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#### **Publication/Creation**

London: Macmillan, 1875.

#### **Persistent URL**

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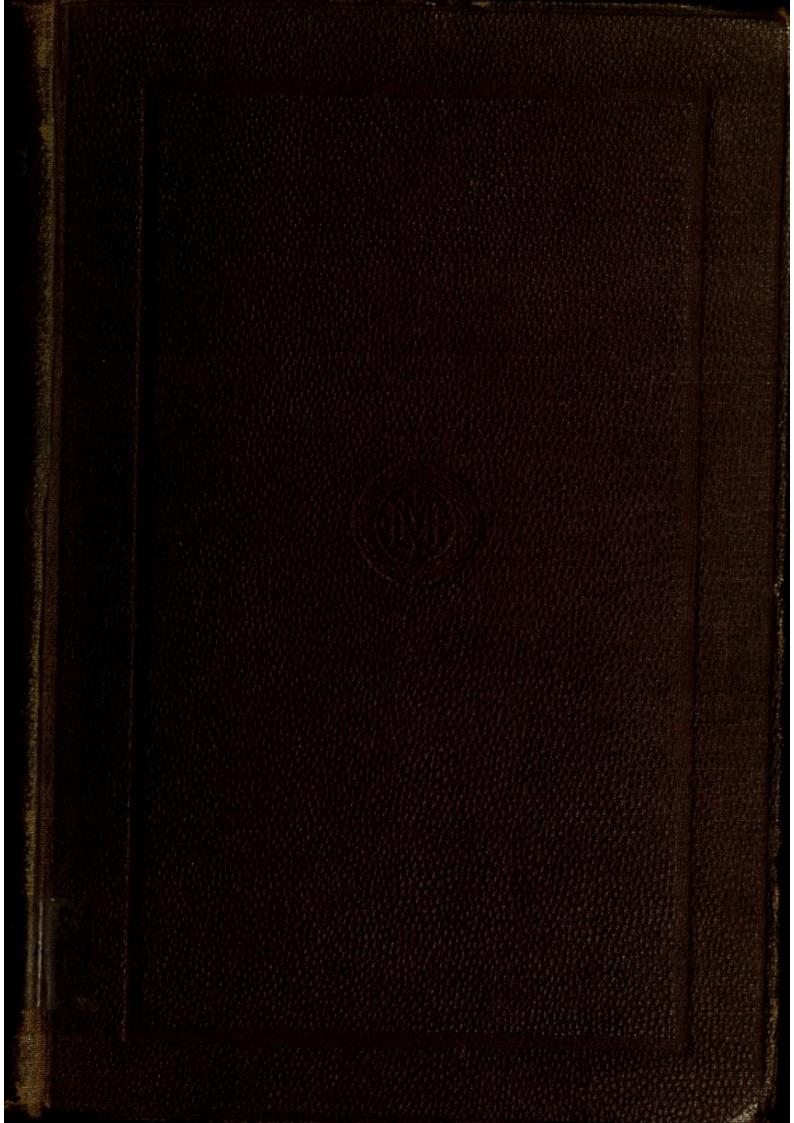
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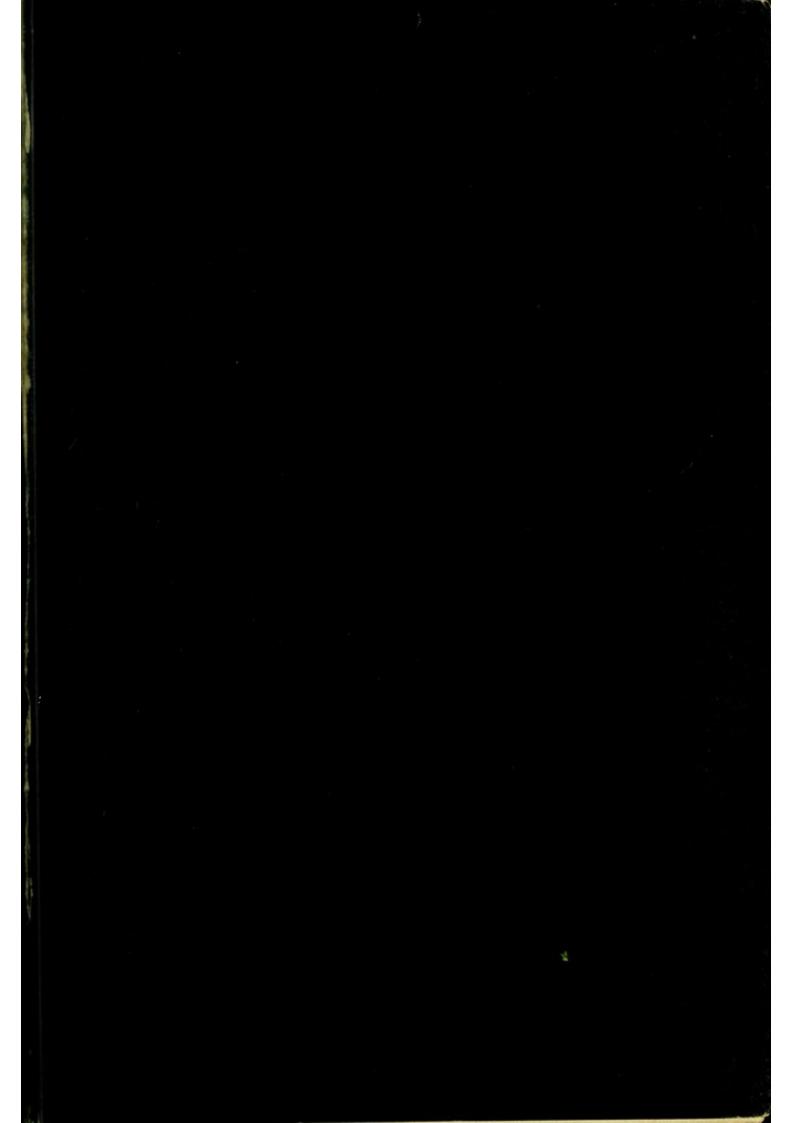




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## ON PARALYSIS

FROM

# BRAIN DISEASE

IN ITS COMMON FORMS.



## ON PARALYSIS

FROM

# BRAIN DISEASE

IN

## ITS COMMON FORMS.

BY

## H. CHARLTON BASTIAN, M.A., M.D., F.R.S.,

Fellow of the Royal College of Physicians,
Professor of Pathological Anatomy in University College, London,
Physician to University College Hospital, and
Senior Assistant-Physician to the National Hospital for the Paralysed and
Epileptic.

WITH ILLUSTRATIONS.

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MACMILLAN AND CO. 1875.

#### LONDON:

R. CLAY, SONS, AND TAYLOR, PRINTERS, BREAD STREET HILL.

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## PREFACE.

THESE Lectures were delivered in University College Hospital last year, at a time when I was doing duty for one of the senior physicians, and during the same year—after they had been reproduced from very full notes taken by my friend Mr. John Tweedy—they appeared in the pages of *The Lancet*.

They are now republished at the request of many friends, though only after having undergone a very careful revision, during which a considerable quantity of new matter has been added. It would have been easy to have very much increased the size of the book by the introduction of a larger number of illustrative cases and by treatment of many of the subjects at greater length, but this the author has purposely abstained from doing, under the belief that in its present form it is likely to prove more acceptable to students, and also perhaps more useful to busy practitioners.

Notwithstanding its defects and many shortcomings, the author is not without a hope that this little book may be considered in some measure to supply a deficiency which has long existed in medical literature. No department of medicine stands more in need of being represented in a text book of moderate compass; so that, imperfect as it is, this small work may perhaps be of some service till it is superseded by something better. In it the author has endeavoured to treat the subject with more precision than has hitherto been customary, and whilst the Lectures contain some novelties in method and mode of exposition, he hopes they may also be found not unfaithfully to embody the principal facts at present known concerning this very important class of diseases.

20, QUEEN ANNE STREET, CAVENDISH SQUARE, April, 1875.

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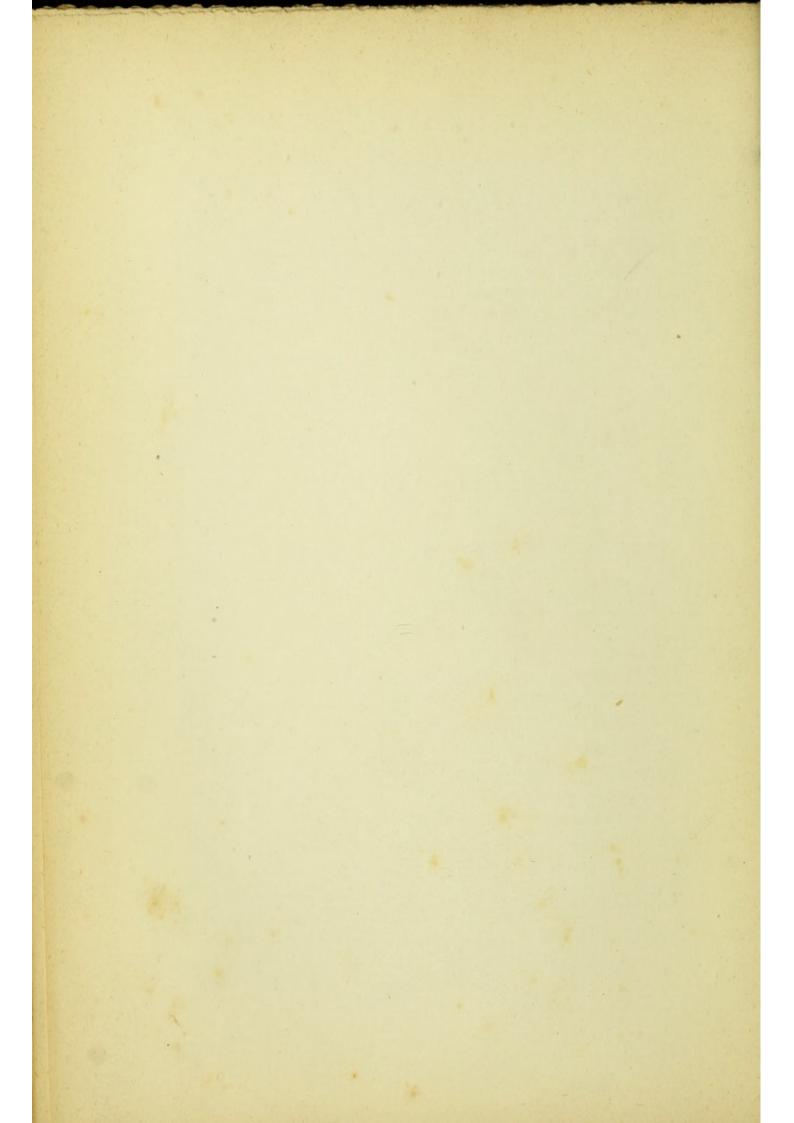
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# PARALYSIS FROM BRAIN DISEASE IN ITS COMMON FORMS.

## LECTURE I.

INTRODUCTION -- CAUSATION OF HEMIPLEGIA-RUP-TURE OF VESSELS -- OCCLUSION OF VESSELS --SPASM OF VESSELS.

- INTRODUCTORY—Inadequate Descriptions of Hemiplegia—Divisions of the subject for Consideration—Causes of Hemiplegia in general —Scope of present Lectures.
- RUPTURE OF VESSELS Most frequent Sites of Meningeal Hæmorrhages—Distribution according to Age—Cerebral Hæmorrhage— Reasons for Frequency of—Liability to, increasing with Age— Most frequent Sites of—Lesions in Medulla Oblongata.
- Occlusion of Vessels—Consequences of—Both Functional and Structural—Softening of Secondary or Inflammatory Origin—Softening from Disease of Minute Vessels—Functional Effects of Softening and of Hæmorrhage very Similar—So-called 'Simple Apoplexy'—Thrombosis occurs in Arteries or Veins—The Process favoured by General and by Local Causes—Sometimes extends to many Vessels—Embolism—Most frequent Sites of—Occurrence at different Ages—Nature of Emboli—Multiple Minute Embolisms.
- SPASM OF VESSELS—A Co-operative Cause—Associated with Altered Molecular States of Nerve Tissue—Varieties of Functional Hemiplegia—Characteristics of Epileptic Hemiplegia—Its supposed Nature—Its mode of Production—Similarity between it and some Forms of Emotional Hemiplegia—Separation of Cases from latter Category—Choreic Hemiplegia—Not a mere Functional Disease—Its mode of Production—Hysterical Hemiplegia—A typical case.

#### LECTURE I.

GENTLEMEN,—During the present session we have had several very interesting cases of Hemiplegia under our observation, and I have been asked to give a few lectures on this subject — dealing with the common forms of the affection in such a manner that you may not only learn the principal variations which are to be met with in the grouping of symptoms, but also how far such variations are indicative of lesions in different parts of the brain. The request seemed reasonable, and not unnatural considering the very brief and altogether inadequate descriptions of the hemiplegic state which are at present accessible to you in the various medical text-books.

The subject is, however, one of very great extent and importance, since lesions of various kinds in different parts of either half of the brain all tend to produce, on the side opposite to that on which the brain is damaged, a more or less complete hemiplegia or paralysis of one lateral half of the body. By reason of the extent and complexity of the subject, therefore, it is one which could not be dealt with in an adequate manner in the few pages which might be devoted to it in a text-book of medicine,—even in the event of its receiving this formal recognition. I speak in this alternative manner, gentlemen, because, strange as it may appear, in more than one of the larger and more important works of this class you will find that the symptomatology of hemiplegia does not even receive such small amount of recognition as might be contained within the compass of a few pages: the subject is often only incidentally alluded to in sections on Hæmorrhage, Softening, Abscess, or other pathological affections of the brain. In these works brain-disease is dealt with primarily from a pathological standpoint, and, as I venture to think, with far too little reference to the locality of lesion.

But for clinical purposes the very reverse arrangement should be adopted. The brain is an exceedingly complex organ, made up of numerous functionally dissimilar though intimately related parts. In each of its halves we have a cerebral hemisphere composed of different lobes, bounded externally by numerous and variously endowed

convolutions; we have, moreover, important ganglia at the base, the crus cerebri, one half of the pons Varolii and one-half of the medulla oblongata. In addition, we have the cerebellum and its peduncles as other possible sites of pathological change. We ought always to endeavour to discriminate between the effects of injury to these several parts, since variations in the seat of the lesion give rise to so many of the differences in the total grouping of symptoms daily encountered at the bedside. And we should more especially strive to make this localization now that we know the weakness of the old point of view. The nature of the lesion must, in fact, be matter for secondary rather than for primary consideration. For experience has clearly shown that the clinical grouping of symptoms may be almost indistinguishable when a given brain region is differently injured, either by the occurrence of Hæmorrhage or by an equal amount of damage due to Softening.

I am also aware that many very important additions have, of late years, been made to our knowledge of the hemiplegic state, though since these can at present only be gleaned piecemeal from numerous contributions scattered through our own and foreign journals, or from memoirs and other more substantial works produced both in this country and abroad,

such new facts are rendered practically inaccessible to most of you.

For these reasons, gentlemen, and because it seemed desirable that some one who has enjoyed good opportunities of becoming familiar with many of the Protean forms of brain-disease should take stock of our present knowledge concerning the hemiplegic condition, I have the less hesitation in complying with the request made to me. I shall, therefore, give a few lectures on the common forms of hemiplegia-or perhaps I should say more definitely, on those forms in which the onset of the disease is more or less sudden. This limitation has been decided upon partly with the object of ensuring greater clearness in my exposition than would otherwise be possible, and partly in order that it may be comprised within some reasonable bounds. I shall take up the subject from a purely clinical aspect, although, for reasons already stated, it seems best to endeavour to deal with it in as systematic a manner as possible.

In thus attempting to collect, sum up, and perhaps put together from new points of view, our existing knowledge on this important subject, I have set myself a difficult and somewhat laborious task. It is one, moreover, which I should have been less ready to undertake did I not feel that, however im-

perfectly it may be accomplished, you may yet become acquainted with many facts and some aspects of this many-sided subject, with which you were not previously familiar.

In endeavouring to give you a connected clinical view of the subject of Hemiplegia, I shall first consider the Causes or various pathological conditions which may produce such an assemblage of symptoms -though even here we shall study the subject principally from its clinical aspects, and with the view of obtaining data available for the purposes of diagnosis. We shall then more fully examine the Signs and Symptoms produced by these pathological conditions; and having done this in a general manner, I shall afterwards strive, however imperfectly, to show how these signs and symptoms are grouped when different portions of the brain are injured. Our knowledge is still, unfortunately, very defective in this direction, yet it does in many instances enable us to come to a differential diagnosis as to the region of brain implicated. The clinical groupings of symptoms which present themselves have, in fact, to be interpreted by the aid of what knowledge we possess of cerebral anatomy and physiology. We thus strive, in any given case, to arrive at a Regional or Anatomical Diagnosis. And this is not a matter of mere

physiological interest; it is often of great importance in relation to questions of prognosis more especially. At the bedside this should always constitute one of our attempts in the direction of diagnosis-such an inquiry should, in fact, always go hand in hand with our endeavour to determine the probable nature of the changes taking place in the affected region. This latter consideration constitutes the second part of our diagnosis-what may be called the Pathological Diagnosis, as distinguished from that which is regional or anatomical. I shall have to point out, therefore, how we are to apply our knowledge of the causes of the disease towards the elucidation of this aspect of the question. In performing this part of our duty we have to bring our clinical and pathological knowledge to a focus; we have to take into account the different ages at which the several pathological states are most apt to be met with; the general condition of other organs of the body with which such states are most frequently associated; and the extent to which differences in the mode of onset of the symptoms may speak more strongly in favour of one or other kind of pathological change. We shall then, further, have to consider the actual state of the patient whose case we are considering, his age, and the mode of onset of his disease, in order to form an opinion as to the probable nature of the lesion.

Lastly, gentlemen, I shall consider the important questions involved in *Prognosis*, as well as those relating to the *Treatment* to be employed in the various forms of Hemiplegia.

#### CAUSATION.

Turning first to the mode of *Causation* of Hemiplegia, we may tabulate its proximate causes in the following manner:—

- a, On the surface of the brain.
  bloodvessels b, Into its substance . . . Hæmorrhage.
- 2. Occlusion of vessels  $\begin{cases} a, & \text{Thrombosis} \\ b, & \text{Embolism} \end{cases}$  Softening.
- 3. Spasm of vessels and Functional Disturbance.
- 4. Fibroid indurations of portions of brain.
- Tumours of brain and its meninges, including acephalocysts.
- 6. Abscesses.
- 7. Traumatic injuries and their sequences (Meningitis, &c.)
- 8. Congenital Atrophy.

Now I propose to speak to you especially concerning the Hemiplegias of rapid onset—those most commonly met with, and which are due to Rupture, Occlusion, or Spasm of one or other of the cerebral bloodvessels. Much that I shall say will of course be applicable also to the rarer forms of hemiplegia—to those due to Indurations, Tumours, Abcesses, Injuries, or Congenital Atrophies of the brain-substance. I cannot, however, now attempt to follow up all the details relating to these more slowly-arising and more irregular forms of hemiplegia.

Confining ourselves within the limits just mentioned, we must look a little more closely at each of our three principal modes of causation.

Rupture of Vessels, producing Hæmorrhage.—
Hæmorrhage into or upon the surface of the brain varies most widely both in situation and in amount in different cases. There are, nevertheless, particular regions in which it is more especially prone to occur. And even where giving rise to obvious symptoms, the hæmorrhage may vary in amount from a minute clot not larger than a pea occurring in the pons Varolii, to an effusion of many ounces of blood which tears up the substance of one hemisphere and subsequently fills the lateral ventricles. Effusions of blood may also be single or multiple in the same individual.

With regard to the most common situations in

which hæmorrhage is met with, I will especially name the following sites, saying nothing at present concerning the relative frequency with which ruptures occur in these various situations:—

- I. Meningeal Hæmorrhages; that is, effusions of blood on or pressing upon the surface of the brain. These effusions are often of traumatic origin. They are due either to rupture of one of the meningeal arteries, or to a giving way of some venous or arterial trunk in the pia mater. At other times a meningeal hæmorrhage is due to the bursting of an aneurism on one of the larger vessels near the circle of Willis.
- II. Cerebral or Cerebellar Hæmorrhages; that is, effusions of blood into some portion of the substance of the brain. Under this last category we may recognise the following sites:—
- I. In the substance of one of the hemispheres, with or without implication of the cortical substance; each hemisphere being roughly divided into (a) an anterior, (b) a middle, and (c) a posterior portion.
  - 2. In or just outside the corpus striatum.
  - 3. In or just outside the optic thalamus.
- 4. In either of the last two situations, together with effusion into the lateral ventricles.

- 5. In the crus cerebri.
- 6. In the pons Varolii. (a) In its centre; (b) in the upper part of one lateral half; (c) in the lower part of one lateral half.
- 7. In the cerebellum. (a) In one of its lateral lobes; (b) in its middle peduncle; (c) in its median lobe.

I have made no undue subdivision here. I have mentioned only those regions the injury of which we ought to endeavour to discriminate at the bedside. Lesions in some of these situations we are able to diagnose with considerable certainty, though between others we shall often have very great difficulty in deciding, even if we do not find it quite impossible.

With regard to the Age at which these ruptures of vessels are apt to occur, a considerable difference exists between meningeal hæmorrhages and those into the substance of the brain, though the latter do not in this respect differ much amongst themselves. Meningeal Hæmorrhages tend to occur at a decidedly earlier age than cerebral hæmorrhages. In very young children, whose vessels are healthy, bleeding into the arachnoid may occur from any unusual amount of strain. This occurs occasionally at the time of birth, especially during prolonged labours. Indeed, according to Cruveilhier, arachnoid

hæmorrhage is the cause of the death of about onethird of those infants who die almost immediately after birth. A little later on in life a similar accident may occur during paroxysms of whoopingcough, or during other spasmodic conditions in which the return of venous blood from the head is impeded. Later still an arachnoid hæmorrhage not unfrequently follows falls on or blows about the head, or it may result from the rupture of aneurisms on some one or other of the vessels at or near the base of the brain-the latter being an event which very rarely takes place in individuals under the fourteenth year, though thence onwards it is apt to occur pretty equally at different ages. These ruptured aneurisms are decidedly more common in males than in females; and in respect to other sources of meningeal hæmorrhage, the disproportion in the same direction is still greater. Thus, of 163 recorded cases of meningeal hæmorrhage analysed by Gintrac, nearly three-fourths were males. If we add to these cases the other 77 instances of hæmorrhage upon the surface of the brain analysed by the same author,-viz. those produced by rupture of aneurisms on the larger cerebral vessels—we get a total of 240 cases of meningeal hæmorrhage, which are thus distributed in respect to age :-

I	day	to	10	years		 10	cases.
II	years	,,	20	,,		19	,,
21	,,	,,	30	,,		27	,,
31	,,	,,	40	,,		52	,,
41	,,	,,	50	.,,		33	,,
51	,,	,,	60	,,		41	,,
61	,,	,,	70	,,		32	,,
71	,,	,,	80	,,		26	,,
						-	
						240	

Putting aside for the moment the question as to the period of life at which Cerebral Hæmorrhage is most prone to occur, I would call your attention to the very great frequency with which bleeding takes place into the substance of the brain as compared with the frequency of such an occurrence in other organs. This liability to internal hæmorrhage is doubtless due in great part to the softness of the brain, and to the consequent small amount of support to the vessels which ramify through its substance. No other organ but the spleen can compare with the brain in this respect; and that softness of tissue is one important factor in the production of cerebral hæmorrhage seems to be further indicated by the comparative rarity of bleeding into the substance of

the spinal cord, where the connective tissues giving support to the vessels are much more notably developed. The canals through the nerve substance, in which the vessels run, are also much less wide in the spinal cord than they are in those parts of the brain where hæmorrhages are most apt to occur. So that this peculiarity of the cerebral vessels—the fact that the small arteries lie almost unsupported, in canals of a larger diameter, which ramify with them through the brain-substance—is doubtless another special cause rendering these vessels peculiarly liable to undergo degenerative changes, and also to be ruptured under the influence of increased pressure from within.

The canals which ramify through the substance of the corpora striata are often three times as wide as the vessels which they contain, as I have myself ascertained by actual measurement. Now this anatomical arrangement seems peculiarly to favour the production of a change which has of late years been shown, by the observations of Charcot, Bouchard, and others, to be especially frequent in cases of cerebral hæmorrhage. I allude to the frequent occurrence of those miliary aneurisms (varying in size from a small pin's head to a mustard seed) which recent researches have shown to be so

common in elderly people who have died from cerebral hæmorrhage—occasioned by the rupture of one or more of them. The production of these vascular dilatations is especially favoured by the previous occurrence of fibroid changes in the walls of the vessels. Such changes, however, as well as fatty or atheromatous degenerations, which are also, as you know, very frequent in the cerebral vessels, tend to increase in direct proportion to the age of the individual.

Thus, the softness of the brain and the further lack of support to its vessels of which I have spoken might be supposed to favour the occurrence of their rupture in persons of any age, where their coats are subjected to any very considerable amount of strain from within. And we may also expect that these influences would become more and more operative with advancing age, owing to the increase of vascular degeneration of the fibroid and of the fatty type which this so commonly entails. Judging from the elaborate statistics collected by Gintrac, this seems to be practically what does occur. defatigable worker has collected and analysed the records of 658 cases of hæmorrhage into different portions of the substance of the brain, and he found them to be thus distributed according to age :-

Birth	to	10	years		15	cases.
11 years,,		20	,,		24	,,
21 ,	, ,,	30	,,		44	,,
31,	, ,,	40	,,		74	,,
41 ,	, ,,	50	,,		98	,,
51 ,	, ,,	60	,,		129	,,
61,	, ,,	70	,,		152	,,
71,	, ,,	80	,,		110	,,
81,	, ,,	90	,,		12	,,
			•		658	

Childhood, youth, or early adult age does not, therefore, by any means preclude the diagnosis of cerebral hæmorrhage in any given case, though reference to the figures which I have previously given will show you that at these periods of life meningeal hæmorrhages are proportionally much more frequent. On the other hand, as we approach middle life and old age, hæmorrhages into the substance of the brain become more and more frequent. The diminution in numbers after, the seventieth year is, as Sir George Burrows long ago pointed out, only apparent, owing to the much smaller number of people who live after this age has been attained.

With regard to the most common seats of hæmorrhage within the skull, it is not easy to arrive at correct information, because we are apt to find an undue proportion of rare cases put upon record. Still, if we look to large numbers, we obtain data of a certain value. In a total of 751 cases of hæmorrhage within the cranium, Gintrac found it recurring in different situations in the following manner:—Meninges, 172; cortical substance of the hemispheres, 45; middle lobes of the brain, 127; posterior lobes, 33; anterior lobes, 17; corpora striata, 72; optic thalami, 38; pons and cerebral peduncles, 76; medulla, 2; cerebellum, 55.

The great frequency here indicated of hæmorrhage into the middle lobes of the brain is a very
striking fact. It is in part explicable by the far
greater bulk of the middle portion of the brain—
roughly spoken of as its 'middle lobe'—as compared
with that of the anterior or of the posterior segments
of the hemisphere; though it is still further explicable by reference to the mode of distribution of the
vessels to these different parts. The latter consideration also suffices to explain the greater frequency
with which the posterior lobes are the seat of hæmorrhage as compared with the anterior.

The frequency of the occurrence of hæmorrhage in the pons and cerebral peduncles is also striking, especially when compared with its extreme rarity in the medulla oblongata. Hæmorrhages here are always very minute, rarely coming up to the size of a pea. I may indeed remark, that in respect to its diseases, the medulla is much more closely allied to the spinal cord than to the brain; and for this reason, although it forms part of the encephalon, I do not propose to take its lesions into consideration in these lectures. I will merely state that morbid processes in the medulla occasionally give rise to a form of paralysis allied to 'spinal hemiplegia.' One of the principal characteristics of each is the absence of facial palsy, though in the latter affection sensory and motor paralysis also occur on opposite sides of the body.

We turn now to the next common cause of hemiplegia — Occlusion of Vessels, whether by Thrombosis or by Embolism. This event leads to pathological consequences and clinical symptoms just as important as those produced by rupture of bloodvessels; and whether the occlusion is due to gradual coagulation of the blood in any given vessel, or to the impaction in the same site of some foreign substance brought from the heart or elsewhere, the pathological consequences are much the same—some amount of 'Softening' occurs in each case, so long as the occlusion is in one of the vessels situated on the distal side of the circle of Willis. The portion

of brain-tissue previously supplied by the occluded vessel is cut off from its blood-supply; the peripheral portions of the vascular area become hyperæmic and serum is effused, whilst the central portions at once begin to undergo fatty and degenerative changes, resulting in the course of a few days in the production of a focus of 'softening,' or cerebral 'ramollissement.'

Occlusion of one or other of its large vessels, indeed, seems to be by far the most frequent cause of Softening of the brain, though in some instances, more especially in old people in whom there are wide-spread atheromatous changes in the smaller cerebral vessels, a patch or patches of degenerative softening may be found where no occlusion of larger vessels can be detected. Where the condition is of inflammatory origin, it is secondary to other changes, such as traumatic injuries or the presence of adventitious products, which with the exception of blood clots do not now come under our consideration.

Several patches of Softening may be found coexisting in the same brain, and the foci themselves may be either very minute, or so extensive as to involve nearly the whole of one hemisphere, with, perhaps, part of the other.

Now, clinically, it matters very little—that is, so far as the symptoms ultimately produced are concerned—whether a given portion of brain-tissue is

broken up by an effusion of blood into its substance, or is rendered functionally inert by a process of softening. You must not forget, however, that in the latter class of cases the hemiplegic symptoms are at first produced by the cutting off of the bloodsupply from a certain portion of the brain-tissue, and that 'softening' is only one of the after-effects of structural changes resulting from this deprivation of blood. It is important to bear this in mind, because, should death occur within forty-eight hours from the onset of apoplectic or hemiplegic symptoms, no softening may be discoverable, simply because there has not been sufficient time for this pathological condition to become established. If, therefore, in such a case you find no hæmorrhage, or no patch of softening, you must not merely on this account come to the conclusion that you have to do with a case of 'simple apoplexy.' You must search diligently for some vascular occlusions-more especially in many of the minute arteries—rather than rest content with such a very questionable diagnosis.

I must now say a few words to you concerning each of the common causes of softening of the brain.

Thrombosis is a process which may occur either in arteries or in veins, but as a cause of hemiplegia we have almost always to deal with it in one or other of

the cerebral arteries, or else in several of them simultaneously. Occasionally, however, ill-defined forms of paralysis are produced by thrombosis in the longitudinal sinus, extending into the cerebral veins over the vertex, on one or both sides. The local coagulation or separation of fibrine in certain vessels, of which the process of thrombosis consists, is determined in part by general causes tending to diminish the force of the heart's action and to alter the quality of the blood, and in part by local degenerative states in the coats of the vessels themselves.

As 'general' causes, may be specified certain cachectic conditions of the system, in which, whilst the heart is weak, the blood is altered in quality and rendered more than usually prone to coagulate. This seems to occur occasionally in the period of convalescence from acute diseases, and also in delicate women after parturition. The process is, moreover, always favoured by a slow, irregular, and weak action of the heart.

The chief 'local' causes of thrombosis are pathological changes in the coats of the vessels, whereby their inner wall is roughened and their calibre is narrowed. Thus it is favoured by all fibroid thickenings and indurations of vessels, both syphilitic and non-syphilitic; and by atheromatous or calcareous changes, which roughen the vessels' surface or expose

the blood to the coagulating influence of not-living materials in the vascular wall.

Wherever it happens that general determining conditions are powerfully aided by local degenerative changes, thrombosis is of course most prone to occur; but in some cases general conditions alone seem to suffice without the determining influence of marked degenerative change, and this is the reason why thrombosis is almost as common in early adult life as it is at later periods. This process may occur in any of the vessels at the base of the brain, or in several of them at the same time—as we not unfrequently find when a thrombus forms in one of the internal carotid arteries. The clot then often extends not only into the middle and anterior cerebrals, but perhaps also into the posterior communicating and the posterior cerebral artery of the same side.

Embolism may be encountered in any of the vessels beyond the circle of Willis, though it is especially prone to occur in the Sylvian or middle cerebral arteries. It is met with more frequently in the artery of the left than in that of the right hemisphere, so that hemiplegia from this cause is most frequently right-sided in its distribution. Embolism may take place at any period of life, and is, indeed, much more frequent under the fortieth year than after that age.

The emboli, or obstructing masses, vary both as to their nature and origin. They may consist of fibrinous masses swept away from 'vegetations' on the valves of the heart; or from bodies of the same nature on the lining membrane of the left auricle, or on any part of the aorta anterior to the origin of the great vessels which carry blood to the brain. More rarely the embolon may be a detached fragment of a thrombus formed in the pulmonary vein, which enters the left cavities of the heart, and is thence swept onwards to the cerebral vessels.

At other times numerous embolic particles or fragments are derived from the crumbling of a fibrinous concretion which has formed in connection with the left auricle or ventricle. In instances of this latter description, or where a large atheromatous depôt in the aorta discharges a quantity of débris into the circulation, we may have multitudes of small embolisms in certain vascular areas instead of a stoppage of one of the main arteries by a larger occluding mass. These accidents, in which the embolisms are both numerous and widespread, are particularly liable to produce a rapidly fatal apoplectic condition.

In those cases in which the occlusion of a main vessel is not perfectly brought about by the impacted body, the stoppage is soon completed by a process of thrombosis taking place on the distal side of the embolon.

I have lastly to speak of Spasm of Vessels as one of the causes capable of rapidly producing a hemiplegic condition. And, unfortunately, there is a great deal of uncertainty about this part of the subject.

Spasm of the vessels may not be the efficient cause, although most likely it has always something to do with the condition of 'functional' derangement to which I now desire to call your attention. There probably exists, in these cases, an altered molecular state in the brain-tissue of the affected region, which for a time renders it unfit to discharge its proper functions—although if we could examine the part at the time there might be no change visible to the naked eye or even by the aid of the microscope. There can be no doubt that many cases of hemiplegia must be placed in this category; and if for the sake of brevity and convenience I now speak of them under the head of 'spasm of vessels,' you must not suppose that I regard such a condition as the sole explanation of the molecular state itself with which it may be associated and which perhaps more immediately gives rise to the paralysis.

We here group many of the cases of 'Epileptic

Hemiplegia' and of 'Emotional Hemiplegia,' as well as a few rare cases of an hysterical character.

We may take Epileptic Hemiplegia as a type of the condition. A patient suffers from unilateral convulsions from time to time, and after one of these attacks, which has been perhaps a little more severe than usual, he is found to be paralysed on the side which was previously affected with convulsions. The hemiplegia is of the ordinary kind, except that it is altogether temporary in its duration. It lasts for a few hours, for a few days, or perhaps for a week or more, and then the patient rapidly and completely regains power in the paralysed limbs. Such a hemiplegic state may frequently recur with subsequent fits, though in many cases there is merely some slight weakening of the affected limbs which lasts about an hour or two. Although a hemiplegic condition of this kind is most frequently associated with a typical case of unilateral convulsion, it occasionally happens that hemiplegia follows where the convulsive condition has been general, though most marked on the side which subsequently becomes paralysed.

Now in the most typical of these cases—those where the convulsions have been unilateral—we find that the convulsions had previously affected the same muscles as are subsequently found to be

paralysed, and had affected them too in nearly the same relative proportion. The rapid and complete recovery from the paralytic condition, however, compels us to believe that a mere molecular damage of a recoverable kind must have taken place in the nerve-elements, a damage or derangement which for a time hinders their proper life-changes and so puts a temporary stop to their functional manifestations.

The result may be very similar in certain cases of fright, sudden surprise, anger, or other emotional disturbance. In these cases a large amount of nervous energy is liberated in the cerebral hemispheres, and a rapid downrush occurs upon the motor ganglia at the base of the brain, as we see by the cries and sudden muscular movements which accompany such states. This sudden downrush of energy, or 'discharge' upon the motor ganglia, induced by emotional causes, whilst it often produces chorea, seems occasionally to induce mere loss of speech, or the same temporary paralysis as that which is produced by the nervous discharges in an attack of unilateral epilepsy-which then goes by the name of Emotional Hemiplegia. In each case, if we have to do merely with spasm of vessels and molecular changes in nerve-tissue, the condition is one in which recovery or molecular restoration speedily tends to occur.

We must not forget, however, that the exciting causes of such attacks as we have last referred to, may entail further changes. They may at the time give rise not merely to spasm, but to actual rupture of vessels in any of the basal motor ganglia, or they may set up irritative changes in the nervetissue which may subsequently lead to the establishment of thrombosis in the part. Thus, whilst we may have a simple and rapidly recoverable form of hemiplegia, induced by an epileptic fit or by a sudden emotional disturbance, we must not forget that in other cases these forerunning events may lead to more permanent damage in the parts of the brain secondarily affected, which would compel us to remove such cases from our present category. And of this we have recently had a good example under our observation. I allude to the case of a woman, lately in our wards, in whom a much more permanent hemiplegic condition was suddenly established under the influence of strong emotion, occasioned by seeing her child have a series of rapidly succeeding epileptic attacks. There is good reason for believing that the functional and other changes to which I have just been referring take place principally in the corpus striatum, though in some instances they may principally involve the pons Varolii. Judging from several symptoms present, this seemed to be the case, indeed, in the instance to which I have just referred.

There is a group of cases included under the name 'Choreic Hemiplegia' to which I ought also here to allude. You know that Chorea, like Epilepsy, sometimes affects one side of the body only, and that in these instances of Hemichorea the same parts are affected with clonic spasms as are paralysed in cases of hemiplegia due to lesions of the corpus striatum and its neighbourhood. Well, it happens in a certain small proportion of the cases of hemichorea that the spasms cease after a time, and give place to a hemiplegic condition which may be more or less marked, though it is usually of comparatively brief duration. Now we have here to do, as I believe, with something more than mere spasms of vessels, and altered molecular states of nerve tissue.

It is doubtless true, that in many cases of chorea we have only to do with mere functional derangements, that is, mere altered molecular states and activities in nerve elements. There is good reason for believing, however, that in other cases minute vessels become blocked in the corpus striatum and adjacent parts of the brain. This I have myself found on three or four occasions. These stoppages

are, I believe, in the main caused by minute thromboses rather than, as generally believed, by embolism. At all events, when present, such multiple minute occlusions may at times spread by a process of thrombosis so as to involve larger vessels, and thus the hemichoreic affection may give place to a hemiplegic condition. Should this be its real pathology, Choreic Hemiplegia cannot be admitted into that category of functional nervous affections with which we are now concerned.

Finally, it remains for me to say a few words concerning what has been called 'Hysterical Hemiplegia.' This is a very rare and altogether anomalous condition, for hysterical paralysis is most frequently confined to a single limb-arm or leg. Occasionally both legs are affected; but it is much more rare for an arm and a leg on the same side simultaneously to exhibit this peculiar loss of power. When such a combination does exist, the weakness is often more marked in the leg than in the arm, and it is much more frequently met with on the left than on the right side of the body. The late Dr. Todd laid much stress upon the absence of facial paralysis and of paralysis of the tongue in these cases. The patient, too, is apt to drag the foot along the ground in walking in a manner altogether differ

rent from the method of circumduction by which the ordinary hemiplegic swings the paralysed leg forwards.

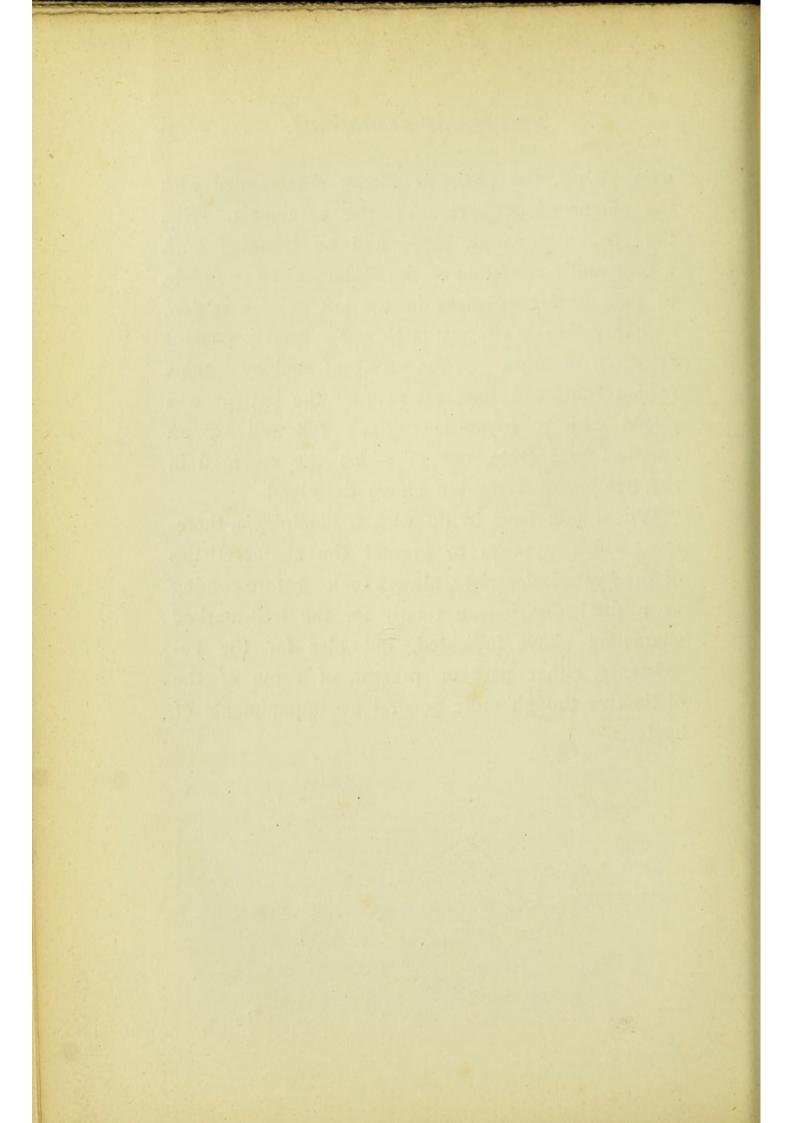
I cannot say that I have ever met with an unmistakable example of this affection, though I have frequently seen cases in which hysterical symptoms have been presented by paralysed patients, where the palsy was obviously due to actual organic lesion in brain or spinal cord. Without doubting that cases may occasionally be met with which might appropriately enough be classed as 'hysterical hemiplegia,' I would strongly warn you not to be led away by the mere existence of hysterical symptoms, and never to forget that such symptoms may coexist with grave organic lesions to which the paralytic condition may be due.

The most definite information known to me concerning hysterical hemiplegia is to be found in M. Charcot's valuable lectures. He admits the great rarity of the affection, but gives an admirable description of a typical case. Its principal characteristics were the absence of paralysis of the face and of the tongue, the existence of a profound and well-defined hemi-anæsthesia on the paralysed side; together with the presence of a peculiar form of rigidity in the paralysed limbs. The rigidity began in the leg, and was of an altogether exceptional and

characteristic nature. The lower limb was in a state of rigid extension, with marked inward deviation of the foot and a similarly powerful contraction of the adductors of the thigh. This bar-like rigidity of the lower extremity was, moreover, permanent. It had existed for nearly two years without daily fluctuations, without disappearing even in the most profound sleep; and yet the nutrition of the limb had not sensibly suffered, nor had the electric irritability of its muscles undergone any very appreciable diminution. Such a form of contraction, though it seems to be the rule in hysterical hemiplegia, is almost if not wholly unknown as a result of ordinary lesions in the brain —and it is almost equally exceptional in these cases for contraction to exist to a marked extent only in the leg. Yet in this case seen by Charcot there was for a long time no contraction whatever in the arm, and when it did occur it supervened not gradually but suddenly after one of the epileptiform attacks to which the patient was liable. The contraction in the arm, however, was more after the ordinary hemiplegic type—that is it was drawn to the side of the chest, whilst the elbow, wrist, and fingers were firmly flexed.

These are certainly very marked characteristics, though others equally important existed in this case. Thus, the patient's illness commenced with the climacteric cessation of the catamenia. She then began to vomit blood and be troubled with a tympanitic condition of the abdomen, to complain of pain and tenderness in the left ovarian region, and very shortly afterwards to suffer from persistent retention of urine. In the midst of such symptoms (which continued long afterwards) the patient was seized with a convulsive attack followed by an apoplectiform state, out of which she emerged in the hemiplegic condition above described.

Where you have to do with a hemiplegia, therefore, which appears to present the characteristics of the hysterical variety, always look—before coming to a final decision—not only for the well-marked characters above indicated, but also for the coexistence, either past or present, of some of the distinctive though more general accompaniments of hysteria.



## LECTURE II.

HEREDITARY INFLUENCE — DISTRIBUTION OF EN-CEPHALIC VESSELS — NATURE OF SYMPTOMS IN BRAIN DISEASE—PRODROMATA—APOPLECTIFORM ONSET OF HEMIPLEGIA.

- Influence of Heredity in favouring Hemiplegia—May act through the Nervous System or through the Vascular System—Association of Hemiplegia with chronic Bright's Disease.
- Importance of Knowledge concerning Cerebral Vessels—New Investigations of Heubner and Duret—Distribution of the Basal System of Vessels—Distribution of the Cortical System—Vascular Supply of Cerebellum—Some facts concerning Localization of Cerebral Hæmorrhages—Importance of a Knowledge of Cerebral Convolutions.
- Symptoms in Brain Disease are Direct or Indirect—Conditions which Modify them—Diagnosis must be Two-fold—Regional Diagnosis based upon Regional Symptomatology—Difficulty in building up this latter Knowledge—Its importance in Prognosis—The Physician and the Cerebral Physiologist deal with different Problems—Importance of this distinction in face of Brown-Séquard's Views.
- Premonitory Symptoms—Numerous Variations in—Modes of Onset of Hemiplegia—The Apoplectiform Onset—Meaning of Term—The so-called Simple and Serous Apoplexy—Symptoms of the Apoplectic Condition—Stupor and Delirium—Extent and Suddenness of Lesion almost as important as its Seat in Production of Apoplexy—Nature of Lesion—Lesions with which the Apoplectic Phenomena are most marked.

## LECTURE II.

GENTLEMEN,—The last lecture was altogether devoted to a consideration of the Causes of Hemiplegia, and I entered into some details concerning those Ruptures, Occlusions, and Spasm of Vessels which are the immediately determining conditions in the commoner forms of paralysis from brain disease. There is one other part of the subject, however, to which I must briefly allude before dismissing for a time the question of causation. How far, it may be asked, is hemiplegia an hereditary disease?—that is, how far may a tendency to the common conditions underlying its occurrence be transmitted from parent to offspring?

This question is not so simple as it appears, and it is one concerning which we are still in need of much further information. In the first place, it should be remarked that such a tendency does appear to exist, though if we put on one side the cases belonging to our third category—those in which

Spasm of Vessels and functional disturbances are the efficient causes,—the other common forms of Hemiplegia, due to Hæmorrhage and Softening, are much more likely to be heritable through transmitted defects of the vascular system than through transmitted defects of the nervous system proper. Although there are many facts known which seem to show pretty conclusively that a tendency to hemiplegia is capable of being transmitted from parent to offspring, the proportion of these cases is very small when compared with others in which no such inherited tendency is traceable.

Thus, inheritance may, I believe, come into operation in the production of hemiplegia in two ways. It takes place through the nervous system when, in some of the many cases in which peculiarities of the nervous system itself are transmitted from parent to offspring, we find handed down as one of such peculiarities, an inherent instability of nerve-tissue which favours the occurrence of hemiplegia through spasm of vessels and functional changes. In other cases it takes place through the vascular system. A defective condition of this system is then inherited of such a nature as to favour early degeneration and rupture of the cerebral vessels, producing hæmorrhage, and as a consequence hemiplegia.

But, as I have already hinted, in a very large proportion of the cases of hemiplegia from cerebral hæmorrhage you will not be able to trace the influence of heredity at all; and still less will you be able to trace it in those persons in whom hemiplegia is caused by softening of brain-tissue and its antecedents. A little reflection will make this plain to you. Taking the common causes of softening to be Embolism and Thrombosis, it is at once obvious that embolism is to be regarded as a pathological accident. And, for all practical purposes, we may regard thrombosis of a cerebral vessel in much the same light, if we look to the complex influences which frequently determine it—though in a few cases it may be due in the main to heritable conditions of the vascular system. Enough, however, has been said to show you the impropriety of citing, in a routine manner, the occurrence of hemiplegia as an evidence of inherited defect on the side of the nervous system.

But although we may find the influence of heredity to be slight in the determination of hemiplegia, there is one condition of the body especially with which this set of symptoms is very apt to be associated, and that is chronic Bright's Disease. The tendency to this conjunction of morbid states should not be forgotten. Chronic Bright's disease is, as as you know, very often accompanied by hypertrophy of the left ventricle of the heart, and by an increased vascular tension. Then, again, where a granular condition of the kidney exists, such as so often forms the basis of this affection, we almost always find more or less marked degeneration of the vascular system. The co-existence of these conditions strongly predisposes to the occurrence of cerebral hæmorrhage, as one of the many pathological accidents or sequelæ prone to reveal themselves in the course of chronic Bright's disease.

Vascular Supply of the Brain.—What I have already said to you concerning occlusions of vessels by embolism and thrombosis, as causes of hemiplegia, will have been sufficient to show the great importance of studying the mode of distribution and the degree of inosculation existing between the several vessels of the cerebrum and of the cerebellum. Knowledge of this kind will prove very useful to you, not only in explaining the extent and distribution of foci of softening, and the relative frequency with which it implicates different parts of the brain, but also in accounting for some of the associations of symptoms met with at the bedside. Some light is also thrown by it upon the cause of the frequency

of hæmorrhage in particular sites, and upon the variations in amount of blood poured out. I have, therefore, no hesitation in calling your attention to the following anatomical details, more especially as some important additions to our knowledge in this direction have of late been made by Dr. Heubner and by M. Duret.

Dr. Heubner's results have been founded upon injections of the different arterial regions with coloured fluids, in thirty human brains. He divides the entire arterial circulation of the encephalon into two systems-a basal and a cortical. The basal system is represented by the circle of Willis, and the principal trunks of the cerebral arteries for a distance of one-third of an inch to one inch from this circle. From these trunks numerous very small vessels pass off at an acute or right angle, and sink almost immediately into the various important ganglia at the These vessels are true terminal base of the brain. or 'end' arteries, which do not anastomose, so that their distribution can be accurately determined by means of coloured injections.

The major part of the intra-ventricular portion, or nucleus caudatus, of the Corpus Striatum is usually supplied by a small vessel from the anterior cerebral, though occasionally by filaments from the choroid artery of the posterior cerebral; but, with this

exception, the blood-supply to the corpus striatum is derived from numerous small twigs given off from the first two-thirds of an inch of the middle cerebral artery. Thus an embolon lodging, or a thrombus forming, in this situation almost completely deprives the corpus striatum of its blood-supply. On the other hand it is a rupture of some of these vessels in the white matter just outside the corpus striatum, which gives rise to the large hæmorrhages occasionally occurring in the centrum ovale. The mass of the white matter itself is only supplied by small vessels, so that hæmorrhages occurring in it nearer the convolutions rarely exceed a small almond in size. There is one important exception to this statement, however, concerning the posterior lobe. For as M. Duret has pointed out, larger hæmorrhages do occur in the midst of the white substance in the occipital region, owing to the fact that the posterior cerebral artery gives off large branches in the calcarine fissure, which almost immediately penetrate into the adjacent white substance.

The Optic Thalamus is, however, nourished in a different fashion. Only the anterior and external portions of the thalamus are supplied from the middle cerebral artery,—that is, by prolongations of some of the twigs which go to the lenticular nucleus of the corpus striatum. And when these vessels are

ruptured, we get a hæmorrhage involving the posterior part of the corpus striatum, and the anterior part of the thalamus. The posterior portions of this ganglion are supplied by rather large filaments from the posterior cerebral, whilst its anterior and internal portions, together with the soft commissure, are supplied by small branches passing off from the anterior communicating artery, which almost follow the course of the anterior pillar of the fornix.

Rupture of one of the posterior vessels of the thalamus often gives rise to a rather large hæmorrhage, which besides tearing up the end of this body extends into the cerebral peduncle of the same side, and occasionally reaches even to the pons. Again, the triple source of blood-supply of the thalamus might lead us to expect that softening would invade this ganglion less extensively than it does the corpus striatum, and also that it would be absolutely rarer in the thalamus, because this body is in the main dependent upon the anterior communicating and the post-cerebral arteries—vessels which are much less frequently occluded than the middle cerebrals. I turned, therefore, with some interest to the valuable statistics collected by Gintrac, and found this anticipation to be well grounded. In a total of 60 cases of softening of these parts of the brain (ranged by

him partly under the head of 'ramollissement' and partly under 'encephalite'), the corpus striatum was implicated 39 times, one corpus striatum and one optic thalamus together in 9 cases, and one optic thalamus alone only in 12 cases.

But, looking to other important parts at the base of the brain, I must mention that the Crura Cerebri and the Corpora Quadrigemina are also almost wholly supplied with arterial twigs from the basal system. Numerous minute vessels for the supply of these parts pass off from the commencement of the posterior cerebral artery. Some penetrate more or less immediately into the crus, whilst others proceed to the corpora quadrigemina, which are very richly supplied with blood by means of three arteries on each side—the two anterior of these being derived from the posterior cerebral, though the third or hindermost vessel comes off from the superior cerebellar artery. Thus, in certain cases of softening of the posterior lobe of the brain from thrombosis of the posterior cerebral artery, we have the association of blindness of one eye occurring on the hemiplegic side; and I need scarcely say that this is an association having an important diagnostic significance when the onset of the unilateral blindness is sudden and synchronous with paralysis of the limbs.

The pons Varolii\* is also very richly supplied with minute vessels, which penetrate into its substance from the basilar artery—some directly along the middle line, and others, after a short course, on either side into its lateral regions. An extensive thrombosis in this artery is particularly apt to give rise to severe apoplectic symptoms proving rapidly fatal; and the same clinical consequences are produced by any large effusion of blood into the substance of the pons from rupture of one or more of its many mesial twigs.

The cortical system of vessels commences where the principal trunks of the anterior, middle, and posterior cerebral arteries begin to divide into branches of the second order. The vessels of this system supply nearly the whole of the convolutional grey matter of the cerebrum and its related white substance in the hemispheres. Its smaller vessels pass off from the larger ones nearly in the direction of the blood-stream, and, subdividing again and again, they pursue long courses through the pia mater whilst multitudinous branchlets pass almost

<sup>\*</sup> It may be as well to state here that I use this name in the sense in which it is usually employed by English writers, as equivalent to the whole 'mesocephalon' or 'annular protuberance,' though the name is now often more strictly limited to the superficial transverse fibres of this body.

perpendicularly from larger and smaller trunks into the cortical substance.

With regard to the extent and freedom of anastomosis between the branches of the three main arteries in their distribution to the cortical grey matter, further researches seem needed. According to Dr. Heubner, such anastomoses are free and abundant, whilst according to M. Duret the inosculations are few and very minute. The fact that in many cases of occlusion of the middle cerebral artery, softening does not invade any large part of the territory of this vessel, but may be limited to the corpus striatum and to its immediate neighbourhood—that is, to parts supplied by the terminal twigs of the basal system—would seem to show that in many cases, at all events, the inosculations are pretty numerous and easily opened up between the cortical branches of the middle and those of the anterior and posterior cerebral arteries.

The distribution of these vessels is as follows:—
The middle cerebral artery divides in the Sylvian fissure into four or five branches, which in their course over the island of Reil send numerous twigs into its substance. One of these branches supplies the tip and the anterior and upper part of the temporo-sphenoidal lobe, another supplies the third frontal convolution, whilst the remaining branches

are distributed anteriorly to the second and part of the first frontal convolution, and posteriorly to the parietal convolutions, to the supra-marginal lobule, and thence onwards to the annectant gyri (Fig. 1). Thus the branches of the middle cerebral supply almost all the convolutions on the outer aspect of the hemisphere. They anastomose anteriorly and along the inner margin of the hemisphere, skirting the great longitudinal fissure, with offsets from the anterior cerebral-a vessel which supplies the convolutions of the orbital surface of the anterior lobe, as well as those of the inner surface of the hemisphere as far back as the internal parieto-occipital fissure. Lastly, branches of the posterior cerebral artery are distributed to the under and posterior part of the temporo-sphenoidal lobe, where they anastomose with twigs of the middle cerebral. And the main trunk turns round to the inner side of the hemisphere, in a position corresponding with the internal parieto-occipital fissure, whence its ultimate branches are distributed over the occipital lobe, anastomosing externally with terminal ramifications of the middle cerebral and internally with those of the anterior cerebral artery.\* All these details I have myself recently verified.

<sup>\*</sup> See Figs. 7—9, where the distribution and nomenclature of these vessels of the cortical system are given after Duret upon diagrammatic representations of the convolutions after Ecker.

I have only to add that the little brain, or Cerebellum, is also supplied by three vessels on each side, which freely anastomose on its surface, and thence send multitudes of minute twigs into its substance. The largest or posterior inferior cerebellar artery arises from the end of the vertebral or from the basilar, and supplies branches to the under surface and to the posterior part of the upper surface. The anterior inferior cerebellar is given off from the middle of the basilar artery, and supplies the anterior part of the under surface of the organ; whilst the superior cerebellar artery is derived from the basilar close to its termination, and passes backwards to the upper surface of the cerebellum, over which it is distributed. Here it anastomoses with its fellow of the opposite side and with the two inferior cerebellar arteries on the same side.

Very large hæmorrhages are occasionally met with in the cerebellum, and these, as M. Duret points out, are due to the rupture of a large artery, derived from the superior cerebellar, which enters the grey nucleus (corpus rhomboidale) on each side by its hilum, and there breaks up into radiating branches for the supply of this organ and of the white substance around. Ruptures of this artery may produce so large an extravasation of blood that it occupies

the whole of the corresponding lobe, or, more rarely still, nearly the whole of the cerebellum. A rupture of one of its radiating branches, however, for the most part gives rise to a small extravasation contained within the rhomboidal body itself. Hæmorrhages into the white substance outside this body rarely exceed an almond in size.

The superior and the inferior peduncles of the cerebellum only contain vessels a little larger than capillaries, and are very rarely the seats of hæmorrhage. The middle peduncle is, however, supplied with larger vessels, derived almost exclusively from the middle cerebellar artery, and here larger extravasations of blood are also occasionally met with, which, following a longitudinal direction, separate its fibres, and may reach as far as the pons.

A familiar knowledge of purely anatomical and practical details like these, gentlemen, will often prove serviceable in helping you towards an accurate regional diagnosis. And again, after death, it is only by keeping such facts well in mind, and by minute attention to the particular convolutions or parts of the brain implicated (with the view of comparing lesions with symptoms), that we can hope to advance our knowledge of regional symptomatology in brain disease, and thus obtain an intelligent insight

into the meaning of those most varied groups of symptoms which frequently prove so perplexing when we stand by the bedside of our patients. But I must pass now to a detailed consideration of these symptoms, and to certain preliminary remarks.

## SYMPTOMATOLOGY.

The symptoms or signs met with in brain disease are very variously produced. Some are due to stimulation or irritation of a portion of brain-tissue; others are produced by the destruction of braintissue; and in each case these symptoms may be classed as 'direct.' In other instances an injury to one portion of the cerebrum or cerebellum, besides giving rise to its own set of direct symptoms, may also produce symptoms of a stimulating or of an inhibitory type upon more or less remote parts. And such 'indirect' effects or symptoms may occasionally be of a more obvious nature than the direct effects. They may be brought about by simple mechanical means, as by pressure, or may be occasioned in a so-called 'reflex' manner under the influence of structural or functional relationships, the precise nature of which we are often unable to fathom.

But, however produced, the groups of symptoms actually met with in different cases of hemiplegia are variously dependent upon the situation, the extent, and the suddenness of the lesion, although it is often extremely difficult, if not impossible, for us to unravel the respective influences of these three factors. The nature of the lesion is a question of subordinate importance for our present inquiry, because almost the same set of symptoms may be produced by the destruction of a given region of the brain by hæmorrhage, as by the cutting off of its bloodsupply through embolism or thrombosis. Thus all cases of brain disease have to be looked at from a double point of view, and our diagnosis must be twofold. As Dr. Russell Reynolds pointed out twenty years ago, we must first endeavour to ascertain the 'locality' of the lesion, and then inquire as to its 'nature.' No one has worked more steadfastly in this direction than Dr. Hughlings Jackson, and both the principle and the practice are now being very generally recognised and adopted.

If the first part of our diagnosis deals with the consideration of the region implicated, this can only be based upon the cumulated results of numerous careful observations. The signs and symptoms met with have, in fact, to be interpreted by clinical and pathological experience as well as by the some-

what obscure light derived from cerebral anatomy and physiology. This part of the subject is therefore beset with intrinsic difficulties of a most baffling description. Advance, and that at a very slow rate, can only be made by accurate clinical observation, going hand in hand with the most careful post-mortem examination of the brain, wherever the opportunity occurs. But the difficulties experienced in following up these indications are very great. In cases with well-marked or well-defined groups of symptoms, the patients often either recover or lapse into a chronic stage, and after a time disappear from observation. And even where pathological do succeed clinical observations, we may not be able to draw any positive conclusionseither owing to the co-existence of several lesions, or because—where a single region only is affected —the last stage of the injury may be such as to blot out all accurate traces of the nature and extent of the earlier pathological conditions with which, perhaps, the most definite symptoms may have been associated.

Again, we not unfrequently labour under the still further disadvantage of allowing lesions to escape our observation—both in instances in which the brain may appear to be sound and in others where some obvious lesions are discovered. In an organ of

great extent and complexity like the brain, we cannot always detect a mere patch of degeneration, and still less can we tell when minute vessels supplying important regions of the brain are occluded, if no sufficient time has elapsed for a focus of softening to manifest itself. The fact also of the existence of two almost similar cerebral hemispheres renders it often still more difficult to draw conclusions as to the physiological effects of unilateral superficial lesions, because, to a not inconsiderable extent, the functions of such damaged parts may be carried on by corresponding portions of brain-tissue in the opposite hemisphere.

With numerous difficulties of this nature besetting our path, progress in the regional symptomatology of brain disease might almost be despaired of. And yet we have made the most solid advances during the last thirty years in our knowledge of this part of the subject, and also in our acquaintance with the various phenomena connected with or which go to make up the hemiplegic state.

In order to form an opinion as to the effects pertaining to lesions of different parts of the brain, it is our duty to study the particular association of symptoms in different cases—that is, the many modes in which symptoms may be grouped; and if we find that some particular grouping is especially

apt to be met with when a given region of the brain is injured, and that certain other groupings occur having distinctive characters of their own when different portions of the organ are implicated, we obtain data for diagnosis such as we require. We may as a consequence be enabled to form conclusions respecting our patient which will be of great value from the point of view of prognosis, and therefore in enabling us to reply with more certainty to the anxious inquiries addressed to us by friends of the patient.

We, as practitioners of medicine, are indeed in a measure independent of those difficult questions which at present perplex the cerebral physiologist questions as to whether the symptoms in cerebral lesions depend upon the mere abolition or stimulation of function of the part involved, or whether they are due to a stimulating or inhibitory effect upon functions of distant parts, brought about either by obscure reflex agencies, by unknown anatomical communications, or by pressure indirectly occasioned. We are at least compelled to believe that some of the symptoms in brain lesions are directly produced by stimulation or arrest of function; and we surely have an equal right to believe that any indirect symptoms occasioned in one case would also be apt to recur in subsequent cases where the injuries

were similar. Like causes should produce like results—and this dictum ought, of course, to hold good for the 'indirect' as well as for the 'direct' effects of brain lesions.

I have been led to make the foregoing remarks, gentlemen, in consequence of recent discussions as to the explanation of the important experimental results obtained by Hitzig Ferrier and others; but more especially in view of the recently expressed opinions of Brown-Séquard concerning the frequency and importance of the 'indirect' symptoms occasioned by brain lesions. It seems to me that on this occasion Brown-Séquard has been speaking to physicians concerning points in practical medicine from the point of view of a physiologist, without giving them the necessary warning that his speech was thus limited. And, moreover, from the lack of a few explanatory statements, many of his readers might suppose him to believe, on the one hand, that no 'direct' effects were produced by lesions in portions of brain-tissue, and that the 'indirect' effects upon which he lays so much stress make their appearance without any definite plan or order. This, however, is surely far from being the view which Brown-Séquard would wish to impress upon his readers.

After these more or less necessary introductory

remarks, gentlemen, we now pass on to a detailed consideration of the modes of onset of different hemiplegic states and of their symptomatology.

Premonitory Symptoms.—These are often absent altogether, especially in instances of embolism or hæmorrhage, though in many cases belonging to the latter category, and in still more of those in which the hemiplegia is due to thrombosis, prodromata may manifest themselves for days and even weeks before the actual onset of the attack. The explanation of this is to be found in the fact that rupture of very minute vessels or alterations in blood-supply in different parts of the brain may precede, for a longer or shorter period, that graver event which ultimately suffices to establish the hemiplegic condition.

These forerunning symptoms are more apt to present themselves in the old or middle-aged than in younger persons, and when they do occur, they may be very various in nature. Vertigo, headache, drowsiness, confusion of mind, defective memory, noises in the ears, defects of sight, numbness in the hand or foot, muscular twitchings of the face or of some portion of the upper or lower limbs (especially of the fingers or toes), mistakes in talking or writing, difficulties in articulation, vomiting—some

of these signs may exist variously combined, in different cases, as forerunners of the hemiplegic condition, though of course they may also occur, at times, without any such grave sequence as a paralytic attack. None are to be relied upon before others as especially indicative of an approaching hemiplegic affection; neither does the presence of either of these signs foreshadow anything definite as to the nature or severity of an approaching attack. Such symptoms may, in fact, as I have already indicated, be wholly absent before the gravest of hemiplegic seizures, or they may be well marked and long continued where slight attacks subsequently supervene. When such symptoms do occur, however, they are alarm-signals which should always receive our attention, since, by warning us as to what may follow, they occasionally enable us to do something to avert an otherwise impending attack.

Modes of Onset.—The actual mode of onset of hemiplegia differs very much, though for facility of description I shall divide the cases into three principal categories. They are (1) the Apoplectiform Onset, in which there is more or less sudden loss of consciousness with resolution of limbs; (2) the Epileptiform Onset, in which there is loss of

consciousness plus convulsions; and (3) the Simple mode of Onset, where there is neither loss of consciousness nor convulsions. This seems to me to be the most useful kind of division, although you must understand that many cases are to be met with of an intermediate character which are not capable of being definitely ranged under either of these categories.

I. Apoplectiform Onset.—This is the mode of onset popularly know as a 'fit of apoplexy.' Taken in its proper and original sense, the word 'apoplexy'-as used by the early Greek writers was meant to imply a sudden loss of consciousness and volition, with more or less disturbance of the circulation and respiration. A certain vagueness is inseparable from this use of the term, because the state itself, as increasing knowledge soon showed, is one which may be induced in various ways. But when, in the early part of this century, certain physicians began to use the word 'apoplexy' as synonymous with hæmorrhage into the brain, simply because such hæmorrhage was a very frequent cause of apoplexy, the way was paved for an actual abuse of this term which was decidedly objectionable. Hæmorrhages into other organs came to be spoken of as 'apoplexies,' and thus the original meaning of the term was entirely lost

sight of by those who used it in this illegitimate manner.

Whilst restricting ourselves to the use of the term in its original sense, however, we must always remember that the assemblage of symptoms included under this word 'apoplexy' may be very variously produced, and may or may not be followed by a condition of hemiplegia. On the different causes of the apoplectic condition I cannot now dwell-it would require a separate clinical lecture to do even faint justice to such a subject. I will only beg you to remember that most of the cases which in former days were spoken of as 'serous' apoplexy or 'simple' apoplexy (because no satisfactory cause for the grave symptoms present during life could be subsequently recognised in the brain), were instances of death in an epileptiform attack of the kind spoken of by Trousseau under the name 'apoplectiform cerebral congestion,' or they were cases of multiple and wide-spread embolism of minute arteries in which incipient softening of large extent was overlooked, or else they were instances of death during uræmic convulsions and coma.

In a small number of cases the onset of Apoplexy may be absolutely sudden, though as Trousseau insists, in a much larger number of cases it supervenes upon slighter symptoms which have preceded it by a few minutes, or even by an hour or more. When the apoplectic attack is well marked the patient drops into a condition of profound coma, and is insensible to stimuli of all kinds. His senses are for a time extinguished. The face is often flushed and swollen and the lips livid, though at other times the face may be pale and clammy; the head and neck feel warm, and the carotid and other arteries throb violently; the eyelids are closed, the conjunctivæ injected, and the eyeballs immoveable, with the pupils sluggish, though variable as regards their state of contraction; the respiration is generally deep and regular, with or without stertorous inspiration and protrusion of the cheeks during expiration; the pulse is usually full and not frequent; the limbs when raised drop like inert bodies, so that there is either complete muscular resolution or else the powerlessness is more marked on one side of the body than on the other.

Described in general terms, these are the classical symptoms of the apoplectic condition, and in this state, where the condition itself has been brought about by some severe lesion of the brain, the patient may die after a few minutes, a few hours, or a few days. On the other hand, in slighter

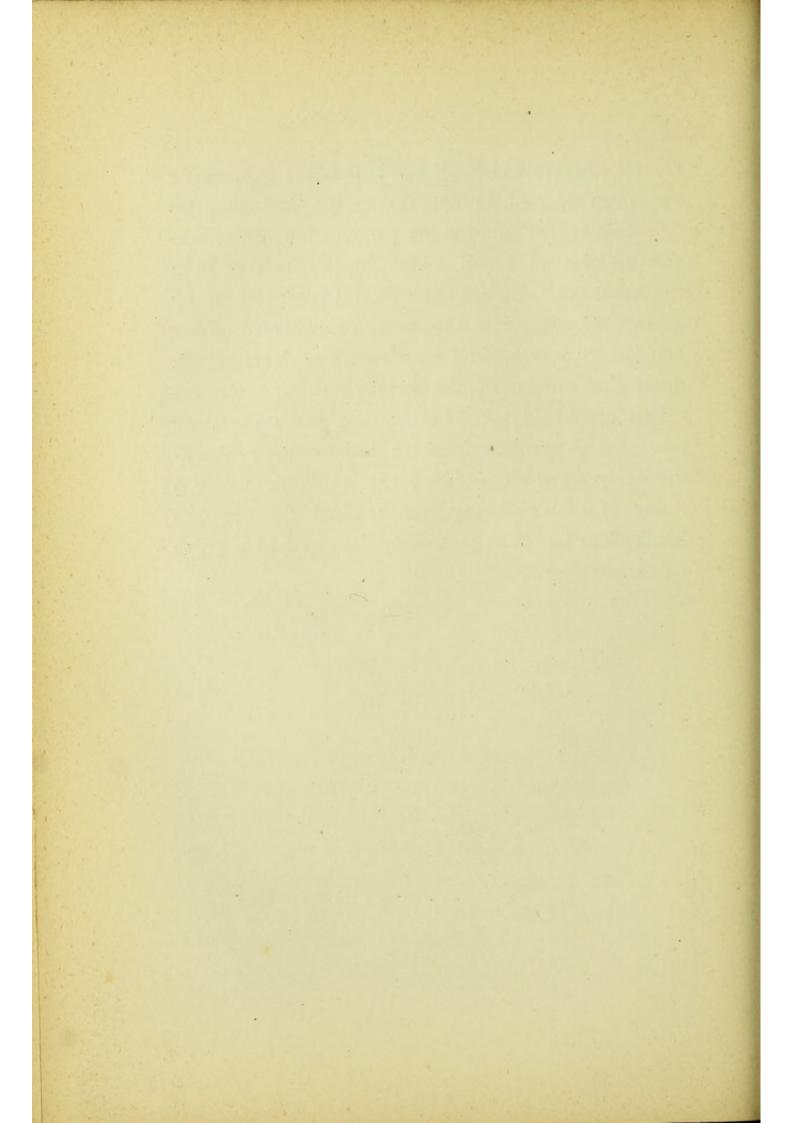
cases, the apoplectic condition may last only half an hour or less, or it may gradually pass off after a few hours; and in these less severe cases other related symptoms manifest themselves. For you must understand that consciousness and that higher directive power, by which the patient not only receives impressions and replies to questions but also directs his thoughts upon particular subjects, may be lost in various degrees. We may recognise, in fact, every kind of gradation between the profoundest coma and a mere slight mental incoherence-the gap being filled up by various states of stupor, of delirium, and of mental wandering. For instance, where the coma is not very profound, the patient, when loudly spoken to, raises his eyelids for a moment or two, though he perhaps takes no further notice; or he may reply in a monosyllable when loudly pressed with any question, and then again almost instantly lapse into his condition of stupor. Should the insensibility be still less profound, the patient's replies may be longer, though still incomplete, vague, and slow. At other times such patients are restless and more or less delirious-especially where we have to do with slight hæmorrhages upon the surface of the brain or with changes in its cortical grey matter.

Turning now to a very brief consideration of the

conditions under which this apoplectiform mode of onset presents itself, at once let me say that this set of symptoms, of which loss of consciousness is the chief, is as much dependent upon the extent and suddenness of the lesion as upon the particular region affected. The occurrence of the apoplectic condition, therefore, of itself gives us only a vague clue as to the region of brain injured. A large and sudden outpouring of blood, for instance, into almost any part of the brain-substance may give rise to apoplectic symptoms; whilst, on the other hand, a small effusion, more slowly poured out, may occur in any situation without entailing loss of conscious-You must not forget, moreover, that the sudden and complete stoppage of one of the large cerebral arteries by embolism may give rise to much the same effects as a sudden moderate outpouring of blood into the vascular area of such an artery.

But although we are not able to say that the injury of any given region of the brain always or necessarily entails loss of consciousness, still past experience does enable us to say something as to the conditions with which these symptoms are most frequently associated. The region which above all others, when extensively damaged or cut off from its blood-supply, gives rise to profound coma is that

central knot to which all cerebral fibres converge—the pons Varolii. More frequently, however, we find this condition of coma produced by an extensive effusion of blood, which breaks its way from the brain-substance into the lateral ventricles, or by a large bleeding into the centrum ovale of either hemisphere; or again by an extensive hæmorrhage upon the surface of the hemisphere into the so-called arachnoid sac. Lastly, the same comatose condition is produced where lesions occur simultaneously on both sides of the brain, or where a lesion of one side supervenes when the opposite hemisphere has been previously damaged to a grave extent.



## LECTURE III.

EPILEPTIFORM ONSET.—TEMPERATURE VARIATIONS IN APOPLEXY.—CONJUGATED DEVIATION OF THE EYES. — SIMPLE ONSET. — TYPICAL HEMIPLEGIC STATE.

- The Epileptiform Onset—Two Classes of Cases to be distinguished—
  Prodromata—Illustrations of this mode of Onset—Terminations
  —Convulsions Unilateral or Bilateral—Paralysis and Convulsion
  on opposite sides of the Body—Andral's Explanation of this—
  Prolonged Stupor—Relation of Epileptiform Onset to Lesions of
  Surface of Brain—Its Occurrence with Lesions in other situations.
- Temperature Variations in Different Stages of the Apoplectic Condition
  —Bourneville's Observations Period of Initial Lowering—
  Stationary Period—Ascending Period.
- Other Signs met with in the Apoplectic State—Conjugated Deviation of the Eyes with Rotation of the Head—Relationship existing between these Signs—Description of the State—Its Diagnostic Value—Its usual Duration.
- Simple Mode of Onset of Hemiplegia—May occur during Sleep—May not be quite Sudden—Pain and other Sensations as Initial Symptoms—Variations in mode of Onset.
- Typical Hemiplegic State produced by Lesion of Corpus Striatum— Progress of Case where favourable—Paralysis of Face in Hemiplegia—Paralysis of the Limbs—State of Nutrition and Electric Irritability of the Muscles.

## LECTURE III.

GENTLEMEN,—We turn now to the consideration of another mode of onset of the hemiplegic state—

(2) The Epileptiform Onset.—This is little more than a variety of the apoplectiform mode of onset which we considered in the last lecture. When seized in this manner the patient drops down, either with or without prodromata, not merely insensible, but actually convulsed. He has, in fact, a kind of epileptic fit or a series of them, though he may never have had one before; and after a time, when consciousness is restored, or perhaps sooner, it is discovered that the patient is paralysed on one side of his body.

This mode of onset of hemiplegia is interesting in many respects, but principally on account of its complicated relations with the epileptic condition. You must not forget, however, that there are two distinct classes of cases included under this head. There are, first, the cases referred to in a previous lecture, in which a very brief and temporary hemiplegia is apt to follow certain severe attacks of unilateral convulsions; the hemiplegic condition in these cases being due merely to vascular spasms plus certain minute but undiscoverable molecular changes in definite parts of the brain. Secondly, there is the much more formidable group of cases in which we have to do with distinct and obvious organic lesions in the brain, giving rise both to the epileptiform mode of onset and to the more persistent hemiplegic condition thus initiated.

There is no reason, of course, why this second state of things, due to the occurrence of a new and obvious brain lesion, should not take place in persons who have been previously epileptic—even although, in their case, an epileptic fit has never previously been followed by hemiplegia. Actually, however, this mode of onset occurs more frequently in those who, previous to the commencement of the paralysing lesion, had not been epileptic—simply because such persons are so much more numerous than those who have been subject to epileptic attacks.

Although in this class of cases prodromata may be absent altogether, or, if present, may be most variable in nature, still I think that two sets of symptoms, either singly or in concert, are especially not to precede the epileptiform mode of onset:

these are, pains in the head and muscular twitchings in different parts of the body. Where they have been present a fit may subsequently occur, and (though the person has never previously had one) this may be followed by other epileptiform attacks at short intervals, after one of which the patient is discovered to be hemiplegic. A good instance of this mode of onset occurred in a patient who was under my care a few years ago.—A woman, thirtyfour years of age, had a first fit whilst in the street; but she was able to walk home, and during the next forty-eight hours she had twelve other attacks. She was then brought to this hospital, and was not at the time paralysed; but soon after her admission she had another series of epileptiform convulsions affecting both sides of the body, though the twitchings were more marked in the right limbs and on the right side of the face. During the intervals between these latter convulsive attacks it was found that the face was partially paralysed on the right side, that the right arm was completely paralysed, and that the leg was powerless to a less extent. The convulsions then ceased, but the patient remained hemiplegic for several months, and probably longer, though she was after a time lost sight of.

In another patient, a girl only eight years old, prodromata occurred in the form of a slight

maniacal condition. This was brought to a close at the expiration of three weeks by the occurrence of four fits, one after another in quick succession, in each of which there was a general convulsive condition. The patient was then found to be paralysed on the right side of the body, and the paralysis persisted for more than twelve months. In this child there was hypertrophy of the heart, together with loud basic systolic and diastolic murmurs. initial attack may be characterised by unilateral convulsions. An instance of this occurred in a woman who was under my care at the National Hospital for the Paralysed and Epileptic. She was forty-two years of age, and, whilst dressing, was suddenly seized with convulsions affecting the left limbs and face. She became unconscious at once, and the convulsions continued, with occasional intermissions, for an hour and a half. She was then found to be paralysed on the left side, and she remained in a more or less unconscious state for fourteen days. The paralysis persisted, and her speech continued indistinct, for a long time.

Some of the patients whose hemiplegic condition is initiated in this manner speedily die, whilst others are subsequently liable to a recurrence of epileptiform attacks at variable intervals. In many cases, however, no subsequent attack occurs, even though

the patient may live for many years: from which we may conclude that the initiating epileptiform symptoms were probably due to the early irritative stage of a brain lesion which by its rapid advance leaves no tendency to the recurrence of similar epileptiform attacks. The early pathological state, in fact, becomes blotted out by the fully developed lesion by which it is more or less immediately followed and upon which the persistent paralysis depends.

As we have seen, the convulsions in this mode of onset of hemiplegia may be general—and this is the rule, — though more rarely they are limited to one side of the body. This unilateral limitation is most frequent in cases, associated with spasm of vessels, such as I described to you in the last lecture, and which, with the view to brevity, I may now class under the term "functional hemiplegia." And where the convulsion does occur wholly or principally on one side of the body, this side, in the great majority of cases, corresponds with that which is either known at the time or subsequently found to be paralysed.

Where convulsions occur, however, after paralysis has already declared itself, it occasionally happens that the non-paralysed side is the one which is agitated or affected with clonic spasms. But the rule laid down by Andral in explanation of such

cases is decidedly that which finds most support from a careful scrutiny of the facts. He maintained that convulsion occurring on one side of the body with paralysis of the other is indicative of a co-existing though unequal damage to both hemispheres of the brain. There is a grave lesion in one hemisphere causing the hemiplegia, and a slighter lesion in the opposite hemisphere giving rise to the convulsions or spasms of the non-paralysed side—spasms which may affect the whole of the side or only one part of it, such as the face and the arm or the arm alone.

I could cite many cases in support of this view—cases especially in which the second or minor lesion has occurred in or upon the surface of the brain. And here slight hæmorrhages or softenings of the cortical grey matter, or limited patches of meningo-cerebritis, will be the coexistent conditions most frequently met with as the causes of convulsion on the opposite non-paralysed side of the body. Occasionally, however, symptoms of this type are also produced by hæmorrhages into the ventricles, when the effused blood irritates the corpus striatum of the opposite side. Where such spasms are of a partial character and dependent upon a superficial lesion, their appearance is not necessarily attended by any loss of consciousness, though as a matter

of fact they most frequently manifest themselves during the early stages of the disease, at a time when the patient may be still in a state of stupor.

And this reminds me that there is another peculiarity of the class of cases with an epileptiform mode of onset to which I ought to direct your attention -namely, the frequency with which very prolonged periods of stupor or partial unconsciousness are met with. I have already mentioned (p. 68) a case of hemiplegia thus initiated in which such a condition persisted for fourteen days, and the period is occasionally even much longer than this. The condition is, however, far from being one of complete coma. The patient may remain in a dull, lethargic state, with eyes closed or only half open, taking no notice of anything that is going on around. He does not always lie still, however. There is often much restlessness, whilst the pulse may be slow, and beating perhaps only about 60 per minute. After a time he begins to display more signs of intelligence in look and manner. His attention may now be slightly arrested; efforts to speak are made, and the full possession of consciousness is gradually regained.

Now this kind of stupor is quite different from coma in more ways than one, though the difference is more especially notable from a prognostic point of view. A patient who continues deeply comatose for forty-eight hours only very rarely recovers; whilst, on the other hand, the state of stupor following the epileptiform mode of onset may exist for three, four, five, or even six weeks, and yet the patient may ultimately do well. You know, however, that an attack of epilepsy, and more especially a rapid succession of epileptiform attacks, even where no paralysis is produced, is not unfrequently followed by stupor for many hours or even for several days. And now we find that where a somewhat similar 'status epilepticus' occurs in an individual whose brain is at the same time damaged so seriously as to entail a lasting hemiplegia, the condition of stupor may be much more remarkably prolonged—that is, it may last even for weeks.

As to the cause of the epileptiform mode of onset little that is definite can be said. We cannot say why lesions should at one time produce effects of an irritative nature leading to the evolution of general or partial epileptic convulsions, whilst apparently similar lesions in other cases produce no such results. Where such differences are met with, however, there may be slight differences in the area involved, or others due to the mode of commencement of the lesion. At all events, this mode of onset is not strictly related to the occurrence of lesions in any

one part of the encephalon: it may present itself where the most dissimilar regions are injured.

Clinical experience, however, suffices to show the very great frequency with which convulsions occur in association with lesions of the surface of the hemispheres, as compared with their frequency when injuries exist in other encephalic regions. An extensive study of the literature of the subject suffices to confirm this view, which was definitely announced by Dr. Wilks, in 1865, and which has of late, moreover, been still further strengthened by the admirably worked out conclusions of Dr. Hughlings Jackson as to the almost invariable association of epileptiform attacks, cephalagia, and double optic neuritis in cases where tumours or new growths implicate the cortical substance of the hemispheres. The connexion between convulsions and lesions of the surface of the brain is still further supported by the highly important experimental results which have been obtained by irritating different portions of the cortical grey matter by Fritz and Hitzig, and more recently and thoroughly by Dr. Ferrier. For, however difficult it may be to find the correct interpretation of these latter experiments-that is, to explain them in accordance with physiological principles—the facts themselves have great significance. From what I said to you in the last lecture

you will understand that we, as practitioners of medicine, may appropriate the facts as they exist, since we are in a measure rendered independent of those more subtle considerations which must necessarily weigh with the physiologist in his search after their interpretation. Looked at from a professional point of view, the simple question for us is—Does irritation of the cortical grey matter on the upper and outer part of the cerebral hemispheres tend to produce convulsions? And this question may now undoubtedly be answered in the affirmative.

If I could end here, gentlemen, you would be left with a simple rule for your future guidance, concerning the region involved in the class of cases to which we are now directing our attention; but unfortunately this cannot be done. As it was with regard to the apoplectic condition, so now I am compelled to say that the epileptiform mode of onset may be met with in association with injuries of almost any part of the encephalon. Lesions in the substance of the hemispheres, in the opto-striate bodies, in the crura cerebri, in the cerebellum, in the pons Varolii, and in adjacent parts, may each occasionally give rise to hemiplegia with an epileptiform mode of onset. Of these various encephalic regions, however, it is the pons Varolii whose injury, next to that of the cortical substance of the hemispheres, is perhaps most prone to be associated with an epileptiform attack at the onset. Where we have to do with injuries in other situations such symptoms are only occasionally encountered, though, as Mr. Callender has pointed out, they seem very frequent with large lesions of the white substance of the right hemisphere just outside the thalamus and corpus striatum.

The occurrence of an epileptiform mode of onset therefore cannot alone guide us with any certainty to the first stage of our diagnosis—namely, the discovery of the region affected. The particular combination of symptoms present—the mode in which convulsion is combined with other symptoms—must be carefully studied before we are entitled to come to any conclusion on this important question.

Before proceeding to a consideration of the third or simple mode of onset of hemiplegia, I ought to direct your attention to certain important modifications of the temperature of the body, and of the pulse and respiration, which have been observed during the apoplectic state, as well as during the more or less profound stupor following an epileptiform onset of hemiplegia; and also to an important sign known by the name of 'conjugated deviation' of the eyes, which is often met with at the same period. These data we owe almost entirely to the

careful observations of MM. Charcot and Vulpian and their pupils, at the Salpêtrière, who have fully utilized their excellent opportunities for carefully studying the clinical phenomena of the apoplectic condition and its sequential states. Important facts in regard to temperature more especially have been recently embodied and published by Bourneville, one of the most distinguished of M. Charcot's former pupils. These investigations are all the more valuable because of the scanty and vague information hitherto available on this subject.

## TEMPERATURE VARIATIONS IN THE APOPLECTIC CONDITION.

Looked at from the point of view of the variations in temperature that are to be met with, Bourneville divides cases of Apoplexy due to cerebral hæmorrhage into four categories. (I) Cases produced by very large cerebral hæmorrhages, or in which two or more bleedings rapidly succeed one another. Here death often occurs in an hour or two, and during this period the temperature of the body is decidedly lowered. (2) Cases in which death takes place in ten to twenty-four hours, and where the tempe-

rature is at first lowered, though the temporary depression is soon followed by a rapidly increasing rise of body-heat. (3) Cases terminating in death at the end of a few days. Here there is (a) an initial lowering of temperature; (b) a stationary period in which, after regaining its physiological standard, the temperature of the body undergoes slight oscillations above and below this point; and (c) an ascending period in which the temperature steadily rises. (4) Cases in which the patient recovers. Here we have an initial lowering of temperature, as in the other divisions, followed by a temporary rise and succeeded by a pretty speedy return to the normal standard.

Now, taking as our type the cases which belong to the third category, I shall be able to lay before you the facts ascertained by Bourneville concerning the several thermometric stages and the modifications of pulse and respiration which they entail, or which, at all events, accompany them. These details refer to temperatures taken in the rectum, and they have to be studied for each of the three periods—for the period of initial lowering, for the stationary period, and for the ascending period of body-heat.

(a) Period of initial lowering.—When the temperature is taken about a quarter of an hour after the onset of the apoplectic attack, it is usually found to be already below the normal standard (probably at about 97½°), though there may be no notable alteration in the character of the pulse or of the respiration. Occasionally the fall of temperature takes place a little later, but in either case it may sink as low as 96°. Any recurrence or continuance of the bleeding into or upon the brain causes the temperature to remain for a longer period at this low point, or will again depress it should it have begun to rise. During this period all the more familiar apoplectic symptoms are well marked—coma, resolution of limbs, stertor, with or without occasional vomiting and relaxation of the sphincters, are to be met with.

(b) Stationary period.—In an hour or two, should the patient survive, we may begin to get more distinct evidences of the hemiplegic condition. The state of coma passes perhaps into one of stupor, in which the patient is less profoundly insensible. The alterations in temperature, however, now to be described may still supervene even where the coma continues without diminution. In this stage, then, the temperature returns to the normal 99\frac{2}{5}, or it may rise to 101°, and it continues to oscillate rather irregularly between these two points for two, three, or four days. During this time the respiration is not sensibly altered in frequency, and the fluctu-

ations of the pulse are slight and irregular—the rising and falling in the number of pulse-beats being by no means always harmonious with the variations in temperature.

Now, as I have already stated, in the most severe cases of apoplexy the patient dies in less than two hours—whilst the temperature is still low. where the patient is destined to die in from ten to twenty-four hours the comparatively stationary period of body-heat just described is also absent -the primary depression of temperature being followed by a steady and continuous rise till it reaches at the time of death some point ranging between 104° and 108°. On the other hand, in instances where the stationary period of body-heat is present, if the patient is destined to recover from the immediate effects of the already existing brain lesion, and if no visceral disease of an inflammatory nature (in the lungs, for instance) should complicate the case, the temperature after two or three days of slight elevation sinks to the normal grade, and there remains. Where, however, the brain lesion is such that the patient is likely to die in the course of a day or two more, the stationary period is followed by another change in the temperature of the body.

(c) Ascending period.—A rise of temperature, of a

rapid and continuous character, is just as unfavourable a sign when it sets in after a 'stationary period' as when it occurs immediately after the period of 'initial lowering.' As a rule, death is then not far distant, and the temperature may rise, as before stated, to any point between 104° and 108°-the maximum being attained at the time of death, or very shortly afterwards. During this stage, too, the pulse and the respiration are much more constant in their characters than they are during the earlier stages of the malady. The pulse becomes small and very frequent — 120 to 136 per minute. respirations increase in frequency (48 to 64), and are also laboured, noisy, and often complicated with laryngo-tracheal râles. At the same time the extremities become dusky, especially on the paralysed side; whilst the face is red, swollen, and covered with an abundant clammy sweat. Finally, there is the most absolute muscular resolution in all parts of the body-even where more or less rigidity had previously existed—which continues till death closes the scene.

You will remember that the temperatures which I have cited have been taken in the rectum, and it is also necessary for me to warn you here that the temperature on the paralysed and on the non-paralysed sides of the body may be different even during the continuance of the apoplectic condition, so that there is a real advantage in obtaining the rectal temperature if we wish to study the changes in body-heat which occur during the apoplectic state. To the question of the difference in temperature between the two sides of the body I shall have to return further on.

## CONJUGATED DEVIATION OF THE EYES, WITH ROTATION OF THE HEAD.

I now wish to speak to you concerning a frequent and very definite sign of the hemiplegic condition which may be present from the first both in the apoplectic and in the epileptiform mode of onset. I allude to a condition to which attention has been more particularly given of late years, though it was previously incidentally referred to by several French writers. It was first definitely described in France by Vulpian, and in this country by Lockhart Clarke, and is now commonly known by the name of "conjugated deviation" of the eyes. Both eyes are turned as they would be if the individual were looking upwards over one or other shoulder, and this appearance is still further simulated in the majority of cases by a rotation of the head and neck in the same direction.

The condition is supposed by M. Vulpian and other physiologists to be related to the phenomena of 'rotation' frequently observed in the lower animals after many unilateral lesions of the brain. On examination of a patient in whom such signs exist, it will be found that there is frequently a certain rigidity of the muscles of the neck, and that attempts to bring the head back into the median position may produce some pain. When it has been brought back in this way, as a rule it lapses into its previous attitude as soon as the pressure is withdrawn. The eyes are usually motionless, though they occasionally exhibit the minute oscillations known as 'nystagmus.' In either case the pupils are lodged close to the palpebral commissures away from the paralysed side, one being against the outer whilst the other is against the inner canthus. The pupils themselves are altogether variable as regards their state of contraction. If the patient (when conscious) be told to move the eyes in the opposite direction, it is found that he can scarcely bring them to the middle line, and as soon as the effort ceases they return to their original position.

In some cases this conjugated deviation of the eyes is much less marked than I have here described it: there may be only a tendency towards the condition, and this may exist alone or without

any obvious rotation of the head. And however well marked the deviation of the eyes and head may be, the spasmodic condition of the muscles to which the phenomena are due, almost always relaxes, as Dr. Reynolds pointed out, when the patient falls asleep. Both eyes and head then come back to the normal position, though as the patient awakes the spasm again declares itself.

M. Prevost has devoted much attention to this particular sign, and has written an interesting memoir on the subject. He showed that it occurs in the human subject from lesions in various parts of the cerebrum. It derives its diagnostic value from the fact that the lateral deviation of the eyes and head, when present, is always towards the side on which the brain lesion exists - that is, away from the paralysed side of the body. To this rule only two or three exceptions have been met with, and in each of these exceptional cases the lesion has been situated in the pons Varolii. So that, according to Prevost, the sign is somewhat uncertain in its indications when we have to do with lesions in the pons Varolii, though for all parts of the brain above this region it follows the rule already mentioned with undeviating regularity. Whilst it is encountered occasionally where we have to do with lesions in or upon the surface of either hemisphere, this sign occurs with greater proportional frequency as the lesion approaches the cerebral peduncles. It is especially apt to present itself in attacks whose onset has been abrupt. It often lasts but a very short time, though it may persist for many days, or, more rarely still, for several weeks.

Having concluded my account of the apoplectic and epileptiform modes of onset of hemiplegia, I must now turn to the remaining mode in which this affection is apt to commence.

3. Simple Onset.—In cases of this kind a person may be sitting at meals, walking, reading, or otherwise engaged, when - perhaps after a momentary giddiness, though without other sensation-he suddenly loses power over one side of the body. If standing, he drops down, owing to a giving way of the leg, and at the same time finds himself unable to move the corresponding upper extremity. When aware of the powerless state of his limbs, the patient may experience no pain or sensation other than a feeling of 'numbness' in this half of the body or in the limbs themselves. quite conscious, and may be able to speak more or less intelligibly, though at other times he is only able to communicate with his friends by means of signs. Now such a mode of onset is very

frequent indeed, especially in the slighter cases of hemiplegia.

Again, not unfrequently we are told by the patient that on awaking one morning he felt a sense of 'numbness' on one side of his body, and was thus led to the discovery that the limbs of this side were paralysed. The injury to the brain takes place without producing any sensation sufficient to awake the patient, and this may occur even in cases where pain has been a marked symptom amongst the prodromata. Thus, a woman aged forty-seven, who was under my care at the Hospital for the Paralysed and Epileptic, was seized rather suddenly with vertigo and pains in the back of the head. After half an hour she became sick and the sickness frequently recurred, whilst she still suffered from excruciating pains in the occiput, for two days. On awaking the next morning she found herself quite paralysed on the left side of the body and also unable to speak.

Sometimes the onset of the hemiplegic symptoms is not absolutely sudden—the paralysis advances and spreads more slowly. In these instances the attack usually begins with a weakness in the hand or arm, though more rarely the loss of power is felt first in the leg. At other times the hemiplegia is ushered in by loss of speech and drawing of the face to one

side. As examples of these latter modes of origin, I may cite the following brief details. One of my patients, a man sixty-four years of age, whilst walking, felt a tingling in the end of his fingers, followed by weakness in the left arm, which increased, and afterwards extended to his leg and face: at the expiration of half an hour he was quite hemiplegic. Another patient, sixty-eight years of age, on arising one morning did not feel well and went to bed again, when he was seized with a feeling of numbness in the left side, which was followed in about half an hour by complete paralysis of the corresponding arm and leg. Another patient, a woman aged fifty-two, whilst taking breakfast felt giddy and found herself weak in the right leg; she gradually lost power in both leg and arm during the next twenty-four hours. The paralysis of these parts had then become complete, and though there was no loss of speech, a thickness of articulation continued for some time. Again, a man, sixty-one years of age, awoke one night and found himself unable to speak. no pain nor any unusual sensations, and in half an hour he regained his power of speaking. He went to his work the next day, but on awaking the following morning he found he had quite lost the use of his right arm and leg.

It happens, moreover, in certain rare cases that

a sensation of pain, either in the head or in one of the limbs about to be paralysed, has occurred as the initial symptom. Thus one of my patients, a man forty years of age, the subject of left hemiplegia, gave the following account of its mode of onset:-He had suffered occasionally from sharp darting pains in the right side of his head, and also in his left arm, for about a month, when suddenly (whilst walking along a road) he felt an acute pain through the same part of his head, and thought he "heard a noise." Owing to the sudden and complete loss of power in his left arm and leg, he would, at the same time, have fallen, had he not caught hold of a support. There was no loss of consciousness, but he spoke thickly for several hours. Another patient, a man sixty-seven years of age, after supper suddenly felt a stab-like pain in his right hand; he cried out, and fell down insensible, paralysed on his right side. I may state that in both these last-mentioned cases the sum-total of symptoms seemed strongly to indicate that the pons Varolii was the part of the brain damaged. In another patient who was under my care a hemiplegic attack was ushered in by a still more special sensation, also tending in a measure to reveal the seat of lesion. He was a man sixty-two years of age, who suddenly lost the use of his right side,

without loss of consciousness, and without any sensation except a "stupefying smell" which lasted for two or three minutes.

Now, although I have described the three modes of onset of hemiplegia-the apoplectiform, the epileptiform, and the simple—as though they were quite distinct from one another, you may easily imagine that these varieties become occasionally more or less merged so as to be represented during the same attack. Loss of power unattended by loss of consciousness may exist for a very short time, and then the patient may either suddenly or gradually lapse into a condition of complete coma, owing to a rapid or gradual increase of the original lesion. Again, an epileptiform attack may supervene shortly afte. the onset of a hemiplegia which commenced either with or without loss of consciousness. Many varieties, indeed, in the mode of onset and subsequent progress are to be met with, which will, I hope, be more easily understood after what I have already said.

We must now turn to a consideration of the hemiplegic state itself, and as a starting point I shall take one of the most ordinary forms of the disease. I shall describe the grouping of symptoms occasioned by a lesion in and just outside the right corpus striatum, in order that this may serve as a type of the hemiplegic condition generally.

HEMIPLEGIA FROM A LESION IN OR JUST OUT-SIDE THE RIGHT CORPUS STRIATUM.

If a loss of consciousness has been occasioned by the shock to brain-function produced by the first occurrence of the lesion, we must suppose our patient to have recovered from this conditionwhether it has lasted a few minutes or a few hours. A temporary loss of consciousness, however, does not necessarily exist in these cases: in many instances it is absent altogether. By careful examination we may be able to note that our patient presents the following peculiarities:—(1) an absence of any decided mental disturbance; (2) slight 'thickness' of speech; (3) more or less deviation of the tip of the tongue to the paralysed side when it is protruded; (4) partial and incomplete paralysis of the facial muscles on the side on which the paralysis of the limbs exists; (5) more or less complete loss of voluntary power over the left arm and leg; (6) a flaccid state of the muscles of these limbs, which are found to respond naturally, or perhaps even a little too readily, both to the faradaic and voltaic currents; (7) some actual loss of sensibility as well as a feeling of 'numbness' on the paralysed half of the body; (8) slight elevation of temperature on the paralysed as compared with the non-paralysed side of the body—the difference being seldom more than one degree of the Fahrenheit scale.

Now the progress of a patient in respect of these symptoms, where the lesion does not increase and everything progresses favourably, is apt to be as follows:—

The mental condition continues undisturbed, or at most there is slight weakening of the general mental power of the individual, which may be accompanied by an increased readiness to the display of emotional manifestations—more especially to crying—on the occurrence of inadequate and often trivial incidents. This slight emotional weakness, with undue irritability and diminished power of attention, may, however, entirely wear off after a time.

The *speech*, after a few days, or at most a week or two, loses its previous 'thickness.' The slight difficulty at first experienced in the articulation or utterance of words gradually passes off.

The tongue also soon begins again to be protruded with its tip pointing straight forwards, owing to the recovery of power in the genio-hyoglossus muscle on the paralysed side. Mistakes are apt to be made in reference to this point by a superficial observer if he does not allow for any marked deviation or lack of symmetry which may still exist about the mouth.

In a day or two, also, though perhaps not for a week or two, the *sensibility* of the paralysed limbs again returns to its normal condition. The subjective feeling of 'numbness,' however, may persist for a longer period.

The temperature of the paralysed limbs soon sinks to the level of that of the opposite side, and after a time it is apt to become even a degree or two lower. The paralysed limbs then are, and feel to the patient, colder than those of the opposite side.\*

The paralysis of the face begins to disappear sometimes after a few days, though more frequently after a few weeks. It may persist to some extent, however, for many months. Now, it is important that you should understand the kind of facial paralysis which is met with in these cases. It is, as I told you,

<sup>\*</sup> Dr. Lombard has now placed at the disposal of medical men a small portable thermo-electric apparatus of great value, capable of yielding the most accurate results in the determination of any differences of temperature which may exist on the two sides of the body. (See British Medical Journal, Jan. 23rd, 1875, pp. 98 and 116.)

both partial and incomplete—that is, it only affects, to an appreciable extent, some of the muscles supplied by the portio dura of the seventh nerve; and those which are affected are weakened rather than wholly paralysed. Those chiefly affected are the buccinator and other straight muscles going to the angle of the mouth and lip on the paralysed side of the body; and in consequence of the weakening of these muscles the cheek on this side looks flat, the corresponding naso-buccal fold is less marked, the upper lip is less arched, and the angle of the mouth on the same side is decidedly lower than its fellow. As patients and their friends often say, the face is 'drawn,' though by this expression they refer more especially to the opposite or non-paralysed side, where, owing to the unresisted action of the muscles on this side, the angle of the mouth is This difference in level becomes marked when the patient laughs or speaks. may also judge of the extent of the paralysis when in answer to your request, the patient attempts to show his upper teeth.

You may note, however, that he can easily frown as usual, can lift his eyebrow and eyelid, and can close his eye on the paralysed side almost as well as on the other side of the face. It is only rarely that a trifling weakness of the orbicularis palpe-

brarum is apparent for the first day or two after the attack.

In judging of the existence or amount of facial deviation in any doubtful case you must not lose sight of the fact that an unsymmetrical condition of the mouth naturally exists in some persons. This may occasionally be due to a comparative absence of teeth on one side, or it may be a mere individual peculiarity which has long existed in the person under observation. These may seem trifling matters, gentlemen, but they are oftentimes of great importance. You will hereafter have to form your opinion as to the state of patients who, in addition to slight symptoms of impending brain disease, seem actually to present some amount of paralysis on one side of the face. For you must know that such paralysis may, on rare occasions, exist alone for a time, as the first instalment or harbinger of a complete hemiplegia. You will see the importance, therefore, of recognising the kind and amount of facial paralysis which goes with hemiplegia, in order that you may be able at once to distinguish it from the mere local paralysis of the facial nerve which is of so much less importance, because of its being due to comparatively external causes.

The last sign of the hemiplegic condition to disappear is the motor paralysis of the limbs. Recovery

in this respect commences after a very variable period-weeks or months; and whilst in some few cases it may go on to a complete restoration of power, in by far the larger number recovery is almost arrested after the patient's condition has more or less improved. The patient is soon able to get about, though in a more or less shuffling manner, keeping the knee straight and the toe pointing downwards, so that drags on or scarcely clears the ground, whilst the leg is thrown more or less outwards and forwards. At the same time the arm hangs helplessly by the side, so that during his attempts at progression the patient recovering from hemiplegia presents a thoroughly characteristic appearance. It is not always, however, that a definite circumduction of the leg is seen. And after a time the patient generally moves the leg straight forwards in walking, though the knee is not bent and the toes are mostly directed inwards.

Now it is important to note that there is a certain order observed in the succession in which power is restored to the paralysed parts. The parts which are paralysed least are the first to show signs of improvement, and the parts which are paralysed most are the last to recover. And in reference to the degree in which the several parts are implicated, I may say that in all such cases of hemiplegia in

which the paralysis is not absolute, it is found that the arm suffers more than the leg; whilst of the several segments of each limb, the muscles about the proximal parts are least paralysed, whilst those concerned with the movement of the distal joints are the most affected. So that, in accordance with what I have already told you, improvement, as a rule, begins to manifest itself by an increased ability to move the limb from the hip-joint, and is followed successively by a recovery of power over the muscles which move the knee-joint, the ankle joint, and the toes. At some variable stage of this recovery of power in the lower extremity, improvement also begins to manifest itself in the upper extremity. This shows itself first in some increase of power in the muscles about the shoulder, and subsequently (if improvement should go so far) in a resumption of power in the muscles which move the elbow, wrist, and fingers. Thus, the parts which are habitually employed in the most complex and purely voluntary movements are usually the last to be restored to their normal condition.

In ordinary cases, such as we are now considering, the muscles remain throughout in a flaccid state, and they undergo no special wasting—that is, no wasting beyond what may be accounted for by longcontinued disuse or functional inactivity, whereby

they are also rendered more soft and flabby than the corresponding muscles on the opposite side of the body. The electric irritability of the muscles in cases of hemiplegia was long ago studied by Marshall Hall and afterwards by Dr. Reynolds; and the latter showed that whilst during the first few days of the disease, or longer in some cases, the electric irritability, both for faradaic and voltaic currents, might be slightly increased, as a rule it was normal in the paralysed muscles of a hemiplegic patient. This fact many of you have now had frequent opportunities of verifying for yourselves, and it is one which often proves of much use to us in our endeavours to ascertain the cause of any given paralysis where its distribution is at all anomalous-since in many cases of spinal cord disease and in diseases of nerve trunks, the electric irritability of related muscles is often rapidly impaired or even lost.

## LECTURE IV.

DEGREE OF MOTOR PARALYSIS.—SENSORY IMPAIR-MENTS.—VARIATIONS IN HEMIPLEGIC STATE.— RIGIDITY AND TREMOR IN PARALYSED MUSCLES.

- Distribution of Paralysis in Hemiplegia Parts exempt therefrom —
  Dr. Broadbent's explanation—Most automatically acting Muscles
  suffer least—They act in concert with Related Muscles on
  opposite side of body—Their related, double Spinal Nuclei
  can receive Cerebral Stimulation on either side—Unilateral
  Stimulation of Muscles of Articulation.
- Sensory Impairments in Hemiplegia—Explanation of certain Exemptions—Connected with Bilateral Activity of Senses—The case of Touch or Common Sensibility—Analogous Regions in each Sensory Apparatus Impairments of Hearing Defects of Sight—Decussation of the Optic Nerves complete—Blindness from Lesions of Corpora Quadrigemina—Functional Defects in same Region—Impairment in sense of Smell—Sense of Taste —Tactile Sensibility Uncertainties concerning its Lower Centres—Degree of Impairment of Common Sensibility in different Brain Lesions.
- Imperfect Forms of 'Hemiplegia'—Implication of Face only—Implication of Face and Upper Extremity—Cases in which Leg is more paralysed than Arm—Trousseau's Opinion thereon—Such Cases divisible into two Categories—First Set explained—Instance of—Nature of Second Set—Illustrative Case—Hemianæsthesia—Unilateral Numbness—Irregular Bilateral Paralysis—Variations in Implication of Cranial Nerves—Paralysis of Sphincters.
- Early and Late Rigidity in Paralysed Limbs—Nature of Early Rigidity
  Often co-exists with Increased Reflex Excitability—Its Mode of
  Distribution—Its Import—Late Rigidity—Its Distribution—
  Its Mode of Production—Choreic Movements and Tremor of
  Paralysed Muscles.

## LECTURE IV.

GENTLEMEN,—The description which I gave you in the last lecture of an ordinary attack of Hemiplegia due to a lesion in and just outside one of the striate bodies, is at present incomplete. I have hitherto dealt almost wholly with those positive deviations from the normal condition which characterise the disease; but the negative signs are here also of great importance. You will not possess a rational acquaintance with the hemiplegic state unless you know what parts escape paralysis, why they are thus exempt, and why certain sensory functions are so rarely interfered with.

Exemptions from Complete Unilateral Paralysis.—Although the condition we have been studying is spoken of as one of hemiplegia, you will have
seen that the paralysis by no means involves the
whole of one lateral half of the body. It is limited
to the arm, the leg, and part of the face. The

muscles of the neck escape, and so also do the muscles of the back, of the thorax, and of the abdomen. Different theories have been put forward to explain these exemptions, because they have been regarded as somewhat contradictory to the generally received view, that each corpus striatum is the organ through which 'the Will' acts upon all those muscles of the opposite side of the body which are capable of being employed in voluntary acts. I shall refer only to the last of the explanations which have been advanced, because, in my opinion, it happens to be also the best. This is the hypothesis of Dr. Broadbent, which, whilst it goes far to explain the facts, is also based upon sound physiological principles.

Referring to those exemptions above noted, which cause the unilateral palsy to be only partial where it might have been expected to be complete, Dr. Broadbent maintains that the key to this apparent anomaly is to be found by a comparison of the muscles paralysed with those which escape paralysis. They are then seen to fall naturally into two distinct classes. The arm is completely paralysed, and this is a part whose movements are always independent of those of its fellow. The leg, which is often less paralysed, can also be moved quite independently of its fellow—though the two limbs so very frequently move in concert. But the muscles of the

eyes, neck, and trunk—those which escape paralysis—act in pairs and are almost always bilaterally combined. Corresponding or related muscles on the two sides of the body are, in fact, here simultaneously called into play. Thus, as Dr. Broadbent says: "We move one arm or one leg whilst the other is quiet or executing a totally different action. We find it impossible to expand one side of the chest without the other, or to move one eye without the other, and extremely difficult to throw into action the muscles on one side of the abdomen without the other—impossible, indeed, to do this forcibly."

Now, it is very probably an anatomical fact that when muscles habitually act together, and rarely or never independently, the spinal nuclei of their nerves are connected by commissural fibres. Dr. Broadbent's explanation, therefore, of the exemption of certain muscles from paralysis in cases of hemiplegia is based upon the supposition that the nerve-nuclei, on opposite sides of the cord, of such pairs of bilaterally acting muscles are so intimately connected by means of commissural fibres as to be in effect a single nucleus. He supposes that "this combined nucleus will have a set of fibres from each corpus striatum, and will usually be called into action by both; but it will be capable of being

excited by either singly, more or less completely, according as the commissural connexion between the two halves is more or less perfect."

Keeping this very luminous principle in view, you will easily be able to understand the reason of the exemptions from paralysis above noted, and will also more readily comprehend the several degrees of implication of those parts which are paralysed in hemiplegia. If the bilaterally acting muscles escape, it is because they are still capable of being called into action by the undamaged hemisphere acting upon their common nucleus; and if the parts most used in voluntary actions suffer most, it is because the muscles of these parts have no communication whatever with the uninjured cerebral hemisphere. This is partly owing to such muscles being habitually called into activity by regions of the brain accustomed to act independently, and partly because - as a consequence of this independent action—the spinal nuclei of the nerves going to these muscles are unconnected with their duplicates in the opposite side of the cord.

Indeed I think it may be laid down as a general rule, that actions which are automatic in various degrees, or which can take place without a conscious guiding superintendence, involve, for the most part, related muscles on the two sides of the body; but actions which are more completely voluntary are generally concerned with muscles on one side of the body only, and often, if not always, take place under the immediate superintendence of parts of the brain which are only very imperfectly connected with corresponding parts of the opposite hemisphere.

Before leaving this subject, however, I will call your attention to one apparent exception to the general law concerning the possibility of the stimulation of bilaterally acting groups of muscles through either hemisphere of the brain. You will easily perceive that no muscles more eminently belong to the bilateral category than those concerned in the ordinary motor acts of speech or articulation; and yet, as we shall afterwards find, these groups of muscles seem only capable of being excited volitionally by stimuli which pass through the corpus striatum of one side (p. 206.) The explanation of this apparent anomaly is, however, doubtless to be found in the deeper fact-also exceptionalthat these particular bilaterally-acting muscles are habitually called into play by voluntary rather than by automatic stimuli.

But the application of this hypothesis of Dr. Broadbent does not end with its explanation of the distribution of motor paralysis in an attack of

hemiplegia. Although he has not fully developed it in the direction which I am now about to indicate, I have long thought that a somewhat similar explanation might be given of certain apparently anomalous exceptions to the law of unilateral paralysis, as regards the sensorial functions, when we have to do with large lesions of one-half of the brain.

Degrees and Modes of Sensory Impairment in Hemiplegia.—In taking up this part of the subject, however, I must point out to you that it will be necessary now to cease to speak specially of injuries in the corpus striatum. I shall have to refer to the effects of lesions of varying extent in different parts of one or other hemisphere of the brain.

The brain is by some physiologists regarded as a completely double organ, so that all its higher functions—those of Perception, Thought, and Volition—it is presumed *may* be carried on by one hemisphere alone, though usually the two hemispheres are associated in these acts. There is good reason, indeed, for believing that one cerebral hemisphere may take cognisance of the visual impressions made upon both eyes, since there are many cases upon record in which, although one hemisphere has been nearly totally destroyed, the sight of each eye has

remained almost entirely unaffected. A statement of the same kind may be made with reference to the sense of hearing, and with certain exceptions (to be referred to further on), also for the less special senses of smell, taste, and touch.

What, then, is the meaning of these facts? We are compelled to fall back, as it appears to me, upon the same kind of reasoning as that employed by Dr. Broadbent to account for the exemptions from paralysis of the neck and trunk muscles on one side in cases of hemiplegia. Thus, in the case of motor phenomena, we find a unilateral outgoing stimulus impinging upon one side of double and united ganglia in the medulla or cord, and thence spreading to related muscles on both sides of the body. Conversely, in the case of sensory phenomena, we seem to have double ingoing impressions impinging upon sense organs on each side of the body, converging to double and united sensory ganglia, and being then taken cognisance of by a single cerebral hemisphere, when it happens that the other is seriously damaged. And the rule which obtains with regard to the perfection of this sort of action in the case of the more automatic motor acts would be likely also to obtain in the case of sensory phenomena; that is, the perceptive action of the one hemisphere for sensorial impres-

sions from the two sides of the body, would be likely to take place perfectly or imperfectly according to the frequency with which the two halves of the sense-centres at the base of the brain are habitually stimulated in concert: because upon the frequency of this synchronism in action would depend the completeness of commissural union existing between them. As matter of fact, however, we know, from the admirable researches of Lockhart Clarke, how intimately the nuclei of the two auditory nerves are knit together in the medulla; and also we know that a similarly close relation exists between the nuclei of the two optic nerves in the corpora quadrigemina. We know, moreover, that the two eyes and the two ears are habitually called into simultaneous activity.

But it is precisely these modes of sensibility—hearing and seeing—which are most certainly known to be exempt from unilateral impairment, in cases where even a very large amount of damage exists in one cerebral hemisphere. It is undoubtedly the rule that lesions of one hemisphere do not affect the sense of hearing or the sense of sight on either side of the body. There are exceptional cases, it is true, with reference to the sense of sight, though, as I shall shortly point out, these are occasional and indirect, rather

than constant direct results of damage to one cerebral hemisphere.

As to the degree or frequency of impairment of the sense of smell or of taste in lesions of one hemisphere of the brain, our knowledge is very limited. So far as I am aware, however, there are no facts to show that these modes of sensibility are implicated in lesions of the hemisphere alone—that is, where the olfactory bulb or the fifth and glossopharyngeal nerves of the same side are not interfered with. According to the rule I have been endeavouring to explain and enforce, this is as it should be, since we may certainly say that the nuclei of these nerves are almost invariably called into simultaneous activity.

Physiologically, however, the case is quite different with regard to the sense of touch or common sensibility. In this mode of sensiency impressions derived from one side of the body engage our attention more frequently than simultaneous impressions on the two sides of the body; and even where the latter event occurs, the impressions on the two sides are discriminated from one another. In its ordinary activity, therefore, this is a mode of sensibility differing altogether from the others. From the nature of the difference, moreover, we should not expect, in accordance with the principles

already laid down, that the commissural connexions would be so intimate between the two halves of the lower nuclear centre for these impressions: and consequently we should not expect that there would be anything like the same amount of non-interference with this mode of sensibility in lesions of one hemisphere, as we find in the case of the higher or more specialised senses. And, as matter of fact, it is found that common sensibility is variously interfered with in cases of hemiplegia due to lesions in one half of the brain. Physiological indications are, therefore, so far harmonious with our everyday clinical experience.

Having laid down these general principles in reference to sensory impairment in cases of hemiplegia, let us deal with the matter a little more specifically, and study the modes in which each sensory function may be interfered with—whether directly or indirectly—by different intracranial lesions.

It may be well, however, to premise in the first place, that there is a similar anatomical plan in the central connexions of each sensory apparatus, and that similar rules will be found applicable for each of them in reference to the effects of injury of corresponding parts. We have in each sensory

apparatus nerves, made up of bundles of fibres proceeding from certain peripheral organs, which come into connexion with variously united ganglion-cells in their respective nuclei at the base of the brain. From these lower nuclei (or 'sense-centres,' as I have elsewhere proposed to term them), fibres ascend, in a mode which we know very little about, into each cerebral hemisphere on their way to comparatively unknown regions of the cortex, which act as 'perceptive centres' for the several kinds of sensory impressions. Now, to put the case more definitely, what I have already pointed out is, that injury of the perceptive centres of one side, or of the fibres connecting them with the lower or basal ganglia, do not produce any hitherto appreciated effects on the senses of hearing, sight, smell, or taste, though it does produce some effect upon common or tactile sensibility. The result is, however, in each instance altogether different if we have to do with damage to the basal nuclei ('sense centres'), or to the nerve trunks connecting them with the peripheral parts or sense organs proper.

The sense of *Hearing* may, therefore, be more or less lost where the nuclei of the two auditory nerves are damaged by a lesion near the junction of the pons with the medulla, or where one auditory

nerve trunk is injured near its origin. Again, deafness has been known to have been produced, on one or both sides, in cases of incomplete thrombosis of the basilar artery (where death has not rapidly occurred), owing to the fact that the minute vessels which supply the auditory nerves and the internal ear are given off from this trunk, and are therefore liable to be occluded at their point of origin.

The sense of Sight is much more frequently affected in intracranial disease than the sense of hearing, this being due in the main to the much greater length of the optic-tract and nerve within the skull, and to their varied blood-supply—circumstances which expose these parts to much greater risks of functional disturbance from pressure or impaired nutrition than those to which the auditory nerves are liable. Thus, it not unfrequently happens that the retina or some part of the optic trunk in its course to the corpora quadrigemina of the opposite side becomes implicated, compressed, or otherwise affected by some pathological lesion, which at the same time acts as the determining cause of a hemiplegic condition.

This is the case, for instance, when with a thrombosis existing in the carotid and middle cerebral arteries on one side, a branch or prolonga-

tion of the thrombus also extends into the ophthalmic artery. Here the sight of the eye becomes affected on the side opposite to that of the paralysis of the limbs, owing to the cutting off or interference with the vascular supply of the terminal portion of the optic nerve and of its retinal expansion, on the side of brain lesion. The occurrence of such a complication more or less simultaneously with the onset of hemiplegia has, therefore, in some cases an important diagnostic significance: just as the simultaneous onset of loss or decided impairment of sight on the same side as paralysis of the limbs, points to a lesion with implication either of the optic nerve after its decussation or of the corresponding pair of quadrigeminal bodies. The latter is a combination which presents itself not unfrequently where thrombosis of one of the posterior cerebral arteries exists as a cause of hemiplegia. I have seen examples of each of these combinations, and several others are on record, especially of the first variety.

Pressure upon the optic tract may occur from lesions of various kinds, and, amongst others, from hæmorrhages into the lower part of the sphenoidal lobe, or into the substance of the crus cerebri. In such cases, however, the rule is that any defect of sight or blindness thus occasioned, exists only for the eye on the opposite side of the body.

Thus the data furnished by disease point, as Brown-Séquard maintains, to the conclusion that the visual impressions from the whole of one eye, and from that only, pass by way of the corresponding optic tract to the opposite pair of quadrigeminal bodies-or, in other words, the evidence from pathology points to the conclusion that the decussation of the optic fibres at the chiasma, instead of being partial, as Wollaston supposed, is complete. Such an arrangement, as Flourens and Vulpian have shown by means of carefully performed experiments, undoubtedly obtains amongst the lower vertebrate animals - that is to say, amongst fishes, amphibia, and birds. Many preserved pathological specimens seem also to lend additional testimony to the fact that a similar arrangement occurs in man. Thus, I carefully examined the brain of that distinguished mathematician the late Professor De Morgan, who happened to have been blind almost from birth in the right eye. The corresponding optic nerve was atrophied, and so was the opposite optic tract, whilst the optic tract of the same side presented a perfectly healthy appearance. We have another very conclusive specimen of the same kind in our museum (No. 2090).

The principal reason in support of the supposed partial decussation, as held by Wollaston, is to be

found in the explanation which such an arrangement is presumed to offer of the mode of production of hemiopic defects of sight. This, however, is an obscure and difficult subject; and, in our attempts to throw light upon it, we must not lose sight of the more definite facts to which I have just called attention-namely, those supplied by pathological anatomy and by experiments upon lower animals. On the other hand, it must be remembered that it is in the anterior of the two quadrigeminal tubercles of one side and in the external geniculate body, that the corresponding optic tract ends. We know little or nothing positive concerning the functions of the posterior quadrigeminal tubercles with which the internal geniculate bodies are in relation.

Where lesions seriously implicate the two anterior quadrigeminal tubercles the blindness is double and complete. We had an interesting illustration of this in the hospital about two years ago, in a man who was under the care of Dr. Reynolds. A long illness in which polyuria and extreme prostration were the most prominent symptoms was brought to a close by blindness and some degree of stupor for about fourteen days; and after the death of this patient I found a patch of softening almost limited to the anterior pair of

quadrigeminal bodies. Other cases of the kind are on record.

It is not, however, always necessary that a discoverable lesion or definite structural change should exist where blindness suddenly supervenes with the onset of some cerebral affection. Just as we may have a temporary paralysis of the limbs, without obvious cerebral change, following certain epileptic attacks, so a temporary blindness not unfrequently occurs after injuries to the brain, with or without epileptic phenomena. On these occasions the optic centres appear to be acted upon in an 'indirect' manner, by lesions occurring in other and perhaps distant parts of the brain. Blindness on both sides may thus be produced for several days, or even for one, two, or more weeks. And such an event is, I think, more apt to occur where the hemiplegia has been initiated by an epileptiform attack, or by a series of them, than when such initial phenomena have been absent. For even in instances where no hemiplegia occurs, I have several times found an otherwise ordinary epileptic attack produce blindness for several days or even for a fortnight.

In a curious case which came under my care about five years ago at the National Hospital for the Paralysed and Epileptic, the blindness lasted for an unusual time—as long as six weeks—and

was associated with a total inability to speak, though the patient's "intellect was perfectly clear." This condition supervened in a middle-aged man who had been a commercial traveller. Whilst in a state of previous good health he had five attacks of 'numbness' in the left arm during a few days. Three occurred on the same day, and after the last of them he found himself in the state above described, and also powerless in the left arm. The recovery of sight after its commencement was brought about rapidly, and continued permanent; though for weeks it had been so complete that he had been unable "even to see daylight."

The co-existing inability to speak in this case did not appear to have been due to spasmodic closure of the jaws. This is generally a phenomenon of much shorter duration, though I have known an inability to speak thus occasioned, associated with temporary blindness. This combination occurred in a still more remarkable case, and in conjunction with fits of tonic spasm instead of a persistent paralytic condition. The patient was forty-five years of age, and whilst recovering from a severe attack of rheumatic fever, four years before he came under my care, he had the first of a series of fits to which he had been very frequently subject ever since. He had them almost daily during the first twelve

months, then at much longer intervals, though of late they had been recurring even more frequently than at first. Throughout they had been substantially the same in character. After a slight feeling of vertigo, all his limbs became contracted with tonic spasms, slightly intermitting at times. His mouth continued firmly clenched, so that he was quite unable to speak, and he became totally blind, though hearing all that was said and otherwise preserving his consciousness. These attacks lasted from twenty to thirty minutes, or even more.

These are the various modes in which unilateral or complete blindness is apt to be associated with hemiplegia due to hæmorrhage or softening, or to mere functional changes. Hæmorrhage and softening have of themselves little tendency to set up optic neuritis, which, when it occurs, may occasionally terminate in optic atrophy and blindness. These pathological conditions thus differ very notably in their tendencies from tumours within the cranium, as Dr. Hughlings Jackson and Dr. Allbutt have fully shown. It must, moreover, be clearly borne in mind that an optic atrophy so determined supervenes as a more or less remote sequential condition, and not as a primary accompaniment of the hemiplegic state.

An abolition of the sense of *Smell* on the side opposite to that of paralysis of the limbs would almost certainly occur from all lesions of the anterior lobe which either pressed upon or damaged the olfactory bulb of the same side. This is a sign to which more attention ought to be paid than has been hitherto given to it, on account of its localizing value.

Subjective 'smells' are complained of occasionally by hemiplegic as well as by epileptic patients, though it is a sign of less value than that afforded by loss of the sense of smell on one side. Their occurrence would not necessarily point to a lesion about the olfactory bulb on either side, since they might be occasioned by an unstable condition of any part of the olfactory tract between one of the bulbs and its corresponding olfactory perceptive centre somewhere in the convolutional grey matter.

The question of the presence or absence of the sense of smell should be tested by the use of assafætida, musk, or other non-irritating substances; and the examination, if it is to yield trustworthy results, must be conducted with extreme care. The fact that some persons are congenitally deficient in respect of this sense should not be forgotten.

The sense of Taste is impaired on one or both

sides in cases in which the glosso-pharyngeal nerve or its root, or the fifth nerve and its root, is damaged by paralysing lesions in the floor of the fourth ventricle, or in the pons Varolii. For, as you know, the palate and posterior parts of the tongue owe this endowment to branches of the glosso-pharyngeal, just as the anterior and antero-lateral parts of the tongue derive their gustatory powers from some of the filaments of the fifth nerve. But paralysis of other portions of these nerves, showing itself by more obvious signs, is almost sure to be associated with paralysis of the gustatory division in either of them; so that the state of this particular sensory function comes to be of comparatively minor importance so far as diagnostic indications are concerned.

Turning now to the question of the interference with Common and Tactile Sensibility on the paralysed side of the body, many points have to be considered. I have already referred to this subject generally, and shown how this mode of sensibility differs from the others in which a simultaneous bilateral excitation of the centres is almost invariably involved in their ordinary activity. The precise situation of the other more special 'sense centres' is also well known, though the same cannot be said with

reference to the centres towards which impressions of common sensibility converge from different parts of the body. The still widely received notion that such a function is to be allotted to the optic thalami I have long ago rejected. The facts supplied by disease seem entirely to discredit this hypothesis, and it has been similarly discredited by the anatomical researches of Dr. Broadbent. I should not omit to state, however, that Meynert still adheres in part to the old view, which he does not consider to be altogether discountenanced by his own very elaborate anatomical investigations.

On the other hand, both physiological and pathological data appear to me to show that the lower ganglia or 'sense centres' for common sensibility are situated on either side of the upper or posterior part of the pons Varolii. This view has long been adopted by many eminent physiologists, and amongst others by Longet and Vulpian, whose opinions on such a subject must have great weight. Certainly we find that lesions involving the upper or posterior strata of the pons on one side (about midway between its lateral border and the middle line) produce a complete paralysis of sensibility on the opposite half of the body, involving the face and trunk as well as the limbs. This paralysis is not only complete but very lasting; in fact, it corre-

sponds altogether with the kind of paralysis observed in the more special senses when the basal nuclei in connexion with their nerves have been injured.

But when we have to do with cerebral lesions of about the same or of proportional extent situated higher up in the nerve centres—that is, in the crus, in the optic thalamus, or corpus striatum, in some portion of the white substance of the hemisphere, or in its cortical grey matter,—we meet with affections of sensibility which become less and less marked, and of progressively shorter duration.\*

Now, in reference to such facts, it seems to me that whilst defects in common sensibility resulting from lesions of portions of either cerebral hemisphere above the pons, may be partly due to an 'indirect' or inhibitory effect of the lesions, they may also in part be directly caused thereby. The relative importance of the direct and indirect modes of interference with common sensibility in cases where paralysing lesions occur above the pons Varolii, cannot at present be estimated, though there are good reasons for believing that the indirect influence

<sup>\*</sup> Some important exceptions to the general rule, furnished by cases of persistent hemi-anæsthesia, will be referred to further on (page 142). Such cases are somewhat puzzling and difficult to understand. At present we need more precise information as to the exact sites in which the occurrence of a lesion suffices to produce such persistent hemi-anæsthesia.

is very powerful, and that deviations from the general rule, stated above, as to the diminishing paralysis of common sensibility as the lesion approaches the cortex of the hemisphere, are due principally to unexplained irregularities of this indirect influence.

On the other hand, it seems probable that the more or less speedy return of sensibility in the cases we are now considering (often taking place as it does long before there is any corresponding improvement in motor power), is due in part to the fact that channels of communication are soon perfected by means of new commissural fibres between the two halves of this sense-centre in the pons, so that the perception of impressions from both sides of the body may then be effectually achieved by one sound hemisphere. The condition in respect to this sense would in such a case become, in a measure, approximated to that normally existing for the more special senses. No other kind of explanation than this seems possible, with regard to common sensibility, in those numerous cases in which we have the record of an extensive atrophy or loss of substance in one cerebral hemisphere, dating perhaps from early childhood-where the limbs of the opposite side are more or less paralysed and rigid, though no appreciable loss of sensibility is to be detected.

Before leaving this part of the subject, I should

state that irregularities often occur in the distribution of the paralysis or lack of common sensibility. In the slighter cases of hemiplegia it is apt to be little marked in the side of the face or over the trunk. Occasionally it is pretty well marked in the arm, but absent in the leg. But when we come to unilateral lesions in the pons Varolii affecting the 'sense-centre' for common sensibility on one side, or near the termination of the cerebral peduncle, the most complete hemi-anæsthesia may exist, extending to the mid-line of the face and trunk.

In these cases, moreover, the different modes of sensibility may be variously implicated, just as we find where we have to do with certain severe lesions in the spinal cord. In illustration I will cite a few details concerning a woman who was under my care for a long time as an out-patient at the National Hospital for the Paralysed and Epileptic in the year 1868. C. C-, was fifty years of age, and the mother of five children. Her general health had always been very good till the onset of her illness, nine months before she came under my observation. The initial attack was sudden, but without loss of consciousness. She first felt numbness in the tips of the fingers, and in the thumb of the left hand, and in a minute or two afterwards entirely lost the use of her left arm

and leg; sensibility was also said to have been lost over the whole of this side of the body—even in the left side of the face and the left side of the tongue—though the loss was most marked in the arm and in the leg. There was also partial deafness on the left side. The face was much paralysed for the first few weeks on the right side (and some remains of this existed when she was seen); she spoke very thickly also, and with much alteration of voice for several weeks.

The patient kept to her bed for three months, but after that began to mend both as regards motor power and sensibility. The improvement commenced about the same time (she said) in arm and leg, and certainly when first seen by me she seemed to have rather more power over the upper than over the lower extremity. She could move the hand and arm freely in different directions, and her grasp, measured by the dynamometer, was almost half as powerful as with the right hand. Tactile sensibility was at this time found to be still very deficient on the left side, though it was not tested with any very great care till about six months later, when she had improved considerably in her general strength and in her power of walking.

The following conditions were then noted. Over the left arm and outer part of thigh, two æsthesiometer points at four inches apart could not be discriminated, and were felt only vaguely as a single pressure, even when strongly applied. On the outer side of the left calf, the two points could not be discriminated at  $3\frac{1}{2}$ ", but were recognized at 4", and there also she was unable to feel at all, unless firm pressure was made. On the right calf, however, the two points were readily discriminated at  $2\frac{1}{2}$ ", and with light contact.

The tactile sensibility of the trunk on the left side was still very much impaired, though not to the same extent as in the limbs. On the left side of the face the impairment was still less marked.

Hot water in which I could not with any composure bear to immerse my own finger, when applied with a sponge to different parts of the patient's arm and fore-arm was not felt, or else only produced sensations of the vaguest kind, which she would scarcely have noticed if her attention had not been aroused. The application of a spoon dipped in ordinary cold water to the same limb, caused an intense "pricking" sensation, which passed downwards to the fingers, and caused involuntary contractions of nearly all the muscles of the limb. On application of the same spoon to the right arm it was at once appreciated as cold, though it simultaneously revived a "pricking sensa-

tion" in that part of the left arm which had been last tested, and also caused the reflex muscular contractions to be reproduced in this limb. On again applying the cold spoon to the paralysed arm, more marked and extensive reflex contractions were produced, which even involved some of the muscles of the lower extremities. Similar phenomena were observed on the application of heat and cold to the left lower extremity, and there was an inability to localize the vague sensations produced by heat—thus when touched on the back of the foot, she said it was "somewhere on the leg."

The sensibility to painful impressions (from the prick of a needle) were found to be about equal and normal on the two sides of the body.

On tickling the soles of the feet an increased reflex excitability was found to exist on both sides, though this was much less marked on the paralysed than on the non-paralysed side. Tickling of the sole of the right foot, in fact, produced irrepressible convulsive twitchings of the same leg.

Electro-sensibility (faradaic current) was diminished in both arm and leg on the left side, though not to any very marked extent. Electro-motility was also somewhat diminished in the muscles on the paralysed side.

This woman had been subject to slight fits at

times since the commencement of her illness. She complained much occasionally of pains in her left foot. She continued to improve steadily for about six months after the date of the examination recorded above. She then, however, had an unusually severe fit, lasting about twenty minutes with convulsions confined to the left side of the body. She suffered much from sickness for several hours after the fit, and was so weak for several days that she could scarcely move about the house even. The special weakness of the left side was also much increased after this fit. Shortly afterwards she was lost sight of, and has not been seen again.

Similar variations as regards the degree of implication of the different modes of sensibility have been observed in other instances. Thus in the case of a middle-aged man who had been paralysed on the left side for two weeks, I found tactile sensibility practically abolished on the left side of the face and in the corresponding arm and leg, though the power of tasting was preserved on both sides of the tongue. A hot body on the face was recognised as "pressure" only; on the arm it was felt as such, though the sensation was not distinctly localized; whilst on the left leg the same hot body was recognised correctly as regards situation, though it gave rise only to a feeling of "tingling." In another

case, that of a young woman thirty-three years of age, who had been paralysed on the right side for six months, a light touch was not felt at all on the paralysed limbs, the prick of a pin seemed "something hot"; and sensibility to differences of temperature was almost abolished. These defects were all much less marked, however, on the corresponding side of the face.

Where the implication, direct or indirect, of the sensory centre on one side is only partial, corresponding irregularities are produced. Thus in a man fifty years of age who had been suffering for many months from left hemiplegia, I found a very marked diminution in sensibility to touch, temperature, and pain, in the right arm and leg, but especially in the former—although no such diminution existed over the corresponding side of the chest and face.

Where a lesion in adjacent parts produces irritation rather than destruction of one of the sense-centres for common sensibility, an actually tender and painful state of one or both limbs may be associated with a condition of more or less marked hyperæsthesia. Occasionally unilateral hyperæsthesia may exist as the most prominent symptom. This was so, for instance, in the case of a delicate and badly fed girl, twelve years of age, who was under my care as an outpatient at this hospital in

1870. She had been suffering from left unilateral convulsions for twelve months, increasing in frequency. A touch anywhere on the left side of the body—face and head included—caused pain. Heat was also felt much more distinctly on this side. No actual difference of temperature was perceptible on the two sides, though she complained much of subjective burning sensations. She was also decidedly weaker in the left than in the right limbs. This little girl was afterwards admitted into the National Hospital for the Epileptic and Paralysed, under my care, where she soon almost completely recovered under the influence of tonics, bromide of potassium, quietude, and better food.

## IMPERFECT OR IRREGULAR FORMS OF HEMI-PLEGIA.

I now proceed, gentlemen, to consider other variations in the hemiplegic state, which are also dependent partly upon the extent and partly upon the locality of the lesion.

Imperfect or abortive forms of hemiplegia are very frequently met with. The symptoms may stop short at various stages. And these incomplete hemiplegic conditions may be more or less speedily recovered from, or they may be the mere heralds of a not far distant complete attack.

- I. Sometimes we meet with a very limited set of symptoms. A patient feels giddy, for instance, finds himself either unable to speak at all for a few minutes or else only able to articulate badly, and at the same time his face may be 'drawn' to one side, though he experiences no weakness in the arm or in the leg. I have seen two such cases lately. and when examined a few days afterwards these patients were found to have completely recovered their power of speaking distinctly. There was no appreciable weakness in the limbs of either side, though the angle of the mouth was distinctly lower on one side, and the tongue deviated as distinctly in the same direction when protruded. The eve however, could be closed quite readily on the side of the face in which the partial paralysis existed.
- 2. In other instances we may find that kind of paralysis of the face and tongue which usually goes with the hemiplegic condition, in association with a more or less complete paralysis of the upper extremity on the same side, whilst the leg wholly escapes. Not only can the patient move his lower extremity freely in all directions when lying down, but he is able to walk without any trace of lameness or dragging of the foot on the side affected. This

latter test is the most important; for, as Dr. Hughlings Jackson has very properly insisted, a patient may show no weakness or inability to execute the various movements of the limb when lying down, and yet may display an obvious amount of lameness when he attempts to walk. symptoms of this kind past experience teaches me that we have some right to expect a lesion of the surface of the opposite hemisphere involving an area of convolutional grey matter and some amount of subjacent white substance, either in the parietal or in the hinder part of the frontal region. kind of lesion I have seen two or three times on post-mortem examination where the patient has died, not from the effects of the brain lesion itself, but from some serious complications or intercurrent maladies; and in scrutinising the details of many cases of this sort, recorded by different observers, I have found such a lesion mentioned very frequently.

3. Between this comparatively rare condition in which the leg escapes altogether, and the ordinary forms of hemiplegia in which it is at first completely paralysed, all gradations are encountered in practice. But still more rarely we meet with an irregular form of the affection, in which from the beginning the leg is more decidedly implicated than the arm. We are

able to recognize this anomaly at once, should the paralysis of the upper extremity be from the first incomplete; though in cases of complete paralysis of the hemiplegic type the existence of the disease in this irregular form cannot, of course, reveal itself until the patient begins to recover power. Then it is that we find the arm beginning to improve more rapidly than the leg-so that after a time the patient completely regains strength in the upper extremity, though the leg, instead of improving, may only become more stiff and powerless. Trousseau passed a very gloomy verdict upon such cases. He thought that severe pains were apt to occur in the paralysed limbs, that dementia was very prone to ensue, and that the patient often died within twelve months. He did not profess to give any explanation of these events, or of their connexion with one another, and unfortunately he did not tell us upon how many cases his opinion had been formed. I cannot help thinking, indeed, that there is no necessary association between dementia or early fatality and this form of hemiplegia.

Out of about two hundred cases of hemiplegia of which I have notes, I find only seven in which there was a tendency, more or less marked, for the arm to recover before the leg. Five of these seven cases were, after a time, lost sight of, and my notes concerning them are rather meagre; but one of the others was under my care until complete recovery occurred, and the seventh, which was a very well-marked instance of this irregular form of the affection, was seen from time to time for two years, and has of late been seen again after an interval of four years. I am strongly disposed to believe, however, that the cases in which the order of recovery, as between the arm and the leg, is reversed, will be found to be divisible into two categories quite distinct from one another, and of which the two cases just alluded to and presently to be recorded may—so far as they go—be regarded as illustrations.

First (a), we shall find cases in which the leg may be from the commencement of the attack more paralysed than the arm, and, therefore, later to manifest signs of returning power. And such a state of things, I believe, will be found to be most frequently associated with certain unilateral lesions of the pons Varolii or with pressure upon this part produced by lesions of the cerebellum. I say this because I think it may be almost laid down as a general rule that the extent and completeness of paralysis of the lower as compared with that of the upper extremity, other things being equal, tends to diminish as the lesion approaches the cortex of the cerebral hemispheres, whilst it tends to increase as the lesion

draws near the medulla oblongata. The movements of the leg are more habitually automatic or sensorimotor than those of the arm, and therefore are most apt to be seriously interfered with as the lesion approaches the pons, medulla, and cord. The movements of the arm and hand, on the contrary, are more habitually voluntary, and, therefore, take place more thoroughly under the instigation and superintendence of other parts of the brain, such as the cortical grey matter and the corpus striatum. It may happen, therefore, that a particular lesion in or pressure upon the pons Varolii causes a more complete paralysis of the leg than of the arm, simply because such lesion interferes with a greater proportional amount of cell and fibre structure naturally existing in this situation in remote relation with leg muscles.

I do not, of course, wish you to understand that complete paralysis of the arm does not occur in lesions of the pons Varolii; or, indeed, that lesions in this situation generally cause more complete paralysis of the leg than of the arm, though I do think that occasionally we get such a result. I can refer to cases verified by post-mortem examinations in confirmation of this opinion, the possibility of which is still further supported by the topographical separation of the strands of motor fibres pertaining to the

arm and leg respectively in this region, as ascertained by Brown-Séquard.

I have lately had under my care what I believe to be a very interesting example of this class of cases. Kate S-, a delicate woman, thirty-one years of age, and the mother of seven children, was admitted into the National Hospital for the Paralysed and Epileptic, on May the 5th, 1874. Five months before, on hearing of her husband's failure in business, she had a sort of 'fainting fit,' and remained in a confused half-unconscious condition for three weeks. During this time she was unable to speak, suffered from spasmodic closure of the jaw, and at times from pains in the top of her head, and was paralysed on the right side of the body. The arm and the leg were perfectly powerless, whilst sensibility was also very much impaired on this side. face was not very much 'drawn' from the first. After three weeks she began to improve rapidly the spasm of the jaws relaxing and the power of speech returning, though she spoke very indistinctly at first. A month afterwards she began to regain power in the arm, and continued to improve very quickly in this respect, so that at the end of another month she was able to knit and sew.

During the whole of this time, there had been no improvement in the leg, the motor paralysis continued complete, and its sensibility did not improve. When first seen by me, after five months, she had regained some sensibility in the leg, though she was still unable to move it more than at first. Its muscles contracted less readily to faradization than those of the left leg, and there was a much more marked diminution of sensibility. The right arm seemed completely to have regained its power, and the right angle of the mouth was only very slightly lower than the left. She complained of no pains. She was ordered a mixture containing quinine and iron, a nourishing diet, and faradization of the muscles of the right leg three times a week.

After three weeks (May 26), improvement in the motor power of the leg had commenced, so that she could move the toes a little, and after faradization on the last two occasions had been able to lift the foot a little from the couch. Sensibility was, however, still very much impaired in her right foot, leg, and thigh—firm pressure, the prick of a pin, and a hot or cold spoon not being felt in these situations. On May 31 the improvement was more marked—she could just make the extensors and flexors of the knee contract, though with a scarcely appreciable power. Sensation was still dull—a touch being not at all, and a pinch scarcely felt. After this she steadily improved and was discharged on July 3

quite well—motor power and sensibility having been completely regained in seven months from the date of the onset of hemiplegia.

I see no reason for calling such a case as this one of hysterical hemiplegia, though some might be disposed to do so. Irrespective of certain peculiarities in her paralysis, the woman presented no signs of hysteria. In all probability some slight structural damage had occurred in or near the base of the brain—though its nature and exact situation were rather obscure.

Many of the cases, however, in which the leg appears to suffer more than the arm will, as I believe, be found to belong rather to (b) our second category. This would include cases in which the leg is not originally more paralysed than the arm—in which, in fact, recovery may even begin to take place in the usual way—though, as in the case about to be cited, it is soon arrested so far as the leg is concerned. Such cases are, I suspect, not so much due to any peculiarity of the brain lesion as to certain quasi accidental extensions of 'secondary' changes which the original lesion has set up in the spinal cord.

To this subject I shall have to return, though I will now relate a few details concerning what appears to be a characteristic example of the genus. In

brief, the particulars of this case are as follows:-A young man, twenty-one years of age, began to have 'fits' three weeks before the onset of hemiplegia. He had about eighteen of them within this period, although they did not recur after he became paralysed on the left side. He lost his sight for about one month after this attack, and seems to have continued in a state of incomplete stupor with mental incoherence for about six weeks. The face was not very notably paralysed, and the sensibility on the palsied side of the body was only very slightly impaired. About two months after the onset of the paralysis he began to regain strength in the limbs, which up to this time had been completely powerless. The improvement commenced first in the arm, whilst the leg, after recovering to a very limited extent, soon ceased to improve any further. It is particularly recorded in my notes concerning the previous condition of this patient (who was very intelligent, and was most carefully questioned), that he regained the use of his arm in the usual way-first being able to move it from the shoulder, then from the elbow, and afterwards beginning to move his hand and fingers. When I first saw him, eight months after the commencement of the paralysis, he could perform all ordinary movements with this left upper extremity, although the left leg could only be lifted so as to raise the heel about twelve inches from a couch, the left knee could only be slightly flexed, and the foot could not be moved at all. He had never suffered from any pains in either limb.

Twelve months afterwards he was in much the same condition. The arm was then quite well and strong, but the leg was in nearly the same state as that described above. He walked with difficulty, keeping the left leg stiff, so that there were no movements of the knee or ankle-joint, and the foot was turned strongly inwards whilst it was being slung forwards in the usual way. He still made no complaint of pains in the limb, and there was no evidence of any mental weakness. Quite recently I have seen this patient again, after an interval of nearly four years. Still he does not complain of pains in arm or leg, and his mental condition is as bright as ever, although little change has taken place in the state of the left leg. The arm has completely recovered; his grasp with the left hand (measured with a dynamometer) is as powerful as that with the right hand, and he has no difficulty in executing the most delicate movements. He still walks in the same way, and has some trouble in bringing the left heel to the ground. When lying on a couch this foot points downwards and inwards, and he can scarcely bend it at all, though he can now move the leg from the thigh well, and bend the knee with some strength. There is a slight diminution in the amount of reaction to faradization in the muscles of the left thigh and leg as compared with those of the opposite side; the left calf looks decidedly smaller, and it measures half an inch less than the right in circumference.

To a consideration of the precise significance of these facts I shall subsequently return (p. 177), and will merely point out for the present that they are strikingly opposed to Trousseau's anticipations.

In another of my cases in which the arm first regained power, I find it noted that the several parts of this limb recovered in an order the reverse of that which is usual—namely, that power was first regained over the movements of the fingers and wrists, then over those of the elbow, and lastly over those of the shoulder-joint. I call your attention to these details because I believe Dr. Hughlings Jackson holds the opinion that this inverse order of recovery of power in the upper extremity obtains in all or in almost all the instances in which the arm recovers before the leg. This is a matter which requires further investigation. My own experience hitherto does not enable me to share this opinion. Moreover, in cases where there

is no reversed order of recovery as regards the leg and the arm, I have occasionally found the several parts of the arm recover in the inverse order already mentioned, without being able at all to explain the reason of this departure from the general rule.

4. In another irregular form of hemiplegia, which is rather rarely met with, we find the loss of sensibility altogether in excess of the loss of motor power, or, in some of these cases, persisting long after the motor paralysis has disappeared. These are unusual conditions which require some additional light to be thrown upon them. To designate the unilateral loss of sensibility itself, the term 'hemianæsthesia' is usually employed.

Cases of this kind were some years ago recorded by Türck; and experiments have, of late, been made in Vulpian's laboratory and at his instigation, by Veyssière, which seem amply to confirm the fact that lesions in the situation indicated by the post-mortem examinations of Türck, are capable of inducing marked hemi-anæsthesia. Veyssière's experimental observations were made upon dogs and rabbits. The observations of both investigators go to show that complete hemi-anæsthesia may be produced by a considerable lesion just outside the optic thalamus, involving the peduncular expansion (internal capsule) just at the part where it begins

to unfold into the foot of the radiating crown of Reil. In Türck's cases injuries in and about this region gave rise to a hemi-anæsthesia which persisted for many months or even one or two years after the motor paralysis had almost or wholly disappeared.

Whilst we have yet to learn whether injuries of the sensory division of the peduncle itself, as well as of the sensory region of the pons, will not give rise to a similarly marked impairment of sensibility, Veyssière found that no such effect followed the complete removal of one cerebral hemisphere above the level of the thalamus, and similarly that no alteration in sensibility was produced when the greater part of the optic thalamus had been destroyed. So far, therefore, the experimental facts are in accordance with clinical and post-mortem observations.

The cases above referred to lead me to mention others, met with perhaps rather more frequently, in which a unilateral 'numbness' only is complained of, extending to the middle line of the trunk and face. It is not, however, simply a subjective feeling of numbness. I have ascertained in some of these cases that there is a positive diminution in tactile and other modes of sensibility—an incomplete condition of hemi-anæsthesia in fact. This state of things may persist for some time, and ultimately

yield to the influence of remedies; or it may subsequently become associated with a more or less markedly motor paralysis of the limbs on the same side.

A more profound condition of hemi-anæsthæsia, which may involve not only the skin but also the deeper structures of the limbs (with or without motor paralysis) is also occasionally met with in hysterical patients. This most commonly shows itself on the left side of the body, and is almost invariably associated with marked tenderness in the left ovarian region and a liability to the occurrence of epileptoid attacks. M. Charcot has lately made an important contribution to our knowledge concerning this very interesting combination of morbid conditions.

5. Large lesions in the central parts of the pons Varolii frequently produce various irregular forms of paralysis, in which power may be lost either unequally or equally on both sides of the body (p. 184). These forms of paralysis are accompanied by much or little loss of sensibility; and they further differ amongst themselves by the variable though often characteristic nature of the paralysis of the cranial nerves with which they may be associated. Unequal paralysis of the two sides of the body is, however, also caused not unfrequently by simul-

taneously occurring lesions in different or similar parts of opposite cerebral hemispheres; whilst in other cases a second attack of hemiplegia may supervene after a considerable interval, where, owing to the occurrence of the new lesion in the previously sound hemisphere, a generalised paralysis is the result.

Variations in Degree of Implication of Cranial Nerves .- Different cases of hemiplegia vary much amongst themselves in respect to the amount of paralysis of the seventh, ninth, and other cranial nerves with which they may be associated. As a rule, in lesions of the corpus striatum, or of any part of the hemisphere above this level, we meet only with that partial and temporary paralysis of the seventh and ninth nerves which I have already described (p. 93). But as the cerebral lesion causing hemiplegia approaches closer to or actually invades the pons Varolii, so is the paralysis of the muscles supplied by these nerves apt to be both more complete and more lasting. Muscles under the influence of the eighth and other cranial nerves may also become simultaneously affected. The tongue often cannot be protruded at all for a time, and there may be difficulty of deglutition, with a more or less copious flow of saliva from the paralysed

side of the mouth. The paralysis of the face, too, is much more marked; the corresponding angle of the mouth is notably lower; the buccinator is quite powerless; and food tends to collect between the teeth and the paralysed cheek. There is also some obvious weakening of the orbicularis palpebrarum, so that the corresponding eye can only be partially closed. In conjunction with such phenomena as these—that is, with the set taken as a whole—there almost always exists a marked difficulty in articulation.

This very striking group of signs may present itself (a) with the paralysis of the face on the same side as the limb-palsy, though at other times it occurs (b) with an implication of the face on the opposite side of the body; and it is in these latter cases that the paralysis of the face attains its maximum. It is here almost as complete as if the nerve-trunk itself were injured or diseased, as in the ordinary cases where facial palsy exists alone.

This form of 'cross paralysis' or 'alternate hemiplegia' (b) was pointed out by Romberg, though it was first carefully investigated by Gubler, and is now generally recognised as being indicative of a lesion in the pons Varolii. But this is not enough; it is indicative of a lesion in the lower or posterior part of one lateral half of the pons, because in this

place a single lesion may implicate the outgoing fibres of the facial (that is, its root-fibres) after their decussations in the pons, and also the channels for the conveyance of motor stimuli to the limbs before their decussation in the medulla. A single lesion thus situated, therefore, suffices to paralyse the facial muscles on the same side and the limbs on the opposite side of the body.

Should the lesion occur in the upper or anterior part of one lateral half of the pons, the fibres of the facial and the motor channels for the limbs are alike implicated above their respective sites of decussation, and consequently (a) the facial paralysis, though still very well marked, occurs on the side on which the limbs are paralysed. Thus, whilst 'alternate hemiplegia' is indicative of a lesion in the pons Varolii, it by no means follows that the lesion is not situated in this region simply because we have to do with a paralysis of the face and of the limbs on the same side of the body. This is a fact which some writers and practitioners appear to lose sight of.

Again, should the nucleus of the facial in the posterior or ventricular aspect of the pons be itself implicated on one side, we encounter an alternate hemiplegia, with the additional sign of a paralysis of the external rectus muscle—also on the side of

the brain lesion. This association is due to the fact, made known by the researches of Lockhart Clarke, that the nerve which supplies this muscle—viz., the sixth—arises from a nucleus common to it and the portio dura of the seventh. This paralysis of the external rectus is, of course, easily detected, and it entails a diplopia in which the two images are on the same plane, and tend to approximate, as, instead of looking at an object in front of him, the patient looks at an object which is moved in the direction of the sound eye and beyond it.

In other lesions of the pons, the sensory and motor divisions of the fifth nerve may be more or less involved. In addition to lack of common sensibility in the side of the face and other parts to which the fifth nerve is distributed, there may also be some marked implication of the sense of smell and taste on the same side. In certain cases also we meet with various trophic lesions in parts supplied by this cranial nerve. Paralysis of its motor division is shown by weakness of the corresponding masseter and other muscles of mastication. The patient cannot chew well on this side, because of his inability to contract these muscles forcibly.

On the other hand, the fourth nerve is very rarely implicated by such lesions of the pons Varolii as hæmorrhage or softening, though in some exceptional cases it may be pressed upon and paralysed.\*

The third nerve is, however, implicated in another form of 'alternate' hemiplegia—where hæmorrhage or softening involves the lower part of the crus cerebri on one side. In the early part of the session many of you had an opportunity of watching a case of this kind. You will recollect that we had in the wards a woman who was hemiplegic on the left

\* Although not often met with in the common forms of hemiplegia, it may be as well to state the signs by which paralysis of the fourth nerve is recognised. When the patient looks straight before him at some fixed point the cornea of the affected eye is raised, and turned slightly inwards. You may judge of its being raised best by noticing that the lower edge of the pupil rises slightly above the level of the lower lid, whilst that of the sound eye remains slightly below this level. Again, there is a loss of the power of rotating the eye round its antero-posterior axis, which may be ascertained by placing your hands upon the patient's temples and inclining his head first to the right and then to the left. During this manœuvre, whilst the sound eye rotates in a direction opposite to that in which the head is lowered, the affected eye does not rotate at all—it remains fixed in the orbit and merely moves with the head.

Again when the patient uses both eyes, he suffers from double vision, the two images not being in the same plane but one above the other. This double vision, as in other cases of paralysis of ocular muscles, disappears only when the patient looks in the direction in which the affected eye is drawn (in this case, upwards and inwards). The two images approach one another when the eyes are directed upwards; they recede from one another when the eyes are directed downwards. When the right nerve is paralysed the pseudo-image goes to the right, but comes nearly to the level of the image proper on directing the eyes to the right, whilst it gets almost immediately beneath the image proper on looking towards the opposite direction.

side, the face being implicated as well as the limbs, whilst on the right side there were distinct signs of paralysis of the third nerve represented by ptosis, dilatation of the pupil, and an outward deviation of the eye-ball, with inability to move it inwards as well as upwards and downwards—the paralysis of the ocular muscles having been produced simultaneously with the paralysis of the limbs.

These, gentlemen, are the characteristic signs of a lesion in the right crus cerebri, and they were doubtless thus occasioned in this particular case, although we had no opportunity of verifying the diagnosis. Our patient is fortunately still alive, and considerably better.

You will thus see, from what I have already said, that attention to the particular combinations and degrees of paralysis of cranial nerves, both sensory and motor, met with in association with the various forms of hemiplegic paralysis, may be of the highest importance in assisting us to arrive at a regional diagnosis in cases of brain disease.

Implication of Sphincters.—A word or two should be said on this subject. During the apoplectic condition, as you know, we may expect to meet with the involuntary passage of fæces and urine. After recovering from this state, however,

such events are very rarely complained of by hemiplegic patients. With one exception, in which, for no very obvious cause, a marked incontinence of urine continued for six weeks from the commencement of an ordinary attack of hemiplegia, these symptoms only presented themselves in some cases of double hemiplegia, or where mental dullness and incoherence existed in combination with a unilateral paralysis. Frequent seminal emissions together with imperfect erections and failure in coition have been complained of in two or three cases, even eight or ten months after the onset of a hemiplegic attack.

## Early and Late Rigidity in Paralysed Limbs.

—I will now call your attention, gentlemen, to the various forms of rigidity which may be met with in the paralysed limbs. This condition is seen not only more frequently but also to a more marked extent in the arm than in the leg. It was a sign much dwelt upon by the late Dr. Todd, and he very properly distinguished between two varieties of it whose pathology is altogether different. To the one variety he gave the name 'early rigidity,' and to the other variety that of 'late rigidity.' In some cases hemiplegia comes and goes without the appearance of either form of limb-stiffening; the

muscles continue flaccid, whilst power is gradually regained. In other instances, where complete recovery does not occur, there may have been no early stiffening, although a condition of this kind (late rigidity) is apt to manifest itself after the lapse of many months.

'Early rigidity' may be limited to one or two fingers, or it may be so slight as only to show itself when we proceed to move the limb. When the arm is bent, for instance, if we attempt to straighten it there is resistance on the part of the biceps. On other occasions, if we try to flex the arm, the triceps responds, and partially resists our effort by some amount of contraction of its fibres. Or the rigidity may be very marked; when the arm is drawn to the side of the chest, the elbow and wrist are firmly bent, and the fingers are flexed upon the palm. In the most marked cases any attempts to straighten the limb only seem to increase the spasm, and may cause pain as well as set up some amount of tremor or slight clonic spasms in the limbs of one or both sides; though at other times, as you may have seen in the case of an aphasic man lately in our wards, the spasm yields considerably under steady pressure, and this, moreover, produces no pain.

Early rigidity may affect the leg at the same time as the arm, and when it occurs we find the thigh drawn up and the knee firmly flexed so as to bring the heel close against the buttock. The arm, however, is much more frequently affected than the leg. This kind of spasm may persist for many weeks or months, with only slight variations from day to day. The muscles do not show any signs of wasting for a long time, and they generally respond to faradization rather more readily than those of the healthy side. For one or two hours also after the extensors have been well faradized, the spasm may continue notably lessened. And we occasionally find with such a form of rigidity that the spasm relaxes altogether during sleep, the muscles resuming their rigid condition during the act of awakening, as in other allied spasmodic states.

When this particular form of muscular rigidity exists, moreover, the reflex excitability of the limbs, but more especially of the leg, is often decidedly increased. Tickling of the sole of the foot or contact with a hot spoon may then cause decided reflex movements, so that the leg is quickly drawn up—very much to the surprise of the patient—in cases where he has not the least volitional control over it. Such reflex movements may also occur in the arm, though they are less frequently seen here. Occasionally they take place in either limb without external stimulus—when the

patient yawns or coughs. Such reflex movements are rather dreaded by those who have experienced them, since they are often somewhat painful.

Early rigidity may show itself after some days, or even immediately after the commencement of the attack, whilst the patient is still in a state of apoplectic or epileptiform stupor. When occurring thus early, the arm may be flexed and rigid either persistently or at times; or the rigid condition may alternate with one of clonic spasm. Sometimes, again, the arm may be rigid whilst the leg is affected with clonic spasms, and *vice versâ*.

As to the precise meaning of this 'early rigidity' we know very little. It is said to be indicative of an 'irritative' influence of the brain lesion, although it may occur with injuries of varying nature. It is doubtless in the majority of cases a grave symptom, and its appearance diminishes the chance of the patient's complete recovery. After a time, if the spasm does not relax, changes begin to take place in the muscles, tendons, and joints of the limb, which may ultimately leave it permanently contracted and useless. The lesion of the brain in these cases is often a large one, seriously implicating some extent of the cortical grey matter and destroying a considerable area of subjacent white substance. Lesions in and about the optic

thalamus also not unfrequently produce this kind of rigidity.

'Late rigidity' supervenes in some cases after many weeks or months, although previously all the muscles may have been in a flaccid condition. This form of rigidity is also, like the other, generally more marked in the arm than in the leg. It commences after the muscles have undergone some wasting. It may be limited to the flexors of the fingers, or it may extend to other muscles, so that in the most extreme cases the arm becomes immovably bent at the elbow, the wrist is firmly flexed, and the fingers are pressed against the palm of the hand-so forcibly, it may be, as to cause some amount of ulceration in this situation. Attempts to straighten the contracted parts produce much pain, and are usually ineffectual, owing to the shortening which has taken place in the muscles and tendons. In the leg, a similar contraction of the hamstring muscles may exist, with rigid tendons. At the same time there may be more or less contraction of muscles about the ankle-joint. Whereever it exists to a well-marked extent, the affected muscles always become notably wasted.

Dr. Todd thought that these characteristics of 'late rigidity' were brought about under the in-

fluence of the process of cicatrisation taking place at the seat of brain lesion. The contracting and indurating changes by which such a process as cicatrisation is distinguished were supposed to occasion an 'irritation' of adjacent nerve-tissue, which thence became communicated to the paralysed muscles.

As to the mode of conduction of this influence, or as to the precise degree of casual connexion between the process of cicatrisation in and about a brain lesion and the phenomena of late rigidity, there is much room for doubt. It is unquestionably true that where a limb remains permanently paralysed, certain nutritive changes are apt to be set up after a time in its nerves, its muscles, and its joints, though it is not always easy for us to decide whether these changes are directly occasioned by the brain lesion, or whether they are produced under the more immediate influence of some unusual extension of those 'secondary' changes in the spinal cord which are so often directly occasioned by damage to the motor tract in the brain (p. 172). The latter view, and strong evidence in favour of it, have been forthcoming during the last few years.

Tremors and Choreic Movements in Paralysed Limbs.—These are some of the rarer complications of the hemiplegic condition. They sometimes take the form of a more or less persistent tremor allied to that met with in paralysis agitans. Of this I only recollect seeing one example, and many of you may have seen the same patient, as she was in No. 3 ward during the early part of the session. Her case has already been referred to (p. 149) as presenting the signs of a lesion in the right crus cerebri. She became affected with tremors in the left hand and wrist when she had regained some amount of motor power. They consisted principally of slight, rapid, and partial pronations and supinations of the hand, together with twitchings of the extensor tendons at the wrist. A few weeks later, tremors commenced in the toes of the left foot, consisting principally of rapid flexions and extensions. This condition lasted for many months, but she afterwards improved considerably in general health, and the tremors gradually diminished. They were always decidedly worse when she was under observation, and entirely ceased during sleep.

At other times these movements in hemiplegic patients are more choreic in character, affecting the hand and arm principally, though occasionally extending to the leg and even to the face. The most marked example of this complication which I have seen occurred in a young woman who had

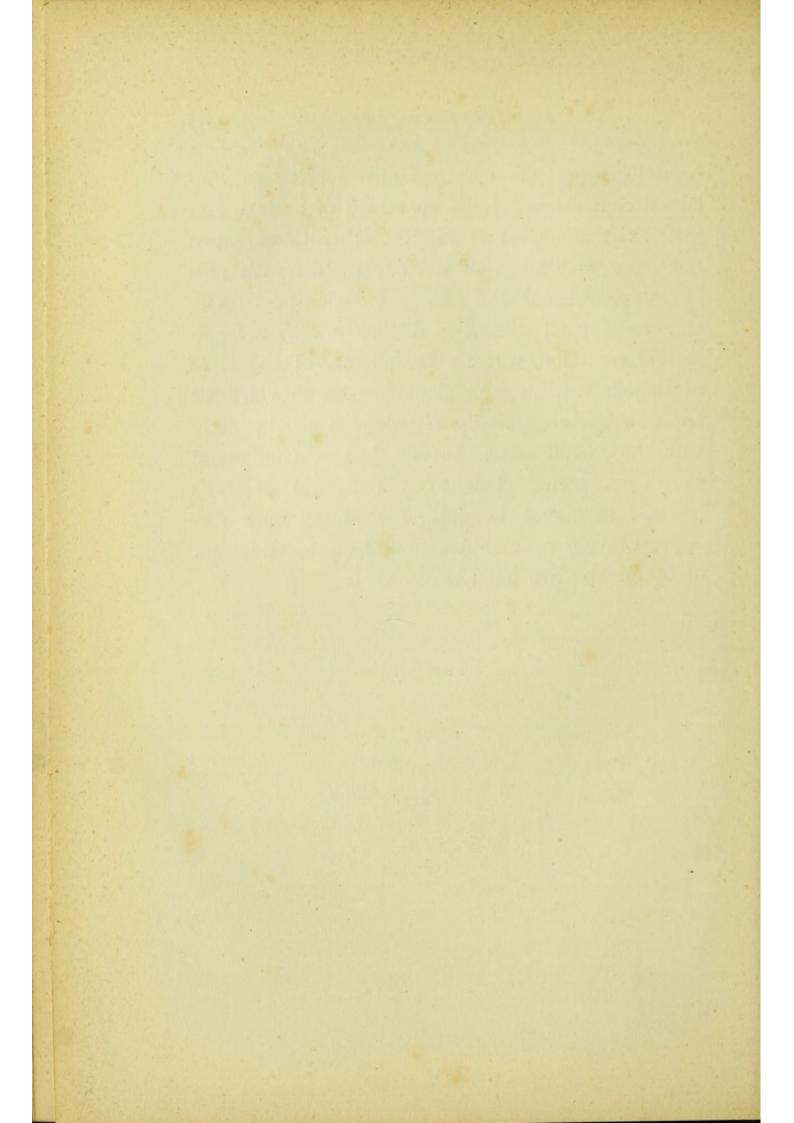
been partially paralysed on the right side from her fifth year, and who was also subject to frequent fits in which the convulsions were unilateral and affected the same side. After some of these attacks her paralysis would at times become more marked, and of late she had been suffering from distinct choreic movements in the hand and arm and also to a slight extent in the leg.

I have also seen a case in which unilateral tremors were well marked, and first in order of appearance. They were associated, not with a definite hemiplegic condition, though with well-marked weakness on the affected side. The tremors had commenced in the mouth and eyelids of the right side. They spread to the hand and arm in the course of a few days, and after another week to the right lower extremity.

In neither of these cases was there any very marked diminution of sensibility on the affected side; though M. Charcot considers hemi-anæsthesia to be a not unfrequent accompaniment of the tremors and twitchings in paralysed limbs to which I am now referring.

These choreic movements which, in patients thus affected, are almost constantly present, are not to be confounded with occasional attacks of tonic spasm, occurring in the arm especially, of certain

hemiplegic patients who are also subject to unilateral convulsions. Such an attack of tonic spasm may exist alone, or it may at other times prove the mere first stage of a complete fit, with unilateral convulsions and loss of consciousness. The alliance of such spasms is distinctly with the epileptoid condition, whereas the alliance of the more continuous twitchings or clonic spasms to which we are now more especially referring, is as distinctly with the choreic state. In fact, just as a unilateral chorea may give place to a unilateral paralysis (p. 29), so may a hemiplegic condition as it disappears favour the manifestation of one-sided tremor or of an abortive hemichorea.



## LECTURE V.

ALTERATIONS IN NUTRITION.—DEGENERATIONS IN THE CORD.—ARREST OF GROWTH IN LIMBS.—APHASIA AND OTHER DEFECTS IN SPEECH.
FUNCTIONAL DIFFERENCES BETWEEN THE TWO CEREBRAL HEMISPHERES.

- Alterations in Nutrition—Acute Sloughing—Congestions and Hæmorrhages—Dropsy of Paralysed Limbs—Inflammation of Joints—
  Changes in Nerve Trunks—Muscular Atrophy—Secondary Degenerations of the Spinal Cord—Their Distribution and Occasional Extensions—Their Relation to Late Rigidity and other
  Changes.
- Arrest of Growth in Paralysed Limbs—Occasion of its Occurrence— Affects Arm more frequently than Leg—Distal Segments principally implicated—Usually co-exists with Atrophy of part of Brain—Not to be confounded with Infantile Paralysis—Arrested Growth of Nails.
- Defects in Power of Speaking—Difficulty in Articulation—Illustrative
  Case—Minor Defects of same kind—Amnesia—Meaning of the
  Condition—Different Degrees of Intensity—Illustrative Letter—
  Previous Defects not specially related to Disease of one or other
  Side of the Brain—Other Opinions on this Subject—Aphasia is
  specially related to Disease of Left Hemisphere—Different Grades
  of Severity—Illustrative Case—Gustatory and Visual Incitations
  often more potent than Auditory—Illustrations—Kind of Speech
  met with in Aphasics—Mental Power in some Cases—Ability to
  write—Illustration—Aphemia—Agraphia—Interesting Case—
  Ataxic Defects in Speech and Writing compared—Different Combinations of Defective Intellectual Expression met with—Importance of Left Hemisphere in initiating these acts—Regions
  principally affected—Third Frontal Convolution—Explanation of
  its importance—Anatomical Investigations.
- Peculiarities attaching to Lesions in the Right Cerebral Hemisphere—
  Greater Fatality of—More frequently produce some of the Various
  Trophic Changes—Convulsions and Tonic Spasms—Hysterical
  Paralysis—Greater Tendency to produce Paralysis or Convulsions
  on same Side of Body—Possible Explanation of this Class of
  Cases—Importance of looking to Secondary Spinal Degenerations.

## LECTURE V.

GENTLEMEN, — In continuation of the enquiry which occupied our attention at the close of the last lecture I shall now proceed to give you a brief account of certain Alterations in the Nutrition of Parts which may be associated with hemiplegia. I shall refer to them in the time-order in which they are apt to show themselves, and shall afterwards endeavour to show how they are related to the lesion which produces the hemiplegic condition.

I. Acute Sloughing.—There is one rare form of lesion that may begin to manifest itself within three or four days after the onset of the apoplectic attack with which the hemiplegia commences. This is an acute process of sloughing, which, quite independently of all external irritating influences, occasionally shows itself, not over the mid-sacral region as in spinal disease, but over the centre of the gluteal region on the paralysed side. I say the pro-

cess may occur independently of all external irritating influences, because M. Charcot tells us he has seen it even where the most minute precautions have been taken with the view of preventing its occurrence—where the patient has from the first been made to lie on the non-paralysed side, and where all contact with urine has been most carefully avoided. At the same time the liability to sloughing on the paralysed side under the influence of irritation is also notably increased in these cases, so that such a process is very apt to occur on the heel, on the inner side of the knee, and, in fact, wherever the skin is exposed to slight though continuous pressure.

The acute slough in the gluteal region commences in the form of an erythematous patch which appears in some cases as early as the second or third day, though generally somewhat later. The red patch is rapidly succeeded by the appearance of bullæ and hæmorrhagic spots, and these are followed by a necrosis of the skin which spreads concentrically. The patient frequently does not live long enough for the slough to separate; for this lesion is a complication seldom occurring except in cases which are about to prove rapidly fatal. Nothing definite can at present be said concerning the situation or nature of the cerebral lesions with which

this trophic change is most apt to manifest itself. It has been known to occur with hæmorrhage, with softening, and also after traumatic injuries of the brain.

2. Congestions and Hæmorrhages.—Other lesions in internal organs are also occasionally produced at this early stage of the disease, though these, on account of their situation, are only recognizable where the malady terminates fatally. Amongst the principal of such lesions are to be included congestions and actual hæmorrhages into the substance of the lungs; though in addition there may be minute extravasations in or beneath the pleura, the endocardium, and the mucous membrane of the stomach, as well as into the substance of the supra-renal capsules and kidneys.

Schiff and Brown-Séquard have often produced experimentally, in some of the lower animals, hyperæmic conditions or actual hæmorrhages in similar situations by traumatic irritation of the pons and middle cerebellar peduncles, and, to a less extent, by irritation of the optic thalami and corpora striata. Brown-Séquard concludes from his experimental observations on animals, and from the fact that these lesions are most readily produced by injuries of the pons Varolii, that they are probably brought about by sudden vaso-motor contractions leading to rupture

of capillaries in the sites of hæmorrhage. He further believes from experimental evidence that this nerve influence or stimulation travels to the lungs, not by the vagus, but through the medulla and upper part of the cord and thence through the first thoracic ganglion of the sympathetic.

The principal practical bearing of such experiments is to be found in the explanation which they afford of the congested and hæmorrhagic condition of the lungs, apt to be met with (in the absence of other determining conditions) in many severe cases of cerebral apoplexy. They are, however, also interesting because they still further strengthen the conclusion supported by other evidence, that the pons Varolii is one of the principal centres of origin for vaso-motor nerves—not only for those supplying the limbs, but also for those of the thoracic and abdominal viscera.

3. Dropsy of Paralysed Limbs.—This condition is occasionally met with in cases of hemiplegia. When present it usually manifests itself in both arm and leg, or in either of them singly, in from three to six days after the onset of hemiplegia. It is mostly a complication of slight significance which tends to abate after a time.

My notes do not supply any very accurate information as to the kind of cases in which œdema of

limbs is most apt to present itself, but if we were to rely upon the physiological data furnished by the experiments of Ranvier and those of Goltz we should be led to expect that it would occur principally in those patients in whom there is a decided increase of temperature in the paralysed limbs (due to paralysis of the vaso-motor nerves), and in addition an anæmic state of the system, or one of incipient renal disease -conditions of body, that is, which of themselves predispose to the occurrence of dropsy. The observers above mentioned have shown by independent experiments that dropsy manifests itself more easily in paralysed limbs, partly because the dilated state of the vessels favours transudation of serum, and partly because an absence or diminution of nervous influence retards the rate of absorption.

We come now, gentlemen, to a consideration of trophic changes taking place at a later period—though, like the condition last referred to, they are also altered states of nutrition presented by the limbs themselves on the paralysed side. You must not, however, look upon the lesions to which I am now about to refer as universal, or even as common occurrences. They are, in fact, met with more or less rarely—though it is none the less desirable that you should clearly understand their nature in the cases in which they occur.

4. Inflammation of Joints.—The first, in order of time, is a subacute inflammation of some of the joints on the paralysed side of the body, which may appear either in the leg or in the arm; most frequently in the latter. Dr. Scott Alison originally called attention to this lesion, though it has been subsequently investigated by Brown-Séquard and Charcot.

Whilst this articular inflammation may show itself as early as the fifteenth day, it more often appears in from three to six weeks after the onset of the hemiplegia, or perhaps even later still. The joints become red, swollen, and painful when moved; and the sheaths of the tendons near the joints are also sometimes affected. The condition much resembles a rheumatic articular inflammation, only it is found to be limited to some one or more of the joints of the paralysed side, and it comes on at a comparatively definite period after the commencement of the hemiplegic attack. On post-mortem examination of the affected joints, M. Charcot has found all the characteristics of a subacute synovitis.

Very frequently, the joint affection after a time gradually subsides; and in some cases it may, from the first, be so slight as only to reveal itself by obscure pains when the limb is moved.

5. Changes in Nerve-Trunks. - In the same class

of cases as those to which I have just referred, tenderness often exists in different parts of the arm or of the leg, instead of being limited to the joints; and where this occurs, as Cornil originally pointed out, we find the pain on pressure more especially marked along the course of the principal nerves. Cornil has ascertained that this tenderness is frequently occasioned by a sub-inflammatory hypertrophy of the nerves and of their sheaths. The nerve-trunks were found to be notably enlarged and much more vascular than natural, the enlargement being due to an overgrowth of the connective tissue elements entering into their composition.

In cases where unprovoked 'pains' are complained of in the paralysed limbs, they are most frequently traceable to the existence of this condition of the nerves, either alone, or to this in combination with the joint affection. But in some rare instances in which such pains are felt, a tenderness of the limb generally may be detected—that is, a tenderness not specially limited to the regions of joints and large nerves—as in the case of the woman suffering from left hemiplegia who is now in ward No. 4.

You must understand that the changes in the joints and nerve-trunks on the paralysed side of the body to which I have just been referring often supervene with, and indeed constitute part of, the

state known as 'late rigidity.' We only have to superadd to such changes certain others taking place in the muscles of the limb affected, in order to complete the state. The nature of the latter changes, and the cause of the set as a whole, I shall now endeavour briefly to indicate.

6. Muscular Atrophy.—It occasionally happens, as in the case of the woman now under our observation and to whom I have just alluded, that an early and altogether special wasting takes place in the muscles of one or other limb within a few weeks from the onset of the paralysis. This event is, however, very rare, and not very many cases of the kind are on record. I can now only refer to two—one narrated by the late Dr. Todd, and one by M. Charcot.

In our case the patient is forty-six years of age. Whilst engaged in household work she dropped down insensible, and did not recover consciousness for two days and a half, when it was found that she was completely paralysed in the left arm and leg. On her admission into this hospital nine weeks afterwards, there was scarcely any trace of facial paralysis and no appreciable diminution of sensibility, though there was still complete loss of power in the left arm and leg. The temperature of the paralysed limbs was ½° higher than that of the non-paralysed

limbs. Some pains and tenderness were already felt in the left upper extremity, and its muscles were at this time also very decidedly wasted around the shoulder, in the arm, and in the forearm. The muscles of the left leg were, however, not at all wasted, and neither were pains nor tenderness felt in any part of the limb. The muscles of each extremity responded a trifle more readily to faradization than those of the opposite or non-paralysed side.

Such was the condition of this patient on admission nearly two months ago. Since then she has regained power considerably in the left leg, and is now able to flex and extend it both from the hip- and the knee-joints. The condition of the arm, however, has become worse. Whilst she has not regained the slightest power over it, the wasting has become even more marked and the whole limb more painful. Pain is felt when we attempt to move it in any way, and there is a distinct tenderness in almost all parts of the limb—not specially confined to the joints or course of the large nerves.

Now this is a condition of great importance. Owing to the rarity of such a sequence, it fixes our attention, and compels us to inquire about its cause and as to its relations with other trophic lesions in the paralysed limbs. You may look upon it as a fact firmly established in pathology that lesions of the

cerebral hemispheres do not of themselves produce an early wasting of muscles in the paralysed limbs—that is, no wasting which may not be explained as a result of the mere disuse of the limb. Hundreds of cases of brain lesion attest the truth of this view: so that when we meet with apparent exceptions to the rule, we are bound to search for some special cause.

Secondary Degenerations in the Spinal Cord often Coexisting with Hemiplegia.—It has been known for the last twenty years that cerebral lesions damaging the motor tract on one side—either in the corpus striatum, the crus, or the pons Varolii—give rise to a well-marked 'secondary degeneration' in the spinal cord, situated in the lateral column, though on the side opposite to that in which the brain lesion occurs. A case of this kind was described and figured many years ago by Cruvelhier in his "Anatomie pathologique."

I cannot now explain fully what we know concerning these very interesting 'secondary degenerations' of the cord. I will merely state that the mode of production of similar lesions in peripheral nerves was first pointed out by the late Dr. Waller, who ascertained that when a motor nerve is cut across, the part below the section (being severed from its central ganglionic connexions) undergoes a rapid process of fatty degeneration throughout its whole length. The white substance of the nerve becomes completely broken up into fatty globules and granules in the course of from five to eight days. In explanation of this remarkable change, it is supposed that the ganglion cells in the anterior horns of grey matter in the spinal cord exercise a sort of necessary or controling influence over the nutrition of the motor fibres with which they are connected, so that any severance of these connexions leads to a simultaneous degeneration throughout the whole length of the nerve-fibres below the point of section.

A process essentially similar to this seems to be initiated when the motor tract is damaged in the corpus striatum or in the lower parts of the brain, as Türck, Bouchard, and others have fully shown. The inferior portions of this tract then become rapidly degenerated throughout their whole extent: so that certain cells in the corpus striatum are supposed to exercise a trophic influence over the fibres composing the spinal motor tract, down to the points where they severally terminate in the before-mentioned motor cells of the anterior cornua. The fatty change in the nerve-fibres which first occurs is also soon associated with an overgrowth

of the neuroglia or interstitial tissue throughout the same motor tract, both in the base of the brain and in the spinal cord.

In this way a well-marked band of sclerosis is after a time produced, which may be traced through the crus, through the pons and anterior pyramid of the same side, and thence into the opposite lateral column of the spinal cord. After a time the tissues in this area of degeneration and fibroid overgrowth diminish in bulk, according to their custom, and then an atrophy or wasting becomes obvious to the naked eye along the whole length of the tract specified. This is the condition so frequently met with in persons dying some months or years after a severe attack of hemiplegia.

Now the process of fibroid overgrowth, thus initiated, may, and mostly does, remain strictly limited in its seat; but in some cases it spreads, as such processes are apt to do, to contiguous healthy regions. In view of this possibility, therefore, you must recollect that the inner and anterior portion of the degenerated tract of spinal cord tissue lies, throughout its whole extent, close along the side of the corresponding anterior grey cornu. We have therefore only to imagine this process of fibroid overgrowth extending inwards or forwards so as to involve the anterior horn of grey matter (either in

the cervical or in the lumbar region, or in both) to account for certain nutritive changes occurring in one or both limbs of hemiplegic patients. An atrophy of the great ganglion cells in the anterior cornu would give us the pathological condition pertaining to, and therefore the real explanation of, the occurrence of rapid muscular wasting in the cases to which I have already directed your attention.

This was the explanation I offered to you of the speedy atrophy of the arm muscles in the case of the woman now under our care. But I have since ascertained that such a mode of causation has been actually verified post mortem in one very important case by M. Charcot, to whom we are also indebted for an increase in our knowledge of the pathology of nervous affections in many other directions. His case may be briefly recorded as follows:—A woman about seventy years of age, was suddenly paralysed on the left side by an extravasation of blood into the midst of the right cerebral hemisphere. The limbs on the paralysed side very soon became rigid; and in rather less than two months from the commencement of the attack the muscles of both arm and leg began to waste. This wasting progressed rapidly, and was associated with a notable diminution in the electric irritability of the muscles. By the time the atrophy of the muscles had become well marked, the

skin of the paralysed limbs presented bullæ here and there, and these bullæ soon gave place to wounds wherever the parts were subjected even to slight pressure. The patient having died shortly after this condition had been produced, sections of the hardened cord were made, which showed the usual band of sclerosis in the left lateral column, though in this case the band had, to use M. Charcot's expression, "extended to the anterior cornu of grey matter on the corresponding side, and had there produced an atrophy of a certain number of the motor ganglion cells."

Now in the above-mentioned case the extension of the morbid process was rapid and probably well-marked; but in the patient now under our own observation there is reason to expect that the extension of the process of sclerosis to the grey matter is less marked, and that it exists only in the cervical region of the cord—or at all events does not affect the lumbar region, seeing that the nutrition of the leg is not at all interfered with. In other cases, however, an extension of the fibroid overgrowth from the lateral column to the contiguous grey matter may take place in the lumbar region more especially, and the process may be so far moderate in extent as not to produce any very decided muscular wasting, though the degeneration

of the grey matter may be sufficient to prevent a return of motor power to the leg.

See, then, how we have traced a very probable relationship between our case of hemiplegia now in the ward with wasting of the muscles of the arm, and those other cases of delayed recovery of power in the leg of which I have already spoken to you (p. 141), and to the consideration of which I promised to return. You will recollect my telling you that, according to Trousseau, pains in the limbs are of very frequent occurrence in these cases, and you will now understand how such pains may be due to sub-inflammatory changes in the nerves or in the joints, such as would be particularly liable to occur if the prolonged paralysis of the leg were due to an extension to the grey matter of the secondary changes in the spinal cord. For, as I should have mentioned to you before, M. Charcot has also ascertained that there is a close, and probably a causal relationship between these degenerative changes in the anterior horns of the grey matter and those pathological changes in the articulations of the same side to which I have already referred.

So that there seems good reason for the belief that the nutritive changes generally, which go with and in great part make up the condition known as 'late rigidity,' are much more likely to be due to irritative influences emanating directly from the spinal cord and occasioned by extensions of the secondary degenerations there existing than to irritative influences proceeding directly from the cerebrum itself as Dr. Todd supposed.

I have only one other suggestion to make concerning the effects of these secondary changes in the spinal cord, and it is this:—Occasionally, where the leg does not recover power as it should do in a case of hemiplegia, the other leg may after a time also become weak, so that a paraplegic condition is slowly brought about without any obvious cause. It develops as the previous hemiplegic condition disappears, and after a time comes to a standstill. We have lately had a case in the wards which appeared to be one of this kind; and though such a clinical sequence may be explained by a slow spread of fibroid degenerative changes into the grey matter on both sides, from its original focus in the lateral column, I cannot at present support this view by post-mortem evidence. The cases presenting this feature are, moreover, exceedingly rare.

Arrest or Retardation of Growth in Paralysed Limbs.—There is another abnormal condition of the limbs occasionally met with, to which I ought to call your attention. It is almost always erroneously

spoken of as one of 'atrophy,' whereas in reality it is a clear instance of arrested or retarded growth. This condition is frequently seen in cases where a lesion of the brain of a severe nature, producing hemiplegia, has occurred at any period between that of birth and the third or fourth year, though it may show itself to a less extent with lesions produced at a still later age. In such cases the arm and leg, or the arm only on the paralysed side, grow much more slowly than on the sound side, so that when the period of growth ceases, the paralysed limbs remain permanently smaller than those of the opposite side.

The arm is more frequently affected than the leg, and when the arrest of growth is well marked in it there is generally also some amount of rigidity about the elbow- and wrist-joints. In thirteen cases which have been under my care, where this diminished size of the limbs has been met with, I have found that the retarded growth has almost always been most marked in the distal portions of the limb. Thus, whilst the length of the humerus or of the femur has been equal on the two sides of the body, the hand and foot on the paralysed side have been notably smaller, and the forearm or leg bones of the same side have been shorter than those of the sound limbs. Occasionally, however, the proximal por-

tions of the limb have also been smaller than natural.

In twelve of the thirteen cases of this kind which have been under my care, the hemiplegic condition became established at different periods between birth and the end of the second year. In the thirteenth case it set in with an epileptiform onset at the end of the fifth year, and here there was only a very slight diminution in the size of the paralysed hand, when the patient—then three years of age-first came under observation. In no less than eleven of these cases it was a right-sided hemiplegia from which the patient suffered, and even in one of the two exceptions, in which the paralysis was on the left side, the little patient, a child five and a half years old, had also suffered from a temporary right hemiplegia. The numbers are small upon which to base any conclusion, but I am inclined to think there may be something more than chance in this comparative frequency of right-sided paralysis in infancy.

In six of these cases the patients were either unable to speak at all, or the period at which they commenced to do so was very much retarded—in one case to the fifth and in another to the sixth year. They suffered also from fits at intervals, in which the convulsions were unilateral and confined

to the paralysed side. In seven cases there was no affection of speech, and in five of these there had been no fit since that which ushered in the paralysis. One of these latter cases was remarkable in another respect. The patient was a boy, and sixteen years of age when he came under observation. Paralysis was ushered in by right-sided convulsions when he was twelve months old, and he has continued to have fits of the same character ever since. He began to speak at the usual time, and there is no arrest of growth in the paralysed limbs, except in the hand which is smaller than that of the left side. The whole of the left side of the face is, however. decidedly smaller than the right. This is the only case of the kind I have met with, and I could discover no clue as to the cause of the retarded growth of the face on the side of the brain lesion.

Where post-mortem examinations have been made in such cases of arrested growth, actual loss of substance or marked atrophy has generally been met with in some part of the cerebral hemisphere.

You must not suppose, however, that a condition of the limb or limbs such as I have just been describing is met with only as a sequence of a hemiplegic attack occurring in infancy. A similar retardation of growth, with diminished size of one or more limbs, may be also due to a previous attack of the so-called

'infantile paralysis.' Here we may have one or both legs affected, but only very rarely one leg and one arm on the same side, and there would usually be differences also as regards the mode of onset. The muscles, too, in these cases of spinal disease are actually wasted, and have often quite lost their irritability under the influence of faradization—or have lost it to a much greater extent than is the case when we have to deal with an arrested growth from brain disease with hemiplegia.

I ought perhaps also to call your attention to the fact that even in ordinary cases of hemiplegia, as Dr. Weir Mitchell has lately pointed out, the nails of the fingers on the paralysed side either cease to grow, or increase at a much slower rate than those of the sound side.

I now pass, gentlemen, to a consideration of some signs or occasional components of the hemiplegic condition which are apt to present themselves—not equally with lesions of either hemisphere, but more especially when one half of the brain is injured rather than the other. Some of these signs are peculiarly associated with injuries of the left, and others with injuries of the right hemisphere.

## FUNCTIONAL DIFFERENCES BETWEEN THE TWO CEREBRAL HEMISPHERES.

The first subject to be considered under this head is one to which very much attention has been given of late years. I allude to the Various Defects in the Power of Speaking or of Writing induced by cerebral disease.

I have already spoken to you of those Difficulties of Articulation which, when constituting part of the hemiplegic state, are most marked where we have to do with lesions of the pons Varolii. In these cases the difficulty is occasionally so marked that no articulate sound can be uttered, and this speechless condition is commonly associated with more or less immobility of the tongue and difficulty in swallowing. But, when a patient is affected in such a manner there is generally no difficulty in coming to the conclusion that we have to deal with a mere difficulty of articulation.

As illustrative of this kind of defect and of several other important points in connexion with extensive disease of the pons Varolii, I am tempted to cite the following details concerning a patient under my care some years ago at the National Hospital for the Paralysed and Epileptic. The previous history was

obtained from the patient himself and from his son, both of whom were very intelligent.

J. W., æt. 54, married, and the father of six children, was a plumber, but never had had lead colic or lead palsy. Never had syphilis or rheumatic fever. Heart healthy. About six months ago he began to feel pain in the front of his head and left side of body, coming on at intervals for about two or three weeks. During the same time, also, he was unable to taste his food properly. One day, whilst at work, he suddenly found he could not use his left hand, so that he gave up working and went to his home which was close at hand. In about ten minutes he is said to have become completely paralysed on both sides of the body, whilst his face was drawn to the right side. The attack was attended by no convulsions, and no loss of consciousness, but he found himself unable to speak or utter a single articulate sound. After this condition of general paralysis had lasted one month, the patient gradually began to gain power over the During the first month he could not right side. move his tongue at all out of his mouth, and had very great difficulty in swallowing. Even still he is obliged to have his food cut up into very small pieces.

During the first three weeks this patient appeared to be quite deaf, though at the same time he could perfectly understand what was written for him on a slate. After the third week he gradually regained his power of hearing, and became also able to utter single words when asking for food. But, even soon after he first began to regain his power of speaking, he could slowly write answers to questions concerning his business—and only rarely had any difficulty in recollecting words. The patient gradually improved in his power of articulating during the second month, though his voice was still muffled: and ever since he has spoken indistinctly and in a low voice, though with no difficulty in finding words so long as he is not excited.

The sight of the left eye was much impaired at first, and still continues bad. The sense of taste was almost lost at first, and he has not yet regained it on the left side. Sense of smell on the left side is also much impaired. At and immediately after the time of attack it was found that he was extremely sensitive, and unable to bear the slightest touch without pain on the left side of the face, the left arm, and the left half of chest and abdomen; but over the left leg the sensibility seemed altogether lost. The sensibility of the right side of the body was natural. This state of things has continued, with very little change, ever since; but during the last two months he has been regaining sensibility on the inner side of the thigh and leg. He now complains of constant,

though exacerbating, pains all over the left side of the head. The whole left side of the body has also generally felt 'colder' than the right.

Having begun to acquire power over the right side after the first month, by the end of the second this was completely restored. Soon afterwards he gained a little power in the left leg, which has since continued slowly to improve. He can now walk about a quarter of a mile with the aid of a stick, dragging his left foot along the ground. Within the last week he has been gaining some slight power over the muscles of the left shoulder, but still cannot move his fingers in the least.

During the whole time of his illness the patient has passed his evacuations naturally. He frequently cries and sobs violently; rarely laughs; this from the first, though it has been more marked of late. Mind otherwise unimpaired. Sleeps badly; appetite good.

This case is an important one in many ways. It affords, in the first place, a good instance of the kind of loss of speech which is met with in disease of the pons Varolii, showing how it is associated with extreme difficulty in deglutition, and gradually gives place to a condition in which mere difficulty of articulation becomes a most unmistakeable feature. The fact of the paralysis for a time being bilateral, of the grave and persistent modifications of sensibility on

the left side of the body, as well as the existence of deafness and the impairment of the sense of taste, when taken together with the difficulty in articulation and deglutition, present a set of symptoms pointing almost indubitably to a lesion of the pons Varolii.

Minor degrees of a similar defect in the power of articulation are often met with in cases where large cerebral lesions are situated higher up than the pons—that is, in parts nearer the cortex of the hemispheres. But where the lesions are in these situations, the difficulty in articulation not only tends to become less marked as the cortex is approached, but it also usually lasts for a much shorter time. When about to disappear, or when existing in their slighter forms, these defects dwindle away merely to some slight 'thickness' of speech.

Other modes of impairment of speech exist which have been roughly classed under the names 'Amnesia' and 'Aphasia;' and it would seem that these defects in articulate speech are to be paralleled by similar defects in the power of intellectual expression by means of writing.

In the defects of speech ranged under the head Amnesia we have to do apparently with a kind of inco-ordination in the action of those higher cerebral centres whose business it is to translate thought into

the corresponding motor acts—there is an irregular carrying out, in fact, of those nerve processes by which the thought of the patient receives that physical expression which renders it intelligible to others. The individual knows what he wishes to say, but there is a defect in the subsequent molecular actions going on in his higher nerve centres of such a nature as to cause hesitation or delay in the utterance of right words—and, what is worse, the substitution occasionally of entirely wrong words, or even of a meaningless set of sounds, in the place of those which he wishes to utter.

Although such a patient may be quite unable to prevent these mistakes or failures, he usually shows by his manner that he is aware of having made them—and yet any attempts to rectify the errors only serve to make matters worse. This defective action in the speech centres and their related parts is very comparable with what occurs in other nerve centres in locomotor ataxy. In this disease a man may have an adequate knowledge of what he intends to do, though when he attempts to move his legs in definite directions he jerks them about in an irregular manner, or even moves them in a way the reverse of what he had intended. In each case, therefore, we have to do not so much with lack of power as with involuntary or

misdirected power. All grades of the amnesic defect in speech are met with, varying from mere forgetfulness or slowness in the use of words up to such an entirely wrong use of them as to make speech almost wholly unintelligible.

And such defects in oral speech often coexist with more or less marked difficulties of the same kind in the translation of thought into written speech—that is. in writing. The patient is then able to perform the mechanical act very well, though he cannot group letters correctly into words: he spells altogether wrongly or even writes words which have no resemblance to those he wishes to employ. Such a patient often cannot group words so as to form definite In his attempts to express himself, he propositions. omits some words, or he uses altogether wrong ones, such as he had not intended to employ. As an instance of this kind of defect, I may quote the following letter, which was written to me by one of Sir Wm. Jenner's patients at a time when I was in charge of his wards :-

UNIVERSITY COLLEGE HOSPITAL, Ward 8.

To - BASTIAR, Esq.

Sept. 6/69.

DEAR SIR,—I am said of my illness. As twelfth years has not the loss of my right eye, you had a lad at once reaching of a shell, and quite an accident. \* \* The left eye was just for years, and do say. \* \* If I have of fifteen years, that I write Plays, and contribute that many of the London journals and newspapers. And I write Essays, Come-

dies, Poem, Dramatic Criticism, &c., and a a thousand. \* \* I have twenty years I gave the appointent that "read for press" for "The Examiner." \* \* In the good health in 1863 was the "neuralgia," and go once at once, or that bad that it be is done. \* \* In September 1867 that blood gone by head, and I cannot by that left eye. \* \* In Septemtember 1868 from eye been better, and can write and read! \* \* In on Good Friday, in the night, has a had a "fit"; and the right leg, right arm, and that I cannot say or can about, and of Palalysis and the Tic Dolorens. I have very ill. \* \* In three weeks I come to that the Hospital here. That have does better. The Hospitial goes to Eastbourne, and goes was ill that than has as ever. His "Tic" is bad, and the Doctor than the Hospital. \* \* His has talk have "counchant" and the "Tic" for rampant. \* \* I cannot write as good to need: for it at be further. \* \* And, dear Sir, this note that not do "the Queen's English Grammar." And if be better.

I, dear Sir,
Your obedient Service,
B. W. W.

The handwriting in the above letter was very good, and there were only two trifling erasures. The letter was divided into paragraphs in the manner indicated.

A patient who writes thus may be able to copy quite correctly any writing which is given to him, or he may be able to translate into written characters what he has before him in a printed page. The latter performance is, however, only imperfectly achieved in other cases.

Now, of the defects already referred to, I may at once point out that Difficulty in Articulation may be met with in lesions of either side of the pons, and also with lesions in higher parts of the brain in either hemisphere. Amnesic defects, again, which

are most marked and most frequently associated with lesions of the cortical grey matter, are generally believed to occur about as frequently with lesions of one as of the other hemisphere. Dr. Hughlings Jackson, however, as well as Brown-Séquard, are not exactly of this opinion: they are inclined to think that both difficulties in articulation and amnesic defects in speech are more frequent with lesions of the left than with lesions of the right hemisphere.

Concerning Aphasia and aphasic defects, however, there is not the same difference of view. Almost all authoritative observers now admit that in the great majority of these cases (at least where we have to do with right-handed people) aphasic defects are associated with lesions of the left hemisphere and right-sided paralysis.

Left Cerebral Hemisphere.—Under the name Aphasia we include a group of cases in which the patient is mostly unable to speak, although his occasional distinct utterance of some one or two words shows that his speechless condition is not due to a difficulty in the actual performance of acts of articulation of the kind met with where lesions exist in or near the medulla and pons Varolii.

These cases differ very much in severity amongst

themselves - partly owing to the extent and situation of the brain lesion and partly to the period of the disease at which the patient is seen. In the gravest cases of this kind the amount of general mental impairment is so great that the patient's inability to speak appears only as one form of mental impairment. Such severe cases are almost invariably instances of recent hemiplegia, and the patient may in the course of two, three, or more weeks show a very notable improvement in some or in all respects. In illustration, I may cite some further details concerning a case (p. 69) already referred to-that of a woman in whom right hemiplegia was initiated by a series of epileptiform attacks before her admission into this hospital.

Soon after admission the patient had another series of epileptiform convulsions, implicating both sides of the body, though principally the right—the twitchings also affecting the right side of the face. During the intervals between these convulsive attacks, it was found that the face was partially paralysed on the right side, that the right arm was completely paralysed, and the leg powerless to a less extent. She had had six of these attacks during the three days after admission, and during this time remained in a dull, lethargic state, with eyes half opened, but taking no notice of things going on around her,

or when spoken to. She did not lie still, however, but was restless, with a slow pulse beating 56 per minute. She continued in much the same state till the 13th of October, when the pulse altered in character, becoming much more frequent, and the patient gradually began to regain a certain amount of intelligence in look and manner. On the 19th, her attention could be at once arrested; she made decided efforts to reply to questions, and was able to say 'Yes' and 'No' indistinctly, though not appropriately. When told to show her tongue she opened her mouth, but did not attempt to protrude the organ. Pulse 88. Took food eagerly, and was able to swallow without difficulty. On the 26th, seemed still more intelligent. Did not protrude her tongue when told, but opening her mouth she took hold of it with her fingers with the view of bringing it forwards. Although unable to move it by an unaided volitional stimulus, on a sweet lozenge being applied to her lips she immediately put out her tongue with great readiness, and whilst eating the lozenge laughed and seemed much pleased. On the 28th, looked still brighter, and took notice of what passed around her. Made signs when she wished to attract the attention of the nurse. When asked if she had pain in the head, she nodded assent; when told to place her hand upon the painful part, she either did not move it at all or else moved it in quite a different direction. Paralysis of limbs and face continued much the same.

About ten days afterwards I examined her again carefully. She had continued to improve in the interval, and could by this time say 'Nurse' distinctly, in addition to 'Yes' and 'No.' She could not repeat the simplest vowel sounds, neither could she read single words in large print, either aloud or to herself, so as to comprehend them. She could not, in fact, point out individual capital letters of very large type. When asked to point out M, after a long time, and much pressing, she placed her finger upon W; and when told she was not right, and asked to point out W, after a still longer interval, she laid her finger upon S. She seemed to recognize familiar objects, and know when the right name was given to them. She could not be made to count at all by tapping with her fore-finger, after having been shown in the most deliberate way. She could not be induced to give a single tap, and only looked distressed. She seemed to recollect her own name. And although she did not give any signs of recognition when the name of the street in which she lived was mentioned, she immediately nodded assent when she heard the remaining part of her address, viz. "Fitzroy Square," pronounced. She rarely laughs, but frequently has fits of crying. She utters no additional exclamations when excited, and her vocabulary is confined to the three words above mentioned.

In this case, in which there was considerable mental defect, it may be seen that the inability to act under volitional stimuli was not confined to the case of speech. The patient was unable to do other much more simple things; and, strangely enough, it is frequently found that such patients are more unable to perform certain simple acts voluntarily in response to incitations made upon the auditory perceptive centres, than when corresponding incitations have been made upon the gustatory or visual perceptive centres. The case just cited supplied an instance of the greater power of the sense of taste than of the sense of hearing in evoking a simple muscular act, and you will recollect how the aphasic man, C-, lately in our wards, like many others, constantly made the most abortive and ineffectual attempts to put out his tongue when he was merely told to do so, though it rapidly appeared when he received the same request through his sense of sight—that is after being shown through his visual perceptive centres what we wanted. The performance of the act in each of these cases was just as voluntary as it would have been had the tongue been protruded

after a simple spoken request. The request was merely conveyed in a different manner—through the sense of taste in one case and through the sense of sight in another. The greater power of these latter stimuli is a matter of considerable interest, and is not without its meaning to the student of evolution.\*

\* Some curious instances of the greater potency of the sense of sight as compared with that of hearing, in evoking speech, are on record. Thus the condition of a patient seen by Dr. Hertz is thus described (Psycholog. Mag. vol. viii.) :- "In regard to his speech the following very remarkable circumstance was to be observed: he was able to articulate distinctly any words which either occurred to him spontaneously, or when they were slowly and loudly repeated to him. He strenuously exerted himself to speak, but an unintelligible kind of murmur was all that could be heard. The effort he made was violent, and terminated in a deep sigh. On the other hand, he could read aloud with facility. If a book or any written paper was held before his eyes, he read so quickly and distinctly that it was impossible to observe that there was the slightest fault in his organs of speech. But if the book or paper were withdrawn, he was then totally incapable of pronouncing one of the words which he had read the instant before. I tried this experiment with him repeatedly, not only in the presence of his wife, but of many other people: the effect was uniformly the same." Another case, possessing many points of resemblance, has been recorded by Dr. Hun (American Journal of Insanity, April, 1851). This patient had an obscure cerebral attack commencing with prolonged stupor. few days he began to recover from this condition, and understood what was said; but it was observed that he had great difficulty in expressing himself in words, and for the most part could only make his wants known by signs. There was no paralysis of the tongue, which he could move in all directions. He knew the meaning of words spoken before him, but could not recall those needed to express himself, nor could he repeat words when he heard them pronounced; he was conscious of the difficulty under which he was labouring, and seemed surprised and distressed at it. If Dr. Hun pronounced the word he needed, he seemed pleased, and would say, 'Yes, that is it,' but was unable to

Though aphasic patients are unable to give voluntary and preconsidered expression to their thoughts, words or even short phrases, such as oaths, may occasionally be uttered under the influence of strong emotion—that is, in a more purely reflex manner. We often find these patients able to make use of common words like 'yes' or 'no' in response to questions addressed to them, though they may be quite inappropriately employed. But, as Dr. Hughlings Jackson has pointed out, such a patient is generally quite unable to repeat one of these words immediately afterwards when asked to do so; he cannot utter it in a more purely voluntary manner.

Phenomena closely related to these are also sometimes seen when resident foreigners become aphasic.

repeat the words after him. After fruitless attempts to repeat a word, Dr. Hun wrote it for him, and then he would begin to spell it letter by letter, and after a few trials was able to pronounce it: if the writing were now taken from him, he could no longer pronounce the word; but after a long study of the written word, and frequent repetition, he would learn it so as to retain it, and afterwards use it. He kept a slate on which the words he required most were written, and to this he referred when he wished to express himself. He gradually learned these words and extended his vocabulary, so that after a time he was able to dispense with his slate. He could read tolerably well from a printed book, but hesitated about some words. When he was unable to pronounce a word he was also unable to write it till he had seen it written; and then he could learn to write as he learned to pronounce, by repeated trials. At the end of six months, by continually learning new words, he could make himself understood pretty well, often, however, employing circumlocution when he could not recall the proper word, somewhat as if he were speaking a foreign language imperfectly learned."

During recovery it is found that they are at first only able to express themselves in that language in which they are most automatically versed—namely, in their own native tongue. I have seen this in two patients. One was a German who had been long a resident in this country—yet after an attack of right hemiplegia and aphasia, he was for a long time unable to utter a word of English. When he began to speak he used German words only, and after he had almost recovered, if occasionally in want of a word whilst speaking English, it was always a German equivalent which first presented itself.

There are many instances on record in which, though the aphasic condition itself has been most complete, the mental powers of the patients have been well preserved. Such individuals have been able to read intelligently to themselves, and play games—like draughts or cribbage—perhaps better than their neighbours. The right hand being usually paralysed in aphasic cases, the patient does not write; and often, doubtless, he would not be able to express his thoughts in this way even if he possessed the necessary skill with his left hand. Some of these patients, however, can express themselves in writing, and learn to do it with the left hand. Thus, on the next page, is the copy of a short letter written to me in pencil by an aphasic patient who was and had been almost speech-

less for more than eighteen months. He was a builder by trade, and originally an active, intelligent man.

His illness began with an epileptiform attack, which left him paralysed on the right side and quite speechless at first. His mind was also much con fused for a time; but after two or three weeks he became able to understand what was said to him. The only articulate sounds he was able to utter for the first nine or ten months were 'Bi-bi-bi,' 'Poy-boybah' and 'No.' After fifteen months he became able to say both 'Yes' and 'No' appropriately; though whilst he continued under my care I never heard him utter any other articulate sounds than those above cited,-and, according to the statements of his wife, this was the extent of his vocabulary. He had regained almost the complete use of his right leg, though the arm and hand still continued very powerless. He had practised writing with his left hand, and wrote fairly well with it. I asked him to write a letter to me, and the next time I saw him he gave me a note of which the following is a literal copy:-

SIR, Jany 5 1869

I am extremely obliged to you for the trouble you have taken about my affairs. You did not say thether (sic) you have recovered from the severe cold and cough that you told me you were suffering from. I sincerely hope that you have quite recovered your health and strength.

I am,

ROBERT H---.

This note was written in less than half an hour and without any assistance. Yet since his attack the same patient had only been able to utter the two or three words and sounds above mentioned. He seemed to understand everything that was said to him, and appeared also to understand the newspaper equally well when he read it—which he did habitually. When told to count twenty by tapping on the table with the fore-finger of his left hand, he did it rapidly and always correctly. He could add quite correctly columns containing five figures when the numbers were low, but often got puzzled and made mistakes when the numbers were higher. According to his wife he was very active and intelligent, only much more irritable than he had been before his illness. He has had several epileptiform attacks since the one with which his illness commenced, and on each occasion the convulsions were almost limited to the right side of the body.

This case is certainly exceptional, and belongs to a class to which I have proposed to restrict the term *Aphæmia*—cases that is in which there is an inability to speak only, and not a more or less equal inability to speak and write such as we encounter in Aphasia proper.

It is occasionally found that by the time some amount of recovery has taken place—that is, after the patient begins to speak a little—the aphasic defect is associated with one of an amnesic character. You will, in fact, frequently meet with these two conditions variously intermixed.

In another class of cases, though the patient is able to speak fairly well or with only an occasional use of wrong words, he has lost the power of forming letters, and can only make mere unmeaning strokes. Such patients, moreover, can scarcely read at all, and cannot spell correctly. It was this kind of defect more especially that Dr. William Ogle proposed to include under the term *Agraphia*.

I had an extremely interesting allied case under my care at the National Hospital for the Paralysed and Epileptic, occurring in a woman, in whom there was a most remarkable inability to pronounce even the simplest words which she saw in print, though she could at once repeat the same word with the greatest ease when asked to do so orally.\*

\* This is, therefore, the reverse condition to that which usually exists (see p. 191). There can be little doubt that in this woman the connexions between the visual and the auditory 'perceptive centres' were broken through in the left hemisphere. Speech, therefore, could not be initiated through sight impressions though it was easily done through the sense of hearing. It could not be roused through the sense of sight, simply because the impressions revived in the visual centre were unable to awake their corresponding impressions in the auditory perceptive centre. And without this latter revival the motor incitations to speech cannot occur. This, at least, is my view.

E. L. had an attack of right hemiplegia with pretty complete aphasia in the early part of the year 1868. In the course of some months she improved considerably, though she continued subject to fits at intervals. After twelve months she was able to walk about with a little assistance, though she was still incapable of doing anything with the right hand and arm. She seemed thoroughly to understand everything that was said to her, and had in great measure regained her power of speaking. could repeat almost any word previously uttered in her hearing, and this without any hesitation, though she could not read even the simplest words in large type. Yet the same words could be uttered with ease immediately she had heard them pronounced. She copied the written word 'London' fairly well with her right hand, but could not write down 'cat' or 'dog' though she could spell the same words quite well. She could not even write down the first letter of either of these words.

Twelve months afterwards she was found to be in much the same condition. Could not read aloud even such simple words as 'and' and 'for'; could point out letters which were named to her with the greatest ease, but could not herself name the letters when they were pointed to. She had improved in her power of walking and was also able to talk rather better. She could read a letter silently so as to understand it, though she did not always seem to comprehend when she read from a newspaper or book. She still remains in much the same state and continues to have fits at long intervals.

From what has been previously stated it would appear that just as we have amnesic or inco-ordinate defects which show themselves both in oral speech and in writing, so we have real ataxic\* defects in which there is loss of power in writing as well as in speaking. That is to say, the loss of power of writing—or the agraphic defect proper—may exist in cases where the paralysis of the arm has been recovered from. The ataxic defects in writing and speaking most commonly encountered (though they often do not coexist in the same individual), have moreover a certain similarity in nature.

## Defects in Speech.

- a. Cannot utter a sound, or only mutters inarticulately.
- b. Constantly repeats some meaningless sound or sounds, such as 'tan-tan,' or 'cousisi.'
- c. Uses some single word such as 'yes'; and also mere sounds, such as 'ba-ba,' or 'poi-boi-ba.'

#### Defects in Writing.

- a. Can only make mere meaningless strokes.
- b. Can form separate letters, but cannot combine them in a suitable manner into words.
- c. Can write his or her own name, or some other very familiar word, without a copy.

<sup>\*</sup> The word being here used in its true sense, signifying 'powerles.'

- d. Uses some short phrase habitually, such as 'List complete,' or 'I want protection.'
- e. Uses three or four words or expressions—though in themselves meaningless or irrelevant in a constant and definite manner; as in one of the original cases (that of Lelong) recorded by Broca.
- d. When a polysyllabic word is dictated can write the first syllable, though the rest is generally all confusion. Or can write a single word, such as 'London,' alone, but fails to write more than the first syllable of this when told to write 'London is very big,'—commences, looks puzzled, and throws down the pen in despair.

A careful investigation of the subject we are now considering has sufficed to convince me that cases of defective intellectual expression, either by speech or writing or by both, are from time to time met with belonging to each of the following varieties, Some of these conditions are much more common than others, and though they are most frequently associated with a hemiplegic condition, it must not be forgotten that they occasionally occur—some of them more especially—without paralysis of the limbs. The first three varieties will include *Amnesic Defects* only, and are as follows:—

- Amnesic defect in speaking, but power of writing correctly preserved.
- 2. Amnesic defect in writing, but power of speaking correctly preserved.
- 3. Amnesic defect both in speaking and in writing.

We have then two groups in which Amnesic and Ataxic Defects are conjoined:—

- 4. An amnesic defect in speaking, with loss of power of writing.
- 5. An amnesic defect in writing, with loss of power of speaking.

And, lastly, we have three groups in which Ataxic Defects only are met with:—

- 6. Loss of power of speaking, but power of writing preserved.
- 7. Loss of power of writing, but power of speaking preserved.
- 8. Loss of power both of speaking and of writing.\*

Though the theoretical explanation of these various states is a subject of great interest, its full consideration would detain us too long. Very different opinions are, moreover, entertained upon the difficult and intricate questions which lie at the root of the problem, so that the subject does not admit of a brief or concise treatment.†

I pass on, therefore, to speak of certain anatomical facts which have been made out in reference to the seat of the pathological changes usually associated with Aphasia.

In the great majority of instances in which it presents itself, we have to do with right-sided paralysis produced by a lesion in the left hemisphere.

- \* The varieties 6, 7, and 8 correspond strictly with the conditions which should, I think, be named respectively:—Aphæmia, Agraphia, and Aphasia. Variety 5 would also mostly be considered as an instance of Aphasia, just as variety 4 may be regarded as an instance of Agraphia. Nos. 1, 2, and 3 are different varieties of Amnesia. Nos. 3, 5, and 8 are the varieties commonly met with—the others are much rarer.
- † My own views are expressed in two articles in the British and Foreign Medico-Chirurgical Review for January and April, 1869; and in them references will be found to the works or memoirs in which the reader may also find the views of other principal writers on this subject.

In fact, a long series of observations has compelled us to recognize the greatly superior activity of the left hemisphere, as compared with the right, in initiating motor acts subservient to intellectual expression. Just as the left hemisphere has undoubtedly to initiate the muscular acts by which writing is effected in right-handed individuals, so it would appear that from this same half of the brain the incitations habitually pass over which are destined to excite the motor acts of speech—even though the muscles concerned are bilaterally disposed, and always act in concert on the two sides of the larynx, fauces, tongue, and lips.

There is reason to believe that this initiatory action of the left hemisphere in relation to speech-movements is connected with a very slight precedence in its development as compared with that of the opposite hemisphere; and that this precedence is itself a more or less remote consequence of an inherited tendency to right-handedness. Accordingly it has been found, in a few well-marked cases, that aphasia when occurring in left-handed persons goes with left- rather than with right-sided hemiplegia. The facts, however, are not quite uniform, and more are needed thoroughly to clear up this subject.

Something definite may also be said with reference to the situation or part of the left hemisphere which is especially affected in aphasic individuals. In such cases the lesion has been principally met with either (I) in or around the third frontal convolution, (2) in the white substance between this convolution and the left corpus striatum, or (3) in this latter body itself.

Looking to the records of carefully-sifted cases, there can remain little doubt, I think, as to the importance of the third frontal convolution on the left side (Fig. I., I,I,I), in regard to the power of intellectual expression. This, being the region originally pointed out by M. Broca, is often spoken of as 'Broca's convolution'; and though we need not regard it as the seat of any supposed 'faculty of language,' we may easily imagine that the volitional stimuli destined to incite the motor acts of speech would pass off from a part of the brain which was in intimate functional relationship with many other regions of the hemisphere.

There must be some one spot from which such motor stimuli habitually pass over, if, as we have every right to expect, a definite order is observed in the discharge of brain functions. It is also easy to imagine that lesions in and around this spot, would, according to their situation, variously interfere with one or other functionally related part, and so tend to occasion considerable variety in the kind of

defect actually produced. And here, gentlemen, I am happy to tell you that the facts disclosed by minute anatomical investigation strongly tend to confirm the views upon this subject which we have derived from clinical and pathological observation of aphasic cases.

Dr. Broadbent has found that the third frontal convolution, whilst it is connected with the corpus striatum below, and with the opposite hemisphere through the corpus callosum, has more abundant and complicated relations with superficial parts of the same hemisphere than any other convolution in the brain. He says:-"It receives fibres from the convolutions of the island of Reil, from the inframarginal convolutions of the fissure of Sylvius, and from parts posterior to it in the supramarginal convolution, from the second frontal and first frontal gyri, and from the orbital lobule, also from the two great longitudinal commissural systems—the axial and fasciculus uncinatus-by which it will be brought into relation with the convolutions on the convexity of the hemisphere, and on the under surface of the temporo-sphenoidal lobe."

The third frontal seems therefore to be a convolution having those complicated relations with other parts of the brain which we should have a right to expect would exist in the event of its being immediately concerned in the determination of the motor acts of speech; it is proper to add, moreover, that it appears to have similarly complicated relations with other parts on each side of the brain. The functions of the two convolutions are doubtless the same, and in all probability they habitually act in unison by means of their commissural connections (through the corpus callosum), the only difference being that the outgoing or volitional motor stimulus for speech seems, in right-handed individuals at least, to pass off from the convolution of the left side.

But whilst the left hemisphere is more especially concerned in the performance of the voluntary motor acts of speech, it would appear that there are also certain peculiarities of function pertaining more especially to the right hemisphere.

Right Cerebral Hemisphere.—It seems from the observations of Brown-Séquard, and also from those of Mr. Callender, that lesions of the right hemisphere are, other things being equal, (1) more frequently and more rapidly fatal than lesions of the left hemisphere; and also that the hemiplegic symptoms resulting from such lesions are proportionately more severe and more lasting than when they result from similar morbid states in the left hemisphere. This opinion has been arrived at in

part from a careful comparison of cases occurring in the human subject, and in part from a study of the results of experiments upon the lower animals.

- (2) The preceding conclusion seems, moreover, to be strengthened by the fact that disorders of nutrition on the paralysed side of the body are decidedly more frequent with lesions of the right than with those of the left half of the brain. Thus Brown-Séguard has found that rather more than two-thirds of the recorded cases of bed-sore or acute sloughing in cases of hemiplegia, have occurred where the paralysis of the limbs has been on the left side. The same authority also believes that there is a greater frequency of those hæmorrhagic or inflammatory conditions of the lung which appear to be dependent upon the brain disease, when the lesion exists in the right than when it occurs in the left hemisphere. Dr. Hughlings Jackson, moreover, is of opinion that double optic neuritis is more frequently associated with disease of the right than with disease of the left hemisphere.
- (3) Callender and Brown-Séquard have adduced some valuable evidence tending to show that convulsions and tonic spasms of the limbs are very much more frequently met with in association with paralysing lesions in the right than with others in the left hemisphere. And similarly Brown-Séquard

has found that the peculiar spasmodic affection, described under the head of "Conjugated Deviation of the Eyes," occurred in about two-thirds of the recorded cases in association with lesions in the right hemisphere.

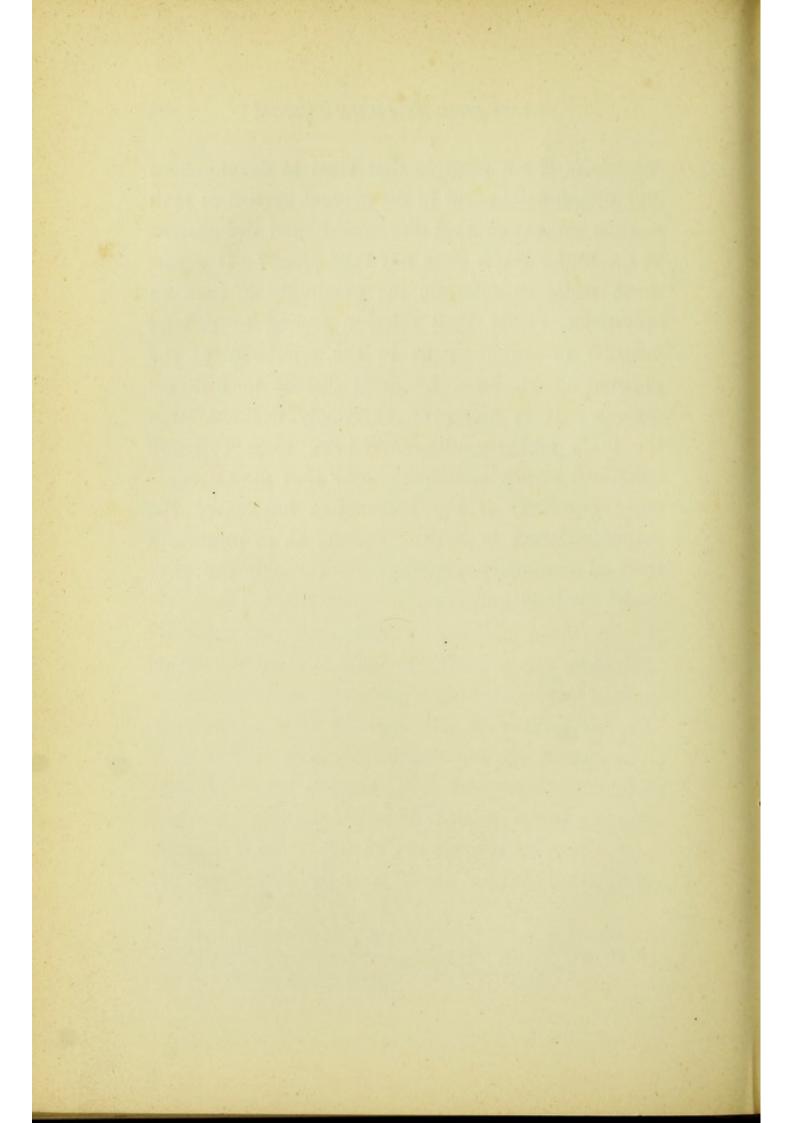
- (4) According to the same authority, again, it appears from a careful analysis of recorded cases that the various forms of hysterical paralysis are far more frequent in the left than in the right limbs. Having analysed 121 cases reported by different observers, Brown-Séquard found that limbs on the left side of the body were paralysed in 97, whilst the right limbs were only affected in 24 instances.
- much more apt than lesions of the left hemisphere to give rise to a paralysis or to convulsions involving the limbs and face on the same side as the brain lesion. I have not hitherto referred to the existence of these anomalous cases, but in the face of records given to us by very competent observers, it cannot be denied that they do occasionally occur. We are unable to explain away such instances by supposing that mistakes have been made, in substituting 'right' for 'left' or vice versa, in the writing out or dictation of post-mortem records—though perhaps some of them may be thus accounted for.

It appears, however, that these very anomalous cases are more apt to be met with in association with lesions in the right than with lesions in the left half of the brain. At present I inform you of this as a mere empirical fact. The occurrence of paralysis or of convulsion on the same side as the brain lesion is with our present state of knowledge quite inexplicable; still more mysterious, therefore, is it when we find one hemisphere apparently more apt than the other to produce such an anomaly.

In reference to this subject generally, it must not be forgotten that the fibres in the outer part of the anterior pyramids of the medulla do not decussate—they descend in the anterior columns of the cord on the same side, and concerning their functions we know little or nothing positively. It seems certain also, that variations in the place and manner of decussation of both motor and sensory impressions exist amongst the lower animals; and it is further generally recognized by physiologists that the motor decussation is less complete in dogs and other mammals than in man—and still less complete in birds.\* But, if the anatomical structure of the cord and medulla varies amongst vertebrate

<sup>\*</sup> These conclusions are based upon the different effects produced by unilateral lesions of the corpus striatum and other parts of the cerebral hemisphere in man as compared with such lower animals.

animals, is it not possible that vices in development may occasionally occur in the nervous system of man to such an extent that the accustomed decussation of the motor tracts does not take place? It seems worth while to entertain the possibility of such an occurrence, rather than allow a feeling of general mistrust to spring up as to the regularity of the phenomena produced by brain disease in different regions. It is, moreover, extremely desirable that the brain and medulla should be most carefully examined in any subsequent case that may occur—more especially if the lesion has implicated the corpus striatum in such a manner as to produce a tract of secondary degeneration through the peduncle, medulla and cord.



# LECTURE VI.

REGIONAL DIAGNOSIS IN BRAIN DISEASE.

- Regional Diagnosis—Difficulty of Subject—Importance of definitely ascertaining the extent of our knowledge concerning—Method adopted.
- Signs of Disease in the Pons Varolii—In its Central Parts—In the Lower Half of Lateral Region—In the Upper Half of Lateral Region—Other characteristics of Disease in the Pons.
- Signs of Lesions in the Crus Cerebri—When in its Upper and Outer Part signs closely resemble those from Injury of Thalamus—Lesions in its Lower and Inner Part very characteristic—Signs of Paralysis of the Third Nerve—Nature of the accompanying Hemiplegia.
- Lesions in or near the Optic Thalamus—Often implicate the Upper Part of Crus Cerebri—Difference between grouping of Symptoms from Injury in this Region and that produced by Injury of Corpus Striatum.
- Hæmorrhage into the Lateral Ventricles—Two sets of Symptoms How they differ from Symptoms due to Lesions in the Pons—Diagnosis from certain Bilateral Lesions.
- Lesions in the Cerebral Hemispheres—Signs of in the Frontal Lobe— Lesions in the Occipital Lobe—Greater Impairment of Intellect— Lesions of the Cortical Grey Matter—Principal Symptoms met with—Different Modes in which they are grouped.

### LECTURE VI.

HAVING pretty fully considered the modes of Causation and the Semeiology of the different kinds of hemiplegia, I began in the last lecture to point out certain differences which are apt to be encountered in the sum-total of symptoms, according as the paralysing lesion occurs in the left or in the right cerebral hemisphere. Following up this part of our subject, I shall now endeavour to indicate the mode in which the signs and symptoms are grouped, as the lesion producing hemiplegia occurs in different parts of the encephalon—that is, I shall endeavour to supply such data as may serve to guide you to a more or less definite 'regional diagnosis' in the cases of brain disease which may subsequently come under your own observation.

## REGIONAL DIAGNOSIS.

This part of our subject is still involved in great obscurity; it can, in fact, at present only be sketched out in a rude and tentative fashion. But in this, as in all other difficult inquiries, it is eminently useful clearly to ascertain the real boundaries of our knowledge. For even a small amount of information firmly based upon trustworthy clinical and pathological observations will, when separated from all entanglements and doubtful questions, afford a positive basis for further observation. We must, in short, know clearly what are the definitely ascertained facts relating to the association of particular groups of symptoms with brain lesions in different localities—we must know in what directions new conclusions appear to be forming and also what old opinions are being modified, before we can hope successfully to widen the boundaries of our knowledge on the subject of regional diagnosis.

In endeavouring to lay before you such positive data as we at present possess, I must necessarily proceed in a simple and comparatively artificial manner. The brain must be mapped out into arbitrary regions; and we must consider separately the effects of a single lesion in each one of these regions—though in actual practice you will often have to do with simultaneous injuries of more than one part, or with lesions situated on the confines between some two of these artificial encephalic departments.

We shall find it most convenient to commence by a consideration of the effects of lesions in different parts of the pons Varolii, and then to pass in review the different combinations of effects produced by injury to parts which successively intervene between this lower centre and the surface of the cerebral hemispheres. Afterwards we may with more advantage study the effects of lesions in the middle peduncles and in the lateral and middle lobes of the cerebellum. Treating the subject in this order, we shall pass gradually from definite or easily comprehensible modes of grouping to others in which the combinations of symptoms are more and more variable or even vague. For we shall thus first consider the effects of lesions in parts whose damage is most apt to reveal itself by 'direct' symptoms or signs (p. 50); and shall gradually pass to the study of the effects of injuries in brain-regions where the signs and symptoms not only belong in increasing proportion to the 'indirect' category, but also become more and more uncertain in their mode of grouping.

Lesions in the Pons Varolii.—Central Parts.— Large lesions here give rise to profound 'apoplectic' symptoms—characterised by deep coma, complete resolution of limbs on both sides, flapping of cheeks during expiration, insensibility of conjunctivæ, and notably contracted pupils. With such lesions in this situation (especially when suddenly produced) death may take place in a few minutes, a few hours, or in a day or two. Where there is a speedily fatal result, this occurs whilst the patient is still in the stage of collapse, with a temperature lower than normal—perhaps as low as 96° F. in the rectum. But where the life of the patient is prolonged for several hours, a day or more, the temperature of both sides of the body steadily rises, till at the time of death it may have attained 109°, or even 110°—a condition of the profoundest coma continuing throughout.

When a central lesion in the pons is slighter in extent, the patient after a time recovers from the first shock of the injury and consciousness is gradually regained. We find, however, a generalised paralysis more or less equally distributed over the two sides of the body; sensibility also may be very notably diminished or perverted in one or more of the limbs. When, in addition to such signs, there is well-marked but irregular paralysis about the face, involving eyelids, mouth, and tongue—and when there is also difficulty in deglutition, associated with a decided impediment in articulation or actual speechlessness not of the aphasic kind — we may be pretty

sure that we have to do with a lesion involving the central parts of the pons Varolii (p. 184). Of course, many sub-varieties of this particular type of paralysis are to be met with, though in each of them we may recognise the characteristic combination of irregular bilateral motor paralysis of the limbs and face, well-marked paralysis or alteration of sensibility in some parts of the body, together with some distinct difficulty in deglutition as well as in articulation.

Lower Half of one Lateral Region.—An injury of this part is characterised by the production of what is called 'alternate hemiplegia,' in which we have an unusually well-marked facial paralysis on the side of the brain lesion, and a more or less complete motor and sensory paralysis of the limbs of the opposite side. Such a hemiplegia may commence with apoplectic symptoms, or there may be an epileptiform mode of onset; whilst in other cases it supervenes more gradually, without either loss of consciousness or convulsions. After the effects of the first shock have disappeared, the temperature of the paralysed limbs is generally found to be about 2° higher than it is on the paralysed side.

Upper Half of one Lateral Region.—Injuries to this part of the brain produce a hemiplegia of the same kind as that last described, with the sole exception that the well-marked paralysis of the face exists on the side opposite to the brain lesion—that is, on the same side of the body as the paralysis of the limbs; for here the fibres of the facial are implicated above their point of decussation in the pons, just as the motor channels for the limbs are implicated above their decussation in the medulla.

But whether the lesion be in the upper or in the lower part of one lateral half of the pons, the facial paralysis is generally very well marked, so as to involve the orbicularis palpebrarum, as well as the orbicularis oris and the straight muscles going to the angle of the mouth. It is also most frequently associated with some distinct difficulties in deglutition and articulation, and there is often a very copious overflow of saliva from the paralysed side of the mouth—partly owing to an increased secretion, and partly to the fact of the paralysis favouring its escape and impeding its deglutition. The degree of impairment of sensibility on the paralysed side of the body is very variable, this symptom being more marked according as the lesion approaches near to or actually involves one side of the ventricular aspect of the pons; and, where it exists, it is apt to be more marked and more durable than in the great majority of cases of hemiplegia due to lesions further away from the base of the brain.\* More rarely a condition of unilateral hyperæsthesia (often limited in its distribution) may occur instead of anæsthesia; and either state may be associated with painful sensations in the limbs or with peculiar subjective sensations of 'coldness,' even when the temperature of the part is actually higher than natural.

The fifth nerve is frequently implicated in these cases of lesion in the lateral region of the pons, so that we may have anæsthesia, hyperæsthesia, painful or anomalous sensations on the corresponding side of the face, and perhaps also a partial unilateral impairment or perversion in the sense of taste. There will moreover be a weakening of the temporal, masseter, and other muscles of mastication on the same side, where the motor division of the fifth is damaged or in any way interfered with.

In addition to the presence of the before-mentioned symptoms in various degrees and in various modes of combination, lesions in the pons are especially apt to be associated with what is commonly known as 'emotional weakness.' There is an undue proneness shown by the patient to burst into tears or to laugh without adequate cause, and often, it may

<sup>\*</sup> The only cases which can compare with these in this respect, are alluded to on p. 142, that is cases where there is a lesion of the 'internal capsule' just outside the optic thalamus.

be, quite inopportunely. The tendency to cry is generally more marked than the tendency to laugh. The comparative frequency of the existence of this emotional weakness in connection with injuries of the pons is, moreover, quite in harmony with what we know concerning its functions as a centre under whose influence the external manifestations of emotional states are regulated. The pons is not itself the centre principally concerned in the genesis of emotional states, as some writers have supposed, though it does seem to contain the nervous mechanism by means of which such states, when actually aroused, reveal themselves by characteristic external signs.

Again, where lesions of the pons cause irritation of parts of the surface of the fourth ventricle we may find sugar in the urine. In other cases there may be polyuria (diabetes insipidus), or albuminuria, when lower portions of the fourth ventricle are implicated—though we here get into the region of the medulla oblongata rather than of the pons.

Whilst suddenly occurring and extensive lesions in any part of the pons always produce an apoplectic attack of a profound character, slighter lesions are not unfrequently ushered in by an epileptiform onset, though in other instances (and especially where the damage occurs slowly) there is neither loss of consciousness nor convulsions. Early rigidity or spasmodic states of the paralysed limbs are also very frequently encountered where we have to do with lesions in this part of the brain. But, in addition to a spasmodic condition of the muscles of one or both limbs on the paralysed side, there may be trismus or rigidity of some of the muscles of the neck—which may or may not be associated with conjugated deviation of the eyes.

Lesions in the Crus Cerebri.—Some lesions in this part of the brain can be diagnosed with the greatest certainty; though in other cases the diagnosis can only be made in a provisional and more doubtful manner. These different degrees of certainty depend altogether upon the precise site and extent of the lesion. Thus, should the inner and inferior part of the crus near the pons be injured, or should there be a larger lesion implicating this as well as contiguous parts of the crus, the third nerve on the same side becomes paralysed simultaneously with the establishment of a hemiplegic condition in the opposite half of the body: the diagnosis should then be easy. If, on the contrary, the lesion implicates only the upper and outer part of the crus (that is, the part next the cerebral hemisphere), the diagnosis becomes much more difficult. There is

no distinctive sign of a lesion in this situation, and the grouping of symptoms approximates very closely to that met with in lesions of the optic thalamus. It so happens, also, that lesions of the thalamus not unfrequently extend downwards into the upper part of the crus cerebri (p. 43).

Under the present heading, therefore, I shall only dwell upon the combination of symptoms produced by lesions in the lower and inner part of the crus—a combination whose significance ought to be readily recognised, and which was described with great fidelity by Dr. Hermann Weber about twelve years ago.

The condition induced is a peculiar form of 'alternate paralysis.' The third nerve is paralysed on the side of the brain lesion, the existence of this condition being shown by ptosis or dropping of the eyelid on the same side; by dilatation and sluggishness of the pupil; by external squint; and by an impossibility of moving the eyeball except slightly in two directions—viz., slightly further outwards, owing to forced contraction of the external rectus; and a little around its own axis in one direction (from outwards upwards), owing to forced contraction of the superior oblique muscle. All the muscles of the eyeball, in short, are paralysed, except the external rectus and the superior oblique which are

supplied by the sixth and fourth nerves respectively. There is consequently more or less diplopia when the eyelid is raised or when the ptosis is only partial-at least, till the patient learns to use one eye only. The co-existing hemiplegia on the opposite side of the body approximates in its general characters to that produced by a lesion in the upper part of one lateral half of the pons Varolii. Thus the tongue generally deviates distinctly to the paralysed side, and the facial paralysis about the mouth is well marked. There is often some difficulty in articulation, or perhaps only a mere thickness of speech for a time. The power of deglutition may not be interfered with. Sensibility is mostly very decidedly impaired on the paralysed side, the impairment continuing for some time, and being more marked in the limbs than in the trunk. The temperature of the paralysed limbs, moreover, may be as much as two degrees higher than that of the non-paralysed side.

Lesions in or just outside the Optic Thalamus.—Injuries in this site, as I have already stated, not unfrequently involve at the same time the upper part of the crus cerebri—especially where we have to do with hæmorrhage into the substance of the thalamus. It will be found, however, most advantageous to compare the effects of injuries to the thalamus with those produced by Lesions in or just outside the Corpus Striatum. The grouping of symptoms occasioned by lesions in this latter situation have already been carefully described (p. 91), since they were taken as typical of the hemiplegic state in general. It remains for me now, therefore, merely to point out the principal differences, or departures from such a mode of grouping, observable with lesions in or just outside the optic thalamus. Our knowledge is, unfortunately, still very incomplete in this direction, although some distinctive features are known to exist.

In the first place, it seems that the motor paralysis occasioned by injuries in or about the thalamus is generally less pronounced than that which would have been occasioned by lesions of equal extent in or about the corpus striatum, and in some cases it may be even almost entirely absent. The paralysis of the face especially is said by Gintrac to be less distinctly marked, though this is a point about which I have not as yet been able to come to any definite conclusion—since the evidence has appeared to me conflicting.

It does seem, however, to be undoubtedly true that early tonic and clonic spasms in the paralysed limbs or about the face and neck are especially frequent with lesions of the thalamus. I have often noted this myself, and we had a very good illustration of it in the case of a man lately in the wards, where the presence of this and other symptoms led to a regional diagnosis, which was subsequently completely verified by post-mortem examination. Spasms of this kind have, in fact, been noted in nearly three-fourths of the recorded cases of lesions in or just outside the thalamus, whilst they are quite exceptional where we have to do with lesions of the corpus striatum.

Though there seems to be no very distinct difference as to the degree of impairment of sensibility in lesions of the thalamus and corpus striatum respectively, the impairment is, perhaps, slightly more marked in cases of lesions of the thalamus.\*

Former physiological notions concerning the functions of the thalamus are, however, by no means borne out by what we know of the effects produced by disease in this part.

The difference in temperature between the limbs on the paralysed and on the sound side of the body is generally more marked in lesions of the thalamus than in those of the striate body, and in

<sup>\*</sup> Lesions of parts of the peduncular expansion just outside the thalamus are, as before stated (p. 142), especially apt to be associated with one of the most permanent forms of hemianæsthesia.

explanation of this I may hazard the following conjecture:—The pons containing some of the principal regulative centres for vaso-motor nerves, the closer proximity of the thalamus would probably entail (in cases of lesion of this body) a more powerful reflex inhibitory influence upon the vasomotor centre on the same side than would be occasioned by lesions in or about the more distant corpus striatum. Thus, whilst in the latter class of cases the temperature of the limbs on the paralysed side is rarely more than one degree higher than that of the limbs on the sound side (and even this difference soon diminishes), in cases of lesions in or about the thalamus the difference may be one and a half or even two degrees, and it persists for a much longer time-often for many weeks or months.

Again, aphasic difficulties in speech, which are so common when we have to do with lesions in or just outside the left corpus striatum, are not, as a rule, met with where we have similar lesions in or about the left thalamus. It not unfrequently happens, however, that these two bodies are damaged simultaneously by some single lesion; and then we should have the combination of aphasic symptoms, together with early rigidity and other signs more especially indicative of a lesion in the thalamus.

Nothing more definite, I believe, can at present be said concerning the differential diagnosis of lesions in these contiguous though very different brain regions.

We have now to consider certain complications occasionally attendant upon lesions in the brain regions last referred to—these being of sufficient importance to deserve a separate study.

Hæmorrhage into the Corpus Striatum or Thalamus, followed by Effusion of Blood into the Ventricles .-- An accident of this kind is often divided into distinct stages. Thus we may have at first to do with an ordinary hemiplegic condition, commencing either by an apoplectiform, an epileptiform, or a simple mode of onset. But, after an interval of varying duration, a fresh hæmorrhage occurs, and the blood then poured out may tear its way through the brain-substance into the lateral ventricles, so that a condition of profound coma with stertor and general paralysis quickly supervenes. In other cases there is no preliminary attack of any kind. A large hæmorrhage occurs suddenly, and the effused blood at once lacerates the brain-substance and opens up a channel through which it is copiously poured into the lateral ventricles. In such a case we should have from the

first an apoplectic attack of the most marked kind, characterised by profound coma, general paralysis of limbs, and dilated pupils.

In each set of cases the laceration of brain-tissue and effusion of blood into the lateral ventricle is associated with a decided lowering of the temperature of the body. The rectal temperature sinks to 96° or thereabouts, and remains at this point for an hour or two, especially where the bleeding into the ventricles slowly continues. Should a fatal result not speedily occur, we find that the primary depression of temperature is followed after a time by a rapid rise, which slowly continues, in cases about to prove fatal, till the death of the patient. Such fatal result very often takes place before the expiration of three days from the onset of the attack, though occasionally it does not occur till a later period.

In these cases of ventricular hæmorrhage we very frequently indeed meet with tonic spasms of one, two, or more limbs; or tonic may alternate with clonic spasms in the same parts. In other instances we have a condition of rigidity in the limbs of one side, combined with clonic spasms in one or both extremities of the opposite side.

In this class of cases 'conjugated deviation' of the eyes is sometimes present from the firstpointing to the side of the brain on which the hæmorrhage with laceration has occurred. And if the coma is not too profound, the conclusion about to be drawn from this indication may be confirmed by the discovery of some slight signs of sensibility on the side of the body towards which the eyes are turned. Touching the conjunctiva on this side, for instance, may produce some closure of the eyelids, whilst irritation of the conjunctiva on the other side (opposite to that of the brain lesion) gives rise to no corresponding reflex movements.

The grouping of symptoms just described is tolerably distinctive of hæmorrhage into the lateral ventricles. When such symptoms are present we should in the majority of cases be justified in arriving at this diagnosis. More rarely, however, we find these combinations of symptoms pretty closely imitated by the results of lesions in the pons Varolii, when we may have to do with injuries, either (a) small at first and subsequently increasing, or (b) large from the first—where the symptoms resemble those produced by a copious effusion of blood bursting at once into either of the lateral ventricles.

In the former class of cases we might be guided to a correct diagnosis if the hemiplegia which had previously existed was of such a nature as to make it referable to a lesion of the pons. And it should be borne in mind that when the condition of coma with general paralysis has become established as a result of an extensive lesion in the pons Varolii (whether this lesion be primary or secondary), the condition itself is very apt to be associated with contracted and motionless pupils, as in opium poisoning, whereas in cases of ventricular hamorrhage the pupils are usually dilated. The diagnostic indications afforded by the condition of the pupils in brain disease are only too often vague and indefinite, though the relations above stated, from their comparative uniformity, afford important exceptions to this general rule.

Tonic spasms are also more frequently absent in severe central lesions of the pons Varolii than where we have to do with ventricular hæmorrhage. In those very severe lesions of the pons which in their symptomatology otherwise closely resemble ventricular hæmorrhages, muscular spasms are usually absent; we find instead a complete resolution of all the limbs, and of the trunk muscles.

Other cases of brain disease occasionally occur in which the resemblance of the symptoms to those of secondary ventricular hæmorrhage may be extremely close. I allude to instances where a hemiplegic condition becomes complicated by the occurrence of a fresh lesion (either softening or hæmorrhage)

in the previously sound hemisphere. Here we have the establishment of paralysis on both sides of the body, and generally a condition of profound unconsciousness. I have seen such a case almost exactly simulate one of secondary ventricular hæmorrhage; and in this instance the only indication of its real nature was the simultaneous occurrence of conjugated deviation of the eyes towards the previously sound side of the brain.

The power of diagnosing between these conditions is occasionally a matter of some importance from the point of view of prognosis.

Lesions in the Anterior, Middle, and Posterior Parts of the Cerebral Hemispheres.—It is a rule to which, as you know, there are only very few exceptions, that a lesion in either hemisphere of the brain, if of sufficient extent, induces a paralysis of the limbs on the opposite side of the body. Whilst we may, therefore, in cases of hemiplegia, at once arrive at a conclusion as to the side of the brain injured, it is by no means easy for us, in the great majority of cases, to determine in what precise part of the hemisphere the lesion is situated. In the records of cases of brain disease we mostly find the site of lesion roughly described—it is spoken of as being in the

anterior, the middle, or the posterior lobe; and anyone who examines such records carefully will soon find that the 'middle lobe' is cited much more frequently as the site of lesion than either of the other lobes. How is this apparent selection to be explained? There can be little doubt, I think, that it is principally attributable to the fact that a very much larger bulk of brain-tissue is included under the designation 'middle' lobe than enters into the formation either of the 'anterior' or of the 'posterior' lobe. What is roughly spoken of as the middle lobe is a mass of brain-tissue having both greater depth and breadth than the portions of the hemisphere in front or behind, and it includes what in more scientific phraseology we should term the 'parietal' and the 'temporosphenoidal' lobes. In addition to this greater mass of the so-called middle lobe affording a greater chance for the occurrence of accidents therein, you must also recollect that the opto-striate arteries frequently give rise by their rupture to lesions situated, in the main, in this great department of the cerebrum (p. 42).

To discriminate during life between lesions occupying these different situations is at present often extremely difficult, if not impossible, though doubtless, as time goes on and more careful attention is given to this subject by a numerous band of workers, our knowledge will increase. Now, however, a few hints only can be given towards the solution of such difficult problems in cerebral diagnosis.

Anterior or Frontal Lobe.—Large lesions in the anterior lobe may either actually implicate the olfactory bulb on one side, or may interfere with its functional activity by bringing about an undue pressure upon the part of the brain in which it is situated. Thus such lesions are apt to be associated with the very significant sign of loss or decided impairment in the sense of smell on the side of the body on which the brain lesion occurs, and, therefore, on the side opposite to that in which the limb-paralysis exists. Such a sign may be encountered in association with hæmorrhage into the anterior lobe, or with softening of the same part caused by embolism or thrombosis of the anterior cerebral artery-by some of the branches of which the olfactory bulb is fed.

Again, lesions of the posterior part of the frontal lobe on the left side, where we have to do with right-sided hemiplegia, are not unfrequently indicated by the coexistence of a typical aphasic condition.

On the other hand, there is no one sign or set

of signs, so far as I know, which would point at all conclusively to an acute lesion in the form of hæmorrhage or softening in the Parietal or in the Temporo-Sphenoidal Lobe. Almost the same kind of statement has to be made in respect to lesions in the Occipital Lobe. I say 'almost' because there is certainly one exception to this rule. Owing to the fact that the corpora quadrigemina are fed by arterial twigs which come off on each side from the posterior cerebral artery, it not unfrequently happens that a softening of the posterior lobe of the brain, due to occlusion of the corresponding posterior cerebral artery, gives rise to some loss of sight in the eye of the opposite side as well as to paralysis of the opposite limbs. The unilateral loss or impairment of sight and the hemiplegia being different results of the same cause (the cutting off of blood-supply from the territory of the posterior cerebral artery), it is important to note that the two become established simultaneously. Should the impairment of sight not be extreme, its existence may escape the notice of the patient. It is a sign, therefore, for which you should inquire whenever a doubtful case presents itself.

After a time we may perhaps learn something more definite than we at present know concerning the degree and kind of impairment in the mental powers due to lesions in the anterior, middle, and posterior parts of the brain respectively. Now there is nothing certain to be said on this subject. I will merely add that various reasons led me, some years ago, to the conclusion that the posterior lobes of the brain had more to do with higher intellectual functions than the anterior lobes—a conclusion which, however contrary it may be to generally received opinions, has since been strengthened by observations made independently in different directions and by different persons. It seems to agree, moreover, with clinical and pathological evidence. Dr. Hughlings Jackson and other authorities on the subject of braindisease agree with me in thinking that mental impairment or derangement is apt to be more especially marked where we have to do with injuries of the posterior lobe of the brain-that is, such signs are prone to be more marked than with lesions of the anterior lobe of the brain, notwithstanding the popular notion as to the decidedly greater importance of this part in respect to intellectual functions.

Dr. Hughlings Jackson even goes so far as to consider that injuries of the right posterior lobe produce more marked impairments in intellectual function than similar injuries of the left posterior lobe. More extended observations, however, seem

needed before we can safely arrive at a positive conclusion on this difficult subject.

Lesions limited to the Cortical Grey Matter, or causing Pressure upon this substance.—It is only to be expected that the precise combinations of symptoms produced by lesions of the cortical substance should differ immensely, according to the nature and also according to the extent of the injury in different cases. The groupings of symptoms due to lesions in this situation are indeed, as a matter of fact, most varied in their character. I cannot attempt to describe one tithe of the different modes of combination which may present themselves, and shall therefore merely proceed to indicate the kind of symptoms most frequently present, as well as those most frequently absent where we have to do with lesions of the cortex of the hemispheres.

As in dealing with other parts of this subject, the statements I am about to make will be based partly upon my own observations, and partly upon a careful study of a large number of cases recorded by other observers, both in this country and abroad. The published cases to which I have referred have been recorded by their respective observers under the head of cortical hæmorrhage or cortical softening, of meningo-encephalitis, and of submeningeal hæmorrhage.

Cephalalgia is often present to a notable degree where the meninges are especially implicated, though it is much more frequently absent when the lesion is limited to the cortical substance itself.

Loss of consciousness, more or less prolonged, may mark the onset of a superficial affection of the brain, though just as frequently incoherence or delirium is manifested as one of the initial symptoms. At other times we have to do merely with obtuseness of intellect, or with more or less marked stupor.

Convulsions may usher in an acute affection of the cortical grey matter, though this does not seem to be so frequently the case with such affections of the cortex as the recent experiments by Hitzig and Ferrier on lower animals might have led us to expect. More limited tonic and clonic spasms, without loss of consciousness, are, however, frequent accompaniments of such affections, in some part of their course. And actual convulsive attacks also occur with great frequency in a class of cases with which we are not at present specially concerned—namely, in association with new growths involving the cortex.

No definite paralysis exists in some cases; there may be merely great general weakness, with a slow vacillating mode of walking or actual inability to stand. In other instances there are signs of partial hemiplegia—that is, we may have more or less

complete paralysis of one arm, together with a very slight lowering of the angle of the mouth on the same side, though without any very appreciable weakening of the leg.

Should the lesion occupy the third left frontal convolution or parts immediately adjacent, aphasia is met with, either alone or in association with partial hemiplegia. With injuries of the grey matter in other parts of the hemispheres, the power of speech and of intellectual expression generally may be variously interfered with. We may meet with well-marked amnesic defects, or there may be merely a slow and laboured utterance—with or without some amount of mental incoherence.

In other instances we have defects of speech belonging to some one of these categories in association with a much more complete hemiplegic condition—that is to say, the paralysis in the arm and in the leg may be well marked, and present the usual peculiarities, though paralysis of the face is either absent or so slightly marked as to be scarcely appreciable. This last is a sign which occasionally has great significance.

The loss of sensibility, again, in these cases of hemiplegia due to superficial lesions, is generally very slight and often scarcely appreciable even within a few days from the date of onset of the malady. In

some instances, instead of a loss of sensibility, there may be even slight hyperæsthesia in parts of the paralysed limbs.

Another point worthy of note is, that the difference in temperature between the paralysed and the non-paralysed side of the body is rarely very marked seldom exceeding 1° F., and tending soon to disappear.

If we bear in mind, therefore, the peculiarities I have mentioned with regard to the distribution of the motor paralysis, its frequent association with mental changes, and with tonic or clonic spasms in the limbs, the absence of distinct loss of sensibility, or of any notable elevation of temperature on the paralysed side where we have to do with lesions of the surface of the brain, we get an assemblage of signs and symptoms which frequently prove very characteristic of the hemiplegic condition thus induced. It is not to any one of these signs singly, but to a combination of them, that we must look for guidance. Thus, if we find a patient showing some amount of mental incoherence or delirium suffering from a partial hemiplegia, with little affection of the leg or face, and if at the same time there are muscular twitchings in one or more limbs, or rigidity about the neck or other parts of the body, our regional diagnosis

becomes comparatively easy. Such a combination of symptoms would point strongly to the existence of a lesion involving the surface of the brain on the side opposite to that on which the motor paralysis exists.

Looking upon such a case as I have just referred to as one of medium severity, many other groups of symptoms of a slighter kind may present themselves, as well as combinations of a more serious nature. In the slighter cases we may have no actual paralysis, but rather tonic or clonic spasms in association with symptoms indicative of slight mental disturbance. And, although we seem advancing on the road towards a more definite localisation in some of these cases, the efforts which have been made must be considered to be still merely tentative.

In instances of more severe superficial lesions—cases due to wide-spread embolisms, or to very copious arachnoid hæmorrhages—a profound apoplectic condition is produced in which nearly all differential characters are for the time lost, and during the continuance of which the patient often rapidly sinks.

## LECTURE VII.

THE CEREBELLUM AND ITS FUNCTIONS.—SYMPTOMA-TOLOGY OF DISEASE IN ITS DIFFERENT PARTS. —PATHOLOGICAL DIAGNOSIS IN APOPLEXY AND HEMIPLEGIA.

- The Cerebellum.—Uncertainty about Functions of.—Is more concerned with Motility than with Mind.—Its Anatomical Connections imply Community of Action with Cerebrum and Cord.—Its Action Subordinate to that of Cerebrum, and also Complex. This, the first cause of Difficulty in Discovering the Symptoms produced by its Injury.—Second and Third Causes due to its Situation and Anatomical Connections.—Great Diversity in Symptoms, Direct and Indirect.—Hemiplegia on opposite side an Indirect Symptom.—Direct Motor Signs should show themselves on same side.
  - Symptomatology in Disease of the Cerebellum.—Clinical Signs of Lesions in its Lateral Lobes.—Great Variations in.—Lesions in One of its Middle Peduncles.—Rotation with characteristic Ocular Deviations.—Lesions in the Median Lobe.—Many Indirect Symptoms.—Excitation of Genital Organs.—Blindness.—Ventricular Dropsy.
- Pathological Diagnosis.—Difficulties of.—Considerations on which to depend.—Diagnosis of the Cause of an Apoplectic State.—Blows upon the Head.—Narcotic Poisoning.—Intoxication.—Uræmic Coma.—Epileptic Coma.—Its Varieties and Characteristics.—Differential Diagnosis of Embolism, Thrombosis and Hæmorrhage.—Indications supplied by Mode of Onset of Paralysis.—By Nature of Attack.—Multiple Embolisms and Thrombosis in Basilar Artery may simulate grave Hæmorrhage.—Evidence furnished by Subsequent Course of Disease.—Variations in Temperature in Hæmorrhage and in Softening respectively.—Further Work needed in this Direction.—Miscellaneous Indications.

## LECTURE VII.

GENTLEMEN,—It remains for me now to speak to you concerning the diagnosis of lesions in the Cerebellum. This is an extremely difficult and obscure division of our subject, partly because the functions of the cerebellum are so very imperfectly known, and partly because the situation and anatomical connections of this organ almost necessarily entail a more than usual amount of functional disturbance of other important and related parts when lesions of its substance are met with.

As you must be all aware, the exact nature of the functions of the cerebellum still remains very uncertain. This is one of those problems concerning the correct solution of which there is no unanimity of opinion amongst physiologists. The uncertainty of our knowledge in this direction is due to several causes, to some of which I will very briefly call your attention, since in so doing I shall be also explaining the nature of the disturbing influences

which tend to obscure or complicate our clinical knowledge of cerebellar disease in its various forms. Before doing this, however, it may be premised that the knowledge we at present possess does enable us to come to one very important conclusion with respect to the functions of the cerebellum—it enables us to say that this organ has no independent function either in the province of mind or in the province of motility. And we may, perhaps, safely affirm still further, that the cerebellum is much more intimately concerned with the production of bodily movements than with the evolution of mental phenomena.

The anatomical distinctness of the cerebellum from the larger brain and other parts of the nervous system is more apparent than real. Do not its roots (under the name of 'peduncles') penetrate into the cerebral hemispheres, into the pons, and into the spinal cord through the medium of the medulla oblongata? The cerebellum is, as it were, an outgrowth from these several parts, and just as there is an anatomical interblending between it and them, so, as we may fairly presume, must there be a community of action in the bringing about of certain physiological results.

That there is an habitual community of action between the cerebellum and the spinal cord is, I believe, doubted by none, and the fact that an

intimate functional relationship exists between the cerebrum and the cerebellum is shown by the circumstance that atrophy of one cerebral hemisphere entails a corresponding atrophy of the opposite half of the cerebellum. The subordinate or supplementary nature of the cerebellar function, however, in this latter relation seems equally well shown by the fact that atrophy of one side of the cerebellum (when it occurs as the primary event) does not entail any appreciable wasting in the opposite half of the cerebrum. What other conclusion can be drawn? If the cutting off of certain cerebral stimuli leads to a wasting of the opposite half of the cerebellum, this would seem to show that each half of the cerebellum is naturally called into activity in response to or conjointly with the opposite cerebral hemisphere. Whilst conversely, if atrophy of one half of the cerebellum does not entail a relative diminution in the opposite cerebral hemisphere, this would go to show that the cerebral hemispheres do not act in response to cerebellar stimuli, since their nutrition does not suffer when such stimuli are certainly absent. The action of the cerebrum is therefore shown to be primary, whilst that of the cerebellum is secondary or subordinate in the performance of those functions in which they are both concerned.

If, however, the cerebellum acts as an abettor of the cerebrum, and the two together act upon or through the spinal cord, we ought to find that injuries to the cerebellum—in the absence of all disturbing causes—display themselves not so much by the lack of certain powers or faculties as by some diminution in the perfection with which they are exercised. But a partial effect of this kind may easily escape detection when slightly marked, just as the precise amount of the defect in any given case may be very difficult to estimate.

Here, then, is cause number one why the functions of the cerebellum should have so long remained wrapt in obscurity. And it may be easily imagined that the difficulty just referred to would be increased rather than diminished if this subordinate function of the cerebellum should prove to be twofold rather than simple in its nature. I must confess, however, to the belief that its functions are of a twofold nature.\*

<sup>\*</sup> Although I cannot now enter upon the grounds of my belief, and merely make mention of it incidentally, I may state that I regard the cerebellum as in part a well-spring of motor force, and as in part an organ for the discharge of certain of the functions hitherto supposed to depend upon the so-called 'muscular sense'—though in respect to these latter attributes I maintain (British Medical Journal, May 1869) the somewhat novel doctrine that the different impressions upon which they depend lie altogether outside the sphere of consciousness. By the combined operation of both sets of functions, afferent and efferent, the cerebellum—acting in conjunction with the cerebrum and with lower

A second reason, however, why the functions of the cerebellum should have so long remained in obscurity is to be found in the fact that its situation and connections are very unfavourable for the elucidation of its functions by means of experiments upon the lower animals. The loss of blood and shock from the operation are mostly severe before the cerebellum can be uncovered. And when to this disturbing cause we add the fact that no attempt to remove the organ can be made without irritating most excitable and important parts of the nervous system to which it is adjacent, some idea may be formed of the difficulties besetting this line of investigation. The animals experimented upon have often died before they had had time to recover from the shock of the operation, or at all events before the effects of irritation of adjacent parts had subsided. No wonder, therefore, at the difficulty experienced by different observers in arriving at any uniform conclusions from evidence derived from such experiments.

A third reason for the backward state of our knowledge concerning the functions of the cerebellum is to be found in the fact that the experiments naturally made by disease of this organ in man also speak to us in a confused manner, owing to a similar contem-

centres in the pons and medulla—comes to be largely concerned with the co-ordination of muscular movements both voluntary and automatic.

poraneous interference with the functions of other anatomically related parts. This again is due to the position and anatomical relations of the cerebellum. Shut in as it is, within the narrow limits formed by the comparatively unyielding tentorium and the occipital bone, in company with the pons and medulla, injuries of the cerebellum are especially apt to compress or irritate one or other of these parts—to say nothing of additional disturbing influences which may be exerted in the cerebral hemispheres through the fibres of the superior cerebellar peduncles. Another cause of complication is, moreover, apt to present itself in some cases. Alterations in the bulk of the cerebellum may occasion more or less impediment to the proper emptying of the venæ magnæ Galeni into the straight sinus, and may thus disturb the nutrition as well as the functional activity of those important central regions of the brain whose venous outflow takes this direction.

The different modes of operation of these disturbing influences are, of course, most various in individual cases of cerebellar disease, owing to the different causes of complication which respectively come into operation, and to variations in their relative activity. Hence it is that we have such great diversity in the symptoms present in different instances of disease of the cerebellum; and hence it is

that we are unable to say at all positively which of the symptoms apt to present themselves are referable directly to destruction or to irritation of the cerebellar substance, and which to secondary or indirect effects of those injuries upon adjacent or related parts. We cannot positively decide this point even with reference to the hemiplegia which so frequently occurs in association with lesions in one lateral lobe of the cerebellum. But although some uncertainty still exists, the balance of evidence seems to point strongly to the conclusion that the more or less complete paralysis of the opposite side of the body is an 'indirect' effect of injury to one lateral lobe of the cerebellum —an effect really induced by the pressure which such lesion occasions upon the corresponding side of the medulla oblongata.

We seem almost irresistibly led to this conclusion when we find the hemiplegia, if present, habitually occurring on the side of the body opposite to that on which the pressure of the medulla and pons is presumed to exist, though we know that the functional relations of each lobe of the cerebellum must be chiefly with the half of the cord and the limbs on the corresponding side of the body.\* This latter relation is obvious from the fact that atrophy of one hemisphere of the cerebrum leads to atrophy of the

<sup>\*</sup> See note, p. 258.

opposite lateral lobe of the cerebellum, and, as I have previously told you, also to atrophy of the opposite half of the spinal cord. We are entitled to conclude, therefore, that all direct effects resulting from lesions in one lateral lobe of the cerebellum would (so far as they were concerned with motility) manifest themselves principally upon the side of the body corresponding with the lesion. And this is a conclusion of some importance.

Concerning 'functional' diseases of the cerebellum we have no certain knowledge; therefore what I have to say will refer to the grouping of signs and symptoms produced by hæmorrhage, or by occlusion of vessels leading to softening of some portion of the organ. We must consider the effects of such injuries separately, according as they implicate one or other of the lateral lobes or middle peduncles, or the middle lobe of the cerebellum. As I have before insisted, we, as practitioners of medicine, have to study the mere grouping of symptoms produced by such lesions, almost irrespective of questions as to the physiological genesis of the several symptoms. We approach these problems of regional diagnosis from the point of view of the physician or clinical observer, and not primarily from the point of view of the physiologist.

Lateral Lobe of the Cerebellum.-Lesions may occur in this region with or without loss of consciousness, according as they are sudden and extensive, or the reverse. Severe pain in the occiput, or it may be in the frontal region, is often complained of, mostly paroxysmal in character; and vomiting occurs perhaps rather more frequently than with injuries in other parts of the brain. Paralysis of the opposite arm and leg is very often met with, though it is not usually accompanied by much diminution of sensibility. The paralysis is apt to be more marked in the leg than in the arm, and it is also generally absent from the face. The condition differs therefore in both these respects from the more ordinary forms of hemiplegia; it differs also from the kind of paralysis produced by superficial lesions of the cerebral hemispheres, where, though paralysis of the face may be absent, the loss of power usually falls more upon the arm than upon the leg.

There may or may not be some early rigidity in the paralysed limbs; perhaps also some trismus, spasms about the face or of the ocular muscles, or marked rigidity of the neck. No definite observations have, I believe, as yet been made, in cases of this kind, as to the relative temperature of the two sides of the body. Speech is generally not affected, and the movements of the tongue are mostly unimpeded.

In the majority of cases also deglutition is not interfered with; but should there be well-marked pressure upon the medulla there may be dysphagia as well as difficulty in articulation.

Blindness may supervene, especially where the superior peduncles are affected by the lesion—these parts being, as you will recollect, in close topographical if not functional relation with the corpora quadrigemina, or visual centres. In the course of the affection there is usually no delirium or mental impairment. At the most there may be some intellectual slowness or dulness, or perhaps more or less drowsiness—though of course coma may supervene towards the close in fatal cases, owing to an extension of the lesion or to some independent mischief. The pulse and respiration are not affected in any uniform manner.

At times, however, the symptoms resulting from lesions in one lateral lobe of the cerebellum are still less definite. There may be no trace of a hemiplegic condition, but rather a general muscular weakness of a progressive character associated with a peculiarly unsteady and vacillating gait (titubation). This weakness is mostly more marked in the legs than in the arms. Sometimes the amount of general weakness and prostration has been extreme, so that the patient has

been quite unable to stand or even to raise himself in bed.

Middle Peduncle of the Cerebellum. - As I informed you in an early lecture, hæmorrhages occasionally occur in this situation, though such an event is very rare. In one instance recorded by M. Nonat the situation of the lesion was correctly diagnosed during life owing to the similarity between the condition of the patient and that of animals in whom the middle cerebellar peduncle has been cut. Such animals almost invariably roll round their longitudinal axis in one uniform direction-namely, from the sound towards the injured side of the body-and show at the same time a remarkable divergence of the eyes. The eye on the injured side is directed downwards and inwards, whilst that of the sound side is turned as strongly upwards and outwards. They may be perfectly motionless in these directions, or may exhibit a certain amount of nystagmus.

In M. Nonat's case an effusion of blood was after death found in the *right* middle peduncle. The patient presented the deviation of the eyes above described, and though in an apoplectic condition, she lay on her right side with the head strongly turned in the same direction—showing a strong tendency at all

events towards rotation. Unfortunately the published details of this case are very scanty.

Similar signs may be met with where the root of the peduncle is implicated in the substance of the lateral lobe of the cerebellum. And in these cases generally if any paralysis is produced, you should look for it on the same and not on the opposite side of the body.\* As I have already mentioned, the lateral lobe of the cerebellum may be considered to be functionally related to the half of the spinal cord on the same side, and the phenomena of rotation produced by section of the middle cerebellar peduncle are, according to Vulpian, best explained by the supposition that there is a cutting off of some motor stimulus from the muscles of the side of the body corresponding with the lesion, and consequently an unbalanced action of those of the uninjured side.

Median Lobe of the Cerebellum.—When a lesion exists in this part of the cerebellum, there is also great latitude with regard to the symptoms produced—the differences encountered being due for the most part to the extent and suddenness

<sup>\*</sup> It is generally admitted that the middle cerebellar peduncles decussate in the pons varolii. So that the fibres of the right side (for instance) penetrate into the left half of the pons and there come into relation with motor fibres. Such motor fibres would be those influencing the right half of the body—above their point of decussation.

of the injury; though they are also in some degree attributable to the variable amount and variable incidence of pressure upon parts of the subjacent medulla and pons. Thus with a large hæmorrhage in this situation causing marked pressure upon such important parts we may have sudden and distinct apoplectiform symptoms, terminating in speedy death. More frequently, however, the lesions are less severe and the effects altogether slighter. The grouping of symptoms is, in fact, generally very indefinite.

Signs of hemiplegia are more frequently absent than when we have to do with lesions in the lateral It so happens, also, that in almost all the cases of disease of the cerebellum in which excitation of the genital functions has been noted the lesion has been situated in its median lobe. Symptoms of this type have, indeed, been observed in about one-third of the recorded cases of disease of the median lobe of the cerebellum. In both sexes there has appeared to be an increase in sexual desires, and in male patients there have been frequent erections, with or without seminal emissions. Such symptoms in connection with lesions of this part have all the more significance because they do not present themselves where only the lateral lobes of the cerebellum are involved. With the limitation

thus indicated, therefore, there would appear to be some foundation for the old phrenological doctrine as to the function of the cerebellum.

The sense of sight is very apt to be affected by a large lesion in the middle lobe, owing to the proximity of the superior cerebellar peduncles, and the anatomical relations of these to the corpora quadrigemina.

Tumours in this situation are more likely than hæmorrhage to lead to ventricular dropsy and perhaps a certain amount of central softening, by causing some impediment to the return of blood from the veins of Galen into the straight sinus. Where such effects are induced, the signs more strictly referable to the cerebellar affection may be masked by vague and variable combinations of symptoms, principally due to the ventricular dropsy and impaired nutrition of the central regions of the brain.

## PATHOLOGICAL DIAGNOSIS.

We turn now, Gentlemen, to another aspect of the problems of diagnosis. We have to inquire into the cause or nature of the change in brain-tissue, giving rise to one or other of the various sets of symptoms previously described. The question is no longer as

to the region of brain implicated, but about the considerations which should guide us in coming to a trustworthy conclusion as to the nature of the change in a given brain-region. Have we to do with hæmorrhage, with thrombosis, with embolism, or with some slighter functional derangement in the particular region presumed to be affected? These are the pathological states which, as you will recollect, are the common causes of the more suddenly initiated cases of hemiplegia - to which our attention has been confined. We for the present therefore dismiss from consideration those other causes of the hemiplegic state which usually produce their effects more slowly - such as indurating overgrowths, tumours, abscesses, and certain sequences of external injuries to the head in the form of severe unilateral meningitis.

Whatever difficulties beset our path in attempting to discover rules for a correct regional diagnosis—and they are, as you are now aware, only too numerous—we do not find them diminish when we strive to formulate rules for the discovery of the nature of the change which has occurred in brain-tissue.

In this inquiry no certain or absolutely distinctive signs exist. We have always to do with probabilities more or less remote from certainty. Sometimes, it is true, we are able to be almost sure about our pathological diagnosis; though only too often it is quite

impossible for us to come to a decision—the probabilities in favour of hæmorrhage and those in favour of softening being so evenly balanced. Those whose experience with this class of diseases is limited are often much disappointed at this. They expect the point to be readily settled, and very frequently are not slow to come to conclusions on such a subject for But the problem of the differential themselves. diagnosis between cerebral hæmorrhage and cerebral softening is regarded in a very different light by those physicians who have had the widest experience with this class of affections. Trousseau, for instance, spoke of the question as "one of the most difficult problems in pathology," and others, whose opinions carry great weight, have expressed themselves in much the same terms.

As a general rule, it may safely be said that, in attempting to come to a definite pathological diagnosis, we must be guided by considerations of the age and general state of health of our patient, of the condition of his heart and larger arteries, by the presence or absence of prodromata, by the actual mode of onset of the attack, and by its progress, as regards general symptoms, during the first few days. We do not now look so much to the signs of the actually developed disease—that is, to the presence or absence of early rigidity, of pains in the paralysed limbs, or

to the degree of impairment of sensibility. These latter considerations are, I believe, most treacherous and uncertain guides for the establishment of a pathological diagnosis, since variations in the relative intensity of such signs are always far more dependent upon the site and bulk of the braintissue injured than upon the particular pathological changes which the injured regions may have undergone. In fact, in attempting a pathological diagnosis we concentrate our attention just as steadfastly upon the mode of onset of the affection and the general pathological state of our patient, as we do upon all the signs of the fully established disease when striving to arrive at a regional diagnosis.

But, though the principal sources of information towards which we look for the solution of these two problems are different, it is none the less true that each source of knowledge is occasionally capable of supplying valuable collateral information towards the solution of that side of the diagnostic problem to which it is not specially related.

The views above expressed, Gentlemen, are not universally held by those who have spoken with most authority concerning diseases of the brain. The contrary opinion of some physicians on this subject is, however, as I venture to think, attributable principally to the comparatively slight attention

which they bestow upon the questions of regional symptomatology. They consider the symptoms of cerebral hæmorrhage and of cerebral softening as it would be necessary to do if the brain were a small simple organ with a definite set of functions, and scarcely in a manner consistent with the fact that it is a large and complex organ made up of parts having very dissimilar functions — injuries of which should therefore, according to their situation, reveal themselves more or less differently.

In order to treat the subject of pathological diagnosis properly, we must look first to the apoplectic condition itself, and to the varied causes which may occasion it.

Diagnosis of the Cause of an Apoplectic State.

—A medical man is often hastily summoned to see a person who has been found in an apoplectic or comatose state, concerning whose antecedents and the mode of whose attack little or nothing is at the time known. In such a case, therefore, the first question for consideration would be as to the cause of the comatose condition. The practitioner must endeavour to satisfy himself whether he has to do with a case of external violence causing internal injury, with a case of narcotic poisoning, of ordinary intoxication, of uræmic coma, of stupor following an

epileptic fit, or of cerebral disease from rupture or occlusion of some of its vessels.

In many cases there is not much difficulty in at once arriving at a satisfactory conclusion—one, I mean, which seems to carry some certainty with it. In other instances, however, it is almost impossible to come to a definite opinion at the time; we are compelled to wait so as to form a more certain judgment by the light of subsequent events. Very much might be said upon this subject, though at present I shall only attempt to give a few hints as to the considerations which should guide you in an emergency of the kind to which I have alluded.

Are there any evidences that the patient has fallen, or that he has received a blow upon the head? Clues of this kind should be sought and inquired after. Though, even if you do find that the patient has fallen and perhaps also struck his head, there will still be room for doubt whether a fit of some sort determined the fall, or whether the comatose condition has been entirely a consequence of the accident—either from mere brain-shock, or because the blow determined a large hæmorrhage upon, or (as more rarely happens) into the substance of the brain.

Where there is no evidence in favour of a previous fall or of a blow, the question should always present itself as to whether the patient is suffering from the effects of *some narcotic poison*—either from opium, prussic acid, or preparations containing them, or even from a too free use of alcoholic fluids.

The close proximity of a bottle containing poison may, in some cases, go far to solve our doubts; or, in the rare cases in which we have to do with prussic-acid poisoning, the diagnosis may be made with even greater precision where its odour is detectable in the breath. It is very dangerous and wrong, however, to rely too much upon the same kind of test, in justification of the conclusion that a person found in an insensible condition is 'merely drunk.' It may and does often enough happen that apoplectic symptoms due to cerebral hæmorrhage supervene in a person more or less immediately after he has been taking spirituous liquors. An acceleration, thus induced, of the cerebral circulation may have been the immediately determining cause of the rupture of some vessel in the brain; and unless there are some definite guiding symptoms present-such as conjugated deviation of the eyes or unilateral spasms, general or partial,-it may be extremely difficult, if not impossible, even for the skilled observer to at once decide whether he has to do with a case of brain disease or merely with one of profound alcoholic intoxication.

The difficulties are sometimes just as great to separate cases of opium-poisoning from those of hæmorrhage into the central parts of the pons Varolii. I have in a previous lecture already alluded to the similarities presented by these different conditions—to the fact that we are apt to get in both sets of cases the most profound coma, with general resolution of limbs, and pupils contracted almost to the size of a pin's point. For the discrimination of such states, however, it should be borne in mind that in poisoning from opium the onset of the coma is slow, whilst in cases of hæmorrhage into the pons it is generally rapid or absolutely sudden in its development. In the latter class of cases also the coma may be occasionally associated with convulsions or one-sided spasms-signs which are almost invariably absent in opium-poisoning.

Poisoning from prussic acid and its preparations is much less frequent, and may generally be discriminated with less difficulty. In addition to the possibility of detecting the poison by the breath, we almost always have to do with more or less marked convulsive phenomena in these cases, in combination with a cold and dusky or cyanotic skin, deep catching respirations, and dilated pupils. In taking into consideration the size of the pupils, however, in these and in other instances, you must not forget that

there are congenital peculiarities in some individuals, partly on the side of habitual dilatation and partly on that of habitual contraction.

Should you be able to eliminate external violence, narcotic poisoning, and ordinary intoxication from the list of possible causes of the comatose condition you will still have to inquire whether your patient's state is due to internal blood-poisoning (as in uræmia), to an epileptiform attack, or to rupture or occlusion of cerebral blood-vessels.

And, first of all, with reference to *uræmic coma* we must look partly to the characters of the attack itself, and partly to the absence or presence of certain bodily peculiarities with which this state is usually associated — namely, a pale, pasty complexion, with puffy eyelids, swollen ankles, and albuminous urine.

Here, however, it must be recollected that, should it be a person beyond thirty or five-and-thirty years of age who presents these characteristics testifying to the existence of renal disease, possibilities other than blood-poisoning are opened up. The general condition of such a patient would also warrant our assuming the possible existence of a degenerated condition of his cerebral arteries—capable of favouring either hæmorrhage or thrombosis. The estab-

lishment of the existence of Bright's disease, therefore, in a person past the meridian of life should not by any means incline us to pronounce too decidedly in favour of uræmic ccma as against an apoplectic condition due to cerebral hæmorrhage or softening. In order to form a definite and reliable opinion we must always look, in this as in other cases, not only to the nature of the attack itself, but also, when possible, to its mode of onset. What, then, are the leading peculiarities of uræmic coma?

An attack of uræmic coma almost always begins with convulsions, following prodromata in which drowsiness and headache are usually marked symptoms. The coma is not profound. Patients in this state may be momentarily roused with comparative ease, though they quickly relapse into their old condition when no longer spoken to or otherwise disturbed, as in cases of narcotic poisoning. There is, again, as Dr. Russell Reynolds has pointed out, much twitching of the limbs and often marked rigidity, though, as he says, 'the locality of these symptoms changes from side to side, and is not accompanied by fixed paralysis.' The muscles also, according to the same observer, show a decided increase in irritability when lightly tapped. Further, there is the very important fact ascertained by M. Bourneville, that the temperature of the body begins to fall with the onset of uræmic coma, and continues to sink as long as this condition persists, so that it may fall as low as 90° F. in fatal cases. On the other hand, in the coma due to cerebral hæmorrhage or softening the lowering of the temperature is slighter in amount, and, in cases not fatal within this period, rarely lasts longer than twelve to twenty-four hours. Peculiarities as to the kind of stertor (its oral rather than guttural character), first pointed out by Dr. Addison, and also peculiarities in the mode of variation of pulse and respiration, have been cited as more or less distinctive of uræmic coma, though they are signs which seem to me to have less positive value than those previously mentioned.

With reference to *epileptic coma*, I have to tell you that taken by itself it presents no distinguishing peculiarities. Nay, more, even if we learn from surrounding friends that our patient has been subject to epileptic attacks, there is only a strong presumption raised in favour of his present condition being a sequence of such an attack. It may, of course, be otherwise. A patient who has been previously subject to epileptic fits is not thereby protected from the occurrence of ruptures or occlusions in some of his cerebral vessels—far from it: so that an apoplectic condition met with in any such person may always have been determined by one of these pathological accidents.

With regard, however, to persons concerning whose antecedents nothing is known, you should recollect that such distinct pathological accidents as I have just mentioned are less likely to operate as causes of the comatose condition in patients who are youthful than in those who have passed the meridian of life; so that the chances in favour of a given case being one of epileptic coma decidedly increase with the youth of the patient.

There are, however, certain organic diseases of the nervous system occurring either in young or middle aged adults, or in old people, in which we are apt to meet with sudden epileptiform attacks (often continuing with intermissions for many hours), or else with attacks more distinctly apoplectiform in character. They occur, for instance, in association with disseminated sclerosis of the brain and cord. and also in persons suffering from general paralysis of the insane. Or similar attacks may be met with in persons who have already been suffering for some time from hemiplegia produced by softening or hæmorrhage-and perhaps more especially, as Charcot has suggested, in those cases where secondary degenerations in the pons, medulla and cord have been produced by the primary lesions.\*

<sup>\*</sup> These very different affections present, therefore, in a large number of cases—however different they may be in other respects—the common

The distinguishing clinical peculiarity of these attacks is that the temperature of the patient almost invariably begins to rise from the first, so that in two hours it may be 101°, and in four or five hours more—or even within a shorter period—it may have reached 104°. Where the convulsions cease and the patient begins to regain consciousness, the temperature may after a few hours gradually subside. In cases not about to terminate favourably, however, it rises still higher, and death may take place in the course of a few days.

During such attacks the pulse is also increased in frequency, and the convulsions, when present, are mostly unilateral. After death in these cases no recent lesion is to be met with at all accounting for the convulsions or the coma.

The sudden elevation of temperature is a sign of great diagnostic significance in enabling us to discriminate such functional or so-called 'congestive attacks' from apoplectic conditions due to hæmorrhage and softening on the one hand, or to uræmia on the other.

We have now narrowed the questions demanding consideration in regard to the cause of an apoplectic

character of an irritative sclerosis of the pons and medulla; and this pathological condition M. Charcot considers may act as the exciting cause of the epileptiform attacks—however inexplicable the sudden onset of the convulsions at intervals may be.

or comatose condition presented by a patient seen for the first time. If, in any given case to which you have been called, you have been able to come to the conclusion—and you may often do this very rapidly—that your patient is suffering neither from the effects of external injury, from narcotic poisoning, from 'drink,' from the accumulation of effete materials in the blood, nor from epileptic coma, you are thrown back upon the consideration of the possible existence of one or other of the ordinary grosser varieties of brain disease.

Is your patient's condition due to rupture of a vessel or vessels, with bleeding into or upon the surface of the brain? Is it due to simultaneous occlusion by embolism of many small vessels, or to the impaction of larger embolic masses in one or more important arteries near the circle of Willis? Or, lastly, is it due to a slow coagulation or process of thrombosis, which has been taking place in one or more of the larger vessels near the base of the brain? In short, have we to do with a case of cerebral Hæmorrhage or of incipient Softening; and, if the latter, is the nutritive and functional change due to embolism or to thrombosis? These are the problems which now remain for our consideration; and, in looking to them, it will be found most convenient no longer to direct our attention

exclusively to the apoplectic state. We may suppose that we are brought face to face with the patient at a later period, so that we are able, in addition, to have regard to the mode of development of the established disease—that is, of the paralytic condition and its attendant phenomena. We must, moreover, now include the consideration of cases with a simple mode of onset—cases in which no coma has pre-existed.

## Differential Diagnosis of Embolism, Thrombosis, and Hæmorrhage.

The indications in favour of the occurrence of Embolism are occasionally most precise, and, therefore, I will allude to them first. If a child, or young adult, known to be suffering from valvular disease, suddenly becomes hemiplegic, with or without loss of consciousness or convulsions, there would be good primâ facie ground for coming to the conclusion that such an attack is probably due to cerebral embolism. For it is important to recollect in this connection how large a proportion of cases of cerebral embolism occur in individuals under forty years of age, and that, on the other hand, cerebral hæmorrhage especially tends to occur with increasing frequency in subsequent decades. The probabilities in favour of embolism, therefore, would diminish as

we had to do with older patients, because, even though such older patients are the subjects of valvular disease, their cerebral vessels may also be in such a condition as to favour the occurrence of hæmorrhage or of thrombosis—and the chances would be also increased that the valvular disease might not be of such a kind as to favour the pathological accident now under consideration.

In any given case, however, the diagnosis of embolism would be strengthened if there were previous or subsequent evidence pointing to the probability of the occurrence of a similar accident in either of the kidneys, in the spleen, or in some part of one of the limbs. Again, we should not lose sight of the fact that embolism in the brain is most frequently met with in the middle cerebral arteries or their branches, and that of the two vessels it occurs more frequently in the artery of the left than in that of the right sylvian fissure.

With regard to *Thrombosis* and *Hæmorrhage*, they have both the greatest tendency to occur in elderly people, and each event may be preceded by more or less marked prodromata. The onset of hæmorrhage is, however, often almost as sudden as the onset of embolism. And although in cases where long and well-marked prodromata have been pre-

sent this fact alone would strongly tend to incline the diagnosis towards thrombosis, yet it appears incontestable from many recorded observations that the commencement of symptoms may likewise be altogether sudden in some instances of thrombosis.

The difficulty in deciding between embolism, thrombosis, and hæmorrhage, in many instances, therefore, becomes extreme—however attentively we may regard the mode of initiation of the malady, and the general condition of our patient. All that we can say is, that a very abrupt onset, in a young person more especially, and in association with the conditions already named, tells strongly in favour of embolism; and that long or well-marked prodromata terminating with an attack of hemiplegia in an elderly person (especially when he or she possesses a weak heart and rigid arteries) points almost as strongly in favour of thrombosis. But the establishment of the existence of renal disease with arterial degeneration by no means points so strongly as some writers have represented towards the probability of cerebral hæmorrhage-since the same conditions would, almost as strongly, favour the occurrence of thrombosis.

Now that I have pointed out the bearing of the indications to be derived from (1) the mode of onset of the affection, and you have learned how unsatisfactory these often are, you may naturally ask, Are there no means of deciding or of gaining further information from (2) the nature of the attack itself, and from (3) its subsequent course, whether we have to do with rupture, with embolism, or with thrombosis of cerebral vessels? These are the points to which I would now direct your attention.

Looking first to the question of the data for diagnosis to be obtained from (2) the nature of the attack itself, I may say that we do in many cases get indications which tell strongly in favour of Hæmorrhage. This is the case, for instance, when we have to do with a very profound and lasting coma from which the patient cannot be roused even for a moment—more especially where such a condition has not been ushered in by convulsions. A sudden hæmorrhage into the centre of the pons Varolii, or a very large bleeding into the lateral ventricles, into the centrum ovale of one hemisphere, or into the arachnoid sac, will give rise to such symptoms—and these may terminate in speedy death within a few days or hours, if not still earlier.

An attack of this kind is almost distinctive of cerebral hæmorrhage. Some years ago it might have been said to have been absolutely distinctive, but the combined experimental researches and clinical observations of Prévost and Cotard have gone far to show that multiple embolism occurring simultaneously over a wide area of the brain may lead to symptoms of the same kind, and to a similarly speedy death.

This latter occurrence may be met with as one of the ordinary accidents of life when, for instance, a fibrinous 'polypus' in either of the left cavities of the heart has undergone central softening and a giving way of its external layers suddenly liberates a large quantity of detritus; or when the contents of some large atheromatous depot near the commencement of the aorta is suddenly (from the giving way of its lining membrane) swept into the bloodstream—so that some considerable part of the débris is carried on to the brain, and there simultaneously blocks up a large number of its smaller vessels. Death often takes place in these cases before Softening has had time to become established, and nothing more than an irregularly congested and anæmic appearance may be seen by an ordinary nakedeye examination of the brain. Fortunately these are rare, and by no means ordinary, pathological events, though doubtless many instances of so-called 'nervous' or 'simple' apoplexy, recorded by earlier observers, would receive their most feasible explanation by being placed in this category.

Owing to the rarity of events of the kind to which

I have just been alluding, our knowledge of the possibility of their occurrence does not detract very much from the value of the indication in favour of cerebral hæmorrhage, to be drawn from the existence in any given case of sudden and very profound coma. There is, moreover, only one kind of thrombosis known to me which is at all likely to give rise to similar symptoms, and that is also a rare event. I mean complete occlusion by thrombosis of the basilar artery in nearly its whole length. Here, too, however, on occasions when it has occurred, the symptoms have presented themselves with tragic severity, although the onset has generally been to a trifling extent more gradual.

But in those numerous instances in which a hemiplegic attack begins either with a slightly marked apoplectic condition or with no loss of consciousness at all, the difficulty of diagnosing between hæmorrhage and softening only too often becomes supreme. We have to except, of course, those cases in which there is good evidence for believing that embolism has occurred, and those other less distinctive cases in which the long continuance of prodromata, combined with the general condition of the patient, point in the direction of thrombosis. But in all the remaining multitude of cases, if we are to decide at

all, we are driven back upon other considerations—upon such indications, in fact, as may be found in (3) the subsequent course of the disease, during the first week or ten days.

Looking at the question from this point of view, clinical observations do supply us with some important additional help.

In many cases of cerebral softening, for instance, and more especially in those due to embolism, we are apt to meet with a distinct remission of symptoms, and a diminution in the amount of paralysis, three or four days after the commencement of the illness. By this time the cerebral circulation on the confines of the affected area has been in part re-established by the opening up of collateral channels, so that a smaller bulk of brain-tissue thereby remains incapacitated for work.

Again, in some instances of commencing cerebral softening due to thrombosis, the symptoms go on increasing by successive stages in a rather characteristic manner during the first week or ten days, owing to the tendency so often shown by a thrombosis to extend backwards and gradually implicate more and more of the arterial region in whose ultimate provinces the process may have first commenced.

In hæmorrhage, on the other hand, we very rarely meet with the distinct remission of symptoms after three or four days, of the kind mentioned as occurring in embolism; a sort of febrile reaction, indeed, often sets in about this time, or a little earlier, leading to a slight exaggeration rather than to a remission of symptoms. It happens occasionally, however, in cerebral hæmorrhage, that there is an increase in the severity of symptoms, taking place by successive stages (especially where slight additional bleedings occur at corresponding intervals)—and where this takes place you may easily understand how very closely some instances of softening from thrombosis are simulated.

It remains for me still to call your attention to certain important thermometric observations, made by Bourneville in the hope of finding temperature-curves which should be distinctive of cerebral hæmor-rhage and of cerebral softening respectively. Some diagnostic indications have been obtained already as a result of these investigations, and, doubtless, others would be yielded by further pursuit in this direction.

Variations of Temperature in Hæmorrhage and in Softening respectively.

Bourneville found that, with the exception of a few altogether unusual cases, the 'period of initial lowering' of temperature commonly met with in cerebral hæmorrhage, is either absent or much less slightly marked where we have to do with cerebral softening. During the first two hours after the attack, the rectal temperature in cases of cerebral softening may range between 985° and 100°; whilst during the same period in cerebral hæmorrhage it is almost always below 985°. But after the first two hours in a case of softening, the temperature may suddenly rise to 102°, or even 104°, though it soon again descends to the normal standard, and afterwards exhibits altogether irregular oscillations-sometimes remaining the same for two days, and sometimes presenting evening or morning remissions of nearly two degrees. Amongst the exceptions to this order of events in cerebral softening we have to include most instances in which the pons Varolii is thus affected—this being a part of the brain which, as I have already had occasion to mention, is known to contain most important vasomotor centres.

In cerebral hæmorrhage, whilst the 'initial lowering' of temperature is generally much more marked than in cerebral softening (owing, doubtless, to the greater shock, from laceration of brain-tissue), it is found that if the temperature rises soon after the attack to a point beyond 102.5°, it rarely sinks again to the normal standard, except under the influence of a shock resulting from fresh hæmorrhage. The

sudden rise to 102° or 104°, followed by a speedy fall, becomes therefore very suggestive of the existence of softening rather than hæmorrhage, unless there is reason to believe that the fall has been occasioned by a fresh accident within the cranium. Again, in the so-called 'stationary period' the oscillations of temperature, according to Bourneville, are more regular and slighter in cases of cerebral hæmorrhage than in those of softening. Such a period lasts for a variable time in instances of cerebral softening, though where one of these cases is about to prove fatal, the temperature-wave becomes characterised by an 'ascending period': even here, however, in the majority of instances, the rise in temperature takes place more slowly than where we have to do with the terminal period of a case of cerebral hæmorrhage. Ramollissement may terminate fatally, in fact, when the rise of temperature has only been very small-not more than to 102° or 104°. Though in other instances, as where we have to do with extensive softening of the pons Varolii, it may rise, as I have myself had occasion to observe, to 109° or even 110° at the time of death—just as in some cases of death from cerebral hæmorrhage.

The variations in the pulse and respiration afford no positive indications which may aid us to distinguish between cerebral hæmorrhage and cerebral soft-

ening. And although I have been able to lay before you the foregoing temperature results obtained by so careful and accomplished an observer as Bourneville, it is desirable that they should be extended and verified by himself and other workers. We ought to know whether more extended observation will show that the indications with which he has already supplied us will prove constant for the great mass of cases of hæmorrhage and of softening respectively. But, above all, we ought to know how far these respective temperature-curves will hold good for lesions in different encephalic regions - that is, whether regional differences will prove subordinate or otherwise, in their power of altering the temperaturecurves, to differences in the nature of the pathological process.

With the exception of a few general or miscellaneous statements, which it may be useful to bear in mind, I have little more to add concerning the problems which present themselves when we strive to come to a correct pathological diagnosis.

You will recollect that, when speaking of the causes of hemiplegia, I directed your attention to the comparative frequency of arachnoid hæmorrhages in children, and also to the frequency with which such lesions follow blows upon the head in persons of

any age—and to the consequent greater frequency of these pathological accidents in males than in females.

Again, you must recollect that hæmorrhage into the brain-substance may and does occur at times in young adults—even in children; and also that paralytic symptoms are occasionally met with in young subjects after debilitating acute diseases, when the blood has happened to have been rendered præternaturally prone to coagulate or to permit of the more easy separation of its fibrine. Such states of the blood, as you know, greatly favour the occurrence of thrombosis, even where there is very little in the way of local arterial degeneration to determine its occurrence. General causes of this kind seem also to operate, at times, in producing cerebral thrombosis in delicate women soon after parturition.

Again, with regard to the region most prone to be affected in the two sexes, it would appear that softenings in districts supplied by the branches of the carotid artery are actually more common in females than in males; whilst the reverse is true for districts supplied by branches of the vertebral and basilar arteries—this latter fact being in part due to the decidedly greater frequency of softenings of the cerebellum in men than in women. I may further mention that there is agreement in this respect between

the pathological proclivities of the spinal cord and of the cerebellum in the two sexes; these being parts of the nervous system which undoubtedly have a very close functional relationship, and which, as a general rule, are called into greater activity amongst men than amongst women.

You may recollect, also, that hæmorrhage is more prone than softening to occur in the middle lobe of the cerebellum; that softening of the pons is more apt to be central than lateral; and that signs of acute disease in the optic thalamus are more likely to be caused by hæmorrhage than by softening. These are facts which may be gathered from past experience; and whilst they are harmonious enough with anatomical data, they also show us how a correct regional diagnosis may at times cast a reflected light upon the solution of the no less difficult problem of pathological diagnosis.

## LECTURE VIII.

PROGNOSIS IN APOPLEXY AND HEMIPLEGIA—TREAT-MENT IN THE DIFFERENT STAGES OF PARALYSIS FROM BRAIN DISEASE. Prognosis in Brain Disease-Importance and Complexity of the Problems-Questions to be considered-Danger of Death during the Apoplectic State-Depends much upon Locality and Extent of Lesion--Liability to Death shortly after-Much influenced by General Health-Signs of Evil Import-Recovery of Power of Articulation-Import of Aphasia and Amnesia-Degree of Recovery of Mental Power-Occurrence of Dementia-Semi-Idiotic Condition from Brain Lesion in Infancy-Probability of Return of Motor Power-Unfavourable Indications-Delayed Recovery of Power in one Limb-Limitation of Paralysis to Face and Arm-Such cases sometimes associated with Mental Impairment or Epileptiform Attacks-Recovery from Sensory Impairments-Favourable Indications-Liability to Recurrence of Attack-Fatality of 'Third Seizure'-Liability dependent upon Age-Not diminished after one Accident-Liability to Recurrence of Embolism.

TREATMENT in Apoplexy and Hemiplegia-Preventive Treatment-Must vary much in Individual Cases-Careful study of Patient's General Health needed-Indications where there is a Weak Heart and Arterial Degeneration-In Hypertrophy of Heart with Arterial Disease-Means to be adopted in Cases of Syphilis or threatened Embolism-Treatment of threatened Attack during Recovery from Acute Disease-Management of Case in Impending Hæmorrhage - Treatment in the Apoplectic Condition-Heroic Measures-General Management of Case-Treatment of a Complicating Epileptoid Condition-Often no Drugs required -Necessity for quietude-Rest for Organ of Deglutition-Artificial Feeding - Rest for Organs of Articulation-Subsequent Measures-Treatment of the Hemiplegic Condition-General Management-Treatment of Epileptiform Attacks-Galvanization of the Brain-Strychnia and Tonics-Faradization-Conditions in which it should be used - Impaired Sensibility-Conclusion.

## LECTURE VIII.

GENTLEMEN,—Having in previous lectures completed a brief survey of the Causation, Symptomatology, and Diagnosis (both regional and pathological) of the various forms of rapidly-developing hemiplegia to which we have been restricting our attention, it now remains for us to study the all-important problems of Prognosis and Treatment.

## PROGNOSIS.

In no class of diseases will your prognosis be more anxiously sought by relatives and friends than where you have to do with what is popularly termed a 'fit of apoplexy,' a 'seizure,' or a 'stroke of paralysis.' These affections still seem to inspire a sort of mysterious awe or dread in the lay mind, decidedly above that attaching to diseases of the lungs or of the heart. The cause of such a feeling

is, moreover, not difficult to find. The brain is the organ principally concerned with mental operations, so that over and above the known fatality of 'fits of apoplexy' and allied affections, there is the dread of the mental wreck or feebleness which may result from such attacks, to say nothing of the miserably helpless condition the paralysis itself entails for months or years.

The problems of prognosis are, however, nowhere more many-sided and complex than in these affections. Your verdict in any particular case will depend much upon the age and state of health of your patient, taken in conjunction with the view which you are enabled to form as to the situation of the mischief, as to its pathological nature, and as to its extent. Hence it is that the correctness or reality of your prognosis in these cases of brain disease will depend, gentlemen, so much upon your ability to arrive at an accurate regional and pathological diagnosis.

The opinions which you form on these difficult subjects must, of necessity, very greatly influence your verdict on the several questions which go to make up a complete prognosis in a case of apoplexy, or of paralysis from cerebral disease. There are, indeed, many separate questions to be answered, the principal of which are as follows:—(I) Is the

'seizure' or fit of apoplexy itself likely to prove fatal? (2) Or, this stage having been recently passed through, are the present symptoms such as to augur an approaching fatal result? (3) What are the chances of recovery of speech or of mental power, where these are impaired? (4) Or, mental power not being much affected at the time, what are the prospects of its subsequent failure? Further, with regard to the motor paralysis, you have to consider (5) the chances of recovery of power in leg or arm-will the limbs again prove serviceable, or will they remain useless, and finally become more or less contracted? (6) How long are sensory impairments likely to continue? (7) Lastly, should the patient get over this attack, what amount of probability is there that it may be repeated?

Now, as may be well imagined, where questions of this kind have to be considered, each individual case must be made the subject of special study, because the different combinations of determining conditions presented at the bedside are almost endless in their variety. We have to do with patients presenting wide and multitudinous variations in age and in their state of general health, suffering from lesions which in different cases afford the utmost diversity in respect of their extent and suddenness, their pathological nature, and their

situation in this or that encephalic region. In the face of such complexity, therefore, inherent in the nature of our present subject, I can only give a few hints, partly general and partly special, for your future guidance in respect to those principal prognostic questions which I have already specified.

(1) Will death take place during the apoplectic condition? In weighing this question, we look to the depth or degree of coma which the patient presents. If he cannot be roused at all, even for a moment-if there are no signs of reflex action when the conjunctivæ are touched—and if there is wellmarked stertor, together with unheeded passage of fæces and of urine, the patient may die rapidly within a few hours or even a few minutes. And in cases where either death or recovery does not soon take place, the persistence of such signs as I have just mentioned, without abatement, for twenty-four hours is an indication of extreme gravity. The large proportion of such patients are destined to live at most only two or three days, the comatose condition persisting to the end.

Much disturbance of respiration, especially where it becomes slow and laboured in character, and also quickness with marked irregularity of pulse, are signs of evil import. Again, it has been pointed out by M. Charcot that a very marked and persistent depression of temperature, setting in with or occurring immediately after the commencement of the attack, is a sign of almost certainly fatal augury.

The speedily fatal cases belonging to the category we have just been considering are furnished by very large hæmorrhages into the centrum ovale (either with or without rupture into the ventricles), by very large bleedings upon the surface of the brain, or by hæmorrhage into the pons Varolii. Much more rarely, multiple minute embolisms of the brain, or complete thrombosis of the basilar artery, may produce similarly disastrous results.

(2) The apoplectic condition itself being survived, are the symptoms such as to augur an approaching fatal result? In the set of cases last considered the prognosis is comparatively little influenced by the age and general condition of the patient—since in them the result depends to such an overwhelming degree upon the situation, the extent, and the suddenness of the lesion. But where our patient has sailed through the perilous period comprised within the first few days of the 'seizure,' then both sets of considerations have to be fully weighed. In these less severe cases, in fact, the general condition of the patient and the state of his other principal organs

may very much influence the result. That is to say, the coexistence of grave disease of the kidneys or of the liver, or a general state of malnutrition, howsoever induced, may produce a tendency towards a fatal result in cases where the extent and situation of the cerebral lesion itself would by no means threaten an evil termination had it occurred in a more healthy individual.

When the patient is suffering from no inflammatory complications, a sudden rise of temperature in cases of cerebral hæmorrhage to 102°-104° is a most grave indication. If occurring within three or four days of the commencement of the attack, such an initial rise of temperature may go on steadily increasing so that the patient dies in the course of a few days. Another sign, also belonging to the irritative type, is said by M. Charcot to be of great significance, and that is the appearance of a process of acute sloughing of the buttock on the paralysed side, commencing within a few days after the onset of the apoplectic attack (p. 163). The cases which present this complication almost always end badly, though I have again to remind you that, fortunately, it is a pathological complication not often met with.

Conjugated deviation of the eyes is a condition which, as you know, exists principally in the more severe cases of hemiplegia, especially in those whose origin is abrupt. Otherwise it is a sign which in itself has no special significance from a prognostic point of view. Very decided difficulty in deglutition is always a grave symptom, since where it is most marked we almost invariably have to do with severe lesions near the medulla oblongata. Extreme emotional weakness is also (and for a similar reason) of rather serious import.

A sudden depression of temperature, with increase or renewal of a pre-existing comatose condition, is usually indicative of a fresh hæmorrhage, though the same symptoms may occur in cases of softening as a result of new and sudden vascular occlusions.

Febrile reactions are apt to manifest themselves within a few days after the onset of an apoplectic attack due to cerebral hæmorrhage, partly owing to a sub-inflammatory or irritative influence of the blood-clot, though often, even more, in consequence of an inflammatory condition which has been initiated in the manner already described in one or both lungs. You will recollect that I called attention to this condition (p. 165) when speaking of the nutritive changes which are apt to be associated with an attack of hemiplegia. I may remind you also that in the opinion of Dr. Brown-Séquard these sympathetic inflammatory conditions are more common

where we have to do with injuries of the right than with those of the left cerebral hemisphere.

Should the patient survive the first ten or twelve days, we have comparatively little reason to expect a fatal result from the brain lesion itself, or from its more immediate complications. We still, however, have to consider the degree of improvement likely to take place in our patient's power of articulation and of speaking, the extent to which his mental faculties will be restored, and the probability of a restoration of motor power in the paralysed limbs.

(3) What I have already said concerning the prognostic significance of profound difficulty in deglutition also holds good for very marked difficulties in the mere articulation of words or sounds. These two disabilities, indeed, often go together, and a considerable proportion of the cases in which they are present to a marked extent do badly. But where the patient is not carried off by the more immediate effects of the lesion, or later by concurrent inflammatory mischief, he may gradually, though slowly, regain his power of easy swallowing and also of speaking with distinctness.

The existence of 'aphasia' confers no extra gravity upon the case apart from the unfortunate nature of the symptom itself, and however marked this defect may be at first, there is always some chance of its almost complete disappearance after a time. Our prognosis on this head, however, must be cautious, and guided by signs of improvement which have already taken place (if any), and by the rate at which they manifest themselves.

The co-existence of any marked 'amnesic' defect rather increases the gravity of the case, and where such a condition shows no sign of abatement after a time, there is reason to fear a subsequent further impairment of mind.

And, looking now more especially to the mental condition of the patient in cases such as we have just been considering, I may say that in the majority of instances, after the first loss of consciousness or stupor (with or without delirious wandering) has passed away, the patient is left free from any very decided mental defect. The mental weakness which does exist shows itself principally in an increased tendency to emotional displays. The patient is more easily affected than of old, and more easily worried by small things; he shows less power of control, whilst some of his finer sensibilities are more or less blunted. The memory is often appreciably weakened, and the power of application or steady attention also fails. Yet these defects may after a time to a great extent disappear.

(4) In other, though rare, instances, however, it happens that a hemiplegic attack becomes the starting-point of a chronic maniacal condition—of a mental degradation which may go on to the establishment of a more or less complete condition of dementia. This unfortunate sequela is most apt to ensue where we have to do with lesions in or about the cortex, especially in elderly people in whom there has been already a tendency towards the occurrence of degenerative changes in this region of the brain.

Again, large lesions occurring in infancy or at the time of birth, either in the substance or upon the surface of the brain, not unfrequently entail a demented or rather a semi-idiotic condition. The child may never learn to speak, and may be quite unable either to stand or walk. In other instances the motor defect is more distinctly limited to one side of the body, and then, in those cases more especially where the lesion occurs before the third or fourth year, the growth of the paralysed limbs may be also more or less retarded. Such patients are often subject to fits, and they may be habitually restless and destructive, whilst, not unfrequently, they give further vent to their feelings by screams and an unintelligible gabble. No more distressing or hopeless cases than these can often come under the notice of the physician.

(5) We turn next to the question of the probability of the return of motor power in the paralysed limbs themselves. However complete the paralysis may be for the first week or ten days, we need not altogether despair of an ultimate recovery of power. Still it may be laid down as a general rule that the later the first signs of returning motility are in manifesting themselves, the less probability is there of the recovery being complete.

The outlook is, therefore, decidedly bad if the patient does not begin to regain some slight power over the lower extremity in the course of three or four weeks. In such cases, recovery, when it commences, may advance very slowly in the leg, but after a time no further progress is made, so that the patient is henceforth only able to walk with difficulty, when aided by a stick. Recovery of power in the arm is found to be still more sluggish and partial. The patient only gains the power of moving it a little from the shoulder, whilst the limb hangs almost powerless by the side, wasted from disuse and colder than its fellow. Gradually, if great care be not taken (and at other times even in spite of it), a more or less marked 'late rigidity' becomes established.

The cases in which 'early rigidity' manifests itself are certainly unfavourable from our present point of view. Where this sign is well marked and persistent, either in the upper or in the lower extremity—but more especially in the former—the limb is peculiarly liable not to recover its motor power; and then the early rigidity, which at first relaxes during sleep, gradually passes into the more persistent late rigidity, with its accompanying organic changes in nerves, muscles, and joints.

The early advent of nutritive changes in the paralysed limbs has already been spoken of as one of the rarer occurrences of the hemiplegic state (pp. 167-172), and I have now to inform you that it is also one of unfavourable omen as regards the ultimate recovery of motor power. I allude especially to those subacute inflammatory changes in the joints and principal nerve trunks, as well as to the early wasting of muscles which may occur in the paralysed limbs, either singly or in concert, within a very few weeks after the commencement of the attack.

You must recollect, moreover, that there is no necessary parallelism in the degree of recovery which manifests itself in the upper and in the lower extremity. Thus, the leg may recover more or less rapidly, whilst the arm, at the same time, or rather later, undergoes comparatively little improvement—the fingers and elbow becoming ulti-

mately more or less hopelessly contracted. Or, in other instances, it happens that the arm recovers power whilst the leg remains useless. I have already alluded to these cases pretty fully (pp. 133-141), and have informed you of the gloomy prognosis which Trousseau pronounced respecting them, so that I need say nothing further on this subject at present.

The cases in which paralysis is from the first almost limited to the arm and face, are not always the less grave for this restriction. In some of them it happens that the paralytic symptoms, such as they are, do not readily disappear; and whilst these cases are sometimes characterised from the first by a more than usual amount of mental change, they are also apt to be rendered still more grave by the recurrence, from time to time, of epileptiform attacks, which may have commenced with or soon after the onset of this partial paralytic condition.

(6) Of the sensory impairments which may complicate the hemiplegic condition, those in the power of smelling, tasting, and hearing are usually of little importance in hemiplegic affections, either on account of their rarity or of their temporary duration. Defects of sight, however, are more frequent, and are also apt to be more lasting. Still, you will recollect that where even perfect blindness at

first accompanies a hemiplegic condition which has been initiated by convulsions, the defect is often one of brief duration, and may altogether disappear at the expiration of ten days or a fortnight at the latest. Blindness of one eye, however, either on the sound side or on the side paralysed, occurring simultaneously with a hemiplegic condition (p. 113), is more likely to be due to actual organic changes in some portion of the lower visual track, and is therefore much more likely to prove a lasting defect.

The diplopia due to paralysis of one or more of the ocular muscles disappears as the paralytic condition itself subsides.

With regard to the impairment of common sensibility, we may safely say that, in the majority of cases, there is a natural tendency for this symptom speedily to disappear. But in those exceptional instances in which actual loss of sensibility or disagreeable subjective sensations persist after the first two months, these discomforts may continue to trouble the patient for an indefinite time.

Hitherto, gentlemen, I have alluded principally to the signs of evil import. I have dwelt, perhaps, too long on the gloomy side of the picture; but it remains for me now to assure you that cases in which such evil portents are not met with are by no means

rare. In very many instances, as most of you must be aware, patients not only recover from the apoplectic condition itself, but they subsequently regain power over the limbs which have been paralysed. It will be easily understood, moreover, from what I have already said, that the graver and more immediate question of prognosis will, as a general rule, be favourably answered or not in proportion to the slightness of the apoplectic attack; that subsequent death within the first week or two is much less likely to occur in young, or otherwise healthy individuals, than in those who are already the subjects of renal disease or other grave organic affections; and that, so far as the recovery of power in paralysed limbs is concerned, the likelihood of this being complete is in direct proportion to the speediness with which an improvement of this kind begins to manifest itself.

(7) It now remains for me to say a few words as to the probability of the recurrence of a similar apoplectic or paralytic attack. That there is some tendency to the return of such attacks is commonly known, and upon this knowledge another notion has been grafted and widely diffused as to the fatality of a third 'seizure.'

Now, I need scarcely tell you that if two or

three seizures do occur, they are independent accidents linked only by their relation to one or more common causes or underlying pathological conditions. The severity of a third attack, therefore, or the actual amount of cerebral damage produced, is not influenced by the fact that two other pathological accidents have previously occurred.

Yet I do not mean to suggest that statistics might not bear out the notion that fewer persons in proportion recover from a third than from a first or even a second attack. In all probability this is the case, and the fact itself—if true, as I believe it to be—would admit of easy explanation. If third attacks are just as likely to be severe as first attacks, they are surely more likely to prove fatal, seeing that the lesion by which such an attack is occasioned occurs in a brain already weakened by two previous accidents.

Turning now to the question of the degree of liability to recurrence, I may say that this is undoubtedly greatest where we have to do with a patient over sixty years of age, who is at the same time the subject of much arterial degeneration. In such a person minute miliary aneurisms are very likely to be present on the cerebral vessels, and where the action of the heart is powerful the occurrence of cerebral hæmorrhage is decidedly favoured.

Any liability thus occasioned of course continues, even though the patient may have already had one or two apoplectic 'seizures.' And a similar remark also holds good concerning the possible repetition of cerebral softening in an aged patient with a feebly-acting heart in addition to narrowed and roughened arteries.

Again, with regard to cerebral embolism occurring in younger subjects, it is well to bear in mind that patients in whom endocardial vegetations exist are in continual danger from the possibility of a fresh Some sudden emotional disturbance or attack. violent exercise may at any time so exaggerate the heart's action as to sweep off an embolic fragment, which may straightway be conveyed to and block one of the middle cerebral arteries. Then, again, old vegetations are apt to grow by surface additions during or after many acute diseases, or whilst a patient is notably lowered in health from various causes—so that where we have to do with this kind of mitral or aortic disease, the risk of cerebral embolism is liable to be increased from time to time, in accordance with particular variations in the patient's general condition.

Thus, whatever cause may have given rise to the first paralysing lesion, patients are always liable to its recurrence. The second attack, however, may take

place either from a similar or from a different cause, because many elderly patients are about equally liable to an attack of hæmorrhage or an attack of cerebral softening. And speaking generally, it may certainly be said that the liability to recurrence increases very notably with the age of the patient in whom paralytic symptoms have already shown themselves.

### TREATMENT.

This part of our subject has to be considered from three points of view. We must look—(I) to the best means of averting a threatened attack of paralysis; (2) to the treatment of the apoplectic condition; and (3) we have to look to the therapeutic measures most calculated to restore power to the palsied limbs, or to improve other related anomalies which frequently accompany the hemiplegic condition.

(1) Under the first of these heads it is almost impossible to give any but very general directions, since the most prudent course to adopt in different cases depends so much upon the particular symptoms presented and upon the age and state of health of individual patients. Each case, therefore, as it occurs, must necessarily be made the subject of

special study. You must strive to learn the relative value to be attached to the different premonitory symptoms which I have previously mentioned; and you must, above all, seek to ascertain what pathological process such symptoms, together with your patient's general state of health, may indicate as possibly imminent.

Much will depend upon the correctness of your judgment in these respects. Should your patient be aged, with signs of arterial degeneration, and should the action of his heart be intermittent and weak, he would need stimulants, cardiac tonics, and the frequent administration of easily assimilable fluid nutriment. Your preventive treatment in such a case would in fact require to be altogether different from that which would be called for in a less aged patient with a full and bounding pulse and signs of hypertrophy of the left ventricle.

Where a patient is suffering from syphilis, or from the cachexia which it has left as a sequela, you have especially to look to the rectification of the general health by specific or other remedies—bearing in mind that there may exist not only a thickening and narrowing of the calibre of some of the cerebral vessels from a nuclear overgrowth in their various coats, but also a state of blood which causes it to be unduly prone to coagulate. And similarly you have

to look especially to the general health and mode of life of those younger patients who may be suffering from endocarditis, in order that by good food, duly moderated exercise, and freedom from sudden excitements, you may diminish as much as possible the chance of the occurrence of cerebral embolism.

Again, should there be signs of some cerebral complication in a young or middle-aged person during the period of recovery from one of the acute diseases, you must recollect that commencing thrombosis is most likely to be the occasioning cause—a thrombosis, too, which may be almost if not quite independent of degeneration in the vessels, but which is essentially determined by an abnormal condition and influence of the tissues outside acting upon slightly altered and more slowly moving blood within. White blood-corpuscles are more prone to lag and adhere to the walls of the vessels where certain states of the tissues exist, and fibrine may be also more prone to separate when particular alterations in the blood are present. A stoppage thus produced in a minute vessel, under the influence of favouring conditions may more or less rapidly spread to larger trunks; whilst, on the other hand, the commencing process of occlusion may be resolved and the tendency itself cut short under the influence of stimulants, good food, quinine and other tonics.

Thus you may see what considerable differences in treatment and general management are needed, according to the condition of your patient and the nature of the process which you may wish to avert. Your mind, therefore, must not be too much enthralled by the notion that cerebral hæmorrhage is about to occur; and neither must you, as a matter of routine, subject your patient to such general treatment as the threatening of such an occurrence would demand. Yet, where this is really the accident most to be feared, you cannot keep the patient too quiet in mind and body. You must also keep him cool, see that his head and shoulders are well raised, give him nothing but light and easily digestible food, whilst at the same time you may endeavour to calm any undue excitement about the heart's action and diminish the flow of blood through the brain by the administration of bromide of potassium, of aconite and ergot, or, by what may be still better, two or three grain doses of bromide of camphor. When hæmorrhage is threatened, moreover, you must always administer purgatives or emetics with the utmost caution, and by no means resort to them as routine measures. Keep well before your mind the risk which may be entailed by an indefinite amount of muscular straining in the act of vomiting, for instance, so that you may not resort to such a measure except in the face of a strong necessity for emptying an overloaded stomach.

(2) Our treatment of the Apoplectic Condition itself is now wholly different from what it was in the last generation when our predecessors bled their patients again and again, and were by no means sparing in their use of blisters and of drastic purgatives. The teachings of the late Dr. Todd, of Trousseau, and many others, on this subject, have fortunately been very widely received and adopted, so that it is almost needless for me to say anything against a method of treatment which, however firmly established it may have been at one time, is now almost wholly discontinued. We have happily learned to recognise the inutility of, and also to fear the risks attendant upon, this routine method of bleeding and of active interference generally - we are content, indeed, to trust more to the natural course of events, and carefully to subordinate our own direct efforts at cutting short the comatose condition.

Still, much may be done for a patient in this state by watchful care and attention. Should there be much heat of head, with violent throbbing of

vessels, you may apply pounded ice in a bladder or caoutchouc bag, or evaporating lotions, whilst your patient lies in a cool airy room, with head and shoulders raised and everything loose about the neck. At the same time you may wrap the feet in flannels, and apply hot bottles to them—or, if you choose, mustard-plasters may be applied to the calves of the legs.

Where the patient is able to swallow without much difficulty you may attempt to tone down an excessive force and frequency of the heart's action by moderate doses of bromide of potassium combined with tincture of aconite. But where the pulse is very rapid and irregular without undue force, it will be most prudent to abstain from the administration of drugs. When the breathing is very slow, however, and the pulse flickering and weak whilst the patient's face is cold and clammy, you must have recourse to a very cautious administration of stimulants.

Should an epileptoid condition supervene, in which there are rapidly recurring convulsive attacks, these should be met by rather large doses of bromide of potassium, as the medicine most likely to exercise a favourable influence upon the patient if the condition is at all amenable to the influence of remedies. Unfortunately, however, this is too often not the

case. The same remedy in smaller doses is often very useful where there is a restless condition with more or less delirious wandering—or, in such cases, small doses of bromide of camphor may often be administered with advantage.

Where we have reason to believe that a prolonged status epilepticus is not due to such obvious lesions as hæmorrhage or occlusion of vessels - where it supervenes, for instance, in a person already suffering from some severe organic affection of the nervous system, and where the patient's temperature steadily rises from the first (p. 272) — we may attempt to diminish the intensity of or cut short the convulsive condition by putting the patient under the influence of chloroform. In the convulsions of uræmia, however, where the temperature as steadily declines from the first, a totally different treatment must be had recourse to. Purgatives and the extraction of several ounces of blood are here often of the greatest use in cutting short the convulsive and comatose condition

Where there are good reasons for believing that thrombosis or embolism of some large artery exists, occasional inhalations of nitrite of amyl may be had recourse to with the view of bringing about its characteristic physiological effects upon the small vessels of the brain. We may thus aid the thorough establishment of a collateral circulation, and thereby bring the most direct and potent curative influence to bear upon our patient's condition. This means must, however, only be had recourse to with great caution and where the diagnosis of impending softening can be almost certainly made. The indications would, of course, be quite opposite in a case of cerebral hæmorrhage, since for some time after such an accident had occurred we should rather strive to diminish the calibre of the small cerebral vessels by ergot, bromide of camphor, or other drugs—in order to lessen the risk of any extension or repetition of the original accident.

In very many cases, however, no drugs will be required during this stage. The patient's state must be carefully watched, and if it seems desirable to unload the bowels, this may be often best accomplished by means of an enema containing castor oil or castor oil and turpentine. Where these means are not successful two drops of croton oil on the tongue may be administered. The state of the bladder must also be looked to, and if necessary the urine drawn off by catheter.

For many days after the condition of unconsciousness has wholly or partially passed away, the patient must still be kept as quiet as possible in body and

mind, and this rule more especially holds good where there is reason to believe that hæmorrhage has occurred. The diet and secretions must be carefully regulated, and in those exceptional cases in which there is very great difficulty in deglutition, the most prudent course for a time may be to feed the patient through an ordinary œsophageal tube, or else by means of a large gum elastic catheter, with its end cut off, passed through one nostril and down into the commencement of the œsophagus. The latter means is most applicable where there is any spasm or difficulty in opening the mouth, though it is contra-indicated in some instances owing to the sneezing to which the operation gives rise. Fortunately however, in cases like those to which I am now referring, the sensibility of the nasal mucous membrane, on one side at least, is often considerably blunted.

By the adoption of one or other of these means, or, as a last resource, by injections into the rectum, we may secure rest for the damaged nerve-centres, and so contribute towards the patient's earlier recovery. And, similarly, where there is very great difficulty in articulation, the patient should for a time be kept absolutely quiet, and not allowed to attempt to speak. A little later on, when there is less danger of inducing irritation at the seat of injury, such

a patient must be encouraged to swallow properly prepared food, and he must also be encouraged in his attempts to repeat or utter simple words. For when the effects of the shock have thoroughly subsided and an amendment is beginning to take place, a judicious calling into play of weakened powers will, of course, materially assist in effecting their complete re-establishment.

In the early days of the disease, and indeed throughout the time that the patient is confined to bed, the greatest care must be taken to prevent the occurrence of bed sores on the paralysed side, by securing extreme cleanliness and constant attention to the state of the bedding. In severe cases where there is any danger of such an accident occurring, the patient should be put upon a water bed from the first.

You must be extremely careful also thoroughly to protect your patient from cold draughts of air at the commencement of his illness, because of the natural tendency which exists in many such cases to the occurrence of bronchial inflammations and low pneumonic affections. These will, unfortunately, sometimes supervene and, moreover, greatly assist in bringing about a fatal termination, even where no exciting causes have been in operation.

The treatment of such cases must be similar to that to which we have recourse when pneumonic affection occurs in asthenic individuals; we should rely indeed principally upon nutritious fluid food, the cautious administration of stimulants, and an occasional use of hot fomentations.

(3) One of the most important means of bringing about or facilitating a return of power to the palsied limbs lies undoubtedly in a thorough attention to the general health of the patient. We must strive to rectify or mitigate general ill-health from whatsoever cause it may arise. We must see that the patient has easily digestible and nutritious food, that he sleeps well, that he is freed as much as possible from all mental perturbation, and, as far as may be, surrounded by an atmosphere of general cheerfulness. These important measures should after a time be combined, in the majority of cases, with passive movements of the paralysed limbs, with frictions of the skin by means of a flesh-brush or flannel, and with occasional volitional efforts to move the parts to which power is already returning—always bearing in mind that such attempts should stop short of the production of fatigue. Tonics such as quinine and iron, either separately or in combination, or fivegrain doses of iodide of potassium, are often desirable

at this time. These remedies, after a time, when the patient's digestive powers are good, may be combined with small doses of cod liver oil.

See that your patient is not confined overmuch to the house as soon as it is safe for him to get out. Open-air exercise in a chair or carriage, whenever the weather is suitable, may do much to improve the patient's general health; and later still, when he has regained some power, much good may also be done by shower baths, or sponging with salt water—either tepid or cold. Stimulants may often be taken in moderate quantities with advantage, though the amount taken at any one time should always be small. Smoking need not, as a rule, be absolutely interdicted in persons accustomed to the use of tobacco. The habit must rather, where necessary, be diminished and carefully regulated.

In cases where epileptiform attacks have been set up at intervals since the onset of the paralysis you will do well to administer iodide of potassium in combination with the bromide in some bitter infusion such as calumba or cascarilla. Where relief does not follow the administration of such remedies, you may have recourse to bromide of camphor, oxide of zinc, or even to four minimum doses of liquor arsenicalis.

Do not in the early stage of hemiplegia resort to

electricity. Harm may be done by a too early faradization of muscles, and, as for galvanization of the brain (i.e., the passage of a galvanic current through the head), I would not counsel you to have recourse to it at any stage. An early adoption of such a method might do actual harm, and where it is had recourse to later, it is, to say the least, not likely to do any good. I am not aware of any sound principles upon which such haphazard attempts at amelioration could be recommended.

Where the paralysis has already existed for a long time and the muscles are in a flaccid condition, the patient often derives great benefit from vegetable tonics in combination with minute doses of strychnia - beginning with one-thirtieth of a grain three times a day. Phosphide of zinc in doses of onetenth of a grain seems also to be beneficial in such conditions. And in the same class of cases faradization is often of the greatest service. It alters and improves the condition of the muscles, so that they become more capable of responding to those weak volitional stimuli which are alone capable of emanating from the diseased cerebral hemisphere, but which previously had no effect in calling the altered muscles into activity. Obviously, if the cerebral stimulus is weakened, we should do all in our power to improve the nutrition of the muscles, and thus

render them more receptive or amenable to incitations of diminished strength.

Where a comparatively early contraction of the hand and wrist sets in, this ominous condition may occasionally be arrested or retarded by cautious faradization of the extensors of the fore-arm three or four times a week. Or, if this alone should not suffice, it may be combined with an application of the constant current to the flexors—with the view of more directly attempting to diminish the spasmodic tendency.

In those rare cases, moreover, in which an early wasting of the muscles of the paralysed limbs is met with, much good may be done by faradization. And we need have the less fear in resorting to such a measure, because there is good reason to believe that this process of early wasting is brought about under the influence of changes which have been secondarily induced in the spinal cord, rather than as an immediate result of any 'irritative' influence emanating from the injury in the brain itself. The cases, therefore, have to be treated in respect of this condition much as we should treat a paralysis with wasting due to disease of the spinal cord.

The same mode of causation also obtains for those other nutritrive changes which go to make up the pathological condition known as 'late rigidity.' And,

as a consequence, these are best warded off by passive movements and frictions of the palsied limbs, combined with careful faradization of the muscles. By these means we are most likely to exert a healthy influence upon related parts of the spinal cord, and thus to do the best in our power to check the progress of degenerative changes in this centre, as well as to arrest those further alterations in nerves, muscles, and joints which tend to occur as concomitant events.

With regard to loss or diminution of sensibility on the paralysed side, this is usually a condition requiring no very direct or special attention. tendency is for this impairment gradually to diminish of its own accord; or at all events the same measures as suffice for the mitigation of the defective power of movement suffice to ensure the diminution of any existing sensory impairment. Occasionally, however, it happens that patients complain very much of subjective feelings of 'coldness' or of disagreeable 'numbness' on the paralysed side long after very decided improvement has taken place in motor power. Or, as you know, this feeling of unilateral numbness may exist almost alone from the first. And in these latter cases I have often obtained marked relief, even where the symptoms have been of long standing, by moderate doses of iodide of

potassium alone, or by a mixture of the iodide and the bromide. In the former class of cases, however, I have found the symptoms far more obstinate—often resisting the influence of remedies, and yielding only in the course of months or years to the ameliorating influence of time.

Though we have to trust, gentlemen, greatly to natural processes in the treatment of a patient who is suffering from the destructive effects produced by cerebral hæmorrhage or by cerebral softening, yet I hope you will have learned that by watchful care and attention we are able to do very much to favour the action of these processes. By strict attention to any unusual symptoms as they arise, by looking well after the patient's general health, by a judicious rousing of the otherwise dormant functions of nerve and muscle, and generally by preserving our patient from all influences and measures which might prove actually harmful, we may do much to facilitate the curative processes in all cases of paralysis from brain disease.



## ILLUSTRATIONS.

TOPOGRAPHICAL ANATOMY OF CEREBRAL CON-VOLUTIONS — DISTRIBUTION OF CEREBRAL VESSELS.

### FIG. I.

## Profile View of the Brain of a Bushwoman described and figured by Prof. Marshall. (After HEATH.)

C Central Lobe, or Island of Reil.

F Frontal Lobe.

P Parietal Lobe.

O Occipital Lobe.

T Temporo-sphenoidal Lobe.

d d Fissure of Rolando.

c c Fissure of Sylvius, anterior division.

e e Fissure of Sylvius, posterior division.

ff Parallel Fissure.

g g Inferior Temporal Fissure.

I Lower, 2 Middle, and 3 Upper Frontal Convolution.

4 Ascending Frontal, and 5 Ascending Parietal Convolution.

5' 5' 5' Lobule of Ascending Parietal Convolution.

4"-5" Supra-marginal Convolution.

A A A Lobule of Supra-marginal Convolution.

666 Angular Convolution.

777 Upper, 888 Middle, 999 Lower External Temporal Convolution.

10 Upper, 11 Middle, and 12 Lower Occipital Convolution.

α First, β Second, γ Third, δ Fourth External Connecting Convolution.

### FIG. 2.

## Profile View of Right Hemisphere, from the Brain of a Scotchman, æt. 23. (After Turner.)

Fr Fr Frontal Lobe.

Par Parietal Lobe.

Oc Occipital Lobe.

TS Temporo-sphenoidal Lobe.

R R Fissure of Rolando.

S S Horizontal.

'S'S Ascending Limb of the Sylvian Fissure.

I P Intra-parietal, and P P Parallel Fissure.

I I I Lower, 2 2 2 Middle, 3 3 3 Upper Frontal Convolution.

444 Ascending Frontal, 555 Ascending Parietal Convolution.

A Supra-marginal Lobule.

5' Outer part of Postero-parietal Lobule. 66 Angular Convolution.

77 Superior, 888 Middle, 999 Inferior Temporo-sphenoidal Convolution.

10 Superior, 11 Middle, 12 Inferior Occipital Convolution.

α βγδ First, Second, Third, and Fourth Annectent Convolution.

FIG. I.

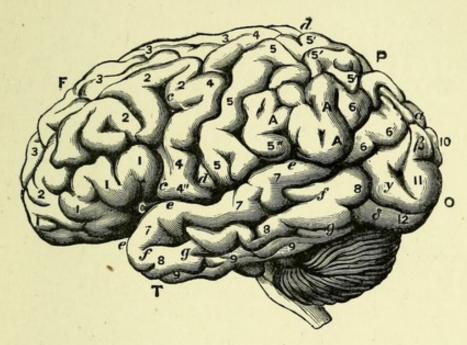
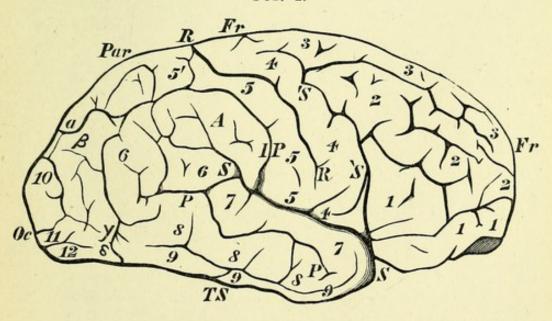


FIG. 2.



### FIG. 3.

### Vertex View of the Brain of a Bushwoman.

(After MARSHALL and HEATH.)

F Frontal Lobe.

P Parietal Lobe.

O Occipital Lobe.

dd Fissure of Rolando.

h Parieto-occipital Fissure.

A A Supra-marginal Lobule.

2 Middle, and 3 Upper Frontal Convolution.

4 Ascending Frontal, and 5 Ascending Parietal Convolution.

5' 5' Lobule of Ascending Parietal Convolution.

66 Angular Convolution.

10 10 Upper, and 11 11 Lower Occipital Convolution.

α α First, and β Second Annectent Convolution.

### FIG. 4.

### Vertex View of the Brain of a Scotchman, æt. 23.

(After TURNER.)

Fr Frontal Lobe.

Par Parietal Lobe.

Oc Occipital Lobe.

SF Supero-frontal, IF Infero-frontal Fissure.

R Fissure of Rolando.

IP Intra-parietal, and PO Parieto-occipital Fissure.

S Horizontal, and S' Ascending Limb of the Sylvian Fissure.

A Supra-marginal Lobule.

1 1 Inferior, 22 Middle, and 333 Superior Frontal Convolution.

44 Ascending Frontal, and 55 Ascending Parietal Convolution.

5' Outer, and 5" Inner part of Postero-parietal Lobule.

6 6 Angular Convolution. 10 Superior Occipital Convolution.

α a First, and β Second Annectent Convolution.

FIG. 3.

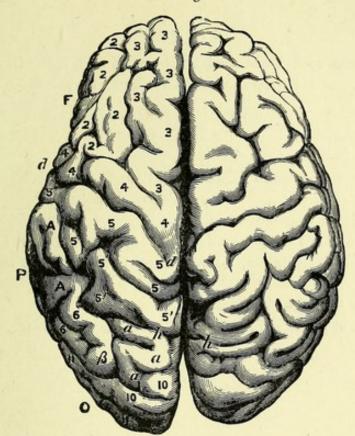
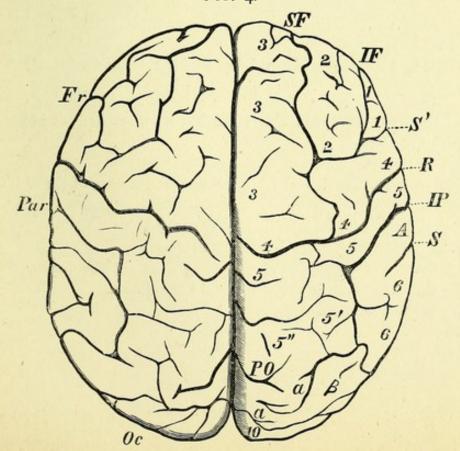


Fig. 4.



### FIG. 5.

### Inner Face and Tentorial Surface of the Left Cerebral Hemisphere. (After TURNER.)

Fr Frontal Lobe.

Par Parietal Lobe.

Oc Occipital Lobe.

TS Temporo-sphenoidal Lobe.

PO Internal Parieto-occipital Fissure.

iii Calloso-marginal, and 11 Calcarine Fissure.

m m Dentate Fissure.

nn Collateral Fissure.

17 17 17 Marginal Convolution.

18 18 Convolution of Corpus Callosum. 18' Quadrilateral Lobule.

19 19 Uncinate Convolution, of which 19' is the crotchet, or recurved part.

25 Cuneus, or Occipital Lobule.

99 Inner face of Inferior Temporo-sphenoidal Convolution.

### Fig. 6.

### View of the Orbital Lobule and of the Island of Reil.

(After TURNER.)

Most of the Temporo-sphenoidal Lobe has been removed in order to display the Island.

O Olfactory Sulcus.

TR Triradiate Sulcus.

1" Posterior, 1" Internal, and 1" External Convolutions of the Orbital Lobule.

C Island of Reil, with its radiating Convolutions.

11 Under Surface of Inferior Frontal Convolution.

4 Under Surface of Lower end of Ascending Frontal Convolution.

5 Under Surface of Lower end of Parietal Convolution.

17 Marginal Convolution.



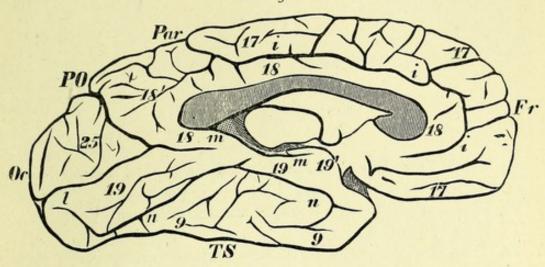
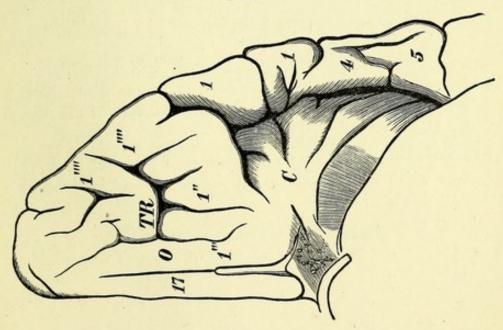


FIG. 6.



### FIG. 7.

### View of the Brain from below.

### (After ECKER and DURET.)

F1 Gyrus Rectus.	
F2 Middle Frontal Convolution. F3 Inferior Frontal Co	nvolution.
f4 Sulcus Olfactorius. f5 Sulcus Orbitalis.	
72 Second or Middle Temporo-sphenoidal Convolution.	
73 Third or Inferior ,, ,, ,,	
T4 Gyrus Occipito-temporalis lateralis (Lobulus fusiformis).	
T5 ,, ,, medialis (Lobulus lingualis).	
t4 Sulcus Occipito-temporalis inferior.	
t3 Sulcus Temporo-sphenoidal is inferior.	
t2 ,, ,, medialis.	
po Parieto-occipital Fissure. oc Calcarine Fissure.	
H Gyrus Hippocampi. U Gyrus Uncinatus.	
Ch Optic Chiasma. cc Corpora Albicantia	
KK Crura Cerebri. C Corpus Callosum.	

### DISTRIBUTION OF VESSELS.

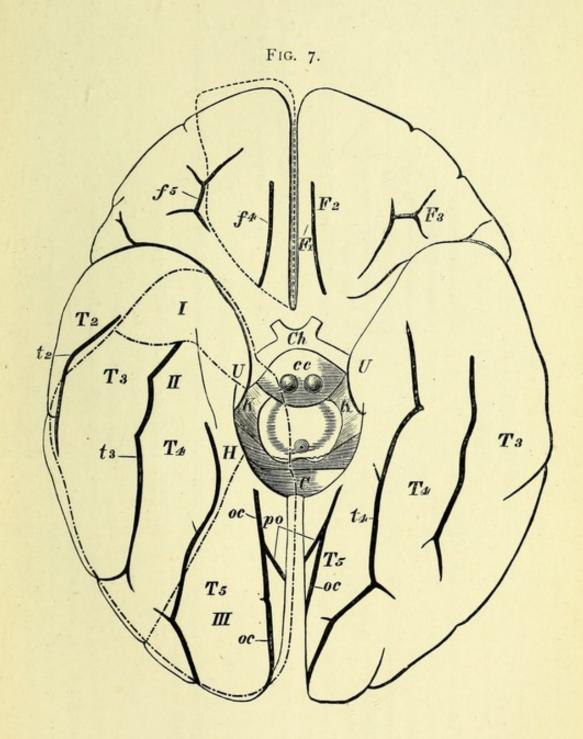
The region bounded by the line (----) represents the territory over which the *Internal and Inferior Frontal Branches* of the Anterior Cerebral Artery are distributed.

The regions bounded by the line (-----) represent the territories over which the branches of the Posterior Cerebral Artery are distributed.

I. Is the region of the Anterior Temporal Artery.

II. ,, ,, Posterior Temporal Artery.

III. ,, ,, Occipital Artery.



medialis (Lobulus lingualis.)

FtG. 8.

## Inner Surface of Right Hemisphere.

(After ECKER and DURET.)

Gf Gyrus Fornicatus. C C Corpus Callosum, longitudinally divided.

II Gyrus Hippocampi.

U Uncinate Gyrus.

F Median aspect of First Frontal Convolution.

c Terminal portion of the Sulcus Centralis.

c m Sulcus Calloso-marginalis.

h Sulcus Hippocampi.

B Posterior Central Convolution.

Oz Cuneus.

A Anterior Central Convolution.

P' Præcuneus.

po Parieto-occipital Fissure.

D Gyrus descendens.

o Sulcus Occipitalis transversus. oc Calcarine Fissure: oc' Superior, oc" Inferior ramus of the same.

T4 Gyrus Occipito-temporalis lateralis (Lobulus fusiformis.)
 T5 Gyrus ,, medialis (Lobulus lingualis.)

DISTRIBUTION OF VESSELS.

The regions bounded by the line (----) represent the territories over which the branches of the Anterior Cerebral Artery are distributed.

I. Is the territory of the Interior and Anterior Frontal Artery.

Internal and Posterior ,, Internal and Middle

III.

The regions bounded by the line (-----) represent the territories over which the branches of the Posterior Cerebral Artery are distributed.

II. Is the territory of the Posterior Temporal Artery.

Occipital Artery.

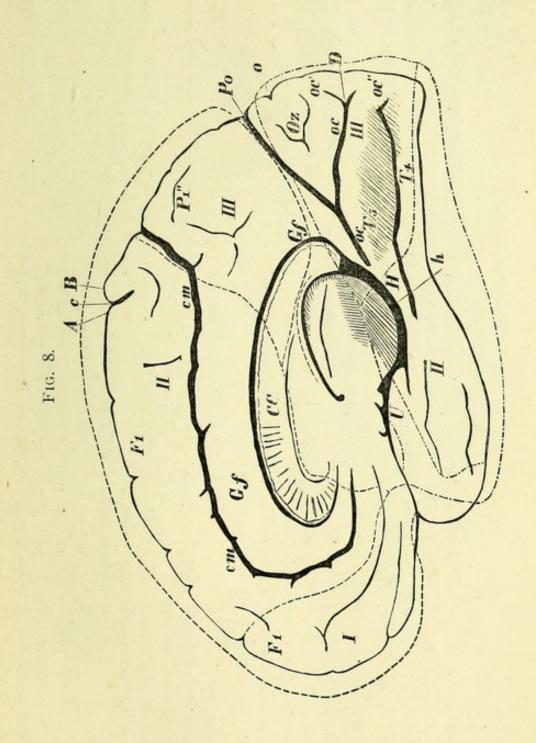


FIG. 9.

# Outer Surface of the Left Hemisphere.

(After ECKER and DURET.)

F3 Inferior Frontal Convolution. T Temporo-sphenoidal Lobe. Pr Superior Parietal Lobule. c Sulcus Centralis. FI Superior, F2 Middle, S' Horizontal, S" Ascending ramus of the same. f3 Sulcus Præcentralis. O Occipital Lobe. A Anterior, B Posterior Central Convolution. P Parietal Lobe. fi Superior, f2 Inferior Frontal Sulcus. S Fissure of Sylvius. F Frontal Lobe.

cm Termination of the Calloso-marginal Fissure. P2 Inferior Parietal Lobule: viz.  $P_2$  Gyrus Supra-marginalis.  $P_2$  Gyrus Angularis.

o2 Sulcus Occipitalis longitudinalis inferior. po Parieto-occipital Fissure. OI First, O2 Second, O3 Third Occipital Convolution. ip Sulcus Intra-parietalis.

T2 Second, T3 Third Temporo-sphenoidal Convolution. or Sulcus Occipitalis transversus. TI First,

t2 Second Temporo-sphenoidal Fissure. ti First,

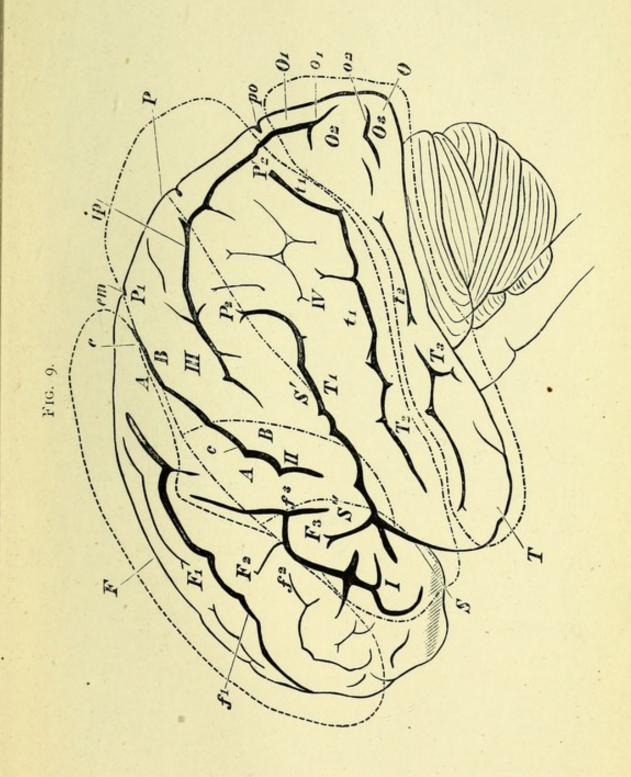
DISTRIBUTION OF VESSELS.

The region bounded by the line (----) represents the territory over which branches of the Anterior Cerebral The anterior regions bounded by the line (-----) represent the territories over which branches of the Artery are distributed.

I. Is the region of the External and Inferior Frontal Artery. Middle Cerebral Artery are distributed.

Parieto-sphenoidal Artery. Posterior Farietal Artery. Anterior Parietal Artery. IV. II.

The posterior and inferior region bounded by the line (-----) represents the territory over which branches of the Posterior Cerebral Artery are distributed.





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