Lectures on the diagnosis of diseases of the brain, delivered at University College Hospital.

ABSTRACT OF RULES

LECTURES

ON THE

DIAGNOSIS OF

DISEASES OF THE BRAIN

DELIVERED AT UNIVERSITY COLLEGE HOSPITAL

BY

W. R. GOWERS, M.D., F.R.C.P.

ASSISTANT-PROFESSOR OF CLINICAL MEDICINE IN UNIVERSITY COLLEGE
PHYSICIAN TO UNIVERSITY COLLEGE HOSPITAL AND TO THE NATIONAL HOSPITAL FOR THE
PARALYSED AND EPILEPTIC

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PREFACE.

MANY readers of my "Diagnosis of Diseases of the Spinal Cord" have asked me for a book, similar in scope and method, dealing with the symptoms and methods of diagnosis of Diseases of the Brain. In response to that request, I publish these Lectures, delivered at University College Hospital.

I have left the Lectures very much in the form in which they were delivered, believing that thus they will be most useful. The account of the diagnosis of the nature of the lesion, contained in the concluding Lectures, is intended rather to impress on the student the methods of diagnosis and the most important distinctions between the various diseases, than to furnish an exhaustive description of these distinctions. At the same time, I believe that those given will enable a diagnosis to be made in the majority of the cases that the student or practitioner is likely to encounter.

QUEEN ANNE STREET, LONDON,
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THE

DIAGNOSIS OF DISEASES OF THE BRAIN

LECTURE I.

INTRODUCTION—MEDICAL ANATOMY OF THE BRAIN; CORTEX, MOTOR PATH, SENSORY PATH.

Gentlemen,—The subject of the diagnosis of diseases of the brain certainly transcends in complexity, and perhaps exceeds in interest, all other problems in practical medicine. In nerve-elements the involution of energy reaches the highest degree known to us, and in the chief organ of the nervous system these elements, vast in number, various in character, are arranged in what seems to us extreme complexity. The number of the nerve-cells of the brain is not known, and if it were, our minds would be powerless to grasp the extent of the array. The cortex alone has been estimated to contain eight hundred millions of cells. Of all the elements that compose the brain, not one is isolated. Each is connected with others, and their anatomical connection subserves a corresponding functional association. A change in the functional state of one involves a change in the functional state of others; and change is constant, rest unknown.

Complex as the arrangement of the elements of the brain appears to us to be, it may be found, with fuller knowledge, that the principles of the plan are comparatively simple. The variety in form of the nerve-elements is small, if we consider how numerous they are. The fibres present little variation. The cells, even when the element of size is taken into con-

sideration, may probably be classed in not more than a dozen varieties. It is the arrangement and connection of these elements that constitutes the complexity of structure of the brain, and subserves its complexity of function. At the same time we must remember that the varieties of form give us no clue to the differences that may exist among the elements of which nervous tissues consist. Neither microscopical examination nor chemical analysis can penetrate beyond the coarsest outlines of the constitution of living matter. It is probably within the facts to assert that each nerve-cell contains as many material atoms as the brain contains nerve-cells. Even the molecules, formed by the grouping of these atoms, are beyond the reach of any means of scrutiny we possess, or of which we have, at present, any promise. The fundamental diversity that must exist, where we can discern only uniformity, is shown to us by the action of poisons, and in the effects of disease. One poison acts on one part of the nervous system, and leaves uninfluenced another part, which a second poison affects alone. We are accustomed to explain this by the difference in the chemical constitution of the poison; but it is evidence of an equal difference in the nerve-elements, that are, as we say, "selected" by it. The selection is no matter of arbitrary choice. It is the result of the nature of the nerve-elements themselves; the expression of differences between them, revealed by such effects alone. This consideration renders many effects of disease more intelligible, though it may not lessen their mystery.

Certain leading facts regarding the structure of the brain must be clearly recognized, if we would understand what is known of the principles of the diagnosis of its diseases. Our present knowledge of the cerebral structure is at once far more than we can use, and far less than we need. The researches of the last quarter of a century—of Lockhart Clarke, Broadbent, and others, in this country; of Stilling, Meynert, Wernicke, Flechsig, among the Germans; of Luys, Foville, and others, in France—have brought to light an

immense number of facts, and have built up a large mass of knowledge, much of which has at present no practical application.

But if we attempt to select from the facts described by different investigators those that are of chief importance, we are at once met by a fresh difficulty. Many of the conclusions reached by different investigators do not correspond,—are even contradictory. This is not surprising. The interlacing tracts of fibres baffle the scalpel, and even the microscope, when we attempt to trace their course. What, then, is to be our guide when investigators disagree? Fortunately we are not dependent only on simple dissection, or even microscopical examination. The selective action of disease affords invaluable help in the study of the structure of the nerve-centres. It was long ago discovered (by Waller) that if nerve-fibres are destroyed at a given point, they undergo degeneration beyond the lesion, and this degeneration extends along them as far as they continue simple fibres. The degeneration is only in one direction, and that is the direction of functional conduction; on the side of the lesion from which the fibre conducts there is no degeneration. By means of this "secondary degeneration" the course of some tracts of fibres may be traced with precision through the brain. Moreover, we have a second guide. A distinguished German investigator, Flechsig, has availed himself of the fact that different tracts of nerve-fibres in the brain acquire their white substance at different stages of fœtal and infantile life. He has shown, by a remarkable series of investigations, that we may learn as much of the course of fibres by studying them in their birth as in their death—in their development as in their decay. His discoveries have thrown new light on many difficult problems, and supply a trustworthy guide in discovering where the truth lies in other and contradictory descriptions.

The experimental researches on the functions of the brain, chiefly of Hitzig and Munk in Germany, and Ferrier in this country, have also given us vast help in diagnosis. Here also, however, we meet with con-

tradictions; and here also more has been ascertained than we can at present apply. Our guide in this department must be the facts of clinical and pathological observation. We must beware of applying wholesale to the human brain the conclusions derived from experiments on animals. The latter are of value to us only as indications for observation on man, and by enabling us to give a fuller interpretation to the facts we learn by our study of disease during life and after death. Some of the experimental facts have at present received no confirmation, and on some points we especially need information, which, from their nature, experiment cannot give.

Before we enter on those details regarding the structure and functions of the brain that are of chief importance for diagnosis, I must remind you of certain important elementary facts. We speak of nerve-cells and nerve-fibres as if they were merely connected structures, essentially distinct. They are not really so. The axis-cylinder of each nerve-fibre is the prolonged process of a nerve-cell, sharing all changes of nutrition that the nerve-cell undergoes, suffering with it when the cell is damaged. This is the secret of the secondary degeneration. If a fibre, or part of a fibre, is cut off from its parent cell, it degenerates; the part still in connection with the cell does not degenerate. If the cell is destroyed, the whole fibre perishes. Although I have said that every fibre is a nerve-cell process, I need hardly tell you that the fact is not proved. It never can be proved by observation. But the relation can be observed of some cells in various parts of the nervous system; the contrary has never been observed; and we may therefore infer, with considerable probability, that the fact is true of all nerve-fibres. We do not know whether any nerve-fibres unite directly the undivided processes of two nerve-cells. It is highly probable that, as a rule, they do not, because we usually find that only one process of a cell becomes directly an axis-cylinder; the other processes divide and ramify in a branching network of the finest nerve-fibrillæ. This structure is probably

intermediate between adjacent nerve-cells, and in it the nerve-fibres may end, that are the prolonged processes of other and sometimes distant nerve-cells.

Another preliminary consideration is of a different nature. It is necessary, in speaking of the functions of the brain, to use the term "centre." Remember that the word is employed in its physiological sense; not in a geometrical, or even in a topographical, sense. It indicates a combination of nerve-cells subserving a given function. Note, first, that this function may not be the only one subserved by the group of nerve-cells. In many groups the nerve-cells are very numerous; they are connected with each other by a ramifying plexus of fibrillæ, and are connected with other and distant groups of cells by nerve-fibres. Such a collection of cells may contain many centres, since only some cells are probably in action at the same time. The possible functional combinations in such a group are almost infinite, since the variations involve not only the number of cells in action, but the relative degree of their action. Only some of these are usually, perhaps ever, in functional activity. Lines of various resistance exist by which the functional combinations are determined. Education is largely the establishment of these lines of least resistance. Habit depends upon them. Although the finest, most complex mechanism of human contrivance is rude compared with the simplest arrangement of the brain, I may illustrate this variation of function by an object with which you are all familiar. Wherever large buildings are being erected, or extensive excavations made, you will see a steam crane. This machine, by a change in the relation of the different parts of the complex mechanism, may be made either to draw up a weight, to swing itself round, or to travel along a line of rails. The motive force is the same —the machinery is essentially the same; the difference in result depends on certain parts of the machinery being in or out of "gear." In the one machine are functional centres for progression, rotation, and turning a windlass.

Not only may one part of the brain contain many centres, but a single functional centre may consist of nerve-elements

that are anatomically distant—even situated in different hemispheres. Cells may act together when the nerve-fibre that connects them is many inches long, as perfectly as if they are only a hundredth of an inch apart, just as the needles of two galvanometers in the same circuit are deflected at the same moment, judged by ordinary standards, whether they are distant a foot or a mile.

One more preliminary consideration. A familiar line asserts that "things are not what they seem," and although the statement is scarcely of the universal application that its form suggests, it is one that should not be forgotten when we study the arrangement of the nervous system. The course of nerve-fibres is often very different from that suggested by the course of the nerves in which they run, and the arrangement and connection of the elements of the brain is often not the same as that suggested by the coarse morphological relations of its parts. Nerve-fibres frequently take a course not only very different from that which might be anticipated, but mysteriously tortuous,—I was going to say unnecessarily tortuous. Doubtless these irregularities have had their necessity; but this lies in the dim past of development, beyond our range of vision. The course of the fibres of the cranial nerves affords many illustrations of these anomalies. A comparatively simple instance is presented by the spinal part of the spinal accessory nerve, which arises from the same grey matter as the cervical nerves, is distributed to the same muscles, but has a course that takes it just within the entrance to the cranial cavity, although it has no business there, as far as we can see, to justify its ascent. The course of the fibres that subserve taste is a more elaborate instance of the same thing, and it has caused difficulties from which neither anatomical nor physiological investigation has yet cleared our knowledge.

I may safely assume, gentlemen, that you are familiar with the chief facts in the topographical anatomy of the brain, and amongst them with the names of the convolutions. But, as I shall have frequently to refer to these, I put before

you a diagram on which the fissures and convolutions are shown and designated.

I need only add one fact to those that are indicated on the diagrams. There is, as we shall see, an important difference

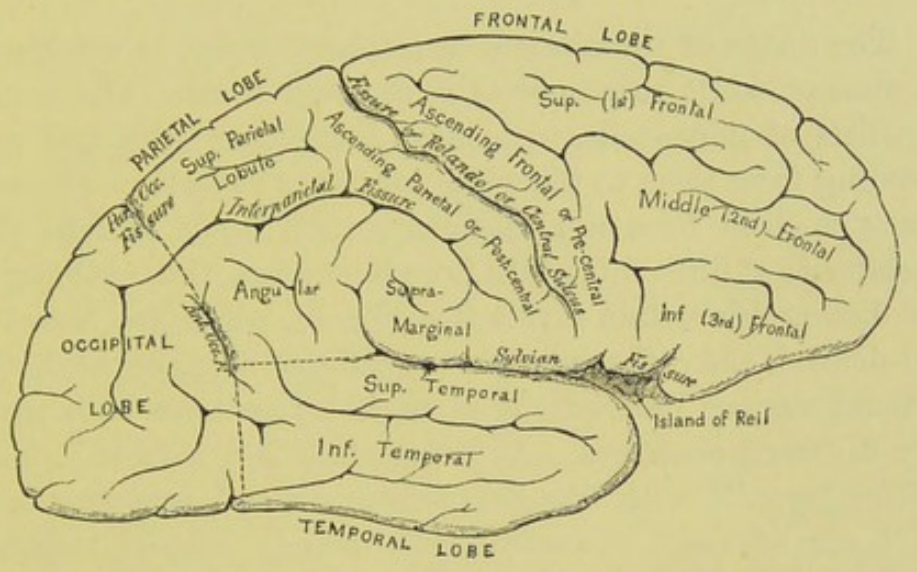

FIG. 1.—DIAGRAM OF THE CONVOLUTIONS AND FISSURES ON THE OUTER SURFACE OF THE RIGHT HEMISPHERE.

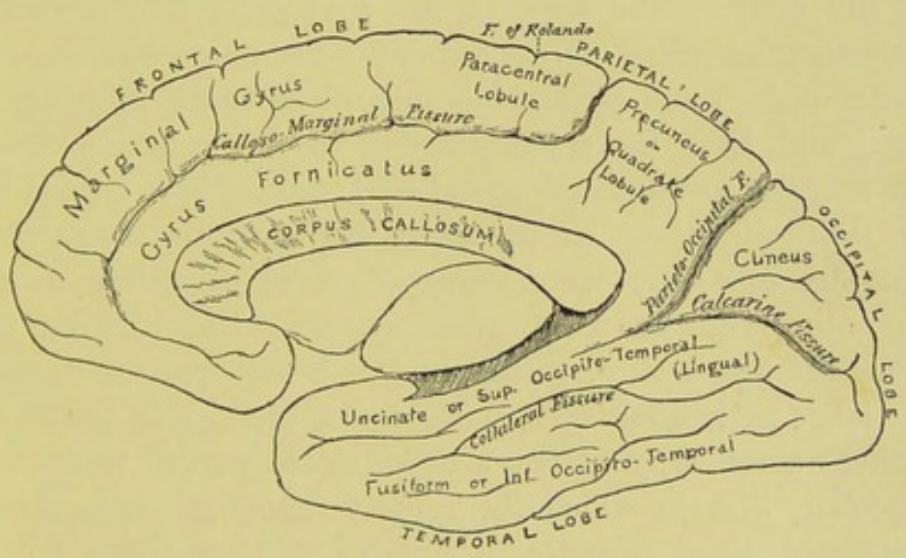

FIG. 2.—DIAGRAM OF THE CONVOLUTIONS AND FISSURES ON THE INNER (MEDIAL) SURFACE OF THE RIGHT HEMISPHERE.

in function between the ascending frontal and the three other frontal convolutions. Hence it is often convenient to speak of the latter without the former. We do so by terming the three antero-posterior convolutions, and corresponding inner

surface, the "prefrontal lobe." Thus we mean by this term the portion of the brain anterior to the ascending frontal convolution. It is, like many others, an inaccurate word, but it has become current, and is convenient—indeed, necessary.

The cortex of the cerebral hemisphere covers, as you know, a mass of white substance—the centrum ovale. The cortex consists of nerve-cells, with fibres connecting them, and proceeding from them to the white substance beneath. The cells vary much in form and size, and certain kinds of cells preponderate at different depths from the surface, thus enabling us to distinguish certain layers (see Fig. 3), which are, however, ill-defined, and differ somewhat in different parts of the cortex. Hence various descriptions of these layers have been given by different observers. At the surface is a narrow "*superficial* layer,"* with few nerve-elements, and beneath this is a layer of *small pyramidal cells* densely massed together. Below this is a thicker layer of *large pyramidal cells*, which are larger in the deeper than in the more superficial part of the layer. The apex of the pyramidal cells is turned towards the surface. Next comes a layer which contains still larger cells, some pyramidal, and others more irregular in form, and, from the resemblance of the latter to the cells of the anterior cornu of the spinal cord, this layer is termed the *ganglion-cell layer*. In the motor region of the cortex these cells are especially large, many of them exceeding in size any other cells in the nervous system. But between this layer and the last, or rather in the lowest part of the last, the large pyramidal layer, small round or angular bodies are rather numerous; and over a considerable part of the cortex, especially that in which the sensory functions seem chiefly developed, these increase in number, and constitute a well-marked *granule layer*. Beneath the ganglion-cell layer, and next the white substance of the hemisphere, is a layer in which *fusiform cells* preponderate.

The white substance consists of fibres passing in various directions. Some pass from one part of the cortex to another

* The italics indicate the names commonly given to the layers.

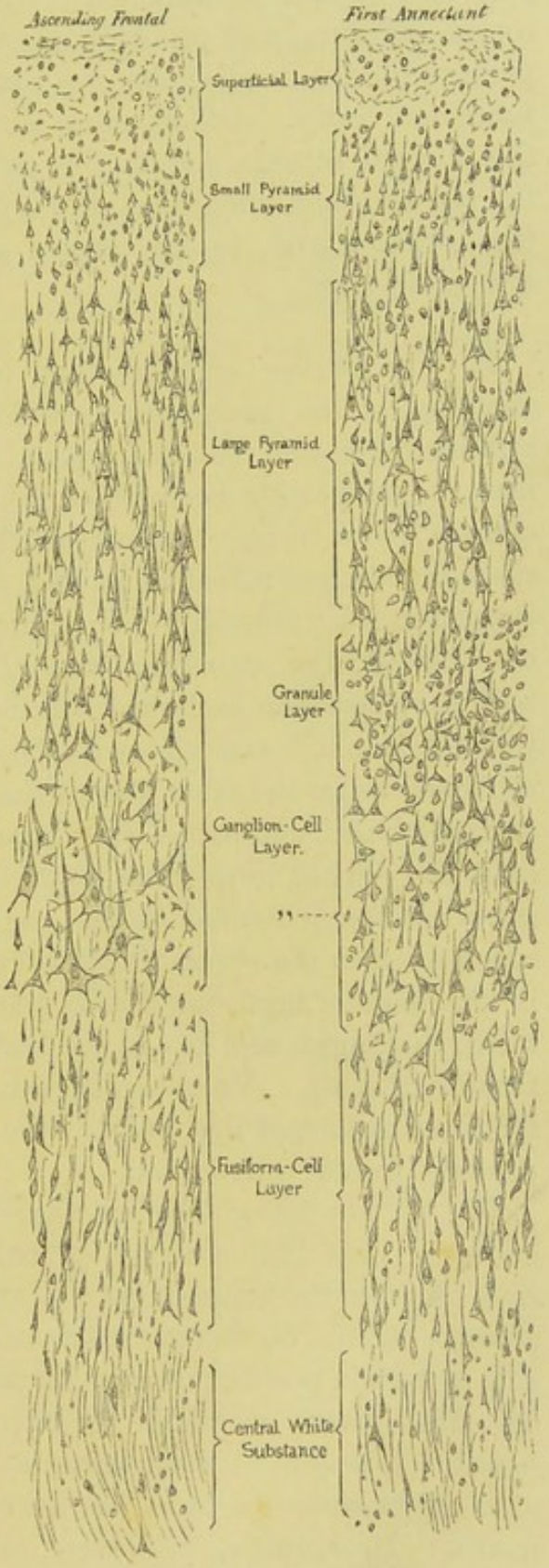

FIG. 3.—DIAGRAMMATIC SKETCH OF THE LAYERS OF THE CORTEX CEREBRI.

The drawings were made from sections of the ascending frontal and first annectant convolutions (the latter near the extremity of the parieto-occipital fissure). The sections were kindly furnished me, as representative of the so-called motor and sensory types, by Dr. Bevan Lewis, whose important investigations into the structure of the cortex are well known. In addition to the facts mentioned in the text, certain others may be noted. No layer is composed exclusively of one kind of cell; most forms are met with in each layer, but some are scarce and others abundant. Almost all the pyramidal cells have their apex turned towards the surface of the brain. The granule layer of the sensory type is formed at the expense of the two adjacent layers, the large pyramid layer and the ganglion-cell layer, and these two layers in the motor type merge one into the other to an even greater degree than the other layers. In the sensory type the very large ganglion cells of the motor type are absent. In the white substance, nuclear bodies lie among the fibres, and near the grey cortex isolated cells occur, similar to those in the grey matter, becoming few as we pass more deeply into the white substance. Many fibres run through the grey substance towards the surface, and they are, to some extent, aggregated into bundles, partially separating the nerve-cells into columns. To avoid confusion these fibres have not been shown in the diagram.

part in the same hemisphere. Others pass inwards to the corpus callosum, and probably, through these, corresponding parts of the two hemispheres are connected, and brought into functional unity. Others, from almost all parts, converge to the internal capsule, and to the optic thalamus; while from the posterior and under part, fibres pass to the crus, and from the posterior part, to the corpora geniculata, quadrigemina, and optic tract.

In our survey of what may be termed the functional anatomy of the brain—the consideration of the structural arrangements that subserve special functions—we may pass next to the parts concerned in voluntary motion. As you already know, in only one part of the cortex can we trace a special relation to voluntary motion. This part consists of the two (so-called "central") convolutions that bound the fissure of Rolando, the ascending frontal and ascending parietal, with the expansion backwards of the latter in the "superior parietal lobule," and also with the medial aspect of these convolutions on the inner side of the hemisphere—the "paracentral lobule," and part of the "precuneus" (Fig. 2). As you have also learned in the class of physiology, voluntary motion is not uniformly related to this region. The leg is chiefly represented in the upper third of these convolutions, the arm in the middle third, the face in the upper part of the lower third, the tongue and lips in the lowest part of the ascending frontal (Fig. 4). Voluntary speech is related to this centre for the tongue and lips, and to the adjacent part of the third (lowest) frontal convolution.

We do not know whether there is any sharp limitation between these limb-centres; probably there is not, at any rate between the centres for the arm and for the leg. In animals certain movements have been found specially excitable at certain points, and representations of the human brain are often given, to which these centres have been transferred. But clinical facts have not at present confirmed in man the existence of these limited centres for certain movements. In animals, moreover, centres are found on the

posterior parts of the first and second frontal convolutions. These are not only unconfirmed so far as the human brain is concerned, but the balance of evidence is against their existence.*

This region of the cerebral cortex is certainly motor, but it is certainly also not exclusively motor. Its destruction by disease causes persistent paralysis, corresponding in distribution to the part destroyed; irritation of it causes convulsions, that begin in the limb corresponding to the part irritated.

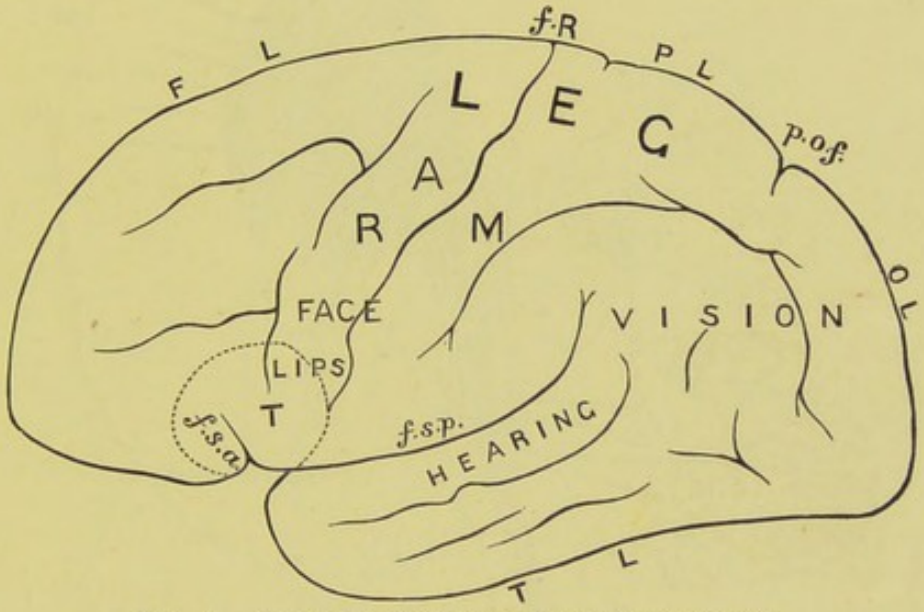

FIG. 4.—CORTICAL CENTRES: LEFT HEMISPHERE.

The position of the limb-centres is indicated by the letters composing their names. T, tongue-centre. The dotted line encloses the motor speech-centre. F L, frontal lobe; P L, parietal lobe; O L, occipital lobe; T L, temporal lobe; *f.* R, fissure of Rolando; *f.s.a.*, anterior limb of the fissure of Sylvius; *f.s.p.*, posterior limb of the same fissure; *p.o.f.*, parieto-occipital fissure.

But destruction of these parts causes also some loss of sensation, chiefly in the extremity of the limb most paralysed, and accompanied by an inability to recognize the position of the extremity, hand or foot—an inability which may be out of all proportion to the loss of cutaneous sensibility, and even, strangely enough, may exist alone. Of this peculiar

* Schäfer and Horsley find, in monkeys, centres for the trunk muscles in the highest part of the motor region (especially on the inner aspect), and in the posterior part of the first frontal convolution. Ferrier found centres for the movement of the head and eyes on the first and second frontal.

loss I shall have more to say hereafter. Another fact of similar significance is that convulsions produced by disease in this region often commence by a sensory "aura."

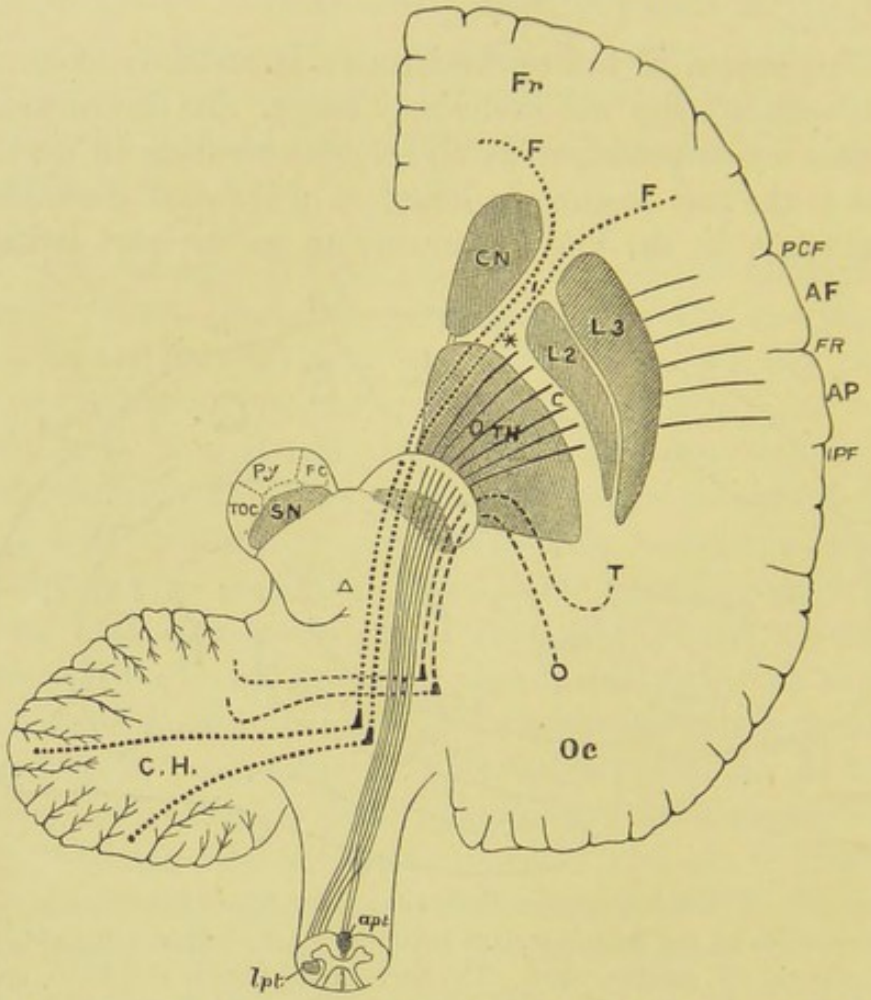

FIG. 5.—DIAGRAM OF THE COURSE OF THE MOTOR TRACT AS SHOWN IN A DIAGRAMMATIC HORIZONTAL SECTION THROUGH THE CEREBRAL HEMISPHERE, PONS, AND MEDULLA.

Fr, frontal lobe ; Oc, occipital lobe ; A F, ascending frontal, and A P, ascending parietal convolutions ; *P C F*, precentral fissure in front of the ascending frontal convolution ; *I P F*, interparietal fissure. A section of the crura is lettered on the left side : S N, substantia nigra ; Py, region occupied by the pyramidal fibres (motor tract), which on the right are shown as continuous lines, converging in the white substance of the hemisphere, to pass through the posterior limb of I C, the internal capsule (the elbow of which is shown at *)—through the crus and pons, and to divide in the medulla into the decussating lateral pyramidal tract (*lpt*), and the non-decussating or anterior pyramidal tract (*apt*).

From this motor region of the cortex, fibres pass down to the spinal cord, and conduct the motor "impulses," as we

call them, from the motor mechanism of the convolutions to the motor mechanism of the grey matter of the cord. Leaving the cortex, these fibres pass through the white substance of the hemisphere, converging to the "internal capsule," which, as you know, lies between the lenticular nucleus (or extraventricular part of the corpus striatum) on the

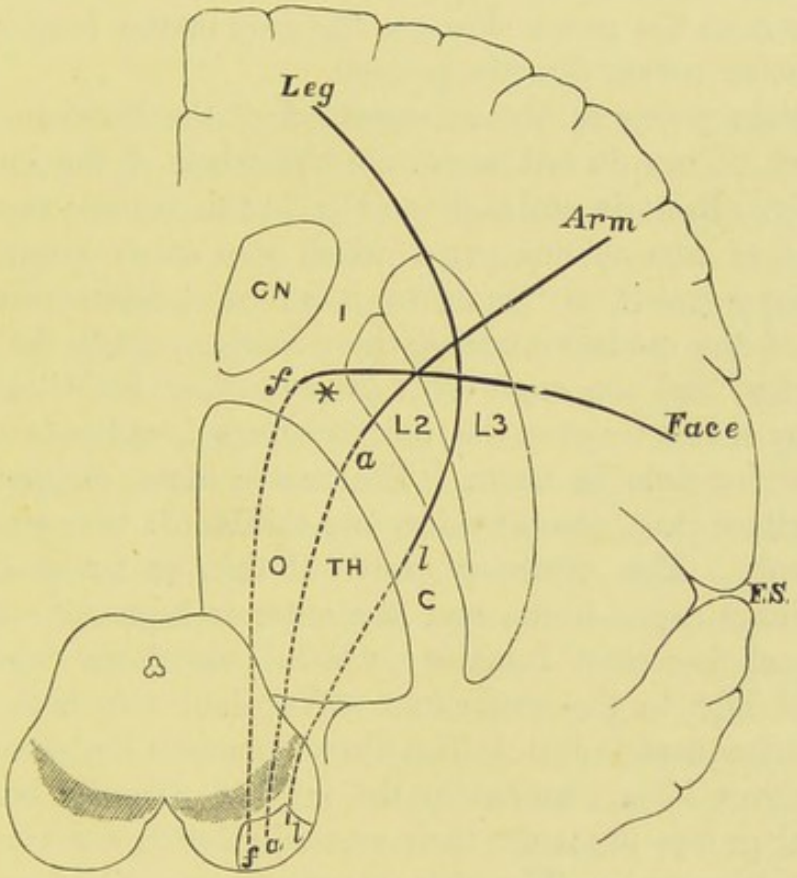

FIG. 6.—DIAGRAM TO SHOW THE RELATIVE POSITION OF THE SEVERAL MOTOR TRACTS IN THEIR COURSE FROM THE CORTEX TO THE CRUS.

The section through the convolutions is vertical; that through the internal capsule, I C, horizontal; that through the crus is again vertical. C N, caudate nucleus; O TH, optic thalamus; L 2 and L 3, the middle and outer parts of the lenticular nucleus; *f*, *a*, *l*, face, arm, and leg fibres. The words in italics indicate the corresponding cortical centres.

outside, and the caudate nucleus, and optic thalamus, on the inside. Thence the fibres pass into the crus, through the anterior part of the pons, and constitute the anterior pyramids of the medulla. Crossing for the most part in the decussation of the pyramids, they continue down the

cord, where we have seen them as the pyramidal tracts,* lateral and anterior. This is a long course; the longest of any fibres of the central nervous system. It was formerly thought that this course was broken in the grey matter of the corpus striatum; but this seems to be an error. The cells of the corpus striatum have no direct relation to voluntary movement; the motor tract passes in an unbroken course from the cortex down to the grey matter from which the motor nerves directly proceed.

Certain points in the arrangement of the fibres must be noticed. They do not constitute the whole of the internal capsule. In horizontal section (Fig. 5) this capsule seems to consist of two oblique parts, which join at an angle; the anterior segment, or "limb," as it is termed, lies outside the body of the caudate nucleus, the posterior outside the optic thalamus, and the angle or "elbow" (the "Knie" of the Germans) lies between the thalamus behind, and body of the caudate nucleus in front. The motor fibres occupy only the elbow and the anterior two-thirds of the posterior segment. The posterior third of this, as we shall see, contains sensory fibres, and the anterior segment contains fibres of uncertain function, which pass down from the frontal lobe to the cerebellum. We shall return to these in a subsequent lecture. The fibres from each limb-centre in the cortex keep together in the internal capsule, but the several groups exchange their vertical relation for one that is antero-posterior (Fig. 6); the fibres for the face, and probably for the tongue, occupy the elbow of the capsule, then come the fibres for the arm, and, most posterior, next to the sensory fibres, come the fibres for the leg.

From the internal capsule the fibres descend into the crus, lying in the anterior or lower division (crusta), and occupying the middle two-fifths of this region, but separated by some other fibres from the locus niger above them. To their inner side lie the fibres that formed the anterior limb of the internal capsule, and to their outer side is another bundle of fibres, of which I shall have more to say. The relative

* "Diagnosis of Diseases of the Spinal Cord," 3rd Ed., p. 10.

arrangement, which was vertical in the cortex, and antero-posterior in the capsule, here becomes transverse (see Fig. 6), the fibres for the face lying to the inner side, and those for the leg to the outer side, of the fibres for the arm. The fibres for the tongue probably lie close to those for the face. The latter two sets of fibres leave the others in the pons, and cross the middle line to the nuclei from which the facial and hypoglossal nerves proceed.

We know much less of the sensory path. Even in the spinal cord it is uncertain. The older theory (of Brown-Séquard), that sensation is chiefly conducted in the grey matter, is not disproved, but there is reason to believe that some sensation is conducted by the fibres of the lateral column in front of the pyramidal tract, and by fibres of the posterior columns. In the medulla and pons the path probably passes up in the posterior half, above the "fillet," perhaps chiefly in the curious network of fibres called the "reticular formation." It has been conjectured that some sensation may be conducted through the cerebellum; but this seems, on the whole, improbable. The path passes up beneath the corpora quadrigemina, through the tegmentum of the crus above the locus niger, and enters the internal capsule, where we are able exactly to determine its position. It occupies, as I have already told you, the posterior third of the hinder limb of the capsule. These fibres were once thought to be the same as those which, in the crus, occupy the outer fifth of the crusta; but Flechsig has shown that this is an error. These outer crustal fibres radiate into the white substance of the occipital and temporo-sphenoidal lobes, and connect those lobes with the cerebellum. As these fibres leave the crus, their place is taken by sensory fibres from the tegmentum, which thus form the posterior part of the internal capsule. Hence you will perceive how uncertain is our knowledge of the greater part of the course of the sensory path. Between the posterior roots of the spinal nerves and the internal capsule we have no definite facts regarding its position. We know only that it crosses the middle line in the spinal

cord, not far above the level at which the nerves enter, and that it passes up the pons on the same side as that which it occupied in the cord. In the upper part of the pons the path from the fifth nerve—from the face, etc.—joins it, so that the posterior part of the internal capsule conducts sensation from the whole of the opposite half of the body and head, skin and mucous membranes, as far as the middle line. Moreover, the facts of pathology show that the path of cutaneous sensibility is here contiguous to the path of special sensibility from the organs of special sense—of taste, hearing, smell, vision—which receive sensory impressions from the opposite side. Hence Charcot has termed this region the "sensory crossway." I say "from the opposite side" rather than "from the nerves of the opposite side," because, in the case of vision, the impressions that pass by this sensory region are those which come from the half of each field of vision corresponding to the side from which the other sensory impressions come. We shall have presently to consider this point more fully.

The ultimate destination of the sensory path is still to a large extent uncertain. Some fibres enter the optic thalamus, but their function is unknown, as there is not at present any evidence to show that impressions which influence consciousness pass through the thalamus, except, perhaps, in the case of visual impressions. The sensory fibres pass into the white substance of the hemisphere, and go towards the parietal and central ("motor") region; roughly speaking, towards that part of the cortex that lies under the parietal bone (Flechsig). We have already seen that disease of the motor cortex often causes impairment of tactile sensibility; and that when convulsions are caused by disease of this part of the surface, they often begin with a sensory aura. Thus clinical evidence, as well as the facts of anatomy, points to the outer part of the hemisphere in the middle region as being the part concerned in sensation. A case which has been recorded by Demange* shows that the indication is correct, and places the conclusion beyond doubt. This case is so

* "Revue de Méd." May, 1883, p. 391.

important that I must mention its leading facts:—A woman, aged 72, after suffering for two weeks from tingling and numbness in the left arm and leg, was found to have almost complete loss of sensation of touch, pain, and temperature in the whole of the left side, face, and limbs; with amblyopia and loss of colour-vision in the left eye. (We will consider subsequently the significance of this loss of sight.) The mucous membranes were not then anæsthetic, but a fortnight later were found to have lost sensibility, and the cutaneous anæsthesia was complete up to the middle line. Smell and taste were also lost on the left side, and the impairment of vision was greater. There was also considerable weakness in the left limbs; this gradually increased, and involved the face and the tongue. These symptoms continued until death, six months after the onset. An extensive area of softening was found, involving a large part of the outer surface of the right hemisphere and subjacent white substance; the inner surface of the hemisphere, central ganglia, and internal capsule were unaffected. The precise area of cortical damage was—lower two-thirds of the ascending frontal and ascending parietal, with the posterior extremities of the middle and lower frontal; island of Reil, inferior parietal lobule, superior parietal lobule adjacent to the intraparietal sulcus, the angular gyrus, and the whole of the occipital and temporo-sphenoidal lobes on the outer surface. The softening of the inferior parietal and angular convolutions seemed of rather older date than that elsewhere.

This case affords a crucial demonstration of the correctness of the indications afforded by the anatomical researches of Flechsig. We shall have to return to it again when we consider the central relations of vision.

LECTURE II.

MEDICAL ANATOMY OF THE BRAIN (*Continued*): NERVES OF SPECIAL SENSE—OTHER CRANIAL NERVES.

GENTLEMEN,—In our survey of those points in the anatomy of the brain that are of chief medical importance, it may perhaps be most convenient to study, next, the central relations of the cranial nerves. We will consider first, however, those nerves that subserve the special senses, and will begin with that which is most difficult, of which we have most knowledge, and yet (perhaps therefore) see most clearly how imperfect our knowledge is—the nerve of sight. At the optic chiasma these nerves undergo a partial decussation, rather more than half the fibres crossing. Fifteen years ago this semi-decussation was regarded as satisfactorily proved; but in this questioning age few doctrines seem unassailable, and it has been maintained that there is a total decussation. The result of the discussion that has taken place is to show that the old doctrine is correct, and to place it upon a firmer basis. In some animals there is a total decussation; but this occurs in those creatures in which the eyes are so placed that they never act together, and in which the fields of vision are entirely separate. In proportion as the two eyes are used together, and the fields of vision correspond, the fibres cross, so as to bring the corresponding regions of each

retina into relation with one cerebral hemisphere. In man the inner half of each field of vision is smaller than the outer half, because it is limited by the projecting nose.

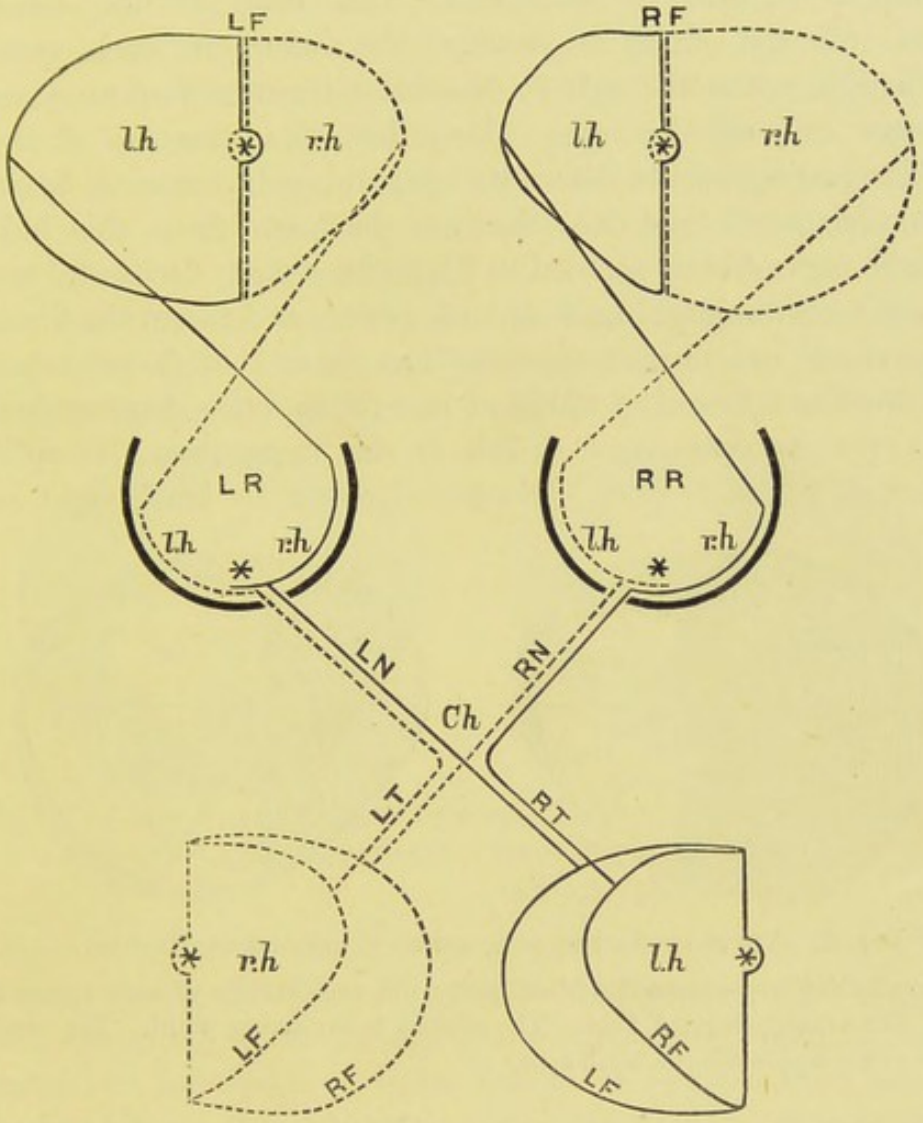

FIG. 7.—DIAGRAM OF THE RELATION OF THE FIELD OF VISION, RETINA, AND OPTIC TRACT ON EACH SIDE.

R F, L F, right and left fields—the asterisk is at the fixing point; R R, L R, right and left retina—the asterisk is at the macula lutea; *l.h*, *r.h*, left half and right half of each retina, receiving rays from the opposite half of the field; R N, L N, right and left optic nerves; *Ch*, chiasma; R T, L T, right and left optic tracts; below, the halves of the fields from which impressions pass by each optic tract are superimposed.

Although the two halves of the *retina* are of equal size, the sensitiveness of the outer, temporal half (which receives rays from the inner, nasal half of the field of vision)

does not extend so far towards the front as does that of the inner, nasal half. This is shown by examining the field—first when the eye is directed forwards; secondly, when it is directed outwards. The limit of the nasal half of the field is nearly the same in each case, although, when the eye is directed outwards, the nose no longer cuts off the rays. Doubtless the structure of the retina corresponds to this; its temporal half contains fewer nerve-elements than does the nasal half, and from this half fewer nerve-fibres proceed. The fibres that decussate are those from the nasal half of each retina, and hence the fibres that cross are more numerous than those that do not cross. If the fibres from the whole of one retina have degenerated, the opposite optic tract shrinks in size more than the optic tract on the same side. Disease of one optic tract, right or

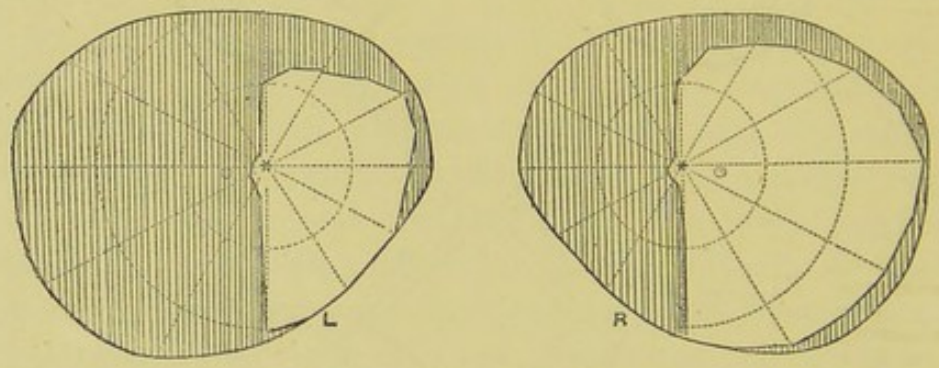

FIG. 8.—FIELD OF VISION IN A CASE OF LEFT-SIDED HEMIOPIA.

The shading represents the blind part; the oval outline of each figure is the average normal field. The asterisk is the fixing point. The small circle represents the blind spot.

left, therefore, arrests the conduction from the same-named half of each retina, and causes loss of vision in the opposite half of each field—"hemiopia," or "hemianopsia," as it is termed. (Fig. 8.)

The division between the two half-fields is usually in, or very near, the vertical line passing through the fixing point (corresponding to the macula lutea of the retina). Sometimes it passes through the fixing point; more often it diverges and passes round the fixing point, a little distance from it, to return again to the middle line, and this whichever side is

blind. The balance of evidence at present collected seems to show that this difference does not depend on the seat of the disease causing the hemiopia; and it can therefore only be explained by individual differences in the decussation. In many individuals, it would seem, fibres from a small area around the centre of the macula pass by each optic tract; while in some persons, each tract contains fibres from only one half of the macular region, as it does from the rest of the retina. Another form of variation seems to show also that there are considerable differences in the character of the decussation. Above and below the fixing point, the line of division is also often irregular, sometimes, for instance, sloping

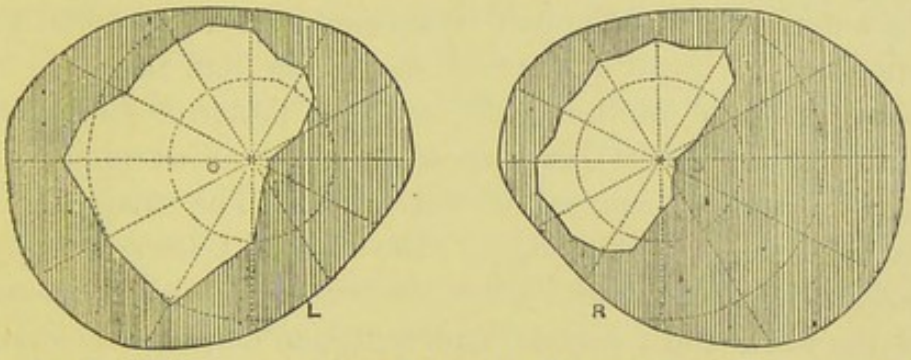

FIG. 9.—RIGHT HEMIOPIA FROM CEREBRAL DISEASE, SHOWING AN IRREGULAR OBLIQUE LINE OF DIVISION, PROBABLY DUE TO AN UNUSUAL FORM OF DECUSSATION.

off to one side above, and to the other side below, the two fields corresponding closely. In an extreme example of this, such as is shown in Fig. 9, the division becomes irregularly oblique instead of vertical. It is impossible to explain this, except on the theory of variations in the decussation. Some facts suggest that there are similar variations in some other decussations in the nervous system, especially in that of the sensory tract from the skin.

The visual path passes in the optic tract, by the corpora geniculata, through the white substance of the hemispheres, to the cortex of the occipital lobe on the outer surface. If it is destroyed anywhere in this course, the result is hemiopia of the same character. Disease of the cortex of the occipital lobe has a like effect. There is no complementary decussation at the corpora quadrigemina, as has been fancied. Whether

the fibres from the tract to the cortex are interrupted by grey matter, or are continuous, we do not know. Fibres from the tract enter the external corpus geniculatum, the optic thalamus, the corpora quadrigemina. Fibres from these pass into the white substance of the hemisphere, and the grey matter of these bodies *may* be interposed in the optic path. It is probable, however, that the fibres that enter the corpora-quadrigemina substance only guide the movements of the eyeball. On the other hand, some fibres from the tract pass directly into the hemisphere, and these may continue the visual path, or part of it, directly to the cortex.

The experiments of Munk lead him to believe that in dogs the anterior half of the half-vision centre of the occipital lobe subserves the upper quarter of the retina (and therefore the lower quadrant of the field); and the posterior half of the centre subserves the lower quarter of the retina (and therefore the upper quadrant of the field). The frequency of partial hemiopia in man, *e.g.*, the loss of a lower or upper quadrant, makes it certain that there must be an arrangement of the fibres and a central representation on a system similar to that in the dog, although whether the correspondence holds good in the details of the plan we do not yet know.

But the central relations of vision are still more complex. The subjects of that strange disease, hysteria, sometimes suffer from hemianæsthesia and loss of the special senses on the anæsthetic side, together with—not hemiopia—but what is termed "crossed amblyopia," dimness of sight of the eye on the anæsthetic side, peripheral constriction of the field of vision, and often loss of colour-vision. There is usually also a far slighter limitation of the field of the other eye. The curious symptoms of hysteria probably depend on some morbid action in high cortical centres. They may teach us much of the functions of the brain, much of the association of its elements on which those functions depend; although they furnish no indication of the coarser anatomical relations. The occurrence of this amblyopia shows that there must be a functional centre, capable of being inhibited, in which is represented chiefly the whole field of one eye, not the half-

fields of both eyes. It does not, however, show that this field is situated in one hemisphere, since the half-vision centres in the two hemispheres might be so connected as to be susceptible of such combined partial inhibition as would produce the symptoms. But the experiments of Ferrier have shown that lesions of the angular gyrus in animals will cause dimness of sight of the opposite eye, which rapidly passes away. In rare cases in man, in which there is certainly organic disease, similar crossed amblyopia has been observed. I have seen a few instances of this, and several others have been recorded. Moreover, the case I have quoted (p. 16) is proof that such amblyopia may result from disease of the outer surface of the hemisphere. It will be noted that in this case the lesion involved the occipital lobe, and should have caused, therefore, hemiopia also, recognizable in the eye on the side of the lesion.* But the pathological appearances suggested that the disease of the occipital lobe was later than that of the angular gyrus; and such hemiopia might well have escaped detection during the later stages of the patient's illness. The original report of the case gives no indication that it was sought for.

Hence it is in a high degree probable that (as Ferrier has suggested) in or near the angular gyrus there exists a visual centre, higher than the half-vision centre, in which the whole of the opposite field is represented. For this relation there must be a connection between this centre on one side and both occipital lobes, that with the opposite occipital lobe being probably by means of the fibres of the corpus callosum. Indeed, the arrangement is probably even more complex. The crossed amblyopia is accompanied by a much slighter restriction of the field of the other eye, *i.e.*, on the same side as the lesion. Hence, in each higher visual centre both fields must be represented, that of the opposite eye, however, far more extensively than that of the eye on the same side. Another difference may be traced

* I have recorded in "Medical Ophthalmoscopy" (2nd Ed., Case 30, p. 311) a case in which total blindness of the left eye, with left hemiopia in the right, accompanied left hemiplegia—all due, no doubt, to an extensive lesion of the right hemisphere.

between this and the half-vision centre. A permanent lesion of the latter causes permanent hemiopia; but the crossed amblyopia soon lessens, and before long becomes slight. This is probably by the substitutionary action of the centre of the opposite hemisphere, since in each, as we have seen, both fields are represented. Lastly, a lesion of the higher centre, whatever its exact seat or nature, seems always to cause the same form of impairment (amblyopia, restricted fields, loss of colour-vision), which differs in degree, but not in form. A partial lesion seems to lower the function as a whole—an indication that the function is diffuse,—and this is probably a characteristic of all the higher cortical centres.

Our knowledge of the central relations of the olfactory nerve is much less extensive. The only indication afforded by experiment is that there is a cortical centre for smell at the anterior extremity of the uncinate gyrus on the inner surface of the hemisphere (Ferrier). Towards this some of the fibres of the olfactory nerve seem to pass directly. Moreover, disease adjacent to these fibres has caused loss of smell on the same side as the lesion. On the other hand, disease involving the "sensory crossway" has impaired smell on the side opposite to the lesion. Hence, it seems probable that the olfactory path passes first to a cortical centre in the same hemisphere, and thence to the other hemisphere, although where it crosses, and what is its ultimate cortical destination, we do not know, except that the latter is on the outer surface of the hemisphere. This is shown by the case described on p. 16.

The auditory nerves pass to nuclei situated at the junction of the pons and medulla oblongata. Their exact position we shall consider presently. They have an extensive connection with the cerebellum, to which, indeed, some fibres of the auditory nerves seem to pass directly. You doubtless remember that one part of the auditory nerve (from the semicircular canals) has nothing to do with hearing, but conveys information, as to the position of the body, to the centre

for equilibration, probably situated in the cerebellum. The auditory path to the hemispheres probably passes up the pons, crossing early, and, in the crus, occupies the superior portion of the tegmentum. Some have thought that it does not pass up the pons, but passes through the cerebellum; but this is, on the whole, improbable. Its course in the cerebrum is by the "sensory crossway," through the white substance, to the first temporo-sphenoidal convolution, which all evidence shows to be the cortical centre for hearing, each centre receiving impressions from the auditory nerve on the opposite side.

Of the path of taste, we know only that it passes by the sensory crossway; of its cortical centre we know nothing. Strangely enough, even the nerve of taste is still somewhat uncertain. It is commonly supposed that the glosso-pharyngeal is the nerve of taste of the back of the tongue, and that the chorda tympani of the facial, which is certainly the nerve of taste for the front of the tongue (and probably comes from the fifth by the Vidian nerve, that passes from the spheno-palatine ganglion to the facial), may ultimately be derived also from the glosso-pharyngeal. But I believe that it will be found that taste-impressions reach the brain solely by the roots of the fifth nerve, and that the doctrine that the roots of the glosso-pharyngeal nerve have anything to do with taste is a curious physiological myth, due to too wide an induction from certain anatomical facts, and from dubious experiments on animals. Some time ago* I published a case in which there was an isolated palsy of one fifth nerve, motor and sensory portions, due certainly to disease of the root at the surface of the pons, in which taste was entirely lost on that side, not only at the front of the tongue, but also at the back, on the soft palate, and on the palatine arches—lost to every form of stimulation, whether by sapid substances or by the voltaic current. The symptoms in this case are permanent, and I have repeatedly demonstrated the loss to

* "Journal of Physiology," vol. iii., p. 229.

those who have attended my practice at the Queen Square Hospital. Since the publication of that case I have met with only two other cases in which there was intracranial disease of the fifth without evidence of any interference with the nerves of the medulla, and in each of these there was the same absolute loss, at the back as well as at the front, demonstrable with ease, and most striking by comparison with the other side. It is possible that the nerve-fibres for taste on the back of the tongue may be *distributed* with the glosso-pharyngeal, reaching them from the otic ganglion of the fifth by the small petrosal nerve and tympanic plexus. This explains the remarkable fact, pointed out by Urbantschitsch, and which I have several times observed, that taste may be lost on the back as well as the front of the tongue, in consequence of caries of the walls of the tympanum. This course seems, I confess, strangely circuitous, but it is scarcely more circuitous than that which is certainly taken by the taste-fibres of the front of the tongue. If this arrangement is ultimately proved beyond question, it will be of much interest, as bringing the sense of taste into the functions of one nerve, and that one the nerve that is so intimately associated with the other special senses of smell, sight, and hearing.

We may conveniently consider next the arrangement of the nuclei from which the other cranial nerves arise. These are situated in the grey matter that surrounds the highest part of the central canal of the spinal cord, the continuation of this canal between the fourth and third ventricles, and that which lies beneath the floor of the fourth ventricle. The nuclei are columns of nerve-cells, small in transverse section, but prolonged in the direction of the axis of the medulla. (See Fig. 10.) The upper part of the hypoglossal nucleus lies close to the middle line in the point of the calamus scriptorius; lower down it lies on each side of the middle line in front of the central canal. In corresponding position behind the canal is the column of cells of the vago-accessorial nucleus. In the calamus this lies outside the hypoglossal, and gives origin to the pneumogastric; while below, part of the spinal accessory springs from it. The

spinal part of the accessory consists only of spinal fibres that rise into a temporary companionship with a higher nerve, but have the same origin, and subside to the same distribution, as the other spinal nerves of the same level. Note, however, the proximity of the nuclei of the hypoglossal and accessory nerves; both supply muscles that act together in articulation. Moreover, the muscle that raises the palate and shuts off the posterior nares in articulation is certainly supplied from one of the nerves of the medulla, probably from either the glosso-pharyngeal or the spinal accessory. All

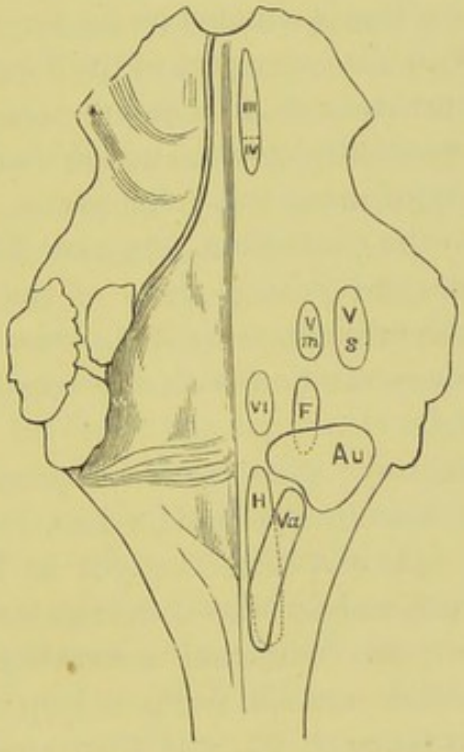

FIG. 10.—DIAGRAM OF THE RELATIVE POSITION OF THE NERVE NUCLEI BENEATH THE FLOOR OF THE FOURTH VENTRICLE.

III, third nerve nucleus; IV, fourth; V *s*, middle sensory nucleus of the fifth; V *m*, motor nucleus of fifth; VI, sixth; F, facial; Au, auditory; H, hypoglossal; V a, vago-accessorial nucleus, the upper part giving origin to the pneumogastric, the lower to the highest fibres of the spinal accessory. Where one nucleus lies beneath another, its outline is indicated by a dotted line.

these three parts—tongue, palate and vocal cord—are paralysed together from disease at the surface of the medulla, damaging the roots of the nerves. All these parts are paralysed

together in degeneration of these nuclei, with the addition also of the lips, constituting the chronic form of "labio-glosso-laryngeal paralysis," or "bulbar paralysis." Why the lips are also affected we shall see presently. The pneumogastric nucleus in the floor of the ventricle corresponds nearly in position to the respiratory centre, and no doubt also to the cardiac centre. Its upper and outer limits are not definable, since it blends with a more diffuse area of delicate grey matter that extends as far as the auditory nucleus. Outside, and rather in front of the upper part of the pneumogastric nucleus, is the small column of nerve-cells from which the glosso-pharyngeal nerve arises.

The auditory nucleus is situated at the level of the auditory striæ, and chiefly in the outer part of the floor of the ventricle, just over the commencement of the inferior cerebellar peduncle, but it extends inwards almost to the middle line. It is a double nucleus, consisting of inner and outer parts. Fibres are said to pass from it to the cerebellum, and some fibres of the nerve seem to turn aside, and joining those of the restiform body, pass directly to the cerebellum by its inferior peduncle. The significance of this connection with the cerebellum has been already mentioned.

Above the auditory striæ, under a prominence on the eminentia teres, close to the middle line, is the nucleus of the sixth nerve. It was once thought to be the common nucleus of the sixth and facial,—a strange combination, since these nerves have no functional association. The facial nerve ascends to this nucleus, forms a loop round it (some fibres, indeed, go through it), and then passes downwards, forwards, and outwards, to a column of cells more deeply placed in the medulla than any other nucleus in the lower part. A rare case, in which there was total palsy of all the ocular nerves, and no paralysis of the facial, afforded me an opportunity of proving beyond doubt that the facial nerve has no real origin from the nucleus of the sixth. All the cells of this nucleus were degenerated, but the fibres of the facial were perfectly healthy, and could be traced unchanged through the nucleus. The cells of the true facial nucleus cannot be traced far below the level of the

auditory striæ, but it is probable that some fibres of the facial nerve, those that innervate the orbicularis oris, descend to the level of the hypoglossal nucleus, and may even arise from this nucleus. The transverse muscle of the tongue and the orbicularis act together. Neither can contract or relax without the other. Try, yourselves, gentlemen, when you are alone with a looking-glass, and you will find that this is true. Both, moreover, suffer together in degeneration of the nuclei, while the other parts of the facial nerve escape.

The chief nucleus of the fifth nerve lies above the sixth, and at the outer part of the floor of the ventricle. The fibres of the nerve pass backwards and slightly inwards, and end in a sensory nucleus on the outer, and the motor nucleus on the inner side. This is, however, but one part of the sensory nucleus. Some fibres pass down the medulla, and can be traced as low as the commencement of the spinal cord. They have grey matter on their outer side, in which they probably end. No doubt these fibres bring sensory impressions from the tongue, etc., into relation with the motor nuclei for these parts. Moreover, the distribution of the fifth nerve to the skin joins that of the cervical nerves both on the face and back of the head, and the continuity of the cutaneous distribution is no doubt subserved by a continuity of the grey matter, in which the lower root arises, with that from which the cervical nerves arise. Another group of fibres of the fifth passes up beneath the corpora quadrigemina, no doubt subserving the intimate connection between the fifth nerve and the organ of vision, of which so obtrusive an instance is presented in the photophobia of conjunctivitis.

The third nerve, entering the inner side of the crus, passes back to a column of nerve-cells beneath the aqueduct of Sylvius, not far from the middle line. The combined teaching of experiment (especially of Hensen and Voelcker) and of clinical observation shows that there are three centres in this column of nerve-cells, distinct at least in function and in pathological liability. The most forward of these is the centre for the ciliary muscle (accommodation); the second is the centre for the light reflex of the iris; the third, which

occupies the greater part of the nucleus, is the centre for the external muscles supplied by the third nerve.

The fourth nerves differ in two respects from any other of the cranial nerves. First, they arise above the fourth ventricle, their origin being from the valve of Vieussens, but they curve round the lower part of the aqueduct of Sylvius to a column of nerve-cells that is really the lowest part of the third nerve nucleus. Secondly, alone of all the cranial nerves, the fourth nerves decussate between the surface attachment and the nucleus. But the whole of the fibres do not come from this nucleus. Some descend the pons, and probably arise from the nucleus of the sixth nerve. Indeed, there seems to be an extensive connection, by large nerve-

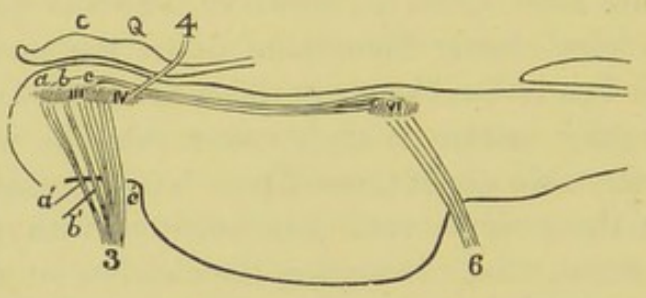

FIG. 11.—DIAGRAM OF LONGITUDINAL SECTION THROUGH THE PONS, SHOWING THE RELATION OF THE NUCLEI FOR THE OCULAR MUSCLES.

C Q, corpora quadrigemina ; 3, third nerve ; III, its nucleus ; 4, fourth nerve ; IV, its nucleus, the posterior part of the third ; 6, sixth nerve. The probable position of the centre and nerve-fibres for accommodation is shown at *a* and *a'* ; for the reflex action of iris at *b* and *b'* ; for the external muscles at *c* and *c'*. The lines beneath the floor of the fourth ventricle indicate the fibres that connect the nuclei.

fibres, the "posterior horizontal fibres," between the three nuclei for the nerves of the external ocular muscles (Flechsig). It is possible that some of these are fibres of the nerves which really pass by their proper nucleus, and arise from one of the other nuclei, and the visible decussation of the fibres of the fourth nerve may be the indication of an extensive decussation of these connecting fibres. Thus we can understand how the apparently simple arrangement of the nuclei, and their apparently strange separation in three distinct groups of nerve-cells, may cover an extensive and

complex structural association, whereby one nucleus may give origin to fibres that run in several nerves, and the various nuclei may be blended into what is practically a series of centres very different from their apparent form. Thus, too, we can understand how these nuclei may govern the complex movements of the eyes, in which many muscles of both sides act together in the most perfect synchronism and exact gradation of effect.

The third, sixth, and hypoglossal nerves arise at the surface, near the middle line, and pass back to their nuclei between the middle line and the motor pyramidal tracts. The spinal accessory, pneumogastric, glosso-pharyngeal, and facial nerves arise on the surface at the outer part of the medulla, on the outer side of the pyramidal tracts, and pass inwards and backwards to their nuclei. The nuclei of the auditory nerve lie almost immediately above its origin, and one part of the nerve passes directly back to it, while the other curves round and above the outer nucleus to reach the inner nucleus.

From all these nuclei, paths, as yet only partially traced, ascend to the cerebral hemisphere. They certainly cross the middle line not far above the nuclei. The paths, motor and sensory, probably join, or at least run close to, the motor and sensory paths from the limbs. This, as we have seen, is certainly the case with the motor path of the face.

After leaving the brain, the nerves have a short course before entering the dura mater. Most arise in the posterior fossa. The nerves to the orbit and the fifth nerve leave the skull in the middle fossa, but as they leave the posterior fossa they pass into the dura mater. Remember that the sixth nerve has by far the longest course before it enters the dura mater, from the posterior border of the pons to near its anterior border, and that before it enters the wall of the cavernous sinus it passes very near the fifth nerve. Remember also that in its course over the convexity of the pons it readily suffers from pressure, if there is any cause of pressure beneath the tentorium. For this reason, paralysis of both sixth nerves is a very common symptom in disease of this region.

LECTURE III.

MEDICAL ANATOMY OF THE BRAIN (*Continued*): CONNECTION OF CEREBRUM AND CEREBELLUM — BASAL GANGLIA— CEREBELLUM—BLOOD-VESSELS OF THE BRAIN.

GENTLEMEN,—We must now return to some points that we passed over in considering the anatomy of the cerebrum. What is the course of the fibres that constitute the anterior limb of the internal capsule, lying between the body of the caudate nucleus and the lenticular nucleus? They pass, on the one hand, to the cortex of the prefrontal lobe, *i.e.*, the frontal lobe in front of the ascending frontal convolution. On the other hand, they descend into the crus, and occupy the inner (medial) portion of the crusta, lying to the medial side of the pyramidal tract. They descend to the pons, and there seem to end in the grey matter which is so abundantly scattered among the white fibres, longitudinal and transverse, of the anterior region of the pons. It is, however, probable that other fibres, proceeding from this grey matter to the cerebellum, continue the path to the cerebellar hemisphere, especially to the lateral and posterior regions. Thus this tract consists of fronto-cerebellar fibres. They degenerate downwards, and therefore probably conduct downwards; but this degeneration only extends to the pons, being arrested, as secondary degeneration always is, by the grey matter that interrupts their course. When the cerebellum is congenitally

absent these fibres are also absent (Flechsig). It is probable that this connection is a crossed one, the frontal lobe on one side being connected with the cerebellar hemisphere of the opposite side.

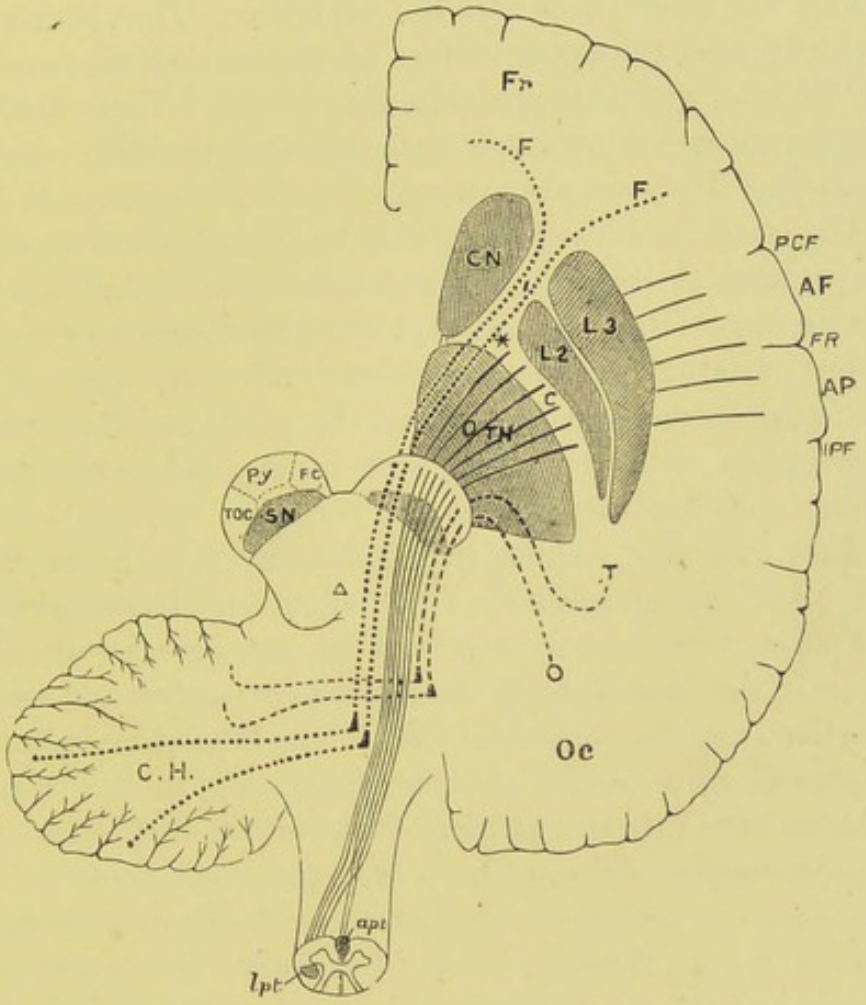

FIG. 12.—DIAGRAMMATIC HORIZONTAL SECTION, THROUGH THE CEREBRUM, PONS, AND OPPOSITE HEMISPHERE OF THE CEREBELLUM, TO SHOW THE COURSE OF THE FIBRES CONNECTING THE FRONTAL AND OCCIPITAL LOBES WITH THE CEREBELLUM.

In the section of the crus, on the left side, T O C shows the position of the fibres to the temporo-occipital lobe from the cerebellum (shown by dotted lines), and F C that of the fibres from the frontal lobe to the cerebellum (shown by broken lines) that occupy the anterior limb of the internal capsule. The other letters are explained on p. 12.

I told you that the outer fibres of the crusta of the crus cerebri, the fibres that lie outside those of the pyramidal tract, do not pass up into the internal capsule, but radiate into the occipital and temporal lobes of the brain, passing from the

crus, partly beneath the posterior extremity of the lenticular nucleus, partly between it and the external corpus geniculatum. These fibres connect these lobes with the cerebellum in the same way as the fibres of the inner part of the crus connect the frontal lobe with the cerebellum. Their connection is chiefly with the upper surface of the cerebellum, near the middle lobe, and it is probably also crossed. These fibres do not degenerate downwards, and therefore probably conduct upwards. They are also absent when there is no cerebellum.

The small bundles of fibres that lie behind, or rather above, the pyramidal tract in the crusta, between it and the substantia nigra (Fig. 5), consist of fibres that descend from the corpus striatum, caudate nucleus, and outer part of the lenticular nucleus, and, reaching the pons, probably connect those parts with the cerebellum in the same way as the other tracts connect with it the cortex.

This extensive connection of the cortex with the hemispheres of the cerebellum is a fact of extreme interest, because the parts of the convolutions thus connected are those in which there is the least definite localization of motor and sensory function. In the prefrontal lobe of the human brain there is at present no evidence of motor function. In the temporal lobe we have only the auditory centre in the first convolution. In the occipital lobe we have only the visual centre, which, although not yet accurately defined, probably occupies only a part of it. In these portions of the brain a lesion may exist, and cause neither motor nor sensory symptoms; their function is more diffuse, so to speak, and thus capable of supplementary substitution. It is here that we must look for the processes concerned in the higher intellectual operations. These parts, whatever other connection they may have, seem to be connected with each other through the cerebellar hemisphere by the downward path from the frontal and the upward path to the temporal and occipital lobes. This very curious fact revives the old idea, that the cerebellum is, in some way, concerned in intellectual processes—an idea suggested by the proportion that the cerebellar hemispheres bear to intellect as we ascend the scale of animals. Moreover, the cerebellar

hemisphere has this in common with the parts of the cerebral cortex with which it is connected, that a lesion may exist in it without motor or sensory symptoms.

You may have observed with surprise that I have said nothing about the great central ganglia of the brain, which were formerly supposed to have such important connections with the motor and sensory tracts. It is probable that some of them have a connection with motor centres, but it is certain that their relations are very different in nature from those formerly attributed to them.

The optic thalamus receives fibres from below that come from the tegmentum of the crus, and probably these are derived from the upward sensory path from the spinal cord. It also receives fibres from the superior peduncle of the cerebellum; perhaps also from the optic nerves. From it fibres radiate to all parts of the cerebral cortex. Some go to the lenticular nucleus; but we do not know whether they end in it, or pass through it to the convolutions. Nevertheless, it does not seem to be in the path of those common sensations that affect consciousness. These pass, as we have seen, outside the hinder part of the thalamus, in the posterior third of the internal capsule. They are close to the thalamus, its lesions often involve them, but when they are unaffected there is no loss of sensation. It is highly probable, however, that the thalamus is concerned with some of the higher reflex processes.

The grey masses of the corpus striatum, on the other hand, seem to have no connection with the cortex. The caudate nucleus is connected with the cerebellum, as we have seen, by fibres that enter the internal capsule, either directly, or after passing through the lenticular nucleus, and lie in the crus close to the locus niger, reaching the cerebellum by the middle peduncles, after interruption by grey matter in the pons. No fibres from the caudate nucleus seem to join the pyramidal motor path. The lenticular nucleus, like the optic thalamus, receives fibres (chiefly

collected into the "lenticular loop") from the tegmentum and from the opposite superior cerebellar peduncle. Thus both parts of the corpus striatum have an extensive connection with the cerebellum of the opposite side, and the descending fibres from the caudate nucleus degenerate (and therefore conduct) downwards, while those from the cerebellum to the lenticular nucleus probably conduct upwards. Thus this connection presents considerable analogy to that between the cortex and the cerebellum. In congenital absence of the cerebellum the corpus striatum is reduced to a third of its ordinary size (Flechsig). It seems probable that the two parts of the corpus striatum are central organs, analogous to the cortex itself. The analogy to those parts of the cortex that are connected with the cerebellum is rendered still greater by the fact that a lesion, even an extensive lesion, may exist in either the caudate or lenticular nucleus, and so long as it does not interfere with the functions of the motor or sensory parts of the internal capsule, it causes no symptoms.

Although the middle lobe of the cerebellum is continuous with the hemispheres, and the peculiar foliated cortex has a similar structure in both parts, it is certain that there is an essential difference in their function. In the white substance of the middle lobe are several curious nuclei of grey matter. As we descend the scale of animals the hemispheres become smaller, until the cerebellum, of birds for instance, corresponds only to the middle lobe. The hemispheres may be diseased without recognizable symptoms, but this is not true of the middle lobe. The disease of this causes the peculiar unsteadiness of movement long known to be characteristic of cerebellar disease, and lately shown (by Nothnagel) to be characteristic only of disease of the middle lobe. The reason why the latency of lesions in the hemispheres was so long unrecognized is because the common lesion is tumour, and this almost always presses on the middle lobe. We must thus regard the middle lobe of the cerebellum as in some way concerned with the main-

tenance of equilibrium, perhaps by combining the afferent impressions, and arranging for harmonious centrifugal impulses. This view is supported not only by the effects of disease, but by the facts that certainly towards this lobe, and probably to it, fibres pass from the posterior columns of the cord, from the direct cerebellar tract (fibres from the lower trunk-muscles), and from the auditory nerve. Ferrier found that stimulation of the middle lobe caused movements of the eyes. Although we cannot conceive that centres for the voluntary movements of the eyes exist here, yet the eye-ball muscles furnish important guidance to the centre that regulates the maintenance of equilibrium, and the centre for these muscles may thus be so related to the central lobe of the cerebellum, that its stimulation may cause indirectly the contraction of these muscles.

No part of the brain has excited more interest and received more study than the olivary bodies of the medulla; varied functions have been, in turn, ascribed them, but these hypotheses, destitute of foundation, have successively sunk out of view, and we are still ignorant of the function of these structures. Their connections are peculiar. The bodies resemble closely the dentate nuclei of the cerebellum; and the resemblance is certainly not accidental, for the two structures are connected by fibres, and atrophy together. Each olivary body receives fibres from the posterior columns of the cord on the opposite side, and is connected with the dentate nucleus, also of the opposite side. From each dentate nucleus fibres pass to the superior cerebellar peduncle and tegmentum of the opposite crus. Thus the tegmentum of one crus cerebri may be connected with the olivary body of the same side through the opposite dentate nucleus.

A large number of cerebral lesions are produced by disease of the cerebral vessels, by their occlusion or their rupture. To understand many facts about these lesions, you must know the arrangement of the cerebral vessels and the

conditions of the cerebral circulation. I will briefly mention to you the facts of chief importance. Those that concern the arterial system have been ascertained by the investigations of Duret in France, and Heubner in Germany.

The blood-supply to the brain comes from the carotid and vertebral arteries. The left carotid arises from the aorta nearly in the direction of the current of blood that courses through the aortic arch; while the right carotid comes from the innominate, and this arises from the aorta nearly at right angles to the current of blood. Hence solid particles are rather more readily carried into the left than into the right carotid—a circumstance that explains the somewhat greater frequency of embolism on the left side of the brain. There is a similar but still greater difference in the origin of the two vertebrals, and it is probably for this reason that the left vertebral is often larger than the right. This does not determine any difference in the frequency of vascular lesions in the part of the cerebrum supplied, because the blood brought by both vertebrals has to pass through the common basilar. The internal carotid, on each side, divides into a small anterior cerebral, and larger middle cerebral. The latter continues the direction of the internal carotid, and hence plugs carried from the heart readily pass into it. The "circle of Willis," you will remember, is formed by these vessels, together with the anterior communicating artery, between the anterior cerebrals, and two posterior communicating (one on each side), between the middle and posterior cerebrals.

From the circle of Willis and the commencement of the three cerebral arteries (anterior, middle, and posterior) small branches arise which supply the central ganglia and adjacent white substance of the hemisphere, while the three arteries ramify over the surface of the brain, and supply the grey cortex and the chief part of the white substance. Thus there are two systems of branches, central and cortical. Between these two systems there are no anastomoses. The central branches do not communicate with each other, and hence obstruction of one causes necrosis of the region supplied, no

collateral supply of blood being possible. The cortical branches vary in this respect in different individuals. In some persons there is enough communication between the cortical branches to maintain nutrition if one is obstructed; in other persons there is no communication. Hence obstruction of the middle cerebral at its origin always causes necrotic softening of the part of the central ganglia that it supplies, and sometimes also softening of the cortex; while in other cases, with a similar obstruction, we may find that the cortex is intact, although the central ganglia are extensively damaged.

The branches to the ganglia must engage our attention in further detail, since the pathological importance of these "central arteries" is very great. They are divided into six groups, of which two are medial and small, and four (two on each side) are lateral and very important. The anterior medial group is given off by the anterior cerebrals and anterior communicating artery, and supply the anterior extremity of the caudate nucleus. The posterior medial group consists of twigs given off by the posterior cerebrals near their origin. Passing through the posterior perforated spot, they supply the inner part of the optic thalamus and the walls of the third ventricle. These two medial groups are insignificant in the extent of the brain supplied by them, but of some pathological importance, since hæmorrhage, from their rupture, is apt to burst into the ventricles. The lateral groups furnish the blood to the chief part of the central ganglia and the internal capsule. The anterior lateral group consists of numerous small arteries that arise from the middle cerebral in the first inch of its course, pass through the anterior perforated space, and supply the caudate nucleus (except its head), the lenticular nucleus, internal capsule, and part of the optic thalamus. Some pass through the inner part of the lenticular nucleus to the internal capsule; while others pass first outside the lenticular nucleus, and then through its outer portion to the capsule. They supply the caudate nucleus and optic thalamus after passing through the capsule. These vessels are prone to rupture, perhaps because their direct origin from a comparatively large vessel exposes them to

a high blood-pressure. Hence the frequency of cerebral hæmorrhage in this situation. The arteries of the posterior lateral group arise from the posterior cerebral, and supply the hinder part of the optic thalamus. Hæmorrhage from their rupture usually damages the posterior (sensory) part of the capsule, and often extends into the crus. Branches from the posterior cerebral supply also the crus and corpora quadrigemina.

Of the blood-supply to the cortex, that from the middle cerebral is both the most extensive and the most important, embracing, as it does, the central (motor) convolutions. The general arrangement of the branches of each artery is the same: each divides and ramifies, and from the branches, and the ultimate ramifications in the pia mater, twigs are given off to the cerebral substance—some short, that end in the grey cortex; others long, that pass through the grey cortex to the white substance, extending in it to various depths. The regions supplied by the several vessels are as follows:—The anterior cerebral, curving round the corpus callosum, supplies, by three branches, part of the orbital lobule, and the inner surface of the hemisphere as far as the quadrate lobule (precuneus). It also supplies, on the outer surface, by branches that come over from the inner surface, the first and second frontal convolutions, and the highest part of the ascending frontal. The middle cerebral or "Sylvian artery" divides in the fissure of Sylvius, opposite the island of Reil, into four branches, which lie in the sulci of the insula, and then pass—the first to the inferior frontal convolution; the second to the ascending frontal, except the highest part, which is supplied by the anterior cerebral; the third to the whole of the ascending parietal, and the adjacent part of the inferior parietal lobule; the fourth to the convolutions about the posterior limb of the fissure of Sylvius, supramarginal and angular, the hinder part of the superior parietal lobule, and the first temporal. From this vessel, near its origin, one or two large branches arise that supply the greater part of the second and third temporal convolutions. The posterior cerebral supplies the occipital lobe, and also the inferior aspect of the temporal lobe, by three

branches, of which one goes to the lower part of the uncinate convolution, a second to the inferior part of the temporal lobe, and a third to the cuneus, lingual convolution, and the inner and outer surfaces of the occipital lobe.

Thus the middle cerebral supplies the motor region, both central and cortical, except in part the leg-centre; it also supplies the part of the cortex that subserves cutaneous sensibility, the cortical auditory centre, and probably the higher visual centre; it supplies all the cortical regions concerned in speech-processes in the left hemisphere, motor, auditory, and visual. The anterior cerebral supplies only a small part of the motor region, viz., the part of the leg-centre that occupies the paracentral lobule and highest part of the ascending frontal. The posterior cerebral supplies the visual path, from the middle of the tract backwards, and the half-vision centre in the occipital lobe; it supplies also the corpora quadrigemina, and the sensory part of the internal capsule.

The pons and medulla receive small arteries from all the adjacent vessels, vertebrals, basilar, and cerebellar arteries. These branches are in two sets—median, near the middle line; and lateral or radicular, that pass in at the side, near the chief nerve-roots. Both pass back to the nuclei near the floor of the fourth ventricle, but the chief blood-supply to these nuclei comes from the median branches. Of the cerebellar arteries, the superior and inferior supply the corresponding regions of each hemisphere, and the upper surface is also supplied by a large branch from the basilar opposite the middle of the pons, the middle cerebellar artery. These arteries communicate freely, and hence necrotic softening of the cerebellum is rare. The branches to the medulla and pons do not communicate, and hence here necrotic softening is common.

The venous circulation of the brain presents several important peculiarities. The veins from the greater part of the cortex pass upwards to the longitudinal sinus, and open into this in a forward direction. This arrangement involves two very unusual conditions. Elsewhere, the blood from ascend-

ing arteries passes into descending veins, so that the feeble pressure that passes through the capillaries is supplemented by the influence of gravitation. Elsewhere, ascending veins convey blood that has been brought by descending arteries, and the venous flow is aided by the liquid pressure, which, according to the well-known law of hydrostatics, tends to make the blood rise in the veins. But on the brain, the blood from ascending arteries passes into ascending veins. The openings of these veins into the longitudinal sinus being directed forwards, the entering blood is opposed in direction to the current in the sinus, and the effect must be to retard the flow in both veins and sinus. Moreover, in the erect posture the anterior half of the longitudinal sinus has also an ascending course, while the trabeculæ that occupy the lumen of the sinus must offer some hindrance to the movement of the blood. These circumstances help us to understand the readiness with which clots form in the cortical veins and longitudinal sinus, when other circumstances favour the coagulation of the blood. Indeed, the marvel is that thrombosis is not more common than it is.

The veins of Galen, conveying blood from the ventricles to the straight sinus, pass above the corpora quadrigemina and middle lobe of the cerebellum, and are readily compressed by tumours in this situation. The course of the veins at the base is not of much medical importance. The blood from the internal ear passes into the cavernous sinus, that from the mastoid cells into the lateral sinus; and septic clots may thus extend when there is caries of the temporal bone. Many sinuses receive veins from the diploe of the skull.

There is very little communication between the individual veins of the surface, and hence obstruction of one causes grave damage to the cerebral tissue. The sinuses, however, communicate freely, and there are certain communications between the intracranial veins and those outside the skull. The veins of the nose communicate with the anterior extremity of the superior longitudinal sinus, and hence epistaxis relieves venous congestion within the skull. The ophthalmic

vein (going to the cavernous sinus) communicates with the facial, and hence pressure on the sinus causes little or no distension of the retinal veins, because the pressure is quickly relieved. The mastoid veins effect a communication between the lateral sinus and the occipital veins. Moreover, many emissary veins pass through small foramina in the skull, and connect certain sinuses with external veins, the most important being between the longitudinal sinus and the veins of the scalp, and the inferior petrosal sinus and the deep veins in the neck. A further communication, variable in degree, is effected by the veins of the diploe. These communications are important, because they explain, first, the extension of morbid processes from the exterior to the interior of the skull, and secondly, the occasional occurrence of external tumefaction when intracranial sinuses are obstructed by thrombosis.

LECTURE IV.

SYMPTOMS OF BRAIN DISEASE: MECHANISM OF THEIR PRODUCTION—MOTOR PARALYSIS—HEMIPLEGIA.

Gentlemen,—You may remember that in describing to you the principles of the diagnosis of diseases of the spinal cord, I insisted on the importance of keeping not only distinct, but separate in your mind, the two parts of the diagnosis—the seat of the disease and the nature of the disease—the former indicated by the symptoms present, the latter by the mode in which they came on. The distinction is equally important in the diagnosis of diseases of the brain; but the separation is not equally practicable. The symptoms themselves are influenced by the nature of the disease to a far greater extent than in the case of diseases of the spinal cord. After we have ascertained the seat of the disease, we have to determine its nature, and then to consider how far the symptoms present are further explained by the character of the lesion. It is convenient to follow the method adopted in the case of diseases of the spinal cord, and, having considered in the last lecture the most important facts at present ascertained regarding the structure and functions of the brain, to study next the symptoms produced by its diseases, and afterwards the relation of these symptoms to the seat and nature of the morbid change.

Before we study the several symptoms, it is well to know the various mechanisms by which they are produced—mechanisms

common to many morbid processes. The first of these is by the destruction of cerebral tissue. The function of the part destroyed is necessarily lost, and the loss is permanent unless it can be compensated by the action of some other part of the brain. In some parts of the brain the function is, as I have said, diffuse, and extensive compensation may occur. Although a loss of tissue, however small, doubtless has its effect, the evidence of the loss may be scarcely appreciable unless the lesion is extensive. When the loss does occur, it is manifested rather by a general lowering of function than by any special loss. This is the case with those regions of the brain that are probably concerned with the higher intellectual processes, as the prefrontal lobe. Some other functions are performed only by certain structures, and if these are destroyed, that function is permanently lost. Between these two groups there is another, in which special functions may not be permanently lost, even when the part of the brain subserving them is destroyed, because these functions are so related to both hemispheres that the corresponding part of the unaffected hemisphere can supplement that which is destroyed, and act for it. If, however, this second centre is also destroyed, the function is entirely and permanently lost.

Secondly, symptoms may depend on loss of function due to damage that falls short of destruction. The chief mechanisms of this damage are compression and defective supply of arterial blood. In compression both these mechanisms are combined; the pressure necessarily interferes with the flow of blood through the capillaries, and causes anæmia. Hence we do not know how far compression acts mechanically on the nerve-elements, and how far it acts by narrowing the vessels. A moderate compression of a nerve soon arrests conduction through it. If you compress your ulnar nerve behind the elbow, you soon cease to feel in the fingers supplied by it; but even here the compression must render the nerve anæmic, and we cannot infer that the effect on the fibres is simply mechanical. In the brain there is a curious fact regarding pressure which you should remember. Pressure is very much more effective when it is suddenly produced than when it is

slowly produced. The pressure of a cerebral hæmorrhage causes symptoms (that we can certainly refer to the pressure) of much greater intensity than does a tumour, although the latter may be of larger size. It is easier to explain this difference if we assume that pressure acts mechanically than if we suppose that it only causes symptoms by producing anæmia.

A diminished supply of arterial blood also causes loss of function. If the supply is altogether cut off, the loss of function is immediate and absolute. This is true of nerve-fibres as well as of nerve-cells. For a short time, perhaps a day, function may return if the blood-supply is restored; afterwards structural disintegration occurs, and the nerve-elements perish. Remember that arterial blood may be deficient when there is no absolute diminution in the amount of blood in the part. If there is a hindrance to the return of blood by the veins, the over-filled vessels cannot receive blood from the arteries, and so the symptoms of mechanical congestion are to a large extent the same as those of anæmia.

The nerve-elements may be damaged or destroyed by more minute morbid processes, either beginning in the interstitial tissue or in the nerve-elements themselves, such as the various processes of inflammation and degeneration.

The second disturbance of function that results from brain-disease is that which we call "irritation." Irritation causes two effects. First, there may be a morbid increase of activity instead of a diminution; there is evidence of an excessive, although abnormal, liberation of energy. This disturbance may be sudden and paroxysmal, or persistent. The former is often spoken of as "discharge," by an obvious metaphor, which is, indeed, more than a metaphor. Discharge implies a preceding charge. The nerve-energy liberated in the discharge must have been ready for liberation, but restrained. It must have been in a state of "tension"—"held," that is to say. But "held" by what resistance? We do not know. Nevertheless, the fact of a resistance and restraint, co-extensive with the production of nerve-force, helps us much in understanding the phenomena of nerve-action in both health

and disease. It helps us, for instance, to comprehend what has seemed to some so strange a paradox, the fact that disease should cause over-activity. If we conceive, as by all analogy we may, that the restraint is the highest function of nerve-cells,—that the self-control, and the capacity for being controlled, are higher functions than liberation of energy,—we can understand that when, by disease, there is deterioration of function, one effect of this is excessive activity. When the brain is suddenly deprived of blood, one effect often is to cause convulsions: thus the first result of failing function may be the liberation of energy. No doubt in irritation the same process is operative. At the same time we must not deny that some influences may directly augment the energy-producing action of cells, although we have no means of proving such an augmentation.

These considerations enable us to understand something of another and remarkable fact—the second effect of irritation. It may not only cause over-activity of nerve-elements, it may lessen their activity, and even arrest it. This arrest is an example of what physiologists term "inhibition." It may be conceived as an increase of the resistance or restraint. It is remarkable that the same process should sometimes prevent and sometimes permit the liberation of nerve-force, but instances of this are familiar to physiologists. The same stimulus, in different degrees, will either arrest or produce reflex action. In irritation the nerve-tissues directly affected may be inhibited or discharged, or their irritation may inhibit or discharge connected nerve-cells at a distance. It is probable that the nervous system is full of mechanisms whereby the action of certain centres is controlled by that of other centres, and it is probable that the chief mechanism of this association is control, and that what we call the excitation of one centre by another may be very often simply a lowering of control, permitting activity. Thus we can understand that inhibition, as well as excitation, may be a result of the pathological process that we call irritation.

Almost all organic lesions of the brain involve these two processes—damage, complete or incomplete, and irritation.

Their relative degree varies, and still more does their relative duration. In a sudden lesion there is immediate damage and immediate irritation. The irritation soon passes off, unless it is maintained by a secondary more chronic process. The damage that is incomplete also passes away, and with it the symptoms that it has caused. The damage that is complete persists, and its symptoms persist. Hence the symptoms of an acute lesion of the brain are at first far wider and more severe than correspond to the actual destruction. The excess due to slighter damage (as by compression) and to irritation, soon passes away. It has become customary (in Germany especially) to distinguish the two classes as the "direct" and "indirect" symptoms. The terms are convenient, although they are not exact, since almost all the so-called indirect symptoms are, in one sense, the direct effect of the lesion.

In disease that is gradual in development and course, such disease as a tumour, slighter damage and irritation are constantly occurring. The symptoms due to these accompany those due to the destruction by the disease. Hence the symptoms of such disease are often complex, and far more extensive than might have been anticipated. You may now see the meaning of the statement I made just now, that the nature of the lesion has far more influence on the character of the symptoms in disease of the brain, than it has in disease of the spinal cord.

The division into direct and indirect symptoms is founded on the mechanism by which they are produced, and the distinction, as we have seen, is not merely theoretical, but is based on the important fact that, in the case of acute lesions, the indirect symptoms, however obtrusive at the onset, soon pass away, while the direct symptoms persist. Unless the loss can be compensated, it persists as long as life endures. We have now to consider another important division of the symptoms, founded, not on their mechanism, but on their character. Some symptoms, such as local palsy, are due to, and indicate, interference with the function of a definite part of the brain. These are termed "*focal*" symptoms, because

they are due to disease at a given spot. It is not quite in harmony with the modern sense of "focus," although there is always a tendency to associate concentration with limitation; but the sense is not altogether alien to the original meaning of "focus," which, you will remember, is that of a fireplace. Other symptoms, such as loss of consciousness, or delirium, indicate a widespread interference with the function of the brain, and are called "*diffuse*." This distinction, although important and useful, must not be conceived as absolute. Few of our distinctions are absolute. Some symptoms may be diffuse in one case, focal in another, as, for instance, convulsions. Moreover, diffuse processes may cause focal symptoms, and *vice versâ*. Both direct and indirect symptoms may be either focal or diffuse; but it is much more common for direct symptoms to be focal than diffuse, and somewhat more common for indirect symptoms to be diffuse than focal.

In considering in detail the symptoms, irrespective of their cause, we will not follow a strictly logical order, but will take first the symptom that is one of the most common, which we know most about, and therefore should be able to understand best—loss of the power of voluntary motion, motor paralysis. The loss of power may be complete or partial in degree; both have always been, and still are, commonly termed "paralyses." It has of late become fashionable to call the partial loss "paresis," a term of doubtful value except as a means of giving a questionable satisfaction to patients, who find comfort in the mysterious word, and think well of its donor—at any rate until the next physician whom they consult assures them that the disease *is* "paralysis," and that "paresis" is only Greek for weakness. But what is altogether unjustifiable is to assert that partial loss of power is *not* paralysis.

Impairment of motor power is due to interference with the motor centres in the cortex of the brain, or the motor path from them by the internal capsule, crus, pons, and pyramids of the medulla, in the course that we have already traced. Above the pons the two paths are separate, and a

lesion in one crus, or one hemisphere, affects only one motor tract, causing paralysis of the opposite half of the body—"hemiplegia." In the pons the two paths are near together; both may be affected by a single lesion, and yet they are far enough apart for one to be often affected alone. In the anterior pyramids of the medulla they are so near that both often suffer. A lesion here, on one side, affects the arm and leg on the opposite side. Above the medulla the path from the hypoglossal nucleus, having crossed the middle line just above the nucleus, is associated with the tract for the limbs, and so the tongue is paralysed on the same side as the limbs. Above the middle of the pons the facial path joins that of the limbs, and the hemiplegia involves the face. Thus affection of face, tongue, arm, and leg on the same side is the characteristic of complete hemiplegia that results from disease anywhere between the cortex and the middle of the pons. The fibres of the motor path spread out in passing from the crus into the internal capsule, and still more widely in passing through the white substance to the cortex (see Fig. 5, p. 12), the tracts for tongue, face, arm, and leg being to a considerable extent separate, and the separation being greatest at the cortex. Hence, a lesion, even of some size, in the cortex, or in the white substance beneath the cortex, may affect only one or two of these parts, the others escaping. The arm, for instance, is often thus paralysed alone. But even a small lesion of the internal capsule usually affects all the tracts; it must be very small indeed to damage only one of them. If the lesion is in the crus, although it is very small, all are involved. Theoretically, it is true, a lesion in the crus or pons may be so small as to damage only one part; but practically this is scarcely ever met with.

The hemiplegia is always on the side opposite to the cerebral lesion. A few cases have been recorded in which the hemiplegia was on the same side as the lesion, and these have exercised very much the minds of pathologists. But a much more frequent event is to meet with hemiplegia without any discoverable lesion in either hemisphere. Some morbid change must exist in such cases; and a similar un-

discovered lesion in the hemisphere opposite to the paralysis is the most probable explanation of the cases in which the only discovered lesion is on the same side as the hemiplegia. Coexistence does not necessarily involve causation.*

In a case of severe hemiplegia—"complete hemiplegia," as it is called—the paralysis affects one side, but not the whole of one side. The arm and leg are powerless; the face is paralysed chiefly in the lower part; the upper part of the face moves almost as well as on the unparalysed side. The tongue, when protruded, deviates towards the paralysed side (being pushed over by the opposite unopposed genio-glossus); but the muscles of mastication contract equally, or almost equally, in ordinary action, and the two sides of the thorax move equally in ordinary breathing, or if there is an inequality it passes away in a few days. But if the patient makes a strong effort, the masseter on the paralysed side does not contract quite so strongly as the other, and if he takes a deep breath, and brings into action the extraordinary muscles of respiration, there is a distinct defect of expansion of the corresponding half of the thorax. I remember when I was a student, learning from two distinguished physicians—from one that the masseters and respiratory muscles are always weakened in hemiplegia; from the other, that they never are. Both were right, or nearly right. The one had observed only ordinary movements, the other extraordinary movements. Other muscles of the trunk are also weakened—those, for instance, of the back and of the abdomen,—but the degree of weakness is always slight.

Thus some muscles are completely paralysed, some are merely weakened, others are usually not paralysed at all, and are never paralysed much. Moreover, this does not depend on the extent of the disease in the brain. It occurs when the whole of one motor tract is destroyed. This is best explained by an hypothesis, first suggested (in a slightly

* The hypothesis of Morgagni, that there is no decussation on the medulla in these cases, does not receive so much support as might seem from the discovery of the variability of this decussation, because it is certain that the medullary decussation, when deficient, is supplemented in the spinal cord.

different form) by Broadbent. Some muscles are habitually used without their fellows on the other side—as the muscles of one arm. Others are often used with their fellows, but often also alone, as the muscles of extraordinary respiration. Others are never used without their fellows, as the intercostals, the frontales, and the masseters. The degree of paralysis in hemiplegia corresponds roughly to the degree of unilateral use. The muscles of bilateral use are represented in both hemispheres of the brain, and the degree of bilateral representation corresponds to the degree of bilateral use. It may be that the representation of these muscles is rather greater in the opposite hemisphere than in the hemisphere of the same side, or that the nervous arrangements are in greater functional activity in the opposite hemisphere. Hence there is sometimes slight weakness for a short time after the onset of hemiplegia; but the hemisphere of the same side is soon able to innervate them in full degree. It is possible, although not yet proved, that the innervation is affected by pyramidal fibres that do not decussate.

I have said that though muscles of bilateral use may be at first weakened, the weakness soon passes off. In muscles that are of partial bilateral use the weakness also lessens, although less quickly. The legs are used much together, although capable of unilateral use, and the loss of power in the leg never remains absolute, although it may remain absolute in the arm. As a rule, however extensive the lesion, the patient regains ultimately some power in the leg, and commonly enough power to enable him to stand. When hemiplegia occurs in early life, the leg always recovers, and becomes as strong as the other, even when its growth is retarded. Since this occurs when the lesion involves the whole of the motor tract of one hemisphere, it must be through the unaffected hemisphere gaining by use that power over the leg of the same side for which, doubtless, structural arrangements always exist, although they are not called into complete functional activity under normal conditions.

Another effect follows from this double representation of the leg. The lesion of the brain causes at first some weakness

of the leg on the same side as well as on the other, but this soon passes off. Doubtless, if we could measure their strength, the other bilateral muscles would exhibit the same bilateral weakness.

These phenomena present also another aspect. Most of these bilateral movements are automatic—need little or no voluntary effort. Movements are lost in proportion as they require will; persist in proportion as they are automatic. Again, emotional movements are automatic: the will is needed not to cause, but to restrain them. Emotional movement may be preserved when voluntary movement of the same muscle is lost. For instance, if the patient tries to show his teeth, the mouth may be motionless on the paralysed side, and yet if he smiles there may be little or no difference between the two sides. Emotional movements are probably innervated from either hemisphere.

Certain movements are normally effected by non-corresponding muscles of the two sides. Such, for instance, are the lateral movement of the eyes, and the rotation of the head. In the latter the head is turned to one side by some muscles of that side acting with the sterno-mastoid of the other. In hemiplegia these movements are affected, but chiefly during the early stage. It is the movement towards the paralysed side that is lost, and the unopposed antagonists may even cause a slight deviation of the head and eyes towards the unaffected side, *i.e.*, towards the side of the brain diseased—this is termed "conjugate deviation." It shows us very clearly that movements rather than muscles are represented in the brain. The fact that the conjugate deviation occurs, and that it passes away, shows us two things. It proves, first, that these movements, though effected by muscles of both sides, are habitually innervated from the opposite hemisphere, *i.e.*, the head is turned to the right by the left hemisphere of the brain. Secondly, it shows that the movements are also represented in the hemisphere of the same side, by nervous arrangements that may readily be called into effective use.

If a patient is unconscious, we can no longer call his will into action, and are thus deprived of the direct evidence of loss

of voluntary power. If the patient is restless, the absence of movement on one side may be observed; and a pinch of the skin may cause a movement on one side only. There may also be flaccidity of the muscles of the paralysed side, and the limbs, when raised, fall more suddenly than on the unaffected side; or, on the other hand, they may present a distinctly abnormal rigidity. The conjugate deviation of the head and eyes, if present, also indicates a one-sided loss. Moreover, we may sometimes obtain help by observing the state of reflex action.

It is very common, although not invariable, for the reflex action from the skin to be lessened or abolished on the paralysed side. The loss may be observed in the plantar, cremasteric, and abdominal reflexes. It exists from the onset. Why this symptom exists in some cases and not in all, we do not yet know.

The myotatic irritability, evidenced by the so-called "tendon-reflexes," is often unchanged during the first week, and at the end of that time becomes excessive, so that the foot-clonus can be obtained. Sometimes, however, a clonus can be obtained a day or two after the onset, and there may be, immediately after the onset, complete loss of these reactions, so that even the knee-jerk cannot be obtained. The early change is to be ascribed to an influence exerted by the lesion on the spinal centres; the later increase to the secondary degenerative changes in the pyramidal tracts reaching these centres.*

At some period the muscles of the paralysed limbs become rigid, stiffening the limbs in certain postures, and opposing passive movement. Todd first distinguished between "early" and "late" rigidity. The former comes on a few days after the onset, and lasts for a few weeks. The posture of the limbs is that of rest. It is probably due to the irritation of the fibres by inflammatory changes about the lesion. But there is sometimes an "initial rigidity," which developes immediately, and lasts for a few hours or for a day or two.

* See "Diagnosis of Diseases of the Spinal Cord," 3rd Ed., p. 23, for a fuller discussion of this subject.

It is probably due to the irritation of the fibres by the lesion itself. When these forms of rigidity are considerable in degree we can often obtain the foot-clonus. The rigidity is the result of that state of the muscle-reflex centres in which myotatic irritability is increased. Late rigidity comes on in the course of a few weeks, and persists as long as the palsy. The shoulder is adducted, the elbow flexed, the wrist pronated and slightly flexed; the fingers are strongly flexed at the middle and distal phalangeal joints by the contracture of the long flexor. When the wrist is passively flexed, so as to shorten the course of the flexor tendons, the fingers can be straightened without difficulty. Although the flexor contracture preponderates, the extensors also present some rigidity. In the leg the rigidity is more nearly equal in the two sets of muscles, so as to fix the limb in the position of extension. This rigidity depends on active muscular contracture. It lessens much during sleep and when the limb is warm. It can be overcome, for the time, by gentle prolonged extension, especially if the muscles are simultaneously rubbed. After some years tissue-changes take place in the muscles, and they can no longer be extended. Thus we ought to distinguish from the late rigidity an ultimate structural contracture, making in all four varieties—initial, early, late, and structural rigidity. The late rigidity coincides with degeneration in the pyramidal tracts of the cord. Excess of myotatic irritability accompanies it, as it does the degeneration of spinal origin, no doubt for the same reason. The foot-clonus and rectus-clonus can readily be obtained; in the arm, a tap on a muscle, its tendon, or the bone to which it is attached, causes a momentary contraction, and sudden tension may develope a clonus in the flexors of the fingers, and sometimes in the flexors of the elbow, and even in the trapezius.

LECTURE V.

SYMPTOMS (*Continued*): HEMIPLEGIA (*Continued*)—CONVULSION.

GENTLEMEN,—We considered, in the last lecture, the chief characters of hemiplegia, and the condition of the muscles in lasting palsy. Some other points regarding the state of the limbs remain for consideration, the first being the changes that may occur in their nutrition.

The nutrition of the muscles may be unchanged, even after the paralysis has existed for years, or slight general wasting may set in a few weeks after the onset, sometimes slowly attaining a considerable degree, although never comparable to that in progressive muscular atrophy. The electric irritability of the muscles may present no change, or a slight increase in irritability may occur at the end of one or two weeks, and continue for a few months, to give place to a slight and permanent diminution. The change is the same to both forms of electricity, faradaism and voltaism, and is the same in the nerve-trunks as in the muscles. It occurs chiefly when there is the change in nutrition just described, and each is probably the consequence of the irritative character of both the cerebral lesion and the resulting secondary degeneration of the pyramidal tracts in the cord. Although this degeneration never invades the motor nerve-cells as a destructive change, it seems to influence, in slight degree, their nutrition, and therefore that of the motor nerve-fibres and muscles.

Vaso-motor and trophic changes may be absent or very marked. They appear to depend in part, like the changes in muscular nutrition, on the irritative character of the cerebral lesion; but there are centres in the cortex that influence the vaso-motor state of the limbs, and disease of these centres, or of the downward path from them, the precise position and course of which are still undetermined, may be the cause of considerable disturbance of this character. During the early weeks there is often increased warmth of the paralysed limbs, amounting to from half a degree to a degree and a half Fahrenheit, at first uniform, afterwards intermitting. With this there may be increased redness and lividity, sometimes with marked œdema, especially if the kidneys are also diseased. Often there is a tendency to graver trophic changes: blisters readily form, filled with dark serum; the skin sloughs from slight pressure in those parts on which pressure chiefly acts in the recumbent posture—the gluteal region, over the trochanter and malleolus. Rarely there is inflammation of joints.

When recovery occurs, power returns in the proximal parts of the limbs sooner than in the distal parts, and in the leg before the arm. Indeed, as already stated, some recovery in the leg is invariable. There is, moreover, more use of the leg in association with the other, than in its separate movements. The flexion of the foot is that which remains longest defective, and hence the patient cannot get the toes off the ground in bringing the foot forward in the act of walking, and swings the leg round. In the arm, the shoulder recovers before the elbow, and the elbow before the hand. The extensors remain weak longer than the flexors, and the supinators longer than the pronators. In rare cases the hand-movements return first; and it is singular that these cases sometimes also present another exceptional feature—the arm improves faster than the leg. No doubt this peculiarity depends on a special position of the lesion.

The distribution of the palsy in cases of hemiplegia depends on the position and extent of the lesion, and certain

forms need special notice. I have already mentioned the escape of the tongue on the affected side, when the lesion is in the medulla, and of the face when it is in the lower half of the pons; that is to say, when the lesion occurs before the cerebral path from the hypoglossal and facial nucleus has crossed the middle line, and has become associated with the path to the limbs. But the cranial *nerves*, from the third to the hypoglossal, in passing from their nuclei to the surface, may be damaged by disease that damages also the motor tract. This paralyses the nerve on the same side as the lesion, but the limbs on the side opposite to the lesion, thus causing what has been badly termed "alternate hemiplegia." Certain nerves are more frequently paralysed in this way than others: the most frequent are the facial, sixth, and third nerves; less frequently the hypoglossal, auditory, and fifth. The facial and sixth are sometimes affected together on the side opposite to the limbs. The paralysis of the face resembles that due to other diseases of its nerve; all parts of the face are paralysed, and there is loss of faradaic and preservation of voltaic irritability. When the lesion is in the crus, the face is affected on the same side as the limbs, but the third nerve on the opposite side—on the same side as the lesion. Do not imagine that this crossed palsy of limbs and cranial nerves is invariable when hemiplegia results from disease in these regions. The lesion may be so placed, or so small, that the nerves escape. Thus the association of palsy of cranial nerves on one side, with that of the limbs on the other, gives us one class of varieties of hemiplegia.

Another class depends on the incomplete extent of the palsy, the seat of the disease being in the cerebral hemisphere, where the constituent elements of the motor tract have so far diverged that its damage may easily be partial. We have already seen that this must be rare in lesions of the internal capsule, on account of the proximity of the several paths, and that it may more readily occur in the white substance, and most readily in or beneath the convolutions. The paralysis may involve only the face, or the arm, or the leg: or it may involve the face and tongue; face, tongue, and arm; or face

and arm. The distribution depends on the relative position of the centres and paths. Thus the tongue and arm are never affected by a single lesion without the face, because the face-centre and path intervene between the two others. Similarly, the face and leg are never affected without the arm, because the centre and path for the arm intervene between the others. This partial hemiplegia is sometimes called "monoplegia," distinguished, according to its seat, as lingual, facial, brachial, or crural, while combinations receive compound names, as brachio-facial monoplegia. It may seem to you rather anomalous to call the latter a monoplegia, but the whole nomenclature is inconsistent. Strictly, "monoplegia" should designate double hemiplegia; but we call this "diplegia." Thus two "half-palsies" make, not a "one-palsy," but a "two-palsy," and a "one-palsy" is less than a "half-palsy."

In these cases of partial hemiplegia, the paralysis never remains absolute, and usually is not absolute, even at first. The state of the limb resembles that of a hemiplegia that is recovering. The coarse movements in the upper part of the limb are preserved, while the movements of the extremity are impaired or lost.

The affection of sensation that often accompanies hemiplegia I shall describe presently. Before leaving the subject of motor palsy one curious class of symptoms must be mentioned. These are the disorders of movement that sometimes come on some months after the onset. The rigidity of which I have already spoken is fixed, varying but little during the waking hours. But the muscular contractions we are now considering are versatile and changing. They also vary much in different cases. Sometimes there is tremor; fine, quick, rhythmical contractions of the muscles. Rarely there are slow and rhythmical movements, wider in range, and chiefly met with in the hands or fingers. Most common of all are irregular muscular contractions, irregular both in time and in degree, rarely quick, far more often slow. The quick movements somewhat resemble those of chorea, and hence the term, "post-hemiplegic chorea," has been applied

to the whole class of movements—unwisely, because they have nothing to do with chorea, and, moreover, resemble chorea only in rare cases. Generally, the movements are far slower than are seen in true chorea. If not constant, they are readily evoked by an attempt at voluntary movement, or even by attention, and by these they are always increased. Incoordination of movement results, peculiar in character—slow, irregular, spreading movements of the fingers, that have been compared, not inaptly, to the movements of the arms of a cuttlefish. "Mobile spasm," it may be conveniently termed. With this there is often some more constant and unchanging spasm, especially in the flexors of the wrist. The arm is always involved in greater degree than the leg. In the latter the effect of the spasm is chiefly to cause inversion of the foot and over-extension of the great toe; spontaneous movements are rare. The arm is usually adducted at the shoulder-joint; the elbow is sometimes flexed, sometimes strongly extended. Often the arm, straightened out, is carried behind the body. The wrist is frequently flexed; the fingers are usually flexed at the metacarpo-phalangeal joints, extended, and even over-extended at the others, the spasm preponderating in the interosseal muscles. There may be a subluxation of the extended finger-joints, the heads of the phalanges projecting on the palmar aspect. Thus there is a remarkable contrast between this spasm and ordinary late rigidity. In the latter the spasm chiefly affects the long flexors of the fingers; the digits are bent at all joints, a form of flexion that is employed in coarse movements of the limb. In the mobile spasm there is the "interosseal flexion" just described, the flexion that is employed in many delicate operations, such as the act of writing. The continuous action of the muscles often leads to their overgrowth, and the limb may be actually larger in circumference than that of the opposite side, when it is less in length. This condition sometimes developes without preceding hemiplegia; and to such a case, in which there was no fixed spasm, but only the slowly-changing irregular movements, Hammond (of New York) gave the name of "athetosis" (=without fixed position). In the vast majority of cases the condition is a

sequel to hemiplegia. It sometimes comes on after hemiplegia in adult life, but is far more frequent after hemiplegia in infancy and childhood, to which it is, indeed, the common sequel. In adults it has been observed chiefly in cases in which the lesion was in or near the optic thalamus. Why, we cannot yet say. In children it does not seem to be related to any special seat; it follows disease anywhere in the motor regions of the hemisphere. The lesion causing the initial hemiplegia is, however, almost invariably softening, and not hæmorrhage. An analysis of adult cases shows this clearly; and in children any other acute lesion than softening is very rare. These considerations suggest that one element in its causation may be the partial recovery of nerve-cells that are damaged, but not destroyed—which recover, but with disordered functions,—and the greater power of recovery and greater capacity for derangement during the period of development may be the cause of the special frequency of this condition after hemiplegia in early life.

From motor palsy, the spasm last considered naturally leads us to that paroxysmal over-action which causes convulsion. Convulsions are frequent and important symptoms of cerebral disease. They occur under two conditions, apparently as the result of two different mechanisms. First, they occur when there is active irritation of the brain-tissue, such as is produced by inflammation of the brain or membranes, a growing tumour, or a sudden lesion. Secondly, they occur in what are termed "stationary lesions," in which the stage of activity is over, and such structural recovery as may be possible is taking place, or has taken place. In this case they are, apparently, due to the imperfect recovery of damaged nerve-cells, which regain the power of evolving nerve-force, but not the higher power of regulating its discharge. By each mechanism, convulsions are produced most readily when the disease is in the cortex. Stationary lesions scarcely ever cause convulsions unless they are situated in or near the motor cortex. Active irritation is most effective

when in the same region, but it may cause convulsions whatever be its seat. They are also produced by general increase of intracranial pressure, and by diffuse processes, such as meningitis.

The diagnostic significance of convulsions depends on their character. They may be general or they may be partial, either in extent or in commencement. General convulsions constitute a "diffuse" symptom. They are often due to a diffuse and widespread morbid process, inflammatory or degenerative disease away from the motor centres, or to general increase of intracranial pressure. Convulsions that are limited in extent or commencement constitute a focal symptom, and indicate disease in or near the motor region, especially the motor region of the cortex. In general convulsions the loss of consciousness is sudden and immediate; there is usually no aura. In partial convulsions, consciousness is lost late, and the patient is usually aware of the local onset. This local onset is due to the fact that the discharge begins in the centre irritated—for the face, arm, or leg. If very slight, it may not spread beyond the one centre in which it began, the convulsion being confined to the corresponding part. If more severe, it spreads to all the centres of that hemisphere, and the convulsion affects the whole of one side of the body. If still more severe, the discharge spreads to the other hemisphere, and the limbs of the other side are also involved, usually after the first side, sometimes, in the most severe fits, simultaneously. Consciousness is usually retained throughout when the convulsion is confined to one limb; sometimes when it involves the whole of one side; scarcely ever when both sides are involved. Different discharges vary in intensity, and the patient often has slight attacks that are local, and more severe attacks that begin locally and become general. The attack usually commences by clonic spasm, which often becomes tonic if the convulsion becomes severe. The spasm almost always begins in the extremity of the limb. But it is common for a sensory "aura" in the part to precede the spasm: the sensation may pass up the limb first affected, along the side of the trunk, and down the second limb affected, and only then may spasm be added. It is as if the

discharge commenced in the sensory cells of the cortex, passed like a ripple through these sensory centres, and only when it reached their limit was deflected to the motor cells. In the very slightest attacks the sensory aura may be accompanied, not by spasm, but by the opposite condition—sudden powerlessness of the limb. The influence of the sensory discharge on the motor cells may thus be to inhibit them, if it is of very slight degree—another instance of the relation of inhibition and discharge mentioned on p. 47. Special sense auræ are very rare in organic disease; when they occur they indicate that the disease is in or near the part of the cortex that subserves the corresponding functions. Thus, I have met with a case in which a flash of light was the aura of fits due to a tumour of the occipital lobe; and another, of a tumour beneath the temporo-sphenoidal cortex, in which the sound of bells was the aura.

A convulsion leaves behind it a transient weakness in the part convulsed, probably due to exhaustion when the spasm is severe, or to motor inhibition when the spasm is slight or the discharge is limited to the sensory cells, sometimes perhaps to both mechanisms. The weakness after a unilateral fit may resemble slight hemiplegia, and, if the convulsions recur at short intervals, the residual palsy accumulates until it may amount to absolute powerlessness of the side, with increased knee-jerk and foot-clonus. It gradually passes away after the convulsions are over.

The characteristic, then, of the convulsions of organic brain-disease is their local commencement. Whether the fit remains limited to the part in which it commences, or spreads to other parts, even the whole body, is a question of degree. Moreover, a local aura, without convulsion, has the same significance as local spasm. It indicates that the discharge commences at one part of the brain, that there is at that part a morbid state, and it is thus a focal symptom. Convulsions that are general from the first are often due to, and symptoms of, organic brain-disease; but they derive their significance from associated symptoms, and not from their character. Without such symptoms they suggest idiopathic epilepsy, not the organic brain-disease that local commencement suggests. I say

suggests, because there is sometimes a local commencement in idiopathic epilepsy. This is, however, rare, and you would not think of idiopathic epilepsy in such a case unless you had ascertained that other symptoms of organic brain-disease were absent, and you could not feel confident, unless the convulsions had existed alone for a considerable time, or there was a strong family tendency to the idiopathic disease.

The first convulsions from organic disease are sometimes excited by a cause outside the central nervous system, a cause that may seem to be adequate to account for them. This is a very important point. Remember that the apparent cause of a fit is scarcely ever more than its excitant—an excitant that is effective only because there is a predisposition; and the predisposition is the important matter to be sought out and to be treated. The spark would be harmless unless it fell on gunpowder. The predisposition may be sometimes an inherited tendency of the nervous system, or it may be an acquired state of morbid irritability, such as forms part of the constitutional disease we call "rickets," or it may be a brain disease that has not reached such a degree as to cause pronounced symptoms. For instance, a child was brought in here who had swallowed a slate pencil, and then had an attack of general convulsions. For this the slate pencil seemed an adequate cause. It was passed by the bowel, and the child seemed well. But six weeks afterwards she was dead, and the cause of death was a large infiltrating glioma of the pons, which must have existed at the time of the convulsion, and no doubt gave rise to the predisposition. As in this case, the convulsion so excited is usually general. But only a few weeks ago, you may remember, we had a man in Ward IV., whose history showed that a convulsion beginning locally may be excited by a local cause, even though it ultimately depends on organic brain-disease. The man, when apparently well, struck his left arm against an iron hook, and a few hours afterwards had a fit which began in this arm, and was heralded by a pain at the part struck. He afterwards had other similar fits. It seemed like a case of what is termed "reflex epilepsy"—a disease, by the way, much

more rare than you may imagine from books. But one day he had a fit which began in the foot, and not in the arm. Then he developed headache, double optic neuritis, and hemiplegia; and when he came in here, he presented the characteristic symptoms of a tumour in the upper part of the ascending frontal convolution. The blow on the arm had determined the time and place of an explosion, of which the real cause was the organic brain-disease. Such a case is the converse of the arrest of a fit, beginning locally, by a ligature round the limb, when the fit is due to organic brain-disease, and the ligature can only stop the fit by acting on the centre in the brain.

Remember, then, that convulsions should not be lightly dismissed from consideration because the fit succeeded an exciting cause.

Sufferers from organic brain-disease who are of the age and sex in which the predisposition to hysteria exists, may suffer from attacks of hysteroid convulsion. It is not surprising that so potent a disturbing influence should evoke into activity a tendency from which few women are altogether free. But the fact is of extreme diagnostic importance, because, when one of a set of symptoms is unmistakable in character, it is natural to take it as an index to the series. You should never do so, gentlemen, until you have satisfied yourselves that no symptom is certainly of a different nature. I insisted on this in speaking of the diagnosis of diseases of the spinal cord, and it is equally true of those of the brain. Both acute and chronic disease may lead to the strange disturbance of function to which we give the name hysteria, and which we might just as well call, as did our forefathers, "fits of the mother." I have seen it, for instance, many times in cases of cerebral tumour, many times in tubercular meningitis, and once at the onset of an attack of embolic hemiplegia. It is a very frequent consequence of the defective development of the brain that results from infantile lesions. Some of you may remember a curious girl who attended here for a long time, whose powerless and

conditions of the cerebral circulation. I will briefly mention to you the facts of chief importance. Those that concern the arterial system have been ascertained by the investigations of Duret in France, and Heubner in Germany.

The blood-supply to the brain comes from the carotid and vertebral arteries. The left carotid arises from the aorta nearly in the direction of the current of blood that courses through the aortic arch; while the right carotid comes from the innominate, and this arises from the aorta nearly at right angles to the current of blood. Hence solid particles are rather more readily carried into the left than into the right carotid—a circumstance that explains the somewhat greater frequency of embolism on the left side of the brain. There is a similar but still greater difference in the origin of the two vertebrals, and it is probably for this reason that the left vertebral is often larger than the right. This does not determine any difference in the frequency of vascular lesions in the part of the cerebrum supplied, because the blood brought by both vertebrals has to pass through the common basilar. The internal carotid, on each side, divides into a small anterior cerebral, and larger middle cerebral. The latter continues the direction of the internal carotid, and hence plugs carried from the heart readily pass into it. The "circle of Willis," you will remember, is formed by these vessels, together with the anterior communicating artery, between the anterior cerebrals, and two posterior communicating (one on each side), between the middle and posterior cerebrals.

From the circle of Willis and the commencement of the three cerebral arteries (anterior, middle, and posterior) small branches arise which supply the central ganglia and adjacent white substance of the hemisphere, while the three arteries ramify over the surface of the brain, and supply the grey cortex and the chief part of the white substance. Thus there are two systems of branches, central and cortical. Between these two systems there are no anastomoses. The central branches do not communicate with each other, and hence obstruction of one causes necrosis of the region supplied, no

collateral supply of blood being possible. The cortical branches vary in this respect in different individuals. In some persons there is enough communication between the cortical branches to maintain nutrition if one is obstructed; in other persons there is no communication. Hence obstruction of the middle cerebral at its origin always causes necrotic softening of the part of the central ganglia that it supplies, and sometimes also softening of the cortex; while in other cases, with a similar obstruction, we may find that the cortex is intact, although the central ganglia are extensively damaged.

The branches to the ganglia must engage our attention in further detail, since the pathological importance of these "central arteries" is very great. They are divided into six groups, of which two are medial and small, and four (two on each side) are lateral and very important. The anterior medial group is given off by the anterior cerebrals and anterior communicating artery, and supply the anterior extremity of the caudate nucleus. The posterior medial group consists of twigs given off by the posterior cerebrals near their origin. Passing through the posterior perforated spot, they supply the inner part of the optic thalamus and the walls of the third ventricle. These two medial groups are insignificant in the extent of the brain supplied by them, but of some pathological importance, since hæmorrhage, from their rupture, is apt to burst into the ventricles. The lateral groups furnish the blood to the chief part of the central ganglia and the internal capsule. The anterior lateral group consists of numerous small arteries that arise from the middle cerebral in the first inch of its course, pass through the anterior perforated space, and supply the caudate nucleus (except its head), the lenticular nucleus, internal capsule, and part of the optic thalamus. Some pass through the inner part of the lenticular nucleus to the internal capsule; while others pass first outside the lenticular nucleus, and then through its outer portion to the capsule. They supply the caudate nucleus and optic thalamus after passing through the capsule. These vessels are prone to rupture, perhaps because their direct origin from a comparatively large vessel exposes them to

a high blood-pressure. Hence the frequency of cerebral hæmorrhage in this situation. The arteries of the posterior lateral group arise from the posterior cerebral, and supply the hinder part of the optic thalamus. Hæmorrhage from their rupture usually damages the posterior (sensory) part of the capsule, and often extends into the crus. Branches from the posterior cerebral supply also the crus and corpora quadrigemina.

Of the blood-supply to the cortex, that from the middle cerebral is both the most extensive and the most important, embracing, as it does, the central (motor) convolutions. The general arrangement of the branches of each artery is the same: each divides and ramifies, and from the branches, and the ultimate ramifications in the pia mater, twigs are given off to the cerebral substance—some short, that end in the grey cortex; others long, that pass through the grey cortex to the white substance, extending in it to various depths. The regions supplied by the several vessels are as follows:—The anterior cerebral, curving round the corpus callosum, supplies, by three branches, part of the orbital lobule, and the inner surface of the hemisphere as far as the quadrate lobule (precuneus). It also supplies, on the outer surface, by branches that come over from the inner surface, the first and second frontal convolutions, and the highest part of the ascending frontal. The middle cerebral or "Sylvian artery" divides in the fissure of Sylvius, opposite the island of Reil, into four branches, which lie in the sulci of the insula, and then pass—the first to the inferior frontal convolution; the second to the ascending frontal, except the highest part, which is supplied by the anterior cerebral; the third to the whole of the ascending parietal, and the adjacent part of the inferior parietal lobule; the fourth to the convolutions about the posterior limb of the fissure of Sylvius, supramarginal and angular, the hinder part of the superior parietal lobule, and the first temporal. From this vessel, near its origin, one or two large branches arise that supply the greater part of the second and third temporal convolutions. The posterior cerebral supplies the occipital lobe, and also the inferior aspect of the temporal lobe, by three

branches, of which one goes to the lower part of the uncinate convolution, a second to the inferior part of the temporal lobe, and a third to the cuneus, lingual convolution, and the inner and outer surfaces of the occipital lobe.

Thus, the middle cerebral supplies the motor region, both central and cortical, except in part the leg-centre; it also supplies the part of the cortex that subserves cutaneous sensibility, the cortical auditory centre, and probably the higher visual centre; it supplies all the cortical regions concerned in speech-processes in the left hemisphere, motor, auditory, and visual. The anterior cerebral supplies only a small part of the motor region, viz., the part of the leg-centre that occupies the paracentral lobule and highest part of the ascending frontal. The posterior cerebral supplies the visual path, from the middle of the tract backwards, and the half-vision centre in the occipital lobe; it supplies also the corpora quadrigemina, and the sensory part of the internal capsule.

The pons and medulla receive small arteries from all the adjacent vessels, vertebrals, basilar, and cerebellar arteries. These branches are in two sets—median, near the middle line; and lateral or radicular, that pass in at the side, near the chief nerve-roots. Both pass back to the nuclei near the floor of the fourth ventricle, but the chief blood-supply to these nuclei comes from the median branches. Of the cerebellar arteries, the superior and inferior supply the corresponding regions of each hemisphere, and the upper surface is also supplied by a large branch from the basilar opposite the middle of the pons, the middle cerebellar artery. These arteries communicate freely, and hence necrotic softening of the cerebellum is rare. The branches to the medulla and pons do not communicate, and hence here necrotic softening is common.

The venous circulation of the brain presents several important peculiarities. The veins from the greater part of the cortex pass upwards to the longitudinal sinus, and open into this in a forward direction. This arrangement involves two very unusual conditions. Elsewhere, the blood from ascend-

ing arteries passes into descending veins, so that the feeble pressure that passes through the capillaries is supplemented by the influence of gravitation. Elsewhere, ascending veins convey blood that has been brought by descending arteries, and the venous flow is aided by the liquid pressure, which, according to the well-known law of hydrostatics, tends to make the blood rise in the veins. But on the brain, the blood from ascending arteries passes into ascending veins. The openings of these veins into the longitudinal sinus being directed forwards, the entering blood is opposed in direction to the current in the sinus, and the effect must be to retard the flow in both veins and sinus. Moreover, in the erect posture the anterior half of the longitudinal sinus has also an ascending course, while the trabeculæ that occupy the lumen of the sinus must offer some hindrance to the movement of the blood. These circumstances help us to understand the readiness with which clots form in the cortical veins and longitudinal sinus, when other circumstances favour the coagulation of the blood. Indeed, the marvel is that thrombosis is not more common than it is.

The veins of Galen, conveying blood from the ventricles to the straight sinus, pass above the corpora quadrigemina and middle lobe of the cerebellum, and are readily compressed by tumours in this situation. The course of the veins at the base is not of much medical importance. The blood from the internal ear passes into the cavernous sinus, that from the mastoid cells into the lateral sinus; and septic clots may thus extend when there is caries of the temporal bone. Many sinuses receive veins from the diploe of the skull.

There is very little communication between the individual veins of the surface, and hence obstruction of one causes grave damage to the cerebral tissue. The sinuses, however, communicate freely, and there are certain communications between the intracranial veins and those outside the skull. The veins of the nose communicate with the anterior extremity of the superior longitudinal sinus, and hence epistaxis relieves venous congestion within the skull. The ophthalmic

vein (going to the cavernous sinus) communicates with the facial, and hence pressure on the sinus causes little or no distension of the retinal veins, because the pressure is quickly relieved. The mastoid veins effect a communication between the lateral sinus and the occipital veins. Moreover, many emissary veins pass through small foramina in the skull, and connect certain sinuses with external veins, the most important being between the longitudinal sinus and the veins of the scalp, and the inferior petrosal sinus and the deep veins in the neck. A further communication, variable in degree, is effected by the veins of the diploe. These communications are important, because they explain, first, the extension of morbid processes from the exterior to the interior of the skull, and secondly, the occasional occurrence of external tumefaction when intracranial sinuses are obstructed by thrombosis.

LECTURE IV.

SYMPTOMS OF BRAIN DISEASE: MECHANISM OF THEIR PRODUCTION—MOTOR PARALYSIS—HEMIPLEGIA.

Gentlemen,—You may remember that in describing to you the principles of the diagnosis of diseases of the spinal cord, I insisted on the importance of keeping not only distinct, but separate in your mind, the two parts of the diagnosis—the seat of the disease and the nature of the disease—the former indicated by the symptoms present, the latter by the mode in which they came on. The distinction is equally important in the diagnosis of diseases of the brain; but the separation is not equally practicable. The symptoms themselves are influenced by the nature of the disease to a far greater extent than in the case of diseases of the spinal cord. After we have ascertained the seat of the disease, we have to determine its nature, and then to consider how far the symptoms present are further explained by the character of the lesion. It is convenient to follow the method adopted in the case of diseases of the spinal cord, and, having considered in the last lecture the most important facts at present ascertained regarding the structure and functions of the brain, to study next the symptoms produced by its diseases, and afterwards the relation of these symptoms to the seat and nature of the morbid change.

Before we study the several symptoms, it is well to know the various mechanisms by which they are produced—mechanisms

common to many morbid processes. The first of these is by the destruction of cerebral tissue. The function of the part destroyed is necessarily lost, and the loss is permanent unless it can be compensated by the action of some other part of the brain. In some parts of the brain the function is, as I have said, diffuse, and extensive compensation may occur. Although a loss of tissue, however small, doubtless has its effect, the evidence of the loss may be scarcely appreciable unless the lesion is extensive. When the loss does occur, it is manifested rather by a general lowering of function than by any special loss. This is the case with those regions of the brain that are probably concerned with the higher intellectual processes, as the prefrontal lobe. Some other functions are performed only by certain structures, and if these are destroyed, that function is permanently lost. Between these two groups there is another, in which special functions may not be permanently lost, even when the part of the brain subserving them is destroyed, because these functions are so related to both hemispheres that the corresponding part of the unaffected hemisphere can supplement that which is destroyed, and act for it. If, however, this second centre is also destroyed, the function is entirely and permanently lost.

Secondly, symptoms may depend on loss of function due to damage that falls short of destruction. The chief mechanisms of this damage are compression and defective supply of arterial blood. In compression both these mechanisms are combined; the pressure necessarily interferes with the flow of blood through the capillaries, and causes anæmia. Hence we do not know how far compression acts mechanically on the nerve-elements, and how far it acts by narrowing the vessels. A moderate compression of a nerve soon arrests conduction through it. If you compress your ulnar nerve behind the elbow, you soon cease to feel in the fingers supplied by it; but even here the compression must render the nerve anæmic, and we cannot infer that the effect on the fibres is simply mechanical. In the brain there is a curious fact regarding pressure which you should remember. Pressure is very much more effective when it is suddenly produced than when it is

slowly produced. The pressure of a cerebral hæmorrhage causes symptoms (that we can certainly refer to the pressure) of much greater intensity than does a tumour, although the latter may be of larger size. It is easier to explain this difference if we assume that pressure acts mechanically than if we suppose that it only causes symptoms by producing anæmia.

A diminished supply of arterial blood also causes loss of function. If the supply is altogether cut off, the loss of function is immediate and absolute. This is true of nerve-fibres as well as of nerve-cells. For a short time, perhaps a day, function may return if the blood-supply is restored; afterwards structural disintegration occurs, and the nerve-elements perish. Remember that arterial blood may be deficient when there is no absolute diminution in the amount of blood in the part. If there is a hindrance to the return of blood by the veins, the over-filled vessels cannot receive blood from the arteries, and so the symptoms of mechanical congestion are to a large extent the same as those of anæmia.

The nerve-elements may be damaged or destroyed by more minute morbid processes, either beginning in the interstitial tissue or in the nerve-elements themselves, such as the various processes of inflammation and degeneration.

The second disturbance of function that results from brain-disease is that which we call "irritation." Irritation causes two effects. First, there may be a morbid increase of activity instead of a diminution; there is evidence of an excessive, although abnormal, liberation of energy. This disturbance may be sudden and paroxysmal, or persistent. The former is often spoken of as "discharge," by an obvious metaphor, which is, indeed, more than a metaphor. Discharge implies a preceding charge. The nerve-energy liberated in the discharge must have been ready for liberation, but restrained. It must have been in a state of "tension"—"held," that is to say. But "held" by what resistance? We do not know. Nevertheless, the fact of a resistance and restraint, co-extensive with the production of nerve-force, helps us much in understanding the phenomena of nerve-action in both health

and disease. It helps us, for instance, to comprehend what has seemed to some so strange a paradox, the fact that disease should cause over-activity. If we conceive, as by all analogy we may, that the restraint is the highest function of nerve-cells,—that the self-control, and the capacity for being controlled, are higher functions than liberation of energy,—we can understand that when, by disease, there is deterioration of function, one effect of this is excessive activity. When the brain is suddenly deprived of blood, one effect often is to cause convulsions: thus the first result of failing function may be the liberation of energy. No doubt in irritation the same process is operative. At the same time we must not deny that some influences may directly augment the energy-producing action of cells, although we have no means of proving such an augmentation.

These considerations enable us to understand something of another and remarkable fact—the second effect of irritation. It may not only cause over-activity of nerve-elements, it may lessen their activity, and even arrest it. This arrest is an example of what physiologists term "inhibition." It may be conceived as an increase of the resistance or restraint. It is remarkable that the same process should sometimes prevent and sometimes permit the liberation of nerve-force, but instances of this are familiar to physiologists. The same stimulus, in different degrees, will either arrest or produce reflex action. In irritation the nerve-tissues directly affected may be inhibited or discharged, or their irritation may inhibit or discharge connected nerve-cells at a distance. It is probable that the nervous system is full of mechanisms whereby the action of certain centres is controlled by that of other centres, and it is probable that the chief mechanism of this association is control, and that what we call the excitation of one centre by another may be very often simply a lowering of control, permitting activity. Thus we can understand that inhibition, as well as excitation, may be a result of the pathological process that we call irritation.

Almost all organic lesions of the brain involve these two processes—damage, complete or incomplete, and irritation.

Their relative degree varies, and still more does their relative duration. In a sudden lesion there is immediate damage and immediate irritation. The irritation soon passes off, unless it is maintained by a secondary more chronic process. The damage that is incomplete also passes away, and with it the symptoms that it has caused. The damage that is complete persists, and its symptoms persist. Hence the symptoms of an acute lesion of the brain are at first far wider and more severe than correspond to the actual destruction. The excess due to slighter damage (as by compression) and to irritation, soon passes away. It has become customary (in Germany especially) to distinguish the two classes as the "direct" and "indirect" symptoms. The terms are convenient, although they are not exact, since almost all the so-called indirect symptoms are, in one sense, the direct effect of the lesion.

In disease that is gradual in development and course, such disease as a tumour, slighter damage and irritation are constantly occurring. The symptoms due to these accompany those due to the destruction by the disease. Hence the symptoms of such disease are often complex, and far more extensive than might have been anticipated. You may now see the meaning of the statement I made just now, that the nature of the lesion has far more influence on the character of the symptoms in disease of the brain, than it has in disease of the spinal cord.

The division into direct and indirect symptoms is founded on the mechanism by which they are produced, and the distinction, as we have seen, is not merely theoretical, but is based on the important fact that, in the case of acute lesions, the indirect symptoms, however obtrusive at the onset, soon pass away, while the direct symptoms persist. Unless the loss can be compensated, it persists as long as life endures. We have now to consider another important division of the symptoms, founded, not on their mechanism, but on their character. Some symptoms, such as local palsy, are due to, and indicate, interference with the function of a definite part of the brain. These are termed "*focal*" symptoms, because

they are due to disease at a given spot. It is not quite in harmony with the modern sense of "focus," although there is always a tendency to associate concentration with limitation; but the sense is not altogether alien to the original meaning of "focus," which, you will remember, is that of a fireplace. Other symptoms, such as loss of consciousness, or delirium, indicate a widespread interference with the function of the brain, and are called "*diffuse*." This distinction, although important and useful, must not be conceived as absolute. Few of our distinctions are absolute. Some symptoms may be diffuse in one case, focal in another, as, for instance, convulsions. Moreover, diffuse processes may cause focal symptoms, and *vice versâ*. Both direct and indirect symptoms may be either focal or diffuse; but it is much more common for direct symptoms to be focal than diffuse, and somewhat more common for indirect symptoms to be diffuse than focal.

In considering in detail the symptoms, irrespective of their cause, we will not follow a strictly logical order, but will take first the symptom that is one of the most common, which we know most about, and therefore should be able to understand best—loss of the power of voluntary motion, motor paralysis. The loss of power may be complete or partial in degree; both have always been, and still are, commonly termed "paralyses." It has of late become fashionable to call the partial loss "paresis," a term of doubtful value except as a means of giving a questionable satisfaction to patients, who find comfort in the mysterious word, and think well of its donor—at any rate until the next physician whom they consult assures them that the disease *is* "paralysis," and that "paresis" is only Greek for weakness. But what is altogether unjustifiable is to assert that partial loss of power is *not* paralysis.

Impairment of motor power is due to interference with the motor centres in the cortex of the brain, or the motor path from them by the internal capsule, crus, pons, and pyramids of the medulla, in the course that we have already traced. Above the pons the two paths are separate, and a

lesion in one crus, or one hemisphere, affects only one motor tract, causing paralysis of the opposite half of the body—"hemiplegia." In the pons the two paths are near together; both may be affected by a single lesion, and yet they are far enough apart for one to be often affected alone. In the anterior pyramids of the medulla they are so near that both often suffer. A lesion here, on one side, affects the arm and leg on the opposite side. Above the medulla the path from the hypoglossal nucleus, having crossed the middle line just above the nucleus, is associated with the tract for the limbs, and so the tongue is paralysed on the same side as the limbs. Above the middle of the pons the facial path joins that of the limbs, and the hemiplegia involves the face. Thus affection of face, tongue, arm, and leg on the same side is the characteristic of complete hemiplegia that results from disease anywhere between the cortex and the middle of the pons. The fibres of the motor path spread out in passing from the crus into the internal capsule, and still more widely in passing through the white substance to the cortex (see Fig. 5, p. 12), the tracts for tongue, face, arm, and leg being to a considerable extent separate, and the separation being greatest at the cortex. Hence, a lesion, even of some size, in the cortex, or in the white substance beneath the cortex, may affect only one or two of these parts, the others escaping. The arm, for instance, is often thus paralysed alone. But even a small lesion of the internal capsule usually affects all the tracts; it must be very small indeed to damage only one of them. If the lesion is in the crus, although it is very small, all are involved. Theoretically, it is true, a lesion in the crus or pons may be so small as to damage only one part; but practically this is scarcely ever met with.

The hemiplegia is always on the side opposite to the cerebral lesion. A few cases have been recorded in which the hemiplegia was on the same side as the lesion, and these have exercised very much the minds of pathologists. But a much more frequent event is to meet with hemiplegia without any discoverable lesion in either hemisphere. Some morbid change must exist in such cases; and a similar un-

discovered lesion in the hemisphere opposite to the paralysis is the most probable explanation of the cases in which the only discovered lesion is on the same side as the hemiplegia. Coexistence does not necessarily involve causation.*

In a case of severe hemiplegia—"complete hemiplegia," as it is called—the paralysis affects one side, but not the whole of one side. The arm and leg are powerless; the face is paralysed chiefly in the lower part; the upper part of the face moves almost as well as on the unparalysed side. The tongue, when protruded, deviates towards the paralysed side (being pushed over by the opposite unopposed genio-glossus); but the muscles of mastication contract equally, or almost equally, in ordinary action, and the two sides of the thorax move equally in ordinary breathing, or if there is an inequality it passes away in a few days. But if the patient makes a strong effort, the masseter on the paralysed side does not contract quite so strongly as the other, and if he takes a deep breath, and brings into action the extraordinary muscles of respiration, there is a distinct defect of expansion of the corresponding half of the thorax. I remember when I was a student, learning from two distinguished physicians—from one that the masseters and respiratory muscles are always weakened in hemiplegia; from the other, that they never are. Both were right, or nearly right. The one had observed only ordinary movements, the other extraordinary movements. Other muscles of the trunk are also weakened—those, for instance, of the back and of the abdomen,—but the degree of weakness is always slight.

Thus some muscles are completely paralysed, some are merely weakened, others are usually not paralysed at all, and are never paralysed much. Moreover, this does not depend on the extent of the disease in the brain. It occurs when the whole of one motor tract is destroyed. This is best explained by an hypothesis, first suggested (in a slightly

* The hypothesis of Morgagni, that there is no decussation on the medulla in these cases, does not receive so much support as might seem from the discovery of the variability of this decussation, because it is certain that the medullary decussation, when deficient, is supplemented in the spinal cord.

different form) by Broadbent. Some muscles are habitually used without their fellows on the other side—as the muscles of one arm. Others are often used with their fellows, but often also alone, as the muscles of extraordinary respiration. Others are never used without their fellows, as the intercostals, the frontales, and the masseters. The degree of paralysis in hemiplegia corresponds roughly to the degree of unilateral use. The muscles of bilateral use are represented in both hemispheres of the brain, and the degree of bilateral representation corresponds to the degree of bilateral use. It may be that the representation of these muscles is rather greater in the opposite hemisphere than in the hemisphere of the same side, or that the nervous arrangements are in greater functional activity in the opposite hemisphere. Hence there is sometimes slight weakness for a short time after the onset of hemiplegia; but the hemisphere of the same side is soon able to innervate them in full degree. It is possible, although not yet proved, that the innervation is affected by pyramidal fibres that do not decussate.

I have said that though muscles of bilateral use may be at first weakened, the weakness soon passes off. In muscles that are of partial bilateral use the weakness also lessens, although less quickly. The legs are used much together, although capable of unilateral use, and the loss of power in the leg never remains absolute, although it may remain absolute in the arm. As a rule, however extensive the lesion, the patient regains ultimately some power in the leg, and commonly enough power to enable him to stand. When hemiplegia occurs in early life, the leg always recovers, and becomes as strong as the other, even when its growth is retarded. Since this occurs when the lesion involves the whole of the motor tract of one hemisphere, it must be through the unaffected hemisphere gaining by use that power over the leg of the same side for which, doubtless, structural arrangements always exist, although they are not called into complete functional activity under normal conditions.

Another effect follows from this double representation of the leg. The lesion of the brain causes at first some weakness

of the leg on the same side as well as on the other, but this soon passes off. Doubtless, if we could measure their strength, the other bilateral muscles would exhibit the same bilateral weakness.

These phenomena present also another aspect. Most of these bilateral movements are automatic—need little or no voluntary effort. Movements are lost in proportion as they require will; persist in proportion as they are automatic. Again, emotional movements are automatic: the will is needed not to cause, but to restrain them. Emotional movement may be preserved when voluntary movement of the same muscle is lost. For instance, if the patient tries to show his teeth, the mouth may be motionless on the paralysed side, and yet if he smiles there may be little or no difference between the two sides. Emotional movements are probably innervated from either hemisphere.

Certain movements are normally effected by non-corresponding muscles of the two sides. Such, for instance, are the lateral movement of the eyes, and the rotation of the head. In the latter the head is turned to one side by some muscles of that side acting with the sterno-mastoid of the other. In hemiplegia these movements are affected, but chiefly during the early stage. It is the movement towards the paralysed side that is lost, and the unopposed antagonists may even cause a slight deviation of the head and eyes towards the unaffected side, *i.e.*, towards the side of the brain diseased—this is termed "conjugate deviation." It shows us very clearly that movements rather than muscles are represented in the brain. The fact that the conjugate deviation occurs, and that it passes away, shows us two things. It proves, first, that these movements, though effected by muscles of both sides, are habitually innervated from the opposite hemisphere, *i.e.*, the head is turned to the right by the left hemisphere of the brain. Secondly, it shows that the movements are also represented in the hemisphere of the same side, by nervous arrangements that may readily be called into effective use.

If a patient is unconscious, we can no longer call his will into action, and are thus deprived of the direct evidence of loss

of voluntary power. If the patient is restless, the absence of movement on one side may be observed; and a pinch of the skin may cause a movement on one side only. There may also be flaccidity of the muscles of the paralysed side, and the limbs, when raised, fall more suddenly than on the unaffected side; or, on the other hand, they may present a distinctly abnormal rigidity. The conjugate deviation of the head and eyes, if present, also indicates a one-sided loss. Moreover, we may sometimes obtain help by observing the state of reflex action.

It is very common, although not invariable, for the reflex action from the skin to be lessened or abolished on the paralysed side. The loss may be observed in the plantar, cremasteric, and abdominal reflexes. It exists from the onset. Why this symptom exists in some cases and not in all, we do not yet know.

The myotatic irritability, evidenced by the so-called "tendon-reflexes," is often unchanged during the first week, and at the end of that time becomes excessive, so that the foot-clonus can be obtained. Sometimes, however, a clonus can be obtained a day or two after the onset, and there may be, immediately after the onset, complete loss of these reactions, so that even the knee-jerk cannot be obtained. The early change is to be ascribed to an influence exerted by the lesion on the spinal centres; the later increase to the secondary degenerative changes in the pyramidal tracts reaching these centres.*

At some period the muscles of the paralysed limbs become rigid, stiffening the limbs in certain postures, and opposing passive movement. Todd first distinguished between "early" and "late" rigidity. The former comes on a few days after the onset, and lasts for a few weeks. The posture of the limbs is that of rest. It is probably due to the irritation of the fibres by inflammatory changes about the lesion. But there is sometimes an "initial rigidity," which developes immediately, and lasts for a few hours or for a day or two.

* See "Diagnosis of Diseases of the Spinal Cord," 3rd Ed., p. 23, for a fuller discussion of this subject.

It is probably due to the irritation of the fibres by the lesion itself. When these forms of rigidity are considerable in degree we can often obtain the foot-clonus. The rigidity is the result of that state of the muscle-reflex centres in which myotatic irritability is increased. Late rigidity comes on in the course of a few weeks, and persists as long as the palsy. The shoulder is adducted, the elbow flexed, the wrist pronated and slightly flexed; the fingers are strongly flexed at the middle and distal phalangeal joints by the contracture of the long flexor. When the wrist is passively flexed, so as to shorten the course of the flexor tendons, the fingers can be straightened without difficulty. Although the flexor contracture preponderates, the extensors also present some rigidity. In the leg the rigidity is more nearly equal in the two sets of muscles, so as to fix the limb in the position of extension. This rigidity depends on active muscular contracture. It lessens much during sleep and when the limb is warm. It can be overcome, for the time, by gentle prolonged extension, especially if the muscles are simultaneously rubbed. After some years tissue-changes take place in the muscles, and they can no longer be extended. Thus we ought to distinguish from the late rigidity an ultimate structural contracture, making in all four varieties—initial, early, late, and structural rigidity. The late rigidity coincides with degeneration in the pyramidal tracts of the cord. Excess of myotatic irritability accompanies it, as it does the degeneration of spinal origin, no doubt for the same reason. The foot-clonus and rectus-clonus can readily be obtained; in the arm, a tap on a muscle, its tendon, or the bone to which it is attached, causes a momentary contraction, and sudden tension may develope a clonus in the flexors of the fingers, and sometimes in the flexors of the elbow, and even in the trapezius.

LECTURE V.

SYMPTOMS (*Continued*): HEMIPLEGIA (*Continued*)—CONVULSION.

GENTLEMEN,—We considered, in the last lecture, the chief characters of hemiplegia, and the condition of the muscles in lasting palsy. Some other points regarding the state of the limbs remain for consideration, the first being the changes that may occur in their nutrition.

The nutrition of the muscles may be unchanged, even after the paralysis has existed for years, or slight general wasting may set in a few weeks after the onset, sometimes slowly attaining a considerable degree, although never comparable to that in progressive muscular atrophy. The electric irritability of the muscles may present no change, or a slight increase in irritability may occur at the end of one or two weeks, and continue for a few months, to give place to a slight and permanent diminution. The change is the same to both forms of electricity, faradaism and voltaism, and is the same in the nerve-trunks as in the muscles. It occurs chiefly when there is the change in nutrition just described, and each is probably the consequence of the irritative character of both the cerebral lesion and the resulting secondary degeneration of the pyramidal tracts in the cord. Although this degeneration never invades the motor nerve-cells as a destructive change, it seems to influence, in slight degree, their nutrition, and therefore that of the motor nerve-fibres and muscles.

Vaso-motor and trophic changes may be absent or very marked. They appear to depend in part, like the changes in muscular nutrition, on the irritative character of the cerebral lesion; but there are centres in the cortex that influence the vaso-motor state of the limbs, and disease of these centres, or of the downward path from them, the precise position and course of which are still undetermined, may be the cause of considerable disturbance of this character. During the early weeks there is often increased warmth of the paralysed limbs, amounting to from half a degree to a degree and a half Fahrenheit, at first uniform, afterwards intermitting. With this there may be increased redness and lividity, sometimes with marked œdema, especially if the kidneys are also diseased. Often there is a tendency to graver trophic changes: blisters readily form, filled with dark serum; the skin sloughs from slight pressure in those parts on which pressure chiefly acts in the recumbent posture—the gluteal region, over the trochanter and malleolus. Rarely there is inflammation of joints.

When recovery occurs, power returns in the proximal parts of the limbs sooner than in the distal parts, and in the leg before the arm. Indeed, as already stated, some recovery in the leg is invariable. There is, moreover, more use of the leg in association with the other, than in its separate movements. The flexion of the foot is that which remains longest defective, and hence the patient cannot get the toes off the ground in bringing the foot forward in the act of walking, and swings the leg round. In the arm, the shoulder recovers before the elbow, and the elbow before the hand. The extensors remain weak longer than the flexors, and the supinators longer than the pronators. In rare cases the hand-movements return first; and it is singular that these cases sometimes also present another exceptional feature—the arm improves faster than the leg. No doubt this peculiarity depends on a special position of the lesion.

The distribution of the palsy in cases of hemiplegia depends on the position and extent of the lesion, and certain

forms need special notice. I have already mentioned the escape of the tongue on the affected side, when the lesion is in the medulla, and of the face when it is in the lower half of the pons; that is to say, when the lesion occurs before the cerebral path from the hypoglossal and facial nucleus has crossed the middle line, and has become associated with the path to the limbs. But the cranial *nerves*, from the third to the hypoglossal, in passing from their nuclei to the surface, may be damaged by disease that damages also the motor tract. This paralyses the nerve on the same side as the lesion, but the limbs on the side opposite to the lesion, thus causing what has been badly termed "alternate hemiplegia." Certain nerves are more frequently paralysed in this way than others: the most frequent are the facial, sixth, and third nerves; less frequently the hypoglossal, auditory, and fifth. The facial and sixth are sometimes affected together on the side opposite to the limbs. The paralysis of the face resembles that due to other diseases of its nerve; all parts of the face are paralysed, and there is loss of faradaic and preservation of voltaic irritability. When the lesion is in the crus, the face is affected on the same side as the limbs, but the third nerve on the opposite side—on the same side as the lesion. Do not imagine that this crossed palsy of limbs and cranial nerves is invariable when hemiplegia results from disease in these regions. The lesion may be so placed, or so small, that the nerves escape. Thus the association of palsy of cranial nerves on one side, with that of the limbs on the other, gives us one class of varieties of hemiplegia.

Another class depends on the incomplete extent of the palsy, the seat of the disease being in the cerebral hemisphere, where the constituent elements of the motor tract have so far diverged that its damage may easily be partial. We have already seen that this must be rare in lesions of the internal capsule, on account of the proximity of the several paths, and that it may more readily occur in the white substance, and most readily in or beneath the convolutions. The paralysis may involve only the face, or the arm, or the leg: or it may involve the face and tongue; face, tongue, and arm; or face

and arm. The distribution depends on the relative position of the centres and paths. Thus the tongue and arm are never affected by a single lesion without the face, because the face-centre and path intervene between the two others. Similarly, the face and leg are never affected without the arm, because the centre and path for the arm intervene between the others. This partial hemiplegia is sometimes called "monoplegia," distinguished, according to its seat, as lingual, facial, brachial, or crural, while combinations receive compound names, as brachio-facial monoplegia. It may seem to you rather anomalous to call the latter a monoplegia, but the whole nomenclature is inconsistent. Strictly, "monoplegia" should designate double hemiplegia; but we call this "diplegia." Thus two "half-palsies" make, not a "one-palsy," but a "two-palsy," and a "one-palsy" is less than a "half-palsy."

In these cases of partial hemiplegia, the paralysis never remains absolute, and usually is not absolute, even at first. The state of the limb resembles that of a hemiplegia that is recovering. The coarse movements in the upper part of the limb are preserved, while the movements of the extremity are impaired or lost.

The affection of sensation that often accompanies hemiplegia I shall describe presently. Before leaving the subject of motor palsy one curious class of symptoms must be mentioned. These are the disorders of movement that sometimes come on some months after the onset. The rigidity of which I have already spoken is fixed, varying but little during the waking hours. But the muscular contractions we are now considering are versatile and changing. They also vary much in different cases. Sometimes there is tremor; fine, quick, rhythmical contractions of the muscles. Rarely there are slow and rhythmical movements, wider in range, and chiefly met with in the hands or fingers. Most common of all are irregular muscular contractions, irregular both in time and in degree, rarely quick, far more often slow. The quick movements somewhat resemble those of chorea, and hence the term, "post-hemiplegic chorea," has been applied

to the whole class of movements—unwisely, because they have nothing to do with chorea, and, moreover, resemble chorea only in rare cases. Generally, the movements are far slower than are seen in true chorea. If not constant, they are readily evoked by an attempt at voluntary movement, or even by attention, and by these they are always increased. Incoordination of movement results, peculiar in character—slow, irregular, spreading movements of the fingers, that have been compared, not inaptly, to the movements of the arms of a cuttlefish. "Mobile spasm," it may be conveniently termed. With this there is often some more constant and unchanging spasm, especially in the flexors of the wrist. The arm is always involved in greater degree than the leg. In the latter the effect of the spasm is chiefly to cause inversion of the foot and over-extension of the great toe; spontaneous movements are rare. The arm is usually adducted at the shoulder-joint; the elbow is sometimes flexed, sometimes strongly extended. Often the arm, straightened out, is carried behind the body. The wrist is frequently flexed; the fingers are usually flexed at the metacarpo-phalangeal joints, extended, and even over-extended at the others, the spasm preponderating in the interosseal muscles. There may be a subluxation of the extended finger-joints, the heads of the phalanges projecting on the palmar aspect. Thus there is a remarkable contrast between this spasm and ordinary late rigidity. In the latter the spasm chiefly affects the long flexors of the fingers; the digits are bent at all joints, a form of flexion that is employed in coarse movements of the limb. In the mobile spasm there is the "interosseal flexion" just described, the flexion that is employed in many delicate operations, such as the act of writing. The continuous action of the muscles often leads to their overgrowth, and the limb may be actually larger in circumference than that of the opposite side, when it is less in length. This condition sometimes developes without preceding hemiplegia; and to such a case, in which there was no fixed spasm, but only the slowly-changing irregular movements, Hammond (of New York) gave the name of "athetosis" (=without fixed position). In the vast majority of cases the condition is a

sequel to hemiplegia. It sometimes comes on after hemiplegia in adult life, but is far more frequent after hemiplegia in infancy and childhood, to which it is, indeed, the common sequel. In adults it has been observed chiefly in cases in which the lesion was in or near the optic thalamus. Why, we cannot yet say. In children it does not seem to be related to any special seat; it follows disease anywhere in the motor regions of the hemisphere. The lesion causing the initial hemiplegia is, however, almost invariably softening, and not hæmorrhage. An analysis of adult cases shows this clearly; and in children any other acute lesion than softening is very rare. These considerations suggest that one element in its causation may be the partial recovery of nerve-cells that are damaged, but not destroyed—which recover, but with disordered functions,—and the greater power of recovery and greater capacity for derangement during the period of development may be the cause of the special frequency of this condition after hemiplegia in early life.

From motor palsy, the spasm last considered naturally leads us to that paroxysmal over-action which causes convulsion. Convulsions are frequent and important symptoms of cerebral disease. They occur under two conditions, apparently as the result of two different mechanisms. First, they occur when there is active irritation of the brain-tissue, such as is produced by inflammation of the brain or membranes, a growing tumour, or a sudden lesion. Secondly, they occur in what are termed "stationary lesions," in which the stage of activity is over, and such structural recovery as may be possible is taking place, or has taken place. In this case they are, apparently, due to the imperfect recovery of damaged nerve-cells, which regain the power of evolving nerve-force, but not the higher power of regulating its discharge. By each mechanism, convulsions are produced most readily when the disease is in the cortex. Stationary lesions scarcely ever cause convulsions unless they are situated in or near the motor cortex. Active irritation is most effective

when in the same region, but it may cause convulsions whatever be its seat. They are also produced by general increase of intracranial pressure, and by diffuse processes, such as meningitis.

The diagnostic significance of convulsions depends on their character. They may be general or they may be partial, either in extent or in commencement. General convulsions constitute a "diffuse" symptom. They are often due to a diffuse and widespread morbid process, inflammatory or degenerative disease away from the motor centres, or to general increase of intracranial pressure. Convulsions that are limited in extent or commencement constitute a focal symptom, and indicate disease in or near the motor region, especially the motor region of the cortex. In general convulsions the loss of consciousness is sudden and immediate; there is usually no aura. In partial convulsions, consciousness is lost late, and the patient is usually aware of the local onset. This local onset is due to the fact that the discharge begins in the centre irritated—for the face, arm, or leg. If very slight, it may not spread beyond the one centre in which it began, the convulsion being confined to the corresponding part. If more severe, it spreads to all the centres of that hemisphere, and the convulsion affects the whole of one side of the body. If still more severe, the discharge spreads to the other hemisphere, and the limbs of the other side are also involved, usually after the first side, sometimes, in the most severe fits, simultaneously. Consciousness is usually retained throughout when the convulsion is confined to one limb; sometimes when it involves the whole of one side; scarcely ever when both sides are involved. Different discharges vary in intensity, and the patient often has slight attacks that are local, and more severe attacks that begin locally and become general. The attack usually commences by clonic spasm, which often becomes tonic if the convulsion becomes severe. The spasm almost always begins in the extremity of the limb. But it is common for a sensory "aura" in the part to precede the spasm: the sensation may pass up the limb first affected, along the side of the trunk, and down the second limb affected, and only then may spasm be added. It is as if the

discharge commenced in the sensory cells of the cortex, passed like a ripple through these sensory centres, and only when it reached their limit was deflected to the motor cells. In the very slightest attacks the sensory aura may be accompanied, not by spasm, but by the opposite condition—sudden powerlessness of the limb. The influence of the sensory discharge on the motor cells may thus be to inhibit them, if it is of very slight degree—another instance of the relation of inhibition and discharge mentioned on p. 47. Special sense auræ are very rare in organic disease; when they occur they indicate that the disease is in or near the part of the cortex that subserves the corresponding functions. Thus, I have met with a case in which a flash of light was the aura of fits due to a tumour of the occipital lobe; and another, of a tumour beneath the temporo-sphenoidal cortex, in which the sound of bells was the aura.

A convulsion leaves behind it a transient weakness in the part convulsed, probably due to exhaustion when the spasm is severe, or to motor inhibition when the spasm is slight or the discharge is limited to the sensory cells, sometimes perhaps to both mechanisms. The weakness after a unilateral fit may resemble slight hemiplegia, and, if the convulsions recur at short intervals, the residual palsy accumulates until it may amount to absolute powerlessness of the side, with increased knee-jerk and foot-clonus. It gradually passes away after the convulsions are over.

The characteristic, then, of the convulsions of organic brain-disease is their local commencement. Whether the fit remains limited to the part in which it commences, or spreads to other parts, even the whole body, is a question of degree. Moreover, a local aura, without convulsion, has the same significance as local spasm. It indicates that the discharge commences at one part of the brain, that there is at that part a morbid state, and it is thus a focal symptom. Convulsions that are general from the first are often due to, and symptoms of, organic brain-disease; but they derive their significance from associated symptoms, and not from their character. Without such symptoms they suggest idiopathic epilepsy, not the organic brain-disease that local commencement suggests. I say

suggests, because there is sometimes a local commencement in idiopathic epilepsy. This is, however, rare, and you would not think of idiopathic epilepsy in such a case unless you had ascertained that other symptoms of organic brain-disease were absent, and you could not feel confident, unless the convulsions had existed alone for a considerable time, or there was a strong family tendency to the idiopathic disease.

The first convulsions from organic disease are sometimes excited by a cause outside the central nervous system, a cause that may seem to be adequate to account for them. This is a very important point. Remember that the apparent cause of a fit is scarcely ever more than its excitant—an excitant that is effective only because there is a predisposition; and the predisposition is the important matter to be sought out and to be treated. The spark would be harmless unless it fell on gunpowder. The predisposition may be sometimes an inherited tendency of the nervous system, or it may be an acquired state of morbid irritability, such as forms part of the constitutional disease we call "rickets," or it may be a brain disease that has not reached such a degree as to cause pronounced symptoms. For instance, a child was brought in here who had swallowed a slate pencil, and then had an attack of general convulsions. For this the slate pencil seemed an adequate cause. It was passed by the bowel, and the child seemed well. But six weeks afterwards she was dead, and the cause of death was a large infiltrating glioma of the pons, which must have existed at the time of the convulsion, and no doubt gave rise to the predisposition. As in this case, the convulsion so excited is usually general. But only a few weeks ago, you may remember, we had a man in Ward IV., whose history showed that a convulsion beginning locally may be excited by a local cause, even though it ultimately depends on organic brain-disease. The man, when apparently well, struck his left arm against an iron hook, and a few hours afterwards had a fit which began in this arm, and was heralded by a pain at the part struck. He afterwards had other similar fits. It seemed like a case of what is termed "reflex epilepsy"—a disease, by the way, much

more rare than you may imagine from books. But one day he had a fit which began in the foot, and not in the arm. Then he developed headache, double optic neuritis, and hemiplegia; and when he came in here, he presented the characteristic symptoms of a tumour in the upper part of the ascending frontal convolution. The blow on the arm had determined the time and place of an explosion, of which the real cause was the organic brain-disease. Such a case is the converse of the arrest of a fit, beginning locally, by a ligature round the limb, when the fit is due to organic brain-disease, and the ligature can only stop the fit by acting on the centre in the brain.

Remember, then, that convulsions should not be lightly dismissed from consideration because the fit succeeded an exciting cause.

Sufferers from organic brain-disease who are of the age and sex in which the predisposition to hysteria exists, may suffer from attacks of hysteroid convulsion. It is not surprising that so potent a disturbing influence should evoke into activity a tendency from which few women are altogether free. But the fact is of extreme diagnostic importance, because, when one of a set of symptoms is unmistakable in character, it is natural to take it as an index to the series. You should never do so, gentlemen, until you have satisfied yourselves that no symptom is certainly of a different nature. I insisted on this in speaking of the diagnosis of diseases of the spinal cord, and it is equally true of those of the brain. Both acute and chronic disease may lead to the strange disturbance of function to which we give the name hysteria, and which we might just as well call, as did our forefathers, "fits of the mother." I have seen it, for instance, many times in cases of cerebral tumour, many times in tubercular meningitis, and once at the onset of an attack of embolic hemiplegia. It is a very frequent consequence of the defective development of the brain that results from infantile lesions. Some of you may remember a curious girl who attended here for a long time, whose powerless and

withered arm was the relic of a severe infantile hemiplegia. She suffered from unilateral epileptiform convulsions, and also from pure hysteroid fits of intense severity, as well as from laryngeal spasm, phantom tumour, and various other symptoms of the same class.

Tonic spasm, in the form of muscular rigidity, occurs as a chronic symptom chiefly after hemiplegia, and as an acute and subacute symptom in irritating lesions, especially in inflammation of brain and membranes, either primary, or secondary to necrotic softening. It is almost always one-sided. Severe tetaniform spasm is rare. It is generally paroxysmal, and has been met with chiefly in disease of the cerebellum, but only in cases of tumour, and it is probably due, not to the damage to the cerebellum, but to the pressure of the tumour on the pons. Similar spasm may be caused by tumours of the pons. Forced movements, as a tendency to rotation, are extremely rare. They resemble those produced in animals by injury to the semicircular canals or cerebellum, and have been observed chiefly when disease involved the middle cerebellar peduncle. Of their precise mechanism we are still ignorant. Fine tremor is rare except after hemiplegia, but sometimes attends muscular weakness in chronic disease. It is without special significance. Inco-ordination of movement is also rare except after hemiplegia. Insular sclerosis of the brain and cord is attended by jerky inco-ordination, sometimes wild in its irregularity and in the extent of its range. It is important to know that a precisely similar form of inco-ordination is sometimes met with in cerebral tumour. I have seen it in cases of tubercle of the pons Varolii and crus cerebri, when the tumour has compressed the motor tract. There is usually hemiplegic weakness; and the movements of the leg, as well as those of the arm, may be irregular. Charcot believes that the mechanism of this inco-ordination in disseminated sclerosis is an unequal morbid resistance in the nerve-fibres passing through a sclerosed area, and a similar effect may, conceivably, result from compression by a tumour.

LECTURE VI.

SYMPTOMS (*Continued*): DISTURBANCE OF SENSATION—AFFECTIONS OF SIGHT.

Gentlemen,—From the motor symptoms that occupied our attention in the last two lectures, we pass to the corresponding disturbances of sensation. The best instrument for testing sensibility is a simple one, a quill pen—the feather for touch, and the point for pain. We will consider first the loss of sensation that is analogous to the motor palsy, and often attends it. Hemiplegia may exist without any sensory loss, or with every degree of this loss. Complete hemianæsthesia may exist alone, or may be combined with every degree of motor palsy. It is rare, however, for the two to exist in high degree: usually a considerable degree of one is combined with a slight degree of the other. I am speaking now and always of organic disease, and have nothing to do, beyond a passing reference, with the mysterious hemianæsthesia that occurs in some cases of hysteria.

The one-sided loss of sensibility may involve all forms of sensibility—of touch, pain, temperature—or may involve one more than the others. In its complete form it extends up to the middle line, the absolute loss, however, usually stopping short of the middle line by a centimetre or so. It involves the mucous membranes as well as the skin. It always depends on interference with the sensory path, the course of which I

described to you (p. 15); but if the lesion is in the pons, the parts supplied by the fifth nerve escape. The most frequent seat of disease causing hemianæsthesia is the posterior part of the internal capsule, between the posterior extremities of the optic thalamus and lenticular nucleus. Disease here, you will remember, may involve the special senses on the same side as common sensibility, the affection of sight being, however, hemiopia, due to arrested conduction from the half of each retina. Although the destination of the sensory path seems to be the cortex under the parietal bone, hemianæsthesia only results from cortical disease when this is very extensive, and then (as the case related on p. 16 shows) the special senses may be involved, and instead of hemiopia we may have loss of sight in the eye on the anæsthetic side, and perhaps hemiopia also in the eye of the same side, if the disease involves the occipital as well as the parietal lobe (see p. 23, note).

The slighter forms of defect may be characterized, not by any absolute loss, but by slight qualitative change. A patient, for instance, often avers that there is a difference throughout one side, that a touch or a prick does not feel the same as on the other side, although there is no place at which the slightest touch is unfelt.

The second form of loss of sensation often accompanies hemiplegia. The loss exists chiefly or only in the limbs, and is greatest in, often confined to, the extremities. Tactile sensibility is lost in greater degree than sensibility to pain, and there is often an inability to recognize the position of the hand or foot, even when the skin is perfectly sensitive. Thus, the patient's eyes being closed, the hand and fingers are moved about, and finally held in some posture which the patient is asked to describe, or to imitate with the other hand. If there is no tactile loss, the fingers should be firmly grasped so that the direction of pressure may not inform the patient of the posture. This should be repeated several times, since too much weight must not be laid upon a single error, especially if the patient is unintelligent. It is remarkable that in some cases in which the patient can

feel the slightest touch on the hand he has not the least idea of the posture in which it has been placed. This loss occurs in disease of the motor cortex. It depends, according to Munk, on the loss of the structures that subserve conceptions of movement. We cannot yet say, however, that it does not result from disease elsewhere. Indeed, some of you may remember a case of hemiplegia that was in the hospital a few months ago, in which this loss was as distinct as it can ever be in an unintelligent patient, and the cortex was intact, the lesion being confined to the internal capsule and corpus striatum. Hence the diagnostic value of the symptom as evidence of cortical disease is probably not great.

It is a curious fact that lesions of the brain occurring in infancy or early childhood seem never to cause permanent loss of sensibility, although they must sometimes involve the sensory path or centres. This fact shows that, in the growing brain, compensation by other parts, perhaps by the other hemisphere, is possible in regard to this function in very complete degree.

The hemianæsthesia that occurs in hysteria may be misunderstood and ascribed to organic disease, but careful attention to the other symptoms in the case will generally prevent error. Remember that in hysteria one-sided loss of sensibility, although usually complete, is not always so; the special senses, and even the head, may escape. If it is combined with motor palsy, the loss of feeling may be much greater in the weakened limbs than elsewhere. The risk of error is greater in such cases than it is when the hemianæsthesia is complete and the special senses are affected—vision as "crossed amblyopia." Perfect as is the correspondence of this sensory loss to that which may result from organic disease of the cortex, the slight risk of erorr that there is may be avoided if you remember that the symptom can be produced by organic disease only when this is very extensive —so extensive that its nature could not admit of question. If you doubt whether there is organic disease, you may feel sure that there is not. In the only situation in which a small lesion

can cause hemianæsthesia with affection of the special senses (the posterior part of the internal capsule), the affection of sight is always hemiopia.

Perhaps the greatest risk of ascribing hysterical loss of sensation to an organic cause is in the cases of infantile lesion of the brain that I have just mentioned. I told you, in the last lecture, how prone the female subjects of such disease are to develope hysteria. If you are not aware that such lesions do not permanently impair sensibility, you may easily ascribe hysterical hemianæsthesia in such a case to the organic disease. It is one of the most subtle of the many traps that hysteria delights to set for the unwary diagnostician. I had myself to pay for my experience. Some years ago a girl came to me with infantile hemiplegia of the ordinary type, with shortened limbs, and mobile spasm in the arm. She complained of no symptoms suggestive of hysteria. On testing sensibility I found that the whole of the paralysed side was anæsthetic; the special senses were blunted on that side, vision was much impaired, and colour-vision was lost. Here, I thought, are two most interesting things: first, there is a lasting loss of sensibility from a lesion of the brain in infancy; and secondly, there is crossed amblyopia from organic disease. I made most careful notes, and told her to come again in a week. She did so; and on the paralysed side sensation was now perfect, and all the loss of feeling had gone over to the other side! Only a few months ago, in one of the medical papers, I saw an account of a similar case, which was published as hemianæsthesia of organic origin.

Symptoms due to irritation of the sensory tract or centres are not uncommon in central disease. Tingling, formication, and the varied sensations called "numbness" are frequently associated with hemiplegia, as prodromata and as accompaniments. They are common both with and without actual loss of feeling. When the lesion occupies the motor centres or path, these sensations, like the anæsthesia in the same cases, occupy chiefly the extremities. Such sensations, extending over the whole of one side, up to the middle line, attend disease in or near the sensory path, and may herald or accompany

hemianæsthesia. The anæsthesia may be transient, and the sensations persistent, no doubt because the lesion is so placed near the sensory path or centres as to irritate but not to interrupt them. In the same class of cases there may be persistent pain in the limbs, usually dull and wearying. The patient whose fields of vision are represented in Fig. 8 suffered severe pains of this character for many years, the result of a lesion which caused transient hemiplegia and persistent hemiopia.

Paroxysmal sensations of tingling, "pins and needles," or pain, may result from such discharges as, in motor structures, cause convulsions, and the sensory discharge often accompanies the motor. I spoke of these sensations in the last lecture. It is also very important for you to know and remember that such one-sided sensations of tingling, etc., may occur as part of the strange phenomena of migraine. They are then more deliberate than those that herald a convulsive attack or constitute a minor seizure, lasting for half an hour or an hour. When on the right side, aphasia often attends them. Followed, as they are, by violent headache, they may cause much alarm, not only to the patient, but also to his medical adviser, if the latter is unaware of the occurrence of such sensory disturbance in this association. If the patient has not had such an attack before, he is certain to have suffered from paroxysmal headache, and probably from slighter sensory symptoms, especially in vision; from these you may feel sure of the nature of the attack.

We pass from the disturbance of common and tactile sensibility to the symptoms that depend upon disturbance of the nerves of special sense. These are cranial nerves, and it will be most convenient to go through the cranial nerves *seriatim*, considering the symptoms within the range of their functions in the anatomical order, which is nearly the order we adopted in the description of the anatomical relations of the nerves. Those relations you must bear in mind, if you would understand the important symptoms that are produced by disease of the nerves, or of the central tracts that continue their path to the cortex.

We commence, then, with the nerve of smell. Remember that the olfactory nerve subserves not only the sensations that we recognize as odours, but also those that are termed flavours. We call the sensation an odour when its cause reaches the olfactory membrane by the anterior nares, a flavour when its cause enters the nose by the posterior nares. In the latter case the sensation is combined with a true gustatory sensation (of sour, sweet, bitter, etc.) from the tongue and fauces, and the two are so blended that we seem to taste flavours when we really only smell them. Hence those who have lost smell always say that they have also lost taste, although you will find, on trial, that the true gustatory sensations are unimpaired. In examining the sense of smell you must employ only odorous substances that are not pungent; *i.e.*, do not irritate the fifth nerve, or the patient may perceive that which he cannot smell, and the result is confusing. Loss of smell is an infrequent symptom of brain disease. Its most common cause is disease of the olfactory mucous membrane, polypus, chronic catarrh, and the like, the absence of which must always be ascertained before the symptom is allowed significance. Mechanical injury, by damaging the delicate filaments that come from the olfactory bulb, is another occasional cause. Diseases of the anterior fossa of the skull, tumour, aneurism, meningitis, sometimes cause it, and it may result from the pressure on the bulbs in extreme internal hydrocephalus. From central disease it is very rare, except in association with complete hemianæsthesia; it is then on the side opposite to the lesion. It probably may be produced also by disease in the temporo-sphenoidal lobe, near the inner part of the fissure of Sylvius, and will be then on the same side as the lesion. It occurs occasionally in degenerative diseases, as general paralysis of the insane, perhaps in consequence of an atrophy of the nerve analogous to that of the optic nerve. Subjective sensations of smell are almost confined to functional diseases (as the aura of epilepsy, etc.), but have been noted from tumour of the temporo-sphenoidal lobe.

Disturbed function of the optic nerve is a very common symptom of brain disease. In acute lesions it is generally due directly to the cerebral lesion; in chronic disease it is often the result of optic neuritis, and is thus a secondary effect of the brain-mischief, of significance chiefly as to the nature of the disease. In almost all cases, however, an ophthalmoscopic examination is necessary to ascertain whether the affection of sight is of intra-ocular origin, before its significance can be determined. There is only one form of impairment that can be referred with certainty to central disease, and that is hemiopia. Even in acute lesions of the brain, which do not, as a rule, entail optic neuritis, coincident retinal changes, such as albuminuric retinitis, hæmorrhage, and embolism, sometimes cause impairment of vision, the origin of which can also only be ascertained by the ophthalmoscope. If optic neuritis is found, we are not justified in immediately ascribing the impairment of sight to its effect. If the visible neuritis is slight in degree it will not account for affection of sight. The latter may be due either to a more considerable retro-ocular inflammation of the nerve, or to the central disease, and between the two we can only decide by the symptoms.

I have already mentioned the chief points in the anatomical relations of the optic nerves that determine the form of loss of sight from disease in the several parts of the visual path. Loss of sight of one eye means an affection of its nerve between the eye and the chiasma, *i.e.*, in the orbit, at the optic foramen, or within the skull. Temporal hemiopia—loss of the outer half of each field of vision—means damage to the middle of the optic chiasma, affecting the fibres that cross from the nasal half of each retina, and leaving unaffected those at the sides of the chiasma that do not cross. Its most common cause is a tumour, distension of the third ventricle, and inflammation. Affection of only the non-decussating fibres, by damage to each side of the chiasma, causes nasal hemiopia, but is extremely rare. Disease anywhere in the optic path, from the chiasma to the occipital cortex, causes hemiopia, which may be complete or incomplete, but is always symmetrical,

although not always minutely symmetrical. When incomplete, a part only of each half-field is blind. Amblyopia of one eye, with considerable peripheral restriction of the field of vision, occurs from extensive disease of the cortex of the opposite hemisphere, and perhaps from a small lesion in the angular gyrus. As I have told you (p. 24), there is usually loss of colour-vision, and there is also slighter restriction of the fields (white and colour) in the eye on the same side as the disease. When caused by acute non-progressive lesions, this loss usually soon lessens, perhaps by compensatory action of the centre in the other hemisphere, or of the undamaged part of the affected centre. Thus, affection of sight of one eye may be due to disease at either extremity of the visual arrangement—the optic nerve, or the highest cortical centre. The two are distinguished by the following indications: (1) the reaction of the pupil is lessened in disease of the nerve, and not in disease of the hemisphere; (2) the other eye suffers in a similar but slighter way in cortical disease; and (3) the associations of the two are wholly different, cortical disease being accompanied by hemianæsthesia and no affection of the cranial nerves; whereas, in disease of the optic nerve, hemianæsthesia is always absent, and the motor nerves to the eyeball are very often involved. Remember, however, that for one case of crossed amblyopia and hemianæsthesia from organic disease, you will see many that are of functional origin, and the evidence of an organic lesion must be very clear before you can ascribe the symptom to this cause.

A simple peripheral restriction of the field of vision occurs also in optic nerve atrophy, both in simple atrophy and in that which follows neuritis. In the latter the fields are often irregular; sometimes there are islets of loss ("scotomata," as they are termed) within the field, and such islets may vary for different colours. A central defect is a consequence of damage to the fibres in the axis of the nerve. It is common in tobacco-amblyopia, but is scarcely ever met with in brain disease.

In searching for these defects of sight you should first ascer-

tain whether there is any considerable error in the refraction of the eye. A slight amblyopia, which you might otherwise ascribe to an affection of the optic nerve, may be due to this cause. It is beyond my present task to describe the methods by which the examination may be made with precision, but for the purpose in view a rough estimate may be formed in the course of the ophthalmoscopic examination. When you throw the light into the eye, as for the indirect examination, if you can see the retinal vessels distinctly, the refraction is abnormal: move your head to one side, and the vessels, thus seen, appear to move: if this movement is in the same direction as your head, the eye is hypermetropic; if in the opposite direction, the eye is myopic. You may further ascertain the degree of each defect in the direct ophthalmoscopic examination, by observing the strength of concave glass necessary for clear vision of the fundus if the eye is myopic, and the greatest strength of convex glass that does not blur the image if the eye is hypermetropic. Of course if your own eye is of abnormal refraction you must allow for this. In the further examination of vision each eye must be separately tested. Acuity of vision is ascertained by test-types, numbered according to the distance in feet (or metres in the case of Snellen's test-types) at which the type should be distinct. The result is expressed in the form of a fraction, of which the denominator is the number of the test-type, and the numerator the farthest distance at which the type is read by the patient. Thus, $\frac{1}{6}$ means that the type which should be read at a distance of six feet can only be read at one foot. Colour-vision is examined by the method of comparison (with wools, etc.), for an account of which I must refer you to books on ophthalmology. It may also be tested by asking the patient to name colours—a method chiefly valuable when you wish to compare the colour-vision of one eye with that of the other. By the method of comparison you escape the errors arising from the common inability to name colours with precision, and from the uncertainty of the natural appreciation of colours, and of the sense attached by the patient to the names he gives. This element, which we may term the personal colour-equa-

tion, does not come in when we merely compare the sense-impressions of one eye with those of the other, and we may therefore, in this case, ask the patient to name the colours, and often learn much regarding his perception of colour that can be ascertained in no other way.

The examination of the fields of vision is of especial importance in the diagnosis of brain disease. For an exact examination it is necessary to use a "perimeter," an instrument in which an arm, in the form of a quadrant of a circle (graduated in degrees), rotates on one extremity, and thus may be made to occupy successively the various radial arcs of an imaginary hemisphere, of which its pivot is the centre. The patient's eye being opposite and looking at the pivot (his other eye being closed), a small white or coloured object is moved along the arm in each radial position, and the point at which the object

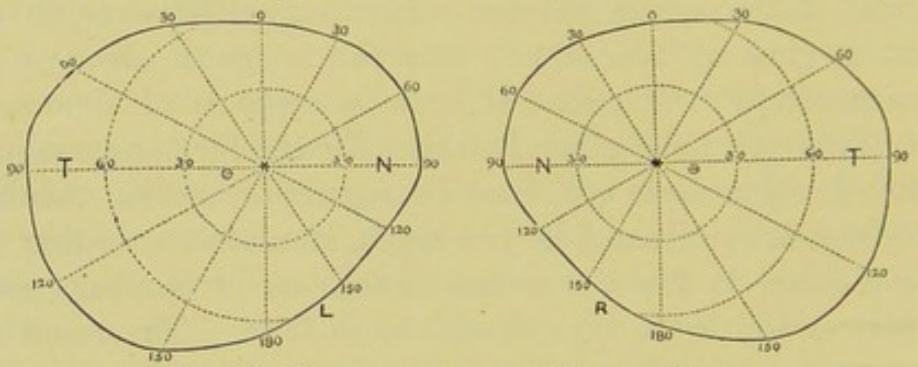

FIG. 13.—DIAGRAMS OF THE NORMAL FIELDS.

The asterisk is at the fixing point; the small circle indicates the blind spot (optic nerve entrance); R, right; L, left; T, temporal or outer side; N, nasal or inner side. The radii and concentric circles are each 30° apart.

ceases to be seen is the limit of the field in that direction. The results are marked on a chart, which consists of radial lines (corresponding to the positions of the arm) and concentric circles (corresponding to the divisions on the arm). A line joining the points in each radial line, at which the object ceases to be seen, indicates the peripheral limit of the field of vision. This, as already stated (see p. 20), is not a circle, being less extensive inwards than outwards, and above than below, on account of the projecting nose and eyebrow, and the influence of these on the functions of the corresponding parts of the retina. In Fig. 13, which represents an average

normal field, the circles and radii are not represented beyond the limits of the field. These circles and radii may be as numerous as you like; in the figure only those at 30° distance apart are represented. The size of the object may be a centimetre or half a centimetre square. To ascertain the colour-fields, a coloured object must be used, and the points noted at which the patient ceases to see the colour, not the object. The latter can still be seen outside the region in which the colour is recognizable, the fields for colour being smaller than that for white, and their order, beginning with the smallest, is violet, green, red, yellow, and blue. An examination with the perimeter requires time, and need only be employed when you have reason to suspect that there is a limitation of the field. You may ascertain whether there is any considerable defect by a much rougher method of examination. Let the patient stand opposite to you, and about two feet away. Make him place his hand over the eye you do not wish to examine, and keep the other fixed on your eye that is opposite his, your other eye being closed. Next place your hand, midway between yourself and him, in various places towards the limit of your own field of vision, and ask him if he sees it; move your fingers, if you like, and ask him if he sees them move. Still better, place a small piece of white paper on the end of a dark-coloured stick (a penholder answers well), and hold the paper in various positions all round the outer part of the field, or nearer the centre if you suspect any central loss of vision, holding it so that the light falls well upon it, and the surface, not the edge, is towards the patient. In this method the patient uses your own eye as a fixing point, and you are able to see at once if he looks away from the fixing point and at the object, as he is almost sure to do at first. You check his field of vision by your own, and do not move the object into positions in which it is beyond the natural range of sight.* A still rougher method of ascertaining that there is no symmetrical

* For testing the colour-fields in this manner, Messrs. Pickard & Curry make small coloured metal discs, that can be fixed on a pen, and carried in an ophthalmoscope case.

hemiopia is the following:—Let the patient stand opposite to you with both eyes open, looking at you. Hold up both your hands, one on each side, as far apart as you can well see them. Then put them down, and ask him if you held up one hand or two. Do not ask him this while the hands are up, or he is almost sure to look first at one and then at the other, and say "two." If both hands were seen there is no defect in the outer half of either field, and therefore no ordinary hemiopia, since, in this, one outer half must be defective.

Remember that the subjects of hemiopia are not always conscious of it; especially when the line of blindness does not go very near the fixing point, the loss is often unnoticed. One woman with such hemiopia was indignant at the suggestion that she had any defect of sight, declaring

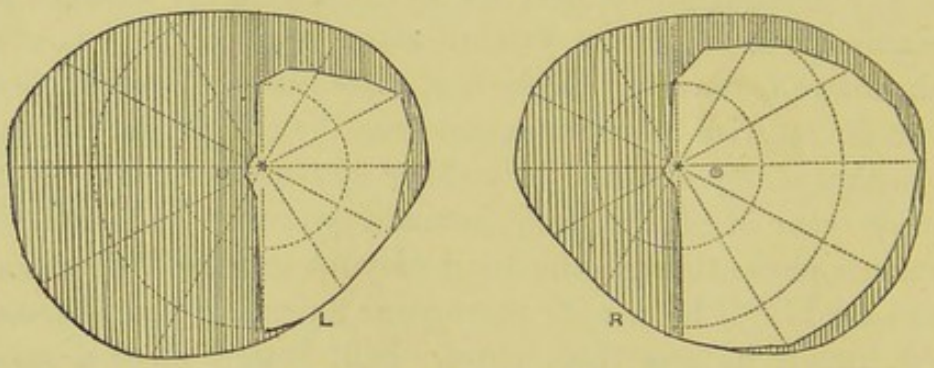

FIG. 14.—FIELD OF VISION IN A CASE OF LEFT-SIDED HEMIOPIA.

The shading represents the blind part; the oval outline of each figure is the average normal field. The asterisk is the fixing point.

that she "could see her husband coming across the common before any one else could." In another patient the defect was not discovered until the nurse noticed that the man never ate his potatoes at dinner. They were always put on the same side of his plate, and he never saw them. The same patient once demonstrated his hemiopia to a gentleman in the street, who was walking with a lady. In the patient's effort to avoid the lady he walked right on to the gentleman. The subjects of this defect often hold the head inclined towards the blind side, so as to bring objects further into the remaining part of the field, and thus see the relations of objects better. A hemiopic cabman was thus able to drive his

hansom about London quite well. But if he wanted a fare he had to go on a rank, because, if he looked about him, he was sure to run into something.

The chief facts regarding the form of hemiopia are five, and I have already alluded to some of them in speaking of the indication they afford of the relation of the optic path. The first is that the division may pass through the fixing

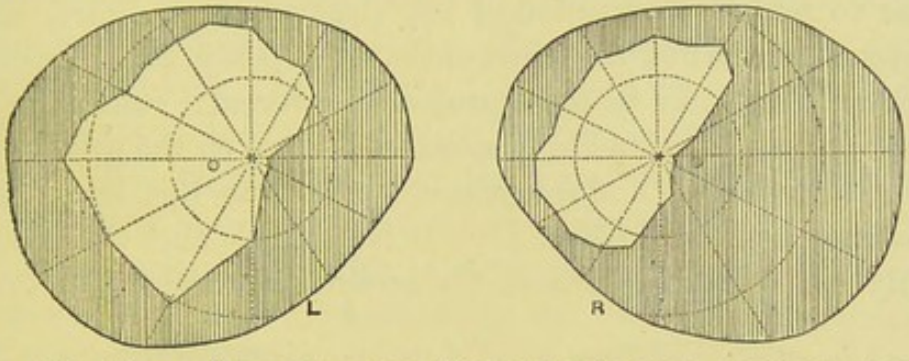

FIG. 15.—RIGHT HEMIOPIA FROM CEREBRAL DISEASE, SHOWING AN IRREGULAR OBLIQUE LINE OF DIVISION, PROBABLY DUE TO AN UNUSUAL FORM OF DECUSSATION.

point, or may diverge so as to leave it within the region of vision (Fig. 13). Secondly, the line of division is often slightly irregular: the divergence that leaves the fixing point

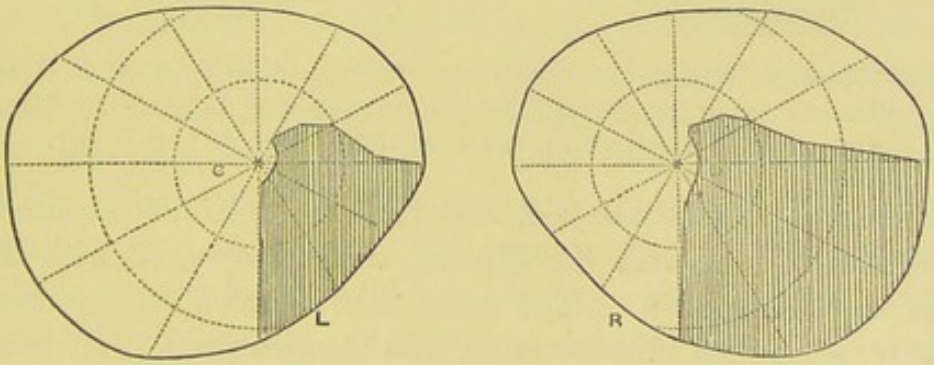

FIG. 16.—PARTIAL RIGHT HEMIOPIA FROM DISEASE OF THE LEFT CEREBRAL HEMISPHERE, THE LOSS BEING NEARLY QUADRANTIC. (Note the close correspondence of the shape of the blind area in each field.)

in the region of vision may commence just above, and end just below the fixing point; or may begin some distance above and extend below this. Thirdly, the line of division often inclines to one side or the other, above and below (as in Figs. 14 and 17), and this inclination may be in opposite

directions above and below, so as to give rise to an oblique hemiopia (Fig. 15). All these variations are probably due to individual variations in the decussation of the optic nerves, and not to the seat of disease. The next fact is that the hemiopia may be incomplete, only a portion of each half being lost. It is usually a segmental defect; often an irregular quadrant, as is shown in Fig. 16. This partial loss is due to partial destruction of the visual path or centre, and depends on the limited extent of the lesion (see p. 22). Lastly, the hemiopia may be accompanied by concentric restriction of the remaining half-fields, greatest in the eye on the side opposite to the lesion, and sometimes confined to this eye. An instance is shown in Fig. 17. It is probable that this occurs only when the disease is in the cerebral hemisphere, and not

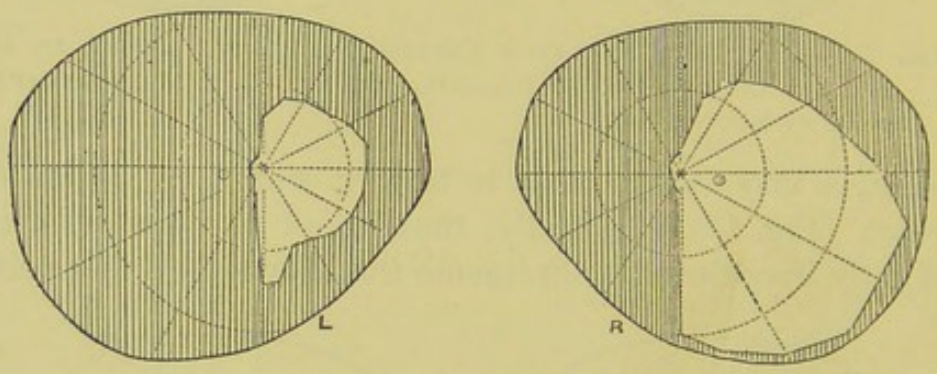

Fig. 17.—Left Hemiopia from Disease of the Right Cerebral Hemisphere, with Concentric Restriction of the Remaining Half-fields, much greater in the Left Eye than in the Right.

when the tract is diseased, and that the restriction is due to an interference with the highest visual centre, or with the fibres passing to it. The restriction is not always present even when the disease is in the hemisphere. You must remember, however, that concentric restriction of the fields may result from optic neuritis, and it may then be even greater than that which results from the cerebral lesion. The only difference between the characters of the two is that the restriction from neuritis is more nearly equal in the two eyes than is that from central disease.

Transient loss of sight occurs in some functional diseases of the brain, but its paroxysmal character precludes almost

all danger of mistake. In epilepsy the loss is complete, but is usually followed instantly by loss of consciousness. In migraine, hemiopia is common, and total loss is rare, but the symptom lasts, as a rule, only a quarter or half an hour. The affection of sight that occurs in hysteria I have already mentioned; there is usually considerable amblyopia of one eye, with slighter amblyopia of the other, and extensive impairment of the other special senses and cutaneous sensibility on the side of greatest visual loss. The distinction I have just described to you. In hysteria there may be also complete loss of sight of one eye, the other being unaffected; but this is rare. The perfect action of the pupil sufficiently distinguishes it. It is doubtful whether hemiopia is ever of hysterical origin; and this is strange, because, in migraine, hemiopia is common. Apparently the two affections are on different functional levels, and keep to them. Persistent hemiopia is almost always due to organic disease, and its significance is therefore decided. Of a large number of cases of hemiopia of which I have notes, there is only one which I think may have been hysterical.

Irritation-symptoms in the function of the optic nerves are rarely due to organic disease, while they are common in functional disturbance. Intolerance of light occurs in meningitis, but is rather a cerebral than an ocular symptom—light seems to distress the head rather than the eye. True photophobia is scarcely known in brain disease, or in diseases of the optic nerve. Visual discharges, subjective lights or colours, occur in epilepsy and in migraine; in the former, flashes or points of light are common; in the latter, the zigzag "fortification" appearance is characteristic. Disease near the visual centre in the cortex may cause subjective sensations of light, but only as symptoms of a discharge that quickly bursts into a convulsion.

LECTURE VII.

SYMPTOMS (*Continued*): OCULAR NERVES—FIFTH NERVE—FACIAL NERVE.

GENTLEMEN,—We may conveniently consider together the symptoms that depend on the three cranial nerves that supply the eyeball muscles—the third, fourth, and sixth. All supply external muscles; the third nerve also some internal muscles. Paralysis of the external muscles is indicated by symptoms of five kinds: (1) limitation of movement; (2) non-correspondence of the direction of the two eyes, *i.e.*, strabismus; (3) double vision; (4) erroneous projection of the field of vision of the paralysed eye; (5) secondary deviation of the unaffected eye. The defect of movement is always in the direction of action of the paralysed muscle, and is termed the "primary deviation." It is most conspicuous when the unaffected eye "fixes" the object, and the affected eye is prevented seeing it by the interposition of the hand, or a piece of paper, in such a manner that the eye can still be observed. The "secondary deviation" is an excessive movement of the sound eye, when *this* is prevented seeing the object at which the affected eye looks. If then the interposed hand be moved so as to cover the affected eye, the sound eye, to "fix" the object, has to move back again, and the degree of backward movement indicates the degree of previous excess, that is, of secondary deviation. Both primary and secondary deviation

depend on the circumstance that the muscles of the two eyes reserve an equal amount of innervation from the centre. That which suffices to move the sound eye into a given position is insufficient to move the affected eye into the corresponding position, and the innervation necessary for *this* causes an excessive movement of the sound eye. Thus, the primary deviation being a defect, the secondary deviation is an excess; they are in opposite directions, but both are in the line of action of the affected muscle. In slight palsy the secondary deviation is often more readily observed than the primary deviation. The patient habitually fixes with the unaffected eye, and hence the primary deviation, the paralytic defect of movement, occurs whenever the eyes are so moved as to bring the affected muscle into action. The difference in the direction of the two eyes constitutes strabismus, and from it there results double vision. The paralytic strabismus may be convergent or divergent, or there may be a difference in the height of the two eyes, according to the muscle affected. The strabismus is distinguished from that due to muscular spasm by the fact that the latter is the same in all positions, whereas the former only occurs in the positions necessitating the action of the affected muscle; in spasmodic strabismus no secondary deviation can be obtained. Double vision is commonly present in paralysis, at least when this is recent, and absent in the common spasmodic strabismus. This difference does not depend on the nature of the two affections, but on their duration. In chronic deviation of the eyes, from whatever cause, the patient learns to neglect the image seen with the affected eye (termed the "false image"), and to attend only to that seen with the sound eye ("true image"), and may be quite unaware of a diplopia which is at once conspicuous to him if a coloured glass is placed before one eye so as to tint one of the images. It is best to place the coloured glass before the sound eye. When the images are so near together that they overlap, the image appears to be blurred rather than double, but the coloured glass distinguishes the two. If a coloured glass fails at first to reveal the double image,

the observer's hand may be placed before first one and then the other eye, and the object will appear to change its position as well as its colour with the change in the eye obscured. Double vision may be "crossed" or "simple." In crossed diplopia the image in the left eye is seen to the right of that in the right eye. In simple diplopia (usually called "homonymous" or "same-named") the image in the left eye is seen to the left of the other. When the strabismus is divergent, the diplopia is crossed; when convergent, the diplopia is simple. If you will make a diagram for yourselves of the eyes in the two positions, and of the rays of light from an object, you will understand this without difficulty. Should you find it hard to keep in mind the character of the diplopia in the two cases—and these simple alternatives often give much trouble to the student—remember that when the prolonged axes of the eyes would cross, the double vision is *not* crossed.

The recent paralytic diplopia exists only in positions of the eyes needing the contraction of the weak muscle, and the farther the object is moved in the direction of action of the muscle, the farther apart are the two images. In old-standing cases some secondary contracture of the antagonists may give rise to diplopia in other positions, and even at rest. In many positions of the eyes the two images are not parallel. This depends on the complex associated action of the oblique and straight muscles, with which you are doubtless already familiar. If a paralysed straight muscle, in a certain position, has a tendency to rotate the globe, which tendency is normally counteracted by one of the oblique muscles, the latter, producing its effect alone, causes an abnormal rotation of the eye.

The "erroneous projection of the visual field" is not quite so abstruse a subject as some old associations with the word "projection" may lead you to fear; and it is a question of much interest. We judge of the relation of seen objects *to each other* by the relative position of their images on the retina, *i.e.*, by their relative position in the visual field; but we judge of their relation to our own body

by the relation to it of the whole visual field. This depends on the position of the head and of the eyes, and we know *this* by the degree of innervation of the muscles that move the head, and especially of those that move the eyes. We are only now concerned with the latter. When the innervation of the ocular muscles is at a minimum, the eyes are in mid-position, and we know, to use popular language, that an object at which we are looking is opposite our face; that is, that a line from the centre (fixing point) of the field of vision to the macula lutea would stand at right angles to the plane of the face. If we turn the eyes to one side, we know that an object *now* in the centre of the field is to the side of the position of the former one; how far to the side, we judge by the degree of movement of the eyes, estimated by the amount of innervation that we have given the muscles. I say "we judge," but there is no conscious judgment; the estimation is by nerve-processes of which we are unaware. If we want to touch this second object, we adjust, also unconsciously, the degree of contraction of the muscles of the arm, so as to enable us to effect the required movement with instant precision. But if the eye muscle concerned is weak, the increased innervation necessary for the movement gives an impression of a greater movement than has really taken place, and therefore an impression that the object looked at is farther on that side than it really is, and if the patient attempts to touch the object, his hand goes beyond it. His arm moves, in accordance with the innervation of the weak muscle, too far in the direction of the action of this muscle, just as the other eye moved too far in the secondary direction. Indeed, the erroneous direction of the arm corresponds exactly with the direction of the secondarily-deviating eye. But the knowledge of the relation of external objects to the body, gained from the innervation of the eye muscles when we look at the objects, is one of the most important guides to the centres that regulate the maintenance of equilibrium. The erroneous projection of the field of vision causes a discord between this and other guiding sensations, and one effect of this discord is the sensation that we call vertigo or giddiness. This

giddiness, depending on paralysis of an ocular muscle, is termed "ocular vertigo," and, like other forms of vertigo, is apt to cause mistakes in diagnosis. The nerve-centres, after a time, learn to accommodate their action to the altered circumstances, and both the erroneous projection and the consequent vertigo cease to trouble the patient. When it exists, the patient may try to avoid it by holding his head in such a position as not to call the affected muscle into action, and this will often put you at once upon the track of the affection. Or he may keep the affected eye closed, by contracting the orbicularis. He always closes the affected eye, because, although closing either eye would remove the diplopia, only closure of the affected eye removes the vertigo or uncertainty. He may thus show you which eye is at fault, when you might find it difficult to ascertain. If you cultivate the habit of observing every point, however trifling it may seem, you may often learn much of the nature of a patient's ailment before he has got half-way across your room.

I may briefly summarize for you the chief symptoms of the paralysis of each muscle. I will not ask you to try to remember them now, but it may be convenient to you to have the summary for future reference. We will take, as examples, the muscles of the right side.

Sixth Nerve.—External Rectus.—Defect of outward movement, convergent strabismus on looking to the right, with simple diplopia, the two images parallel and on the same level in the horizontal outward movement, but the false image often on a different level, and oblique, when the eyes are directed above or below the horizontal level. Secondary deviation of sound eye inwards. The head is held inclined to the right.

Third Nerve.—Internal Rectus.—Defect of movement inwards, *i.e.*, on looking to the left, with divergent strabismus, and crossed diplopia, the false image oblique above and below the horizontal plane. The head is inclined to the left.

Superior Rectus.—Defect of movement upwards, and espe-

cially upwards and outwards; the inferior oblique, which habitually acts with the superior rectus, being capable of aiding the upward and inward movement, it may visibly rotate the globe in so doing. Secondary deviation is by the opposite superior rectus moving the eyeball too much upwards. Diplopia occurs on looking upwards; the false image is above the other, and the difference in level is greatest in trying to look upwards and to the right.

Inferior Rectus.—Defect of movement downwards, and especially downwards and outwards (because the downward and inward movement is aided by the superior oblique). There is corresponding double vision, which is crossed. The false image is below the others, and the lower the greater the attempted movement; it is also oblique, especially on the inward movement, from the rotation effected by the superior oblique. Secondary deviation is by the opposite inferior rectus.

Inferior Oblique.—Defect of movement upwards, in which position the muscle is an elevator (just as the superior oblique, in the inward and downward movement, is a depressor). The secondary deviation is also upwards and inwards, by the inferior oblique and the internal rectus of the sound eye. Double vision exists in the same movement; the false image is above the other, and oblique, the obliquity being greatest in looking outwards, the difference in height greater in looking inwards.

Fourth Nerve.—Superior Oblique.—Defect of movement, chiefly downwards and inwards, since in this position the muscle is a depressor. Convergent strabismus exists in looking down, and the corresponding diplopia is simple, the false image being the lower, especially in an inward movement, and oblique, especially in the outward movement; the lateral distance between the two is greatest in the middle line, and lessens when the object is moved inwards or outwards. The diplopia, occurring on looking downwards, often gives the patient much trouble in going down a flight of stairs, which seems to be double, and he does not know which flight to choose.

You must not imagine, however, that it is always possible to determine with exactness the muscles that are affected in the derangement that results from brain disease. It is generally possible when the nerves themselves are diseased, but in central affections there is often a complex association of palsy and spasm that may baffle all attempts to unravel it. I remember, some years ago, one of the most distinguished ophthalmic surgeons in Europe, who knew, perhaps, more of ocular palsies than any one at that time living, visited the Queen Square Hospital and examined some patients for us. After spending some time in the investigation of the defect of movement of the eyes in one case, he said: "I can tell you many things that it is not, but I cannot tell you what it is."

The special symptoms of paralysis of each ocular nerve need not detain us long, since those of the sixth and fourth nerves are the palsies of the muscles they supply, the external rectus and superior oblique. When the whole third nerve is paralysed all the muscles are affected except the two just named; the eyeball cannot be moved upwards or inwards, and only slightly downwards, while the unopposed action of the external rectus usually causes the eye to deviate outwards. There are, however, three other symptoms: the upper lid droops from the paralysis of the levator (ptosis); from the paralysis of the sphincter of the iris, the pupil is midway between contraction and dilatation, and does not contract to light; while the paralysis of the ciliary muscle abolishes the power of accommodation.

Isolated paralysis of these muscles is usually due to disease of the nerves in the pons, in the crus, at the base of the brain, in the orbital fissure, or in the orbit; but curious associated palsies are sometimes met with in central disease. Loss of the upward or downward movements and of convergence (without loss of other action of the internal recti) has been observed, and is probably due to a lesion in or near the third nerve-nuclei or in the corpora quadrigemina, usually degenerative in character. Loss of the movement of both eyes to one side occurs in focal lesions of the brain, and has been already mentioned

(p. 53). I will only now tell you further that this "conjugate deviation," as it is termed, may be the result of either paralysis or spasm. In disease of the hemisphere the eyes deviate in paralysis towards, and in spasm from, the side of the brain affected. But in disease of one side of the pons they may deviate in palsy from the side of the lesion. Sometimes first one and then another ocular muscle becomes paralysed until all have lost power, and the eyes are motionless, with a peculiar unchanging stare. There may also be ptosis. This affection—the progressive ophthalmoplegia of v. Graefe, the ophthalmoplegia externa of Hutchinson—depends on a degeneration of the nuclei of the muscles, analogous to that which, in the cord, causes progressive muscular atrophy in the limbs, and in the medulla oblongata gives rise to what is termed "progressive labio-glossal paralysis."

The muscular mechanisms within the eye are susceptible of four forms of paralysis. First, the ciliary muscle may be paralysed, causing loss of accommodation, so that objects cannot be seen well, except at a distance, and small print, that could only be read near, cannot be read at all. The contraction of the iris associated with accommodation may be lost, either alone or with accommodation. The reflex contraction of the iris, on exposure to light, may be lost alone, and so also may the reflex dilatation on stimulation of the skin. Combinations of these palsies often occur, especially of the associated action of the pupil and of the ciliary muscle, and of the light and skin reflexes. Loss of accommodation may also be combined with loss of convergence. Sometimes all the internal muscles are paralysed—the ophthalmoplegia interna of Hutchinson. These internal palsies are rarely the result of focal lesions. They result from influences acting on the nerve-elements according to their function. Thus, in diphtheritic paralysis the ciliary muscle especially suffers. Apart from diphtheria the chief cause of these affections is degeneration, usually associated with signs of degeneration elsewhere. This is their chief diagnostic indication, and a very important one it is.

Paralysis of the fifth nerve may involve the sensory or the motor parts, or both. The symptom of the former is anæsthesia of the skin from the vertex to the lower jaw, and of the mucous membrane of the nose, tongue, and mouth on that side. Ammonia and snuff no longer irritate the nasal membrane, but odours can be perceived, although, after a time, the sense of smell is blunted from defective secretion. When the patient drinks, the cup, felt only on one side, seems broken. Food is not chewed on the affected side because it cannot be felt, and often because the muscles of mastication are also paralysed: hence fur accumulates on that half of the tongue, as it does whenever food cannot be chewed on one side. There is much discrepancy of evidence as to the influence on sensation at the back of the tongue and palate. In some cases of disease limited to the root of the fifth nerve there is anæsthesia of these parts, including the soft palate and anterior palatine arch. In other cases these parts have been sensitive. We do not know whether the difference depends on personal variations or on the position of the disease: probably on the latter. Similar discrepancies exist with regard to the affection of taste, as I have already told you (see p. 25). Taste is usually lost on the front of the tongue, and is certainly sometimes lost everywhere, even on the palate and its anterior arch. The probable explanation of this I have already given you. Trophic changes may result from paralysis of the sensory part of the fifth. Of these the most important is that in the eye. The cornea becomes cloudy, then opaque; ulcers form upon it, and may perforate, and lead to a destructive inflammation of the globe. This "neuro-paralytic ophthalmia," as it has been termed, probably depends on the irritation of the nerve by the lesion rather than on the anæsthesia or on the mere loss of nerve-influence, and the irritation seems to be most effective when it involves the Gasserian ganglion or the fibres from the ganglion in the nerve in front of it. The symptom is rare when the disease is situated between the ganglion and the pons, and the lesion there has to be more irritative in character to produce the effect.

Paralysis of the motor part of the nerve causes weakness of

the masseter and temporal muscles, easily recognized if you make the patient "bite hard" while your fingers are on the muscles on each side. If the loss of power is slight, the weaker muscle seems to contract a little after the other. The paralysis of the external pterygoid causes a defect in the lateral movement of the jaw; this can be moved towards the paralysed side, but not from it. When the lower jaw is depressed, it deviates towards the paralysed side, because, in depression, the external pterygoids draw the condyle of the jaw forwards; and this movement, occurring only on the unaffected side, causes a deviation. The movement of the hyoid bone is not perceptibly impaired; and although the tensor palati and tensor tympani are said to be supplied by the fifth, they do not seem to suffer when the root is diseased, and hence, perhaps, their fibres come ultimately from some other source. After a time the paralysed muscles waste, the temporal and zygomatic fossæ become flattened, and ultimately a little secondary shortening of the muscles may limit, in slight degree, the downward movement of the jaw.

Paralysis of the face is the result of disease of the fibres or nucleus of the facial nerve, or of the motor path between the facial nucleus and the cortex. This, as we have seen, lies to the inner side of the limb-path in the crus, and in front of it in the internal capsule, occupying the angle at the junction of the anterior and posterior parts of the capsule. The loss of power is on the same side as a lesion of the nerve-fibres, or of the nucleus in the pons; but, since the upward path decussates just above the nucleus, a lesion of the upper part of the pons, of the crus, or of the hemisphere, causes paralysis of the face on the side opposite to the lesion. There is another difference between the effect of disease above the nucleus and that of or below the nucleus. In the latter case all parts of the face are affected; the eye cannot be closed, the forehead cannot be wrinkled, and the mouth can neither be widened nor firmly closed, the one half of the orbicularis being paralysed. Hence the patient cannot

whistle, and he cannot "puff" out a candle, for which complete closure and sudden opening of the lips are requisite. But in the case of a supra-nuclear lesion the upper part of the face is little affected, and the orbicularis is scarcely weakened, the chief palsy being that of the zygomatici and elevators of the upper lip. The explanation of this has been already given in the account of hemiplegia, of which such palsy usually forms part. Another important difference is presented by the electrical reaction. In nuclear, and infra-nuclear disease, the nerve-fibres degenerate, and the nerve-trunk, in severe cases, can no longer be stimulated by any form of electricity. The muscles no longer respond to faradaism, since the intramuscular nerve-fibres, on which only faradaism acts, are degenerated; but the muscular fibres still contract to the more deliberate stimulus of a slowly interrupted voltaic current, and even act to this with greater readiness than in health. This is termed the reaction of degeneration. I have explained its characters more fully in speaking of the diagnosis of diseases of the spinal cord. In the complete form of facial paralysis, the loss of muscular tone and action produces a strange effect on the aspect of the patient. In the young, in whom the elasticity of the skin largely moulds the features, the paralysis is little noticeable at rest, but is grotesquely obtrusive on any movement of the face, one side of which may be stern and unbending, while the other is convulsed with laughter. In the old, however, the skin is inelastic, and is thrown into wrinkles by the contracture in which the muscles stereotype the emotions they have habitually expressed. When the muscles become toneless, the wrinkles disappear, and the palsy reproduces the unseared features of an earlier age. The contrast throws into such relief the effect of age, that patients often refuse to believe that the smoother half can be unnatural, and maintain that the wrinkles are the morbid appearance. Indeed you can never place reliance on the statements of patients or their friends as to the side of the face that has been affected. The paralysis of the zygomatici on one side permits their fellows on the other to draw the mouth over, and far over on emotional

expression. This renders the error an easy one. I have often heard a patient say that the unaffected half of the face "must have been the side that was wrong, because the mouth was drawn right up to the ear." Physicians in their turn are liable to be misled in another way. In cases that do not recover perfectly a curious contracture occurs, and voluntary movements, while less in degree, spread too widely over the face, so that the eye closes unduly in smiling, and the corner of the mouth is drawn up when the eye is closed. This contracture deepens the naso-labial furrow; the normal side of the face may be the smoother of the two, and may be thought to be the paralysed side, until the degree of movement is observed. This contracture, in the young, causes a deformity almost as serious as the earlier palsy, but in the old it has a cosmetic influence, bringing back the natural furrows, and rendering the face symmetrical. The common cause of complete palsy is neuritis of the nerve just within the Fallopian canal, or ear disease, damaging it in its course through the temporal bone, and you would not think of an intracranial cause (within the pons or at the base of the brain) unless there were other symptoms of such disease. When the nerve is affected at the lower part of the canal, the chorda tympani often suffers, and taste is lost in the front of the tongue. It is often said that in disease of the nerve higher up, the palate is paralysed, but I doubt whether this is true. For fifteen years I have been looking out for paralysis of the palate in disease of the facial nerve, and I have never seen it. I am inclined to think that the opinion is due to a misinterpretation of the very common congenital obliquity of the uvula, and inequality of the palate.

LECTURE VIII.

SYMPTOMS IN THE REGION OF THE CRANIAL NERVES (*Continued*): AUDITORY; GLOSSO-PHARYNGEAL; PNEUMOGASTRIC; SPINAL ACCESSORY—PARALYSIS OF THE LARYNX AND OF THE PALATE—HYPOGLOSSAL NERVE—COMBINED PALSY OF THE BULBAR NERVES.

GENTLEMEN,—We will to-day continue and complete our study of the symptoms that are due to disturbance of the function of the cranial nerves. The auditory and bulbar nerves remain for consideration. The term "bulbar" is a convenient designation for those nerves that arise from the medulla oblongata. You will remember that the auditory, facial, and sixth arise at the level of the junction of the pons and medulla, but the facial and sixth ascend to their nuclei. That of the sixth is altogether, and that of the facial in great part, above its surface attachment, while the auditory nuclei are at the level of origin of the nerve. The nerves below the auditory, the glosso-pharyngeal, pneumogastric, accessory part of the spinal accessory, and hypoglossal, arise from, and have their nuclei within, the medulla oblongata, and are therefore those included in the term "bulbar nerves."

Disturbances of function of the auditory nerve are occasionally important symptoms of brain disease, but they

are so far more frequently due to derangement of the delicate and complex organ of hearing that the presumption is always in favour of an aural rather than of a cerebral cause. The symptoms of disturbance are three — deafness, tinnitus, vertigo. Deafness may be the result of impaired conduction in the nerve, or of disease of its centre. Tinnitus aurium, or "noises in the ears," is due to irritation of the nerve or its central or peripheral terminations. Vertigo results from interference with the fibres that are distributed to the semi-circular canals, and subserve, not hearing, but perception of the position and movement of the head—the fibres that Cyon calls the "space nerve" (an inapt designation, although the only one that has been given to this important part of the auditory nerve).

The deafness that is due to disease of the auditory nerve is the same in character as that which is due to disease of the labyrinth in which the nerve-fibres end, but it is sharply distinguished from that due to impaired conduction through the tympanic cavity (middle ear) or external meatus. The distinction is that the latter impairs hearing through the air, but leaves unaffected the perception of sounds conducted through the bones of the skull. The former alters both alike. One mode of ascertaining this is with a tuning-fork, and the test depends on the fact that the hearing through the air is normally rather more acute than that through the bone. A vibrating tuning-fork is held in contact with some part of the skull, say the parietal eminence. As soon as it ceases to be audible it is removed from the skull, and held opposite the auditory meatus. It should be still distinctly heard. If it is not, there is impaired conduction through the meatus or middle ear, and the former can readily be excluded by the speculum. If the tuning-fork is still heard, any deafness must be of nerve origin, *i.e.*, due to disease of the auditory nerve or nerve-endings in the labyrinth. The watch affords another test that is of great practical importance, not merely on account of its convenience, but because it is even more delicate than the tuning-fork, since perception, through the bone, of the short high-pitched sound of a watch is, for some reason, often

impaired out of all proportion to the loss to the tuning-fork. The meatus should be closed by pressing the antitragus over it, the watch held close to, but not touching, the zygoma, and the patient asked if he can hear it. It should then be pressed firmly on the zygoma. In health it can scarcely be heard in the former position, but the sound becomes loud as soon as it is pressed against the bone. This is the case also in disease of the middle and external ear. In disease of the labyrinth and nerve, it is common for the sound to be quite inaudible when the watch is in contact. When this is the case we can be quite sure that there is nerve-impairment. Indeed, a caution is necessary on account of the delicacy of the test. Many persons, especially after middle life, have lost the power of hearing a watch through the bone, when they are conscious of no impairment of ordinary hearing. This is no doubt due to pathological changes in the labyrinth, for the change is often one-sided, and many of these persons suffer from tinnitus or vertigo. If the sound is still heard through the bone, although not so loudly as in health, we cannot infer disease of the labyrinth or nerve, because simple anchylosis of the stapes will lessen perosseal hearing (since some vibrations pass through the chain of bones), but no disease of the middle ear will extinguish all hearing through the bone. Galton's whistle, by which a very high-pitched note is produced, may also be used to test the hearing of such continuous sounds, but the pathological significance of their loss has yet to be defined. Very rarely a limitation of hearing exists, analogous to the limitation of the field of vision. It probably occurs chiefly in degeneration of the optic nerve. Thus, a patient with locomotor ataxy and optic nerve atrophy, who doubtless has also atrophy of the auditory nerve, has gradually become considerably deaf to all sounds, and is absolutely deaf to the loudest musical notes above E of the treble clef, and below the lower G of the bass.*

* Since the above was written, the range of hearing has gradually become further restricted, until only the notes between the two E's of the treble clef can be perceived. Even the lower E flat is inaudible.

Deafness from intracranial disease is usually due to disease of the nerve at the base of the brain, less frequently of its nucleus in the medulla. It very rarely results from disease of the auditory centre in the first temporo-sphenoidal convolution, and is then on the side opposite to the disease. Bilateral deafness may be due to damage to both auditory nerves, and I have also known it to be produced by a tumour of the corpora quadrigemina damaging the upper layer of the tegmentum of each crus cerebri, in which the auditory path lies. From symmetrical disease of the cortical centres it is extremely rare. But deafness on both sides is frequently due to symmetrical labyrinthine disease.

Subjective noise, tinnitus aurium, is, like deafness, usually aural in origin. It may result from almost any disease in any part of the ear. It may be due to an abnormal sensitiveness to the movements that are normal in the ear—of the blood in the vessels (especially in the internal carotid), or of the muscles within the tympanic cavity,—or it may be due to an abnormal increase of these movements, *e.g.*, to an increased vibration of the moving blood in anæmia, or in carotid aneurism. It may be due to pressure on the auditory nerve, or to irritation, functional or organic, of the auditory centres. Its characters are extremely varied. The first point to be ascertained is whether the sound is pulsatory or continuous. In the former case the pulsation will be found to be synchronous with the heart's action, and the sound probably has its origin in the ear. Such a sound has been known to be due to an aneurism, and to be audible on auscultation of the skull. This is the only case in which a murmur is so audible, and so the diagnostic significance of the phenomenon is great, although it is extremely rare. Sounds that are due to irritation of the labyrinth or nerve, or are of central origin, are usually (though not always) continuous, and not pulsating. The next important fact is the elaboration of the sound. Sounds that are of simple character, "rumbling," "buzzing," "hissing," "whistling," may be of either local or central origin, but those that are of higher elaboration, such as bells or music, are generally, the latter

always, of central origin. I have known the sound of bells ringing to be the aura of epileptiform convulsions due to a tumour beneath the first temporal convolution. We have little definite knowledge of the relation of tinnitus to disease of the auditory nuclei of the medulla, but the sound of a loud crash has attended the onset of acute lesions in the vicinity of the nuclei. Whether the sound is referred to the ears, or vaguely to the head, is of little diagnostic importance. Intense bilateral sounds of subjective origin are usually referred to the head, and not to the ears. Tinnitus and deafness are often associated, and must then be ascribed to the same cause. The vertigo that results from affection of the auditory nerve will be considered in connection with other forms of vertigo.

The glosso-pharyngeal nerve and the pneumogastric need not detain us long, since their isolated affection forms a less frequent factor in the diagnosis of brain disease than does a general derangement of their function. This may occur from a severe lesion in any part of the brain, but is most conspicuous when the disease is in the medulla oblongata. We will consider subsequently the chief symptoms that are due to derangement of the visceral functions of the pneumogastric, and deal now only with two effects of the impairment of these nerves—paralysis of the pharynx, and paralysis of the larynx. With these, however, we may, for reasons that will appear as we proceed, most fitly consider the paralysis of the palate.

The motor nerve-supply to the pharynx is derived from the pharyngeal plexus, into which both the glosso-pharyngeal and pneumogastric enter largely, and we do not know from which of these nerves the motor fibres come. In paralysis of the pharynx, swallowing is difficult; food is apt to lodge, or get into the larynx, and particles may even find their way into the lower air-passages, and, after a time, set up chronic disease in the lung. Paralysis of the pharynx is rarely an isolated symptom of brain disease, and its diagnosis is not

difficult. I have known it to be mistaken for cancer, but an examination should prevent the error. Palsy is painless, while cancer rarely is, and the former is usually accompanied by loss of power in adjacent parts.

You will remember that the larynx receives only sensory fibres from the pneumogastric proper, its motor fibres coming entirely from that part of the spinal accessory that joins the vagus. You will remember also that the superior laryngeal nerve contains the sensory fibres for the vocal cords, and for the larynx above them, and motor fibres for the crico-thyroid muscle, while the inferior or recurrent laryngeal supplies all the other muscles that act on the glottis, and contains the sensory fibres for the larynx below the vocal cords. The vocal cords are abducted, and the glottis opened, chiefly by one muscle—the posterior crico-thyroid—which passes upwards and outwards from the back of the thyroid to the outer muscular process of the arytenoid; and, drawing this back, moves the vocal process outwards. The cords are adducted, and the glottis closed, by several muscles, of which the most important is the lateral crico-arytenoid, which, passing backwards from the side of the cricoid cartilage to the outer process of the arytenoid, moves this forwards, and is thus the opponent of the posterior muscle. But the outer fibres of the thyroid-arytenoid muscle, which pass, parallel to the vocal cord, from the thyroid cartilage to the muscular process of the arytenoid, have a similar although feebler action. This closing rotation of the arytenoids is supplemented by the arytenoideus, which, passing from the back of the one to the back of the other arytenoid cartilage, brings the two together. The vocal cords are lengthened and made tense by the crico-thyroid, which draws back and slightly tilts the cricoid cartilage, and they are made tense or lax in parts by the inner fibres of the thyro-arytenoideus, which end at different points along the cord. But the muscular mechanism must be much less simple than this. In the case of some muscles, all the fibres have not the same direction, and may have different actions according to their association. Doubtless, in the delicate and varied

actions that produce vocal sounds, complex associated actions of the fibres are concerned.

The symptoms of paralysis are threefold—altered phonation, deranged regulation of the entrance of air in breathing, and defective movement (observable with the laryngoscope). The phonic and respiratory functions of the glottis are subserved by the same muscles and the same nerves, but by centres that must differ in their anatomical connection, if they do not in their anatomical position. After death, the vocal cords are in a position of slight abduction from the middle line, and this, termed the "cadaveric position," must be regarded as that of muscular relaxation—of that rest which, during life, they never actually attain, since they move with every breath. They move farther apart during inspiration; they come nearer together during expiration; while in phonation they are brought very near together and made more tense. In total paralysis of all the muscles, the cords are in the cadaveric position, and do not move with breathing or on an attempt to produce sounds. Instead of the natural explosive cough there is only a rush of air through the glottis. There is some stridor on a forcible inspiration. If only one vocal cord is completely paralysed, some hoarse phonation may still be possible by extreme adduction of the normal cord, and its abduction prevents inspiratory stridor, but a proper cough is still impossible. Such one-sided palsy may result from disease of the nucleus of the spinal accessory in the medulla, of its roots at the surface of the medulla, of the trunk of the vagus, and even of the recurrent laryngeal, since the escape of the crico-thyroid in the latter case does not materially modify the symptoms. Hence the palsy itself does not help us to determine the position of its cause.

In other cases of bilateral palsy the cords are nearer together than the cadaveric position. They can be approximated for voice or cough, and when the effort is over, their elasticity may make them recede a little; but they cannot be abducted even as far as the cadaveric posture, and the normal separation during inspiration does not occur. This is called paralysis of the abductors, the posterior crico-arytenoids. The longer the

palsy lasts, the closer together are the cords, in consequence of a secondary contracture of the unopposed adductors. The cords being always in the position of phonation, voice is little affected, but the absence of the normal separation during inspiration causes a serious impediment to respiration, since the inrushing air brings the cords still nearer together, and causes a loud inspiratory stridor, and dyspnœa on the least exertion. The absence of expiratory stridor distinguishes the dyspnœa from that due to tracheal stenosis, and, together with the integrity of the voice, distinguishes this from any other laryngeal affection. The condition is one of considerable danger, since the least catarrhal swelling of the mucous membrane may necessitate immediate tracheotomy to prevent suffocation. If this abductor palsy is one-sided, the symptoms are slight or absent, and the diagnosis can be made only by the laryngoscope. The most frequent cause of abductor palsy is central degeneration; but it sometimes occurs, strange to say, in severe hysteria, even, as I have seen, in extreme degree. Equally strange is the well-established fact that it may result from disease of the recurrent laryngeal, which, supplying the adductors also, should cause only a total palsy. This has given rise to much speculation as to its mechanism. Some light seems to be thrown on it by the fact that electrical stimulation of the recurrent laryngeal also causes adduction, although all the muscles, adductors and abductors, must be equally stimulated to over-action. This must be due to the greater power of the adductors, perhaps also to the mechanical advantage at which the chief adductor, the lateral crico-arytenoid, acts, in comparison with the abductor, the posterior crico-arytenoid, since the former passes nearly at right angles, the latter at a very acute angle, to the muscular process of the arytenoid cartilage. A force acts on a lever at greatest advantage when applied at right angles to the lever. The effect of a general under-action may be to impair the effect of the abductors more than that of the adductors, just as the general over-action on electrical stimulation increases the effect of the adductors out of proportion to that of the abductors. According to this explanation, the abductor palsy would be the effect of paralysis of the recurrent, when it is

incomplete in degree, although not necessarily partial in distribution; while the total palsy with cadaveric posture would be the result of complete paralysis. In harmony with this is the fact that in progressive disease of the recurrent an initial abductor palsy has been observed to pass into total palsy (Schech, Rosenbach). In some cases, as Riegel suggests, secondary contracture of the crico-thyroid may aid in causing the adduction; but since adduction is often absent in total recurrent palsy, the influence of this contracture cannot be great. When the palsy is first, and for long, of the abductor type, secondary tissue-changes in the preponderating adductors may perhaps maintain the glottis in adduction, even when the palsy has become complete.

In simple adductor palsy the cords are apart, and cannot be brought together, but further abduction occurs in deep inspiration. The cords are not approximated in speech, and so the patient is voiceless, but they can still be brought together in coughing. Hence it has been termed by Türck, "phonic paralysis."* This is the cause of hysterical aphonia, but is scarcely ever produced by organic disease. It is readily distinguished, by absolute voicelessness and perfect cough, not only from other laryngeal palsies, but also from other diseases, such as catarrhal laryngitis, in which there is a hoarse attempt at phonation.

These laryngeal palsies can be diagnosed with certainty only by the aid of the laryngoscope; but the symptoms themselves often justify a strong suspicion of the nature of the affection, and it may be well to put them before you in the form of a table. You will see that the inability to effect an explosive cough is of great significance, and should lead you to suspect a palsy, probably of organic origin; you will observe that if voice is also entirely lost, the palsy must be double. If voice is preserved and cough lost, you suspect one-sided palsy. Loud inspiratory stridor with preserved voice means double abductor palsy; a normal cough and no voice or stridor signifies an unimportant adductor palsy.

* A much more exact term than his attractive but loose antithetic designation of "respiratory paralysis" for abductor palsy.

SYMPTOMS.	SIGNS.	LESION.
No voice; no cough; stridor only on deep inspiration.	Both cords moderately abducted and motionless.	Total bilateral palsy.
Voice low-pitched and hoarse; no cough; stridor absent or slight on deep breathing.	One cord moderately abducted and motionless, the other moving freely, and even beyond the middle line in phonation.	Total unilateral palsy.
Voice little changed; cough normal; inspiration difficult and long, with loud stridor.	Both cords near together, and during inspiration not separated, but even drawn nearer together.	Total abductor palsy.
Symptoms inconclusive; little affection of voice or cough.	One cord near the middle line not moving during inspiration, the other normal.	Unilateral abductor palsy.
No voice; perfect cough; no stridor or dyspnœa.	Cords normal in position and moving normally in respiration, but not brought together on an attempt at phonation.	Adductor palsy.

The nerve-supply to the palate is one of those points in anatomy that urgently require re-investigation. The most important muscle is the levator palati, which receives a nerve from the spheno-palatine ganglion, but the ultimate origin of its fibres is certainly not from the fifth nerve. They are usually said to come by the Vidian from the facial, but they probably really come from one of the bulbar nerves, either the glosso-pharyngeal or spinal accessory. Paralysis of the palate results from disease of these bulbar nerves (at the surface of the medulla) or of the bulbar nuclei. It is doubtful whether it ever results from disease of the facial nerve, and certainly it does not from disease of the fifth. The centres for the palatine muscles are especially obnoxious to the mysterious influence that diphtheria leaves behind it, and this is the most frequent cause of simple palsy of the palate. In total palsy, the soft palate hangs low and flaccid, the uvula is long, and no movement occurs in drawing a deep breath, in phonation, or on tickling the mucous membrane. The posterior nares are no longer closed. Hence, during swallowing, liquids are apt to come back through the nose; during speaking there is a nasal resonance, and the explosive consonants, as *p* and *b*, are no longer properly articulated, because the patency of the nares prevents the necessary compression of the air in the mouth,

and they are transformed into *m*. In unilateral palsy these symptoms are absent. All the muscles, even of one side, are rarely paralysed, since unilateral palsy is scarcely ever due to disease of the centre (which affects both sides), and the course of the fibres to the different muscles is not the same, and therefore a lesion does not affect all of them. The palate at rest presents little change in unilateral paralysis. On one side it may be a little lower than the other. The uvula may hang a little to one side, but palsy never causes any curve in the uvula, and no abnormal position of the palate or uvula can be due to paralysis, unless it is increased on movement. The most important symptom of one-sided palsy is recognized only on movement. At rest, the palate may appear equal, but if the patient is made to say "ah," the base of the uvula deviates a little to the unaffected side, and a little on this side of the middle line, about midway between the arch and the hard palate, a slight dimple forms, while the unparalysed side remains smooth. The difference is obvious and characteristic. There is no deviation of the uvula. The difference between the two sides, on movement, evidently depends on paralysis of the levator palati. It occurs together with paralysis of the tongue and vocal cord on the same side, when there is disease at the side of the medulla damaging the hypoglossal and spinal accessory nerves. This was first pointed out by Dr. Hughlings-Jackson. Whether the paralysis depends on the disease of the spinal accessory, or on disease of the adjacent glosso-pharyngeal, is uncertain, but the association leaves no doubt that the motor fibres for this, the most important of the palatine muscles, come from one of these bulbar nerves. I have known the same three palsies (tongue, palate, and vocal cord) to result from a deep-seated tumour in the upper part of the neck, which must have damaged the nerves outside the skull.

When the accessory part of the spinal accessory is injured by disease at the foramen magnum, the spinal portion may suffer also, causing wasting and loss of power of the sterno-mastoid and upper part of the trapezius.

Disease of the hypoglossal nerve, outside or within the medulla, causes paralysis of the same half of the tongue; and disease of the motor tract above the nucleus causes paralysis of the opposite half of the tongue. Disease of the nucleus itself almost always causes paralysis of both sides of the tongue, because degeneration affects both nuclei, and the two lie so near together that both suffer in acute lesions. In one-sided palsy, the tongue, at rest, is in its normal position in the mouth, but the base is higher on the paralysed side, owing to lack of tone in the posterior fibres of the hyoglossus. Within the mouth, movement is deficient towards the paralysed side, but on protrusion the tongue deviates from the sound, and towards the paralysed side, being pushed out and over by the unaffected genio-hyoglossus. In bilateral palsy the tongue lies motionless within the mouth, and cannot be moved. In disease of the nerve or nucleus, the tongue usually wastes, and the mucous membrane lies in irregular folds over its surface.

The process of articulation is effected by the muscles supplied by these bulbar nerves, and its impairment always constitutes an important symptom of their disease, or of disease in the part of the brain from which they arise, and in which a complex structural association subserves the conjoined action of the various muscles concerned in the process. Articulation consists in stopping and varying the outgoing current of air, which is often thrown into vocal vibrations, on which the process of articulation effects more elaborate and perfect modulations than it can in merely whispered utterance. In the whole series of movements that can be effected by the muscles of man, there are none comparable to these in exquisite delicacy of adjustment, and in the infinite diversity of result that a few simple muscles can produce by their combined action. It is not surprising that commencing failure of the adjustment of nervous action should be manifested in these sooner and more than in any other mechanism. There are two chief forms of defect of articulation. The first is due to paralysis of the muscles, and in this the loss of power can

be recognized in other movements. The precise form of defect depends on the muscles that are weak, and this I have already alluded to, and will return to in a moment in speaking of the combined palsy. In the other variety there is no actual paralysis, but the delicate adjustment is at fault. Syllables are run together, in what may be termed "confluence of articulation"; the ends of words are not well pronounced, are even elided; or the syllables may be unduly separated, in what is termed, from a musical analogy, "staccato" utterance. The most delicate of all the movements is that involved in the pronunciation of *r*, and it is in this that the commencing defect is often most conspicuous, as by making the patient say "truly rural." Often there is a peculiar drawl, and this may be combined with elision of syllables. In many cases, and especially in the paralytic defect, the patient can utter a word by a deliberate effort much better than he does in habitual speech.

Before leaving the subject of the symptoms of the cranial nerves, one other subject should be noticed. Certain of these nerves are liable to suffer together from disease of their nuclei, usually from degenerative disease. The nerves thus associated in disease are the two groups of motor nerves that are associated in function. The first group is that of the nerves for the eyeball muscles, and its disease causes the progressive ophthalmoplegia that I have already mentioned. The other group is that of the nerves for the complex series of muscles of the orifice and upper part of the respiratory passages, mouth, throat, and larynx,—the fibres of the facial for the orbicularis oris, the hypoglossal, the fibres to the palate, perhaps from the glosso-pharyngeal, those that give motor power to the pharynx, and the laryngeal fibres of the spinal accessory. Before considering the associated palsy that results from nuclear degeneration, I may remind you of the associated one-sided palsy of tongue, palate, and larynx, which I have just described as the result of disease outside the medulla. The tongue deviates to one side; the middle of the soft palate is drawn to one side when it is raised, and

if the larynx is examined, the vocal cord on that side is seen to be motionless. These symptoms are due to damage to the roots of the hypoglossal, spinal accessory, and perhaps also of the glosso-pharyngeal.

In the associated nuclear palsy—"bulbar paralysis," as it is often termed—the paralysis involves the same parts, tongue, palate, and vocal cord; but it is bilateral, and its functional distribution is more complete, since the lips and pharynx are usually also involved. You will remember what I said of the central relations of the nerve-fibres for the lips: although they run in the facial nerve, they must be derived from nerve-cells that have the most intimate connection with those for the transverse muscle of the tongue, and the two may even be derived from the same nucleus. The association of the lips with the other parts caused Duchenne to give to the disease the name "labio-glosso-pharyngeal paralysis," by which it is still often known. The symptoms are, as it were, grouped about the tongue as a centre, and it is in the delicate movements of lingual articulation that the first symptoms occur—a clumsiness in the pronunciation of the lingual consonants *l*, *r*, *n*, *t*, and *s*. Subsequently the degree of protrusion of the tongue becomes impaired, until at last only the tip can be put beyond the teeth. It is often conspicuously wasted. The early weakness of the lips prevents whistling, and the labial explosives *b* and *p* become *f*, and *v* becomes *u*. This transformation is assisted by the weakness of the palate, which ceases to shut off the nasal cavity, and so interferes with the compression necessary for explosive sounds; hence also nasal resonance persists in all sounds. Swallowing becomes difficult; liquids regurgitate into the nose; food gets into the larynx, and this may be so paralysed that the glottis cannot be closed, and an explosive cough is impossible. Ultimately a low, hoarse vocal sound is all that remains of speech; but it is rare for the paralysis of the larynx to be complete.

LECTURE IX.

SYMPTOMS (*Continued*): MENTAL DISTURBANCE—LOSS OF CONSCIOUSNESS—APOPLEXY—DELIRIUM—MENTAL WEAKNESS—LOSS OF MEMORY.

Gentlemen,—The symptoms that have hitherto engaged our attention are the derangements of special functions subserved by special nervous structures. Those that we have now to consider are more general in nature, and the symptoms caused by their derangement are for the most part "diffuse." It is convenient to consider with these some symptoms that are "focal," but which are closely allied to those that are general. Thus it is better to study defect of speech, a focal symptom, after we have considered derangement of mind, a general symptom.

The highest functions of the brain are those concerned in mental processes, and the derangement of these is a frequent and obtrusive effect of organic brain-disease. But these symptoms do not stand in the special and pre-eminent relation to such disease that might reasonably be anticipated. The highest cerebral functions are so readily disturbed, that their derangement is less frequently the result of organic brain-disease than of changes in the blood, on the one hand, or of the minute alterations of nutrition that we term functional disease, on the other hand. The most significant disturbance from organic disease is the coarsest, such as uncon-

sciousness. The slighter disturbance, such as delirium, derives its significance from its associations, not from its simple presence.

We may consider first the profound and important mental symptom that I have just mentioned—loss of consciousness. Remember that the terms "conscious" and "consciousness" are used in two senses: first, to signify subjective knowledge of the occurrence of mental processes; and secondly, outward manifestation of such processes. In medical language the words are chiefly used in the second of these two senses. A patient is said to be "unconscious," or to have "lost consciousness," when there is no evidence of mental action, either spontaneous or in response to attempts to elicit it. The term "insensible" is often applied to the same condition. Another confusion is introduced by the use of the term "conscious of," or "unconscious of," in the sense of cognition, or its absence. Thus a delirious patient is said to be unconscious of what is occurring around him, although he is not said to be unconscious.

Loss of consciousness may occur suddenly or gradually, may be complete or incomplete. The variations may be in the degree of subjective consciousness, or of the external manifestation of consciousness; and it is to the latter that the term "partial loss" is usually applied, as, for instance, to the condition in which a patient lies apparently asleep, but opens his eyes when spoken to, immediately relapsing into sleep. This condition is often termed "stupor." Complete unconsciousness, lasting more than a few minutes, is termed "coma." In both conditions there is usually imperfect control over the sphincters. In stupor, the reflex action on the limbs is preserved, and sometimes increased; the patient swallows automatically liquid placed in his mouth; the pupils act to light. In coma, the reflex action in the limbs is usually lessened, and often lost. Muscular tone gives place to flaccidity, and with this change myotatic irritability often disappears. The pupils may be widely dilated or small, and do not act to light, at any rate when the coma is deep, and then the conjunctival reflex is also lost. The act of swallow-

ing may or may not be possible; in deep coma it is lost, and the palate, sharing the muscular relaxation, vibrates under the current of air, and causes the peculiar "stertor" which is a familiar indication of the depth of coma. Even the respiratory movements are lessened, in consequence of lowered activity of the respiratory centre; they become shallow, infrequent, and sometimes present rhythmical variations of intensity, in what is termed the "Cheyne-Stokes breathing"—alternating periods of decreasing and increasing depth of breathing, separated by a pause. The lessened breathing fails to clear the air-passages of the secretion in them; this accumulates in the bronchial tubes, and is often erroneously regarded as evidence of bronchitis; finally, mucus collects in the trachea, and causes the well-known harbinger of death.

Consciousness may be impaired by almost any one of the many morbid processes to which the brain is liable, whether acute or chronic. It results from chronic and subacute disease chiefly when this damages a considerable area of the cortex, either directly, or indirectly by causing a rapid or extreme increase in the intracranial pressure. It results from sudden lesions in any part of the brain, and is then usually sudden in onset, and termed "apoplexy."

Of all the sudden lesions that cause apoplexy, intracranial hæmorrhage is the most effective, and the most frequent; hence "apoplexy" has come to be a synonym for internal hæmorrhage, whatever be its seat. Next in frequency is the sudden occlusion of a large artery by a plug brought from a distance (embolism), or formed *in situ* (thrombosis). It may result from congestion of the brain, although it does so far less frequently than is commonly supposed. A similar sudden loss of consciousness may occur in the old without any visible lesion of the brain to which it can be ascribed. This has been termed "simple apoplexy." In senility the brain shrinks; the space between the convolutions is occupied by serum. This change, which is common in the old, and without significance, when found in old persons who had died of simple apoplexy was thought to be important, and the

cause of death; hence the condition was termed "serous apoplexy"—a disease that has no real existence, although the name still survives, and now and then finds its way into certificates of death.

The characteristic of apoplexy is sudden loss of consciousness, not due to any cause outside the nervous system, such, for instance, as failure of the heart's action or a poison in the blood. The onset may be sudden; the patient falls as if "struck" down by some unseen hand,—an idea that is fossilized in the name, and in its English synonym, a "stroke." Sometimes the onset is gradual; consciousness slowly fades; stupor slowly deepens into coma: and this has been termed "ingravescent apoplexy." The face may be flushed or pale; it is rarely very pale. Often the heart and arteries pulsate strongly, but sometimes less frequently than normal. The temperature is usually at first depressed; its subsequent course varies with the cause of the apoplexy. An exception to the initial depression of temperature is presented by active lesions of the pons, which, whatever be their nature, are sometimes attended with a rapid rise of temperature, that may, in an hour, reach 105° to 106°. If the attack is one of moderate severity, reflex action soon returns, and in the course of a few hours some indication of returning consciousness can be perceived. On the other hand, the coma may deepen, and the interference with breathing, already described, may come on. In most cases the symptoms of apoplexy are accompanied by those of a local cerebral lesion, commonly by those of hemiplegia, previously detailed.

The symptoms of apoplexy are those of lowered cerebral function, beginning at the highest, and extending downwards to lower centres in proportion to the depth of the coma. Its precise mechanism has been much discussed, but is a matter of theoretical rather than of practical importance. It is easy to frame a simple and satisfactory hypothesis of the way it is produced by any one lesion, but the variety of its causes shows that more than one mechanism may be concerned in its production, and suggests that its origin is complex in every case. Sudden increase of intracranial pressure causes

loss of consciousness, but in what degree the result is due to the mechanical action on the nerve-elements, or to anæmia from the compression of the capillaries, is uncertain. This mechanism is doubtless effective in cerebral hæmorrhage, but it is probably not the sole mechanism, even in this case, because apoplexy may result from a very small hæmorrhage, and consciousness may be lost at the very onset of a hæmorrhage. The sudden occlusion of a vessel will cause apoplexy, but cannot do so by its influence on the intracranial pressure. Attempts that have been made thus to explain it are remarkable chiefly for their ingenuity. In most cases of apoplexy there is a sudden damage to cerebral tissue. Sudden arrest of blood-supply constitutes a damage as effectual as laceration. Hence it is probable that one element in the production of the loss of consciousness is the inhibitory effect of the irritation of the lesion. We have evidence of a downward influence of this character in the initial loss of muscular tone and muscle reflex action (*e.g.*, in the loss of the knee-jerk*). A similar upward action, inhibiting the highest centres, is probably the cause of the initial loss of consciousness, the prolongation of which may be helped by other mechanisms, such as increased pressure. All lesions are effective in proportion to the rapidity with which they are produced. Experiments, for instance, show that the amount of intracranial pressure needed to abolish consciousness has to be ten times greater when slowly than when rapidly produced.

The diagnosis of the cause of apoplexy we shall consider when we come to the last part of our subject—to the indications of the nature of the brain disease,—but I may briefly point out to you some of the chief differences between the coma of cerebral origin and that which may result from causes outside the nervous system, or from cerebral derangement that is merely functional in character. In all cases the most important point is to search carefully for any evidence of a local cerebral lesion, and especially for the indications by

* See "Diagnosis of Diseases of the Spinal Cord," 3rd Ed., p. 31.

which hemiplegia can be recognized during the state of coma. These I have already described to you (p. 54). The reflex actions are of especial importance. If these are all perfectly normal, this is, in the absence of other decided symptoms, against the existence of a cerebral lesion. An abnormal condition of reflex action is in favour of it, and is almost conclusive if the abnormality is unilateral.

We will take first the functional disorders of the nervous system. A patient may be unconscious for an hour or two after an epileptic fit, and this may be mistaken for apoplexy with a convulsion at the onset. Such a difficulty can scarcely arise unless the history of the patient is unknown, since first convulsions are rare, except in those in whom cerebral apoplexy is also rare. Post-epileptic unconsciousness resembles sleep more than it resembles coma. The patient can be readily roused. The temperature is nearly normal—never below normal, as it often is in apoplexy,—and unilateral symptoms are absent. The convulsions that cause transient post-epileptic hemiplegia are always one-sided, and are not followed by such deep sleep as to raise the question of a possible cerebral lesion. The patient soon emerges from the mental obscurity that follows an epileptic fit, and often at once passes into a normal condition, but occasionally is still "befogged" for a longer or shorter time—in a wandering, stupid state of mind, that is itself very characteristic.

The state of unconsciousness that occurs in rare cases of hysteria simulates apoplexy less than it resembles the coma due to less rapid cerebral processes, such as meningitis. A patient, for instance, after a period of headache, becomes unconscious, swallows what is put in the mouth, but cannot be roused. The diagnostic indications are the age and sex of the patient, who is usually either a female or a boy; the absence of any cause of a cerebral lesion; the history of other symptoms of functional nervous disturbance (especially of hysterical convulsions or vertical headache); the cessation of headache when the coma comes on; the absence of all objective symptoms; the inconsistence between the ready

deglutition and the apparent depth of coma; and the uniform course of the affection, which often presents no change for many days.

Of causes outside the nervous system, uræmic poisoning is that which most often causes perplexity, because its frequent cause, chronic kidney-disease in the second half of life, is also a frequent cause of the apoplexy that it resembles. Albuminuria is constant in uræmia, frequent in apoplexy; its absence is therefore more significant than its presence. In many cases the coma is preceded by other uræmic symptoms, especially by convulsions or amaurosis. Severe general convulsions may, it is true, usher in both affections, but at the onset of apoplexy the convulsion is usually single; at that of uræmic coma there are many. Sudden complete amaurosis is almost conclusive evidence of uræmia. An acute cerebral lesion scarcely ever causes total blindness. Elevation of temperature is strongly in favour of cerebral mischief; depression is consistent with either, but continuous depression, lasting for two or three days, is strongly in favour of uræmia. An examination of the fundus of the eye will, of course, be one of the first steps you take. Albuminuric retinitis often proves the existence of renal disease, when an examination of the urine is, for the time, impracticable. It shows, moreover, profound systemic mischief. It always indicates that the patient is in a state in which uræmic symptoms may come on at any moment. The absence of retinal change does not exclude uræmia, any more than its presence proves that uræmia is the cause of the coma.

In profound alcoholic poisoning the diagnosis from cerebral apoplexy may be extremely difficult. When you have no history to guide you, as is often the case with the patients who are brought to a hospital or a police-station, the diagnosis may be impossible. Many a patient with apoplexy has been locked up in a police-cell all night as dead-drunk. Brandy is the universal panacea for impairment of consciousness; and so apoplectic patients often smell of alcohol. If focal symptoms are absent there is no distinctive

indication. A few hours will always decide the question; and it is better to let a drunken man get sober in bed, than to let a patient with ventricular hæmorrhage die in a police-cell. We need, however, more facts regarding the state of reflex action in profound alcoholic poisoning, especially of the muscle reflex action (knee-jerk, foot-clonus, etc.). It is not improbable that some useful guidance may be found in these symptoms. This is one of the many points on which those of you who are hospital residents may make valuable observations.

The danger of confusing opium-poisoning and apoplexy is not great, because the patient who has taken opium usually comes under observation while the pupils are contracted to an extreme degree—conclusive, except as regards the distinction from hæmorrhage into the pons. In the latter there is often a history of sudden onset, and usually objective symptoms are present that permit no doubt as to the nature of the case.

From the lessened manifestation of mental activity we may pass to the disorder and excess that constitute "delirium," the condition in which mental processes are not in accordance with sense-impressions, and there is no consciousness of the discrepancy. The condition is essentially the same as that which constitutes "insanity"; but the term delirium is used when the mental derangement is acute in course, and occurs in consequence of organic brain-disease or of some blood-state. Delirium is commonly distinguished as "quiet" or "active." In the former there are hallucinations, especially of sight, and these dominate the patient's ideas. He often talks continuously, but in a low monotonous voice, and it may be difficult to make out what is said—a condition aptly termed "low muttering delirium." On the other hand, in "active delirium" there is more energy in the manifestation of the mental processes, and the patient tries to act according to his erroneous ideas. Although the elements of delirium are identical in nature with those of what is termed insanity, certain common features of the latter rarely occur in delirium.

Such are the extreme and persistent emotional depression of melancholia, the exaggeration of idea that is common in general paralysis of the insane, the outrageous delusions of personal identity met with in some cases of chronic insanity, and the rhetorical loquacity of acute mania.

Delirium is far less frequently the result of organic disease of the brain than of altered conditions of the blood, especially that in pyrexia. There is rarely anything in the character of the delirium to indicate to what cause it is due. In acute alcoholic delirium, unpleasant visual hallucinations are a marked feature, and there is usually conspicuous tremor; but chronic alcoholic delirium may present none of these characteristics. The delirium due to pyrexia and that resulting from organic brain-disease present no difference. Since pyrexia is by far the most common cause of delirium, this symptom alone is suggestive of organic brain-disease only when there is no pyrexia, or only a slight degree of pyrexia, insufficient to account for it. It must, moreover, be remembered that those who are addicted to alcohol, and those who are old, are rendered delirious by a slighter degree of blood-change than is necessary in the case of the sober and the non-senile. The caution to attribute no weight to delirium, unaccompanied by other symptoms of brain disease, if the patient has considerable fever, may seem a simple rule; but, like many other simple rules in diagnosis, it is often forgotten, and consequent mistakes are frequent. Not long ago I saw a man who was said to have inflammation of the brain, and I found he had only inflammation of the lungs. The delirium had so misled the doctor in attendance that he had not even examined the lungs. Again, a child became feverish and delirious: the medical attendant diagnosed tubercular meningitis, and foretold a speedy death. But the pyrexia was sufficient in degree to account for the delirium: there was no evidence of anything more than a catarrhal febricula, and in a week the child was well. Do not, however, go to the other extreme, as some have done, and conceive that delirium is of no significance as an indication of organic disease. It is significant

when there is no other discoverable cause, and it may both confer and receive significance by association with other cerebral symptoms. These may be various in character, but one of the most frequent is headache. Headache, like delirium, is an effect of fever. But the headache of fever ceases when the delirium comes on; that of brain disease persists. The coexistence and the sequence of the two have, therefore, quite a different significance. If the patient is delirious, and has, at the same time, severe pain in the head, you should suspect organic disease. Of course, pyrexia often accompanies delirium from organic disease, and we have then to depend on the other symptoms for our diagnosis, or on the order of the two. If the delirium precedes the fever, it has evidently the same significance as if it existed alone. The various other symptoms that may give significance to delirium need not be enumerated here.

Mental weakness shows itself in failure of power in all the various mental processes, and of these loss of memory ("amnesia") is the most conspicuous and the most tangible. Memory, like other mental actions, has its physical side. Every functional state of the nerve-elements leaves behind it a change in their nutrition, a residual state, in consequence of which the same functional action occurs more readily than before; and this residual disposition is increased by repetition. This is the basis of motor training, which consists in a sort of motor memory that enters little into the region of consciousness. The same residual disposition in the cells that act during mental processes no doubt influences the revival of those processes in memory properly so-called. The sequence of action of groups of nerve-cells is the physiological aspect of that which, in its psychological aspect, we term the association of ideas. There is no special faculty of memory, physical or psychical, apart from the general cerebral and intellectual processes. But there is, or seems to us to be, a peculiar power of the voluntary revival of these processes—of the re-energizing of the residual tendencies,—a faculty that is popularly termed "recollection."

Any disease of the brain may affect memory, whether it be coarse organic disease or finer degenerative processes. Moreover, temporary malnutrition, as from acute disease, or severe anæmia, may have the same effect. The defect may be seen in the inability to retain new impressions, or in the loss of those that are recent. As Ribot has put it, "the new perishes, the old endures." Strange examples of this are sometimes met with. I have seen a clergyman who had lost all memory of the last twenty years of his life. Those years had passed over him, leaving their marks indelibly on his frame. They had been years of active work, and at the end of them he had an illness. When he recovered, all memory of those years had vanished. In rare cases of the kind, memory has returned, and the lost time comes back in the order from the past towards the present.

Another symptom of mental failure, with which, indeed, loss of memory is closely associated, is deficient power of attention, of excluding all but one subject from the domain of consciousness. This may be one cause of failure of memory, and it sometimes causes a failure to remember when there is no real failure of memory. When one subject dominates the mind, sufficient attention is not given to other subjects to secure their retention. Hypochondriacal patients often present this inability; their minds are constantly occupied with their own feelings, and they do not give sufficient attention to other subjects to ensure their persistence in the mind, and the apprehension of mental failure is added to the other sources of mental distress. Reassure the patient as to his fancied ailments, and the unmeaning character of his various sensations, and his loss of memory will vanish.

Closely connected with failure of the power of attention is incoherence of idea. Instead of the definite sequence of mental processes that we recognize as normal, one mental image excites another by some accidental association, which would be unnoticed in health. Often the connections that determine sequence are so subtle as to evade detection. The mental processes change rapidly, and when one is only half expressed, another has possession of the mind. This incoher-

ence is conspicuous in delirium, and is also frequent in simple mental failure.

Defect of moral sense is also common in mental failure. The slighter defects are relative, rather than absolute, and must be estimated by their deviation from the normal conduct of the individual. Many actions would be more distinctly pathological in a man of refinement than in an ill-mannered man of the lower classes. Other actions are unequivocal. Urine and stools are often passed into the bed in cases of brain disease, in consequence of this mental failure, when there is no loss of power over the sphincters. Under these circumstances it is an indication of a considerable degree of mental change—a greater degree than is suggested, perhaps, by the other indications of the mental state. Of similar significance, in these cases, is a disinclination to swallow. There is no real difficulty in deglutition, but when food is placed in the mouth, the patient lets it lie there, and after some time, perhaps half an hour, spits it out again. Particles may get into the larynx, and suggest that there is a pharyngeal paralysis that does not really exist. It is important to note that, in children, slight mental defect is often shown by a lack of the sense of propriety, rather than by failure of mere intellectual processes. They are unabashed by the presence of strangers, are disobedient, mischievous, meddlesome. Indeed, the lack of capacity for restraint often leads to undue manifestation of what mental power they possess, and parents constantly consider such children unduly precocious, and possessed of mental faculties above the average.

LECTURE X.

SYMPTOMS (*Continued*): AFFECTIONS OF SPEECH.

Gentlemen,—From the mental symptoms that we considered in the last lecture, we pass now to another group, intermediate between the mental symptoms on the one hand, and simple motor and sensory symptoms on the other—affections of speech. We must consider them at some length, because the phenomena are complex, their relations are intricate, and if I were to attempt to be brief, I should succeed only in being unintelligible. As it is, I fear I must ask for your somewhat close attention. There is difficulty, not only in the theoretical study of the subject, but also in the practical application of our knowledge. No two cases of speech-defect are alike; and you can only unravel the phenomena of each case by having a firm grasp of the laws that govern both normal speech and the derangement that is produced by disease.

The brain contains upper and lower mechanisms for expression by articulate speech, the upper in the cortex, the lower in the medulla. The latter transfers to the peripheral nerves the impulses that come down from the cortex, perhaps adjusting their form in minor details. It is in the cortex that the elements of speech are arranged. In disease of the

lower mechanism, the elements of expression are correct in nature, number, and arrangement, but their form is defective; "articulation" is at fault—the jointing of the elements. In disease of the higher cerebral apparatus, the form of the constituent elements may be correct, but they are wrong in nature, in number, or in arrangement. The error in the arrangement of the elements often causes great error in the form of words, although the form of the elements may be correct. You will understand this better as we proceed. We have already considered the effect of disease of the lower mechanism in the medulla, and are now concerned only with the cortical mechanism.

Speech is the expression of mental processes; but it is not the only mode of their expression. They may also be expressed by writing; but writing is merely expression by speech translated into symbols of a different kind. The nervous processes are elaborated in the same cortical mechanism, although they leave the cortex at a different place, and do not pass through the lower mechanism for articulate speech, but pass by it, to still lower mechanisms in the spinal cord.

A different and very important mode of the expression of mental processes is by simple muscular movements in various parts of the body—face, limbs, and even trunk; we call these "gestures." Gesture-symbols are much simpler than speech-symbols. They are the first to be acquired by the race and by the child—the most uniform in different races. Speech-symbols are acquired later, and are diverse in different races.

By these methods we express two classes of mental processes—"ideas" (which are expressed as propositions) and emotions. Emotional processes are by far the simpler, and they are expressed by the more simple and automatic methods, chiefly by gesture. This is the chief use of gesture. It is true, gesture can express propositions, but only in a very limited degree, and only those that are extremely simple in character. An instance is the expression of affirmation by a nod. Gestures that express propositions are called "signs."

In vocal speech there are two elements—articulation and

phonation. Articulation forms the words on the outgoing current of air; the larynx adds voice to speech, and enables it to be heard at a distance. Voice is merely material for articulation. It is indeed a means of expression, by its variations, but it is only an expressional gesture accompanying speech, and, like other gestures, it expresses chiefly emotions. Emotions may also be expressed by words, as by interjections, and by many phrases that have a propositional form, but only an interjectional meaning—"dead propositions," they have been aptly termed by Hughlings-Jackson, to whose philosophical investigation of aphasia every student of the subject is profoundly indebted.* Most oaths are such "dead propositions," and so are many familiar expressions, as when, to express mere surprise, we formally deny, as in the phrase, "You don't say so!" Real propositions, as such, cannot *express* emotion, they can only state the fact of its existence. Although tone chiefly expresses emotion, it has, like other gestures, a limited power of conveying propositions, as when a negation is converted into a question by an interrogative tone.

The expression of emotion is essentially involuntary and automatic. The will is needed, not to effect it, but to restrain it. The expression of propositions is chiefly voluntary, by an effort of the will. But propositions differ much in their speciality. The more special, the less frequently expressed they are,—the greater is the volition required, although we may be scarcely conscious of it. The less special, the more frequently employed they are,—the less voluntary, the more automatic is the utterance. In vocal music, words are chiefly used as the vehicles for tone. The propositions that the words formally convey are scarcely ever really expressed as such.

* Of Dr. Hughlings-Jackson's writings on the subject, the most important are his papers in "Brain" (vol. i., p. 305 ; ii., p. 203, 323). The student who is interested in the subject is strongly advised to read these articles. Those who are acquainted with them will see how largely the views expressed in this lecture have been moulded by those of Dr. Jackson, and how extensively I have adopted the phraseology that he has made, not only current, but indispensable.

Intellectual processes are aroused by language as well as expressed by language—aroused through the senses of hearing and of sight, and in blind persons by touch. This necessarily involves an intimate connection between the nervous processes for these sensations and those for language. Of the links of this connection we can recognize some, because disease occasionally separates them. When a word is heard, the processes thus aroused in the auditory centre excite others that subserve the recognition of the sound as a word, and these, in their turn, excite those that subserve the image corresponding to the word. So too with visual word-symbols. Thus the nervous processes for language have both motor and sensory relations. Of these, the sensory (auditory) processes are developed earlier than the motor processes. The child understands many things said to him, long before he can utter a single word.

We have seen that a sensory word-process must intervene between that for the sound and that for the image of the thing symbolized. So motor word-processes intervene between those for the image and the motor impulses for the muscles. There is a sort of internal revival of words before they are uttered, and this may occur without utterance. A revival of word-processes, motor or sensory, often accompanies deliberate thought.

The motor and sensory word-processes leave behind them residual states, which facilitate the subsequent revival of the same arrangements. These residual states subserve the memory for words. We have seen that memory of other kinds is subserved by similar residual states (p. 117). Thus there may be said to be both a motor and a sensory memory for words. In the revival that precedes speech, we are more conscious of the motor process, of a sort of "internal speech." But it is probable that both motor and sensory word-processes occur together, and that the sensory (auditory) word-process actually leads in the revival. This process, as we have seen, is first established. It may be perfect when the motor memory is lost, and its loss interferes with thought far more than does the loss of the motor memory.

In the act of reading words, the process is analogous to that which takes place in hearing spoken words. Nerve-processes are aroused successively for the simple sensation, the word-symbol, and the image of the thing symbolized. There is an intimate connection between these sensory processes and the motor process. In those unaccustomed to reading, the motor processes are energized in the act, and even the lips may be observed to move, mental images being no doubt aroused more readily by the double than by the single process. In the act of writing, the motor processes for articulate speech are first energized, and these excite the processes for the movement of the hand, and the formation of the written symbols. Even in writing, however, it is probable that the auditory nerve-processes are revived before or with the motor processes. If you attend to the mistakes you make in writing—writing wrong letters, for instance—you may often trace the influence of these auditory and motor associations by the character of the error.

We may now ask what we know as to the parts of the brain concerned in these functions. The chief facts have already come under our notice in our review of the anatomy of the brain. In each hemisphere the lower part of the ascending frontal convolution contains the centres for the movements of the muscles concerned in articulation. From these centres, conducting fibres pass down to the lower mechanism. Hence motor processes for words must leave the cortex at this part. The adjacent posterior part of the third frontal convolution also contains structures that subserve speech, perhaps somewhat higher processes than those of the motor centres in the ascending frontal, and this region is usually regarded as the chief speech-centre. Whether the island of Reil contains similar structures is still uncertain. The first temporo-sphenoidal convolution contains the structures that subserve the auditory perception of words. Those for the visual perception of words are probably contained in or near the angular gyrus. But there is an important difference in the functions of the two hemispheres. Voluntary speech-processes go on chiefly in the left hemisphere in

right-handed persons, in the right hemisphere in left-handed persons. The sensory word-processes, perhaps influenced by the motor, also go on chiefly in the left hemisphere. Disease of the left motor speech-region causes loss of the power of uttering words voluntarily; and that of the first temporal convolution, loss of the power of understanding spoken words—"word-deafness;" whereas disease of the corresponding regions of the right hemisphere produces no such effect. The power of understanding words that are seen is also localized in the left hemisphere. Thus, although the sensory centres for hearing and sight are double, one in each hemisphere, it is only in the left that they subserve the recognition of words.

And yet the left hemisphere has by no means a monopoly of speech-function. The right hemisphere contains structures of similar position and similar connections. These structures can supplement those in the left hemisphere. Loss of speech, due to permanent destruction of the speech-region in the left hemisphere, has been recovered from; and that this recovery was due to the supplemental action of the corresponding region of the right hemisphere, is proved by the fact that, in some of these cases, speech has been again lost when a fresh lesion occurred in this part of the right hemisphere. This supplemental action occurs in the sensory as well as the motor functions. It occurs far more readily in children than in adults. Permanent aphasia in children from disease of the left hemisphere is almost unknown. The loss of speech rarely lasts longer than a week. Then the child speaks almost as well as ever. Hence it is probable that speech-processes go on more equally in the two hemispheres in childhood than they do in adult life. It is also highly probable that there are individual differences in this respect among adults. Certainly, with a lasting lesion, speech is recovered more readily by some than by others. But in all persons the right hemisphere takes some share in speech-processes. Much emotional expression and automatic use of words is effected by it. This is shown by the fact that such emotional and automatic use of words remains, although

the voluntary use of words is lost by disease of the left hemisphere. But since emotional and automatic expression is not lost from disease of the right hemisphere, it follows, as Hughlings-Jackson has insisted, that such expression must be effected by both hemispheres. Hence we may say that expression is one-sided, that is left-sided, in proportion as it is voluntary; is both-sided, that is either-sided, in proportion as it is involuntary and automatic. We have seen that the same law holds good of motion generally. Dr. Jackson believes that the preliminary energizing of motor word-processes that precedes utterance takes place in the right hemisphere. Perhaps, however, it occurs in both.

A curious illustration of the analogous motor impairment was presented by a man who had right hemiplegia and aphasia. In these cases there is usually more loss of simple voluntary movement of the tongue than in similar disease of the right hemisphere, as if the speech pre-eminence of the left hemisphere carried with it a greater representation of the simpler movements of the tongue. I told the man to put out his tongue. He made many attempts, but could not. Then he put out his tongue and licked his lips—an automatic action to facilitate the process,—and tried again to put it out voluntarily, but failed. An analogous speech-defect was that of a girl who, after many vain attempts to utter the word "no," said, "I can't say 'no,' sir."

The extent to which automatic word-processes may be subserved by the right hemisphere is strikingly exemplified by a case that was under my observation many years ago. A man had embolism of the left middle cerebral artery, and, as we afterwards found, the whole of the motor speech-region of the left hemisphere was destroyed. From the attack till his death, a few weeks afterwards, he said only "yes," "no," and once uttered "ning," when the house physician wished him good-morning. But one day another patient in the ward began to sing a song,—"I dreamt that I dwelt in marble halls." The speechless patient joined in, sang the first verse with the other patient, and then sang the second

verse by himself, uttering correctly every word. Of course this was not speech. No one intends to express the propositions contained in the words of the song. The words are used automatically, and this automatic utterance must have been effected by the right hemisphere.

In all cerebral affections of speech there are two elements to be distinguished: some speech is lost, and some speech is preserved, but is deranged. The loss is the effect of the destruction by the lesion; the derangement of the remaining speech is due to the imperfect action of the speech-structures that remain. The word "aphasia" is current as a general designation for all forms and degrees of loss of speech, as "anæmia" is for all forms of defect of blood. Other general terms have been proposed, equally inexact, and less convenient. Indeed, the whole subject has afforded abundant scope for word-makers, who have flooded its literature with a new terminology, to a large extent needless, and to some extent injurious, fostering a harmful tendency to divide where it is desirable only to distinguish. One recent writer alone proposes fifty special designations for varieties of aphasia.

The two most important symptoms of speech-defect correspond to the motor and sensory functions already described. (1) In some cases there is loss of the motor processes for speech. The patient can understand what is said to him, but he cannot speak, or can only use voluntarily one or two words; he cannot even repeat words. (2) In other cases the patient is unable, sometimes absolutely, to understand what is said to him. In this case, he has usually considerable power of speech, but makes mistakes in words and in their form, especially in unfamiliar expressions. The error may be so great that his speech is unintelligible. In the first case the patient is conscious of his errors; in the second he is not, because he does not understand his own utterance. These two leading varieties of aphasia are best styled (with Wernicke) motor and sensory aphasia.

But another defect is very common. There is an inability

to revive voluntarily the word-images, or these are revived wrongly. The patient cannot "recollect" the word, or he recollects it wrongly. Defect of memory being termed "amnesia," this special form is termed "verbal amnesia," or sometimes "amnesic aphasia." We have seen that in the revival of words both the motor and the sensory memories take part, but the sensory (auditory) is the more important, and takes the lead. Doubtless, in educated persons, the visual memory assists; but we may, for the present, leave this out of consideration. Word-deafness involves loss of the auditory word-processes—loss, that is, of the leading mechanism for the subjective revival of words. It always, therefore, involves verbal amnesia. In motor aphasia there is much less verbal amnesia, because the sensory processes are intact; the loss of the motor memory probably causes some impairment of the power of recalling words, some verbal amnesia, although the loss is difficult to ascertain on account of the loss of speech. But verbal amnesia may exist without word-deafness, without any impairment of the power of expressing words. The patient cannot "think of a word"; but if it is told him, he utters it correctly at once. As a clinical variety of speech-defect, this form is often met with, but it is not a separate pathological form, as it has sometimes been regarded. It has been described as the loss of a special memory for words; but there is no function or seat of memory for words, except that of the word-processes, motor and sensory. Moreover, the defect in word-revival always occurs in a certain order, from the special to the general. Its indication is that the speech-processes go on in structures that are relatively inadequate. The loss is never absolute; and it is always a defect in the *voluntary* revival. Automatic utterance is largely in excess of the voluntary utterance. Conclusive evidence that it is not a distinct pathological variety is afforded by the fact that the most perfect example of this amnesic aphasia is presented by cases in which there has been, at first, complete motor aphasia, and, the lesion persisting, the patient has slowly recovered a considerable power of speech, by a re-education of the right hemisphere. Here it is manifestly a residual

state, the result of the relative inadequacy of the structures in which the speech-processes go on.

We may now consider the varieties of aphasia, and the various symptoms in detail, premising that they are often combined, and the resulting symptoms are often extremely complex.

In extensive disease of the motor speech-region, the derangement usually extends beyond the limits of vocal speech. The power of writing is lost, even when there is no paralysis of the hand. This shows, as I have already said, that in writing, the word-processes, although they must leave the cortex at the hand-centre, are first energized in the speech-centre. In severe cases, expression by gesture is at first impaired, so far as the expression of propositions is concerned. The patient nods for "no" as well as for "yes." But all power of expression is never lost. That which remains varies much in different cases, but is always the more automatic—the least special forms of expression. Emotion may be expressed by gesture perfectly, and even by words, interjections, or phrases, such as oaths, that have a propositional form but not a propositional meaning. The oath uttered in anger cannot be repeated without the emotion. Further, most patients speedily regain one or two of the lowest propositional utterances, as "yes" and "no." The word "yes" seems to be a less automatic utterance than the word "no." Children, as a rule, say "no" long before they say "yes." Perhaps these words are first regained because they are often used as interjections; they may be first regained as interjections, and this may facilitate their propositional re-acquirement.* These patients may also sing, as did the man whose case I

* "The words 'yes' and 'no' are propositions, but only when used for assent and dissent; they are used by healthy people interjectionally as well as propositionally" (Hughlings-Jackson). A child under my observation, in learning to speak, first uttered "no," and then expressed assent by the common infantile affirmative "m'm." This he next elaborated into "am," and for many months after he had become able to speak well, he still used "am" to express assent. When he began to say "yes," he employed it for a long time only as an interjection, *e.g.*, when he was called, retaining "am" for assent.

have mentioned. In extensive and persistent disease of the left hemisphere, all the utterances must be effected through the right hemisphere. In rare cases, under strong emotion, a proposition may escape that is much higher than the habitual utterance of the patient, forced out, as it were, by the emotion (Hughlings-Jackson). Slowly, more power of speech returns, by the re-education of the right hemisphere; and, as I have said, more readily in some persons than in others, and most readily in children.

Motor loss, such as this, usually results from disease of the

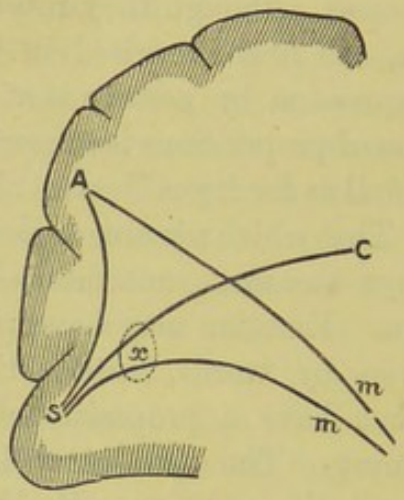

FIG. 18.—DIAGRAM OF PROBABLE COURSE OF FIBRES FROM MOTOR SPEECH-CENTRE.

A, hand-centre in the middle of the ascending frontal convolution; A *m*, fibres from this to internal capsule; S, motor speech-centre; S C, fibres from this to the corpus callosum; and S *m* to the internal capsule. S A, fibres from speech-centre to hand-centre. A lesion at *m m* will cause only transient aphasia, the speech-processes being able to pass by S C to the corpus callosum and opposite speech-region; a small lesion at *x* would cause permanent aphasia, since it involves both the fibres to the corpus callosum and internal capsule, but would not abolish expression by writing, the fibres S A (connecting the speech- and hand-centres) escaping.

cortex; but it may also be caused by disease just below the cortex. If the disease involves the motor path some distance below the cortex, it may cause transient defect of speech; but this is soon recovered from, probably because the left region is able to act through the right by means of the commissural

fibres of the corpus callosum. When the disease is just beneath the cortex these fibres are also damaged, and the aphasia is as lasting as when the cortex itself is destroyed. In the extremely rare cases in which a patient can write and cannot speak, the disease is probably so placed as to interrupt the fibres that go to the motor tract, and those to the corpus callosum, but has not destroyed the speech-centre itself, or the connection between it and the hand-centre (see Fig. 18).

The next important point to be considered is the error that occurs in the speech that still remains, or has been regained. This speech is frequently wrong, both as regards the words employed and their form. The error in form is not in articulation, but in the elements of the word. One patient, for instance, said "int" for "ink," "tinors" for "scissors"; another asked for some "pagne-cham" when she wanted some champagne. The error in form may be so great that the speech is unintelligible jargon; one patient could only say "drumlandee." The resulting disorder is often termed "ataxy of speech." Hence, this form of speech-defect is often termed "ataxic" or "atactic aphasia," an objectionable designation for the pathological variety, because equal "ataxy" may result from the opposite defect, a sensory loss, word-deafness. Moreover, the term is apt to mislead, suggesting that there is a centre or faculty for the co-ordination of speech other than the motor speech-centre.

Without error in form there may be error in use: if "yes" and "no" are the only words that can be uttered they may be used wrongly. When more speech is retained, an unintended word may be used. Errors occur, just as loss occurs, in proportion as speech is special and voluntary. There is always less error in automatic utterance and in signs. A patient who can only say "no," and utters the word on all occasions, may indicate correctly by signs, by nodding or shaking his head, whether he means "yes" or "no." It is often difficult, sometimes impossible, to understand the mechanism of these wrong utterances, but they may be, to some extent, understood by certain general principles. The first is the tendency to repetition. This depends on the fact,

that a nervous arrangement, after functional activity, remains for a time in a condition in which it is more readily energized than other nervous arrangements. This often leads to error in health. If Smith has been talking to Jones of Robinson, he is very likely to call Jones "Mr. Robinson." It is by this tendency to repetition that recurring utterances of what Dr. Jackson calls "low speech value" become established. In the speech-structures that remain, if one arrangement has been energized, it is apt to be re-excited on every attempt, to speak, and the more often the words have been uttered, the greater is their tendency to come out. It is easily conceivable that the right hemisphere may suffice for recurring utterances of low propositional value, such as "no, no." But in some patients the recurring utterance is of high propositional form, although used without meaning. The character of this utterance is often so special as to suggest, irresistibly, that it has its origin in some former utterance of the patient. Dr. Hughlings-Jackson has suggested that it is the expression that the patient was about to utter when taken ill, and that such recurring utterances occur only in the cases in which the onset was deliberate, believing that it is the result of a preliminary speech-process in the right hemisphere. I am inclined to think that it is usually the last words actually uttered by the patient, just before the attack. These cases support the view that in ordinary speech the right hemisphere takes a part in the preliminary energizing of the speech-processes, since such a recurring utterance can only be effected through the right hemisphere. In the absolute arrest of voluntary speech-processes that follows an extensive lesion, causing aphasia, the process last energized in the right hemisphere, if energized not long before the attack, is re-energized when any attempt to speak is made and is uttered, no fresh arrangement being possible to the will for a long time. Many facts support this explanation of these recurring utterances. Thus, the recurring utterance of a signalman who was seized when on duty, was "Come on to me"; that of a girl attacked when riding on a donkey, "Gee, gee" (Hughlings-Jackson); that of a librarian was "List complete"

(Russell). One of my patients was taken ill in a cab; on entering the cab she had told the cabman to drive her to "Mrs, Waters," and these were the last words she spoke. Her recurring utterance was "Missis." The recurring utterance may be an unmeaning combination of syllables. Dr. Jackson has suggested that the disorder is due to the struggle of various word-processes that arise simultaneously, and of which only fragments succeed in achieving expressional predominance. Some of the error in form may also depend on the fact that relics of previous word-processes get mixed up with those that the patient intends to utter, in consequence of the readiness with which the former are re-excited. This is often obvious on an attempt to repeat the alphabet, which one patient uttered thus: *a*—a, *b*—be, *c*—ce, *d*—de, *e*—de, *f*—def, *g*—de, *h*—a, *j*—da, *k*—ka, *l*—kel, *m*—em, *n*—den, *o*—do, *p*—be, and so on. In the same way, the elements of the recurring utterances are apt to crop up when the patient begins to say other words, and so also, perhaps, are parts of words that have been familiar in previous life. Another curious and common form of error is the reversal of the syllables of a word, as in the instance of "pagne-cham," already mentioned. As I have said, words are revived subjectively before they are uttered, and it would seem that the part of the word last energized is the most vivid and most readily uttered. I have heard a very young child say "I got for," instead of "I forgot." The reversal is sometimes partial; an obtrusive consonant in the middle of the word may be uttered also at its commencement: one patient said "Lalice" for "Alice." Often the recurring utterance is a repetition of the same consonant with varying vowel sounds. One patient, whenever he tried to speak, always uttered, "da, de, da, do, de, da," with extreme rapidity, like an engine-wheel flying round on a slippery rail. This is evidently explicable on the principle of repetition. What consonant is the basis of this utterance is doubtless determined by circumstances similar to those that determine the form of the recurring utterance. The woman whose chief utterance was "Missis," when she acquired some additional power of speech, and tried to

utter other words, was apt to run off into "so, so, sa, se, so, si."

The loss of writing may be absolute; the patient can only make unmeaning strokes; or letters may be formed, but combined at random. Lastly, words may be written, but erroneously. The errors may be similar to those that occur in speech; repetition of words and syllables, and the substitution of one letter for another, often of one that is similar in sound, as *p* for *b*, sometimes one that is written in a similar manner, as *g* for *a*. Sometimes the order of the letters of a word are reversed; one patient wrote "tae" for "tea." The mechanism of these errors is doubtless the same as that of the similar errors that occur in speech. The same law of order of loss is traceable. It is common for a patient to be able to sign his own name when he can write nothing else, just as he can tell you his own name when he can say nothing else. A man's own signature is the most automatic of all his writing. One patient, being told to write his own name, wrote "James Slim," correctly. He was then told to write how he came to the hospital, and he wrote "cgng kgig kiyan." The errors in writing are usually greater than in speech, as indeed they are in health. In the double symbolism there is more room for error, and there is more time for error. Thus, when the patient who wrote as above was asked to *say* how he came to the hospital, he said "rail"; asked "how else?" he said "clab"; asked again, "no way."

Patients who cannot write a word can often copy writing or copy print in printing characters, sometimes in writing characters; but they do not understand it, and copy mistakes without correcting them. I remember a sad instance of this. The son of a distinguished poetess would copy page after page of his mother's poems, although he clearly could not understand them. He would also copy, with perfect exactness, page after page of a complicated almanack—always in printing characters.

In the defect in the subjective revival of word-processes that is termed verbal amnesia, the order of loss is also from the special to the general. Proper names are lost first, and

nouns are lost before verbs and adjectives. This loss for nouns has been described as a special variety of aphasia, but it is only a special example of a general and almost universal law. Among nouns, *cæteris paribus*, the more special are lost first, the more general being retained and more easily recalled. Thus, one patient, being shown a shilling, and asked what it was, replied "money," then, after a pause, "coin," and at last, after another pause, "shilling." There is often error in utterance in this form of speech-defect. The patient uses a wrong word; asks for a spoon when he wants a fork. Sometimes, with a little care, we can trace the mechanism of these wrong words. Thus, one patient with some word-deafness, but no motor loss, on being shown a pen-knife and asked what it was, said "Cornwall." Further investigation made it clear that the patient had only used a similar knife for the purpose of cutting his corns: hence the sight of the knife led to the energizing of the nerve-processes for the word "corn." He had once lived in Cornwall, and so the processes for the word "corn" led to the energizing of "wall" in association with "corn," rather than of "knife." In considerable degree, especially when there is loss of the auditory word-processes (word-deafness), there is considerable error in the forms of words, because the patient is thrown upon his motor memory, and this is apt to mislead him when it has not the co-operation of the sensory memory. No doubt this mechanism was at work in the energizing of "Cornwall," just mentioned. These patients have often an extensive power of expression by familiar phrases, if they bring them out quickly and almost automatically; if they hesitate they get wrong. "If I want to say a thing I must say it in a hurry, or I cannot say it at all," one patient said. Errors in writing are more marked in these cases than are the errors in speech, for the reason I have already mentioned, and we can often trace their origin. A patient who made no mistakes in speech wrote "disacreeable" for disagreeable, "glag" for glad. The former was evidently due to an error in the motor memory, substituting for one letter another for which the motor process is nearly

the same; the second was due to the principle of repetition. The nervous arrangements are not under perfect control, and some unintended arrangement is energized, in which, for one of the reasons already mentioned, the resistance is lower. I may quote an instance of the greater readiness of familiar automatic utterance than that which is less familiar, and therefore requires a greater voluntary effort. I asked the patient who had made the remark just quoted about saying things in a hurry, to write down the name of a boat-race that had taken place the day before. He said, "Boat-race? Oxford and—Oxford and Cate—Ama—Abramidge—Oxford and Baxford—I ought to know, I have been there often—what is it?—Oxford and—now I cannot tell you—Oxford and Batham." He then took the pen and wrote "Oxford," and said, "Now I cannot say that other," and wrote "bab a tha," and then "C a t b h," and exclaimed "I am a bigger fool than ever. Oxford and Ca—Cab—Caba—Cambridge—there, I have it at last." But after repeating it correctly several times, he wrote "Cabrage."

In partial word-deafness, the same order of loss is observed. The patient may understand familiar words, and not those that are unfamiliar. He may understand signs when he does not understand speech. If told to put out his tongue, he may make no attempt to do so; but if shown, he may put it out at once. Putting out the tongue is not, however, a good test. When a patient comes to a doctor, he expects to be told to put out his tongue, and may guess that this has been said to him. He may put out his tongue to order, but if told afterwards to shut his eyes, he may simply again put out his tongue. It is, perhaps, hardly accurate to speak of this as guessing. Expectation facilitates comprehension. This statement is a platitude: but the physiological aspect of the platitude is that the physical side of "expectation" is a lowering of resistance in certain nerve-processes. Hence it is better to ask the patient to do something unexpected, or, as Dr. Hughlings-Jackson suggests, to ask him some absurd question, the comprehension of which is readily shown by the obvious amusement it excites. The same condition is

observed in those who are recovering from word-deafness. Slow recovery occurs from word-deafness, as from other forms of aphasia, the lesion persisting. Doubtless this also is by the re-education of the right hemisphere, perhaps aided by the visual word-centre. A very intelligent man, who had slowly recovered from complete word-deafness, told me that he still often had a difficulty in understanding a word spoken to him; but he would repeat it a few times, and at last he seemed to see the letters of the word, and then its meaning flashed upon him.

Patients with motor aphasia usually cannot read. We cannot infer from this that there is word-blindness, in the strict sense of the word. The co-operation of the motor speech-centre seems necessary for understanding the visual word-symbols. The best way of testing the power of reading is to write down a direction to perform some simple action. Patients often try to read, and even seem to think that they understand, when they certainly do not. This loss usually lessens in time, other word-processes that remain aiding the visual centre. But in true word-blindness the patient is absolutely unable to understand the words that he sees, and the loss is the more striking in that the motor processes for speech are often preserved.

I may briefly recapitulate the chief points to be attended to in examining a case of aphasia, and the indications they afford. Remember that some weeks must elapse, from the time of the onset, before we can infer, from the character of the symptoms, the position of the lesion. The connection between the different centres concerned in speech is such that an acute lesion in one of them causes a temporary derangement of the others. Only when this has passed away, in the course of two or three weeks, can we infer the position of the lesion. The action of the speech-centres may also be interfered with, for a time, by disease that is near them but does not actually involve them or the fibres from them.

It is well to begin by ascertaining whether the patient can

understand what is said to him—whether there is any word-deafness. Try him first with simple orders, and then with sentences that are more complex. If he cannot understand even simple things, there must be disease of the first temporal convolution, or beneath it. If there is mere impairment, and not loss of auditory perception, it may be the result of partial damage to the same parts, or may be the result of old disease, from which the patient is recovering by the action of the right hemisphere.

Next observe the character of the motor loss, the degree of speech that remains, and whether the patient can repeat what is said to him. If speech is limited to a few automatic utterances, and the patient, while understanding, cannot repeat words, there is disease of the motor speech-region—third frontal and lowest part of the ascending frontal convolutions. The disease may not be in, it may be just beneath, the cortex. If utterance is only unintelligible jargon, or if there is a recurring utterance, the indication is the same. If, however, there is word-deafness, great formal error in speech does not prove disease of the motor speech-region. If the motor loss soon passes away—in the course, for instance, of two or three weeks—although hemiplegia persists, the disease is probably some distance below the cortex, in or near the internal capsule. If recovery takes place in the course of two or three months, hemiplegia persisting, the lesion is probably in the white substance near the cortex, and has damaged, but not destroyed, the fibres to the corpus callosum and opposite speech-region. This is the more probable if the patient can write better than he can speak. It is often difficult to ascertain the patient's power of writing, because the right hand is frequently paralysed, and the left hand is awkward, while to form words with moveable letters is a much more severe test than writing.

Inability to read can only be regarded as evidence of disease of the visual word-centre when there is no motor aphasia. The power of voluntarily recalling words is best tested by asking the patient to name objects that are shown to him. Defect of this may be due to past or present word-

deafness, or it may be a residual condition left by a past motor aphasia. In neither of these conditions has the symptom any diagnostic value. It only indicates the relative inadequacy for voluntary action of the structures in which the speech-processes go on—in most instances those of the right hemisphere acting with those of the left that remain. If the condition is a primary one, its significance is uncertain. It is probably due to a small lesion near the sensory speech-regions of the left hemisphere, lowering their function generally, sometimes to one in the parietal lobe. There is no evidence to support the statement that is often made, that the symptom is due to, and indicates, an interruption of the connection between the sensory and motor speech-centres. Indeed, it is not easy to conceive how such an interruption could produce the effect ascribed to it.

Motor aphasia is often combined with hemiplegia from disease of the motor centres adjacent to the motor speech-region, and especially with paralysis of the face, the centre for which is the nearest. On the other hand, in sensory aphasia, hemiplegia is often absent, and, if it occurs, is usually slight.

In conclusion, one aspect of the question of speech-defect is of considerable practical importance—its relation to the capacity for making a will. In pure motor aphasia, in which the auditory word-processes are intact, words that are heard are perfectly understood, and assent or dissent can be expressed, although only by gesture, the patient could certainly make a will. If there is considerable word-deafness, it is always so doubtful whether the meaning of what is said to him is correctly perceived, that there is probably no "testamentary capacity" unless written words are perfectly understood, and all communications are thus made. *A fortiori*, if there is both word-deafness and word-blindness, a valid will could not be made.

LECTURE XI.

SYMPTOMS (*Continued*): HEADACHE—VERTIGO—VOMITING.

Gentlemen,—We will consider next three symptoms that stand very much on the same level of diagnostic significance,—symptoms that are often combined, although they are very different in form and in nature—headache, giddiness, vomiting. The first of these is, of all the many symptoms that organic disease may produce, the most obtrusive, the most distressing; and at the same time the most equivocal, the most apt to mislead both the patient and the physician. Local pain is a symptom of universal incidence; wherever it occurs, it absorbs the attention of the sufferer, and to him at least, suggests irresistibly a local cause. But of all seats of pain, the head is the most common, and pain is here the most suggestive of local disease, and yet it is the place in which pain is most frequent without local disease. In consequence of some mysterious relation, pain in the head is the common result of most varied causes—of stomach disturbance, of anæmia and plethora, of every kind of morbid change in the blood, febrile conditions, acute specific diseases, kidney disease, toxæmia. The nerves that supply the walls of the skull are prone to neuralgic pain of every kind. Headache occurs also in various functional disorders of the nervous system.

It readily results from mechanical congestion of the brain, however produced, and is sometimes so obtrusive that the patient notices the cause less than the effect. Thus I have known a patient to come for treatment on account of headache, and to complain of nothing else, when the headache was the result of cough, and the cough of phthisis. I think it is no exaggeration to say that for one case of headache due to organic disease of the brain, you will meet with fifty that are due to other causes.

There is not much in the character of the pain to indicate, or even to suggest, its origin. Pain that follows closely the course of a cranial nerve is probably neuralgic in nature; but it may be due to irritation of the fibres of the nerve: so that, while it does not suggest, it does not exclude, intracranial disease. Pain that is very limited in area is more often of functional than of organic origin. Thus, pain limited to one supra-orbital region, or to a spot at one temple, on which the patient can put his finger, or to a spot at the vertex, is commonly of functional origin; the last is common in hysteria, and is termed, from the frequency with which it is likened to a nail being driven in, the "clavus hystericus."

Of much greater significance, though still not pathognomonic, are the course and severity of the pain, taken together. A common feature of the pain of organic disease is its constancy. The pain varies in severity from time to time, but there are rarely periods of perfect freedom. It is usually a severe pain; often at times most intense, causing the patient to shriek from the suffering. In mere severity, however, the pain of organic disease is rivalled by some functional headaches, as by that of migraine; but these are paroxysmal—attacks of intense pain are separated by days or weeks of freedom, comparative or perfect. The pain of organic disease persists during the night, and often prevents sleep, or rouses the sufferer. Functional headaches rarely prevent sleep, which, indeed, often ends the pain. Whenever, therefore, a patient complains of severe pain in the head, ask him if it keeps him awake at night; and if it does, you should think of organic disease as possible, and search most care-

fully for any other symptoms. But severity and constancy of pain, even together, only *suggest* an organic cause; they do not prove such cause. Proof of this—or even the high degree of probability that, in practical medicine, we have often to be content with—is only supplied by the association of headache with other symptoms, diffuse or focal. Of the diffuse symptoms, optic neuritis and vomiting are the most significant. An ophthalmoscopic examination should never be neglected in any case of severe headache. If you can exclude three constitutional states—considerable anæmia, kidney disease, and lead-poisoning,—the coincidence of considerable neuritis and headache may be regarded as proof of organic intracranial disease. To this, and to the significance of vomiting, we shall presently return.

Always remember, gentlemen, that you can never assume the converse of diagnostic rules. The presence of a symptom may be strong positive evidence, while its absence has very little significance, and perhaps none at all. This is true in every department of diagnosis. There is hardly a disease, usually attended by some prominent and characteristic symptom, that does not occasionally occur without that symptom. Acute peritonitis, of which intense agony is a characteristic, may run its course without any pain. Neither the absence of pain in the head, nor its trifling character, enables you to exclude either intracranial disease in general, or any special form of such disease in particular. Cancer is usually a most painful malady, wherever it occurs; all tumours of the brain usually cause intense suffering: and yet cancer of the brain is occasionally almost painless. I do not say that the absence of symptoms has no negative value; it justifies you in attaching less weight to equivocal symptoms that are present, but it does not lessen the significance of any unequivocal symptoms.

We know little of the way in which pain in the head is produced. The brain of an animal can be cut or torn without the creature showing signs of suffering. But the significance of this fact may be over-rated, since other structures, that seem normally insensitive, become acutely painful

when diseased. It is certain, however, that the membranes are very sensitive, and highly probable that much of the pain of intracranial disease is produced in them. Some diseasés, as tumour, have been thought to cause pain by the pressure they produce, but the evidence that this is commonly the effective mechanism is insufficient, since other diseases may increase the intracranial pressure, locally and generally, without causing pain. Nor can we reasonably invoke the ready explanation of "congestion" as a universal mechanism; in many conditions of headache this is out of the question. In short, until we know more than we do at present, speculations as to the way in which headache is produced are scarcely profitable.

The question may perhaps have occurred to you—Is there any relation between the seat of pain and the position of the lesion? All that can be said is, that there is sometimes no correspondence, sometimes there is a general correspondence, and occasionally there is a close correspondence. Pain at one part of the skull is rarely caused by disease at an opposite part of the brain; one-sided pain is usually due to disease on that side; disease beneath the tentorium generally causes pain at the occiput, which may pass down the neck; and disease at the surface of the brain often causes pain corresponding closely to the seat of the lesion. Light percussion sometimes elicits tenderness over the seat of the disease—pain being produced by a tap there, and not elsewhere. This is most frequently the case when the disease is superficial. The relation of pain to the nature of the lesion we shall consider in a subsequent lecture.

Unpleasant cephalic sensations, other than actual pain, are very rarely caused by organic disease. When they occur without pain, they are almost always of functional origin, and suggest the absence of organic disease. The sensations are, however, frequent elements in the diagnostic problem, because they distress the patient, are apt to be brought on by brain-work, and, if allowed to do so, may practically incapacitate their subject. They generally occur in hypo-

chondriacal persons, who attend much to their sensations, and to whom these feelings are fertile in suggestions of evil. Indeed, hypochondriasis often results from, when it does not cause, these sensations, and it is always increased by them. They are common in those whose brains are overworked—in clerks, in lawyers, in commercial men,—and are predisposed to by neurotic inheritance, and by the condition of defective tone of the nervous system that it is fashionable now to dignify with the name of "neurasthenia," at once more alarming, and more gratifying, to the weakened mind, than the older equivalent, "nervous weakness." These sensations are various in character, but feelings of fulness in the head, heat, "rushing" sensations, and pressure on the vertex are the most common. The vertical pressure is especially frequent and troublesome. These feelings apparently depend on an abnormal consciousness of processes that should not rise into the sphere of consciousness. An infinite number of impressions must be continually reaching the brain from various parts of the body, engendered by processes of function and nutrition. Of most of these we are normally unaware, but of some we may become conscious by attention. Fix your attention on any part of the body, and in a few moments you will be conscious of some feeling there of which you were previously unaware. Let it be the vertex: in a few moments many of you will be conscious of a distinct sense of pressure there, most readily if you are tired—if you have been listening, for instance, to two or three lectures—or if you are somewhat out of health. You can understand how such a sensation, by continued attention—aided by that potent intensifier, concern—may be cultivated; the sphere of consciousness, as it were, being extended in that direction, until the sensation becomes a real trouble, and the sufferer is convinced that there must be an organic cause for that which is so distressing. This conviction, I regret to say, is sometimes strengthened by members of our profession who find it profitable to pander to these morbid tendencies by a diagnosis of some actual disease in harmony with the patient's fancy—a diagnosis for which there is not the slightest justification. "Congestion

of the base of the brain" is one of the most common of these diagnoses. There is a current manual of diseases of the nervous system, in which the symptomatology of congestion of the brain has been largely written from the history of these sufferers from cephalic sensations. Endeavour to convince the patient that there is no actual mischief in brain or circulation; encourage him to disregard the sensations, and they will slowly cease to trouble him.

There is only one condition in which these sensations, other than pain, are suggestive of real disease, and that is when they are distinctly paroxysmal and the paroxysms are accompanied by vomiting. Not long ago I saw a man who complained of occasional sensations of a "rushing" feeling passing from the occiput to the vertex. These gradually increased in intensity, until the sensation became actually painful, and with this he would sometimes vomit. There were no symptoms of organic disease, with one important exception—there was slight optic neuritis. This gave additional significance to the vomiting, and made some form of tumour highly probable. A few weeks afterwards he complained suddenly of acute pain in the head: while describing it to his doctor, he lost speech; he became unconscious, and died in a few hours. Most likely he had an intracranial aneurism, the rupture of which caused his death.

We pass next to a symptom that has excited much interest, on account of its peculiar character, its significant associations, and its relation to the disturbances that can be produced experimentally in animals. "Vertigo," I need not remind you, means "a turning." The word is strictly applied only to those sensations in which the patient feels as if he were turning round, or at least moving, or has the related objective sensation that other things are moving before him in a definite direction. But it is often, and its English equivalent "giddiness" is still more frequently, applied to all sorts of sensations that involve a sense of uncertain equilibrium, and even to vague feelings of mental

confusion, or to dimness of sight—sensations that are also termed "dizziness." Some mental confusion is no doubt always involved in the derangement of the perception of the exact relation of an individual to his environment that constitutes true "vertigo." It is not surprising, therefore, that the same term should be applied more widely to a similar mental state, irrespective of the precise mechanism by which it is produced, especially since our vocabulary is inadequate to designate, or our language to describe, a hundredth part of the varied sensations to which our frame is subject.

We are now concerned only with the sensations that do involve so distinct a sense of disturbed equilibrium or actual movement as to come under the general designation. In definite vertigo we may distinguish three elements, which are unequally prominent in different cases. The most pronounced, and most constant, is a sense of movement in the patient himself—to one side, backwards or forwards, up or down, or a sense of oscillation. Next in frequency is an apparent movement of objects at which he is looking: these may seem to move in the same direction as that in which the patient seems to move, or in the opposite direction. The third is less common than the others—an actual movement in the patient; it is usually a fall, or a visible tendency to fall: the fall is commonly in the same direction as the subjective sense of movement, but sometimes it is in the opposite direction. It is of great importance to ascertain, as exactly as possible, the precise character of each of these elements. Although we cannot, as yet, use all these facts in practical diagnosis, they enable us to understand something of the nature of the symptom, and will probably, in the future, teach us much of its mode of production.

The first significant fact is that the patient usually falls, if he does fall, in the direction in which he feels a tendency to fall. At first sight this may seem natural, even necessary; but the fact makes it probable that the sensation of vertigo is really (as Dr. Hughlings-Jackson has suggested) a motor sensation, the effect on the sensorium of motor processes, which, in greater intensity, cause an actual movement.

I told you that objects sometimes seem to move in the same direction as the patient, sometimes in the opposite direction. This difference is not very easy to explain. It has been thought that stationary objects should appear to move in the opposite direction to the subjective sense of movement, because, if the eyes move, say to one side, objects will pass across the field of vision towards the other side. Thus, if you move your eyes to the right, an object that was before in the middle of the field will pass to its left side. But our sense of the movement of visible objects does not depend only on their position, or change of position, in the field of vision; it depends on this, combined with the consciousness of our own condition of rest or movement. If, for instance, we remain still, and an object remains in the same part of the field, we conclude that it is stationary, by a process of unconscious inference, if the term may be allowed. But if an object remains in the same part of the field while our eyes are being moved to the right, we know that it is moving to the right. It is the same if the head or body is turned instead of the eyes. Hence, if we have, in vertigo, a sense of movement without actual movement, an object that remains in one part of the field seems to move in the direction of the sense of movement. When an actual movement occurs, this is probably always far slighter than the sense of movement; and the reverse movement of objects in the field, due to the actual movement of the eyes, may be insufficient to compensate for the greater sense of movement; so that they still appear to move in the same direction, although, if the patient could analyse his feelings in what is usually a moment of supreme confusion, the objects would probably seem to move less rapidly than himself. We cannot at present give an explanation of the apparent movement of objects in the opposite direction to the sense of movement of the patient. It is probable, however, that the motor and sensory relations, disturbance of which causes vertigo, are extremely complex and numerous, extending through almost all grades of motor and sensory cerebral processes, from low to high, and may be deranged at various functional levels. It may be that in certain

derangements the sense of movement, even if it is attended by actual movement, does not involve visual impressions in the resulting error of judgment. Again, the tendency to movement may cause a compensatory and opposite action of the centres to preserve equilibrium, and this may, in some cases, exceed its cause. But I will not take you farther into the obscurity in which this part of the subject is involved.

The centrifugal impulses that maintain equilibrium are regulated by certain centripetal impulses that give guiding information regarding the relation of the body to its environment. One class of these consists of the various sensory impressions from the parts on which the body is resting, and of the impressions from the muscles that maintain the body in its posture (or the sense of the innervation bestowed on these muscles). A second is derived from the innervation of the ocular muscles, and from those of the head and neck. From this, as we have before seen (p. 85), we derive, unconsciously, our perception of the relation of our body to seen objects. A third consists of the impressions derived from the semicircular canals—impressions that vary according to the pressure of the endolymph in the ampullæ of the canals, under the influence of gravitation and movement. If any one of these impressions is suddenly deranged, vertigo results. The most effective is the derangement of that derived from the ocular and aural sources of information; and the resulting derangement constitutes, respectively, ocular and aural (or labyrinthine) vertigo. The same symptom may be also produced by a very different mechanism, disturbance of the stomach.

It would be foreign to my present task to describe to you in detail all these varieties of vertigo. I mention them to you, because the symptom results from these causes far more frequently than it does from central disease, and you cannot ascribe it to the latter until you have excluded a peripheral origin. Evidence of such origin is afforded by other symptoms of derangement of the organs concerned in its production, and for these you must search. In ocular vertigo there is diplopia; in aural vertigo there is commonly the "nervous

deafness" of which I described the symptoms in a previous lecture (p. 95), and there is often also a noise in the ear. In gastric vertigo there are other symptoms of stomach disturbance. Giddiness occurs also, but usually in vague form, in anæmia, and other states of nervous weakness. It is sometimes met with when no cause for it can be discovered—a form which, to conceal our ignorance of its nature, has been termed "essential vertigo." Definite vertigo, in which the sense of movement is well marked, is far more frequently due to disease of the semicircular canals than to any other cause; and when it does result from any other cause, as, for instance, from stomach disturbance, you will often find some deafness or other indication of an affection of the ear,—as if the impairment of the auditory nerve had given a more definite character to vertigo that was *excited* by another mechanism. A gentleman, a member of our own profession, has suffered all his life from prostrating attacks of dyspepsia. Some years ago, he became deaf from labyrinthine disease; ever since, his attacks of dyspepsia have been accompanied by severe vertigo, from which he never suffered until he became deaf. The paroxysms of auditory vertigo may be very severe, and are often ascribed to some brain lesion. For this reason they may excite considerable, but groundless, alarm.

Another form of vertigo that may give rise to diagnostic difficulty is that of epilepsy. Vertigo is a frequent warning of the minor seizures, and sometimes of the severe attacks. The almost invariable concurrence of loss of consciousness sufficiently distinguishes this from the vertigo of peripheral origin. Remember, however, that this warning is not confined to idiopathic epilepsy. The convulsive seizures that result from organic brain-disease are often heralded by vertigo. I have already described the diagnosis between the two.

Severe vertigo, whatever its origin, is usually attended by vomiting. There must be a close connection between the pneumogastric centre and the nerves or centres that regulate equilibrium. The auditory and vagus nuclei are not far apart in the medulla, but we do not know whether the connec-

tion between the two is established there or in the cerebellum. The latter is not improbable. A connection certainly exists, and enables us to understand that stomach-disturbance may produce, and intensify, vertigo, as well as that vertigo may cause vomiting. Sea-sickness is not improbably produced by the oscillations of the endolymph in the semicircular canals.

Diseases of various parts of the brain, and lesions of various kinds, may produce vertigo, and it has therefore in itself little diagnostic significance. It is produced by chronic irritating disease, but rarely by stationary lesions. It occurs at the onset of acute lesions. The most intense vertigo, however, is caused by lesions of the middle peduncle of the cerebellum, and it may then be accompanied by its true motor form, a forced rotation.

Vomiting is occasionally a most important symptom of cerebral disease, but it is a symptom that derives its significance chiefly from its associations. It has, indeed, certain characteristics, but these, which are chiefly negative, are common to the vomiting of functional nervous disorder and of organic disease. The act of vomiting is reflex, effected through a centre in the medulla, probably part of the vagus centre. It may be produced by an undue impression on the nerve-endings in the stomach—an impression that is felt as pain, discomfort, or nausea. Such is the vomiting of gastric origin, as that of ulcer of the stomach, or of simple indigestion. But vomiting may also occur from exalted irritability of the centre; and, in this condition, stimulation of the nerves of the stomach by the mere presence of food may be sufficient to excite the irritable centre. Such is the vomiting of hysteria, in which food is rejected as soon as it enters the stomach; and such is the vomiting of cerebral disease. The increased irritability of the centre may be so great that vomiting may occur without any peripheral irritation, when the stomach is empty.

Nausea may or may not attend this increased reflex excitability. In hysterical vomiting it is generally absent, and it is often absent in the vomiting of organic cerebral disease. We

know very little about nausea. It is an effect on the sensorium of the nervous irritation that excites vomiting, but is, as it were, a collateral disturbance, and is not due simply to the action of the reflex centre, since vomiting may occur without it. It is referred as much to the fauces as to the stomach, and is prominent when vomiting is excited by tickling the fauces, and when the excitability of the centre is augmented by some other peripheral irritation, as that of the uterus. Hence, while its absence suggests, its presence is compatible with, a central cause.

Organic disease in any part of the brain will cause vomiting, and it is therefore probable that the higher central relations of the pneumogastric nerve are very extensive—a fact that is also suggested by the varied functional disturbance of the cerebral centres that may result from gastric disturbance, and by the singular readiness with which vomiting may be excited by olfactory impressions and by some psychical influences. You may perhaps know Weir Mitchell's story of the man who was so much disgusted by his wife's vomiting during her first pregnancy, that he vomited too; and ever after, when his wife became pregnant, he became sick. Although vomiting may be produced by disease in any situation, it is caused with especial readiness by disease of the pons and medulla, and most readily of all by disease of the cerebellum. It has been thought that this is due simply to pressure on the subjacent medulla, but it is probable that a functional mechanism is concerned in the effect. Of all the pathological relations of vomiting, the most remarkable is its relation to vertigo. Of this relation I have already spoken, and will only further remark that this association, coupled with the curious disturbance of equilibrium that results from disease of the cerebellum, suggests that the vomiting of cerebellar disease is due to an intimate relation between the gastric centre and this part of the brain.

Cerebral vomiting results chiefly from irritating disease. It is rarely due to a stationary lesion. Meningitis, tumour, abscess cause it; acute lesions only at their onset, or during the period of secondary irritation. There are two

facts regarding cerebral vomiting that I would impress upon you. The diseases that cause vomiting usually cause also pain in the head, and the vomiting often attends paroxysms of pain, and adds much to their significance. They give a similar significance to pain that is slight, and would not, in itself, suggest organic disease. The second fact is that brain disease, which exalts the excitability of the gastric centre, does so gradually, and the vomiting may be at first excited by a peripheral cause—a cause that might not be effective were it not for the exalted excitability of the centre. In such cases the exciting cause may be accepted too readily as an adequate explanation. This caution is especially necessary in the case of children. I have more than once known the vomiting of meningitis or of cerebral tumour to be, at first, excited by some injudicious food, and to be therefore thought to be of no importance, until graver symptoms gave significance to it. Remember that indigestion, sufficient, by itself, to make children vomit, usually makes them ill; and if a child vomits food without seeming otherwise indisposed, do not at once assume that there is nothing more the matter with it than indigestion, but watch it carefully.

LECTURE XII.

SYMPTOMS (*Continued*): TEMPERATURE—PULSE—DISTURBANCE OF RESPIRATORY, DIGESTIVE, AND URINARY ORGANS—SYMPTOMS OBSERVED WITH THE OPHTHALMOSCOPE.

GENTLEMEN,—We have to consider to-day a miscellaneous group of symptoms that are sometimes described as being "outside the nervous system," apparently because they are within the range of other organic systems. They are no more "outside the nervous system" than is paralysis of the arm. We will begin with the changes in the heat of the body that are produced by brain disease.

Elevation of temperature accompanies many diseases of the brain. The relation between the two varies in different cases. In some the pyrexia is simply associated, the result of the cause of the cerebral disease. In others it is the result of the pathological process in the brain. In others, again, it is the result of the lesion, and not of the process that causes the lesion. In each case it is necessary to recognize the precise relation, in order to estimate correctly the significance of the pyrexia. Instances of the first form are presented by the fever that accompanies embolism in a case of ulcerative endocarditis, or a secondary abscess, due to septicæmia. In these cases the pyrexia is chiefly due to the general disease, and is simply associated with the cerebral lesion. The second relation obtains in most cases of intracranial inflammation. The morbid process raises the temperature, as it would if it occupied any

other situation. The pyrexia of meningitis, and the elevation of temperature that occurs a few days after an acute lesion, are instances of this form. These two causes of pyrexia often concur; a general disease causing fever produces a cerebral lesion that has a similar effect. The fever that results from an intracranial cause varies in its degree and course much more than does that which results from a similar process outside the nervous system. Two facts are especially important. Pyrexia may be absent with an inflammation that elsewhere would certainly raise the temperature. Apparently this is the effect of a restraining influence exerted by the irritated nervous system. This influence may even go so far as to lower the temperature under the normal, in spite of the process of inflammation. The second fact is that the rise in the pulse-rate which usually attends fever may, in like manner, be restrained, and this, even though the rise in the temperature is not prevented. The pulse may even be lowered to an abnormal infrequency. Thus a normal temperature does not exclude inflammation; and elevation of temperature, if it occurs, derives additional significance from an infrequent pulse.

The third relation between brain disease and temperature is very remarkable. Lesions in the pons or medulla may cause a very rapid elevation of temperature, which, in the course of an hour, may attain a height of 106°, 108°, or 110°. This hyperpyrexia is usually associated with copious perspiration. It occurs not only in various acute lesions, but sometimes in the course of chronic lesions. It may also result, although very rarely, from more extensive disease, such as meningitis, probably from an influence exerted on the lower parts of the brain. This hyperpyrexia is apparently due to the loss of action of some influence that normally restrains the heat-producing mechanisms of the body. As you may know, a similar effect follows experimental lesions in this part of the brain in animals. It is always of very grave significance, usually being followed by death within a few hours.

There are one or two miscellaneous facts regarding the

temperature in diseases of the brain and nervous system with which you should be acquainted, in order to avoid errors into which you might otherwise fall. When convulsions succeed one another with great frequency—the "status epilepticus" —the temperature often rises steadily, and may attain an alarming height—106° or 108°. The rise is the result of the convulsions, and does not indicate any inflammatory condition of the brain. If the patient does not die, the temperature slowly subsides as the convulsions become less frequent. Some degenerative diseases, as general paralysis of the insane, are occasionally attended with slight chronic pyrexia, which appears to have its origin in the nervous system, and does not necessarily indicate any inflammatory condition. In the same class of diseases, paroxysmal disturbances occur, characterized by great heat of the skin, often sweating, sometimes loss of consciousness, sometimes convulsions. The patient may seem for a few hours in a most grave condition, as if some acute inflammation had suddenly developed; but in a few hours more all these symptoms have passed away. These are called "congestive attacks"; we do not know their real nature, or whether the indications of congestion are primary or secondary. Thirdly, children are sometimes liable to attacks, probably of the nature of migraine, in which there are severe headache, fever, and sometimes vomiting. The condition of the child resembles that of one suffering from meningitis, but the symptoms develope more rapidly than those of meningitis, reaching their height in a few hours from the onset. They pass away with equal rapidity. Such attacks may recur at intervals of a few weeks or months. The rapidity of the onset, and the history of previous attacks of the same character, distinguish them from more serious affections. Their recognition is important, otherwise you may make a diagnosis of fatal disease, and the patient may be well at your next visit.

Depression of temperature occurs at the onset of some acute lesions, especially hæmorrhage, and in some chronic diseases in which there is profound lethargy, and all the bodily functions are lowered, in response to the lowered function of the

brain. In meningitis, the influence that prevents an elevation of the temperature may, as I have already mentioned, go so far as to reduce it below the normal, in spite of the inflammation.

Many diseases of the brain have no special influence on the pulse. The most important fact is that which I have already mentioned, that the frequency of the heart's action may not be increased, and may even be lessened, by morbid processes that elsewhere would increase the pulse-rate. In inflammation, variations in frequency occur without any recognizable condition to which they can be attributed, and irregularity in rhythm is occasionally a marked and early symptom. Infrequency is not uncommon in chronic disease, and also at the onset of sudden lesions. When the latter are in the neighbourhood of the medulla, there may be great irregularity in both frequency and force.

Vaso-motor disturbance occurs as part of hemiplegic palsy, and this form has been already described. In meningitis there is often a strong tendency to trophic disturbance in the skin—bed-sores and the like—similar to that which occurs in hemiplegia, and occasionally this symptom has a diagnostic value. I have known, for instance, the readiness with which blisters were caused by a hot-water bottle to first arouse a suspicion of the grave nature of an illness that had been regarded as hysteria. Dilatation of the cutaneous vessels, excited by an irritation of the skin (such as a stroke with the finger-nail), lasts long in many cases of acute brain disease, and much significance was formerly attached to the "tache cérébrale," as the red line thus produced was called. But it occurs also in many general diseases, and is as likely to mislead as to aid the diagnosis.

Respiration may be either quickened or slowed by cerebral disease; but the change is considerable only when the functions of the brain are impaired by extensive disease, such as a large hæmorrhage, or when the lesion is in

the neighbourhood of the respiratory centre, *i.e.*, is in the pons or the medulla. Under these circumstances the respirations are sometimes very irregular, and there is occasionally a peculiar respiratory convulsion, a prolonged convulsive cough, or a hiccough. The rhythmical variations that constitute the "Cheyne-Stokes" breathing (see p. 110) occur towards the end of many cerebral diseases, such as cerebral hæmorrhage or meningitis. In some rare cases of disease of the pons or medulla, in which consciousness has been preserved, there has been intense subjective dyspnœa.

In coma, the respirations are often quickened, not as a direct result of the cerebral lesion, but in consequence of the accumulation of mucus in the air-passages. This is due chiefly to respiratory inertia, but in some cases neurotic congestion may increase the secretion, since lesions near the pneumogastric centre may cause actual hæmorrhage into the substance of the lung.

Of the derangements of the digestive organs, the most important, vomiting, has been already described. Constipation is common in acute and subacute diseases, and sometimes adds weight to the significance of other symptoms.

Alterations in the composition of the urine may be a direct result of the cerebral lesion, but are rare, and of little diagnostic value. Excessive secretion, and the presence of albumen and sugar, have been observed as the result of disease, acute and chronic, in various parts of the brain, most frequently when the lesion was in the pons. The derangement has usually been transient; occasionally, glycosuria has continued for many months. I need scarcely mention that organic renal disease, and previous diabetes, must be carefully excluded before the change in the urine can be ascribed to the influence of the cerebral lesion.

The sphincters are affected as frequently in disease of the brain as in that of the spinal cord, but rarely in the same

degree. Both retention of urine and incontinence are met with, but the incontinence is the result of overflow, and not of complete inaction of the sphincter. It is very important to remember how often there is an apparent incontinence that is merely the result of mental inertia. I have mentioned this before, in speaking of the mental changes; and I told you how significant it is, as an indication of a profound degree of mental change—more profound than the other symptoms might lead you to imagine.

I have left to the last the changes in the fundus oculi, not because they are last in importance, but because they stand to the cerebral lesion in a relation somewhat different from that of the symptoms that have hitherto engaged our attention.

The indications of brain disease that are revealed by the ophthalmoscope are not only of the highest practical importance, but are of interest as a salient indication of the progress of medical science, since thirty years ago they were unknown. Morbid appearances in the eye are frequent in intracranial disease, and often prove the existence of such disease when other symptoms are inconclusive, or afford evidence of its nature that can be obtained from no other source. For this and other reasons, skill in the use of the ophthalmoscope, and familiarity with the conditions it reveals, are indispensable to the physician. I cannot too strongly impress upon you the importance of acquiring dexterity in the use of the instrument as early as possible, in order that you may be able to utilize the opportunities for gaining experience which almost every department of practical work will supply. These opportunities will be lost if you only learn to use the instrument when you proceed, towards the close of your course, to the special study of diseases of the eye.

Some of the changes in the eye met with in brain disease are merely associated, the result of, and evidence of the cause of, the cerebral lesion. Others are consecutive, the

effect of the brain disease, and evidence of its presence and of its nature. The associated and consecutive symptoms are sometimes conjoined.

The associated changes are due to the constitutional condition on which the brain disease ultimately depends. The most important of these are the following: First, albuminuric retinitis, acute or chronic, associated chiefly with arterial degeneration, such as causes cerebral hæmorrhage or cerebral softening. Secondly, syphilitic disease, especially choroiditis, or choroiditic atrophy, associated with lesions of the brain due to acquired or inherited syphilis. Thirdly, tubercular disease—tubercles of the choroid occasionally in tubercular meningitis, very rarely massive tubercle in cases of tubercular growths in the brain. Other forms of associated growth are too rare to be of practical importance. Fourthly, vascular lesions of the retina sometimes coincide with similar lesions in the brain. Thus, embolism of the central artery of the retina may occur in the same patient, and even at the same time, as embolism of a cerebral artery. Miliary aneurisms on the retinal arteries have been observed to coincide with cerebral hæmorrhage due to similar aneurisms in the brain. They are, however, extremely rare. Hæmorrhages in the retina are more common, and are of some significance. They occur especially in albuminuria, in gout that has profoundly affected the system, in leucocythæmia, profound anæmia, purpura, ulcerative endocarditis, and other forms of septicæmia. In the latter they have often white spots in the centre, and are the result of capillary septic embolism. Hæmorrhages, identical in aspect, are sometimes found in the same cases in the cerebral meninges.

The consecutive changes, that are the result of the cerebral disease, comprehend optic neuritis and atrophy. Neuritis or papillitis consists in swelling and increased vascularity of the intra-ocular termination of the optic nerve—the "optic papilla." It will be remembered that the end of the nerve is visible in the fundus as the "optic disc," the boundary of which is the oval opening in the sclerotic and choroid. The fibres radiate from the nerve on all sides, but not equally; they are few on

the temporal side. The nerve-elements are so translucent that they do not obscure the edge of the disc, except in some cases and to a slight extent, above and below, where they are more numerous. The disc is rarely uniform in its surface, because the separation of the nerve-fibres leaves a central hollow—the "physiological cup"—in which there are few or no vessels, and which is therefore pale, while the periphery of the disc has a rosy tint from the minute vessels that lie among the nerve-fibres. This physiological cup varies much in size, and may be absent. The vascular portion of the disc also varies in extent (inversely as the cup), and varies so much in tint that no inference can be drawn from its colour unless this is observed to change from time to time. Mere congestion, therefore, consisting only of increased vascularity of the disc, is very difficult to recognize, and, although it probably occurs, it is extremely rare as an isolated condition, and is of small practical importance. An actual pathological change, however slight in degree, is usually attended by some swelling of the papilla, and especially by lessened transparency of its structures. The effect of this change is first to lessen the sharpness of the edge of the disc, and then to obscure it altogether. It is, therefore, to this point, the sharpness of the edge, that attention must chiefly be directed. The effect of a morbid change on the edge of the disc is greater when the examination is made by the direct than by the indirect method. On the other hand, if the indistinctness is apparent, and not real—is due to the tint of the disc being nearly that of the adjacent choroid, and not to the lessened transparency of the structures in front of it—the edge is more distinct by the direct than by the indirect method of examination. In the early stage of neuritis the edge of the disc, seen by the indirect method, may appear a little blurred, and surrounded by a pale halo, while by the direct method the halo is resolved into a striated, semi-opaque layer, completely concealing the edge. The early change is greatest on the nasal side, which may be obscured when the temporal edge of the disc, on which there are few nerve-fibres, is still sharp. As the change advances, it involves the whole

circumference of the disc, and the swelling rapidly encroaches on, and ultimately obliterates, the normal white depression in the centre of the disc. The prominence of the swelling is readily recognized by the relative displacement that the different parts appear to undergo on a lateral movement of the observer's head in the direct, or of the lens in the indirect, method of examination. It is also shown by the loss of the central reflection from the vessels where they course down the sides of the swelling, and their plane is no longer at right angles to the line of vision. The tint of the swollen papilla becomes a full red, or, more commonly, a greyish-red, on indirect examination, but the direct method shows a fine striated vascularity. As the prominence increases, the swelling becomes wider in extent, until it may be two or three times the diameter of the normal disc. White spots may appear on its surface, due to the accumulation of products of degeneration. At first the retinal vessels present little change in size, but as the swelling increases, their compression causes the veins to become broader, and the arteries narrower, and extravasations of blood may be visible on the surface or margins of the swollen area. The process varies much in the rapidity with which it is developed; it may reach a considerable intensity in a fortnight, or be still moderate in degree at the end of three or four months. As a rule, the more quickly it is developed, the more intense it becomes. Retrogression is indicated by a diminution in the vascularity, still greater contraction of the arteries, and, later on, contraction of the veins also, if the degree of neuritis has been considerable. The swelling remains, for some time, pale and soft-edged, and slowly sinks until the edges of the disc appear. The substance of the disc is for long, often permanently, occupied by new tissue, which, with the narrowed arteries, affords evidence of the preceding inflammation. Other indications of this are often to be seen in damage to the adjacent edge of the choroid. It is only when the neuritis has been slight that the disc resumes a perfectly normal aspect. When much new tissue has been formed, this, by its cicatricial changes, leaves the disc white and atrophied, in the condition of "consecutive atrophy," or

"neuritic (papillitic) atrophy," as it is also termed. When the papillitis has been very intense, and the swelling wide in area, the adjacent retina may suffer in its nutrition, and products of degeneration may remain as white spots, especially near the macula lutea, simulating the aspect of albuminuric retinitis. The microscope reveals, in the substance of the swollen papilla, various inflammatory changes, of which you will find details and illustrations in my work on "Medical Ophthalmoscopy." Similar, although slighter, changes may be traced back in the substance of the optic nerves, or in their sheaths, even to the chiasma, and they may be more intense in front of the chiasma than they are midway between this and the eye. The outer sheath of the optic nerve in most cases is distended by liquid, so as to form a pyriform swelling behind the globe. The space within this sheath is continuous, behind, with the subarachnoid space around the brain, and in front, with the lymphatic spaces in the optic papilla.

A slight and even considerable degree of optic neuritis may cause no symptoms; acuity of vision may be unimpaired, colour-vision normal, the visual fields unrestricted; but when intense, sight suffers in each of these characters, and may be entirely lost. The damage to vision is often greater during the stage of subsidence than it is during the active period of inflammation, probably because the nerve-fibres are compressed by the cicatricial contraction of the tissue-elements formed during the inflammation. As I mentioned in a previous lecture (p. 73), the affection of sight is not necessarily due to the visible papillitis; it may be the result of more intense inflammation behind the eye, or the effect of the intracranial disease. After the neuritis has quite subsided, improvement in sight often takes place by the recovery of some of the damaged fibres, but there may remain absolute blindness, or considerable amblyopia, and often there are very irregular changes in the field of vision, both for white and colours.

Optic neuritis may result from many diseases of the brain, but the most frequent cause is tumour, and a considerable

degree of neuritis is seldom due to any other lesion. Next in causal frequency is meningitis, especially when it affects the base of the brain; the inflammation caused by meningitis is usually less intense than that of tumour, perhaps on account of the briefer duration of the disease. Optic neuritis does not result from cerebral hæmorrhage, or from thrombotic softening. It has been several times observed in cases of softening from embolism, when the source of the obstructing plug was active endocarditis, perhaps because the material carried from the valves is of an irritating, septic character, and determines a greater degree of secondary inflammation in the softening produced. Neuritis has also been met with, once or twice, in cases in which no naked-eye lesion of the brain could be found, but in which the microscope revealed slight inflammatory changes—a diffuse cerebritis. In all these cases the neuritis is, as a rule, double, although it may develope more rapidly in one eye than in the other. Occasionally it is one-sided, and then it is generally on the side opposite to the lesion; but unilateral optic neuritis is much more frequently due to disease at the back of the orbit, or at the optic foramen.

It must be remembered, however, that optic neuritis may result from other causes than intracranial disease; it occurs in chlorosis, albuminuria, lead-poisoning, and after certain fevers, especially scarlet fever and typhoid. An important fact is that in many of these conditions its occurrence is associated with some cerebral symptoms; thus, when neuritis preponderates over the other retinal changes in albuminuria, there is usually much pain in the head, and in lead-poisoning it is usually associated with the acute cerebral symptoms that have received the name of "encephalopathia saturnina." Remember this concurrence of cerebral symptoms with neuritis due to a general disease, because it often gives rise to a diagnostic difficulty.

The mechanism by which optic neuritis is produced is a subject on which various opinions have been held. A full account of these will be found in "Medical Ophthalmoscopy."* It is sufficient here to say that the early theory that neuritis

* Second Ed., p. 65.

is due to increased intracranial pressure, acting mechanically, has been proved erroneous. Slow increase of intracranial pressure has almost no influence on the occurrence of this symptom. In a large number of cases of neuritis there is distinct evidence of a descending inflammation, either along the trunk of the optic nerve, or along its sheath; and in cases of meningitis, such descending inflammation is invariable. The distension of the optic sheath with serum has been regarded as the chief mechanism, but it is not essential for the production of neuritis; it may be absent, and its occurrence is related especially to the presence of an excess of subarachnoid fluid. The signs of mechanical "strangulation," which are to be observed in cases of intense neuritis, are no evidence that the inflammation was caused by any mechanical process. The cause of the strangulation is the compression of the veins by the inflammatory products within the swollen papilla, and not, as was once thought, their compression within the sclerotic ring or behind it by the distension of the sheath. It is probable that optic neuritis is rarely due to a single factor, and that the most potent element is the descent of a process of tissue-irritation, which, when it reaches the papilla, sets up a more intense inflammation; that in some cases this influence is alone effective; and that in others it is aided by the distension of the sheath, which hinders the escape of effete products, increases œdema, or even conveys irritating material. The distinction between optic neuritis and "choked disc" is one of degree, and not of mechanism. So far as optic neuritis has any single significance, it is that of an irritative process within the skull.

There is an important relation between the chronicity of the neuritis and that of the intracranial process. A chronic cerebral process may cause an acute neuritis, but a chronic neuritis never results from an acute process, and the degree of the chronicity of the neuritis is an indication of the degree of chronicity of the intracranial disease. When the latter begins to improve, the neuritis lessens, and the commencing subsidence of the intra-ocular inflammation is often the first indication of the improvement of the brain lesion.

Optic nerve atrophy is a less frequent symptom of brain disease than is optic neuritis, with the exception of that form of atrophy which is the consequence and evidence of past inflammation. Primary atrophy is rare, and confined to those forms of brain disease that are degenerative in nature. It is met with, for instance, in cases of disseminated sclerosis, and of general paralysis of the insane, as an associated symptom. Secondary atrophy occurs, not infrequently, in consequence of damage to the optic nerve behind the eye, or to the optic chiasma. Disease of the optic tract does not cause marked atrophy of the disc. When the disease is at the chiasma, both eyes may suffer; when it is in front of the chiasma, the atrophy is usually limited to one eye. The characteristic of secondary atrophy is that sight fails without ophthalmoscopic changes to account for the failure, and this is slowly followed by atrophy. The only distinctive features of the failure occur when the disease is at the chiasma (see p. 73). If the lesion of the nerve is inflammatory, some descending inflammation may reach the eye and be visible at the optic disc. Its amount depends on the intensity of the inflammation, and on its proximity to the eye, but it is always insufficient to account for the affection of vision. When the lesion is near the eye, the vessels often present distinct constriction. Of the significance of these various changes as indications of the pathological nature of the cerebral lesion, I shall have more to say when this problem engages our special attention.

You may have observed that, in this outline of the changes met with in brain disease, I have said nothing about congestion or anæmia of the retinal vessels or of the disc, as an indication of similar states of the brain. Exaggerated expectations and, indeed, opinions were formed, when the ophthalmoscope was first introduced, regarding its revelations. It was thought that, since the blood comes to the eye from a cerebral artery, and returns to an intracranial sinus, the circulation in the eye would share, and show, the changes in that of the brain. But experience has not confirmed the opinion. The circulation in the eye is, so to speak, autonomous; the state of the vessels is regulated by independent

influences. When all the veins of the head become over-distended, in consequence of an obstruction to the flow through the superior vena cava, the retinal veins suffer far less than those on the surface of the head, no doubt because, in the closed chamber of the eye, neither distension nor contraction of the vessels can readily occur from a mechanical cause. The information afforded by the ophthalmoscope is important and extensive beyond the dreams of its inventors, but the expectation that was most confidently held has not been fulfilled; and if you find any author professing to diagnose the state of the cerebral circulation from that of the eye, or drawing therefrom indications for treatment, you may safely conclude that he knows very little about the subject.

LECTURE XIII.

DIAGNOSIS OF THE SEAT OF DISEASE: "LOCALIZATION"—GENERAL METHOD OF DIAGNOSIS — CEREBRAL HEMISPHERE, CRUS, THE CORPORA QUADRIGEMINA.

GENTLEMEN,—According to the loose use of words that is still common in medical terminology, we are said to "localize" disease, that is, to make it local, when we infer its locality from the symptoms it produces; and this process of inference is termed "localization." In the previous lectures we have considered the chief symptoms of brain disease, and we have learned in what part of the brain a lesion may be situated to produce those symptoms. If is necessary now to review these facts from the other side; to look at the symptoms from the standpoint of brain-regions. Before we do so, I must remind you of the distinction between the direct and indirect effects of disease (see p. 48), and that only those symptoms which last for some time can be regarded as due to the destruction wrought by the morbid process. Moreover, you must bear in mind that the nature of a lesion, as well as its position, exerts an influence on the character of the symptoms that are produced. Speaking generally, we may say that in acute lesions the symptoms are, at first, wider in range, while in chronic lesions they are, throughout, slighter in degree, than might be anticipated from our knowledge of the function of the parts that are implicated. Moreover, the symptoms produced by

slow processes, such as a growth or an abscess, differ in range, according as the disease influences the grey or the white substance, nerve-cells or nerve-fibres. The fibres are strangely tolerant of slow pressure, especially when this is widely diffused. A tumour or an abscess may occupy the position of the motor path, and yet cause only slight palsy, because the fibres have been merely displaced by it, and still retain much power of conducting, in spite of their compression. When such a disease occupies the grey matter, there is a similar, although slighter, tolerance of pressure, so far as concerns loss of function, but the nerve-cells exposed to the morbid influence are in a state of abnormal irritability, and the connection between the nerve-cells of the cortex is so abundant and extensive, that the functional change may spread over a wide area and to a considerable distance from the primary disease. Discharges, causing convulsions, may commence at various parts of the area thus exalted in irritability. Moreover, centres are sometimes inhibited by the irritation, instead of being "discharged"; and there results loss of function instead of over-action. Hence, the symptoms produced by such diseases are often anomalous, and extreme care is needed in drawing conclusions from them. Some extensive collections of facts relative to this subject have been rendered absolutely valueless by disregard of this consideration.

To return to our immediate subject. Having ascertained what symptoms are present, we must, in the case of an acute lesion, endeavour to distinguish the direct from the indirect. We can only do this with certainty by waiting till the indirect symptoms have passed away, as they will have done in a few weeks. We may, indeed, make a guess at the direct symptoms soon after the onset, from their severity, and from the absence of any indication of early improvement in them; but to attain a high degree of probability in our local diagnosis we must, in many cases, wait. Fortunately we have not thus to wait for indications of the nature of the lesion, on which, and not on the exact seat of the disease, our treatment depends.

Before you can draw any conclusion from the symptoms as to the seat of the disease, you must consider their mode of development, whether slow or sudden, simultaneous or successive. The bearing of this on the problem we are now considering is very important. If the morbid process is one of sudden onset, only those symptoms that came on together can be regarded as due to the same lesion. Take, for example, a not uncommon case, that of a patient who presents paralysis of the limbs on one side, and of the third nerve on the other. These are symptoms of disease of the crus. But to have this significance, if the hemiplegia came on suddenly, the paralysis of the third nerve must have come on at the same time. If you find, on inquiry, that the paralysis of the third nerve existed before the sudden hemiplegia, it cannot be due to the same morbid process, and the affection of the third nerve must be eliminated from the problem of the seat of the disease causing the hemiplegia. But if both were gradual in development, we cannot certainly dissociate them : they may then be due to one lesion or to separate lesions.

Having thus determined what symptoms developed in such a manner that they may possibly be due to a single lesion, we have next to ask, Is there any part of the brain in which a single lesion would produce this group of symptoms? Some combinations of symptoms can be caused only by a lesion in one situation ; others may be produced by a lesion in any one of several places. Others, again, are of such a character that there is no part of the brain in which a single lesion will produce them. To return to the instance we have just taken, hemiplegia on one side, and paralysis of the third nerve on the other, can be produced only by a lesion of the crus. Hemiplegia and paralysis of the third nerve on the same side cannot be caused by a single lesion in any situation, since the motor tract, where contiguous to the nerve, is that to the limbs of the opposite side.

Many of the symptoms of brain disease are of no localizing value, because they may be caused by disease in any situation ; while others are of low value, because, although

produced by disease in various places, they are rather more frequent when it occupies certain situations. Most of the diffuse symptoms (see p. 49) are of this character—headache, vomiting, optic neuritis, mental change, loss of consciousness. In so far as these vary with the seat of the disease, their variations have been mentioned in the account of the symptoms. Some symptoms that are focal, and not diffuse, are produced by disease in so many places that they have but little significance, as, for instance, conjugate deviation of the head and eyes from the paralysed side in severe hemiplegia. This may occur in lesions of the motor tract anywhere between the pons and the cortex.

Bearing in mind these facts, we may proceed to our review of the chief symptoms that are produced by lesions in various parts of the brain, and we may commence with the *cerebral cortex*. In the *frontal lobe*, or rather what is termed the prefrontal lobe, in front of the ascending frontal, lesions are usually unattended by either motor or sensory symptoms. Considerable mental change is rather more frequent than in other situations, sometimes taking the form of chronic insanity, and gives some support to the opinion which ascribes to this region a high psychical importance. But these symptoms are neither characteristic nor invariable. The posterior part of the lowest frontal convolution on the left side constitutes, however, an exception to the negative character of this region, since disease here, around the anterior limb of the fissure of Sylvius, causes, with much uniformity, motor aphasia. Lesions which irritate, and are adjacent to the ascending frontal, often cause convulsions that begin locally. It is possible that the limb-centres are not always strictly limited to the ascending frontal and that disease on the roots of the antero-posterior gyri may cause motor symptoms in some individuals, and not in others; but no symptoms have been observed in man corresponding to the functional centres that you often see marked on these convolutions in diagrams of the human brain, to which they have been transferred from the brains of monkeys.

In the *central area* of the brain (as the motor region—ascending frontal, ascending parietal, and superior parietal—is often termed), disease causes motor paralysis. There is loss of power in the leg when the lesion is in the upper third; loss of power in the arm, when in the middle third; in the face, when the disease is in the upper half of the lower third; in the lips and tongue, when it is in the lowest part of the ascending frontal; and, on the left side, disease in this part causes also aphasia. Partial hemiplegia is much more common than paralysis involving the whole of the side, and the parts that suffer together are those for which the centres are contiguous in the cortex. Convulsions are exceedingly common, and their characteristic feature is the local commencement, already described, and due to the commencement of the discharge in an irritated centre. This is not always the region most diseased. Considerable disease in one centre may cause discharges to start from an adjacent centre less diseased, and therefore capable of greater functional excitement. Thus a tumour occupying the highest part of the ascending frontal convolution (leg-centre) caused at first convulsions beginning in the foot, and afterwards, as the disease advanced, convulsions beginning in the hand (the centre for which was not invaded by the growth), and even sometimes in the face (the centre for which was some distance from the tumour). Thus, also, the disease causing such convulsion may be near, but not in the motor area. Hence local paralysis is a much more decisive indication of the seat of the disease than are the local convulsions. In the case of tumour, with changing convulsions, just mentioned, the leg became paralysed, and this indicated that it was in the leg-centre that the destruction of tissue was in progress. Disease of the motor cortex often causes some defect of sensation, chiefly in the extremity of the paralysed limb, and in the convulsions a local sensory aura often precedes the spasm, and may even pass through the whole side before the spasm comes on (see p. 62). There is never complete hemianæsthesia when the disease is limited to the motor region. With the cutaneous defect in the extremity paralysed, and even without this, there

is usually an inability to recognize the position of the limb; but we do not yet know whether this loss is confined to cortical lesions. Probably it is not (see p. 69).

Disease is rarely limited to the remaining part of the *parietal lobe*, supra-marginal convolution, and there is much uncertainty as to the symptoms produced. The lesion usually extends to the motor region, causing hemiplegia, and there is some reason to think that ptosis accompanies the hemiplegia rather more frequently when the lower parietal lobe is affected. Extensive disease of the outer surface of the hemisphere causes hemianæsthesia, and to this the disease of the lower parietal lobe probably contributes. Defects of speech have been met with, although not with such constancy or uniformity as to furnish diagnostic indications. A lesion of the angular gyrus at the infero-posterior angle of the parietal lobe, probably causes crossed amblyopia, and disease about this region causes word-blindness; but the precise locality on which this symptom depends is not yet known. Only one symptom is known to be produced by disease of the *occipital lobe*—hemiopia,—and we are still uncertain as to the exact part of the cortex of the occipital lobe on which the symptom depends. Small lesions may cause a loss of only part of the half-field, *e.g.*, a quadrantic defect. It is probable that the separation between the preserved and blind halves of the field does not differ from that in the hemiopia produced by disease in the lower part of the visual path, but that there is greater peripheral limitation of the preserved half of the field. Irritating disease of this region may cause convulsions that commence with a visual aura referred to the opposite eye or opposite side. Disease of the *temporo-sphenoidal* lobe causes no motor or sensory symptoms unless the first temporal convolution is involved, and then deafness is produced on the opposite side; and if the disease is on the left side of the brain, there is word-deafness and sensory aphasia. Irritating disease may cause convulsions that commence with an auditory aura. If the lesion is on the inner surface of the lobe, and involves the anterior part of the uncinate convolution, it is probable that loss of smell

may occur on the same side. It is important to remember that the inner part of this lobe is contiguous to the optic tract; tumours are apt to invade the tract, and thus cause hemiopia, which may be followed by hemiplegia from the further extension of the disease into the crus cerebri. This is one of the commonest causes of hemiopia from disease of the tract.

The *island of Reil* is rarely the seat of isolated lesions, and much uncertainty still exists regarding its functions and the symptoms that result from its damage. Defects of speech, similar to those that result from disease of the third frontal convolution, have been attributed to its disease; but the evidence is not altogether satisfactory, and speech-defect has been absent in some cases in which the insula was extensively damaged.

In the *white substance of the hemisphere*, centrum ovale, the symptoms produced by disease resemble, for the most part, those that are caused by lesions of the corresponding part of the cortex, with the exception that convulsions are rare, save in the case of tumours, which cause persistent irritation. The disturbance of speech is transient unless the disease is seated just beneath the cortex, for the reason given in a previous lecture (p. 130). Hemiopia results from disease of the white substance of the occipital lobe, but it is doubtful whether crossed amblyopia occurs unless the angular gyrus is affected. Lesions of the white substance of the lower part of the temporal lobe, and of most of that of the prefrontal lobe, cause no symptoms.

In the *central ganglia*, lesions of either part of the corpus striatum cause no lasting symptoms if limited to the grey matter. I have seen the lenticular nucleus softened from one end to the other, and the patient, before death, presented no indication of hemiplegia. I will not say that the disease caused no symptoms, but her history was very carefully taken, and no account was obtained of any motor or sensory symptoms. Such cases are, however, extremely rare, because, in most instances, the internal capsule suffers also. A lesion of the anterior or middle part of the optic thalamus may also

cause no symptoms, although there is some reason to think that the latter sometimes causes athetoid inco-ordination. When the posterior portion of the thalamus, the pulvinar, is diseased, hemiopia occurs; but it is not quite certain whether it depends on such disease, or whether there is, in these cases, interference with the posterior portion of the optic tract, which enters the corpus geniculatum close to the posterior extremity of the thalamus. The balance of evidence, however, is in favour of the dependence of the symptom on the disease of the thalamus.

The effects of disease of the *internal capsule* vary according to the part damaged. When this is the anterior limb, between the caudate and lenticular nuclei, no symptoms result. Disease of the anterior two-thirds of the hinder limb causes hemiplegia of the ordinary type, and is indeed the most frequent cause of this. It is supplied by branches of the middle cerebral that are apt to be occluded, either from disease of the wall of the vessel, or from embolism, and the arteries adjacent rupture more frequently than any other, causing hæmorrhage, which tears across the fibres of the capsule. Since the fibres for the face occupy, as we have seen, the junction of the two parts of the capsule, an isolated facial palsy may result from a very small lesion in this situation; more frequently the adjacent fibres for the arm are involved also. The leg-fibres, lying behind the others, may escape in a very small lesion paralysing face and arm, while damage to the hinder part of the posterior limb causes a preponderant paralysis of the leg. If the disease involves the hinder third of the posterior limb, occupied by the sensory path, hemi-anæsthesia is produced—loss of sensation on the opposite side, head, trunk, and limbs, extending up to the middle line. The loss is sometimes complete; more often a touch is unfelt, while a strong painful impression is perceived. The special senses are also often involved—taste, hearing, and perhaps smell being lost on the anæsthetic side, while vision to that side is lost in each eye (hemiopia). The last may result either from disease of the external corpus geniculatum, or from damage to the fibres from the tract to the cortex, or from damage to the optic thala-

mus, or from the latter to the cortex. Considerable damage to one part of the capsule is often attended by slighter and transient damage to the other part; hence, hemianæsthesia may occur at the onset of hemiplegia and pass away, and, conversely, hemianæsthesia is often accompanied by transient weakness of the limbs on the anæsthetic side. Convulsions rarely result from disease in this region. Incomplete hemiplegia is, however, often accompanied by mobile spasm (athetosis, post-hemiplegic chorea), but we do not know whether this is to be ascribed to the disease of the capsule, or to that of the adjacent ganglia, especially of the optic thalamus.

The *corpora quadrigemina* are so rarely damaged alone, that there is much uncertainty as to the symptoms that depend upon their disease. The balance of evidence is strongly against the old opinion that sight is impaired. The chief effect is probably derangement of the internal and external ocular muscles, sometimes impairment of certain ocular movements, especially of the upward movement of the eyes and elevation of the lids. An inco-ordination of movement, noted in some cases of tumour, is probably the result of pressure on the adjacent middle lobe of the cerebellum.

Damage to the *crus cerebri* causes paralysis of the face and limbs on the opposite side, similar in character to that which results from disease of the internal capsule, but accompanied by palsy of the third nerve on the side of the lesion, and therefore on that opposite the hemiplegia. Hemianæsthesia may accompany the motor palsy, from implication of the sensory path in the tegmentum of the crus. Hemiopia may result from damage to the optic tract, but is very rare, since the lesion is usually situated close to the pons. Giddiness is common, but is usually ocular, and due to the affection of the third nerve.

These facts of localization are complex, and those that remain are still more so. It may be well, therefore, to postpone their consideration until our next meeting.

LECTURE XIV.

DIAGNOSIS OF THE SEAT OF DISEASE (*Continued*): PONS, MEDULLA, CEREBELLUM; BASE; VENTRICLES—REVIEW OF SYMPTOMS IN RELATION TO LOCALITY—BASAL LESIONS.

GENTLEMEN,—In the last lecture, you will remember, we passed in review the symptoms that are produced by disease in various parts of the cerebral hemispheres, in the crus cerebri, and in the corpora quadrigemina. To-day we resume our survey, and must notice first the effect of lesions in the mesencephalon, where the paths from the hemisphere to the limbs are brought into proximity, where the chief cranial nerves have their origin, and where an important junction is effected between the fibres of the cerebellum and those of the cerebral hemisphere.

In the *pons Varolii* a lesion usually causes very characteristic symptoms, due to the implication of the motor path and of the cranial nerves that arise from the pons. Tumours that grow slowly, however, and especially infiltrating tumours, may attain a considerable size without causing characteristic symptoms. These symptoms may be either unilateral or bilateral, according as the disease affects only one or both sides of the pons. Motor palsy is very common. Hemiplegia results from one-sided disease, the limbs being, of course, paralysed on the side opposite to the lesion. In the upper half of the

pons, the upward facial path having crossed, the face may suffer with the limbs exactly as in disease of the internal capsule; but the chief characteristic of pontine hemiplegia is the affection of the fifth, facial, or sixth nerves on the side of the lesion, that is, opposite to the limbs, constituting the "alternate hemiplegia" already described (see p. 58). The tongue suffers as in ordinary hemiplegia. The palsy of the fifth may involve motor or sensory parts, or both; in the latter case there is usually much pain, referred to the face, and due to irritation of the nerve-fibres. When there is crossed palsy of the face, this is usually unaffected on the side of the limb-palsy, but extensive disease may affect both sides of the face. Paralysis of the sixth may be associated with weakness of the opposite internal rectus, causing a paralytic inability to move the eyes towards the side of the lesion; they are thus in conjugate deviation towards the paralysed limbs, a direction opposite to that usually caused by disease of the hemisphere. In bilateral disease, various combinations of paralysis of limbs and the cranial nerves may occur. In acute lesions of the upper part of the pons, the pupils are often strongly contracted from irritation of the nuclei of the third nerve, and, if these nuclei are invaded and destroyed, the contraction gives place to dilatation and immobility. Difficulty in swallowing and articulation result from interference with the adjacent medulla. Convulsions are rare, except in acute lesions, and are then sometimes peculiar, affecting both arms or both legs. Tonic spasm in the paralysed limbs is a common and often very marked symptom. Rhythmical movements are rare. Anæsthesia is less common than motor palsy, and the two are often disproportioned. The explanation of this lies in the fact that the motor path is in the anterior part of the pons, the sensory in the outer part of the posterior portion, the two being separated by the deeper transverse fibres. Tingling and formication in the limbs are occasionally marked. Deafness is rare. Vision is never directly impaired. Taste may be affected. Giddiness is sometimes severe. Respiration may be irregular and abnormal in acute lesions, and the action

of the heart irregular. Hyperpyrexia may result from acute lesions, even from hæmorrhage, which elsewhere, at first, depresses the temperature. Psychical disturbance is much more frequent than might be expected, and is perhaps produced by the damage to the fibres and grey matter which, as we have seen, connect the cerebellum with the frontal and temporo-occipital regions of the brain. It varies much in character.

The most vulnerable part of the *medulla oblongata* is the region occupied by the nerve-nuclei at and below the apex of the fourth ventricle; and the most characteristic symptoms are those resulting from the damage to these nuclei. It is rare for an acute lesion to cause symptoms in the limbs, because a small lesion rarely occupies the pyramidal region, and any other than a very small lesion usually causes rapid death by interference with the cardiac and respiratory centres. Hence, also, if limb-symptoms do occur, they are slight; they may be unilateral or bilateral. A small tumour may, however, paralyse the limbs on one side, and the hypoglossal nerve on the other; but these symptoms depend more frequently on disease outside the medulla, compressing it and the nerve-trunks. The spinal accessory and glosso-pharyngeal nerves may then suffer with the hypoglossal, causing the group of symptoms that I have already described (p. 104). Of acute lesions, hæmorrhage usually kills with great rapidity. Necrotic softening, from disease of the vertebral, occluding its branches, sometimes causes symptoms of sudden onset in the muscles supplied by the bulbar nuclei. There is palsy of the lips, tongue, palate, pharynx, and sometimes of the larynx, constituting acute labio-glossal paralysis, or acute bulbar paralysis. The loss of power is usually equal in the muscles of both sides, the lesion being seated commonly near the middle line. The palsy is occasionally irregular in distribution. The onset may be attended by subjective sensations in the legs, tingling, formication, etc., and rarely by some motor weakness. Chronic degeneration of the nuclei causes a palsy of similar distribution, but differing in its course, being gradual in onset and progressive in course, and often associated with muscular atrophy elsewhere.

Active disease of the *middle peduncle of the cerebellum* causes very peculiar symptoms, the chief being an involuntary "forced" movement of the trunk on its longitudinal axis, sometimes towards, sometimes from the side of the lesion. It may amount only to an irresistible tendency to lie on one side. There is usually vertigo, and sometimes it is very intense. The eyes may be directed to one side even when there is no tendency to fall, or there may be a difference in their height, or nystagmus. These symptoms are absent in stationary lesions, and so probably depend on active irritation, and not on a loss of nerve-structures. Associated with these there are often other symptoms of disease of one side of the pons—paralysis of the fifth nerve and of the opposite limbs.

The *middle lobe of the cerebellum* seems to be concerned in some way with the maintenance of equilibrium, and its disease causes unsteadiness in standing and walking. The feet are placed wide apart, and the defective balance may cause the patient to progress in a zig-zag manner. Sometimes there is a tendency to fall forwards or backwards. The gait resembles that of a drunken man. There is not the irregular movement of the legs common in locomotor ataxy. A sense of giddiness often accompanies the unsteadiness, but is not its cause, since either may exist alone. The unsteadiness may be present when the lesion is stationary, as well as in active disease. Vomiting is also frequent, but occurs only when the morbid process is active. The intimate connection of the vagus with the nerve-structures concerned in equilibration is seen in the vomiting that attends severe vertigo, such as that from ear disease, and in the curious phenomena of sea-sickness, which probably depend on oscillations of the endolymph in the semicircular canals. This connection probably underlies the vomiting from cerebellar disease. The vomiting may occur apart from giddiness. The same combination of vomiting and giddiness occurs in labyrinthine vertigo, but the giddiness is more distinctly paroxysmal, and the vomiting occurs only in connection with the giddiness. Disease of the *hemi-*

spheres of the cerebellum, away from the middle peduncle, causes *per se* no definite symptoms of diagnostic significance. As Nothnagel first pointed out, the unsteadiness of movement does not result from disease in the hemisphere unless it is of such a character as to compress the middle lobe, and it is on this compression that the symptom depends. Compressing lesions, such as a tumour, in either the middle lobe or hemisphere, exert pressure on the pons, the tentorium limiting the pressure to the subtentorial region. Hence there is often slight weakness of the limbs, with signs of descending degeneration, increased myotatic irritability, etc., and sometimes feelings of numbness and tingling, rarely anæsthesia. When the tumour is in the middle lobe of the cerebellum, the limbs suffer on both sides; when in one hemisphere, the limbs suffer chiefly on the opposite side. The cranial nerves arising from the pons are also sometimes compressed, and the sixth nerves suffer earlier and more than others, on account of their exposed course over the convex surface of the pons. The facial and auditory nerves sometimes suffer from lateral pressure. Convulsions are rare in disease of the cerebellum, but tonic spasm, tetanoid in character, may occur in tumours, and is probably due to pressure on the pons.

In the ventricles of the brain only two lesions are common, hæmorrhage and liquid effusions, and the localizing symptoms are subordinate to those special to the morbid process.

It may be well to recapitulate the chief facts that we have considered, looking at them from the side of the symptoms instead of localities.

Persistent hemiplegia of the ordinary type may be due to disease of the motor path anywhere above the middle of the pons, or to disease of the motor cortex; but transient hemiplegia only shows that the disease is near the motor path or centres, so that it can affect them indirectly. Since the most common seat of disease causing hemiplegia is the

corpus striatum and internal capsule, there is always a presumption that simple hemiplegia is due to a lesion in this situation. If the face and tongue escape, the disease may be anywhere in the path between the medulla and cortex, but there is a presumption that it is above the internal capsule, where the elements of the motor path are wider apart, and partial escape is more probable. The same is true, *à fortiori*, of partial hemiplegia affecting face and tongue, or face and arm. These, if of sudden onset, are most frequently due to disease of the cortex, sometimes to that of the white substance, very rarely to a lower lesion. I say "if of sudden onset," because when a chronic lesion involves the motor path anywhere, the gradual impairment may be at first partial in extent; but if the path, at the seat of disease, occupies a small area, all parts soon suffer, and the initial weakness of one limb, as it deepens in degree, soon extends to the other parts of that side. Considerable loss of sensibility on the extremities of the limbs, not through the whole side, probably indicates a lesion of the cortex or white substance beneath. Well-marked hemianæsthesia with hemiplegia indicates an extensive lesion of the posterior limb of the internal capsule; and conjoined hemiopia indicates that the lesion is extensive at the posterior part of the capsule, and has damaged the optic path, probably at the corpus geniculatum, or optic thalamus. Affection of the other special senses has the same indication of extensive mischief in this situation, but it is more common to have these symptoms without than with persistent hemiplegia, because a lesion in this region does not often destroy the motor part of the capsule. Paralysis of a cranial nerve on the side opposite to the hemiplegia indicates a lesion at the level of origin of that cranial nerve—the crus in the case of the third nerve, the middle of the pons in that of the fifth, the lower third of the pons in the case of the facial or sixth, the junction of the pons and medulla in the case of the auditory, the medulla in the case of the hypoglossal or spinal accessory. It must be remembered, however, that the localizing significance of these associations depends, in the case of acute lesions, on their simultaneous onset.

Ptosis, slight in degree, on the side of the hemiplegia, indicates that the lesion is in or near the cortex, with a slight probability that it involves the parietal lobe, or subjacent white substance. Conjugate deviation of the eyes from the side paralysed, or towards it if there are convulsions or much rigidity of limb, is of no localizing significance; towards the side paralysed, without any indications of spasm, it suggests, but does not prove, an affection of the pons; if there is complete loss of power of the external rectus, and slighter loss of power of the opposite internal rectus, the lesion is certainly in the pons. General convulsions are of no localizing significance: those that begin locally, if they occur at the onset of an acute lesion, suggest, and, if they attend a chronic lesion, practically prove, that the disease is in, or just below, the cortex.

Paralysis of the cranial nerves has a far more definite significance when several nerves are affected, than when one suffers alone. The possible seats of the disease are numerous in the latter case, while in the former they are few, and often there is only one position in which a single lesion can cause the combined effect. We may glance at a few of the more important indications. Palsy of one hypoglossal is probably due to disease outside the medulla, and this is certain if the palate and vocal cord (spinal accessory) are affected also. Paralysis of both sides of the tongue is usually due to disease of the nuclei (very rarely to symmetrical disease of the cortex), and the lips usually suffer with the tongue. If the face is paralysed, we must note first whether the lower part suffers chiefly, and the muscles of the forehead and eyebrow escape, and electrical excitability is normal, or whether all parts are involved, and faradaic irritability is lost. In the first case the lesion is above the nucleus, and is probably above the internal capsule. In the second it may be in the nucleus, the fibres of origin in the pons, at the base of the brain, or in the temporal bone. Paralysis of the lower part of the face and tongue on one side indicates disease of the hemisphere, and probably of the cortex. If the auditory nerve suffers with all parts of the facial, and there is no ear disease, the lesion is at the base.

Paralysis of all parts of the face on each side is due to symmetrical disease of the facial nuclei, or of the nerve-trunks at the base.

Paralysis of one ocular nerve is usually due to disease at the base; that of all the nerves of one eye indicates disease about the orbital fissure; that of all the nerves of both eyes, degeneration of the nuclei. Paralysis of the sixth and facial (without the auditory) is due to disease within the pons. Paralysis of the sixth and fifth indicates disease outside the pons; although the nerves arise at some distance from each other, the sixth nerve, as it enters the dura mater, is nearer the fifth than is any other nerve.

The diagnosis of the seat of disease at the base of the brain has to be made chiefly in the case of chronic and subacute lesions, especially tumour and meningitis. In hæmorrhage the local symptoms are rarely prominent—they are lost in the more diffuse symptoms that attend severe hæmorrhage. In all basal disease the characteristic symptoms are due to the interference with the cranial nerves. In disease of the posterior fossa, the fifth and those below are liable to suffer, and with them the motor tract, causing weakness in the limbs, usually hemiplegic in character. The distinction from disease within the pons depends on the circumstances that the nerves more often suffer before the motor tract to the limbs when the lesion is at the base, and that the combination of nerves affected is different. Thus, at the base, the fifth and sixth nerves may suffer together, the facial and auditory, the spinal accessory and hypoglossal. The nerves are often irritated before they are paralysed, and such irritation of the fifth nerve often causes very severe and persistent neuralgic pains in the region supplied by it, and sometimes (although rarely) trophic changes in the eyeball. Rigidity in the limbs may occur from the same cause, especially when the anterior pyramids of the medulla are exposed. General convulsions are rather more common from disease outside, than from that within the pons. Pressure on the middle peduncle of the cerebellum may give rise to intense vertigo and forced movements.

In the middle fossa, symptoms are chiefly produced by

tumours, and the most important are symptoms of irritation and paralysis of the fifth nerve. The Gasserian ganglion is often involved, and trophic changes in the eyeball are common. In the interpeduncular space, morbid processes affect one or both third nerves, and often also one or both motor tracts in the crus. Less commonly one optic tract is involved, causing hemiopia. Convulsions are rare.

In the anterior fossa, properly speaking, only the olfactory nerves suffer, but disease usually involves also the neighbourhood of the sella Turcica, and damages one or both optic nerves, and the nerves to the eyeball in the wall of the cavernous sinus or at the orbital fissure. Occasionally the optic chiasma is involved; the fibres that decussate suffer chiefly, causing temporal hemiopia.

LECTURE XV.

DIAGNOSIS OF THE NATURE OF THE LESION—PATHOLOGICAL FACTS BEARING ON DIAGNOSIS.

Gentlemen,—We pass now to the last diagnostic problem that we have to consider, that which is, beyond all question, the most important—the nature of the disease. The diagnosis of the seat of the lesion is a problem of fascinating interest to the physician, but the limits of its importance to the patient are quickly reached. It is important to the latter chiefly as affording, in some instances, a guide to prognosis. But on the nature of the lesion depend not only the prognosis for the most part, but the treatment altogether. This pathological diagnosis is, as a general problem, far more difficult than the anatomical diagnosis. It is true, the nature of the disease can sometimes be determined with ease and confidence; more often, however, it is a question of much difficulty and some uncertainty.

We may ask, in the first place, what are the morbid processes that we have to deal with? We may leave out of the question those that are of extreme rarity, and consider only those that you are likely, at some time or other, to meet with —that constitute about ninety-nine out of every hundred cases of organic brain-disease that come under observation.

They are not very numerous, fortunately for us. They are inflammation, chiefly of the membranes, hæmorrhage, necrotic softening, tumours, aneurism, abscess. Congestion and anæmia of the brain, which are not, properly speaking, organic diseases, we shall have to notice incidentally, and likewise three degenerative diseases—the bulbar degeneration that I have already mentioned to you, termed, from its symptoms, "labio-glosso-laryngeal paralysis"; "general paralysis of the insane," which differs from other varieties of insanity in the obtrusive physical symptoms that form part of its manifestations, and often come into diagnostic relation to other diseases; and, lastly, "disseminated" or "insular sclerosis."

To understand many of the indications of these lesions, it is needful for you to know certain facts regarding their mode of production and pathology. It is impossible for me to enter, however briefly, into the pathology of all these lesions. But the diagnostic indications, that I shall have to describe to you, will be clearer if some facts, regarding especially the acute lesions, are fresh in your minds. It may be well, therefore, for me to depart, in this lecture, from the special subject of this course, in order to direct your attention to these points.

Although we shall deal with congestion of the brain only incidentally, you should know that doubt has been thrown on the very occurrence of this condition—doubt that is a reaction from another extreme. The state of the blood-vessels of the brain after death is very little indication of what their condition has been during life. Before this fact was known, post-mortem distension was regarded as evidence of ante-mortem congestion, and an extensive symptomatology was elaborated on insufficient grounds,—a symptomatology that has, to some extent, survived its data. Further, "congestion of the brain," like "congestion of the liver," is a ready diagnostic refuge, seductive in its simplicity, and pleasing in its preciseness; easily affirmed, and not easily disproved. On this basis of pure diagnostic fancy, as I hinted in a previous lecture, a

symptomatology has also grown up, and even statistics have been amassed, the value of which is considerably below zero. It is not surprising that from such reasoning a reaction has arisen, and the influence of cerebral congestion in causing symptoms has been narrowed almost to the vanishing point. Even its pathological possibility has been denied, on an old ground that the total amount of blood within the cranio-vertebral cavity cannot vary because the cavity is a closed one. The truth probably lies between the two extremes. Cases in which the pronounced symptoms can reasonably be ascribed to active cerebral congestion are rare—far less frequent than might be imagined from the place that the condition still occupies in the written and unwritten pathology of the present day. But the state probably does occur, and does cause symptoms. The mobility of the cerebro-spinal fluid permits the condition of the vessels to alter in various parts of the brain. If the cranio-vertebral cavity were hermetically closed, the variations could, perhaps, be only relative, not absolute. But the numerous foramina of this cavity are occupied by structures of little resistance, which may yield in some degree. Moreover, the large surface-veins of the spinal cord, and still more the enormous plexuses outside the spinal dura-mater, must constitute an important means of adaptation. The processes of secretion and absorption of the cerebro-spinal fluid, always in constant operation, must be influenced by the degree of pressure, and may quickly vary with it. The enormous variations in the amount of blood in the nerve-centres after death may be admitted as affording some evidence that variations are possible during life. Lastly, the eye is closed at least as completely as the cranio-vertebral cavity, and variations in the amount of blood within it may be actually seen. If, then, you should hear the possibility of cerebral congestion denied, remember that the grounds on which it is denied are not themselves unimpeachable.

Cerebral hæmorrhage is almost always due to the rupture of an artery, very rarely to that of a vein. Veins rupture

chiefly under such extreme pressure as, for instance, attends strangulation, and, under the same circumstances, capillaries may give way and cause minute extravasations. Arteries, however, rupture only when their walls are diseased, and they may then give way without any extraordinary pressure of the blood. Before they burst, their wall yields before the blood-pressure; becoming extended, it becomes thinned. This bulging of the wall constitutes an aneurism, and hence it is that the hæmorrhage always results from the rupture of an aneurism. A healthy wall never yields before the blood-pressure; local change, lessening the elasticity of the wall, always precedes dilatation. The aneurisms that result may be on the large arteries of the base or surface, and are then of some size; or they may be on the small arterial branches, especially on those that go to the central ganglia, and are then minute. They are often called "miliary aneurisms," from their small size. The larger aneurisms on the arterial trunks are meant when we speak of "intracranial aneurism," without the qualification of "miliary." They are often single, and always few. The miliary aneurisms are always numerous; sometimes there are hundreds scattered through the brain. The difference in name has its justification, not only in the difference in size, but also in the fact that the miliary aneurisms cause no symptoms until they rupture, while the larger aneurisms sometimes cause considerable disturbance, especially when they are seated on a vessel at the base of the brain. The local change in the wall that permits the vessel to yield before the pressure of the blood is usually syphilitic disease or inflammation of the wall—either traumatic inflammation, or more often that which results from imperfect obstruction by a plug that comes from an inflamed cardiac valve, and excites an inflammation in the wall of the vessel similar to that in its source. The aneurism sometimes results from simple fibroid degeneration, but rarely from the fatty degeneration that is termed atheroma. The miliary aneurisms are chiefly the effect of a primary degeneration of the wall, of which an important cause is the strain to which they are exposed, aided by the degenerative tendency that is incidental to age, and that

attends certain constitutional diseases, especially kidney disease. Thus the agent in their rupture, the blood-pressure, is in some measure the agent of their production. The reason why they are so frequent on the branches to the central ganglia is probably because these small arteries come off directly from a large vessel, and so the blood-pressure within them is higher than it is in most arteries of the same size. Although the degeneration differs in nature from the atheroma on the larger arteries of the base, their causes are in part the same, and therefore the two often coincide. Indeed, it has been suggested by Nothnagel that the atheroma may aid the production of the minute aneurisms, by the loss of elasticity in the larger arteries that it involves; this renders the pulse-wave more sudden in the branches. From these facts it follows that, while there is no necessary relation between the two, atheroma is present in three-fourths of the cases in which there are miliary aneurisms (Charcot and Bouchard). It is important to remember this fact. Certain general diseases cause a tendency to hæmorrhage, apparently from an acute degeneration of the walls of the smaller arteries. These are scurvy, purpura, pernicious anæmia, and especially leucocythæmia. They may cause cerebral hæmorrhage. The actual rupture of an artery may be produced by the strain of some muscular effort, or it may occur when the patient is at perfect rest, even, indeed often, during sleep. The frequency of rupture during sleep is remarkable. Perhaps it is determined by the influence of gravitation; or rather by the loss, in the recumbent posture, of this aid to the return of blood from the head. Possibly, moreover, the contraction of the vessels, that is said to attend sleep, is of arteries smaller than those that are the seat of miliary aneurisms, and may even increase the blood-pressure in the latter, and help to determine rupture.

The most frequent seat of hæmorrhage is the corpus striatum, the vessels that rupture being especially the branches of the middle cerebral that pass through the lenticular nucleus to the caudate nucleus, or optic thalamus. The extravasation ploughs up the adjacent parts, and usually tears through the internal capsule, causing hemiplegia. Often

it extends into the white substance of the hemisphere, or bursts into the lateral ventricle. Although hæmorrhages may occur in any part of the brain, they are comparatively rare in the cortex—a fact of considerable diagnostic importance. Meningeal hæmorrhage usually results from the rupture of an internal extravasation, or from traumatic causes, but it sometimes occurs in maladies that entail a hæmorrhagic tendency, and in the course of severe acute specific diseases. Ventricular hæmorrhage is also usually secondary, rarely primary.

The extravasation of blood destroys the tissue-elements by lacerating them, and it compresses the adjacent structures. All mechanical injury irritates nerve-elements at the moment of its occurrence, and the irritation that attends hæmorrhage is probably one agent in the production of the loss of consciousness that usually attends the onset. This is doubtless due also to the pressure to which the whole brain is exposed, striking evidence of which is afforded by the extent to which the falx is sometimes bulged to the other side. It is to this compression that the "indirect" symptoms are chiefly due. But the adjacent damage thus produced is not usually intense in degree, although it is wide in range, and the ultimate recovery from these indirect symptoms is often perfect.

The term "softening of the brain" is used in medicine in its literal sense. You have probably already discovered that the popular use of the word is somewhat metaphorical, and that it is applied to various conditions in which there is a slow failure of mental power—conditions which we call by other names, because there is no actual softening. The chief pathological causes of softening are inflammation and arrest of blood-supply. There is, indeed, a third form, simple chronic softening, but it is excessively rare (not more than half a dozen well-authenticated cases are on record), and we need not, therefore, concern ourselves with it. Inflammatory softening we may also leave for the present; it is rare, except as the result of injury (which is beyond our province), or as the first stage of abscess, and that we shall

consider separately. The arrest of blood-supply is due to the obstruction of an artery, and causes a sudden cessation of all the processes of nutrition that maintain the vitality of the tissue-elements. It is, therefore, often called "necrotic softening." This arrest of blood-supply, whatever be its cause, always involves derangement of the collateral circulation, and considerable secondary inflammation about the necrosed area. Hence the extent of the damage that falls short of destruction is usually relatively greater than in hæmorrhage.

Two pathological processes may cause the occlusion of an artery. A plug, from some distant source, may be carried into the vessel by the blood, and be arrested where the artery is narrower than the plug—"embolism"—or the clot may be formed in the artery by coagulation of the blood at the spot obstructed—"thrombosis." Embolism is the result of a morbid process elsewhere in the vascular system, commonly in the heart. Thrombosis is the result of a local disease of the artery, by which its calibre is narrowed and its inner surface is changed. The alteration in the wall of the vessel is usually the result of atheroma or of syphilitic disease. The process of occlusion is often aided by a change in the blood, rendering it more prone to coagulate, or by a slower movement of the blood, giving it more time to coagulate. These may not only aid arterial disease in causing the occlusion, but may also sometimes cause thrombosis when there is no change in the wall of the vessel.

Although the processes of thrombosis and embolism are thus distinct, and usually occur under different conditions, the two may be combined in a secondary manner, which does not, however, affect the important and essential distinction between them. If an artery is obstructed by embolism, the distal portion may be further occluded by coagulation of the stagnant blood. Again, if a clot forms in an artery, it may be detached, and may be moved a little further on. This is, *de facto*, embolism, although the result of thrombosis, and classed as thrombosis since the clot is formed in the artery occluded, although not at the point of occlusion. Again—

and this is very important,—under certain circumstances the influences that cause thrombosis may also cause embolism. A state of the blood, inclining it to coagulate, may cause a clot to form, not in an artery, but in the heart, especially when there is valvular disease, and the clot may be detached. We shall presently see the importance of this consideration.

In embolism the source of the plug is between the pulmonary capillaries and the artery obstructed, and it usually comes from the left side of the heart, from a diseased valve, or from a clot in one of the cavities of the heart (especially from the auricular appendix), rarely from the aorta or the lungs. The valvular disease that is most frequently the source of a plug is mitral constriction, perhaps because the slow flow during the diastole favours the collection of corpuscular vegetations on the valve, and the rapid flow during the systole of the auricle favours their detachment. When the endocarditis is septic, and the vegetations contain organisms, the obstructing plug is apt to excite similar inflammation in the brain; and if the endocarditis is virulently septic, or the plug comes from a septic focus in the lungs, the inflammation in the brain may go on to suppuration.

The arterial disease that causes thrombosis is chiefly, as I have said, atheroma or syphilitic disease. The latter occurs at all ages at which acquired syphilis is prevalent; it is rare from the inherited disease. The atheroma is essentially a senile change, but its occurrence is facilitated and hastened by Bright's disease, and some other influences. It increases in frequency as life advances. Both atheroma and syphilitic disease of the arteries are sometimes symmetrical, and may thus give rise to symmetrical lesions in the brain, and symmetrical symptoms in the limbs. Remember that traumatic injury, such as a fall on the head, or a blow, may cause arterial disease, which may lead to thrombosis long after the injury, perhaps after it has been long forgotten.

Thrombosis may occur, not only in the arteries of the brain, but also in the cerebral sinuses and veins. That in

the sinuses is an extremely grave affection, not only by reason of its effects, but also because it is generally due to disease that is in itself most grave, and of which the thrombosis forms only a terminal incident. Its causes are of two kinds. The first is general disease, favouring clotting by changing the quality and retarding the flow of the blood. This form occurs with especial frequency in young children, and occasionally in very old age. Now and then it occurs at the end of phthisis, after childbirth, or in the course of cancer. In the young, its most common cause is exhausting diarrhœa, or some other prostrating disease, such as an acute specific. The sinus affected in these cases is almost always the superior longitudinal. I drew your attention, in an early lecture (p. 41), to the conditions of the circulation in the veins and sinuses, that render a general cause so effective. The second great cause of thrombosis in the sinuses is adjacent disease. Almost any sinus may be thus affected. The disease is often in the bones of the ear or of the skull; sometimes it is outside the skull, as erysipelas of the scalp. This form occurs with nearly equal frequency throughout life, but it is rare at those periods at which the primary thrombosis is most frequent—infancy, and extreme old age. Inflammation of the wall of the vessel usually precedes coagulation of the blood within it. You may remember that I drew your attention (p. 43) to the communications between the sinuses and the veins of bones and exterior of the skull, which permit the ready extension of morbid processes.

The clot that forms in a sinus often extends into the veins. The obstruction of a vein causes intense hyperæmia of the part from which the blood should be removed by the vein; the capillaries rupture, and the minute extravasations that result may occupy almost all the area of the affected region. Some softening results, but there is not the wholesale softening that is produced by arterial occlusion.

In text-books on Medicine you will find very little about thrombosis in the cerebral veins, as distinguished from that in the sinuses. The condition is rarely seen post-mortem, but it is now and then met with. I believe,

however, that it is both a frequent and an important lesion, and I must digress for a few minutes to give you the grounds for my belief. The condition is rarely seen in the post-mortem room, because, if a patient dies, the clot extends into the sinus, and the case is regarded as one of thrombosis in a sinus. Thrombosis, limited to a vein, although it may be serious in its effect on the brain, is not a lesion that involves much danger to life. The patients recover, and we can only infer the occurrence of the lesion from the character of the symptoms. I have seen it post-mortem in phthisis, and it has been observed in other conditions of prostration. It is occasionally met with in the course of tubercular meningitis, and may, I believe, occur when there is meningeal tubercle, but no inflammation. When it has been met with as an isolated lesion, one or two large veins over one hemisphere have been found occluded. The symptoms that suggest its occurrence are most frequently met with in children who are suffering from debility, exhausting diseases, especially acute specific diseases, or after blows on the head. Now and then they occur in very young children without any exciting cause; and I have observed that such cases, like infantile spinal paralysis, frequently occur during the hot season of the year.

The symptoms are these:—young children are attacked with hemiplegia, and recover, usually with some persistent weakness in the arm, often with mobile spasm in the limbs,—"infantile hemiplegia," or "infantile spastic hemiplegia," the cases are termed. The onset is often attended with severe convulsions, and convulsions may recur, even through life, affecting chiefly the paralysed limbs, and constituting a distinct variety of epilepsy.* We have already seen, in the lecture on the local diagnosis, that such symptoms indicate a cortical lesion. The conditions under which this hemiplegia comes on make it almost certain that the lesion is thrombosis. Thrombosis in arteries is a very rare post-mortem lesion in childhood, far more rare than is combined

* See "Epilepsy and other Chronic Convulsive Disorders," p. 127, for an analysis and description of this form.

thrombosis in sinuses and veins. The initial symptoms in these cases resemble closely those that have been observed in the rare cases in which a vein of the cortex is occluded in an adult. When children die with such symptoms as I have mentioned, we commonly find thrombosis in the longitudinal sinus, and veins opening into it. As I have said, the thrombosis probably extends into the sinus, and into the veins of the other hemisphere, during the final period. From these considerations it seems to me practically certain that, in the cases that do not die, there is thrombosis in one of the surface veins that ascend to the longitudinal sinus, in which, for the reasons I gave you, the circulation is so slow, and the conditions are so favourable to coagulation. When such cases die in later life, the appearances are in perfect harmony with this view. We do not find any cavity, such as we do find when an artery has been occluded by embolism in childhood, and the subject dies in adult life. We see that in a certain area of the cortex the convolutions are shrunken and indurated—exactly the state that we should expect from the appearances presented by recent cases, and very unlike that observed in cases in which we are sure that there has been occlusion of an artery.

These cases are not at all infrequent. They constitute the majority of cases of sudden cerebral disease in childhood. The hemiplegia may be transient, when the lesion is near, but not in, the motor part of the cortex; and probably, in early infancy, the initial hemiplegia, if slight in degree, may altogether escape observation. Most cases of epilepsy dating from infancy, in which the fits are one-sided, are of this character. We can very often trace slight indications of hemiplegia, in trifling weakness, slight shortening of the limbs, or in a slight degree of the mobile spasm that is so marked in the more severe cases.

There is another frequent form of infantile cerebral palsy about which you will also find little in your manuals. In this form, paralysis of one side, or more commonly of both sides, is what is called "congenital," that is, the child

comes into the world with it. The affection, like most others of a congenital character, is popularly attributed to "maternal impressions," to some emotional disturbance that the mother endured during her pregnancy. The designation is not altogether inapt, for the actual mechanism is a maternal impression, although it is one in which the mother is the agent, and not the subject. The cause is the injury the child suffers during its passage into the world. The affections are most common in first children, and after lengthy labours, and they are more common when the presentation is unnatural than when the head presents. They occur in forceps cases also, but probably the effect is due much less to the instrument than to the conditions that render instrumental aid necessary. The chief pathological mechanism is meningeal hæmorrhage, compressing certain parts of the brain. In children who die, there is usually a thick layer of blood over part of the cortex. The clot often lies over the motor convolutions, and may extend from the longitudinal fissure to the fissure of Sylvius. There is sometimes extensive sub-tentorial hæmorrhage from laceration of the cerebellum, but the blood over the cerebrum seems generally to come from the meningeal vessels, and not from the brain-substance. The child often suffers from convulsions during the first few days of life, and the limbs may even then be observed to be motionless. After a time some movement returns, and the ultimate condition is a combination of palsy and spasm, with spontaneous movements, and an irregular choreoid inco-ordination, constituting what has been variously termed "double spastic hemiplegia," or "bilateral athetosis," or "congenital chorea." The irregular movements are chiefly marked in the arms; in the legs the spasm is chiefly tonic, extensor, but with occasional flexion of the knees, especially on an attempt to walk, and often there is some contracture of the calf-muscles. In many cases there is mental defect, which may amount to actual idiocy. The children do not begin to walk until late, often not until the age of six or eight, and their walk is always peculiar. Adductor spasm in the legs may cause a "cross-legged" progression. You may

sometimes recognize the unfortunate subjects of this defect in the streets by their peculiar gait. In addition to these motor limb-symptoms, there are often symptoms of paralysis of the muscles of the neck. The children are unable to keep the head up during the first two or three years of life. I have observed this neck-palsy chiefly in cases of foot or breech presentation. We do not know whether it is due to injury to the bulbar nerves (as the spinal accessory), or to their compression by extravasated blood, or to damage from the inflammation excited by such extravasation. It is conceivable that the anterior pyramids of the medulla may be directly injured, and that this may cause some of the limb-symptoms; but this is on the whole improbable, since hæmorrhage over the cerebrum and about the medulla may coincide. The symptoms sometimes suggest that the inflammation about the medulla closes the openings into the fourth ventricle, and that internal hydrocephalus may thus be produced. Occasionally the legs are affected in much greater degree than the arms, perhaps because the hæmorrhage is situated over the upper part of the motor convolutions (leg-centre), or these may even be directly damaged when the edges of the sagittal suture greatly overlap. Such cases have been termed "congenital spastic paraplegia." Again, the tongue and lips may be weakened out of proportion to the other parts, and difficulty in deglutition and in speaking may persist through life. Some of you saw such a case a few days ago in my out-patient room. This bulbar palsy may be the result of damage to the bulbar nerves, such as I have just mentioned, or it may be due to symmetrical hæmorrhage over the lower parts of the motor convolutions, which may produce symptoms closely resembling those that are caused by disease of the medulla oblongata.*

Inflammation of the brain, except from injury, or in the form of abscess, is so rare, that we may leave it out of con-

* The relation of these symptoms to injury during birth was pointed out by Dr. Little in 1862. Some valuable facts relating to the question have been recently published by an American lady, Dr. McNutt ("American Journal of Medical Science," January, 1885).

sideration. The chief form of intracranial inflammation is that of the membranes—meningitis. Inflammation of the dura mater is rare, except from injury; the variety of chief medical importance is the inflammation of the pia mater, or pia arachnoid, since both the softer membranes usually suffer together.

Meningitis may be simple, purulent, tubercular, or syphilitic. It may affect chiefly the convexity, or chiefly the base, or may be still more partial. Simple meningitis is rare except from exposure to the sun, and then it is seated over the convexity of the brain; but it occurs in limited extent from local disease of various kinds, such as tumour, or softening that is situated near the surface. Purulent meningitis is due generally to suppuration elsewhere, either adjacent to the membranes, as in caries of the bones of the skull, or disease of the internal ear. Distant suppuration produces it chiefly by the mechanism of pyæmia. It may attend the septicæmia of the puerperal state. Occasionally it occurs in the course of acute general diseases, and in children it may develope when no condition can be discovered to which it can be secondary. Purulent meningitis is often general, affecting both the base and the convexity, and in children the spinal membranes also.

The most common form of meningeal inflammation is the tubercular. It occurs in childhood with especial frequency, generally in families in which there is some tubercular taint. In adults it occurs chiefly in those who are actually suffering from tubercular disease of the lungs, but in youths of both sexes it may be primary. The membranes of the base are usually affected far more than those over the convexity, and the most characteristic symptoms of the disease are due to the damage to the cranial nerves. Remember that tubercle of the membranes is not quite the same thing as tubercular meningitis. Tubercle may exist without inflammation, and probably always precedes inflammation, which may be excited by some influence that may seem to be the cause of the symptoms. If the membranes were previously healthy, the exciting cause would be without effect. Massive tubercle, constituting a tumour, may be attended with intercurrent tubercular

meningitis, and mixed symptoms of a complex character may result.

Syphilitic meningitis is almost always chronic. It may involve the membranes of the base, or of the convexity, but is often partial in distribution. It consists of a combination of inflammation and growth, and often extends about an actual syphilitic tumour, irritating and damaging the adjacent structures. Remember that the nerves are often damaged by syphilitic disease when there is no actual meningitis. They may be affected by a syphilitic neuritis, an inflammation of the sheath of the nerve; or the sheath may be the seat of a syphilitic growth. Such growths on the nerves are sometimes symmetrical; the same cranial nerve may thus be paralysed on each side.

Almost any kind of tumour may occur in the brain, but some, as fatty tumours, which are common elsewhere, are extremely rare in the brain, while others, as glioma, are almost confined to the cerebral tissue. The most common intracranial growths are tubercular and syphilitic; next comes glioma, then sarcoma, and then cancer. Other forms are rare. Tubercular tumours are most frequent during the first twenty years of life. Syphilitic growths occur chiefly during the period of active adult life, from twenty-five to fifty. The only influence that ever seems to *excite* the development of a tumour is injury; this more commonly causes a tumour springing from the membranes than one beginning in the brain-substance. Growths that spring from the dura mater usually compress the brain-tissue. Those that spring from the pia mater, or develope within the brain, may either compress or invade the cerebral substance. The characteristic of glioma is its invasive tendency. It developes in the interstitial tissue of the brain, and encloses and destroys the nerve-elements. It enlarges the part of the brain in which it occurs, and there is no sharp line of demarcation between the tumour and the brain-substance. On the other hand, most other tumours, and among them tubercular, syphilitic, and sarcomatous growths, do not

invade the brain-tissue, in the strict sense of the word; it perishes before them, partly by the compression they exert, partly in consequence of the inflammatory irritation that they produce, and which is shown by the zone of softening that usually surrounds them. In each case, however, the functional impairment of the cerebral tissue does not always correspond to the apparent structural damage. The nerve-fibres that are compressed before the growth in the one case, or are included in the growth in the other case, may retain some functional power, and the symptoms of loss of function may be much less than might be anticipated from the position and the extent of the lesion. Again, the irritation that is caused may be greater in degree, and wider in range, than the size of the tumour would suggest. I mentioned this fact to you on a former occasion, and remind you of it now on account of its importance. Lastly, you should know that one form of tumour, glioma, is occasionally the seat of a sudden hæmorrhage.

Abscess of the brain is usually due to injury to the brain, to adjacent suppuration, especially bone disease, or to suppuration at a distance, especially in the lungs. Curiously, it is rare in general pyæmia. Ear disease is its most common cause. It occasionally results from disease of the orbit or of the nose. The abscess may be in any part of the brain. Caries of the middle ear usually causes abscess in the temporo-sphenoidal lobe, rarely in other adjacent lobes. In caries of the mastoid cells the abscess is often in the cerebellum. The symptoms produced are frequently slight in proportion to the size and position of the abscess, and it is common for the disease to run an almost latent course until acute symptoms arise. These are often due to rupture, which may occur into the ventricles, or into the membranes, and its symptoms are of extreme gravity, quickly fatal.

Did time permit, I might with advantage point out to you many other facts of etiology and pathology that aid us in our diagnostic effort. Some of them I may mention in the

description of the method of diagnosis to which we shall pass in the next lecture. But there is one general law that I must mention before I leave this part of the subject.

Whatever be the morbid process that causes damage to the brain, the symptoms are due to the latter; and the changes in the nerve-elements are much the same, whatever be the nature of the morbid process that causes these changes. We do not recognize this in our mode of speaking of these diseases. We speak, for instance, of "syphilitic disease of the brain," but the damage to the nerve-elements is never syphilitic. The syphilitic disease is outside them, sometimes altogether away from them, and it causes in them simple processes of degeneration, etc., on which the symptoms depend. This is not a mere theoretical refinement; it is of great practical importance, especially in regard to prognosis, and occasionally important in diagnosis. We shall see that we sometimes look to the result of treatment—to the effect of the administration of "anti-syphilitic" drugs—to confirm our diagnosis. But such treatment has no direct influence whatever, so far as is known, upon the changes in the nerve-elements. We may lessen or remove the syphilitic lesion that has produced the changes in the nerve-structures, and then certain kinds of damage will pass away, especially that which results from pressure, as of a growth. Other kinds of damage cannot pass away, as, for instance, necrotic softening, due to syphilitic disease of the wall of the vessel. You may remove this disease, but you cannot restore the brain-tissue that is dead, and no more recovery is possible for it than in a corresponding case of embolism. Symptoms may pass away in each case, but only when they are "indirect" in nature; and "direct" symptoms are as lasting in one case as in the other. Hence, before we can take the absence of improvement under treatment as an indication that a disease is not syphilitic, we must consider whether the change in the nerve-elements is one from which recovery is possible when the cause is removed. Speaking generally, the more quickly the changes are produced (and the symptoms developed), the less

evidence of a similar lesion elsewhere in the system. A third is the position of the disease: certain morbid processes are more common than others in certain parts of the brain. A fourth is the character of the symptoms themselves, which may vary somewhat in the same part of the brain, according to the nature of the disease: this variation is slight at the onset, and is chiefly available when the course of the disease can be ascertained or watched. Lastly, other symptoms of the morbid process outside the nervous system may indicate its nature,—such as the pyrexia that attends inflammation.

The first question, then, is the time occupied by the onset of the symptoms—whether the onset is sudden, acute, or chronic. By "sudden" I mean the development of the symptoms to a considerable degree in from a few minutes to a few hours; by "acute," an onset that occupies from a day to two or three weeks; by "chronic," one that occupies more than a month. We are thus able to form a rough classification of the lesions into three divisions—the first comprehending the vascular lesions; the second, the inflammatory lesions; the third, the chronic inflammations, the growths, and the degenerative lesions. Cases of intermediate course are met with, but they can usually be brought without difficulty into one or other of these types, to which it is convenient to limit ourselves, not only for the sake of simplicity, but because they include nineteen-twentieths of the cases of organic disease. In thus using the mode of onset as a means of classification, we must take care that it is the actual onset that we consider. We shall have also to notice certain exceptions to the rules laid down—exceptions that are, however, rather apparent than real.

We may consider, first, the group in which the symptoms are sudden in their onset, developing often in a few minutes, sometimes occupying a longer time—a few hours. Remember, as a useful working rule, that symptoms of sudden onset, due to an organic cause, indicate a vascular lesion. They indicate the rupture of a vessel or the obstruction of a vessel; the former, as we have seen, causing hæmorrhage, the latter softening. The exceptions to this rule are few. They include the transient paroxysmal symptoms, such as convulsion or

pain, and also inhibitory palsy; but the last is very rare. Again, an abscess of the brain may cause sudden symptoms by its rupture. This is the most important exception to the rule that sudden symptoms indicate a vascular lesion; but even here there are often chronic symptoms before the sudden symptoms that attend the rupture. Moreover, the symptoms that result from the rupture of an abscess are more often acute than sudden, according to the definition that I gave you of the sense in which we use these words.

We will take, then, the symptoms, such as paralysis, loss of sensation, etc., the sudden onset of which justifies us in suspecting the occurrence of a vascular lesion—softening or hæmorrhage. We have not merely to decide between the two, but we have, if the lesion is softening, to determine its form, that is, the nature of the obstruction that has caused it. The softening may be from the occlusion of an artery or of a vein. I told you, in the last lecture, the pathological causes of the various forms of obstruction, but it may be well for me to put them before you again in the form of a table.

A. Hæmorrhage—from rupture.
B. Softening—from obstruction,
    (a) in an artery—from
        (1) local thrombosis, due to
            (*a*) atheroma,
            (*b*) syphilitic disease,
            (*c*) blood-state;
        (2) embolism,
    (b) in a vein—thrombosis.

The actual onset may be preceded by premonitory symptoms. The significance of the presence or absence of these is limited. They are confined to those lesions that result from considerable preceding vascular disease—to softening from atheroma and syphilitic disease of vessels; they are absent in softening from embolism and in simple thrombosis. In ordinary hæmorrhage, premonitory symptoms, as such,

are also absent. The miliary aneurisms cause no symptoms until they burst. Any premonitory symptoms in hæmorrhage are due, not to the cause of the hæmorrhage, but to coexisting atheroma, and have no direct relation to the lesion. In syphilitic disease and atheromatous softening, the premonitory symptoms are transient weakness or tingling in the limbs subsequently paralysed, due to the diminution of the blood-supply afterwards arrested; and in each case there may be headache. In larger aneurisms, previous symptoms are those of a pre-existing lesion, causing pressure on some structure at the base of the brain, and are not true premonitory symptoms. The presence of premonitory symptoms may be of diagnostic importance, but their absence is of very little significance, since there may be none even in the diseases that are most often attended by them.

The actual onset is the next element in the diagnosis. The indication that it affords must ordinarily be used in subordination to the causal indications. Nevertheless it is important to consider it first, because it does occasionally afford a very strong indication, so decided as to override every other consideration. The chief guiding point is the effect on consciousness—the occurrence of apoplexy, its degree and duration. Either softening or hæmorrhage may cause apoplexy, but it is produced by hæmorrhage more readily than by softening. Deep coma, lasting many days, is very rarely caused by vascular obstruction. On the other hand, it is not common for there to be no loss of consciousness in hæmorrhage, unless the extravasation is very small in size. Hence the absence of this symptom is not of much weight unless there is reason to think that the lesion is of large size. All that can be said is, that if there is complete hemiplegia lasting for some days, and no unconsciousness at the onset, the lesion is more likely to be softening than hæmorrhage; and if there is deep and prolonged coma, the lesion is almost certainly hæmorrhage, unless the patient is very old, and then there is some probability of softening, even with prolonged coma. If focal symptoms are slight, and initial apoplexy was well marked, hæmorrhage is much more likely than softening.

When the onset occupies several hours, the symptoms developing gradually without initial loss of consciousness, we must wait for the termination of the onset. If it ends in deep coma (ingravescent apoplexy), there is almost certainly hæmorrhage. If it ends in a stationary condition, without any loss of consciousness, the lesion is probably softening.

We must consider, next, the causal indications. They should not be taken in subordination to the mode of onset, but independently. The two should then be compared, their indications balanced, and their relative weight estimated. The first causal indication is the age of the patient. Hæmorrhage is so rare under forty, that it would not be suspected unless deep coma strongly suggested it. Softening from atheroma is still more rare under forty. This renders the diagnostic problem far more simple during the first half of life, since there remain only three common vascular lesions—embolism, thrombosis from syphilitic disease, and thrombosis from blood-states. We may conveniently, therefore, limit ourselves, in the first instance, to the cases that occur during this period of life. In each case we must begin by searching carefully for any indications of the presence of one of these three causes, always remembering, however, that hæmorrhage, though not probable, is possible, even in childhood.

In the last lecture we considered the chief causes of these lesions, and I told you that embolism, in the vast majority of cases, is from the heart, and is associated with valvular disease. The first point to be ascertained is therefore the presence or absence of such disease. In recent cases you are only justified in suspecting embolism, in the absence of valvular disease, when there is some other condition which may be the source of a plug (such as an aneurism of the aorta). But if the diagnosis has to be made a year or more after the onset of the hemiplegia, we cannot exclude embolism because we find no indications of valvular disease of the heart, if the onset occurred during, or soon after, such an illness as is usually, or occasionally, attended by endocarditis—acute rheumatism, chorea, or scarlet fever. I have

known, for instance, mitral endocarditis during chorea to cause cerebral embolism, and a year later the heart had so far recovered that no evidence of valvular disease could be recognized.

There is one exception to the rule that valvular disease in a young person makes it probable that the lesion is embolism. Valvular disease may lead to cerebral hæmorrhage by causing an aneurism. I told you in the last lecture that the imperfect obstruction of an artery by embolism is one of the most common causes of intracranial aneurism. Hence, if the initial apoplexy is deep and prolonged, or if there is the "ingravescent apoplexy" already mentioned, the presence of heart disease not only does not exclude hæmorrhage, but increases its probability. This is true, even in children.

In thrombosis from blood-states alone, the causal indication is usually obtrusive. In adults this influence is almost confined to the post-puerperal condition. The clot may be formed *in situ*, or, if there is valvular disease, it may be formed in the heart, and carried to the brain. In other conditions favouring coagulation, such as phthisis and cancer, the clot generally forms in a vein. This is true also of the thrombosis that occurs so readily in young children, of which I spoke to you in the last lecture. The fact that the patient is very young and does not suffer from heart disease, or that the attack occurs during a state of considerable prostration, justifies a diagnosis of thrombosis; and if the symptoms are those of a cortical lesion, you may feel confident that the thrombosis is in a vein.

Thrombosis in an artery, from syphilitic disease of the wall of the vessel, occurs from six months to twelve years, or even more, after the primary disorder. In many cases there is a history of syphilis, and no other cause of arterial obstruction can be traced. We may then feel sure that a sudden cerebral lesion is due to this cause. If, however, we have no history of syphilis—as, for instance, in the case of a patient who is unconscious, and unable to give us his history,—we have to rely on the indication afforded by the age, and on the absence of other causes. Often we are aided by the presence of pre-

monitory symptoms, such as headache, or slight symptoms in the limbs that are afterwards paralysed. These symptoms are of especial diagnostic importance in cases in which there is heart disease, and the patient has had syphilis. We then have to decide between embolism and syphilitic thrombosis. In the former, as we have seen, premonitory symptoms are absent. Hence, in such a case, any symptoms that suggest that there was a morbid process at work within the cranium before the occurrence of the vascular obstruction, make it probable that this obstruction is due to thrombosis, and not to embolism. For instance, a man who had old-standing aortic regurgitation was seized with hemiplegia. He had suffered from severe headache for a few weeks before the attack. Many years before, he had suffered from constitutional syphilis. The headache that preceded the hemiplegia could not be explained on the supposition that the lesion was embolism, and made it probable that the obstruction was due to thrombosis from syphilitic disease of an artery. Although there was no opportunity of ascertaining the exact nature of his lesion, yet it is probable that the diagnosis was correct, for a few weeks later some nodes appeared upon the skull, showing that the syphilis was still active in his system.

Syphilis, as well as heart disease, may cause cerebral hæmorrhage, and by the same mechanism, by causing an aneurism. Hence, also, a history of syphilis does not lessen the probability of hæmorrhage, if this is suggested by the character of the symptoms.

In the second half of life, the diagnostic difficulty is far greater. The causes operative during the first half continue during the second (although they become less effective as life advances), and, in addition, the potent mechanisms of arterial degeneration come into operation. Thrombosis from atheroma, and hæmorrhage, increase in frequency up to extreme senility; then, thrombotic softening becomes more frequent than hæmorrhage, and is more probable if other indications are equally balanced.

The most important indications in this period, are those drawn from the state of the vascular system. Arterial ten-

sion and a strongly acting heart suggest hæmorrhage; a soft pulse and an irregular, feeble heart suggest softening. If these indications are strongly marked, their significance is very great. Mere arterial degeneration tells us little. It tells us that the cause of softening exists—atheroma; but this so often coexists with the miliary aneurisms that cause hæmorrhage, that its significance is small. All that can be said is, that if other indications are equally balanced, considerable degeneration in the accessible arteries renders cerebral softening rather more probable than cerebral hæmorrhage. As we have seen, while atheroma is almost invariable in senile softening, it is absent in about a quarter of the cases of hæmorrhage. Advanced Bright's disease causes both atheroma and miliary aneurisms. It may thus be a cause of either softening or hæmorrhage. The former is, however, a rather more frequent consequence than the latter, perhaps because the miliary aneurisms attain an effective degree earlier than does atheroma, unless the patient has reached the period of senile degeneration. Hence, if other indications are equal, and the patient is not very old, Bright's disease suggests hæmorrhage; but if the other symptoms are such as to suggest softening, their significance is not appreciably lessened by the presence of Bright's disease.

As I have said, the difficulty in the diagnosis during the second half of life is increased by the circumstance that the vascular lesions common in earlier life—embolism, and thrombosis from syphilitic disease—occur during this later period. Thrombosis from the state of the blood and circulation also occurs, but the powerful influence of the puerperal state is no longer effective, and the influence of the circulation in causing thrombosis becomes to a large extent merged in that of the arterial degeneration, since it is rarely effective alone. On account of the frequency of the senile lesions, the mere presence of a cause of one of the earlier lesions,—a history of syphilis, or a source of embolism,—is of far less diagnostic significance than it is during the first half of adult life. The diagnosis rests, therefore, in larger degree on other indications than those of a present cause. If such other

indications are absent, the diagnosis is a matter of probability only, or rather, I should say, of probability that is never high. All diagnosis that is founded on reasoning, and not on simple observation, is a matter of probability. Where several and different indications coexist, the degree of probability is often very low. The indications that guide us correctly in two cases may fail us in the third. A woman of fifty-five died with symptoms of a vascular lesion. The age of the patient made it probable that the lesion was either hæmorrhage, or softening from atheroma. Of the two, the symptoms suggested the latter. But the patient also had considerable mitral obstructive disease, and this made embolism a possible lesion. After death there was found, as expected, softening and not hæmorrhage; but the softening was due to syphilitic disease, extensive and characteristic, of the likelihood of which we had no suggestion from the patient's history. Dr. Hughlings-Jackson has recorded a case in which atheroma and syphilitic disease coexisted, and softening that had occurred was due only to the former. These cases illustrate the difficulty in diagnosis that results from the persistence during the senile period of the causes of vascular disease that are operative during the first half of life; and in such the diagnosis must usually be a matter of probability only, and of very low probability, and a conclusion, rightly reached, may unavoidably be wrong.

The second element in the causal indication is the evidence of a lesion elsewhere, of the same nature as that within the brain—evidence that a morbid process is at work in the system, such as may be the cause of the cerebral lesion. Most of the vascular lesions may occur outside the brain, and we may sometimes detect them. If they occur at the same time, or about the same time, as the cerebral lesion, they afford strong evidence of the nature of the latter. The occurrence elsewhere of the process of embolism may be recognized; as, for instance, in the spleen, by the enlargement and tenderness of the organ. The central artery of the retina may be obstructed by embolism, thus affording evidence of this process

in the arterial system to which the cerebral vessel belongs. If, as I have seen, the retinal artery is occluded at the same moment as the cerebral artery, the evidence of the nature of the lesion in the brain amounts almost to demonstration. In syphilis we rarely have a more special indication of the precise process at work in the brain. Neither syphilitic disease of vessels, nor thrombosis therefrom, is to be recognized elsewhere, except in the rarest cases; but other indications that the syphilitic virus is still active may furnish equivalent evidence. Simple thrombosis, from blood-state, is occasionally accompanied by a similar accident in some other part, especially during the puerperal period. A clot may form in a vein of the leg, or some other part. Arterial or venous thrombosis elsewhere, from combined arterial disease and feeble circulation, coincides not unfrequently with cerebral thrombosis, and affords presumptive evidence of the nature of the lesion of the brain. Instances are senile gangrene of the foot, thrombosis in the veins of a leg or of the retina in those who are old and gouty, or feeble and depressed. Hæmorrhage elsewhere is rare in cases of cerebral hæmorrhage, except in the eye, and even there is significant only when the extravasation is large. The small hæmorrhages in the retina, that are so common in albuminuria, do not signify more than that the kidneys are diseased, and afford alone no more evidence that the cerebral lesion is hæmorrhage than does the renal affection itself. The significance of this we considered before. A large intra-ocular hæmorrhage, such as one into the vitreous, makes it highly probable that the cerebral lesion is of the same nature. But extravasations elsewhere, in the course of a general disease that is attended with a hæmorrhagic tendency, such as pernicious anæmia or leucocythæmia, also afford strong reason for believing that a cerebral lesion is of the same nature.

The position of the lesion occasionally helps us. A lesion of the pons, and still more of the medulla, that is not quickly fatal, is much more likely to be softening than hæmorrhage. The basilar artery is a frequent seat of syphilitic disease, and

if other indications are equally balanced, in a case in which the symptoms suggest an obstruction of the basilar, as, for instance, the presence of heart disease and a history of syphilis, the fact that the basilar is the affected artery would turn the scale in favour of syphilitic thrombosis, against embolism. If the symptoms point to a cortical lesion, and other indications are equally balanced, this is much more likely to be softening than hæmorrhage. The sudden extension of the symptoms from one side to both, with renewed coma, that is produced by the rupture of an extravasation into the ventricles, constitutes evidence that the lesion is hæmorrhage, and not softening; but this significance is only decided when the extension occurs within a few days of the onset. Even then it is not a certain indication. Identical symptoms may attend the formation of a clot in a large artery of the other side of the brain in cases of softening, and if such symptoms come on after the first few days, it is even more likely that they are due to a fresh lesion than to the extension of the old one, and they therefore no longer have a definite significance. Hence it also follows that when there is decided reason to believe that the original lesion was softening, the occurrence of symptoms like those of ventricular hæmorrhage does not materially weaken the original diagnosis.

The character of the symptoms, in relation to the position of the lesion, does not give us much help in the case of acute lesions. Convulsions at the onset are of little significance. Those that succeed the onset, or occur during the subsequent course of the disease, are in favour of the lesion being softening, rather than hæmorrhage. An intense degree of secondary irritation is in favour of softening. So also are the athetoid movements, "mobile spasm," that may come on after the hemiplegia has existed for some months.

Here, for to-day, gentlemen, we may conveniently stop. There remains for consideration the diagnostic significance of the symptoms that attend the acute and the chronic lesions.

But the chief acute lesion, according to the classification that I gave you at the outset, is meningitis. I propose, after we have completed our survey of the chief diagnostic indications from the side of the symptoms, to review them from the side of the lesions. It will be convenient to postpone the consideration of the symptoms of meningitis until we come to it in that survey. The symptoms that may result from chronic lesions we will proceed with at our next meeting.

LECTURE XVII.

DIAGNOSIS OF THE NATURE (*Continued*): CHRONIC LESIONS—SYMPTOMS IN THE SEVERAL MORBID PROCESSES.

Gentlemen,—The chronic lesions of the brain are chronic meningitis, tumour, aneurism, abscess, disseminated sclerosis; and we may include among them labio-glossal paralysis and general paralysis of the insane. In all these diseases well-marked symptoms develope, as a rule, slowly, occupying at least several weeks, and often several months, in their progress to a considerable degree of intensity. It is true that one of them (tumour) occasionally, and two of them (abscess and aneurism) frequently, have a latent course, and then give rise to acute and even sudden symptoms. But as pathological processes, they are chronic, and the symptoms of their development, when these exist, are correspondingly chronic in their evolution.

The first point to be considered in the diagnosis of these diseases is the causal indication that can be discovered. This element is rarely of much significance; it is far less important than it is in the sudden lesions of the brain.

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tically confined to adult life; aneurism and disseminated sclerosis being sometimes met with in youth. Tumour and abscess occur in all ages. Sex helps us only in so far as general paralysis is rare in females. Heart disease is a cause of aneurism alone among the chronic lesions. Syphilis may cause tumour, chronic meningitis, or aneurism. It is by far the most frequent cause of cerebral tumour in adult life. Chronic meningitis is very rare as a solitary lesion, and is due usually either to injury, chronic alcoholism, or to syphilis. In the absence of these causes it is very improbable. Injuries to the head may also cause abscess, or, in rare cases, tumour. The most common other causes of abscess are adjacent bone-disease, especially in the ear, and suppuration elsewhere, especially in the lung. If these are not present, abscess is unlikely. Tumours are usually primary, and independent of growths elsewhere. Cancer is occasionally secondary, and hence a malignant tumour in some other part of the body renders it highly probable that symptoms of organic disease of the brain are due to a tumour. Tubercular growths in the brain are also occasionally secondary to tubercular disease elsewhere, but are more often primary, occurring in a subject predisposed by inheritance to tubercle. Hence either hereditary predisposition or actual tuberculosis suggests that a chronic disease is a tumour.

Neurotic heredity, indicated by a history of such diseases as epilepsy, insanity, neuralgia, etc., is rather against than in favour of actual organic disease of the brain. Equivocal symptoms are more likely to be due to functional disease. Preceding anxiety, or mental shock, also render such disturbance more likely than coarse organic disease.

The diagnosis of the chronic lesions of the brain depends chiefly on the symptoms they produce. You doubtless remember the distinction between the diffuse and the focal symptoms. The diffuse symptoms, if any are present, are of much greater importance in the pathological diagnosis of chronic disease than are the focal symptoms, and we may, therefore, consider them first. If the symptoms are chiefly

focal, we have to depend on their mode of development, and on their conformity to certain types. An instance of the latter is the case of labio-glossal paralysis, in which diffuse symptoms are entirely absent, but the focal symptoms correspond closely in all cases.

Of the diffuse symptoms we may consider, first, pain in the head. Headache is a conspicuous symptom in chronic meningitis, in tumour, and often in abscess. It is absent in the purely degenerative diseases. In aneurism it is sometimes marked, sometimes it is trifling, occasionally it is absent. Remember that the characteristics of the pain of organic disease are severity and constancy, and the association with other symptoms. One of these is vomiting, which occurs in the same lesions as headache, and often coincides with the more intense paroxysms of pain. Of even greater significance is the association with optic neuritis. Rare in aneurism, optic neuritis is frequent in abscess and chronic meningitis, and most frequent and intense in tumour, in which it occurs, at some period, in five-sixths of the cases. If there is no cause of abscess to be discovered, persistent headache, and considerable optic neuritis, may be regarded as almost certain indications that the organic disease is a tumour. But remember that the optic neuritis of tumour is not always intense. In cases of slowly growing tumour, it may be slight in degree, and extremely chronic in course. Atrophy of the optic nerves varies in its significance according to its form. "Consecutive atrophy," that which follows neuritis, has the same significance as the neuritis that precedes it. Simple atrophy is rare except in the degenerative diseases, disseminated sclerosis and general paralysis of the insane. Its significance is always that of a degenerative process. Atrophy, that *follows* loss of sight, is due to damage to the optic nerve, and indicates either a tumour pressing on the nerve, or inflammation involving it. Loss of the reflex action of the pupil to light, without loss of sight, has the same significance as primary atrophy; it is due to degeneration, and indicates that a degenerative process is at work. It affords a ground for suspecting that other symptoms are due to a similar degeneration. It does not prove this. A

coarse disease may coincide with a degenerative process. Such coincidence is now and then observed in syphilis, which predisposes to degenerations even during the stage in which it still causes organic lesions. Nystagmus occurs in tumour and in some degenerative diseases, but is not common in either class, except in disseminated sclerosis; in this it is a very frequent symptom.

Mental change may have various meanings, according to its form. Exaggerated delusions occur chiefly in general paralysis of the insane, but the early stage and slighter form of this disease are often attended by a simple optimism, in which, without any false idea, all things are looked at in a favourable light; the patient is happy under depressing circumstances, and is always "better" or "well" when physical weakness is steadily increasing. An unnatural complacency, without actual optimism, is also common in disseminated sclerosis. It is often marked, even in the early stage, and is a significant symptom that should always attract attention. This form of mental change is confined to these degenerative diseases, and is not met with in other organic lesions. Chronic delirium, sometimes active but amenable, may occur in chronic meningitis and tumour; occasionally the delirious condition resembles closely that of chronic insanity. More common in these diseases is simple mental failure—loss of memory, and slowness of speech—deepening to lethargy. In a considerable degree of this condition, fæces and urine are often passed without notice, although there is no paralysis of the sphincters. I have more than once mentioned to you the importance of this symptom.

Convulsions are absent in disseminated sclerosis, and also in most cases of aneurism, until rupture occurs. They are common in tumour and in chronic syphilitic meningitis, but not in alcoholic meningitis. They occur also, although not very frequently, in general paralysis of the insane. In each disease in which they occur, the convulsion may be general or partial, the latter form being, as you know, a focal symptom. Slight partial fits, recurring with great frequency, are almost confined to tumour.

Certain focal symptoms need special consideration, because they characterize certain forms of cerebral disease. Defect of articulation is produced by any disease of the medulla and pons, but it is also an early and characteristic symptom in the degenerative diseases — sclerosis, general paralysis, and labio-glossal paralysis. In the latter the defect is dependent on actual loss of power in the lips and tongue, which can be recognized as soon as there is much impairment of articulation; but in the two former diseases, sclerosis and general paralysis, there is at first no distinct weakness. The alteration in the early stage may be very similar in the two—a tendency to clip words, to run together syllables, which are thus confluent, instead of being "articulated." As sclerosis advances, however, there is usually a tendency to a separation of syllables. The staccato and elisive defect may coexist (see p. 106). In general paralysis, speech usually becomes hesitating, and is interfered with by manifest tremulous twitching of the muscles of the lips and face; often words are drawled out, and sometimes a guttural noise accompanies inspiration.

Another important group of symptoms are those of the eyeball muscles. Any disease at the base of the brain may paralyse these muscles in one or both eyes, but a slow progressive palsy, involving many muscles of both eyes, without other indications of basal disease, shows a progressive degeneration of the nuclei of the nerves, analogous to that of the bulbar nerves in the labio-glossal palsy.

Of the focal symptoms in the limbs, only one class is of sufficient pathological significance to need mention. Tremor is common in general paralysis, but may occur in tumour, and in chronic meningitis. Jerky inco-ordination of movement, accompanying weakness, is a characteristic symptom of disseminated sclerosis, but it occurs also in cases of tumour, chiefly in children (see p. 66).

We may now review these facts from a different side—from the side of the lesions,—and consider the aggregate

of symptoms which indicate the several lesions, *i.e.*, the symptoms that these lesions produce in typical cases. In doing so I shall have to repeat many things that I have already said. I need not apologize for this, because, gentlemen, you have failed to learn the most important lesson in method of study if you object to repetition. You can only learn thoroughly by going over facts many times in their different relations.

We will commence again with the cases in which the onset is sudden, and take first the conditions in which there is loss of consciousness, apoplexy, but in which there are no indications of a one-sided lesion. There may be no symptoms on either side of the body and limbs, or there may be symptoms on both sides. These symptoms are in the state of the muscles, relaxation or contracture, or in the state of reflex action. We may thus divide these cases into two classes, according to the absence or presence of symptoms of bilateral character.

We will take first the cases in which there are no peripheral symptoms recognizable on either side. A patient may suddenly become unconscious, and remain so, and may not only present no indications of damage to one side of the brain, but there may be such restless automatic movements of the limbs on both sides, as indicate with certainty that there is no damage to the motor tract of either side. In most cases of the kind the coma is incomplete—there is stupor rather than coma. The patient, usually between forty and sixty, is of plethoric aspect, the pulse full, the carotids pulsate strongly, the face is flushed, reflex action is normal, the pupils are of medium size and act to light. In the course of two or three days the symptoms pass away entirely, and no indication of any focal lesion can be found, when consciousness has returned, and a thorough investigation is possible. Such an attack is probably due to cerebral congestion. It is customary to ascribe it to this cause, and the balance of evidence is in favour of the correctness of this opinion. The patient often has more than one attack of this character.

In the second class of cases the apoplexy is accompanied by

evidence of interference with the motor centres on both sides of the brain. There is either complete relaxation of the muscles, or bilateral rigidity, or rigidity in some parts and resolution in others. In these cases we have to deal with a lesion either in or outside both hemispheres or in the pons. There may be ventricular hæmorrhage, meningeal hæmorrhage, obstruction of vessels in both hemispheres, hæmorrhage into the pons, or softening of the pons. If universal resolution and deep coma succeed, after a few hours or a day or two, the following symptoms—mental excitement, rigidity, convulsive movements (now in one part, now in another), and sudden headache—there is probably meningeal hæmorrhage, and this is especially probable if the symptoms succeed an injury. If, in a patient under forty, with heart disease or old syphilis, the coma is complete from the first, the limbs relaxed, and the symptoms continue for some hours without diminution, a cerebral aneurism has probably burst. Headache, giddiness, or palsy of cranial nerves for some weeks or months before the onset, makes this diagnosis still more probable. If initial coma is accompanied by indications of a one-sided lesion, rigidity or resolution of the limbs of one side, deviation of the head and eyes, followed in a few hours or a day or two by similar symptoms on the other side, without special affection of the cranial nerves, but with deep coma, the lesion indicated is ventricular hæmorrhage. If the symptoms are from the first bilateral, if there is rigidity or resolution, or the two are associated in different parts, if there are convulsive movements in both arms or both legs, interference with respiration within two or three hours of the onset, strong contraction of the pupils persisting or yielding to wide dilatation, a rise of temperature to 102° or more within an hour of the onset, the indication is hæmorrhage into the pons. If, however, similar symptoms come on gradually in the course of one or two days, the coma at first incomplete, and slowly deepening, without any early rise in temperature, with irregular affection of the cranial nerves, recognizable before the coma becomes deep, the indication is an occlusion of the basilar artery. Whether this is due to

embolism or thrombosis must depend on the more sudden onset in the former, and on the causal indications. In the one case a source of embolism will be found, and perhaps indications of embolism elsewhere. In the other case these are absent: syphilis may be probable or certain; or the patient is in the degenerative period, with atheromatous arteries and a weak heart.

We will take next the case in which a patient is seized with apoplexy, and there is distinct evidence of a one-sided lesion—relaxation of muscles, loss of the skin reflex, change (loss or early excess) of the muscle-reflex action, deviation of the head and eyes. You are sure that a vascular lesion has occurred, on account of the suddenness of the onset; and unless the patient is a child, or is suffering from phthisis or other cause of extreme weakness, you are sure that it is an arterial lesion—an artery has given way, or become stopped up. For further guidance you examine the pulse, arteries, heart, and urine. At any age, after childhood, deep coma, lasting many hours, renders hæmorrhage probable, the more so if the patient is over thirty-five, and is suffering from Bright's disease. The significance of prolonged coma as an indication of hæmorrhage is much greater in early and middle life than it is in old age, since in the latter it is produced by arterial occlusion much more readily than in the former. Mind, I am speaking of actual coma, not of the mere hiatus in conscious memory, which the patient afterwards describes as unconsciousness. He may tell you of a period of unconsciousness lasting for many days, when what we term manifestations of consciousness returned in a few hours. The indications afforded by the prodromata, onset, pulse, arteries, and heart, I have already described, and need not here repeat, beyond reminding you that embolism is suggested by valvular disease of the heart, especially during the first forty years of life, by a sudden onset without prodromata, by the brevity or absence of initial loss of consciousness, and by the evidence of embolism elsewhere. Syphilitic disease is suggested by a history of syphilis, or, failing this, by its possibility combined with the absence of other causes, by the occurrence

of prodromata (headache, symptoms in the limbs afterwards paralysed), by an onset that is sudden or deliberate, but without, or with only brief, loss of consciousness. Hæmorrhage is suggested by the degenerative period of life,—but is not absolutely excluded even by youth; by deep and prolonged coma,—but is not excluded by brief coma, or even by the absence of any loss of consciousness. It is suggested also by high tension of pulse, a strongly acting or hypertrophied heart, the absence of prodromata, an initial fall of temperature, and the presence of Bright's disease. Softening from atheromatous thrombosis is suggested by the degenerative period of life, and, unlike hæmorrhage, is excluded by youth or early adult age. It is further suggested by a dilated, feeble, and especially an irregular heart, by previous slight attacks of the same nature, by prodromata in the limbs afterwards paralysed, by the brevity of initial coma, and *à fortiori* by the absence of loss of consciousness at the onset.

Simple arterial thrombosis, from the blood-state alone, may be suspected if there is a constitutional state known to favour thrombosis, especially the puerperal state, and if no source of embolism can be discovered, and no cause of arterial disease can be traced.

Venous thrombosis is suggested by previous profound prostration and weakness, and especially by the patient being an adult in the last stage of phthisis, or a child under five years of age, and by the occurrence at the onset of convulsions in, or beginning in, the limbs afterwards paralysed,—convulsions that indicate a cortical lesion.

Sinus-thrombosis is indicated by somnolence increasing to coma, and attended by general convulsions, coming on in a young child prostrated by diarrhœa or some other exhausting disease, or in a patient who has external disease adjacent to a sinus. In the latter case, focal symptoms may be present, which vary according to the sinus occluded. The diagnosis is only certain, however, when external tumefaction near the position of the sinus succeeds the other symptoms.

The indications that I have given you hold good, not only

of hemiplegia, but also of other symptoms of an organic lesion, hemianæsthesia, hemiopia, and the like, which cannot, as a rule, be recognized until initial loss of consciousness has passed away.

We may now pass to the chief acute lesion of the brain—meningitis. The symptoms that should lead you to suspect meningitis are the combination of headache, vomiting without gastric cause, pyrexia, and delirium coming on in an acute manner. Remember that the absence of any one of these is of little negative significance. Headache is, however, seldom absent, and perhaps is never absent at all periods of the case, but is sometimes only trifling at the onset. On the other hand, it is usually severe, and the leading symptom. Remember, also, that the presence of only one of these symptoms is of no significance, and that this is true also of the combination of two of them—delirium and pyrexia,—which, without other indications of brain mischief, suggest a general and not a cerebral disease. The addition of moderate optic neuritis, of inequality of pupil, strabismus, palsy of cranial nerves, however slight, rigidity or weakness of limbs, retraction of head, or convulsions, adds very much to the probability of the diagnosis.

The diagnosis of the nature of meningitis is often less easy than the recognition of its existence. A family history of phthisis, or the presence, in an adult, of actual lung disease, suggests the tubercular nature of the inflammation, as, indeed, does the mere circumstance of childhood or youth, in which tubercular meningitis is far more common than any other variety. The absence of a cause of another form of inflammation is also an important negative element in the diagnosis. The indications of the other forms of meningitis are chiefly causal. Purulent inflammation may be suspected if the symptoms of meningitis follow suppuration elsewhere, near or distant, or signs of general septicæmia. The causes of purulent meningitis and of cerebral abscess are for the most part the same, and when an abscess causes acute symptoms, the diagnosis between the two is often difficult. It

depends chiefly on the greater affection of the cranial nerves in meningitis, and on the history of previous more chronic cerebral symptoms in abscess. But it must be remembered that an abscess and purulent meningitis not unfrequently coexist.

In this outline of the most important symptoms of the sudden and acute lesions, I have said nothing of anæmia of the brain, because it is extremely rare for symptoms due to this cause to come into any diagnostic problem. Almost the only cases in which such symptoms are important are those of young children who are profoundly exhausted by diarrhœa. The patient may become somnolent and comatose; may sometimes present convergent strabismus, and even rigidity of the neck. These symptoms may pass away; or the coma may deepen, the pupils dilate, and the child die—the brain after death presenting no morbid change. The state was called "hydrocephaloid" by Marshall Hall from its resemblance to meningitis, which is sometimes called "acute hydrocephalus." The diagnosis from meningitis depends on the absence of focal symptoms and on the circumstances under which the symptoms came on. The distinction from sinus-thrombosis is still more difficult, and depends chiefly on the depression of the fontanelle, the absence of external tumefaction, and of limb-symptoms. Often, however, it is necessary to wait before a confident opinion can be given.

LECTURE XVIII.

DIAGNOSIS OF THE NATURE OF THE LESION (*Continued*): SYMPTOMS PRODUCED BY CHRONIC LESIONS, TUMOUR, ANEURISM, ABSCESS, DEGENERATIVE DISEASES—DIAGNOSIS BETWEEN FUNCTIONAL AND ORGANIC DISEASES—CONCLUSION.

Gentlemen,—In the last lecture we commenced a survey of the groups of symptoms that indicate special lesions of the brain. We considered those that characterize the chief morbid processes that are sudden and acute. We pass now to the symptoms that attend the chronic lesions of the brain—chronic meningitis, tumour, aneurism, abscess, and the degenerative processes that we decided to include in our survey.

Chronic meningitis need not detain us long. The alcoholic form occupies the convexity, and causes diffuse symptoms, especially headache and delirium. Occasionally there is slight optic neuritis. It is probable that the inflammatory changes found in the membranes are part of a slight general encephalitis, rather than the actual cause of the symptoms. Syphilitic meningitis differs from all other forms in being local and never general. Hence its symptoms closely resemble those of a syphilitic growth. A positive diagnosis

between the two is rarely possible, and indeed they frequently coexist. The chief difference is, that in meningitis the symptoms of irritation are greater than is the evidence of destruction, and the symptoms indicate a wider extent of mischief, especially at the base, than a growth would be likely to produce.

The symptoms that suggest the existence of a tumour are severe and persistent headache, vomiting, and optic neuritis, with progressive symptoms of interference with the functions of some part of the brain. These must be searched for, in every case, most carefully. Many of the most important are unnoticed by the patient. The pupils should be compared, their action to light ascertained. The movement of the eyes in every direction should be observed, and if there seems to be any defect, a coloured glass should be used to examine for double vision. The hearing should be tested, the strength of the masseters felt, the movements of the tongue and palate, and the closure of the vocal cords should be tested by making the patient cough. The voluntary, emotional, and associated movements of the face should be carefully observed. In the limbs and trunk the reflex action should be especially examined. A loss of the superficial reflexes in limb and trunk is sometimes the first objective symptom of tumour on one side. If there is evidence of the existence of a tumour, the next question is, Is the growth in and invading the brain, or outside the brain and compressing it? Optic neuritis is produced more readily by tumours in than by those outside the brain. If the symptoms indicate a growth of some size at the posterior part of the base, or over the convexity, and there is no optic neuritis, this is in favour of the growth being outside the brain. If the symptoms are those of a tumour of the pons, and the cranial nerves suffer before the limbs, and on one side before those on the other, the tumour is probably outside the pons, springing from the membranes in the posterior fossa of the skull.

There still remains one of the most difficult problems in cerebral diagnosis. What is the nature of the tumour? The answer to this question can sometimes be given with a high

degree of probability, now and then with certainty. Often, however, the probability is so low that the answer is hardly more than a guess. The chief indications are as follows :— (1) The presence of morbid growths elsewhere, the nature of which can be determined. If the symptoms succeed a cancer or sarcoma elsewhere, it is practically certain that the intracranial growth is of the same nature. In rare cases the presence of an hydatid tumour elsewhere justifies the conclusion that the growth in the brain is of the same character. (2) Evidence of a general disease, of which an intracranial growth is known to be an occasional manifestation. Such diseases are tubercle and syphilis. In adults, signs of phthisis usually precede a tubercular tumour of the brain ; in children there may be no other present indication of tuberculosis. A family history of tubercular or scrofulous disease is usually to be obtained. A physical configuration such as often coexists with a tubercular tendency is also suggestive. The symptoms or history of syphilis, congenital or acquired, render it highly probable that a tumour is syphilitic. The absence of a history of constitutional syphilis, if the patient has had a chancre, should not receive too much weight, since secondary symptoms are often unrecognized, or even absent. Moreover, in an adult, the syphilitic nature of a growth cannot be excluded unless we can exclude the *possibility* of infection, since even the primary disease may have been unnoticed, as in many cases in which constitutional syphilis is patent. These diathetic indications afford a probability of the nature of the growth—often a very high probability, but no more than a probability, since a cerebral growth of other nature may coexist with either diathetic state. (3) The age of the patient affords a little help. If the patient is under fifteen, and presents no indication of inherited syphilis, the tumour is not a syphiloma. If the patient is an adult, and presents no indication of phthisis, the tumour is not likely to be tubercular. (4) The seat of the disease may give some assistance. In the cerebellum or pons, a tumour is likely to be tubercle or glioma, or, also if in the pons, it may be

syphilitic. A cortical tumour, with signs of irritation, is probably syphilitic or glioma. A tumour of the base is probably syphiloma or sarcoma. A tumour outside the brain-substance is probably a sarcoma. (5) The course of the growth may be suggestive. A very slowly growing tumour is not likely to be syphilitic. A tumour that grows rapidly at the onset, and then becomes stationary, is probably tubercular or syphilitic. The occurrence of an apoplectic seizure of moderate severity, with sudden symptoms, is rare except in glioma. (6) Evidence that arrest and retrocession of the growth follow the administration of iodide of potassium or mercury is strongly in favour of the syphilitic nature of the tumour. If these drugs are without influence, and arrest follows a tonic treatment, the growth is probably tubercular. (7) Lastly, tubercle, glioma, and syphiloma are the most common forms of growth in the substance of the brain. There is always an intrinsic probability, therefore, that a growth will be one of these, and this may be allowed weight in the absence of other indications. Neither the intercurrent meningitis nor multiplicity of growth is of diagnostic significance. By a careful comparison of these considerations a probable diagnosis can be made in a large number of cases—that is, a diagnosis which will turn out more often to be right than to be wrong.

The symptoms that indicate the presence of an abscess of the brain are, for the most part, the same as those of tumour —headache, vomiting, optic neuritis, mental dulness, and focal symptoms. The latter are absent, however, far more frequently in abscess than in tumour, on account of the great frequency with which the abscess occupies the temporo-sphenoidal lobe, and on account of the tolerance the nerve-elements exhibit to the pressure of a slowly increasing encapsuled collection of pus. The distinction depends on the course and associations of the cerebral symptoms, and on the causal indications. (1) Instead of the uniform progress of the symptoms of a growth, those of abscess are for a long time slight in what is termed the "latent stage." They then develope

rapidly, in an acute manner, like meningitis, or even in a sudden manner, like hæmorrhage, distinguishable from these by the definite character of the slighter chronic symptoms that preceded. (2) The ophthalmoscope is often of great value, revealing, during the latent stage, or at the onset of the acute stage, an optic neuritis, which excludes alike a mere vascular lesion and a commencing meningitis. (3) The general symptoms that attend suppuration—fever and rigors—are often present. (4) A cause of abscess (ear disease, suppuration about the skull or elsewhere, or an injury) can be found in most cases. With no cause ascertainable, and an absolute latency, an abscess can be suspected only on the indication afforded by the ophthalmoscope. There is no other cerebral lesion in which the patient may plunge, in a few hours, from apparent health into imminent danger, and in which considerable optic neuritis is found at the onset of the acute symptoms, when there is no general disease to which the optic neuritis can be due.

The symptoms of a small tumour at the base of the brain, in the position of a large artery, occurring in a person who is past middle life, or who has heart disease or syphilis, and, in the latter case, not yielding to treatment, justify a suspicion of the existence of an aneurism. The suspicion can only be raised to absolute certainty by the presence of a rare sign, a murmur audible on auscultation of the skull, practically met with only in aneurism of the internal carotid. The diagnosis of the precise artery affected depends on the local symptoms. I may mention a few of these indications, obtained by comparing the symptoms in many recorded cases. Optic neuritis is occasionally met with, but is not common unless the aneurism is adjacent to the optic nerve, *i.e.*, is of the internal carotid or anterior cerebral. The seat of pain is of significance only when occipital; it then points to the basilar as the artery affected. Loss of sight of one eye, sometimes extending to the other, with, or still more without, optic neuritis, with or without loss of smell on the side first affected, occurs in aneurism of the internal carotid and anterior cerebral. The distinction between the two

depends on the occurrence in the former of paralysis of the motor nerves to the eye first affected. Paralysis of the third nerve without affection of sight, or with hemiopia, is produced by aneurism of the posterior communicating, and also, usually, with hemiplegia of the opposite side, by aneurism of the posterior cerebral. Affection of the fifth nerve alone is of little significance, but with bilateral weakness in the limbs, and difficulty of articulation or swallowing, it occurs in aneurism of the basilar. Paralysis of the cranial nerves below the sixth, associated with weakness of the limbs on one side, may be due to aneurism of the lower extremity of the basilar or of the vertebral, and symptoms of bulbar paralysis, of slow or sudden onset, may occur in the same cases. Slight hemiplegia is of little significance, but, if it is considerable, there is not likely to be an aneurism of either the anterior cerebral or posterior communicating artery. Simple general convulsions are very rare. If they begin locally, they suggest that the aneurism is of the middle cerebral artery, on the outer surface of the brain, and if they are opisthotonic, that it is of the basilar. All these symptoms are, of course, only significant in the presence of a cause of aneurism. The severe apoplectic symptoms which attend rupture increase much the probability of the diagnosis; but this is then a matter rather of scientific curiosity than of practical importance. Without preceding symptoms, the rupture of an aneurism may be suspected if sudden and deep apoplexy occurs in a person who has not yet reached the degenerative period of life, and has no renal disease, and especially if there is heart disease or a history of syphilis. If the apoplexy is attended first with unilateral and then with bilateral symptoms, the aneurism that has ruptured is probably of the middle cerebral. Symptoms of hæmorrhage into the pons suggest that the aneurism is of the basilar or of the posterior cerebral.

There remain the three degenerative diseases that stand, to some extent, apart from the other lesions of the brain —bulbar paralysis, disseminated sclerosis, and general para-

lysis of the insane in so far as its physical symptoms are concerned. An alteration in articulation is constantly an early symptom of the first, occasionally of the second, and frequently of the third. Tremor is a usual symptom of the two latter, but is absent in the former. In all, the symptoms are gradual in development, although chronic bulbar paralysis has its acute homologue, which depends on a sudden vascular lesion in the medulla. Although the resulting symptoms of the acute and chronic disease may be the same, the sudden onset takes the cases out of the present category of chronic lesions.

A defect in articulation, and afterwards in swallowing, depending on actual distinct loss of power in the parts concerned, is the distinctive characteristic of bulbar, or labio-glossal paralysis. I told you (p. 107) that the palsy is arranged around the tongue as a centre, affecting the lips, tongue, palate, pharynx, and often the larynx; and I described to you the symptoms that result. The ultimate aspect of the patient, with motionless tongue, open mouth, immobile lower face, and unmodulated phonation on an attempt to speak, is characteristic and unmistakable.

The distinctive symptoms of insular sclerosis are not specially cerebral. There are coarse jerkings in the arms, sometimes in the legs, occurring on movement only. They apparently depend on the development of islets of sclerosis in the motor tract, either within the brain or within the spinal cord, and are often followed by actual loss of power. Sometimes unsteadiness on the legs is one of the earliest symptoms. With these, however, are often associated symptoms that are distinctively cerebral. (1) A change in articulation, in which syllables are unduly marked off from one another, and even separated, and, at the same time, the endings of words are slurred. But with this there is no loss of power in the muscles concerned in articulation, at any rate until the latest stage of the disease. (2) Nystagmus. (3) A slight and inconstant degree of mental change. There may be at first some mental hebetude, but the most characteristic condition is an unnatural cheerfulness, and contentment with a state

of disability which should naturally give rise to grave concern. The change never goes beyond this; but in this degree it is very common, and rarely fails to strike a medical observer as peculiar, although the friends regard it as merely the expression of an admirable resignation.

The early symptoms of general paralysis vary much. They may be mental or physical. The mental change that is most characteristic is the familiar "expansive delirium," as it is called, in which the patient revels in exaggerated personal delusions. Often, however, there is no more than a tendency to regard all things through rose-coloured spectacles, without any actually false idea. There may not even be this optimism; there may be merely mental weakness and failure of memory. The physical symptoms are as much spinal as cerebral. Tremor on movement is often conspicuous; it interferes with the more delicate actions, and is especially conspicuous in the muscles of the lips and face in articulation. Speech is hesitating and drawling, with a tendency to slur word-endings. Loss of the light-reflex of the iris, and inequality of the pupils, are common. With these there may be no spinal symptoms, or there may be indications of either lateral or posterior sclerosis. Cases of what may be termed "pseudo-general paralysis," in which the characteristic physical symptoms exist almost alone—merely loss of memory and slight optimism representing the mental disturbance—are not uncommon, both in syphilitic subjects and in those who have not had syphilis. These cases run a much more benign course than does the classical form of the disease, often remaining stationary for years, and even improving. They do not get into asylums, and therefore are scarcely recognized in descriptions of the disease, although they are far from rare.

In conclusion, we may glance briefly at some of the more salient points in the all-important distinction between the so-called functional and organic diseases of the brain. The diseases that most frequently give rise to difficulty are hysteria, neuralgia, and some forms of epilepsy, but the chief diagnostic indications of the inorganic head-pain and con-

vulsion have been already alluded to, and I need only now speak of the often perplexing symptoms of hysteria.

There are few organic diseases of the brain that the great mimetic neurosis may not simulate. Palsy and spasm, coma and convulsion, pain of every form and degree, giddiness, loss of sight, of hearing, of speech,—almost every symptom of positive lesion finds its counterpart in the repertory of that functional disturbance which lies, latent or manifest, within the potentialities of the nervous system of most women, and of many men, ready, in some, to spring into activity on the slightest touch of favouring circumstance. To lead you through the labyrinth of detailed distinction would occupy as many lectures as we have been able to devote to our entire subject. I must content myself with a briefer course—a course, indeed, that may perhaps be more useful to you,—and merely point out the general principles that must guide you. First remember this fact. Given the condition of age and sex—that is, of state of nervous system, developmental or other—that underlies hysteria, its manifestations may be evoked by any disturbance of nerve-function, whether this comes from without, as in the pure and primary disease, or from within, as in secondary hysteria, which may accompany almost any organic disease of the brain. I mentioned to you in a previous lecture how many organic maladies of the brain may evoke symptoms of hysteria. Therefore, given symptoms of hysteria, we must never infer that this is the primary disease until we have searched for, and excluded, the symptoms of organic disease. *A fortiori* this is true of the mere conditions in which hysteria occurs, and yet the diagnosis of hysteria is often made merely because the patient who presents symptoms of organic disease happens to be a girl. The slightest unequivocal symptom of organic disease is of absolute diagnostic significance, and until the absence of any symptoms of the kind has been ascertained, no other symptoms and no etiological circumstances should be permitted to bias the observer's mind. In a large number of cases, attention to this rule will dispel all difficulty. But there remain cases

in which the only symptoms present are equivocal, and seem compatible with each disease, although not characteristic of either. This difficulty is usually rather apparent than real. The symptoms common to the two classes present differences of detail and grouping, and a thorough knowledge of the characters of organic disease enables the observer to discern these differences without difficulty. In the very rare cases in which the symptoms are absolutely equivocal, the history of other unequivocal symptoms of hysteria may be allowed weight and to turn the scale. For instance, a lad, after a period of excessive study, was suddenly seized with severe pain in the head, which lasted for a day or two, and then gave place to a state of stupor, in which he could only be partially roused to slowly swallow nourishment that was placed in his mouth. The condition resembled that of meningitis; but a similar condition is sometimes of purely functional origin. All other symptoms of organic disease were absent, and it was ascertained that the headache was preceded by a period of excessively frequent breathing (a characteristic hysterical symptom), which ceased suddenly when the headache came on. This justified a diagnosis of hysterical stupor, and two days after the onset the lad woke up free from any serious symptom.

It may be well to glance very briefly at the differences between some of these equivocal symptoms. Speaking generally, this hysterical character is suggested (1) by their onset after emotion, or after witnessing analogous symptoms in another. (2) By their increase on attention, and in the course of examination. (3) By their mutability; grave symptoms of one character will cease suddenly, and give place to others which could not result from the same organic cause as the first. (4) By the differences between the symptoms of hysterical origin, and the corresponding symptoms of organic disease. For instance, we may take the one-sided motor palsy of hysteria—hemiplegia. There is rarely complete paralysis in both limbs, although there may be in one. The face is never affected—an important distinction. There is usually some contracture in the most paralysed limb, and

this, in the hand, involves usually the long flexor, flexing all the phalangeal joints (the metacarpo-phalangeal more than in late rigidity), and the flexion of the wrist does not relax the other joints as it does in organic disease. Moreover, all hysterical contractures present a distinguishing characteristic: when an attempt is made to overcome them, it is felt that the resistance is not uniform, but varies from moment to moment. When the paralysis is incomplete, movement is slow, and is attended by characteristic irregular tremor, far smaller in range than that of disseminated sclerosis, but coarser than simple tremor, and more irregular. If the muscles are felt or watched, it will be found that voluntary movement is interfered with by undue contractions in the opponents of the muscles that should effect the movement. There is no wasting, or change in electrical irritability, except after long disuse, and then only in trifling degree. The skin-reflexes are not lessened on the affected side; the knee-jerks are equal; there is no uniform foot-clonus (unless there is great contracture of the calf-muscles), but what I have termed a "spurious clonus" can occasionally be obtained, characterized by the palpable variations in the half-voluntary contraction of the calf-muscle on which it depends.* The onset of hemiplegia may be rapid, but it is rarely so suddenly complete as in organic disease, and is not attended by loss of consciousness. Anæsthesia is common, either one-sided and complete, or chiefly localized in the most paralysed limb. Hemianæsthesia, coming on without other indications of a cerebral lesion, is almost always of hysterical origin.

The only derangement of the eyeball-movements that occurs in hysteria is convergent strabismus from muscular spasm, and it is easy to observe the absence of any paralysis if you make the patient, with one eye closed, move the other in various directions. Simulated ptosis is sometimes seen; it depends on a very gentle contraction of the orbicularis, that is transformed at once into a vigorous and demonstrative contraction, to keep the eyelid down, if the patient is made to look up with the other eye.

* See "Diagnosis of Diseases of the Spinal Cord," 3rd Ed., p. 32.

The well-known aphonia of hysteria, depending on under-action of the adductors of the vocal cords, is sufficiently distinguished by the absence of any attempt at phonation. This inactivity may extend to the tongue, and cause loss of even whispered speech, a sequence that is pathognomonic. Very rarely a palsy of the abductors may give rise to inspiratory stridor without impairment of expiratory phonation. The symptoms are those of the same palsy when of organic origin, but the association of the two is always sufficiently diagnostic. The convulsive attacks of hysteria ought not to cause a difficulty in diagnosis. These features are, for the most part, distinctive, and the problem is, not whether they indicate organic brain-disease, but whether any other symptoms of a different character coexist with them.

Here, gentlemen, we must end. The problems that we have considered in these lectures are certain to present themselves before you, frequently, in your future work. Their form will vary, but the principles of diagnosis that I have endeavoured to put before you, will guide you, I believe, to a right conclusion in most cases. When you meet with special difficulty, do not be hasty in trying to arrive at a decision. Think over the symptoms; read over the description of the diseases between which the diagnosis lies; examine your patient again; and, if necessary, watch the symptoms for a time. Perplexing as these problems often are, they are rarely insuperable to those who combine, with a firm grasp of the methods of diagnosis, a fair knowledge of the symptoms of organic diseases of the brain.

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