts, proceeding from the central convolutions of the brain; and the fact that both sides of the body are attacked, indicates that the disease is situated at a part where the pyramidal tracts are in close proximity to each other. If the case be seen early, probably all these symptoms, tremors, paralysis, and rigid contraction, will occur before the end; but if the patient do not come under observation until late, only some of them may be observed. Thus, in the case where the optic thalami were gradually invaded, and in the case of cerebellar tumour pressing from above on the medulla, all occurred; whereas, in the case of the large tubercular mass almost completely occupying the pons, only the later stage of persistent rigidity was seen. In determining the exact seat of the tumour, whether in the optic thalami, corpora striata, pons, or cerebellum, we must consider the other symptoms referable to the special nerves involved, which arise at different levels along the base of the brain, and also whether there are, or have been, symptoms which suggest cerebellar disease. Moreover, optic neuritis is very constant, and early in its appearance in cerebellar disease, and often late when the tumour is higher up.

I would not have it understood that all tumours in these positions give rise to the phenomena described, for soft succulent ones often do not; but if the symptoms I have pictured be present, the disease is probably a hard slowly growing tumour in the area mentioned. In all the cases just related, the tumour was a large tubercular mass; and the early stage of such tumours is shown in a specimen on the table, which was taken from the body of a baby who presented no obvious symptoms of cerebral disease. A caseous nodule can be seen (Fig. 12) occupying the lower third of the external segment of the lentiform nucleus. In a drawing of a microscopic section of this mass, a large vessel is seen, and close by the vessel lies a colony of well stained tubercle-bacilli. (Fig. 13.)

But, interesting as this series of cases is from the point of view already considered, they are still more so in their bearing upon the question of the functions of the cerebellum, and its relation to muscular spasm. In the *Lancet* for 1880, vol. i, p. 122, Dr. Hughlings Jackson has written "On Tumours of the Cerebellum," and speaks of "cerebellar rigidity." "I suppose," says he, "the cerebellar rigidity to be owing to unantagonised cerebral influx (rigidity in hemiplegia being owing to unantagonised cerebellar influx), and the tetanus-like seizures to be owing to cerebellar discharges analogous to these cerebral discharges which produce unilaterally beginning (epileptiform) convulsions. But there is the obvious objection that the two symptoms, with the cerebellar disease, may be owing to interference with the corpora quadrigemina (electrical excitation of which produces tetanus-like states), or to interference with the medulla oblongata."

Further on, in the same volume of the *Lancet*, p. 522, Stephen Mackenzie has a paper on the same subject, in which he expresses the opinion, that "the reeling gait and tonic spasms are both expressions of disorder of the locomotory apparatus, of which the cerebellum is the chief governing centre." In comparing the tetanus-like seizures with tetanus, he goes so far as to say, "the range of the two is so much alike as to afford the strongest reasons for the belief that tetanus is an affection of the cerebellum."

Now, if the same rigid contractions and the same tetanic seizures occur in disease of other parts at the base of the brain, they cannot be held to be characteristic of cerebellar disease.

Hughlings Jackson says: "This universal rigidity, as a condition arising from cerebellar disease, I have seen only when the tumour has been in the middle lobe," and again: "In the case of extensive rigidity and tetanus-like seizures, the tumour has been of the middle lobe, and has been large." These remarks really furnish a clue to the matter. I would put the case thus: a tumour of the cerebellum does not produce contractures and tetanic seizures, unless it be in such a position and of such a size as to produce pressure on the posterior Varolii or medulla oblongata.

The following case illustrates this point well, and shows that tumours may be present in the lateral lobes, or in the central lobe,

and that great distension of the lateral ventricles may exist, without these muscular contractions making their appearance.

Case of Tubercular Masses, both in the Central and Lateral Lobes of the Cerebellum, unaccompanied by Tetanic Spasms.—E. C., aged 10 years, was admitted into St. Thomas's Hospital, under the care of Dr. Bristowe, on September 13th, and died on December 5th, 1877. At the age of 7, the patient had a fit, lasting for about two hours, in which her eyes were fixed and her hands clenched. Ever since that time she had been becoming blind. The only other illness she had had was "low fever," two years before admission. About five months before coming to St. Thomas's, she had been suddenly seized with very severe pain in the head and vomiting, which lasted, off and on, for a day and a half. These attacks recurred from time to time for five weeks, during which her sight became worse and worse, but she never had convulsions. On examination, it was found that she could not distinguish light from darkness, and that she had atrophy of the optic discs. She had no deafness, and was very intelligent, and able to give a great deal of information about her illness. Her head was somewhat hydrocephalic in shape, and the forehead prominent. She kept it fixed, and said it hurt her to let it drop backwards. When seated, the legs appeared to be quite strong, and it was impossible to detect any weakness in them when one tried to flex or extend them against her will. When held by the hands, she could walk pretty well, but when she was left to stand alone, she quickly lost her balance. The same occurred when she tried to walk, and she seemed to have considerable inco-ordination of the legs. There was a tendency to fall to the right side, though she was said previously to have usually fallen to the left. When standing up and made to lean a little backwards, she did not fall, but ran backwards.

During the rest of her life she had frequent attacks of severe pain in the head, accompanied by vomiting, and a feeling of great giddiness. Her temperature remained normal, and she had no tetanic spasms or contractions. She was found dead in bed on the morning of December 5th. At the necropsy, there was no evidence of meningitis, but the ventricles of the brain were distended with clear fluid, twelve ounces being removed and measured. The convolutions were flattened, and the whole extent of them collapsed when the ventricles were emptied. The optic nerves were markedly atrophied. The lateral lobes of the cerebellum were both very soft and swollen, and the left was adherent to the dura mater. There were several tumours, both in the central and in the left lobe, which appeared to be tubercular masses with the centres broken down. Their size varied from that of a pea to that of a medium sized marble; their circumference consisted of a pale, rosy grey, semi-translucent, hard tissue, and their centres of a puriform fluid. The other organs were healthy.

In the class of cases which I have been discussing, tremors and paralysis are probably due to slight interference with the functions of the fibres of the pyramidal tracts, persistent rigidity to still greater interference, while the attacks of tetanic spasms may resemble the convulsions which are observed in connection with cortical tumours of the brain. In the case of cerebral tumours, the pressure is exerted upon the cortical grey matter; in the cerebellar cases, upon the grey matter in the floor of the fourth ventricle, and not upon that of the cerebellum itself, for I have seen cases in which the central lobe of the cerebellum has been completely destroyed by tumours, but in which no pressure was exerted upon the medulla oblongata, and no rigidity was observed during life.

It is interesting to note, in relation to this question, that Nothnagel has found that irritation of these parts is particularly liable to produce convulsions; so much so, that he has supposed a special convulsive centre (*Krampfcentrum*) to exist in the pons, which is readily brought into action, either directly or reflexly, by stimulation of a spot in the fourth ventricle. I have said that the tetanic spasms "may" be due to pressure on the grey matter of the floor of the fourth ventricle, because they are capable of another explanation; they may, in fact, be the result of varying pressure upon the pyramidal tracts. In the next lecture, I shall relate a case where a tumour pressing upon the cord from without produced similar spasmodic attacks; and they are also seen in other diseases affecting the lateral columns of the cord.

Foster, in his work on *Physiology*, says: "We must consider the cerebellum as an important organ of co-ordination, though we are unable at present to define its functions more exactly." This probably represents our knowledge of the functions of the cerebellum, and it is in keeping with pathological experience; and I do not think we are justified in considering that disease of it produces spasmodic contraction of muscles, except indirectly, by pressure upon underlying structures.

ABSTRACTS OF THREE LECTURES

ON

THE BRAIN-MECHANISM OF SIGHT AND SMELL.

Delivered at the Royal College of Surgeons.

By ALEXANDER HILL, M.D., M.R.C.S.,

Hunterian Professor to the College; Fellow of Downing College; Demonstrator of Anatomy to the University of Cambridge.

LECTURE III.—THE RELATION OF THE OLFACTORY AND OPTIC NERVES TO THE CENTRAL GREY TUBE AND CEREBRAL CORTEX.

In the scheme of the central nervous system submitted in the two preceding lectures, the neural axis was represented as exhibiting an uniform plan of structure throughout its whole extent. The large cells of which the peripheral nerves are the axis-cylinder processes, accumulate at the point of exit of these nerves; in the same way, the small-celled tissue with which the sensory nerves, after breaking up in the gelatinous substance of Rolando, terminate, exhibits a similar segmental arrangement. The key to the constitution of the system is to be found in its metameric division. Most anatomists are willing to concede this proposition for the whole of the neural axis behind the optic thalamus. At this level, however, a complete alteration in the plan of the system is supposed to occur. On the one hand, two nerves, the olfactory and optic, are left without primary centres in the neural tube; on the other hand, a mass of small-celled grey matter, the optic thalamus, directly continuous with that surrounding the aqueduct of Sylvius, and developed in the same relation to the primitive neural canal, as the rest of the aesthesodic portion of the central tube, is excluded from it. No motor nerves arising from the mid-brain, small-celled plexus only should be formed from its wall. It is impossible to determine the amount of this plexus needed to constitute the primary centres of these two large nerves, for there is no evidence to show that a sensory nerve is connected with only one cell of the neural axis; rather does it seem that the sensory fibre, after its division, is brought into connection with as many cells of the plexus as may be needed for the distribution of its impulses to their appropriate motor apparatus. It is not on account of its structure, its connections, or its mode of growth, that the optic thalamus has been severed from the rest of the grey tube, but because physiologists require centres intermediate between the cortex and the cord, to carry out a certain class of reflex actions. The optic thalamus is therefore allied with the corpus striatum to form a subordinate *bureau*, in which the messages from the front may be submitted to an official censorship before being forwarded to the cortex, and the mandates of the will be put into proper form for transmission to the seat of action. The two ganglia so allied are developed from different cerebral vesicles, and have very different phylogenetic records; but this is overlooked in the desire to satisfy what the lecturer can only term the pernicious doctrine of "centres." Not physiologists only, but also anatomists, have come to regard the nervous system as consisting of a series of centres of ascending grades of authority; societies of cells, differing from one another in structure, but bound together for a common object. It is almost a revelation to find, after submitting the central nervous system to section-cutter and microscope, that, from filum terminale to lamina terminalis, no collection of cells, which could be regarded as a centre in this sense, is to be found. There is no reason for separating the optic thalamus from the remainder of the central grey tube.

Turning now to the two nerves which are left without primary centres, the lecturer explained, with the aid of diagrams, how it is easily to be seen (at any rate, in the brains of ungulates) that the optic nerve grasps the posterior end of the thalamus very much in the same way as the auditory nerve its aesthesodic portion of the medulla oblongata.

The route by which the olfactory nerve reaches its primary centre in the anterior end of the thalamus is a circuitous one. The fibres from the olfactory bulb stream centralwards in three divisions; of these, the inner is inconsiderable. The middle, a thick bundle, grooves the head of the nucleus caudatus as it curves inwards to the anterior commissure. This commissure is not wholly made up of ol-