The pathology, diagnosis and treatment of intra-cranial growths.

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THE

PATHOLOGY, DIAGNOSIS AND TREATMENT

OF

INTRA-CRANIAL GROWTHS.

BY

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Color che vanno
Con cosa in capo non di lor saputa.
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THE Trustees of the Fiske Fund, at the annual meeting of the Rhode Island Medical Society, held at Providence, 12 June, 1890, announced that they had awarded a premium of three hundred dollars to an Essay on "The Pathology, Diagnosis and Treatment of Intra-cranial Growths," bearing the motto —

"Color che vanno
Con cosa in capo non di lor saputa."

The author was found to be PHILIP COOMBS KNAPP, M.D., of Boston, Mass.

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INTRODUCTION.

SINCE this essay was presented for the Fiske prize, in May, 1890, I have been enabled, by the kindness of the Trustees of the Fiske Fund, to make a thorough revision of it. I have not, however, departed from the original plan. At that time I decided to present a collection of new cases, although many of them were defective and did not present characteristic features, rather than to collect more typical published cases from the immense number now reported. The essay is therefore based on the records of forty cases with autopsies. Of these thirty-eight were patients at the Boston City Hospital, and I wish here to thank my colleagues at the hospital for their kindness in permitting me to make use of this material, without which I could not have written the essay. My thanks are especially due to Dr. S. G. Webber, who has aided me with notes and sketches. Some of these cases came under my own observation, and a few of them have already been reported. The greater number, however, have never been published. I have added a case seen by me while interne at the Boston Lunatic Hospital and already reported by Dr. T. W. Fisher, and another case seen in private practice.

Although I have not collected illustrative cases I have of course availed myself freely of the literature of the subject, without undertaking the enormous task of continuing the work of Ladame and Bernhardt. The discussion of special symptomatology, therefore, is based rather upon the literature than upon the data furnished by the few cases reported.

In the chapter on treatment I have departed from the principle above laid down in giving a tolerably complete list of the cases operated upon. I should be glad to have any omissions called to my attention. My thanks are due to Dr. J. T. Eskridge of Denver for his kindness in placing at my disposal the records of an unpublished case.

Boston, May, 1891.

I. ÆTIOLOGY.

By an intra-cranial growth or cerebral tumour we mean any form of new growth within the cavity of the skull. Such a growth may develope primarily from the bones of the skull, the cerebral membranes or blood-vessels, or the brain substance itself, or it may develope in any of these tissues as a metastatic growth secondary to a new growth arising primarily in some other organ. In other cases a morbid growth outside of the cranial cavity may, by direct extension, penetrate the cavity, and thus become intra-cranial.

The ætiology of such growths is as obscure as is the ætiology of tumours elsewhere. The brain from its great vascularity affords an excellent seat for morbid growths, particularly if they be due to infection from micro-organisms; yet the determining cause of their developement within the skull is seldom clear. Certain factors, such as sex, age, heredity and trauma have some influence upon their developement, and these may be briefly considered.

Sex. — The male sex is much more disposed to cerebral tumours, as the following table will show: —

TABLE I.

	Men.	Women.
Personal cases	28	12
Bernhardt [1] and Ladame [2]	440	210
Seppilli [3]	89	40
Lebert [3]	51	38
Friedreich [3]	28	15
Hasse [3]	9	5

It is probable that the different writers above cited have in some instances drawn their figures from the same cases, but

it is manifest that fully two-thirds of the collected cases have occurred in males. Gowers[4] states that tubercle and glioma are even commoner in males than other forms of tumour, and adds that the preponderance in favour of males is seen in children as well as in adults. In fact after the age of fifty the proportion between the sexes is more nearly equal. Other writers have sought to explain this greater frequency of cerebral tumours in males to greater cerebral activity and to the fact that men are much more exposed to certain noxious influences, such as syphilis and trauma, which might favour the developement of new growths.

Age. — The forty cases collected from the hospital reports show the following results: —

TABLE II.

Under 10	1
11 to 20	2
21 to 30	10
31 to 40	9
41 to 50	7
51 to 60	7
Age not stated	4

It must be borne in mind, however, that very few children are received at the Boston City Hospital except those suffering from acute infectious diseases and surgical affections. Gowers' statistics show that one-third of the cases occur before the age of twenty, two-fifths from twenty to forty, and one-fifth from forty to sixty. Steffen[5] cites a case in a four weeks' old infant, and he and Starr[6] have found, in children and youths up to twenty years of age, that cases are commoner before the age of eight than after it.

The frequency with which the different varieties of tumours occur is markedly influenced by age. The majority of cerebral tumours in children are tubercular, and tubercle is not

common except in the young. Glioma occurs chiefly in young adults. Sarcoma and cancer usually develope in the brain, as elsewhere, after the period of maturity. Gumma also is seldom seen except in adults. Cerebral lesions are infrequent in cases of hereditary syphilis, and, when they do occur, they are more commonly diffuse, only rarely taking the form of isolated tumours.

Heredity. — Heredity seems to be of little influence upon the developement of new growths, although it may have some effect upon their nature, especially in the case of cancer. It is still a matter of doubt whether the "invalid brain," subject to some neuropathic taint, is more susceptible to the still obscure causes that tend to the developement of a tumour.

Trauma. — The laity are prone to attribute every form of new growth in any part of the body to injury, but in many cases the injury, on inquiry, proves to be comparatively slight or to have preceded the appearance of the growth by so long a period as to render its potency as a cause rather doubtful. It is, however, beyond question that injury may act as the exciting cause of a tumour, even of an infectious granuloma, for growths have been found arising from the scar of the injury. I have myself seen a case of probable tubercle of the cerebellum, in a patient still living, where the symptoms dated quite definitely from a fall on the head.

Other ætiological factors are of less importance. In some cases the symptoms develope after great nervous strain, debilitating influences, or alcoholic or other excesses, but it is not easy to determine how far such influences may act as a true cause of the growth.

II. PATHOLOGICAL ANATOMY.[7, 8]

INTRA-CRANIAL growths may develope from the skull, from the membranes of the brain (including their prolongations into the brain, such as the falx or the choroid plexus), and from the brain itself.

Tumours developing from the skull itself are distinctly rare. Such growths are usually of the connective tissue type, sarcoma being the commonest. Exostoses of the skull may occasionally develope without causing any symptoms, the brain gradually accustoming itself to the slow growth. Malignant growths are more apt to cause external symptoms as well, than to give rise merely to cerebral symptoms due to the pressure of the growth.

Tumours of the dura are also most frequently of the connective tissue type, although tubercular and syphilitic thickenings are occasionally seen; they may sometimes attain such a size as to cause remote symptoms from pressure. The commonest growth, however, is the sarcoma, which usually forms a flat, rather widely-extended growth, sometimes as large as an apple, spreading over the surface of the brain. In some cases these growths show proliferating granulations, and they may even perforate the skull (fungus hæmatodes). In other cases they grow inwards and give rise to more pronounced cerebral symptoms. The variety of sarcoma called endothelioma not infrequently has its seat in the dura. Bony and cartilaginous growths, when they do not develope from the bone itself, usually develope from the dura or falx. Cancer, too, is not uncommon in the dura as a metastatic growth.

The pia is the favourite seat for new growths of an infectious type. Syphilitic growths almost invariably, and tubercular growths very commonly, have their starting-point in the pia, and follow its folds, or some blood-vessel, into the interior of the brain. Cancer not uncommonly developes from the plexuses. Cholesteatoma is most frequent in the membranes of the base. Other forms of neoplasms may also arise from the pia. All these growths, as they extend, may either exert marked pressure upon the brain beneath them, or actually penetrate into the brain substance.

The tumours which develope primarily in the nervous tissue are the glioma and the neuroglioma ganglionare. Almost all the other forms of neoplasm, however, may occur within the nervous tissue, and it is frequently difficult to determine whether a given tumour has originally developed in the brain substance or has arisen from a fold of the pia, a penetrating blood-vessel, the lining of the ventricle, or from the choroid plexus, and invaded the brain later. In the consideration of the individual forms of new growths further details as to the seat of origin and the appearance of the different growths will be given.

The accompanying tables show clearly the nature of the various new growths found in the brain, the relative frequency of their occurrence, and the frequency with which they are found in the different regions of the brain. The first table is compiled from Bernhardt's treatise,[1] the second is taken from Birch-Hirschfeld[7] and undoubtedly includes many of the same cases, the third is made up from three hundred cases in children collected by Starr[6] who includes some of Bernhardt's cases, and the fourth is made up from the cases I have collected from the hospital reports. In a large percentage of Bernhardt's and of my own cases the nature of the growth is not given.

TABLE III (Bernhardt).

Location of Growth.	Granulomata.		Connective Tissue Tumours.																Epithelial Tumours.			Cysts.		Parasites.		Not Stated.	Grand Total.
	Tubercle.	Gumma.	Sarcoma.	Glioma.	Fibro-sarcoma.	Myxo-sarcoma.	Cysto-sarcoma.	Glio-sarcoma.	Myxo-glioma.	Fibro-glioma.	Fibroma.	Osteoma.	Osteo-fibroma.	Enchondroma.	Myxoma.	Lipoma.	Psammoma.	Neuroma.	Carcinoma.	Papilloma.	Cholesteatoma.	Cyst.	Dermoid Cysts.	Cysticerci.	Echinococci.		
Cortex	19	6	7	7	2	...	...	1	...	1	...	...	...	...	...	...	...	...	2	...	...	...	...	2	1	9	57
Lobes	6	2	18	15	...	...	. .	4	3	...	...	3	...	...	...	...	2	...	6	...	1	8	...	3	15	38	124
Optico-striate region	3	...	4	7	...	1	...	...	1	...	...	...	1	...	...	...	...	...	1	...	1	...	...	...	...	7	26
Crus cerebri	1	...	...	1	...	. .	...	1	...	...	...	...	...	...	...	...	...	...	. .	...	...	...	...	...	...	....	3
Corpora quadrigemina	2	...	1	2	...	...	...	...	...	...	...	. .	...	...	...	1	. .	...	...	...	...	...	...	...	...	5	11
Pineal gland	. .	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	1	...	...	1	...	...	...	1	3
Pons	11	3	2	3	...	1	...	2	...	...	...	...	...	...	...	...	...	...	2	...	...	1	...	...	...	5	30
Cerebellum	16	1	11	10	...	...	1	3	2	...	1	1	...	...	...	...	...	...	3	1	...	7	2	1	...	30	90
Medulla	2	1	1	4	1	...	...	1	...	. .	...	...	...	...	1	...	. .	...	1	2	...	...	...	1	...	6	21
Hypophysis	...	...	2	...	...	...	...	...	...	...	...	...	...	..	...	...	...	...	1	...	...	...	...	...	...	2	5
Base	...	1	4	...	...	...	1	1	...	...	1	..	...	1	...	...	2	. .	...	...	...	...	...	...	...	28	39
Cranial nerves	..	...	2	...	...	...	...	...	...	..	...	...	...	...	..	...	...	3	...	...	...	...	...	...	...	....	5
Multiple	33	1	6	3	. .	...	1	...	...	..	...	...	1	...	...	1	...	...	1	...	...	...	...	8	2	14	71
Total	93	15	58	52	3	2	3	13	6	1	2	4	2	1	1	2	4	3	18	3	2	17	2	15	18	145	485

TABLE IV (Birch-Hirschfeld).

Location of Growth.	Tubercle.	Gumma.	Sarcoma.	Glioma.	Osteoma.	Myxoma.	Lipoma.	Cholesteatoma.	Total.
Cortex	31	9	7	12	1	....	....	....	60
Lobes	12	6	21	22	2	....	....	1	64
Basal ganglia	14	2	6	11	1	...	....	1	35
Corpora quadrigemina	2	....	1	4	....	....	1	....	8
Hypophysis	....	3	3	....	....	....	....	....	6
Pineal gland	....	..	4	....	....	...	....	....	4
Pons	19	3	5	4	....	...	....	....	31
Cerebellum	41	3	20	17	1	6	....	....	88
Medulla	7	1	3	6	....	1	....	....	18
Total	126	27	70	76	5	7	1	2	314

TABLE V (Starr) Children.

Location of Growth.	Tubercle.	Glioma.	Sarcoma.	Glio-sarcoma.	Cyst.	Carcinoma.	Gumma.	Not Stated.	Total.
I. Cortex cerebri	13	6	1	....	....	1	....	...	21
II. Centrum ovale	6	1	5	1	15	1	1	5	35
III. Cerebral axis.									
1. Basal ganglia and lateral ventricle	14	3	5	....	1	1	....	3	27
2. Corpora quadrigemina and crura cerebri	16	1	3	....	....	....	....	1	21
3. Pons	19	10	5	2	1	....	....	1	38
4. Medulla	2	....	....	....	1	2	....	1	6
5. Fourth ventricle	1	1	1	....	...	1	....	1	5
6. Base	....	....	1	1	1	1	....	4	8
IV. Cerebellum	47	15	10	1	9	3	....	11	96
V. Multiple	34	....	3	....	2	....	....	3	42
Total	152	37	34	5	30	10	1	30	299

TABLE VI (Personal Cases).

Location of Growth.	Tubercle.	Gumma.	Sarcoma.	Myxo-sarcoma.	Fibro sarcoma.	Glioma.	Enchondroma (?)	Not Stated.	Total.
Cortex	3	....	1	....	....	1	1	4	10
Lobes	1	2	....	....	....	1	....	....	4
Corpus callosum	....	....	1	....	....	....	....	1	2
Optico-striate region	1	1	...	1	1	....	....	1	5
Corpora quadrigemina	....	....	2[1]	...	....	....	....	....	2
Pons	...	1	....	....	....	....	....	....	1
Cerebellum	2	...	1	...	....	....	....	2	5
Hypophysis	....	....	1	....	....	....	....	....	1
Base	....	2	....	...	...	1	....	2	5
Multiple	2	2	....	....	...	1	....	....	5
Total	9	8	6	1	1	4	1	10	40

[1] One of these also contained cholesteatomatous nodules.

From these tables it will be seen that tubercle is the commonest form of intra-cranial growth; next come the two forms of connective-tissue growths, sarcoma and glioma, with their varieties; and next, with a long interval, gumma, cancer, and parasitic cysts. Other forms of new growth are exceptional.

Tumours of the brain do not differ anatomically from tumours elsewhere, except that glioma and neuroglioma ganglionare are peculiar to the central nervous system. It will be well, however, to note their characteristic features, dwelling upon any peculiarities incidental to their situation, before noting the changes produced by them in the brain itself and their pathological action.

1. Infectious Granulomata.

The infectious granulomata owe their origin to the presence of some form of schizomycetes. In the majority of cases this micro-organism has been positively determined. In the case of syphilis the precise micro-organism is not yet absolutely accepted, but Lustgarten's bacillus is regarded as the possible cause of infection, and no one doubts that the disease is due to some micro-organism. Whether the morbid processes are the direct result of an infectious irritation, or are due to a lesion of tissue caused by the infectious irritation is still uncertain. It is probable that the micro-organisms in their growth give rise to ptomaïnes, which in their turn cause morbid processes in the tissues.

Although anatomically the invasion of the brain by these schizomycetæ always leads to similar processes, nevertheless we must distinguish clinically between acute processes which give rise to more or less extensive lesions of an inflammatory character, where the new formations are of small size, but multiple; and the more chronic processes, where the new formations are present as single large masses, more closely resembling tumours. This distinction is most marked with tubercle and syphilis, and it will be considered more fully in the description of such growths.

The infectious granulomata are usually secondary to an infectious process elsewhere. They are locally progressive growths, and grow from the periphery outwards. The central portion is the oldest, and contains few blood-vessels, and therefore it is prone to degenerate, the degeneration usually assuming the cheesy form. The forms most commonly met with in the brain are tubercular or syphilitic. Other forms occur only very rarely, and none are to be found in the cases collected in any of the tables given above.

Tubercle. — As the tables show, tubercle is by far the commonest form of brain tumour, being found most frequently in the cortex, the pons, or the cerebellum. As I have already said, it is more common in children and in young adults. It is due to an infection of the brain with the bacillus tuberculosis of Koch, the morbid process in the brain being usually, if not invariably, secondary to some tubercular process elsewhere.

Anatomically speaking, we cannot make any distinction in the forms of cerebral tuberculosis, but clinically, of course, such a distinction is apparent. The bacilli enter the brain either through the blood-vessels or through the lymphatics; in the latter case the meninges are first affected. In tubercular meningitis, for instance, we see a collection of round cells and miliary tubercles about the vessels of the pia, following these vessels into the brain. In a part of the cases the infection is general, numerous miliary nodules form in the meninges, an active inflammation is set up, and the patient succumbs before any one of these nodules attains any size. In other cases the process is slower, and some of the nodules attain considerable size. Among the cases I have collected will be found one or two of tubercular meningitis, where such nodules were found. It is, however, not easy to distinguish such cases from others where the solitary nodule is primary, and leads to a secondary general infection.

In other cases we have a different clinical picture, corresponding to that seen with other forms of cerebral tumour. Here the process is more chronic. One or two regions of the brain are infected with tubercle bacilli, the bacilli being probably not very numerous or of very rapid growth. These bacilli enter only one or two small branches of one of the meningeal arteries. Here tubercular nodules are formed, which gradually increase in size and penetrate more or less deeply into the brain substance. The growth of these nodules is slow, and

they may attain a considerable size, a diameter of three or four centimetres or more. These are the so-called "solitary" tubercles, an ill-chosen name, for, as the tables show, such growths are often multiple, and the individual tumour is in many cases formed by the agglomeration of several smaller nodules. Tubercular disease of the dura is rare, except in combination with similar disease in the pia or the skull. These "solitary" growths arise most commonly from the pia, or some penetrating fold thereof; and, when they occur within the brain apart from the pia, they probably spring from a blood-vessel, through which the bacilli have been carried to the affected spot.

These growths are usually irregular in shape, with various projections, indicating the agglomeration of several distinct nodules. The surrounding brain-substance may be softened or sclerosed about the growth, but sometimes the periphery becomes fibrous, forming a capsule to the tumour. In long-standing cases this capsule may become calcified. On section the growth is found to have a grayish-red, translucent periphery and a yellowish centre, which is sometimes cheesy, and occasionally shows a concentric marking. The growth extends along the lymphatic sheaths, and, when blood-vessels are met with, it causes thrombosis. Sometimes, however, the growth may penetrate a vessel, and the blood may become re-infected with bacilli, and thus give rise to a fresh and more acute infection of the brain, a secondary tubercular meningitis.

Under the microscope we find in the periphery of these growths an abundance of newly-formed round cells, nuclei, giant cells with many nuclei, and epithelioid cells. Here, too, may usually be found a certain number of bacilli. The exact origin of these cells, whether from endothelium in the serous cavities, lymph cavities, or adventitia, or from connective tissue or lymph corpuscles is still uncertain. Occas-

ionally we see a reticular network between the cells, the remains of the destroyed connective tissue. The growth, as I have said, contains very few vessels, and the centre, deprived of blood, undergoes coagulation necrosis and cheesy degeneration. Here we find only a fatty-granular homogeneous detritus, with, perhaps, an occasional cell still preserved, and, rarely, a few bacilli.

Gumma. — What has been said of cerebral tuberculosis holds true in large part of cerebral syphilis. The bacilli (?) enter through the blood-vessels and give rise to inflammatory processes and cell-proliferation in the meninges. The favourite seat of this process is at the base of the brain, especially in the loose meshes of connective tissue about the optic chiasma.[9] The products of such an inflammation often form dense masses, composed largely of connective tissue, with some round-cell infiltration, and give rise to symptoms resembling those caused by a tumour. One or two such cases will be cited later. Anatomically such a growth is to be regarded as a meningitis, and not as a tumour; but the process is histologically the same in both cases, and the distinction drawn in tuberculosis cannot be so well maintained. All syphilitic processes in the brain are gummatous; sometimes the process is limited, and the inflammatory products attain a considerable bulk in one spot, — the gumma or syphilitic tumour, as distinct from the syphilitic inflammation.

The discrete tumour or gumma is comparatively rare, much rarer than gummatous meningitis. Such growths are found most commonly in the cortex or the pons, and usually develope from the meninges, starting perhaps from a local meningitis; in rare instances they seem to arise from the vessels. Gumma is rarely seen in children. Starr,[6] in 300 cases, found but one, and that in a youth of eighteen; and Rumpf[10] has collected only two. Cerebral lesions are

rare in hereditary syphilis, and gumma even rarer than other forms. These growths are irregular or wedge-shaped, and may attain considerable size. They are usually surrounded by softened brain tissue, but in some cases they are infiltrated; capsule formation is rare. On section of a tumour of good size and average duration we find a reddish-gray, translucent periphery, the newest part of the tumour, and within this one or more dry, yellowish centres of caseation. The growth contains very few vessels, and the vessels in and near it show periarteritis and endarteritis. In a later stage, perhaps as a result of treatment, there is a considerable developement of connective tissue in the tumour (a characteristic change in syphilitic growths), and the tumour becomes hard, shrunken, and fibrous, — an indurated cicatrix.

Under the microscope the grayish-red periphery is found to be made up of small round cells, spider cells, spindle cells, and even giant cells, with the remains of the elements of the original tissue. A characteristic feature of syphilitic growths is the presence of fibrous tissue, mixed with the round cells. The cell formation is primary, and leads to the formation of fibrous tissue as a sort of cicatrix. The fibrous tissue occasionally has a somewhat alveolar arrangement, enclosing the cells in its meshes; this is commoner in the growths that develope from softer tissues. The cheesy portions are more irregular in their distribution than are those of tuberculosis, and they consist of a fine granular detritus, shrivelled nuclei, and granular cells. The distinction between gumma and tubercle is not always easy. The presence of other syphilitic lesions, especially endarteritis, the irregular shape and the irregular distribution of the cheesy nodules, the characteristic developement of fibrous tissue, and the absence of the bacillus of tubercle will usually render the distinction possible.

Actinomyces. — In rare cases actinomycosis extends from

the face and neck, its favourite seat, through the occipital foramen into the brain, leading to a diffuse purulent inflammation of the pia, with granulating nodules, or, in some cases, to abscess formation. Bollinger[8] reports a case, as yet unique, where a granulation tumour the size of a hazel-nut was found in the third ventricle, and where no other traces of actinomycosis could be found. The growth was, like other actinomycotic tumours, made up of connective tissue and round cells, with the peculiar nodules of the specific micro-organism.

Other infectious granulomata play no part in the consideration of intra-cranial growths. Ziegler[6] states that in leprosy and glanders the brain may become involved, but, in the extremely rare cases where this happens, the process is diffuse and does not lead to tumour-formation. Mycosis fungoides and rhinoscleroma never, so far as is known, attack the brain.

2. Connective Tissue Tumours.

Second only to the infectious granulomata in importance and frequency come the connective tissue tumours, developing from the tissues of the mesoblast. Of these there are many varieties and also mixed forms. They may develope primarily in the brain, or secondarily from metastases within the skull. Among them we find, next in frequency to tubercle, sarcoma and glioma, the latter being in the strictest sense a brain tumour. Other forms are rare, but require a word of description.

Glioma. — The glioma is peculiarly a "brain" tumour, for it developes from the cells which form the foundation of the central nervous system, from the neuroglia, and it is found only in the central nervous system or in its prolongation into the retina. It is usually a solitary growth, and is found most commonly in the cerebrum, and next, although

less frequently, in the cerebellum and the brain stem. It never forms metastases and is probably malignant only from its situation, although, when removed, it has a tendency to recur. It may attain the largest size of any of the intra-cranial growths, sometimes having a diameter of eight or ten centimetres and a weight of a hundred or a hundred and fifty grammes. Developing, as I have said, from the neuroglia, it is primarily sub-pial, and involves the membranes, if at all, only secondarily during its growth. Ordinarily the external form of the brain is unchanged, and only by touch or by some change in colour, or perhaps by some swelling do we recognize that there is a growth beneath. The growth is of varying density, usually not differing very materially in density from the brain substance. In rare cases the tumour is distinct from the brain substance, but usually it is hard to distinguish the boundary between the tumour and normal brain tissue. Occasionally the brain about the tumour is softened, and the softening may go on to the formation of cysts. On section the growth may closely resemble either pale or hyperæmic gray matter, but more commonly it is gray or grayish-red, somewhat yellowish, and translucent; the section having a variegated appearance with whitish and hæmorrhagic spots. The vessels within the growth are often increased in size and number, and sometimes a part or almost the whole of the growth may be destroyed by a hæmorrhage, so that only by a microscopic examination is it possible to detect the remains of the new growth. Under the microscope the tumour is found to be made up of glia cells with fine fibrous prolongations (Spinnenzellen), and a delicate intercellular fibrous tissue. These cells may vary considerably, both in size and number. Osler[11] has described cells occasionally met with which resemble ganglion cells, and he has also found large spindle-shaped cells and translucent band-like fibres. When the cells are small and few

in number the growth is harder and denser. Sometimes some of the cells are collected in groups; others have several nuclei. The tumour probably grows from the periphery by increase and division of the glia cells. The nerve-fibres and ganglion cells in the parts invaded by the growth finally disappear, but they may resist destruction for a long time. When the pia is invaded it usually shows a marked increase of connective tissue. In some cases of hæmorrhage we see agglomerations of cells. Corpora amylacea also occur.

In some cases the vessels are so greatly developed in the growth as to warrant the name of *teleangiectatic* glioma. When the fibrous tissue is increased in the growth we speak of a *fibro-glioma*. If there is within the tumour a developement of mucous tissue we speak of a *myxo-glioma*. In some cases where the cells are very abundant the tumour may closely resemble a sarcoma; in other cases there is a true proliferation of round or spindle-shaped cells, a *glio-sarcoma*.

Neuroglioma ganglionare. — This is a new growth occasionally seen in the brain, and is always to be referred to some disturbance of developement. The growth may be either an increase of substance in the brain without any sharp contour, or it may exist in the form of a somewhat circumscribed nodule. Like glioma it is found only in the central nervous system. The pia is usually not much altered over the growth, but on section the normal distinction between the cortex and the white matter is lost, and the growth has a uniformly white or grayish-white appearance, with occasional gray spots scattered through it. In consistency it is harder than the brain substance. Under the microscope the growth is found to consist of glia tissue, in structure resembling somewhat the nodules of multiple sclerosis. In addition medullated fibres are found in some portions of the tumour.

In other portions large and small ganglion cells are to be found, either isolated or in groups.

Sarcoma. — Sarcoma is about equal to glioma in frequency, being one of the commonest forms of new growth next to tubercle. It may develope from the dura, from the pia, or from within the brain substance; in the latter case it probably arises from the pial sheath of a blood-vessel. Sarcomata are generally solitary but they may be multiple. They may develope primarily within the cranial cavity, when they are either flat, and fungus-like (if of dural origin) or wedge-shaped, or they may arise as metastases from growths elsewhere, when they form roundish nodules. Wernicke [12] states that they are of slow growth and poor in vessels, but this is not universally the case. The intra-cerebral growths are usually distinct from the brain substance, and the interior of the growth may show hæmorrhages, caseation, cyst-formation, or fatty degeneration. Sarcoma is a tumour of the connective-tissue type, made up of cells, the cells predominating over the intercellular substance. The softer forms are white or grayish, with many cells; the harder forms pass over into the fibromata.

Histologically the sarcoma is made up of cells, the character of the cells giving the growth its name. We find large or small round cells, large or small spindle cells, giant cells, stellate cells, and a mixture of the various forms. The small round cell sarcoma is the most malignant (except the melanotic form), but the question of the malignity of an intra-cerebral growth has not thus far been of much importance. It has, however, recently assumed importance in connection with the surgical treatment of intra-cranial growths. This form of sarcoma shows on section a milky white surface from which a juice exudes. The spindle-cell sarcoma is less malignant, firmer, translucent, and of a gray or yellowish white colour, often containing

considerable fibrous tissue. Sarcomata have a granular, fibrous intercellular substance, and some of them have a structure much like that of a lymph-gland (*lympho-sarcoma*). The spindle-cell and polymorphous types are the commonest forms found in the brain. When the fibrous tissue is well-marked we speak of a *fibro-sarcoma*, a transition form between the sarcoma and the fibroma, which is rare in the brain. In a very few cases the inter-cellular substance is found to be mucous in character — *myxo-sarcoma* or *cylindroma*. I have already stated that cysts are in rare cases found in the growth — *cysto-sarcoma*. In some cases the cells contain pigment — *melano-sarcoma*. In these cases the melanotic deposit in the brain is generally multiple and secondary to a growth elsewhere. Melanotic growths, whether sarcomatous or carcinomatous, are intensely malignant, and, when they occur in the brain, the cerebral symptoms are apt to be subordinated to the symptoms caused by the growth elsewhere. Bramwell [13] figures an exquisite specimen where the brain was studded with numerous melanotic nodules. I have spoken above of the *glio-sarcoma*. I am disposed to believe that in a portion of the reported cases of sarcoma the new growth contained also gliomatous tissue, overlooked by the observer. If this be true, sarcoma should be ranked as third in frequency among cerebral tumours. Some sarcomata are highly vascular, *telangiectatic sarcomata*, while others seem to consist of cells assembled about an enlarged plexus of vessels, *plexiform angio-sarcoma*. In some cases these growths become calcareous, *angiolithic sarcoma*.

Sarcomata of the dura are most commonly of the spindle-cell type. They may be of alveolar structure, with clusters of cells within a connective tissue stroma, the so-called *endothelioma;* these may contain endothelial cells from the lymphatics, and occasionally the remains of a

lymph vessel may be traced in them. At times these dural growths develope calcareous deposits, giving rise to the *psammoma*, which may also arise from purely fibrous growths. Sarcomata of the dura are flat or spongy growths, with extensive roots in the dura; they may grow inwards, compressing the brain beneath, or outwards, perforating the skull at times, as one form of the fungus hæmatodes. The precise nature of endothelioma has been in dispute, some writers confusing it with epithelioma. The histological distinction is by no means easy. Ziegler[8] lays some stress on the presence of lymph-vessels. Birch-Hirschfeld[7] states that "all tumours arising from the connective tissue, in which the endothelium stands in the same relation to the bundles of connective tissue or to the vessels as in physiological connective tissue, are to be regarded as sarcomata or connective tissue tumours; but when the newly-formed endothelium is arranged in the alveoli of a vascular stroma after the manner of the epithelial cells of carcinoma (in distinction from sarcomata, where the vessels are also equally distributed among the cells in the alveoli) we should use the term endothelial cancer." The anatomical distinction between alveolar sarcoma and epithelioma is sometimes, however, almost impossible; and we can distinguish only by determining the genesis of the growth.

Sarcomata of the pia are either soft nodules or flat and more extensive growths. They may in rare cases involve a very large extent of the meninges in an endothelial growth. They develope partly from the adventitia and partly from the endothelium of the membranes. The polymorphous or alveolar endothelial forms are commonest, but other forms of sarcoma, and myxo-sarcoma and angio-sarcoma also occur.

Within the brain sarcomata are usually wedge-shaped, sharply defined, and made up of spindle or polymorphous

cells. Hæmorrhages and softening may occur within the growth. The surrounding brain substance is sometimes softened, and, if the growth be sub-pial, the meninges are inflamed and thickened.

Fibroma. — Fibroma is rarely found in the brain. When it does occur it is usually a rounded growth, dense and hard in character. Under the microscope it is found to consist of fibrous tissue containing a few cells. The transition form, fibro-sarcoma, has already been mentioned. In some instances concretions occur in the growth, as we have seen may happen with sarcoma, and then we have one form of the psammoma. The Pacchionian bodies, so common as hardly to be considered abnormal, are small fibromata. Fibrous thickenings are also seen at times in the epithelium of the ependyma, as a sort of organized thrombus.

Osteoma. — As a rule osteoma, composed of true bony tissue, developes from the skull, either as an exostosis or as a more irregular growth. In some cases it may arise from the dura, the falx, or the tentorium, and a few cases are on record where osteomata developed within the brain substance. Mixed forms, such as *osteo-fibroma* or *osteo-sarcoma* may also occur.

Enchondroma. — Enchondroma is also rare. It usually forms a flattened growth arising from the bones of the skull or from the dura and lying like a plate upon the brain. It is composed of ordinary cartilage. Sometimes the growth takes on in part a bony character, *osteo-enchondroma.*

Myxoma. — The mixed forms of myxoma, myxo-sarcoma and myxo-glioma, have already been mentioned. The true myxoma is much less common. It is a soft, round, fairly well-defined growth, and, under the microscope, shows a pronounced mucous ground-substance, containing mucine, with cells.

Lipoma. — A few cases are on record where fatty tumours,

composed of ordinary fat tissue, have been found in the brain. They are so rare as to possess no importance.

Angioma. — I have already spoken of the teleangiectatic forms of sarcoma and glioma. In a few instances growths made up of enlarged vessels have been found; in most of these cases careful examination has revealed also cells which indicate the gliomatous or sarcomatous nature of the growth.

Neuroma. — Virchow gave this name to a heterotopy of gray or white matter, occasionally seen as a congenital malformation. Such a growth is probably more properly to be regarded as a neuroglioma ganglionare. A few cases of neuromata are on record, similar to neuromata elsewhere, developing within the cranium from the cranial nerves. It is not clear whether these growths are true neuromata, composed of nerve fibres, or the so-called false neuromata, that is, fibromata arising from the nerve sheaths. They may attain considerable size and give rise to symptoms like those of any other intracranial growth.

3. Epithelial Tumours.

Epithelial growths develope from the tissues of the upper and lower layers of the blastoderm, and contain epithelial cells as well as a vascular connective tissue stroma. The type of normal growth to which they approximate is that of glandular organs, but in the brain they generally assume atypical forms. They rank third in importance among the new growths of the brain, and it is probable that they are much less common than was formerly held. The most important form is cancer, but even this is rare, occurring in only about five per cent. of Bernhardt's cases.

Adenoma. — Adenoma is one of the more typical epithelial growths, developing from glandular structures and resembling gland-tissue in its composition. It forms a rather soft spongy growth, which, under the microscope, resembles

acinous or alveolar glands, masses of epithelium in a connective tissue stroma, or tubes which show in the stroma on section cavities lined with epithelium. As these growths develope from gland-tissue it is obvious that they can occur in but one part of the brain, namely, in the hypophysis, which developes from the pharynx, and is really a bit of the pharyngeal tissue enclosed within the skull. The anterior lobe of the hypophysis consists normally of a vascular connective tissue stroma, with follicles filled with epithelium. In this adenoma may develope. Weigert[c] also describes cystic degeneration and hyperplasia of the hypophysis, with the formation of cysts containing colloid material. Such glandular hyperplasia may attain the size of a hen's egg.

Carcinoma. — Cancer of the brain is often secondary, but it may be primary, in which case it usually arises from the meninges. In the cases collected there was no case of cancer, but I found in the hospital records one case of cancer of the vertebrae with myelitis, where a secondary nodule had formed in the skull, penetrating the cranial cavity for a depth of two millimetres. There were no cerebral symptoms, the membranes and brain substance were healthy, and therefore I have not reported it. Cancer may arise, as this case shows, from the bones of the skull, when it usually perforates the skull, giving rise to one form of fungus hæmatodes. Cancerous growths of the dura are usually secondary, appearing as rather soft roundish nodules, usually multiple. When cancer developes primarily from the pia it is very apt to take its origin at the base, from the epithelium covering the choroid plexus, and to penetrate into the ventricles. Less frequently it arises from the epithelium of the ependyma. The stroma not uncommonly grows in projecting papillae, hence the name formerly used, of *papilloma*. The growths may, in such cases, attain a considerable size, and are usually of a soft, spongy consistency, often containing hæmorrhages

and being vascular. In some cases they assume a colloid character. Whether cancer may develope primarily elsewhere in the brain is still uncertain; it may possibly do so in the hypophysis or in the transverse fissure.

Cancer may develope in the brain substance as multiple secondary nodules from primary growths in the ventricles, or from primary growths outside the skull. In either case the nodules are small, multiple, and rounded, and are often arranged somewhat symmetrically.

Cancer of the brain is usually soft, and often of a colloid character. Under the microscope may be seen nests of epithelial cancer cells, arranged in a connective tissue stroma, but not displaying any definite glandular structure. The cells are most commonly of a cylindrical type, but other forms may occur. In these nests of cells epithelial "pearls" may sometimes develope, which closely resemble the pearls in epithelial growths of the skin, and are in striking contrast to the cylinder epithelium. The stroma not infrequently undergoes mucous degeneration, and later, the mucus being absorbed, cysts may form.

Cholesteatoma. — We occasionally see in the brain pearly white bodies, usually of small size, situated in the pia at the base or near the great fissures. They consist of epithelial cells arranged in concentric layers, resembling the cells of the epidermis. They contain cholesterine, and in very rare cases minute hairs. Their exact nature is unknown. Birch-Hirschfeld allies them to retention cysts, but most authors consider that they are offshoots of the epithelium of the medullary tube, arising from the outer layer of the blastoderm, and existing in the brain as malformations. At times they may grow to considerable size, and, very rarely, they may assume a clinical significance. Case XXII, however, is peculiar, as showing a connection, possibly fortuitous, between a cholesteatoma and a sarcoma.

4. Aneurysms.

Aneurysms of the arteries at the base of the brain are much rarer than actual neoplasms, and I have been able to find no record of a case at the hospital, and only one case has come under my observation. Many cases, however, are upon record, and they naturally give rise to symptoms resembling those of a new growth. They arise from primary degeneration of the blood-vessels, syphilis, injury, or embolism. The importance of miliary aneurysms in the genesis of cerebral hæmorrhage is familiar. Larger dilatations of the vessels are much rarer. In about half the cases death is caused by rupture of the aneurysm. Aneurysms are rather commoner on the left side of the brain, and, like aneurysms elsewhere, may be true or false. Gowers,[4] tabulating 154 cases, gives the following distribution: —

TABLE VII.

Middle cerebral artery	44
Basilar artery	41
Internal carotid artery	23
Anterior cerebral artery	14
Posterior communicating artery	8
Anterior communicating artery	8
Vertebral artery	7
Posterior cerebral artery	6
Inferior cerebellar artery	3

As there are two middle cerebral arteries it becomes apparent that the favourite seat of aneurysms is in the basilar artery.

5. Cysts.

I have already spoken of the cystic degeneration which many forms of new growths may undergo. In cases of hæmorrhage cysts may also develope from absorption of the clot. These cysts are secondary and therefore need no sep-

arate mention. In addition small retention (?) cysts are often found in the plexuses, but they rarely have any clinical significance. Finally a very few cases are on record where a dermoid cyst (*teratoma*) has been found in the brain, probably of congenital origin, and usually arising from the meninges.

6. Parasitic Growths.

Parasitic cysts, in Bernhardt's table, play an important part, and the Australian journals report numerous cases. In America they are rare, and the hospital records show very few cases of hydatid cysts anywhere in the body, and none in the brain. Parasitic cysts may be due either to *echinococci* (hydatids) or *cysticerci*.

Hydatid cysts may be small or large, single or multiple. They usually lie upon the brain, compressing it, and they may give rise to softening of the contiguous brain substance.

Cysticerci may assume the ordinary form of small bladders with scolices, or the racemose form, with sterile bladders hanging from one another like a bunch of grapes.

7. Changes in the Brain Consecutive to Intracranial Growths.

The growth of a tumour causes, of course, an increase in the contents of the cranium and a consequent increase of pressure within the skull. This is manifested in various ways. The cerebral fluid is diminished and the pia appears dry and anæmic; the convolutions are compressed and flattened, and the sulci are less deep; secondarily to this, the gray matter of the convolutions may become thinner and undergo atrophy from pressure. The growth may also exert pressure on the aqueduct of Sylvius or the venae Galeni, and cause a dropsy of the ventricles, the hydrocephalus thus

increasing the pressure. This increase of pressure may also be manifested in the membranes of the brain and in the bony structures; the dura becomes thinned, and the skull may become as thin as paper and may even be perforated, as in Case XXIII. In such cases the thinning of the bones is irregular, and the marks of the convolutions may be seen on the skull. In some cases the pressure is such as to produce considerable displacement of the brain, and thus it may cause a marked distortion of the brain and the brain stem. The cranial nerves may be directly compressed by the growth, as in Case XXVIII, and thus become atrophied, or they may atrophy by reason of the increased general pressure, as in Case XXXV. It is still a matter of dispute whether the inflammation of the nerves, sometimes seen, is due to pressure or to direct irritation. This will be discussed later in the consideration of optic neuritis.

The new growth may, of course, cause a direct destruction of tissue in the part where it developes, and it may further lead to a softening of the brain substance in the neighbourhood of the tumour, as has already been stated. This softening is due, in the first place, to the pressure on the vessels, with blood-stasis and œdema from pressure. In this form small hæmorrhages are often seen in the softened portions, and thrombosis may occur in the smaller vessels in the neighbourhood of the growth. Furthermore, by the direct irritation of the growth, an obliterating arteritis may develope, with subsequent softening. This is most common with the granulomata.

Larger hæmorrhages are more apt to occur in the tumour itself, especially in gliomata. Cyst-formation, either secondary to the hæmorrhage or due to retention, is sometimes seen. Where the growth reaches the membranes a localized meningitis is not uncommon, and if the growth be of an infectious character, especially in cases of tuberculosis, this

inflammation may become general. With tubercle it is probable that the growth is the primary lesion and general meningitis is secondary, but the reverse is of course not infrequently the case.

In some cases, as a result of the destruction of brain tissue by the growth, secondary degeneration may ensue. This occurs, of course, most frequently where the motor tract is destroyed. It seems to be less common than in cerebral hæmorrhage or in lesions which more suddenly cut across the motor fibres, but Case VIII presents the clinical features of a descending degeneration.

III. PATHOLOGY.

FROM what has been said of the anatomical changes found in the brain in cases of cerebral tumour, the pathological action of such growths may readily be inferred. This action may be classed under the three heads of pressure, destruction and irritation.

Every intra-cranial tumour must add to the contents of the cranium and thus, while it grows, increase the intra-cranial pressure. Of course, by the ordinary physical laws, the mechanical action of pressure can be exerted only by a growing tumour, and when it ceases to grow it ceases to exert pressure, although naturally the effects of past pressure remain. As the brain itself is a solid the pressure is not uniform, but it is more manifest where the brain is forced into recesses of the cranium or against an unresisting surface. This general compression gives rise to many functional disturbances, some of which will be pointed out later. Soft tumours do not exert very much local pressure, but the harder and denser growths, beside causing general compression of the brain, may exert local pressure on adjacent parts, thus giving rise to disturbances of function in those parts.

Intra-cerebral growths, much more than intra-cranial growths which do not involve the brain substance, destroy the portion of the brain in which they grow, and secondarily, by the processes of softening, degeneration, etc., already noted, they cause destruction of adjacent portions of the brain. In this way they give rise to symptoms due to the loss of function of the parts destroyed, symptoms of deficit (Ausfallserscheinungen), such as paralysis, anæsthesia, blindness, deafness, aphasia, etc.

Tumours of the brain, especially during their growth, act as irritants. The irritation thus caused may be mechanical, and exerted chiefly and primarily at the periphery of the new growth, or it may be chemical, the growth causing certain chemical changes in the blood and the lymph; in consequence of such chemical changes irritation may extend to more remote regions of the brain. As a result of these irritations, whatever their cause, there arise various symptoms of irritation (Reizerscheinungen), which, in their nature, are precisely the opposite of the symptoms of deficit; among them may be mentioned convulsions, contracture and pain.

As the tumour grows symptoms of irritation may develope in more remote parts, which become involved in the morbid process (symptoms of invasion); and these symptoms may be followed later on, when the morbid growth has entirely destroyed the part, by symptoms of deficit.

Finally, the altered conditions of the blood supply may have a marked effect on the symptoms presented by the growth. The changes in the vessels in the parts surrounding a tumour have already been mentioned, but very vascular growths are subject to variations in size from the variations in the amount of blood in them. This is seen especially in aneurysms, and, as is evident, it may give rise to temporary variations in the clinical picture.

IV. GENERAL SYMPTOMATOLOGY.

It may be said here once for all that tumours of the brain may exist with hardly any symptoms. It may be, as Hughlings-Jackson has suggested, that they cause a certain slight deficiency in mental power, in apprehension, or in the power for continuous exertion, and that careful study of the highest intellectual faculties may reveal a falling off, or a limitation of the mental field; but symptoms such as would lead to the suspicion of any real disease may be wholly wanting. In other cases there is evidence of disease, but the evidence is not clear enough to warrant the diagnosis of a cerebral tumour. In a third class of cases the existence of a new growth in the brain may be diagnosticated with much probability, but there are no symptoms which warrant a localization of the growth. Finally, the symptoms may be so clear and so definite as to make it probable, not only that there is a new growth within the cranium, but that that growth is located in one definite region. These distinctions will appear in the consideration of the individual cases. Out of the forty cases here collected, six presented no symptoms of cerebral disease, and in five other cases the cerebral symptoms may well have been due to other co-existing affections. In thirteen cases there were symptoms of some cerebral trouble, but the symptoms were not definite enough to permit a correct diagnosis. In eight cases there were symptoms definite enough to warrant the diagnosis or at least a strong suspicion of an intra-cranial growth. Finally, in eight cases it was possible to make a correct focal diagnosis, as well as a diagnosis of the existence of a growth. These rather surprising results are in part to

be accounted for by the fact that the principle of selection here employed was not to select cases dying from brain tumour, but to collect all cases where at the autopsy a new growth was found. In this way eleven cases are reported which are not strictly cases of brain tumour, but cases where the patient died of some other disease, and the tumour was merely a co-existing lesion, giving rise during life to no apparent disturbance. Excluding these cases, however, the figures still give a striking manifestation of the limitations of our ability to diagnosticate correctly intra-cranial growths.

The symptoms presented by intra-cranial growths are of two sorts, first, those due to the presence of a tumour somewhere in the cranial cavity, — general or diffuse symptoms; and, second, those due to the presence of a tumour in some definite region of the brain, — focal or localizing symptoms. The former may, speaking broadly, be ascribed to the general effect which the tumour produces upon the brain, the latter chiefly to the destruction or irritation of certain regions. In some cases, however, a tumour may, either from remote pressure, from some reflex action, from irritation conveyed along association fibres, from chemical changes, or from various unknown causes, give rise to disturbances in some more or less remote part of the brain, and thus lead to errors in diagnosis.

The chief general symptoms to be considered are headache, vertigo, vomiting, optic neuritis, convulsions, psychical disturbances, and disturbances of the general functions.

Headache. — This was noted in 27 cases and is one of the most constant symptoms of an intra-cranial growth. Mary Putnam Jacobi,[14] tabulating the cases of Lâdame and Bernhardt, found it in 401 out of 614 cases, the percentage being the largest with tumours of the cerebellum and corpora quadrigemina. The greater frequency of headache with cerebellar growths she ascribes to the increase of pres-

sure in the narrow space beneath the tentorium. In tumours of the corpora quadrigemina she thinks the connection with the cerebellum has some influence, but in this region a new growth is pretty apt to block up the aqueduct of Sylvius, press on the venae Galeni, and give rise, as in Cases XXII and XXIII, to marked hydrocephalus and to a very great increase of the intra-cranial pressure. The probable cause of headache in a majority of cases is the irritation of the highly sensitive dura by the increased pressure. In some cases the new growth may affect the dura, and here the pain becomes more distinctly localized, and there is sometimes local tenderness on percussion.

The character of the headache in cases of brain tumour may vary exceedingly. Sometimes the pain is slight, temporary, and of a dull character; in other cases it may be most intense and unendurable, causing the utmost suffering and driving the victim to suicide or making him insane; the pain may be dull or sharp, transitory, intermittent, or constant. It is often aggravated by stooping, exercise, mental exertion or any cause that may lead to cerebral hyperæmia.

Oppenheim[15] has found that the location of the headache bears no relation to the situation of the growth, an opinion with which I am disposed to agree. Out of sixteen cases where the headache was more or less constantly localized in one part of the head, in only six did the pain at all correspond to the part of the brain in which the tumour was situated.

With tumours of the occipital lobe and cerebellum some writers think that the pain is more apt to be in the back of the head, and in tumours of the frontal lobe the pain may be in the front of the head, but this rule is by no means without exceptions. With tumours of the base or pons, the fifth nerve may be affected, giving rise to a trigeminal neuralgia which is to be distinguished from true headache.

Oppenheim thinks that localized tenderness is of more value than localized headache as indicating the probable seat of the growth, but, as Case XXIX will show, this is not an invariable rule. Data are lacking, in my cases, to permit me to give statistics on this point.

Vertigo. — Vertigo was noted in 12 of my cases, and in 31 per cent. of those reported by Mills and Lloyd.[11] It is so common a symptom in all sorts of affections that it has comparatively little significance as diagnostic of a tumour. Some writers think that it is most suggestive of tumour when it comes on after rising from a recumbent position; but vertigo is not uncommon on such movements in any case of anæmia or in any disturbance of the cerebral circulation. Vertigo is said to be commoner with tumours in the posterior part of the brain, as in seven out of my twelve cases. The causes of this vertigo are probably numerous. In some cases it may be due to oculo-motor paralysis, in others to the digestive disturbances, and in others still to disturbances in the cerebellum, or to the general pressure which is conveyed to the labyrinth.

Vomiting. — Vomiting was noted in 172 of 568 cases tabulated by Jacobi and in 17 of my own cases. Another of my cases had nausea without vomiting. In 13 of my 18 cases the growth involved the basal ganglia and the posterior portions of the brain, and other writers confirm the idea that vomiting is commoner with tumours in the posterior part of the brain. Vomiting is especially common in cerebellar growths, and here, more than elsewhere, it may be most persistent and severe. Sometimes, especially with cerebellar growths, the vomiting may be unattended with pain or nausea and may be independent of the ingestion of food; but even with cerebellar growths the vomiting may be attended with nausea and very severe pain, as in Case XXIX. In some cases the patient may vomit without warning, and

the vomiting closely resembles the regurgitation of infants. In other cases the vomiting is attended with great pain or distressing nausea.

The vomiting is probably due to various causes. Ferrier[26] ascribes it in the majority of cases to irradiation of irritation by the nerves of the meninges, or to the physical effects of acute pain. Painful impressions conveyed by the branches of the fifth nerve to its nucleus in the medulla may be transmitted to the contiguous vagus nucleus. Vomiting may also arise from vertigo, or from irritation of the vomiting centre in the medulla by pressure or direct irritation, apart from any irritation of the fifth nerve.

Optic Neuritis. — Certain writers, of whom one of the latest is Oppenheim,[15] endeavour to make a distinction between a true optic neuritis and the so-called "choked disc" (Stauungspapille). Oppenheim follows Uhthoff in his distinction between the two, speaking of choked disc when there is a prominence of the nerve of at least two-thirds of a millimetre (a difference of refraction of two dioptrics).

Deutschmann,[16] however, has found, histologically, no difference in the cases of so-called choked disc and cases of optic neuritis. In every case there was a true degenerative inflammation of the nerve fibres. I have therefore not attempted to make any distinction.

Optic neuritis is the most important general symptom of an intra-cranial growth. It is, of course, not pathognomonic of a tumour, but an ophthalmoscopic examination should be made in every case of suspected brain disease. Oppenheim has shown the importance of making such an examination, not merely once, but repeatedly during the progress of the disease, as it may be a symptom of late developement. The presence of double optic neuritis should always lead us to suspect a tumour. The frequency of optic neuritis is not definitely known, owing to the imperfection of many of the

records, and the neglect of examining the eyes. In my own cases, 11 had neuritis, 2 atrophy (probably post-neuritic) and 5 a normal fundus, but in these five cases repeated examinations were not always made. In Cases XIV, XVI, and XXIV, however, examinations were made repeatedly, until shortly before death, but there was no neuritis. Neuritis was therefore present in over two-thirds of the cases examined. Of the other 21 cases two had some dimness of vision noted, but this is not satisfactory evidence for or against the existence of neuritis, for tolerable vision may exist with marked neuritis. Thus Cases XXIII and XXIX had a vision of $\frac{20}{30}$ and $\frac{15}{17}$ respectively, although neuritic changes in the eyes were marked. Annuske and Reich,[3] in 88 cases, found neuritis absent in only five per cent., which is probably a little too large; Gowers[4] thinks that neuritis probably exists in four-fifths of the cases; Bernhardt[1] found atrophy or neuritis in 25 per cent., amblyopia in 20.4 per cent., and normal vision or a normal fundus in 6.8 per cent., 47.8 per cent. of the cases giving no data. He recognizes that neuritis may have existed in some of the so-called "normal cases." Oppenheim found that in 82 per cent. of his 23 cases there was neuritis on one or both sides; in 13 cases he found choked disc and in 5 neuritis, and he accepts Annuske's view as to the frequency of neuritis. Gowers says that neuritis is rather commoner when the brain itself is invaded by a growth, and that the size of the growth has no influence on the existence of neuritis, in other words that increased intra-cranial pressure is not a factor. In four of my cases of neuritis there was a great increase of intra-cranial pressure, in two other cases and in one of the cases of atrophy there was some increase, in the rest no statement is made. In two of the cases with normal fundus there was marked increase of

pressure, in two others the increase was moderate, and in the fifth no statement is made.

The pathology of neuritis has been much discussed, and many theories have been advanced to explain it. These may be classified as (1) the increased pressure theory, (2) the descending neuritis theory, (3) the vaso-motor theory, (4) the irritation-pressure theory.

1. Von Graefe[16] advanced the hypothesis that the increased intra-cranial pressure, so common in cases of tumour, caused compression of the cavernous sinus, checked the outflow from the ophthalmic vein, and gave rise to a venous stasis in the eye, producing the "choked disc." This hypothesis was later disproven by Sesemann, who showed that the ophthalmic vein anastomosed with the facial, so that stasis could not be produced in this way. The increased pressure theory took a new form, which has, in the main, been accepted until recently, after Schwalbe showed that the intra-vaginal space surrounding the nerve communicated with the sub-arachnoid tissue. Hence Schmidt and Manz argued that, with increased pressure, the fluid is forced into this space, compressing the nerve, and they showed that injections into the sub-arachnoid tissue actually reached this space. Kuhnt argued further that there was a lymph stasis from the vessels of the lamina cribrosa, and Ulrich and Parinaud advanced the hypothesis that there was an œdema of the nerve. Schulten claims to have established the hypothesis of Schmidt and Manz by injecting substances into the cranial cavity, thereby causing an increase of pressure, after which he found signs of a beginning choked disc; he supposed from this that a greater pressure would produce the true choked disc.

I have already said that Gowers believes that increased pressure is not the cause of neuritis, and my own cases show that increased pressure may exist without neuritis. Various arguments have been brought forward against this theory,

of which the most conclusive are those of Leber and Deutschmann. Deutschmann[16] claims that choked disc is an inflammatory condition, like the neuritis of meningitis or of Bright's disease, that there is no anatomical difference between it and neuritis, and that there is in the nerve in all cases a true neuritis. Leber found neuro-retinitis in the earliest stages of choked disc. Deutschmann found choked disc without dropsy of the nerve sheath, and with no compression of the central vessels. Even when there is dropsy of the sheath it is slight and the vessels are not compressed. Further experiments by Deutschmann were still more conclusive. He found that by injecting sterilized agar-agar into the sheath of the nerve, with strict antisepsis, choked disc could be obtained only when the vessels were absolutely compressed. Injections of agar-agar into the cranial cavity never produced choked disc, no matter how great the pressure. His further experiments will be considered later.

2. Von Graefe also made an alternate hypothesis that a tumour gave rise to meningitis, which, in turn, set up a descending neuritis; subsequent writers have supported this view. In many cases, however, there is no meningitis, the neuritic changes are chiefly, if not wholly, in the bulbar end of the nerve, and Deutschmann argues that the neuritis may also be ascending. Neuritis may also improve, and, in such cases, relapses are never noted. In unilateral neuritis the neuritis is usually on the side opposite to the tumour. Finally, a descending neuritis in other cranial nerves is, to say the least, rare. Descending neuritis may account for some of the cases, but probably only for a minority.

3. The vaso-motor theory, that the tumour by irritating the vaso-motor nerves in the brain, causes vascular disturbances in the nerve and hence neuritis, has met with little acceptance. The old hypothesis that vaso-motor alterations produce inflammatory changes is well nigh abandoned, and

the presence of vaso-motor nerves in the brain is not yet proven. Other irritations, which might well cause vaso-motor changes, do not produce neuritis, and disturbances are not found in other regions, although we should expect them on such a hypothesis.

4. Leber was the first to advance the hypothesis that the "products of tissue metamorphosis of new growths, mingled with the inflammatory transudations, act as irritants, enter the sheath of the nerve with the cerebro-spinal fluid, reach the bulb and give rise to neuritis and papillitis." Deutschmann supplemented the experiments already referred to by others which support Leber's views. He injected staphylococci into the sheath of the nerve, and produced neuritis. Injections of staphylococci into the brain, however, proved speedily fatal. He was more successful with tubercle, and succeeded in producing a neuritis, not of the descending type, by injecting tubercle into the brain. He finds, moreover, that the lymph-currents of the cerebro-spinal fluid are centrifugal, and that increased pressure favors the process. "The inflammatory affection of the papilla, which increases to choked disc, has nothing to do with a stasis from pressure. It is the effect of inflammatory irritants which enter the optic sheath with the cerebro-spinal fluid from the cavity of the skull, remain at the bulbar end, and set up an infective action." "Pure increase of pressure in the cranial cavity does not lead to disease of the intra-ocular end of the optic nerve; the chief factor is to be found in the inflammatory irritants which enter the sheath from the cranial cavity." Whether these irritants are chemical or parasitic is still uncertain. Deutschmann, having found various cocci in the vicinity of a tumour, is somewhat inclined to the latter view, thinking the immediate neighbourhood of a new growth a good field for the developement of bacteria.

Leber and Deutschmann seem to have overthrown the in-

creased pressure theory. The increase of pressure, however, is regarded by them as very important in the production of neuritis, since it forces the irritants into the optic sheath. Their theory seems to me the most plausible for the majority of cases, but in some there is very likely a true descending inflammation. The irritants which are supposed to cause the neuritis are still unknown. It is a curious fact, which none of the above theories throws light upon, that, in cases of tumour, neuritis of other cranial nerves apparently does not occur, at least not with any great frequency, except when the nerves are directly involved in the growth.

Convulsions. — Ten of my cases had convulsions of a more or less general type; in one of these, however, the symptom was probably due to tubercular meningitis, and in two others the convulsions were undoubtedly uræmic from chronic Bright's disease. These attacks were of a general, epileptiform character. Gowers[4] distinguishes two forms, one a typical epileptic seizure, the other an attack of petit mal. Such attacks may come on with a new growth in almost any part of the brain, and they have no localizing value. They do not seem in any way related to the size of the tumour. Most writers assume that they are caused by the increased pressure, but Gowers has shown very conclusively that this is not the case.

The consideration of partial epilepsy will, perhaps, throw some light upon the pathology of general convulsions. Four of my cases had spasm or convulsive seizures beginning in or limited to a definite portion of the body. This symptom has been considered of very great value in the focal diagnosis of cerebral disease, and Seguin[17] lays much stress upon the study of the special distribution of the spasm which inaugurates the convulsion of Jacksonian epilepsy, as a signal symptom. In some cases there is a sensory aura in the part of the body in which the spasm

begins, followed by a tonic or clonic spasm of a group of muscles, the spasm remaining limited to this region, or extending to the entire limb, to one half the body, or to the whole body. This spasm in its extension follows the distribution of the motor centres in the cortex. If it begins in the face it extends first to the arm and then to the leg, never to the leg and then to the arm; so, also, if it begins in the leg, it extends first to the arm and then to the face. The details of this "march" of the convulsions will be spoken of later. This type of epilepsy was at first supposed to be due to organic disease of the cortex in or near the motor area, and to be almost pathognomonic of such a lesion. This hypothesis, however, no longer obtains. The theory that every epileptic seizure, partial or general, is due to a discharge in the cerebral cortex has steadily gained ground, and seems to be the most reasonable one. The discharge, however, is not always due to disease in the cortex, but it may arise from irritation of the cortex from more remote lesions. The same may be said of partial epilepsy. The researches of Seppilli[18] and Löwenfeld[19] have shown that partial epilepsy, due probably to a discharge in the motor cortex, may be brought about by a gross lesion in the neighbourhood of the cortex or even in the basal ganglia. Furthermore, partial epilepsy may occur, not only as a symptom of organic brain disease, but as a symptom in uræmic convulsions, in hysteria, or in idiopathic epilepsy. The existence of partial epilepsy in the latter affection has until recently been denied, Rolland[20] claiming that the two have nothing in common, but Löwenfeld seems to have established the fact.

It is evident, then, that partial epilepsy cannot be regarded as a decisive focal symptom. Rolland asserts that it is due more often to irritative than to destructive lesions; hence with a large cortical growth the epilepsy might be due to irritation anywhere in its periphery. The most we can say

is that partial epilepsy is most commonly due to organic disease in or near the motor cortex, and that if it is present in any case of cerebral tumour, the tumour very likely involves the special motor centres which preside over the movements exhibited in the convulsions. The growth, however, may be remote from the centres, and may even be situated in the basal ganglia.

Psychical Disturbances. — In 29 cases there was more or less psychical disturbance; and Jacobi[14] records it in 49 per cent. of the cases collected. I believe, however, that in every case some change can be found by a competent observer who has known the patient intimately before; in other words that there can be no gross lesion of the brain without some disturbance, greater or less, in the psychical functions. This may consist only in some slight impairment of the higher intellectual powers. A good French scholar may become unable to read French without translating it; a mathematician may have less power to solve intellectual problems; a musician may have a slightly diminished power to play a new and difficult piece at sight, — powers not present even in health in the average hospital patient. In other cases there may be lack of power of persistent application, undue irritability, or marked inertia. Other patients may be delirious, maniacal, or exhibit various delusions. Others, still, may become dull, childish, and utterly demented, or present the clinical picture of paretic dementia.

Oppenheim[15] lays stress upon a condition of somnolence, which he thinks is very common in cases of tumour of the brain. The patient may be aroused and he will answer questions intelligently but he will drop off to sleep almost immediately after answering. In other cases this somnolence goes on increasing, finally concluding with coma. In fourteen of my cases this condition was present in a greater or less degree, but in a number of these cases it was merely a

terminal symptom, making its appearance only in the last few weeks of life. It depends, I suspect, chiefly upon the degree of intra-cranial pressure, and I am disposed to think it by no means a constant symptom. It is due simply to an increase of general pressure or to destruction of certain portions of the brain.

Surface Temperature. — In only two cases was the surface temperature noted, and here it was above the normal, as laid down by Gray.[21] Mills and Lloyd[11] from an analysis of seven cases conclude that "in brain tumours the average temperature of the whole head is elevated several degrees above the normal, and that the elevation of temperature is usually greatest at the station nearest the seat of the growth." Case XXIX does not warrant such a conclusion. Much more evidence, however, is required in order to settle this question.

Beside these symptoms, which may be present in any case, tumours of the brain present certain more or less definite focal symptoms, according to their situation. These symptoms will be dwelt upon in connection with the special symptomatology of the individual cases. Most of them are symptoms of deficit or irritation, among them being the various forms of paralysis, anæsthesia, and disturbances of special sense. In addition there are other general symptoms of value in corroborating the diagnosis, among which the symptoms of increasing pressure are of chief importance. Beside the steady increase of the symptoms already noted we may observe a slow pulse, disturbed respiration, dilated or contracted pupils, and in the later stages strabismus. Pseudo-apoplectic attacks are occasionally described, the origin of which is uncertain. Some of them are doubtless true apoplexies, from hæmorrhage in or about the tumour. As the disease progresses signs of physical exhaustion appear, anæmia, emaciation, digestive disturbances and the like. The appetite often is poor, but

in a few cases it becomes ravenous. Toward the close of life somnolence and coma, as has been said, may develope. Finally we meet with the ordinary group of symptoms which mark the end of so many nervous diseases — incontinence of urine and fæces, general paretic symptoms, bed-sores, cystitis, Cheyne-Stokes' respiration, and death.

V. SPECIAL SYMPTOMATOLOGY.

It is hardly necessary to repeat the statement already made, which will be confirmed in part by some of the cases that follow, that tumours may exist in almost any part of the brain either without any symptoms referrible to a new growth, or without any focal symptoms. In the forty cases collected, as I have said, in only eight were the symptoms so definite that a correct localization was possible before death.

It is, furthermore, not always possible to determine from records of autopsies made before the question of localization assumed such importance, the precise location of new growths; nor can we speak with confidence of the presence or absence of symptoms from clinical records made before the significance of those symptoms was appreciated, or from records made in haste by hospital internes from hasty or unskilled observation of patients who spent perhaps only a few days in the hospital.

In the tables of Bernhardt,[1] a division is made between tumours of the cortex and tumours of the lobes, but tumours of the occipital cortex are put in the same table with tumours of the central region. It seems to me more important to differentiate between tumours of the various cortical regions than to make a hard and fast distinction between cortical and subcortical growths. Cortical tumours, if of any size, also involve the subjacent white matter, and tumours in the white matter, in many cases, cut off communication with the adjacent cortical centres, and give rise to symptoms of irritation or deficit much like those caused by disease of the cortex itself.

Other growths, more deeply seated, have a similar relation to tumours of the optico-striate region. Therefore, although in the chapter on diagnosis I shall speak of the symptoms which enable us to differentiate between cortical and subcortical growths, I shall not make such a distinction in the classification of the cases here reported.

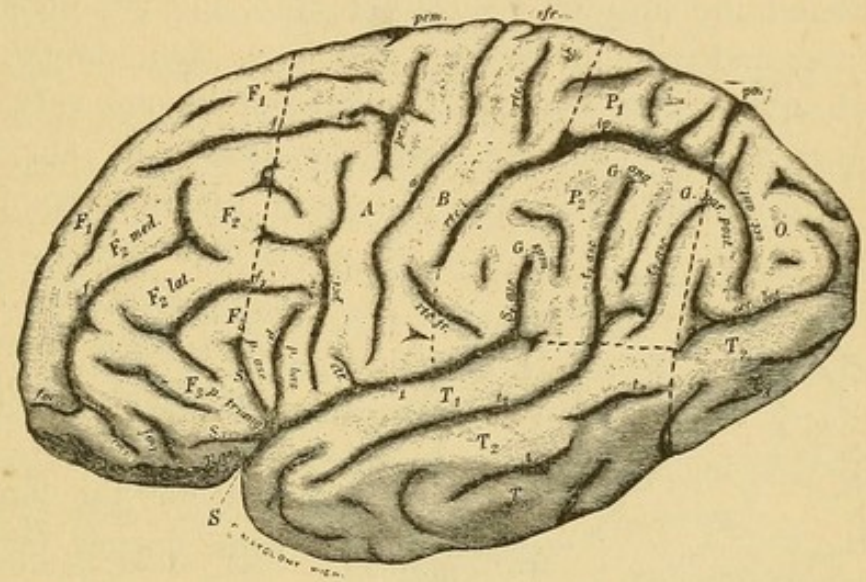

FIG. 1 (Eberstaller). Lateral aspect of the brain. The dotted lines indicate the divisions into regions.

S = Trunk of the fissure of Sylvius.
S1 = Fissure of Sylvius.
S1 asc. = Posterior ascending branch of the fissure of Sylvius.
S2 = Anterior ascending branch of the fissure of Sylvius.
S3 = Anterior horizontal branch of the fissure of Sylvius.
c. = Central fissure.
ctr. = Lower transverse sulcus.
pci. = Inferior precentral sulcus.
pcs. = Superior precentral sulcus.
pcm. = Median precentral sulcus.
rtc. i. = Inferior retro-central sulcus.
rtc. s. = Superior retro-central sulcus.
rtc. tr. = Transverse retro-central sulcus.
sfr. = Subfrontal fissure.
f1 = Superior frontal sulcus.
f2 = Inferior frontal sulcus.
f3 = Median frontal sulcus.
d. = Diagonal sulcus.
r. = Radiate sulcus.
fm1, fm2, fm3 = Fronto-marginal sulcus.
ip. = Inter-parietal sulcus.
po. = Parieto-occipital fissure.
occ. ant. = Anterior occipital sulcus.
occ. lat. = Lateral occipital sulcus.
t1 = First temporal sulcus.
t1 asc. = Ascending branch of the first temporal sulcus.
t2 = Second temporal sulcus.
t2 asc. = Ascending branch of the second temporal sulcus.
A = Anterior central convolution.
B = Posterior central convolution.
F1 = Superior or first frontal convolution.
F2 = Middle or second frontal convolution.
F2 med. = Median part of the second frontal convolution.
F2 lat. = Lateral part of the second frontal convolution.
F3 = Posterior or third frontal convolution.
p. bas. = Basilar part, *p. asc.* = Ascending part } of the opercular part of the third frontal convolution.
p. triang. = Triangular part of the third frontal convolution.
p. orb. = Orbital part of the third frontal convolution.
1 = Lateral root of the first frontal convolution.
2 = Lateral root of the second frontal convolution.
P1 = Superior parietal lobule.
P2 = Inferior parietal lobule.
G. spm. = Supra-marginal gyrus, *G. ang.* = Angular gyrus, *G. par. post.* = Posterior parietal gyrus } of the inferior parietal lobule.
T1 = First temporal convolution.
T2 = Second temporal convolution.
T3 = Third temporal convolution.

I. Tumours of the Cerebral Cortex.

a. Tumours of the Pre-frontal Region.

Case I. M. R. CLVII–52.* William M., 44, salesman, was admitted to the hospital the 17th of February, 1879. There was a history of probable syphilis. Three weeks before he entered the hospital he caught cold, and complained of weakness and pain in the back and legs. On admission he was observed to be weak and quiet, and he ate very little. On the 21st he became semi-comatose and ate nothing. He moved when the skin was pinched, seemed to have no paralysis, and could be roused. The pupils were somewhat contracted and did not react to light. The next day the coma became deeper, the right pupil was dilated, and both pupils were immobile. There was some general tremor of the limbs, and the patient died.

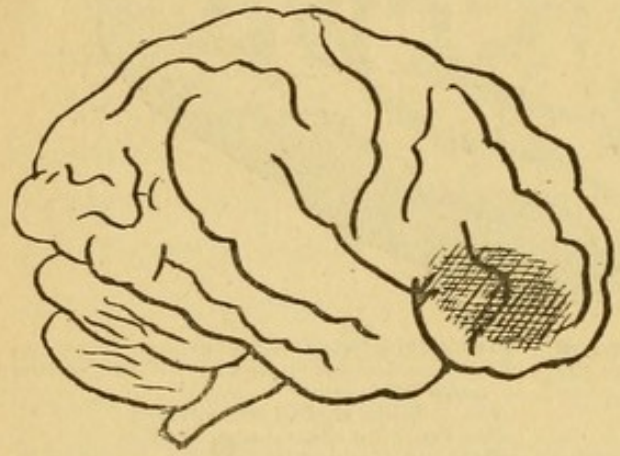

Fig. 2 (Dalton). Case I. The shaded portion indicates the location of the tumour. The fissures are indicated in Fig. 7.

Autopsy. — There was some œdema of the lungs. The convolutions of the brain were flattened, and in the lower part of the right frontal lobe there was a tumour the size of a hen's egg, round, of a mahogany colour, containing several distinct hæmorrhages, and surrounded by yellow softening.

Case II. M. R. CCXLI–16. George S., 40, married, a wool sorter, entered the hospital the 25th of March, 1886. On admission he was found to be wandering, and no history

* These numbers refer to the hospital records, medical, surgical, and nervous.

could be obtained. He muttered, picked at the bed-clothes, and apparently saw imaginary persons about him. His temperature was 102.2° F.; his pulse 104. There were bronchial respiration and fine râles. The abdomen was tender, the pupils reacted to light. On the 28th there was carphologia and subsultus tendinum. He grew more delirious, the pupils became sluggish; he had no paralysis, but there was incontinence of urine and fæces, and he died.

Autopsy. — Tubercular meningitis. The two frontal lobes were adherent, and at the point of adhesion there was a nodule the size of a pecan-nut in the brain substance, pale red in colour, with the vessels of the pia passing through it.

CASE III. N. R. XVI–42. Ella H., 23, married, domestic, entered the hospital the 26th of August, 1882. Five weeks before entrance she began to complain of headache and vomiting, the headache being severe and constant. There was vertigo and blurring of vision on getting out of bed. For four weeks before entrance she had been in bed most of the time, suffering from headache. She complained also of poor sleep and bad dreams. On the 6th of September she had cramps in the right hand and leg and numbness of the right arm. There was optic neuritis. On the 11th the right pupil was dilated, the respiration grew slow and feeble, and she became comatose and died.

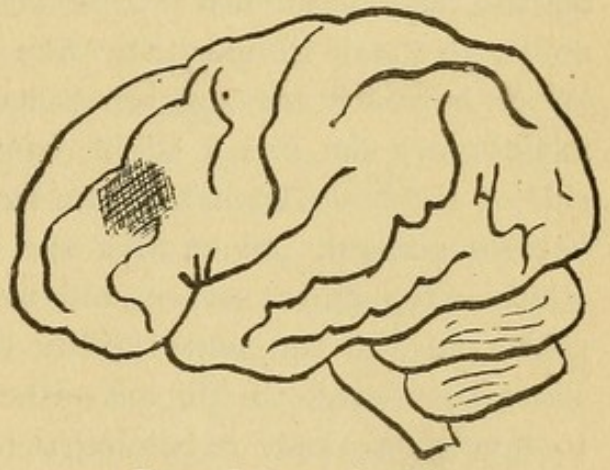

FIG. 3 (Dalton). Case III.

Autopsy. — The dura was found adherent to the pia on the left side; the pia was dry and the convolutions flattened.

The dura was also adherent on the right side. In the central part of the left frontal convolutions there was a sarcoma the size of an English walnut, gray in colour and homogeneous; the white substance surrounding it was softened.

† CASE IV. Mrs. E., 54 years old, was seen by me the 16th of March, 1890. Her parents died of apoplexy, and she had had a severe nervous strain for a year and a half. Three years before she had a bilious attack. In the autumn of 1889 she began to complain of pain across the forehead and failure of vision. The liver was also slightly enlarged. In January, 1890, she had severe vomiting, with pain in the forehead and further failure of vision. She lost control of the bladder. She had a very weak pulse, which varied between fifty and ninety, and at times failed almost entirely, becoming imperceptible at the wrist. She seemed intelligent, but when she spoke her sentences were very short. At that time she was in bed for a week and passed her water in bed. Later on, the right pupil was a trifle larger than the left, and her ability to speak diminished. She used only monosyllables, lay on her back and apparently had much headache. Early in February she had a slight convulsion affecting the right side of the body, including the face; the convulsion did not become general. Since then she had had other convulsions which were more severe and were attended with a tonic spasm in the leg and rigidity of the arms. The face at times was drawn to the left — the pulse was rapid, seventy to ninety, at times becoming very weak and thready, and suddenly failing. She had attacks of stupor lasting for days, during which she would neither answer nor protrude the tongue. At times she was apparently unable to speak. She would occasionally say "no" for "yes," and once in a while she would say a few words, but the speech was

† Cases marked thus came under my own observation at some part of their course.

more or less affected. There was occasional vomiting, coming on without warning, and not accompanied with nausea. There was no control over the bladder or rectum. For ten days the right eye did not close as well, and there was marked mental failure. These symptoms, however, varied, and some days she seemed quite bright. She put her hand frequently to her head as if there were pain there. The rigidity of the right arm was quite slight and disappeared on passive motion. There was double optic neuritis. At my examination she did not answer when spoken to and would not do anything she was asked. The right pupil was larger than the left; both reacted to light. The eyes moved well and there was no ptosis. She used both arms, but the right arm was used less than the left; the reflexes were normal. Her condition continued practically unchanged for some time, with periods of temporary improvement. Later on she had other convulsions, sometimes beginning in the left side but more frequently on the right. At last she had right hemiplegia. During the summer she died without my seeing her again.

Autopsy. — An infiltrating glioma, the size of a hen's egg, was found well forward in the left frontal lobe.

In the pre-frontal region I include all the anterior end of the cerebrum, in front of a line drawn as an extension of the anterior ascending branch of the fissure of Sylvius. This falls a little in front of the pre-central sulcus, and thus the posterior extremities of the frontal convolutions are not included. This boundary has been adopted by Mills.[11]

The region in question lies just in front of the great motor region of the cortex, and is supposed to be the region chiefly concerned in the higher psychical processes. It probably includes, however, the centres for movements of the head and eyes to the opposite side; at least in the monkey's brain,

according to Horsley and Schaefer,[22] these centres lie in front of the boundary line given. This pre-frontal region has been regarded as one of the so-called latent regions of the brain, in which lesions give rise to no definite localizing symptoms. In a certain number of cases, however, the diagnosis of a new growth in the frontal lobe can be made with considerable assurance. In addition to the general symptoms of an intra-cranial growth already discussed, the most marked symptoms of a tumour in this region are of a psychical character. This was especially noticeable in Case IV. In such cases we meet with failure of the power of attention and judgement, mental slowness and uncertainty passing on to marked hebetude, dementia or stupor, and a general and marked decline in all the higher intellectual powers. Less frequently there may be irritability, excitement, or delirium, as in Case II. Such symptoms may occur, of course, in almost all cases of tumour, but where the tumour is in the pre-frontal region they are probably of much earlier onset. Such mental symptoms, then, coming on early and associated with the general symptoms of headache, optic neuritis, vomiting and vertigo, with the absence of any special focal symptoms, may lead us to suspect a tumour in this region.

In addition to these symptoms the anatomical position of the growth may in some instances give rise to other symptoms of much localizing value. By pressure upon or extension of the growth into one olfactory lobe we may find unilateral anosmia, or in rare cases hallucinations or perversions of the sense of smell from irritation. Invasion of one optic nerve may cause neuritis to appear in the eye of the affected side, although this is very rare. Blindness of one eye from pressure on the nerve is also possible. In other cases, when the chiasma is involved, we may discover homonymous, temporal hemianopsia. In other cases pressure on the orbit

may give rise to exophthalmos. Localized headache, as has been said, is of some significance.

Much more important, however, are the symptoms caused by the backward growth or pressure of the tumour — symptoms of invasion of the motor region. Cases III and IV present examples of this. The tumour first irritates the adjacent motor centres, and we get spasm or partial epilepsy of the leg, the arm, or the face; or, if the left hemisphere be involved, symptoms of beginning motor aphasia. The more exact focal significance of these symptoms will be apparent in considering the different motor areas and their arrangement in the cortex. Later on the advance of the growth causes destruction of the motor centres, and paralysis and anæsthesia succeed the spasm and sensory aura. In tumours of the left frontal lobe symptoms of aphasia then become more marked.

Although partial epilepsy, by itself, is of no very exact significance, yet, when it is superimposed upon the clinical picture already given, it leads to a very strong probability, although not to an absolute certainty, that the tumour is in the pre-frontal region. In but two of the above cases is it possible from the data given to diagnosticate a tumour. In Case II the tumour was secondary to the meningitis in its effect upon the symptoms. Case III showed the invasion symptoms clearly, but the record of the psychical state, which might have made the focal diagnosis possible, is unfortunately lacking. In Case IV a focal diagnosis was made before death and confirmed by the autopsy. Here the question of operation was brought forward; but, owing to the weakness of the heart and the probable vascular nature of the growth, it was deemed inadvisable to operate.

b. Tumours of the Central Region.

CASE V. M. R. CXX–8. Henry L., 40, single, entered the hospital the 16th of February, 1884, with advanced phthisis, which he had had for four years. He suddenly became unconscious. There were respiratory disturbances, and he died on the 4th of March.

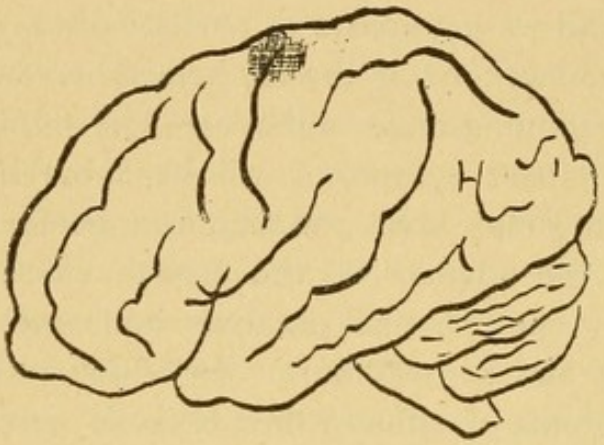

FIG. 4 (Dalton). Case V.

Autopsy. — Pulmonary tuberculosis. Over the left central convolutions, three centimetres from the median line, there was a firm, dense, cartilaginous mass, measuring twelve millimetres each way, and about two millimetres in thickness. The brain was otherwise healthy.

CASE VI. M. R. XCIII–165. Michael M. entered the hospital the 28th of September, 1873. He had been drunk for nine weeks, had taken little food, and had had much vomiting. For three weeks his mind had been wandering — he stared at vacancy and muttered to himself. He was stupid, but he had no active delirium. For two weeks he had dragged the right leg in walking. He passed urine and fæces in bed. Six days after entrance he died.

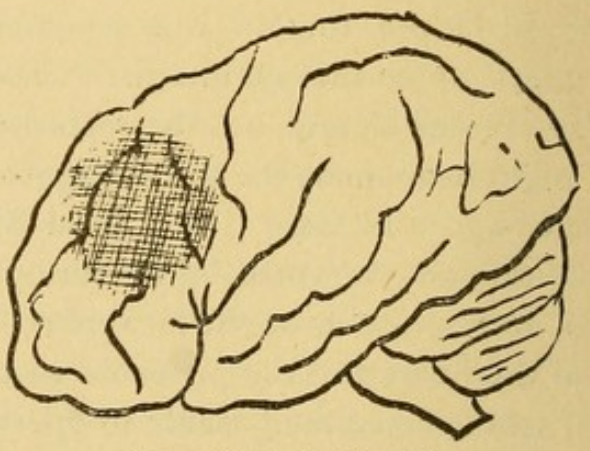

FIG. 5 (Dalton). Case VI.

Autopsy. — The dura was adherent to the skull. The convolutions were flattened. In the left second frontal convolution was a tumour two inches in diameter, harder than the brain, somewhat translucent, of a brownish-gray colour. Beneath this was a cyst containing an ounce of clear yellow fluid. In the upper part of the left medulla was a hæmorrhage the size of a marble.

CASE VII. N. R. IV–218. Simon F., 55, single, a hostler, entered the hospital the 2d of September, 1878. Eight years before entrance he received a blow on the left side of the face, without any bad result. Nine months before entrance he complained of pain in the left temporal region, the pain varying from dull to severe. His memory was poor and his mind sluggish. The left side of the face could be moved better than the right. There was advanced phthisis. On the 7th he became restless and could hardly be aroused. He failed rapidly, and the next day he had carphologia, subsultus tendinum, and twitching of the masseters. He became comatose and died.

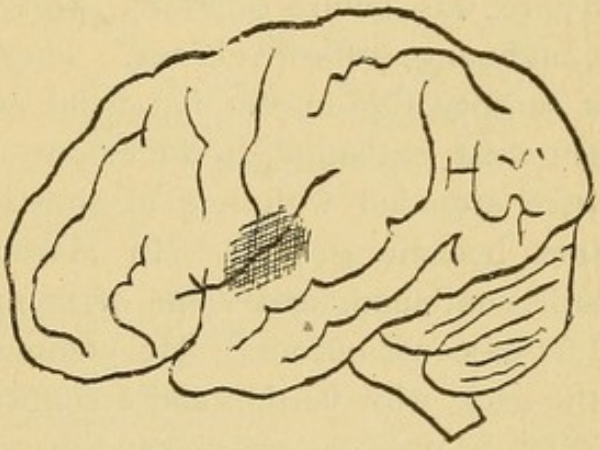

FIG. 6 (Dalton). Case VII.

Autopsy. — There was tuberculosis of the lungs and kidneys. The dura was adherent to the skull and to the pia, and there were opacities of the pia. In the left fissure of Sylvius the artery and vein were surrounded by a dense inflammatory mass, half an inch by an inch in extent.

† CASE VIII.* S. R. CLXIV-9. Arthur G., 32, married, a florist, entered the hospital the 17th of December, 1888. In 1868 he received an injury to his head, followed the next day by seven convulsions. He had no further trouble, and the previous history was otherwise negative. In 1886 he began to have nausea, vomiting, and some pain in the head. In the beginning of 1887 he had stiffness and weakness of the hands. In March, 1887, he had a convulsion, with extension and abduction of the left arm. About the same time there was loss of power in the left arm and leg and numbness in the left hand, and there developed left hemiplegia, with exaggerated reflexes and slight contracture. There was failure of vision, with increase of paralysis and continuance of convulsions. These convulsions began with a sensory aura in the left hand and a clonic spasm of the left wrist, extending to the elbow. Sometimes these seizures were attended with loss of consciousness, and the convulsions became general. In March, 1888, the convulsions still continued, and some of them were not attended with loss of consciousness. He complained of a tired feeling in the back, with vertigo and a suffocated feeling. In January, 1888, he was obliged to give up work. About this time he had very severe pain in the back of the head; his memory failed, and his speech was slow. He had burning in the stomach, and occasional attacks of slight delirium. In November, 1888, when I first saw him, examination showed impaired movement of the eyes to the left. He had double optic neuritis, paralysis on the left side of the body, including the face, with contracture and exaggerated reflexes. There was left hemianæsthesia, most marked in the arm, comprising anæsthesia for touch, temperature, localization,

* Dr. Bradford and I have reported this case in full in the Boston Medical and Surgical Journal for 4, 11, 18 April, 1890, and in the Medical and Surgical Reports of the Boston City Hospital, Vol. IV, p. 103.

position and movement. There was no tenderness of the skull. The surface temperature was somewhat above normal, but there were no differences in temperature over the different portions of the head. A convulsion was observed to begin with spasm of the face, abduction, pronation and tremor of the arm. Another began in the shoulder, another in the wrist. His ears, nose, throat and urine afforded nothing abnormal on examination. He was trephined on the 28th of December by Dr. E. H. Bradford, and a tubercular tumour, weighing 35 grammes, and consisting of two masses partly separated, was removed from the middle of the two ascending convolutions on the right. The tumour measured seven by four by three centimetres and lay about a centimetre below the cortex. The patient died from the shock in about three-quarters of an hour. There was no autopsy.

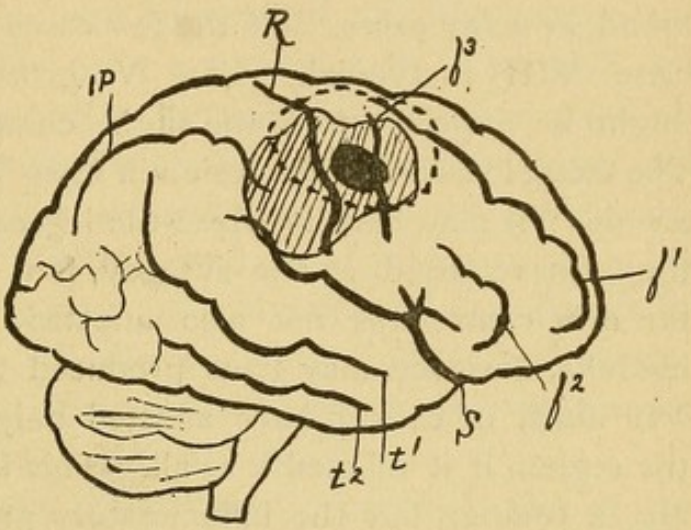

FIG. 7 (Dalton). Case VIII. The black spot indicates the centre of the trephine hole.
S = Fissure of Sylvius.
R = Fissure of Rolando.
IP = Inter-parietal sulcus.
f_1, f_2 = First and second frontal sulci.
f_3 = Precentral sulcus.
t_1, t_2 = First and second temporal sulci.

The anterior boundary of this region has already been given; the fissure of Sylvius forms its lower boundary, and the intra-parietal sulcus, and a line drawn as a prolongation of this sulcus to the upper end of the ascending branch of the calloso-marginal sulcus forms the posterior boundary (Mills[11]). This is the great psycho-motor region of the

cortex, in which lie the centres which preside over movements of the opposite half of the body. It is to be expected, therefore, that a destroying lesion in this region will give rise to symptoms of motor deficit, and an irritating or discharging lesion to symptoms of motor irritation; or, in other words, that a destroying lesion will cause paralysis and a discharging lesion spasm. This is precisely what is found in many cases. Of the few cases here collected only Case VIII is typical. Case V gives no symptoms, as might be expected from the slight character of the lesion. The exact location of the lesion in Case VI is not easy to determine; it may have involved the leg centre, although from the facts reported at the autopsy, it is difficult to see why the arm centre was not also affected. The lesion in the medulla, however, may have produced the paralysis. Case VII must, of course, have affected only the lowest part of the region, if it affected it at all. This is not, strictly speaking, a tumour, but the inflammatory product was so large that it may fairly be regarded as such.

Physiological experiment and clinical and pathological research have firmly established the theory that this region contains the centres presiding over the movements of the opposite side of the body. Furthermore, it is pretty clearly proven that the centres for the face lie in the lower part of this area, the centres for the arm in the middle part, and the centres for the leg and trunk in the upper part. Hence a destructive lesion of one of these regions ought to produce paralysis in the corresponding portion of the other half of the body. The cases under consideration, while few in number, help to confirm this view. It is safe to say that any tumour in this region, excepting slow-growing tumours of the dura which press on the centres without destroying them, will cause paralysis of greater or less extent. Bramwell[13] gives a remarkable example of one of these slow dural

growths in the Rolandic region, which attained a large size and compressed this portion of the cortex without causing any focal symptoms.

These centres, however, are not divided from one another by any sharp boundary. Each half of the body undoubtedly has some motor representation in the cortex of the same side, through the uncrossed fibres of the pyramidal tract, and in the centre for one segment of the body other segments have probably some representation. Luciani [23] was the first to point out that, although, for instance, the chief representation for movements of the leg was in the upper part of the Rolandic region, nevertheless, there was some representation in the middle part, and perhaps very slight representation in the lower part. Horsley, Schaefer and Beevor [22, 24] have confirmed this hypothesis of the overlapping of centres, or, more strictly speaking, of the more general representation of different segments of the body.

Subsequent research has rendered it probable that we can subdivide this great motor area much more minutely. In the neighbourhood of the pre-central sulcus, lying just in front of the sulcus, and possibly a little back of it in the ascending frontal, and running upwards and overlapping on the medial aspect of the hemisphere, Horsley and Schaefer [22] place the centres for conjoined movements of the head and eyes to the opposite side. In the upper portion of the face centre they place the centres for the movement of the eyelids. Below this are the centres for elevation and retraction of the angle of the mouth, for the tongue, for opening the mouth, for mastication and for the muscles of the pharynx and larynx. In the lower end of the ascending frontal anteriorly Semon and Horsley [25] place a centre for the vocal cords. In the arm area Beevor and Horsley [24] find the muscles represented from above downwards, in the order of shoulder, elbow, wrist, finger, thumb, the elbow below the

shoulder and posterior, the wrist next below and anterior, the fingers lower and also anterior, the thumb lowest and posterior, about opposite the lowest point of the intra-parietal sulcus. Even the movements of these joints have been separately mapped out, the shoulder from above downwards showing movements of advance, abduction, rotation outwards and adduction; the elbow, movements of extension, confusion, flexion; the wrist, extension, then flexion and pronation, confusion, and supination. In the

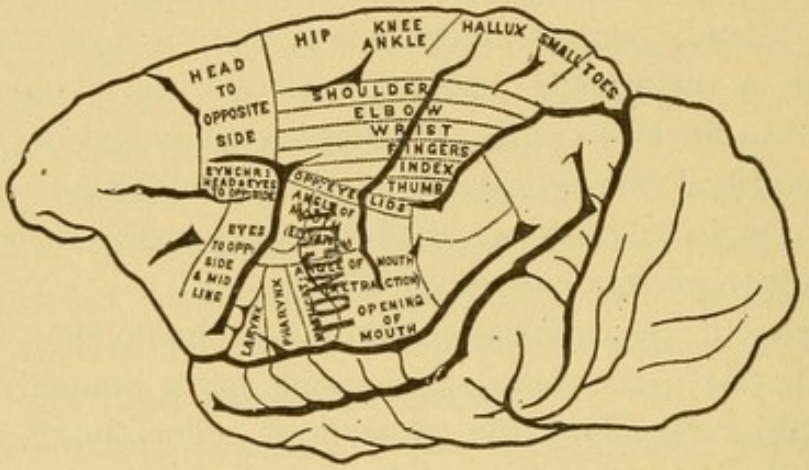

FIG. 8 (Horsley). Motor centres in the monkey's brain.

fingers and thumb they find that extension is more marked in the anterior region and flexion in the posterior. The movements of the leg have a less extensive representation in the cortex and the centres are less highly differentiated. The centre for the hallux is about the upper end of the fissure of Rolando. The toes are represented behind this, the knee and ankle in front; the knee being nearer the median line than the ankle, and the hip still farther forward. The ankle has very little primary representation, and the knee is feebly represented over the whole area. The shoulder and arm are represented slightly in the median aspect just back of the head. Then further back, extending

in monkeys almost to the parieto-occipital fissure, are the centres for extension of the hips, flexion of the knee, and movements of the toes and foot. In this whole region the most distinctive areas are those for the most highly purposive movements, while joints which are very rarely called upon for a primary purposive movement, such as the knee and elbow, are only diffusely represented.

The recent work of Horsley, Schaefer and Beevor is more elaborate than any yet undertaken. It is probably to be accepted in its main lines, although certain special points are still in abeyance. The finest differentiation comes from the results of electrical stimulation, although the results of ablation confirm the main features. It is doubtful whether a destroying lesion in the human brain small enough to cause a deficit of a single motor representation often occurs, or, if it does occur, whether it would give rise to enough other symptoms to render a diagnosis possible. With a growth the size of that in Case VIII we should expect a deficit in all the motor representations of the arm, as actually happened. Nevertheless, a careful consideration of these finer subdivisions of the motor centres may often prove of value in the study of paralysis from cortical growths. Oppenheim [15] has recently shown once more that local paralyses do not necessarily indicate a growth in this region — a fact of which I have already spoken.

I have already spoken of the significance of partial epilepsy as an indication of irritation of the cortical motor centres. These same researches give the anatomical reasons for the order of march of the spasm, which was first laid down by Hughlings-Jackson from clinical observation. In stimulating the upper part of the arm centre, the order of motion, or of spasm, is shoulder, elbow, wrist, and hand; in stimulating the lowest part, the thumb, fingers, wrist, elbow, and shoulder. In the mid-region in the parietal the move-

ment begins with the elbow, in the frontal with the wrist. In the leg the spasm is more apt to begin in the hallux or toes, the order of march being hallux, toes, ankle, hip, knee; in the other and rarer forms of movement, originating in the other joints after stimulation, the order of march respectively was ankle, toes, hip, knee; knee, hip, ankle, toes; and hip, knee, ankle, toes. This is true for the monkey, and clinical observation seems to confirm this march in man; but Horsley and Beevor have failed to produce the march in the orang-outang.

Although spasm following this type is not proof of a gross lesion of the cortex, as I have already said, it is nevertheless more probably due to such a lesion than to any other disturbance. If, therefore, the partial spasm is accompanied with partial paralysis the evidence of a cortical or subcortical growth becomes very strong. The spasm, unlike the paralysis, has comparatively little value for exact localization. It may arise from irritation in almost any part of the periphery of a growth, or even from comparatively remote irritation, and Case VIII shows how the initial spasm may vary in its distribution at different times, owing doubtless to irritation at different points in the periphery of the growth. Spasm helps to localize the lesion in the motor cortex as distinct from other parts of the motor tract, but paralysis is a much more important symptom for the exact localization of the lesion in the cortex.

Case VIII presents another symptom which must be considered here, — namely, the disturbance of sensation. According to Ferrier [26] the functions of the Rolandic region are purely motor, and the centres for sensation lie on the median aspect of the brain in the limbic lobe. This view is supported by Schaefer [27] and Mills,[28] and to a degree by Horsley,[29] who thinks, however, there is some sensory representation in the Rolandic region. Munk [30] and Luciani,[23] however,

hold that the sensory centres are situated in the same region with the motor centres in the central convolutions, and Seppilli,[23] Starr[31] and Dana[32] have supported the results of their experiments by a mass of clinical evidence. I will not discuss the question here, but I hold to the views of Munk and Luciani, without necessarily committing myself to the entire doctrine of Munk that this region is purely sensory and that the motor disturbances are due to the total loss of muscular and other sensibility. A case under my own observation affords one of the most exact experiments on man yet reported.* The patient had cortical degeneration following an old injury. Before the operation his sensibility in all its forms was tested, and found normal — the reaction time, owing to his mental impairment, being a trifle slow. He was trephined, and during the operation the cortex of the left ascending parietal, about on a level with the first frontal sulcus, was somewhat lacerated in the effort to tie a vessel; a small bit of the cortex, about three by six millimetres and two millimetres thick was excised in the same region. A circle of a centimetre and a half in diameter would have covered the whole area where the cortex was injured. Two days later, and for a period of at least seven weeks after the operation, he had anæsthesia of the right arm up to the elbow for touch, pressure, motion and position; sensibility to pain, heat and cold was retained. There was also impairment of highly purposive movements, inco-ordination from loss of muscular sense, and a little athetosis. The tactile anæsthesia persisted for at least seven weeks, but the other symptoms improved somewhat. Beevor and Horsley have found, on the whole, more pronounced motor representation in the ascending frontal than in the ascending parietal. It may be that sensory representation is greater in the ascending parietal. At all

* Trans. Amer. Neurol. Assoc. 1890.

events, I believe that in this central region of the cortex are the centres for tactile sensibility of the opposite half of the body, the leg above and the face below, the arrangement being the same as with the motor centres. The clinical evidence of sensory disturbance from lesion of the limbic lobe seems to me very slight.

In some cases of paralysis we find the post-paralytic disturbances of motion. Contracture is, of course, the commonest, and is found with any destructive lesion of the motor tract, whether cortical or capsular. Other disturbances, athetosis, chorea, ataxia, and the like, are much more rare. They, too, have no special focal significance, occurring with disease anywhere in the motor tract.

Tumours in the left central region may give rise to other symptoms of a very definite localizing character. Tumours situated at the posterior end of the left third frontal convolution in right handed people give rise to true motor aphasia. The power of voluntary speech is lost, unless perhaps for a phrase, an interjection, or an oath, but the comprehension of written or spoken language, and the ability to express ideas by writing or gesture may remain intact. Where the centre is not absolutely destroyed, but merely invaded or irritated by a growth in the neighbourhood the power of speech is not wholly lost, but there is more or less difficulty, either hesitancy of speech or imperfect articulation, or failure to recall names or nouns. It has been suggested, too, that in the posterior end of the left second frontal convolution is a motor centre for written language, differentiated from the motor centres for the hand and arm. Destruction of such a centre would produce inability to write, ability to speak and to understand written or spoken language being retained. As the existence of such a centre is still doubtful no definite rules for diagnosis can be laid down. Should agraphia be found, without any other speech disturbance, we

might fairly suspect a lesion in this region, but it would probably be attended with some motor disturbance of the hand or arm.

c. Tumours of the Posterior Parietal Region.

CASE IX. N. R. IX–124. James P. entered the hospital the 14th of August, 1880. He had suffered with œdema of the feet and dyspnœa for two years. The heart was enlarged and systolic murmurs were heard. There was also crepitation and dullness in the lungs. The urine showed albumen and casts. There was choroiditis and redness of the optic discs, and the vessels of the fundus were not very full; the condition was ascribed to tobacco and not to nephritis. He was sluggish and stupid, and had much diarrhœa. A week after entrance there was some swelling in front of the right ear; his voice was husky, and he had an epileptic convulsion. Three days later, on the 24th, he had spasmodic twitchings and died.

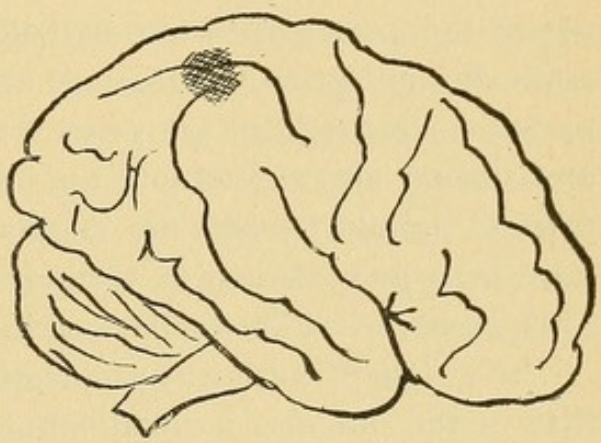

FIG. 9 (Dalton). Case IX.

Autopsy. — There was advanced nephritis. A tumour the size of an English walnut was found adherent to the dura over the right parietal lobe an inch and a quarter from the median line. There was slight compression of the brain.

The antero-superior boundary of this region has already been described. The boundary between the parietal and temporal and occipital lobes is indeterminate. Obersteiner[33] suggests as a boundary a line drawn as a projection of the

internal parieto-occipital fissure, and another drawn from the end of the fissure of Sylvius to the lateral occipital sulcus, which is not very constant, and these boundaries I adopt for convenience.

The present case teaches us little, and all its symptoms may have been due to nephritis. The functions of this region are still in dispute, and exact localization of lesions in it is not easy.

The collection of cases by Naunyn[34] and Starr[35] makes it probable that in the neighbourhood of the left angular and supra-marginal gyri is the visual centre for language. A lesion here will give rise to the symptom of word-blindness, where the power of vision, of voluntary speech, and of the auditory perception of language are retained, but the ability to understand written or printed words is lost. The visual centres may also extend into the lower portion of the inferior parietal lobule toward the angular gyrus, so that a lesion here may give rise to a certain degree of soul-blindness. This, however, will be spoken of in the next section.

The weight of authority seems to be in favour of the view that in the inferior parietal lobule are the centres for the various forms of muscular sense. The growth in Case VIII probably involved this region, but the results of the operation just cited confirm the views of Munk and Horsley that the muscular sense has a representation, perhaps only partial, in the Rolandic region. My patient recovered a part of his muscular sense in the seven weeks that followed the operation. The sense of position was fairly good, that of motion was still diminished. Further research may enable us to speak more definitely. Loss of muscular sense without paralysis might lead to a suspicion of lesion in this region.

In some cases of lesion about the angular gyrus, ptosis has been observed on the opposite side, and some authorities think that the upper facial centre may be in this region.

Munk[30] places the oculo-motor centre near here, in close relation with the visual centre. Schaefer[27] and Brown[36] obtained conjugate movements of the eyes by stimulating the angular gyrus and the occipital lobe. Horsley,[24] however, as I have said, places the centres for the eyelids and conjugate movements of the head and eyes near each other in the Rolandic region. We cannot therefore, as yet, draw any positive conclusions from oculo-motor disturbances, which will enable us to localize lesions in this region.

Ferrier[26] holds that in the angular gyrus is the centre for distinct vision of the opposite eye, and that this whole region is the chief visual centre, lesion of which causes a crossed ambly opia. This subject will be discussed in the next section more fully. Lesions of the gyrus, if deep-seated, may involve the optic radiations of Gratiolet and thus cause hemianopsia, but I do not believe that the gyrus itself, at any rate in man, has much to do with vision, nor does it seem at all probable, in spite of the views of Charcot and Gowers, that the retina of one eye is entirely represented in the opposite half of the brain.

d. Tumours of the Occipital Region.

CASE X. M. R. CCXXXV–129. Johanna S., 40, married, was admitted to the hospital on the 6th of November, 1885. She was very stupid and had indulged freely in beer. For a month she had had pain in the head, weakness and diarrhœa. For three weeks she had had a cough, chilly feelings, thirst and epistaxis. There were dry râles and dullness over the right lung and the liver was enlarged. Two days later she suddenly died.

Autopsy. — There was chronic adhesive pleurisy, interstitial pneumonia, and a fatty and cirrhotic liver. There was a calcareous nodule in the posterior part of the occipital lobe.

CASE XI. N. R. X-262. Mary S., 55, married, entered the hospital the 3d of January, 1881. Ten weeks before entrance she had trouble in the head and vomiting which lasted for three weeks. Four weeks before entrance the pain returned and became intense in the head and face. There was excessive vomiting. For three weeks she had been unable to talk plainly. She knew what she wanted to say and could understand what was said to her but she could not herself be understood. Two or three weeks before entrance she had some difficulty of vision. It was found on examination that she moved the left side somewhat better than the right. Two days later, she would answer questions only by the word, "yes." She was noisy and somewhat delirious. She used her left hand more than she did her right. On the 10th she talked considerably but did not seem to understand what was said. On the 26th there was considerable irregular action of the right arm and hand, and a tumour, which pulsated slightly, was noticed in the neck under the sterno-mastoid. A few days later she died.

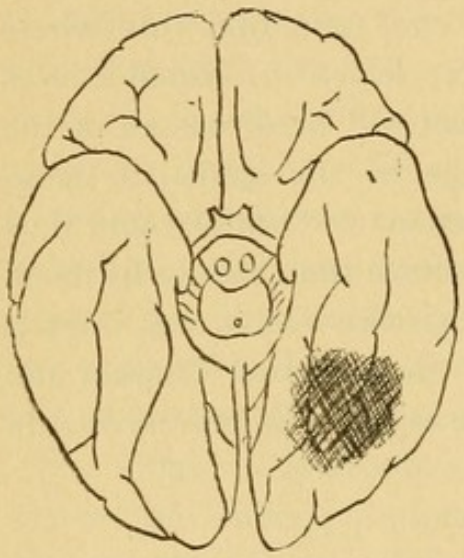

FIG. 10 (Ecker). Case XI.

Autopsy. — There was old pleurisy and some phthisis. The inner surface of the dura was snuff-coloured, with detachable bits of membrane hanging from it. The dura was adherent on the inferior surface of the left posterior lobe, where there was a tumour the size of a lemon, with variegated surface, whitish, with radiating lines and opaque yellow masses. Gumma.

The boundary of this region is also uncertain. The oc-

cipital and temporal lobes run into one another, and there is no distinct division between them. Giacomini[37] assumes as a boundary the line of the upper margin of the petrous portion of the temporal bone, which sometimes leaves a slight, temporary furrow on the fresh brain. Obersteiner's[33] boundary, which I shall adopt, is the projection of the internal portion of the parieto-occipital fissure on the external aspect of the hemisphere.

The two cases just cited afford us little, if any, information. In Case X the tumour was probably an old tubercle which had become calcified and inert, producing no symptoms. In Case XI the location of the tumour, as given in the autopsy records, is vague; and we have no information in regard to certain important symptoms, especially in regard to any disturbance of vision. Whether the disturbances of speech and motion were due to downward pressure on the medulla or not we cannot say. It is not probable from what we know of the localization of cerebral functions that such disturbances should be due to lesion in this neighbourhood, but without further knowledge this case cannot be made use of for any definite contribution to our knowledge of the localization of function.

Ferrier, in the first edition of his work on the Functions of the Brain, held that the centre for vision was in the angular gyrus. Munk[30] opposed that view and maintained that the centres for vision were in the occipital lobes, the right halves of each retina being represented in the right lobe, the left halves in the left; each eye was therefore represented in both hemispheres. Munk's views have received further confirmation from the researches of Luciani[23] and Schaefer,[27, 36] and Ferrier[26] has modified his views so far as to admit that both the gyrus and the cuneus are visual centres. A large amount of support for this theory has been obtained from human pathology, notably by Wilbrand,[38] Seguin[39] and

Hun.[40] The weight of evidence, therefore, goes to prove, as Nothnagel[34] maintains, that the centres for vision of the homonymous halves of the retina are situated in the cuneus and probably in the first occipital convolution on the same side. Luciani failed to confirm Munk's hypothesis that the different segments of the retina were represented in different portions of the visual centre in the cortex, but Hun's remarkable case seems to confirm Munk's view. Here a lesion of the lower half of the cuneus caused blindness of the corresponding lower quadrants of the retinae on the same side, and consequently there was limitation of the field of vision in the corresponding upper quadrants of the field on the opposite side.

It is to be remembered that the image projected on the retina is reversed by mental adjustment, and therefore, although the retina is blind in its homonymous halves on the same side as the lesion, the blind fields are so projected as to seem on the side opposite the lesion. Lesion of the optic tract, anywhere from the cuneus to the chiasma, as will appear later, will give rise to lateral homonymous hemianopsia; but if the lesion be in front of the occipital lobes, there will probably be motor or sensory symptoms in addition. In lesions of the cuneus there are no focal symptoms except hemianopsia. Tumours involving this region, therefore, may be localized if, in addition to the general symptoms, this symptom is present.

Wilbrand[41] and Hun, furthermore, support the view of Munk, that the remainder of the occipital lobe serves as a centre for the higher visual representations, and that, if it be destroyed, soul blindness follows. In such cases the power of sight is retained, but the power to recognize what is seen is lost. Word blindness is, in fact, only a sub-division of soul-blindness. The value of this symptom in the focal diagnosis of brain tumours is yet to be determined, but, when

present without hemianopsia, it should lead us to expect a lesion in the occipital lobe, outside the true visual centres in the cuneus and first occipital.

e. Tumours of the Temporo-sphenoidal Region.

CASE XII. N. R. XVI–80. William W., 40, married, a baker, was admitted to the hospital the 5th of September, 1882. For a year he had complained of pain in both sides of the head, and for two months he had had constant frontal headache. He had occasionally had delirium lasting for a few days. No other symptoms were noted. A week after admission he was seized with convulsions and vomiting. He became comatose; his temperature rose; the pupils were motionless, the eyes turned to the left, the head turned to the right. He remained comatose and died on the 17th.

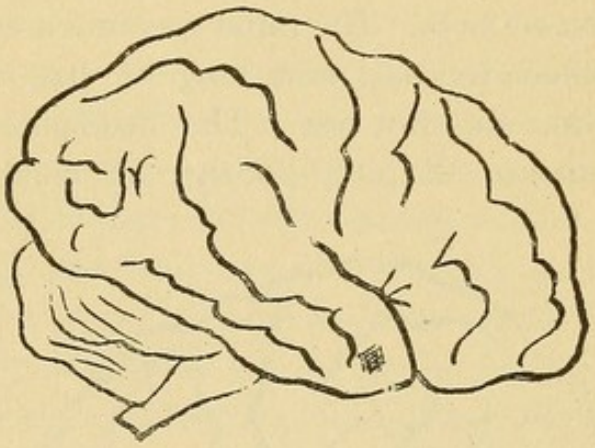

FIG. 11 (Dalton). Case XII.

Autopsy. — The dura was about two millimetres in thickness over a space four by fifteen millimetres over the right convexity. In the right middle fossa the dura was adherent to the pia over an area of two square centimetres. There was a nodule the size of a pea in the cortex of anterior portion of right temporal lobe. There was a soft, pulpy area a centimetre in diameter, with the surrounding tissue red and softened, in the left corpus striatum. There was chronic endarteritis.

CASE XIII. N. R. XXXVIII–124. Daniel H., 49, married, entered the hospital the 11th of June, 1887. There

was a history of some alcoholic excess. Six weeks before entrance he had a sudden attack of vertigo, followed by loss of consciousness which lasted half an hour. Ever since then he has had constant pain in the head, especially on the right side. There was great weakness, but no paralysis. He slept poorly. The vision was good. The appetite was good, but he had lost flesh. He had a cough and some expectoration. There was a systolic murmur at the apex of the heart, and râles at the apex of the right lung. The knee-jerks were somewhat increased; the sensibility to touch was slightly diminished. There was considerable mental impairment. Romberg's symptom was present. On the 17th he was very weak, unable to stand, and several times he fell out of bed. The mind was much affected; he did not know where he was, how long he had been in the hospital, or what month it was. The headache increased. On the 21st he was sleeping poorly; he complained of much frontal headache, and required restraint. An examination of the eyes showed considerable optic neuritis. On the 26th left hemiplegia developed. On the 28th there was no facial paralysis, but he could not get his tongue beyond his teeth. He spoke very little, and that indistinctly. He became drowsy and was very hard to rouse. The drowsiness increased to coma and on the 2d of July he died.

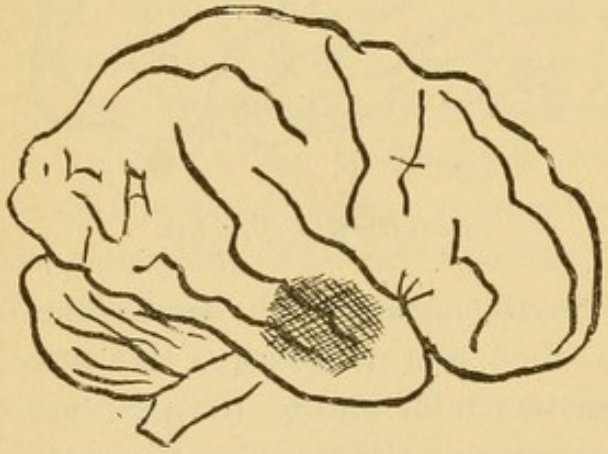

FIG. 12 (Dalton). Case XIII.

Autopsy. — There was pulmonary œdema, hydrothorax, ascites, and an atheromatous aorta. A tumour the size of a small lemon was found in the right temporo-sphenoidal

lobe; at its outer aspect was a clot, and on its inner aspect softening.

† CASE XIV. N. R. XLIII–114. Angus M., 42, married, a carpenter, came to the out-patient department of the hospital the 3d of February, 1888. The history he gave was rather contradictory. His previous history had been good. He denied syphilis, but he admitted an excessive use of tobacco and a moderate use of alcohol. He had had considerable trouble and anxiety. Three years before this time he had poor appetite and thickness of speech, with failure of memory. There was no history of any apoplectic seizure. The memory improved after rest, but within six months the thickness of speech had increased. He had much headache, which was worse at night, and was usually on the left side of the head. There was some pain about the knees but none in the shin bones. For a year and a half he had not done his work as well; and the memory had again failed. The hands were somewhat numb, and he thought the left side was weaker. The speech was jerky, thick and hesitating, the syllables were slurred, and he stumbled over them. He occasionally used the wrong word. He understood what was said but he seemed slow of comprehension. Somewhat later, in the course of conversation, he said a few words but he could not remember what he wished to say to complete the sentence, being unable to recall the name of the object. He could strike a high note with his voice and hold it fairly well. There was no trouble in swallowing. He thought there was a blur before the left eye. There was considerable mental dullness when the headache was severe. His strength failed; he was somewhat depressed but he had no delusions. The vision was tested and found to be good. The movements of the eyes and pupils were normal, and the fundus of the eyes was also normal. There was a little tenderness over the back of the neck to the left of the spine. There was no fibrillary tremor

of face or tongue, or tremor of the hands. His grip was strong; the knee-jerk was normal; the heart was enlarged, with a murmur at the apex. The urine showed a trace of albumen. The hand-writing was quite suggestive of paralytic dementia. Three weeks later, on the 23d, he entered the hospital. At that time the pupils were found to be a trifle sluggish, but equal. The gait was unsteady, he swayed with the eyes shut. The sensibility to touch in the legs was a trifle diminished. The knee-jerk was exaggerated and there was slight ankle clonus on re-enforcement. The speech became more and more incoherent and finally he scarcely spoke. He was stupid, delirious, and required restraint. The appetite was at first good, but finally grew poor. He was costive, and passed his urine and fæces in bed. He had repeated convulsive seizures, with twitching of the arms, especially of the left arm. Bedsores developed; he had strabismus; the knee-jerks were lost; the convulsions became more frequent, and on the 10th of April he died.

Autopsy. — Acute pleurisy; hæmorrhagic infarction of the lung; thrombosis of the pelvic plexus. The calvaria was slightly thickened; the convolutions were flattened. The left hemisphere was less tense, and was flaccid and fluctuating. The cavity of the left lateral ventricle was distorted. In the middle of the centrum ovale there was a cavity the size of a lemon with smooth walls, and containing a clear yellow fluid. From the anterior wall projected a vascular mass the size of a small peach, which, on section, showed alternate grayish gelatinous and also opaque yellow portions. From the wall of the cavity projected numerous sessile masses the size of small peas and of a structure similar to the large mass. The cavity reached to within four millimetres of the fissure of Sylvius and had distorted it greatly. Microscopic examination showed the tumour to be a glioma.

It may be doubted whether this last case can be classed among the tumours of the temporal lobe, but a growth of this size involving the basal ganglia and having these relations with the fissure of Sylvius, would probably involve a considerable portion of the lobe, together with the surrounding parts.

The temporal lobe may fairly be regarded as one of the latent regions of the brain, and most writers agree that lesions here apparently give rise to no focal symptoms. The external aspect, especially the posterior portion of the upper temporal convolutions is believed to be the centre for hearing. Ferrier,[26] Munk[30] and Luciani[23] substantially agree on this point, but Schaefer[27] and Sanger Brown[36] maintain that this region has nothing to do with hearing; that complete extirpation of both lobes causes no deafness. It is believed, moreover, by some anatomists, that, after destruction of the auditory apparatus, degeneration can be traced upwards into the temporal lobe; and one or two cases are on record where disease of both temporal lobes caused deafness. I think it probable that this centre contains the auditory centres. Luciani holds that each ear is represented in both hemispheres, in a manner similar to the representation of the eyes. This would explain why a unilateral lesion does not cause deafness. Our tests for hearing are still so incomplete in comparison with those for vision, that we have no knowledge as to any changes in hearing from disease of one temporal lobe, with one exception. As the centre for visual perception of language is unilateral, so is the centre for auditory perception of language; and the clinical researches of Seppilli,[23, 42] Naunyn[34] and Starr[35] bring much evidence in favor of the belief that the posterior end of the left first temporal convolution contains the centre for the auditory perception of speech. A lesion here, then, will cause word-deafness, where hearing, voluntary speech, and

the ability to read are preserved, but where the power to understand the spoken word is lost. It is, moreover, probable that in this region are also centres for soul deafness, where the ability to comprehend the meanings of sounds apart from articulate language is also lost; but this fact is by no means so sharply to be distinguished from the faculty to understand the spoken word as is soul blindness from word blindness.

I have already spoken of the theory, supported by Ferrier and Schaefer, that the centres for tactile sensibility lie in the median aspect of the temporal lobe and the gyrus fornicatus. Cases XIII and XIV showed some diminution of sensibility, but the distribution in Case XIV did not suggest anæsthesia from unilateral cerebral disease, both legs being affected. In this case, however, the patient's mental condition was such as to render his answers to inquiries about sensibility quite untrustworthy.

There is some reason for supposing that the temporal lobe bears a relation to the sense of smell; the hippocampus is well developed in osmatic animals and less developed in anosmatic animals. Ferrier is disposed to locate the senses of smell and taste in the anterior part of the temporal lobe. Our knowledge of the disturbances of those senses is still too vague to render it of much help in the localization of disease, so that we can lay slight stress upon such disturbances for focal diagnosis.

Cases XIII and XIV present one symptom — the swaying with the eyes shut — which is not very common in brain disease, and may, perhaps, arise from the presence of a lesion near the organs of equilibrium, — the semi-circular canals. This may on further investigation prove of some help in diagnosis. Case XIII presents motor symptoms which may have been due to pressure. Case XIV presents symptoms of slight motor aphasia, with apparently no word deafness.

This, again, seems to me due rather to a slight impairment of some of the speech centres from remote pressure than to actual destruction of the centres by a growth. Beyond this the symptoms in all three cases were general, and all presented, to a marked degree, mental impairment. In only one (Case XIII) was the diagnosis of a tumour made; Case XIV was regarded by every one who saw it as a typical case of paretic dementia. None of them presented any definite localizing symptoms, thus justifying the statement already made that the temporal lobe, with the exception of the posterior end of the left first temporal convolution, is a latent region, in which neoplasms give rise to no focal symptoms.

2. Tumours of the Corpus Callosum.

Case XV. N. R. VIII–17. James W., 42, single, a salesman, entered the hospital the 26th of December, 1879. He had had malaria but he denied having had syphilis. Six weeks before coming to the hospital he had gonorrhœa, and, with that, he had severe pain in the neck extending to the head, a pain which continued at intervals. Four weeks before entering the hospital his knees grew weak, he staggered in walking, and was afraid to go down stairs without assistance. A few days after he had tremor in the feet, extending all over the body, and while it lasted he was perfectly helpless. His memory failed, and he was delirious and violent. There was no numbness or prickling in the legs, and no pain in the legs except that due to varicose veins. There was no awkwardness in using the arms. There was a slight tremor of the legs; the knee-jerks were normal. The pulse was slightly irregular, 102. The urine contained albumen and casts. There was no optic neuritis. On the 1st of January he had nausea and vomiting and some delirium. The headache increased, the appetite failed, and later on the pupils became

dilated, although not immobile, and there was a hesitancy in the movements of the right eye. On the 10th of February he died.

Autopsy. — The convolutions were flattened. There was a tumour the size of a turkey's egg, involving the whole corpus callosum, grayish, vascular, sharply defined, with a soft centre, and containing a yellowish fluid. The corpus striatum and other basal ganglia were flattened. Back of the tumour in the right hemisphere was a small hæmorrhage.

† CASE XVI. N. R. XX-112. Peter M., 58, widower, a cabinet maker, entered the hospital the 13th of August, 1883. The previous history was good. He had used alcohol moderately. On the 3d of July he did considerable lifting, after which he had an attack of vertigo. Since then the vertigo had recurred with considerable frequency, and in some of the attacks everything looked dark, and he lost consciousness and fell. No information could be obtained as to any convulsion. These attacks became less frequent and less severe. There was no tinnitus or diplopia. The vision was good. The memory and mental power were somewhat diminished. He slept well. He complained of numbness and prickling in the hands, and said that the left leg had lost strength, and that he could not use it as he wished in walking. There were no thoracic or digestive symptoms. Micturition was attended with some pain in the back. He was obliged to pass water in the night, the demand being imperative, and, if it were not answered, vertigo would come on. The reflexes were normal. The arms were of equal strength. The left leg was weaker than the right. The urine was normal. He could hear the tick of a watch with either ear at a distance of one foot. The movements of the eyes, field of vision, and fundus were normal. There was no facial paralysis. A week after entrance he complained of headache, a heavy feeling in the head, and some vertigo. He grew somewhat delirious and

confused. He tried to walk but fell on the floor. On the 22d he had left hemiplegia coming on gradually and affecting the tongue, arm and leg. The watch was heard at only one inch from the left ear. He became restless, delirious and demented. Five days after the hemiplegia appeared he had contracture and exaggerated reflexes on the paralyzed side, with vaso-motor paralysis. He complained of a crowded feeling at the root of the nose. He grew suddenly weaker and more demented, restless, and delirious, passing urine and fæces in bed. The left side of the face became paralyzed; he had difficulty in swallowing. On the 6th of September the contracture of the left arm was much diminished, and on the 11th he died, having gradually grown weaker and become comatose.

Autopsy. — There was pulmonary œdema and emphysema, leptomeningitis, ependymitis, and dilated ventricles. The convolutions were flattened, the pia dry. The posterior two thirds of the corpus callosum, the posterior half and pillars of the fornix, and the right gyrus hippocampus were made up of a firm pale-red mass, a vascular sarcoma, which extended into the hemispheres, one centimetre on the right and half a centimetre on the left. The basal ganglia were not compressed.

Tumours of the corpus callosum are rare. Bernhardt[1] cites only three cases in four hundred and eighty-five, and few writers have classified them under a separate heading. Bristowe,[43] however, has reported four cases, and he thinks that the symptoms may sometimes be sufficiently characteristic to justify a diagnosis. The characteristic features, he says, are: "1st, their ingravescent character, a character which they possess in common with other cases of cerebral tumour; 2d, the gradual coming on of hemiplegia; 3d, the association of paralysis of one side with vague hemiplegic symptoms of the other; 4th, the supervention of stupidity,

associated for the most part with extreme drowsiness, a puzzled inquiring look when awake, a difficulty of getting food down the throat, and cessation of speech; 5th, the absence of implication of the oculo-motor nerves, and of direct implication of other cerebral nerves; and 6th and last, death from coma. None of these symptoms, however, seem to be especially characteristic, with the exception of the developement of paralysis, gradually affecting both sides of the body. The other symptoms, as we have seen, are by no means rare with tumours elsewhere.

Case XV seems to present no very definite symptoms. Case XVI corresponds in many respects with Bristowe's description. In several of the cases reported the duration of the symptoms seems to have been very brief, as it was in Case XVI, and it is difficult to imagine that a tumour could have attained such a size in only two months. It must be supposed, therefore, that many of the symptoms, especially the onset of hemiplegia, are due, not to the growth in the corpus callosum itself, but to the invasion of the hemispheres. With our present lack of knowledge in regard to the association tracts in the brain we are unable to recognize any deficit in the great association tract between the hemispheres, but it is possible that in the future we may be able to do so. The mental failure which seems common in these cases is perhaps due to the destruction of these tracts, but the failure is no greater than in other cases of intra-cranial growth. The gradual onset of hemiplegia is to be regarded, as I have said, as due to the invasion of the hemispheres. As optic neuritis and any marked headache and vomiting may be absent, the diagnosis between a tumour of the corpus callosum and a slowly progressing thrombotic softening seems at present impossible, even when the symptoms mentioned by Bristowe are present. If well-marked general symptoms of tumour are present a probable diagnosis can

sometimes be made. It will be seen that Case XVI presented a lesion, probably involving the hippocampus, without any sensory disturbance.

3. Tumours of the Optico-striate Region.

Case XVII. M. R. CXLIX–122. Lizzie S., 5, entered the hospital the 17th of May, 1878. For three weeks she had had a bad cough, with frontal headache and delirium. The pupils were dilated. She had no photophobia or convulsions. On the 19th she was cyanotic, the respiration was irregular, the pulse weak, and she took no notice of anything. On the 20th there was ptosis. The next day she became unconscious, and on the 22d she died.

Autopsy. — There was tuberculosis of the meninges, lungs, spleen, liver, kidneys and peritonæum. In the right lateral ventricle a yellowish-gray mass the size of a bean was found, attached to the ependyma, toward the posterior part of the corpus striatum, opposite the centre of the optic thalamus, but not implicating it.

Case XVIII. M. R. CX–214. Mrs. K. was brought to the hospital the 31st of October, 1874, in convulsions. No history could be obtained. The eyes were turned to one side and at times turned upwards, — a clonic spasm. There was coma in the intervals of convulsions. The jaws were set. There was œdema of the ankles. She could be roused a little, but died in ten hours.

Autopsy. — Advanced nephritis and pleurisy. There were ulcers in the large intestine and congestion of the brain. A tumour the size of a bean, with partly solid contents, was found in each choroid plexus.

Case XIX. M. R. CCIV–132. Andrew W., 53, single, a labourer, entered the hospital the 12th of September, 1882. His parents died of pulmonary disease. He had

had malaria. He had had a cough, with expectoration, for ten days. He had dyspepsia, anorexia and nausea, but no vomiting and no headache. He had chills daily. He became apathetic and then unconscious. On the 24th he refused food. On the 27th he was completely unconscious. He moved his arms and legs freely on pinching; the right cheek was flattened; the pupils were equal but they did not react alike; the extremities were cold. On the 28th he had a convulsion in which his legs were thrown about, the arms being motionless. The left pupil was larger than the right. His head was drawn to the right, the right eye was closed. The arms were not paralyzed; the left side of the face was less sensitive than the right; the face was drawn to the right. The left pupil finally became very much larger than the right. He died the same day.

Autopsy. — Pulmonary tuberculosis, contracted kidneys and œdema of the brain. There was a fibrous mass the size of a pea depending from the fornix.

CASE XX. S. R. CXXXI–270. Mary H., 58, widow, a domestic, was brought to the hospital the 15th of February, 1886. She was run over by a wagon and had a compound comminuted fracture of the great toe, which was amputated. The wound did poorly; she vomited a good deal. She grew more and more stupid. The head was turned to the left. On the 21st of March right tactile hemianæsthesia was discovered, the right eye also being involved; the pupils were contracted, the reflexes were increased. On the 2d of April she had right facial paralysis for a few days with some hemiplegia on the right side. On the same day she was for a time quite rational, but later she grew very stupid, and died on the 6th of April.

Autopsy. — Half an inch below the surface of the occipital lobe there was a recent clot the size of a small orange, surrounded by much softened periphery. In front of this was

an old puckered cicatrix, surrounded by an area of soft, yellowish-red brain tissue. From this, projecting into the left lateral ventricle, was a new growth (gumma) the size of the end of the thumb, quite dense, three-quarters of an inch long.

CASE XXI. N. R. XXXI–114. Edgar M., 22, single, a hatter, entered the hospital the 9th of August, 1886. He had used alcohol somewhat, and there was a suspicion of syphilis. He was said to have had severe and constant pain in the back of the head for several months. Three weeks before entrance, he went out, drank some liquor, returned and fell unconscious, without any convulsion. After that the symptoms increased. The patient stated that three weeks before coming to the hospital he was seized with a sudden and severe pain in the right side of the head, both front and back. He vomited freely, the vomiting having no relation to the taking of food. There was great mental torpor, and also weakness in the legs and sleeplessness. He had a poor appetite, diarrhœa and constipation. There was considerable vertigo. There was a steady increase in the symptoms. It was hard to get an answer; he seemed to understand what was said and to answer rationally, but he was stupid and torpid. Examination of the chest and abdomen was negative. There was albumen in the urine. The temperature was 99.4; the pulse 80. The tongue was protruded straight. The pupils were equal and reacted to light. The movements of the eyes were normal. There was left lateral homonymous hemianopsia; and there was also drooping of the right eyelid. The grasp of the left hand was weaker and he used that hand less than the right. There was much tremor of the right arm, and some tremor of the left arm and tongue. There was left hemianæsthesia for touch and localization, extending not quite to the median line. The elbow and knee jerks were much increased

ankle clonus was present; the skin reflexes were active, and the reflexes were alike on the two sides. On August 10th he was more stupid. There was a slight left facial paralysis. On the 11th he was unable to recognize his friends. He passed urine and fæces in bed. On the 13th there was found to be optic neuritis in the left eye; the right eye could not be examined. On the 14th he was drowsier, and there was less movement of the left arm and leg. The left facial paralysis was greater. On the 15th he recognized his friends. On the 16th he was again somnolent, he could not be aroused, and he did not recognize any one. He moved the right arm in a purposeless way without co-ordination. It was hard to get him to take food. Later in the day he had tonic convulsions; the limbs were rigid and extended. The pupils were unequal and immobile, the right pupil dilated and the left contracted. There was a slight tonic extensor spasm in the arms. He became comatose and died.

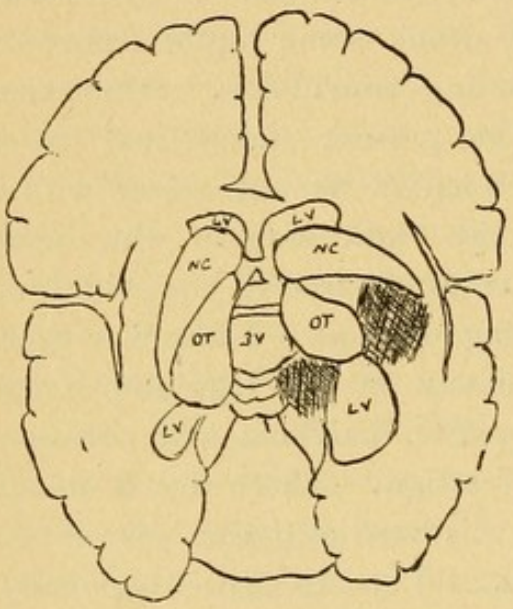

FIG. 13 (Edinger). Case XXI.

LV = Lateral ventricle.
3V = Third ventricle.
NC = Caudate nucleus.
OT = Optic thalamus.

Autopsy. — There was venous engorgement of the organs of the body and small hæmorrhages in the lungs. The calvaria was thin, the inner surface roughened, showing grooves made by the convolutions. The dura was injected, the pia dry; the convolutions were flattened, the sulci obliterated; the fissure of Sylvius was shallow, the brain flabby. The right lateral ventricle was one-half larger than the left, the ependyma

granular. In the middle horn of the right lateral ventricle was a dark red, vascular, gelatinous, globular, lobulated mass, measuring antero-posteriorly 5.5 centimetres, transversely 5 centimetres, vertically 3 centimetres, lying laterally to the optic thalamus, posteriorly to the caudate nucleus, the long diameter of the latter being nearly transverse. Between this and the cerebellum, and behind the optic thalamus, was a similar mass, half a centimetre thick, and three centimetres in diameter. The posterior cornu of the lateral ventricle was the size of a lemon, with a granular surface. A section of the larger growth showed that it was firm, opaque, yellow, with gelatinous areas — the outside was soft, purple, and homogeneous. The smaller growth was purplish, soft and homogeneous. The external capsule, on a level with the anterior tip of the optic thalamus, was yellow and softened. The lenticular nucleus, on a level with the middle part of the optic thalamus, was softened and yellow; behind this, the three parts of the nucleus were soft and yellow. The right centrum ovale next the tumour was soft and yellow for one centimetre. The right crus and internal capsule were intact. The left side of the brain was normal. Under the microscope the central portion of the tumour was found to contain large granular capsules and fatty, degenerated cells. In the periphery were large cells in masses, with fine granular protoplasm and distinct nuclei. There was a mucine reaction. There was great developement of the blood-vessels. The tumour was found to be an endothelioma arising from the choroid plexus, — a tumour of the sarcomatous type developing from connective tissue.

The optico-striate region embraces the corpora striata, with the two divisions thereof, the optic thalami, the external and internal capsules, the claustrum, and the adjacent ventricles. In point of fact, growths in this region most commonly arise

from the choroid plexus, the corpus striatum, or the optic thalamus, involving the capsules only by extension.

Cases XVII, XVIII and XIX are merely examples of the small growths in the choroid plexus, often cystic, which are found at the autopsy and give rise to no symptoms during life. All the symptoms in these cases were doubtless due to the disease elsewhere which caused death. Such growths seldom have any significance, but in rare cases, as Edes[44] has shown, they may interfere with the circulation of the cerebro-spinal fluid in the ventricles and cause an increase of pressure.

Cases XX and XXI present more definite symptoms, the former hemianæsthesia and partial paralysis; the latter hemianopsia, hemianæsthesia and hemiplegia.

It is probable that tumours in this region give rise to no focal symptoms unless the internal capsule be involved. It is probable that the corpus striatum has some connection with motor impulses, and the optic thalamus with sensory impressions and perhaps with inhibition, but our knowledge of the exact functions of these organs is still vague, nor do we know what effect a destructive lesion of either of them produces. At any rate growths limited strictly to the thalamus, the lenticular or the caudate nucleus apparently cause no focal symptoms. As the growth becomes larger and involves the internal capsule, symptoms develope which may enable us to localize it, but only when the posterior limb of the capsule is involved. If the anterior two-thirds are alone affected we have an ordinary hemiplegia, with a more or less gradual onset, often associated with dysarthria or even motor aphasia if there be right hemiplegia. In such a hemiplegia convulsions are rare and anæsthesia is absent, thus differing from paralysis from cortical disease. Should the growth involve the posterior third of the capsule, hemianæsthesia may be present, usually more general than the anæsthesia from cor-

tical disease. In such cases the pulvinar is also apt to be involved, and the fibres of the optic tract, and, as in Case XXI, we get homonymous hemianopsia on the opposite side, differing from cortical hemianopsia in being associated with hemianæsthesia or hemiplegia. With such an association of symptoms we can be reasonably sure of our diagnosis as to the location of the growth.

Beevor and Horsley[45] have very carefully studied the arrangement of the motor fibres in the internal capsule and have found that the most anterior fibres are those for the movements of the eyes; then come fibres for the movements of the head, the shoulder, elbow, wrist, fingers, thumb, trunk, hip, ankle, knee, hallux and small toes, which are the most posterior. A study of their diagrams, however, shows that the lesion which would affect one set of these fibres must be so extremely small as to cause practically no other disturbance; so that the probability of a limited paralysis from a lesion in this region is extremely slight.

Oulmont[46] and Raymond[47] hold that lesions just in front of the sensory tract, toward the posterior part of the optic thalamus, give rise to the symptoms of post-hemiplegic disturbances of movement, especially athetosis or hemichorea, — an opinion which has some support from the recorded cases, and with which many writers agree. Should such symptoms arise in any case presenting the symptoms already mentioned, they would help to confirm the diagnosis. Case XXI presented some motor disturbance but it was on the same side as the lesion. If the disturbance come on at the close of life then I am inclined to believe the symptom due to pressure. I do not, however, believe that post-hemiplegic disturbances of motion by themselves have any distinct localizing value. I have seen athetosis in cases of cortical lesion, multiple neuritis, and chronic hydrocephalus, and I believe

these disturbances may arise from lesions almost anywhere in the motor tract.

4. Tumours of the Crus Cerebri.

Tumours of the crus are rare and no true case has been found in the hospital records, although Case XXIV was sufficiently high up in the pons to present symptoms similar to those of tumour of the crus. The striking feature of new growths in the crus is crossed paralysis — a paralysis of the oculo-motor nerve on the same side as the lesion and of the face and limbs on the opposite side. For a further discussion of these symptoms, however, I must refer to the sections on tumours of the pons and tumours of the base of the skull.

5. Tumours of the Corpora Quadrigemina and Pineal Gland.

† Case XXII.* Frank C., 24, single, was admitted to the Boston Lunatic Hospital the 15th of February, 1884. There was a history of phthisis in the family. At the age of five years he had epileptic attacks. His previous history was otherwise good except that latterly he had had headaches. A year before admission, he had an attack of neuralgia on the left side of the head, the pain being accompanied with vertigo. About the same time he had dimness of vision and diplopia. At times there was vertical diplopia, when the eyes were turned to the right, horizontally, or upwards, but this diminished. The head was carried backward, in order to correct the diplopia. Later on he had some spasm of accommodation. In August, 1883, he began to have scruples in regard to his conduct, and a feeling of unworthi-

* This case has already been reported by Dr. T. W. Fisher, American Journal of Insanity, January, 1885.

ness. After this the headaches returned. At times he lost control of the movements of his hands. With this loss of control there was some difficulty in swallowing, and a loss of power in the right hand. He grew childish and lost his initiative and his power of attention. The vision of the left eye began to fail in September and by December he was entirely blind; about the same time his hearing failed. In September he began to go to the left in walking, and fell backwards to the left. The gait was reeling and the hands unsteady. There was nystagmus and squint. It was hard to determine the condition of his sensibility, but it probably was not much impaired. In February, just before entering the hospital, he lost consciousness and had a convulsion involving both arms. On entrance, the examination showed small pupils and right internal strabismus. There was double optic neuritis. He responded somewhat to irritation of the skin, showing no marked paralysis. The knee jerk was very slight on the left and exaggerated on the right. He apparently was entirely deaf and blind. The skin reflexes were normal. He swallowed his food well. He passed urine and fæces in bed. On the 16th the knee jerks were found to be equal. On the 20th pressure over the cervical vertebrae was apparently painful. There was marked plantar flexion of the feet. He lay perfectly quiet making no response. He spoke voluntarily only once during the time he was in the hospital. On the 22d there was right external strabismus. He grew steadily weaker and died on the 22d.

Autopsy. — Acute pleurisy, broncho-pneumonia, hypoplastic aorta. There was a hæmorrhage between the dura and periosteum in the cervical region of cord. The calvaria was very thin ($1\frac{1}{2}$ millimetres). The inner table was absorbed, and the diploë exposed. The brain weighed 1605 grammes. The pia was pale and dry; the convolutions were flattened; the sulci obliterated. The lateral ventricles each contained

75 cubic centimetres of clear fluid. The region of the corpora quadrigemina and pineal gland was occupied by a soft reddish-gray globular mass measuring thirty centimetres in all its diameters and extending to the corpora geniculata. Over it lay the velum interpositum. There was no trace of the pineal gland or of the anterior corpora quadrigemina. On the posterior surface of the tumour the posterior corpora formed a layer only two millimetres in thickness. The tumour was made up of small round cells in a delicate mesh, with abundant vessels. Other parts showed an alveolar structure. The tumour also contained pearly nodules, cholesteatoma. The growth was a vascular sarcoma with cholesteatomatous portions, growing probably from the velum interpositum.

CASE XXIII. M. R. CC-50; N. R. XV-88; XVI-125. Adolph S., 23, single, a sailor, entered the hospital the 8th of May, 1882. There was a family history of phthisis. He had had typhoid and yellow fevers, but he denied syphilis. About the last of April he began to have severe paroxysmal headache, with pain in the back and vomiting. There was slight photophobia and vertigo. He lost strength. The vision was good. He slept and ate well. He had no disturbance of respiration or micturition. On May 30th it was found that he had diplopia for distant vision. June 2d it was recorded that the headaches usually came on before breakfast and were attended with vomiting. There was beginning optic neuritis. On June 16th he was found to have paralysis of the right external rectus. There was some mental impairment; he acted strangely, but he had no memory of his actions afterwards. On the 28th of June he had less headache and no vomiting. For some weeks his gait was somewhat like festination. The left side felt as if a cloth were between it and the object touched. July 15th, he had marked optic neuritis, but vou $= \frac{20}{30}$. On the 24th he had various delu-

sions — some of them delusions of persecution. His gait was staggering; there was no headache; he was quite stupid, but he knew enough to elude the attendants. He soiled himself regularly. July 30th he was somewhat brighter; he had no memory of his delusions; and said he had been asleep. His headache was more severe. August 13th he was free from headache and delusions. He had moderate hemianæsthesia on the left side, and some vertigo. September 1st, the neuritis was more marked. September 5th, the vision had grown poor. He did not walk as well, which was attributed to his poor vision. On October 4th there was slight paralysis of the lower part of the left side of the face; the left eye did not move outwards beyond the median line; the right eye tended to turn inwards, but it could be moved out beyond the median line; he had diplopia on looking downwards. He had left hemianopsia and slight hemianæsthesia. The knee jerks were equal and lively; the plantar reflex greater on the left. October 5th, he had a tendency to totter backwards, and could not put the toes down to the floor. Hemianopsia was less marked. October 7th, he could not move his arms, and lay in bed, being very stupid. October 10th, there was more paralysis of the right external rectus. October 18th, the right eye moved out better. October 25th, there were slight tremors on the left side several times a day on intended movements. The tongue was tremulous when protruded. He followed the finger with his eyes when it was moved to the right; the left eye moved outwards only to the median line, and he seemed to lose sight of the finger when it was moved to the left. On November 6th the left arm was stiff and he had more left internal strabismus. November 16th, there was spasm of the arms and legs. On November 18th there was nystagmus of the right eye with a tendency of the eye to turn inwards; the left pupil was larger; there was less motion

in the left eye, which could be turned inwards somewhat. On the 18th he became comatose and died.

Autopsy. — The calvaria showed the impress of the convolutions and was very thin, three millimetres in its thickest part, and as thin as paper in the thinnest part, with an actual perforation of the skull. There were pearly nodules, the size of a pin-head on the inner surface of the dura. The convolutions were flattened; the pia dry. The left optic tract and abducens were smaller and flatter than usual, and the abducens was less translucent. The lateral ventricles were twice the normal size. The cavity of the third ventricle, the anterior half of the right lateral ventricle, and the fifth ventricle were occupied by a soft, pale, custard-like mass, eleven by six by three and a half centimetres, adherent only by small vessels to the floor of the ventricle. The growth could not have arisen from brain substance. The corpus callosum and anterior fornix were enormously stretched and lying on the tumour. There was no velum interpositium. The tumour was most adherent near the septum lucidum. The growth had greatly compressed the pineal gland and the corpora quadrigemina, on which it lay. On examination it proved to be a small round cell sarcoma.

The anatomical situation of tumours in this region causes compression of the aqueduct and venae Galeni, and thus produces hydrocephalus, giving rise to greater intra-cranial pressure than growths in any other region.

These two cases present the two symptoms recently laid down by Nothnagel [48] as characteristic of tumours involving the corpora quadrigemina, namely, inco-ordination in walking and oculo-motor paralysis. Although Nothnagel [49] had suggested the importance of these symptoms in the diagnosis of growths in this region in 1879, it is only in his recent paper that, after a study of eighteen cases (including Case XXII) he takes the definite position that this combin-

ation of symptoms, coming on early in the progress of the disease, is characteristic. Gowers, and the majority of writers before this, have ascribed the disordered gait to pressure on the vermis cerebelli, but Nothnagel cites a case where the tumour was too small to exert much pressure, and emphasizes the fact that, as in Case XXII, the disturbed gait was one of the early symptoms. In Case XXIII the disturbance seems to have come on somewhat later, but it is very possible that the corpora quadrigemina were only later involved in the growth. Oculo-motor symptoms have long been recognized as frequent in lesions in this region. We must therefore regard the combination of these two symptoms, if observed early in the disease, as indicative of lesions of the corpora quadrigemina.

The anatomy of this region shows that a large portion of the optic nerve fibres arise from the anterior corpora, and that they also receive or are traversed by many of the fibres of the optic radiations coming from the occipital lobe. Hence, *a priori*, we might expect that a lesion in this region would cause blindness, even before optic neuritis had developed, and various writers, among others Edinger,[50] regard such an amaurosis, when not due to neuritis, as of some value in the focal diagnosis. Nothnagel opposes this view. Case XXIII would seem to support Nothnagel's opinion; but it must be borne in mind that in this case the tumour merely pressed upon the corpora quadrigemina and did not destroy them, and that later, perhaps as the pressure destroyed the subjacent parts, the vision began to fail.

Some of the optic fibres arise from the thalamus and the external geniculate body. A good-sized tumour of the corpora quadrigemina may involve this region or the posterior part of the internal capsule, and thus give rise to hemianopsia or hemianæsthesia, as in Case XXIII. It is doubtful in this case just where the growth originated, but it so dis-

tinctly presented symptoms like those ascribed to tumours of the corpora quadrigemina, that I have ventured to put it in this section.

Flechsig [51] has recently called attention anew to the connection between the acoustic fibres, especially those of the cochlear nerve, and the posterior corpora quadrigemina. The clinical importance of this relation has not yet been worked out, but the deafness of Case XXII may perhaps have some connection with this anatomical condition.

6. Tumours of the Pons and Medulla.

† Case XXIV. N. R. XX-258. Samuel K., 46, married, a waiter, entered the hospital the 8th of October, 1883. He had indulged freely in alcohol, and had had gonorrhœa and a sore, but he denied any secondary symptoms. Eleven months before he entered the hospital he began to have pains across the loins, in the right leg, left shoulder and left leg, the pains being sharp and shooting in the right leg. After this he had numbness in the legs and right arm. Two or three months later he had trouble in walking, the right leg being most affected; and he did not go out much for fear of falling. He also had stiffness and cramps in the leg; the right arm was weaker than the left and was somewhat stiff. He thought that the tactile sensibility was normal. For six weeks he had constant frontal headache, and also lumps on the scalp. He had diplopia and poor vision; he required glasses for reading; his hearing was good. There were no special respiratory, circulatory or digestive symptoms. There was a trace of albumen in the urine, and one or two hyaline casts. About the time he began to have trouble in walking, he had some vertigo but the pains in the legs grew less. On examination the left eye was found to be more prominent; the left pupil was found to be twice the

size of the right; both reacted feebly to light, the right reacting more than the left. The eyes moved freely in all directions, and, at the time of the examination, there was no diplopia. The tongue was protruded to the left. The facial muscles responded naturally to the will. The right arm was weaker; the movements, when the eyes were shut, were irregular and jerky; the left arm moved naturally. The movements of the right foot were less extensive and weaker; the plantar reflex was diminished on the right. The knee jerks were exaggerated, being greater on the right side than on the left. There was patellar clonus but no ankle clonus; all the deep reflexes were exaggerated in the arms and legs. On the 10th he had slight deficiency in the action of the left external rectus. The right eye was open during sleep; the right naso-labial fold was less marked; the left eye moved less readily to the right. The fundus oculi was normal. The right hemiplegia increased; the tongue protruded to the left; the left eye moved only by the action of the external rectus and oblique; there was ptosis of the left eye and the pupil of that eye was widely dilated. The right eye was constantly kept open. The muscles of the face on the two sides reacted alike to a weak faradic current. He was dull and stupid and had much headache. The sensibility was not much affected. He grew steadily worse; bed sores formed; he became comatose and died on the 5th of November.

Autopsy. — Chronic adhesive pleurisy and bronchitis. The calvaria was of normal thickness, and adherent to the dura, which was thickened and opaque. The pia was thickened, opaque and slightly œdematous. Both vertebral arteries were adherent to the medulla; the other vessels were normal. The vessels of the brain were well filled with blood. In the anterior inferior portion of the left lenticular nucleus was a reddened, softened area, measuring about 8 millimetres

antero-posteriorly, 11 millimetres transversely, and 3 millimetres vertically. The antero-posterior length of the pons was 32 millimetres. On the anterior surface were two reddish-gray areas. On the left half was an area 15 millimetres antero-posteriorly, 13 millimetres transversely, overlapping the median line to the right 2 millimetres, and beginning 10 millimetres from the anterior boundary of pons. On the right anterior pyramid of the medulla was a similar discoloured spot measuring six millimetres each way, beginning at the median line and five millimetres from the pons. On the left half of the medulla at the fissure between the anterior pyramid and the olivary body, implicating many of the fibres of the hypoglossal nerve, was a grayish-yellow spot five millimetres by six millimetres, two millimetres from the border of the pons, and four millimetres from the median line. Over these two spots in the medulla the vertebrals were adherent. On section of the pons and medulla these areas were found to correspond with nodules. That of the pons was a single nodule, measuring 15 millimetres antero-posteriorly by 20 millimetres transversely, firmer than the brain substance, reddish-gray for about 6 millimetres about periphery, with an opaque yellow centre. In

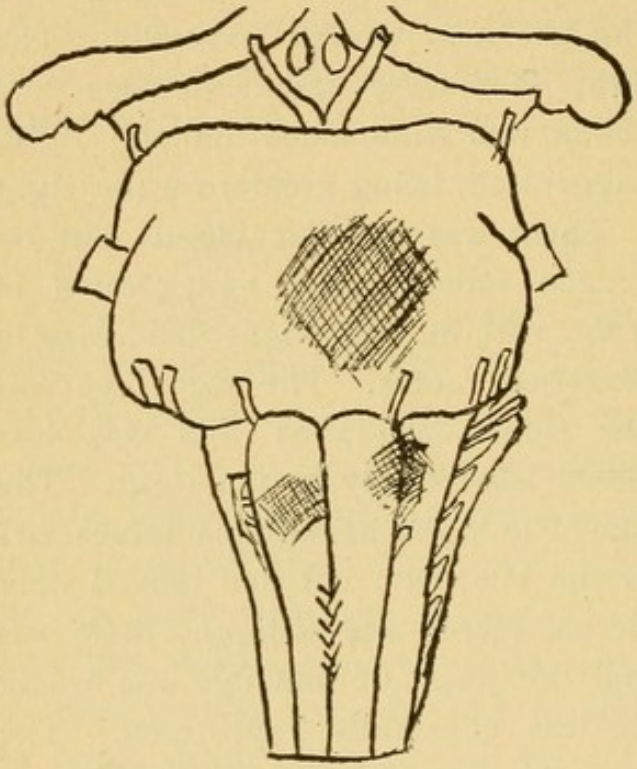

FIG. 14 (Foster). Case XXIV. Frontal aspect of pons.

the periphery were reddish streaks, evidently vessels. The nodule in the right anterior pyramid was six by four milli-

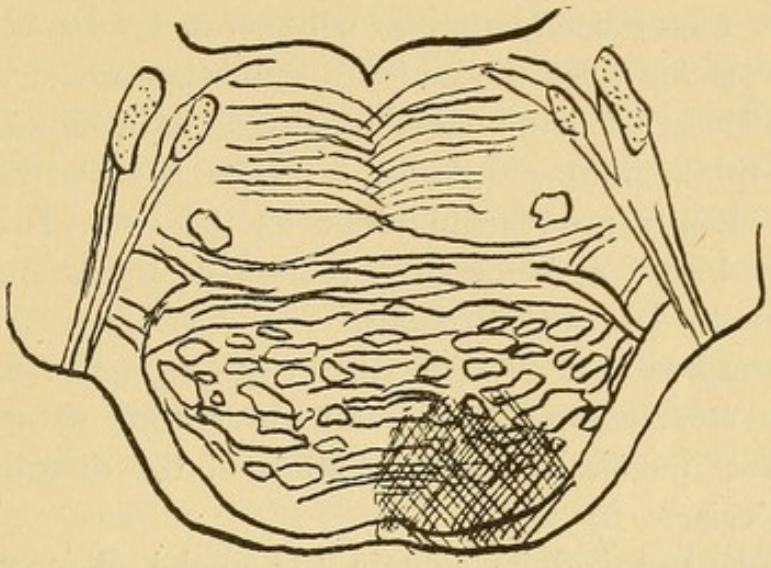

FIG. 15 (Foster). Case XXIV. Section through upper growth.

metres, with a red periphery and yellow centre, occupying nearly the whole of the pyramid. That on the left was six millimetres antero-posteriorly and one millimetre transversely. Gummata.

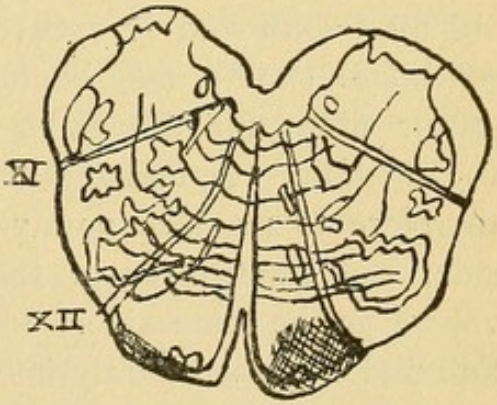

FIG. 16 (Foster). Case XXIV. Section through lower growth.
XI = Spinal accessory nerve.
XII = Hypoglossal nerve.

This case presents many peculiarities; the left third and twelfth nerves were unmistakably paralyzed, and there was right hemiplegia. The hemiplegia was due undoubtedly to the large nodule in the pons, but the lesion in the left lenticular nucleus may also have taken part in it. The lesion in the pons is also responsible for the paralysis of the left oculo-motor nerve. The lesion at the root of the left twelfth nerve in the medulla is the cause of the paralysis of that nerve. These lesions were diagnosti-

cated before death. The lesion in the right pyramid is not as easy to understand. It apparently gave rise to no symptoms. Whether it did not penetrate deeply enough into the pyramid to cut off motor conduction, or whether it developed only at the close of life when the patient's condition was such as to render a slight left hemiplegia unnoticeable is impossible to say. I think, however, that the record is inaccurate, and that the deeper growth should be on the left. This would correspond with a sketch of a section of the medulla furnished me by Dr. S. G. Webber.

The symptoms of new growths in this region of the brain are often most complex. Careful observation may help us to a correct localization after a study of the distribution of the paralysis.

The pons and medulla contain the nuclei of most of the cranial nerves, and the great motor and sensory tracts connecting the cortex with the periphery. The sensory tract probably passes upwards in the tegmentum, the motor tract passes downwards in the middle portion of the anterior border of the crusta. The nerves most commonly affected are the fifth, sixth and seventh; lower down in the medulla the ninth and twelfth may be involved. As is well known, the motor fibres for the limbs decussate at the lower part of the medulla. A unilateral lesion in the region above ought therefore to produce paralysis of the limbs on the opposite side, and disturbance of the nerve whose nucleus was involved on the same side. In the medulla we should therefore find a crossed paralysis of the tongue and limbs, in the lower pons crossed paralysis of the face and limbs; higher in the pons crossed paralysis of the limbs with facial anæsthesia or neuralgia, and still higher in the crus the crossed paralysis of the oculo-motor nerve and the limbs, to which I have already referred.

Small lesions of the pons may, rarely, affect only one or

more nerve nuclei, and spare the great motor or sensory tract. Other lesions may affect the motor tract alone, causing ordinary hemiplegia. With facial paralysis, if the nucleus of the facial nerve itself be involved, we may get electrical changes of degeneration in the facial muscles. Sensory disturbances, except of the fifth nerve from irritation or destruction of its nucleus, seem less common. With tumours lower down in the medulla symptoms of bulbar paralysis may be present, or glycosuria or albuminaria may be present from disturbances in the floor of the ventricle.

One symptom is of some significance in diagnosis of lesions of the pons, — conjugate deviation of the head and eyes. It is not an uncommon symptom in cerebral disease, where, in a case of paralysis, the head and eyes are turned away from the paralyzed side, and the patient looks toward the lesion. In cases of unilateral spasm, however, the condition is reversed, and the patient looks away from the lesion and toward the convulsed side; although this is less constant than with paralysis. In lesions of the pons this state of things may be reversed. The patient looks toward the paralyzed side and away from the lesion; away from the spasm and toward the lesion. The distinction is of some importance and may afford us help in diagnosis, but it is far from constant, and is by no means a certain guide.

7. Tumours of the Cerebellum.

Case XXV. M. R. CLXXXIII–143. John S., 28, single, entered the hospital the 9th of April, 1881. A year before entrance he was attacked with nausea and vomiting, the vomiting coming on after taking food, and usually containing a dark-brown substance. This persisted, coming on regularly four hours after eating, and three times a day. He had dull pain in the epigastrium. He grew thin, pale and

weak. Two months before entrance to the hospital the vomiting ceased but the pain persisted. He was anæmic, and had œdema of the legs. He failed rapidly, without marked symptoms. He had considerable dyspnœa and died on the 15th.

Autopsy. — He had ulcer of the pylorus, penetrating to the pancreas and liver. There was œdema of the lungs, and thrombosis of both common iliac arteries, most marked on the left side. In the posterior extremity of the right lobe of the cerebellum was a mass three-quarters of an inch in diameter, with a hard calcareous shell and putty-like contents.

CASE XXVI. M. R. CXV–154. John G., 28, was admitted to the hospital the 25th of May, 1875. No history could be obtained. The eyes were staring, the head twitching; he threw the bed-clothes off and tried to get out of bed. He passed urine and fæces in bed. He was stupid, tore his hair and slapped himself. His pulse was slow and full. On the 26th he had taches bleuâtres. On the 29th he was very stupid, the pulse grew more rapid, the pupils dilated; he frothed at the mouth, the eyes were motionless and staring, and he did not respond to a touch. His hands were cold, his face livid. On the 30th he was somewhat better and understood what was said, but he failed steadily and died the 1st of June.

Autopsy. — There was tuberculosis of the lungs, peritonæum and intestines. A cheesy mass, the size of an acorn, was found in the cerebellum.

CASE XXVII. N. R. IX–125. Louis H., 30, single, farmer, was admitted to the hospital the 14th of August, 1880. He had twice had acute rheumatism. Two months before entrance he had headache and vertigo, which were increased on sudden movements. He had nausea and vomiting in the morning, vomiting the food taken. He had

severe headache, which was most marked in the vertex. His vision was dim. An examination of the eyes showed that the discs were injected, and the outlines indistinct, but no hæmorrhages or white patches were seen. On the 19th the right pupil was larger than the left. On the 20th he complained of general malaise, and vomited occasionally; he had less headache, but he was dull and heavy. On the 24th he seemed dull, but he answered questions intelligently. A few minutes later respiration became feeble and he died suddenly.

Autopsy. — The brain was soft and waxy. In the right lobe of the cerebellum was a gelatinous tumour, the size of an English walnut, not encapsulated. The cerebellum was softened about it. The growth was a small round-celled sarcoma.

CASE XXVIII. N. R. VII–259. Tyner S., 14, single, entered the hospital the 13th of December, 1879. A sister died of convulsions. Three years before entrance to the hospital he was said to have had his head bumped on the floor and to have been struck in the belly, causing vomiting. After that he had headache and vomiting. Since that time he had vertigo on going down stairs, and he could not walk straight; he walked as if drunk. Every day there was pain in the back of the head below the occipital protuberance. He was sleepy and gaped a good deal. For nine months his vision failed. He had six attacks in which he vomited and could not move, but he had no convulsive movements. These attacks were sometimes preceded by a numb feeling extending over the body, and he was sleepy after them. The left side of the head and tongue at times felt prickly, and these attacks of prickly feeling somewhat resembled petit mal. He was somewhat unsteady on walking with his eyes open, the left foot coming down more heavily; he was more unsteady with his eyes shut. He had atrophy

of both optic nerves. The sensibility of the right hand was less than that of the left. He had an indurated scar on the back of the head. On the 19th of January he had an attack which began with a tickling feeling in the chin, running over the mouth and the back of the head. The body was somewhat stiff. This lasted about ten minutes; he was conscious after it and said that his mouth felt dry and that he had a pain in the back of the neck. He died that afternoon.

Autopsy. — The convolutions were flattened. The ventricles contained 15 ounces of serum. On the under surface of the cerebellum, in the median line between the cerebellum and medulla, slightly more to the left than to the right, was a tumour measuring three inches across, extending into both lobes of the cerebellum. The tumour contained five cysts, two very large, two very small, and one projecting just below the corpora quadrigemina. Several nerves from the medulla were thinner and less white than normal.

† CASE XXIX. S. R. CXCII-112. Robert W., 28, single, a lawyer, was seen by me on the 30th of October, 1890. At the age of seventeen months he had had spinal infantile paralysis, and the left leg was paralyzed and much wasted. The family history was good. He had used tobacco freely. For two or three years he had had more or less indigestion and constipation. Two years before I saw him he began to have headaches; it was hard to get on with him; he disagreed with his family and left home for a time. About fifteen months before I saw him, he began to have severe pain in the right occipital region, and soon after this the vision began to fail. The headache began in the spring of 1889 and continued with increased severity during the summer. Early in October he was in bed for a week on account of it; when he got up again his vision was affected; he had occasionally diplopia — sometimes seeing three or even six objects. An examination of the eyes showed that the

field of vision was good. The vision was $\frac{14}{15}$ after a slight error of refraction was corrected. There was double optic neuritis. Later there were attacks of nausea and vomiting followed by intense pain in the head. Soon after the failure of vision he lost his sense of smell. The mind was perfectly clear throughout his trouble. After that he had several attacks in which he would cry out, froth a little at the mouth and fall on the floor. The headache, nausea and vomiting persisted after that. The right eye failed more than the left, and in August, 1890, he was totally blind. About the middle of September he began to complain of poor hearing, especially in the left ear, and of a peculiar distorted, swollen feeling in the mouth, and some numbness of the hands. He talked perfectly intelligibly, but he said his speech seemed to him thick. He had been taking iodide off and on for a year, and there was some iodide eruption. He said his forehead felt as if there were a bar through it. He had sudden sharp pains in the head, especially on the left side. The left side of the face was a little swollen. He walked rather unsteadily, but until his vision failed he said he walked perfectly well. The eye-lids drooped, but he could open them easily. The pupils were equal and dilated; they reacted to convergence but not to light. The left hand was a little weaker than the right and was used a little more awkwardly. The knee-jerk was normal on the right and absent on the left. The sense of smell was lost. The hearing was very poor in the left ear and somewhat diminished in the right. The deafness was thought by Dr. J. O. Green to be of nervous origin. The sensibility to touch was good. There was no paralysis. There was a little tenderness on pressure over the right temple. He complained steadily after this of numbness in the face, a stiff feeling in the lips, and an uncomfortable numb feeling in the hands. As the headache grew more severe I advised him to enter the

hospital for operation. The focal symptoms were too few to enable me to localize the growth, but it was thought best to trephine simply to relieve the intra-cranial pressure. He entered the hospital on the 12th of January, 1891. He complained rather more of numbness of the hands, and the movements of the hands were at times uncertain, but the muscular sense was good. Observations of surface temperature gave temperatures of 95.5 to 98.3 F., the highest temperature being over the temples, the temperature being equal over the two temples. He was trephined by Dr. E. H. Bradford over the tender spot in the right temple on the 19th of January, the dura being opened, and the brain incised. The wound healed promptly, there was no rise of pulse or temperature, and he did well. On the 27th there was left facial paresis, the tongue going to the left. The hands were unaffected. There was a cerebral hernia the size of half an orange. He returned home the 28th. For about ten days after returning home he was somnolent and irritable, he complained much of soreness at the seat of the operation, but the old headaches had nearly ceased. The paresis increased to nearly complete left hemiplegia and there was left hemianæsthesia. Then the edges of the wound separated, and there was some oozing of serum, blood, and bits of brain matter. On the 14th of February there was an abundant discharge of clear serous fluid from the wound, which continued. On the 16th, he became brighter, sat up, was perfectly clear mentally, and complained only of some soreness about the wound. The headache had ceased. He continued in this condition for some time, but the hernia sloughed more and more; and finally on the 7th of March he began to have severe pain, and he became comatose, and died on the 9th.

Autopsy. — Pericranial ecchymosis near the seat of operation. Trephine opening. A large cerebral hernia involving the right upper temporal and lower frontal convolutions and the

base of the ascending convolutions. Adhesion of the meninges to the trephine opening. A large tumour was found involving most of the left lateral lobe of the cerebellum, lying close to the transverse fissure. The examination of the brain is not yet complete.

The tumour in Case XXV had no connection with the symptoms and had probably existed for years, having long ceased to grow or to give rise to any disturbance. The tumour in Case XXVI also had comparatively little to do with the symptoms presented. Cases XXVII and XXIX presented the general symptoms of a brain tumour, without any localizing symptoms. Case XXVIII presented fairly typical symptoms of a cerebellar growth.

The functions of the cerebellum are still obscure. Mercier[52] holds that it is the organ for the co-ordination of movements in simultaneity, the cerebrum being the organ for the co-ordination of movements in succession. Gowers[53] thinks that the middle lobe exerts no downward influence, but that it is a regulating centre for centripetal impulses, having a special relation to motor processes and the maintenance of the equilibrium; the cerebellum having, possibly, an inhibitory influence on the cerebral cortex. It is to be borne in mind that no centrifugal tracts have yet been discovered coming from the cerebellum. All the sets of fibres which have yet been studied run upwards from the cord and basal ganglia into the cerebellum, or from the cerebrum into the cerebellum. It is therefore difficult to see how any impulses can be conveyed from the cerebellum to the spinal cord or the brain. It is uncertain whether there are any nerve tracts conveying impulses from the cerebellum to the brain or the basal ganglia; at any rate none have yet been discovered. Luciani[54] holds that the inco-ordination seen immediately after ablation of the cerebellum in dogs is due to the phlogistic effects of trauma, and that cere-

bellar ataxia is due to asthenia, caused by the insufficient energy and imperfect tonus of the motor nervous system, which is manifested later by a rapid decline in nutrition. This rapid decline in nutrition, however, has not been especially noted in lesions of the cerebellum in man.

Most observers agree that the lateral lobes are latent regions, and that tumours situated in them, as in Cases XXVII and XXIX, give rise to no focal symptoms. Tumours of the middle lobe, however, give rise to well-defined symptoms, which have recently been reviewed by Seguin,[55] but which have been recognized by other observers previously, especially by Nothnagel.[49] Case XXVIII is a good example. Seguin thinks optic neuritis commoner from tumours in this region than from tumours elsewhere. The headache is often occipital, and in some cases it is most intense. Vomiting is often more frequent than with growths elsewhere, and it may be independent of the ingestion of food, coming on without warning and being unattended with nausea. Nystagmus is not uncommon. Mercier[52] and Hughlings-Jackson hold that the spasm in cerebellar lesions begins with the trunk muscles, extending to the limbs, and being tonic in character. Later observers, however, deny that cerebellar convulsions have this distinctive character. The convulsion in Case XXVIII approaches this type. The convulsion in Case XXIX, however, is at least not typical. The symptom which is said to be characteristic of a tumour of the middle lobe of the cerebellum is the peculiar gait of cerebellar ataxia, titubation, wholly unlike the stamping, throwing gait of tabes dorsalis, and resembling closely the reeling gait of the drunkard. This ataxia is said to be observed only when the patient tries to walk; when in a recumbent position the legs can be moved accurately. This distinction between tumours of the middle lobe of the cerebellum and tumours of the lateral lobes is not, however, fully justified. Cases have been reported in which the character-

istic gait was seen, although the lesion was in the lateral lobes, and in one of the operative cases cited in Table VIII there was a large cyst outside the cerebellum, but pressing upon one lateral lobe, and yet there was a typical cerebellar gait. Whether such a gait may be due to pressure from without upon the middle lobe is still uncertain, but it seems clear that the typical gait is not necessarily indicative of a tumour which involves the middle lobe. Absence of oculo-motor paralysis may aid us in excluding growths in the corpora quadrigemina, where this ataxic gait has also been observed. With the general symptoms, especially with frequent vomiting and with the ataxic gait, if there be no oculo-motor paralysis, we may be reasonably certain that the growth involves the cerebellum, and probably its middle lobe.

Tumours of the cerebellar peduncles are sometimes attended with characteristic symptoms. These will be referred to in the section on tumours at the base of the skull.

8. Tumours of the Hypophysis.

Case XXX. N. R. XXIX–182. Eliza S., 58, came to the out-patient department of the hospital on the 15th of February, 1882. She had always been weak, dyspeptic and costive. At this time she complained of headache in the left temporal region, tinnitus of the left ear, failure of vision, pain in the left eye, and formication in the left cheek. The memory was poor. She had vertigo. Some months before she had numbness of the left side. She had paresis of the left side of the face, and weakness of the left side of the body, with some difficulty in articulation, the tongue being protruded to the left. She was sleepy and easily confused, and had frequent dizzy spells. There was also otitis of the left ear, with symptoms of neuralgia on the left side of the face. Soon after she had pain in the right eye. There was

partial atrophy of the optic nerves, and the field of vision was limited. There was tenderness over the fifth nerve on pressure on the left side. She had left hemicrania, the pain often extending to the left arm and chest. In the middle of April there was tenderness of the left median nerve and transitory hemianopsia. She had nausea and headache. There were pains in the throat and cheek, and numbness in the left ulnar region. On the 18th of April, 1886 she entered the hospital. At that time she said that in 1881 she had a general swelling of the body, and had vomited blood. She had occasional diarrhœa. Three weeks before entering the hospital, the vision of the right eye had failed suddenly and she was nearly blind. At that time she had headache, vomiting, sleeplessness, pain in the stomach, thirst, anorexia, nervousness, deafness, and a poor memory. The urine contained a trace of albumen and a few casts. There were râles heard over the right lung. She was drowsy much of the time, and often complained of headache. The right eye turned outwards, and the lids drooped. The pupils did not react to light. On the 2d of May the right pupil was larger than the left, and both were dilated and immobile. The right eye-lid drooped. She would answer questions rationally; she was drowsier but she could be aroused. On the 4th it was noted that she had optic atrophy in both eyes. On the 7th she had pain in the abdomen, and vomited. On the 14th there was the beginning of a bed-sore. Her strength failed rapidly; she became unable to sleep, and died on the 23d of May.

Autopsy.—Acute general miliary tuberculosis, with pyelonephritis and cystitis. Ulcer of the duodenum with perforation and gangrene of the adjacent parts. There was a depressed yellow area smaller than the little finger-nail in the anterior part of the left caudate nucleus. In the hypophysis the sella turcica was occupied by an ovoid body

the size of a walnut, soft, gray and vascular, which proved to be a sarcoma. Both optic nerves, which lay upon this, were flattened. There was a depression in the chiasma; the left third nerve lay below the tumour and was flattened.

Tumours of the hypophysis of course closely resemble tumours situated in the anterior fossa and can hardly be distinguished from them. In some cases Seppilli [3] thinks they may be differentiated by the early developement of amaurosis, often without neuritis, and of oculo-motor symptoms caused by the pressure on the nerves. If one eye be first affected it points more definitely to a lesion of the base. From the situation of such a growth we should expect as an early symptom a bi-temporal hemianopsia, followed by the symptom of invasion of the oculo-motor nerves. In this case the record as to hemianopsia is vague. The oculo-motor symptoms were slight, but the fifth nerve was much affected; the left third nerve, which was found flattened at the autopsy, seems to have performed its functions very well. The left hemiplegia was an early symptom, but it soon disappeared. Some observers have noted polyuria or glycosuria; — others an increase of subcutaneous fat.

In the autopsies which have been made in the cases of acromegaly there has been a distinct enlargement of the hypophysis; and some writers have considered the enlargement of the hypophysis to be a cause of the acromegaly. Cases, however, of tumours of the hypophysis may exist, like this one, without any acromegaly. It therefore seems more rational to consider the enlargement of the hypophysis in cases of acromegaly to be merely a secondary symptom, an enlargement of an ἄκρον of the body, rather than as a cause of the disease. The chief symptoms of tumours in this region arise from implication of the cranial nerves. I will therefore refer to the following section for further consideration as to localization.

9. Tumours of the Base of the Skull.

Case XXXI. M. R. LXI-248. Julia P., 44, widow, entered the hospital the 6th of February, 1871. There was no known heredity. Four years before entrance she had headache for a year, which returned for a time two years later, and again eight weeks before entrance to the hospital. She had sore throat and necrosis of the palate. For two weeks she had trouble in swallowing, weakness, anorexia, costiveness and frontal headache; she was thought to be hysterical. On the 8th, she was wandering, and became speechless and unconscious. No paralysis was noted. She passed her urine and fæces in bed. On the 9th she died.

Autopsy.—The dura was rough and villous, and was thickened over the left fissure of Sylvius, attaining a thickness of $\frac{3}{16}$ of an inch. The brain was adherent to the dura just anterior to the middle part of the posterior lobe and was softened over a space half an inch in diameter. Below the anterior cornu of the left lateral ventricle, resting on the base of the skull, was a gray tumour, with a yellow centre, $\frac{3}{4}$ of an inch deep and an inch long. The bone beneath was eroded.

Case XXXII. N. R. LIII-268. George D., 33, married, a shipping clerk, entered the hospital the 12th of July, 1889. Two years before entrance, he had probably had syphilis. He complained of headache, and pain in the stomach, and vomited for ten days. For a week he had acted strangely and his memory was very poor. He had great pain in the frontal region. For a few days he was stupid and forgetful. Two days before entering the hospital he had an attack of right hemiplegia. His appetite was poor; he was somnolent. The tongue was protruded to the right; the left pupil was larger than the right and irregular in shape, but the irregularity was due to an old iritis. The knee-jerks were exag-

gerated; the elbow jerk was greater on the left. On the 20th he seemed brighter. On the 25th he was weaker, and could hardly speak. He failed rapidly, became comatose, and died.

Autopsy. — The pia at the base was thick and opaque in patches with puckered radiations. The left anterior cerebral and anterior communicating arteries were nearly obliterated by endarteritis. Beneath the pia in the frontal lobe, close to the anterior communicating artery, was a yellow nodule with a red periphery, the size of a hemp-seed. The anterior third of the left caudate nucleus was depressed and red. The tissue of the caudate nucleus, the internal capsule, and the anterior tip of the lenticular nucleus could be made out, but the whole was softer, with a yellow centre and a red periphery, containing fatty-degenerated ganglion cells, granular capsules, fat drops, and fine granules with broken-down nerve fibres and vessels with ischæmic softening.

CASE XXXIII. N. R. VII–210. Russell R., 35, single, a piano-maker, entered the hospital the 16th of November, 1879. He had used tobacco and alcohol to excess. He was distinctly anæmic. Twelve or fourteen years before he had received a blow on the back of the head, and had had more or less headache ever since. Four years before entrance the vision began to fail. Six or eight weeks before entrance he had severe headache and was unconscious for half an hour. There were no convulsions or paralysis. Three days before entrance he had severe headache, went to bed, and became unconscious. He was unable to answer questions, but he had no paralysis. On the 17th he was able to answer questions; he was found to be nearly blind in the right eye, and wholly blind in the left. The urine contained albumen and casts. On the 21st he was delirious. On the 26th the pupils were widely dilated. He had not answered questions for two or three days, and he died on the 26th.

Autopsy. — At the base of the median line, from the front of the pons to beyond the fissure of Sylvius, was a tumour two by two and a half inches, the size of the fist, with an irregular, lobulated, flaky surface, with blood vessels over the surface. The convolutions were flattened. The brain was soft and contained a puriform fluid over the tumour. The bones at the base of the skull were rough and carious, and the base of the sphenoid and its lesser wings were ulcerated through.

CASE XXXIV. M. R. CCXLI-62. Andrew F., 46, single, painter, entered the hospital the 1st of April, 1886. He had indulged somewhat in alcohol, and had had gonorrhœa. For three weeks he had a cough, with expectoration, dyspnœa, orthopnœa, palpitation, headache, œdema of the feet, indigestion, and frequent micturition. Sibilant râles were heard over the chest. The urine contained albumen, fat and casts. On the 8th he was very violent and abusive, and had Cheyne-Stokes respiration. On the 9th he became unconscious, gradually failed, and died on the 10th.

Autopsy. — Cardiac hypertrophy, interstitial pneumonia, emphysema, and bronchitis. Contracted kidney. Stricture of the urethra and hypertrophy of the bladder. Below the left half of the cerebellum, where the middle peduncle is given off, was found in the pia a firm, translucent, lobulated nodule, the size of a cherry.

CASE XXXV.* M. R. LXI-80. Mary S., 19, single, a domestic, entered the hospital the 2d of January, 1871. For over a year she had had neuralgic headaches, with occasional vomiting, increasing in frequency and severity. For six months she had had neuralgia of the right side of the face. For a year there was a change in her voice. She had much vertigo, and her gait was like that of a drunken person. The

* This case has been reported by Dr. S. G. Webber. Archives of Scientific and Practical Medicine, February, 1873.

head was tremulous, the grasp stronger on the right. A month before entrance the right eye became inflamed, and rapidly became destroyed. There was a general flushing of the skin, and slight facial paralysis on the right. There was nose-bleed; the right nostril was narrower and was full of mucus. The right side of the mouth could not be perfectly closed, and there was an excessive amount of saliva flowing from it. The tongue was protruded straight and was somewhat flatter on the right side. The senses of touch and taste were better on the left side of the tongue. The food stuck in the right side of the mouth, and the right side of the palate was flabbier. The ticking of a watch could be heard at three inches from the left ear; gradually she became totally deaf in that ear. Later there was optic neuritis in the left eye and the pupil of that eye was dilated. There was anæsthesia in the distribution of the right trigeminal nerve. The headache increased. Later she had paresis of the left leg. She suddenly became blind, and the blindness was followed by a slight return of vision and intense pain. Some hours later there was spasm of the right side, she became comatose and died on the 30th.

Autopsy. — The skull was thin in the temporal region. The convolutions were flattened. Just below the tentorium was a gliomatous tumour an inch and a half in diameter, growing from the meninges, and spherical in shape. There was a hæmorrhage in the centre of the growth, the colour of the growth being gray. The pons, medulla, and middle peduncle were pressed upon on

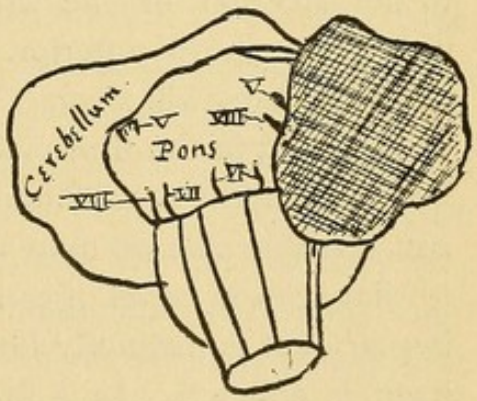

FIG. 17 (Webber). Case XXXV.
V = Trigeminal nerve.
VI = Abducens nerve.
VII = Facial nerve.
VIII = Auditory nerve.

the left side and the pons and medulla were pushed to the right. The right fifth nerve was softened, probably from pressure; the left fifth nerve was displaced but it was not pressed upon; the right nerve was somewhat degenerated. The eighth nerve on the left was involved in the growth, the left seventh nerve was not affected.

Of these cases only one presented any distinct symptoms. Case XXXI presented only vague general symptoms, largely hysterical in appearance. Case XXXII presented no special symptoms of basal trouble, the symptoms being due very largely, if not almost wholly, to the softening of the basal ganglia. Case XXXIII is a striking example of the few symptoms that an extensive lesion may sometimes cause. Case XXXIV had no symptoms that could not be accounted for by the renal disease. In Case XXXV the symptoms were due to remote pressure, and not to the tumour itself. In this case the lesion was thought to be on the right side, involving the fifth nerve upon that side; instead of that, it involved the fifth nerve upon the left and the right nerve was affected only by the remote pressure.

The focal symptoms of tumours of the base are due chiefly to the affection of the cranial nerves. According as the growth lies in the anterior, the middle, or the posterior fossa, the symptoms will vary. Tumours of the anterior fossa may be attended in the first place with disturbances of smell, from involvement of the olfactory nerve. If the tumour attains a considerable size we may expect mental impairment similar to that seen in cases of tumour of the frontal lobe; but this impairment is distinctly less than in the cases where the lobe itself is affected. In a few cases the growth has pressed downwards upon the roof of the orbit, and has either pressed the roof downwards or has penetrated into the orbital cavity, giving rise to exophthalmos. If the tumour extend further backwards in the anterior fossa the optic chiasma will be

involved. Tumours in this region will, of course, resemble closely tumours of the hypophysis; the visual disturbance will be prominent. This disturbance may take the form of unilateral blindness from affection of one nerve; a bi-temporal hemianopsia from affection at the chiasma itself, or homonymous hemianopsia from an affection of the nerve back of the decussation. If blindness begins in one eye and then goes to the other it is suggestive of a lesion at the base involving first one nerve and then the other.

The characteristic symptoms of tumours of the middle fossa arise from pressure on the third and fifth nerves. A tumour situated in the anterior portion of this fossa may involve the optic chiasma, and produce symptoms similar to those spoken of with tumours of the anterior fossa. Isolated paralysis of the fourth nerve, from a growth at the base, is exceedingly difficult to diagnosticate, and it can be done only by careful tests for double vision, when one eye is covered by a coloured glass. The symptoms of paralysis of the third nerve are much more striking and much easier to determine. Where single muscles supplied by the third nerve are paralyzed the trouble is more apt to be due to a lesion of the nuclei, but this is by no means always the case. In syphilitic basilar meningitis transitory paralyses of single muscles are not infrequent, but a tumour affecting the nerve outside of the brain will be much more apt to cause a paralysis of all the muscles supplied by the nerve. Tumours in this region may also exert pressure upon the crus cerebri, and thus give rise to hemiplegia. In such a case the symptoms may closely resemble those of a tumour of the crus, which have already been spoken of. Here the oculo-motor paralysis is more apt to precede the hemiplegia, while with tumours in the crus the opposite condition obtains.

Tumours of the posterior fossa may present symptoms similar to those of the crus, pons or medulla, crossed para-

lyses in various forms predominating. These paralyses are due, of course, to pressure upon the motor tract before it decussates, and upon the cranial nerves which have already decussated. The nerves which go off from the brain stem in this fossa are the fifth, sixth, seventh, eighth, ninth, tenth, eleventh, and twelfth. The symptoms due to lesions of the nerves usually precede the general motor and sensory symptoms. Where the fifth nerve is involved there will be neuralgia in the nerve, followed by anæsthesia in its distribution, and perhaps by neuro-paralytic ophthalmia and weakness of the muscles of mastication, as in Case XXXV. Anatomical considerations will explain certain other features. The sixth nerve may be affected without any conjugate action of the opposite internal rectus, such as is seen in disease of the pons. The facial and auditory nerves are affected together, although it is to be borne in mind that the facial nerve from its denser consistency, is much more tolerant of disease than is the auditory.

I have not spoken under a separate heading of tumours involving the middle peduncle of the cerebellum, yet this peduncle was pressed upon, if not directly involved, in Cases XXXIV and XXXV. Lesions of the middle peduncle of the cerebellum cannot readily be separated from lesions of the base involving the peduncle. In some cases lesions of this region give rise to so-called forced movements or rotatory movements toward the opposite side. Should this occur, especially if associated with disturbance in the distribution of the cranial nerves, which go off near the peduncle, we may reasonably infer that the growth has involved the peduncle; yet these forced movements were absent, or at least not noted, in these two cases.

10. Multiple Tumours.

Case XXXVI. M. R. CCLXXXVI–94. Mary W., 29, married, entered the hospital the 18th of May, 1889. Her previous history is uncertain. For three months she had had pain in the head, neck and abdomen, with obstinate vomiting. Four months before entrance she was confined, and three weeks after that she was attacked by the present trouble and steadily grew worse. Her temperature was 103. The lower portion of the abdomen was tender; the spleen was enlarged, and a round body was felt in the vicinity which could be moved to the right side. There was an offensive leucorrhœa. The uterus was movable, retroverted and much eroded. During the first week she was in the hospital she lay with her face covered. She had much headache, and one or two attacks of loss of consciousness in which the eyes were rolled up. The back of the neck seemed tender. On the 27th she was noisy, cried considerably, and seemed stupid. On the 29th there was retention of urine. She would not answer questions intelligently, but would put out her tongue when asked. The pupils were sluggish; the right was larger than the left. The abdomen was distended and tender. On the 30th there was marked strabismus. On the 31st she became comatose; her pulse was weak and rapid; she had difficulty in swallowing. On the 1st of June she died.

Autopsy. — General tuberculosis, tubercular meningitis. There was an opaque cheesy nodule about the posterior part of the left fissure of Sylvius; another outside the right optic thalamus; another in and below the medulla; and still another in the cerebellum. There were thirteen of these growths in various parts, from the size of a small pea to the size of a cherry.

Case XXXVII. M. R. CCLXVI–38. Robert G., 22,

single, a farmer, entered the hospital the 2d of February, 1888. The family history was good, and he had always been in good health. Five weeks before entering the hospital he was attacked with nausea, vomiting and vertigo. He vomited yellow, bitter matter immediately after eating. These symptoms continued, and he had also constant sweating. There was no pain in the abdomen. There was very severe frontal headache, which was worse at night. His appetite was poor and he was very costive; he slept little. During the month following entrance he had much headache and vomited a good deal. He grew steadily weaker. On the 9th of March there was retraction of the abdomen. Several purple spots were seen over the lower abdomen and tache cérébrale was noted. On the 21st optic neuritis was noted, which was most marked in the right eye. There was slight paresis of the right oculo-motor nerve. On the 21st he failed rather suddenly, his pulse became weaker and rapid, and he died.

Autopsy. — The calvaria was half the usual thickness, and showed the impression of the convolutions. The dura was red and vascular; the pia dry; the convolutions were flattened; the sulci obliterated. The lateral ventricles were twice the usual size. In the floor of the right lateral ventricle, near the tip of the caudate nucleus, was a pale, gray, gelatinous, ovoid, projecting nodule the size of a pea, another the size of a filbert meat. In the floor of the third ventricle was a similar flat nodule the size of a filbert meat. The cavity of the fourth ventricle was occupied by a soft, grayish, translucent growth, adherent to the whole of the floor except over an area the size of a finger nail, and also involving the roof of the ventricle and cerebellum as far as the olivary body. The upper surface of the cerebellum was elevated. The growths were gliomata.

CASE XXXVIII. N. R. XLVIII-150. Charles B., 39,

married, a grocer, entered the hospital the 26th of October, 1888. He denied any venereal disease. For a year he had been in poor health. Six weeks before entrance he found himself very weak on rising in the morning, although he was as well as usual the night before. He could stand and walk, but he could not articulate plainly. He was in a confused mental state, and could not attend to business. He kept about his work for two weeks and then gave up. At that time it was thought that his right arm was not as strong as his left. There was occasional diplopia. He was costive and slept poorly. The pulse was rather slow — 58. The urine contained a trace of albumen. There was a slight amount of paresis of the right side of the face. On the 1st of November he complained a good deal of headache, but his general condition improved. On the 9th his mental condition was duller; his answers were irrelevant, his speech indistinct. The disturbance of speech became more marked; he was unable to whistle; his pulse was still slow. He was given iodide of potassium which diminished the headache. On the 11th he answered still more irrelevantly; the paresis of the face was more marked. That afternoon he became unconscious; he had some expiratory dyspnœa with cyanosis, and died early the next morning.

Autopsy. — The dura was tense; the pia dry; the convolutions flattened; the sulci obliterated. The lateral ventricle was somewhat dilated. In the under surface of the left frontal lobe, at the entrance of the fissure of Sylvius, corresponding to the anterior tip of the temporal lobe, was a reddish-gray nodule with an opaque, yellow centre, the size of a large pea, imbedded in the brain and intimately connected with the pia. The brain surrounding it was yellower and softer. In the brain substance anterior to and outside of the caudate nucleus was a similar growth, the size and shape of a peanut meat; three centimetres back of this was

another, the size of a walnut, just outside of the nucleus and extending to the pia at the base. In the outer segment of the left lenticular nucleus was a cavity the size of a filbert. The white matter of the left centrum ovale, outside the anterior half of the basal ganglia, was pale yellow and the consistency of baked custard. There were hæmorrhages between the circular fibres of the pons four millimetres below the floor of the fourth ventricle. The tumours were found to be gummata.

CASE XXXIX. N. R. IV-163. William B., 31, married, a printer, entered the hospital the 30th of July, 1878. Three months before entrance he had an attack of hiccoughs, which had recurred. There was pain in the legs, left arm, and wrist for two weeks; these pains were increased on walking. The knee-joints were tender. Two days before entrance he had three fits within an hour. The mouth was drawn to the right; he frothed at the mouth and lost consciousness. He had a cough and signs of phthisis in the left lung. On the 2d of August his mind wandered, and he answered questions less quickly. He failed rapidly, and died on the 3d.

Autopsy. — Tuberculosis of the lungs, liver, kidneys, intestines, testicles, and supra-renal capsules. The dura was adherent. There was a yellow band along the fissure of Sylvius. The pia was thick, the vessels injected. There was a cheesy tumour the size of a bean, in front of the precentral sulcus, between the second and third frontal convolutions; another, the size of a bean, in the anterior part of the right first temporal convolution. There was a tumour, half an inch in diameter, on the right side of the pons, on a level with the fifth nerve, between the point of exit and the median line. There was a tumour a quarter of an inch in diameter in the lower part of the olivary body. There were others in the anterior and under surfaces of the cerebellum.

CASE XL. M. R. XXIX–34. Martha B. was brought to the hospital unconscious on the 17th of March, 1886. She was said to have suffered from exposure and privation, and to have been unconscious for four days. She was somnolent but she could be roused to slight consciousness. The respiration was quiet, with some remissions; the pulse was weak and intermittent. The pupils varied independently of light, the right pupil being larger. The eyes at times moved independently. She resisted passive movements of the limbs. The heart was enlarged, and a systolic murmur was heard at the apex. She opened her eyes and moaned; she protruded the tongue straight; there was no paralysis. The temperature was 100.6. On the 18th she was conscious, but slow to answer. She complained of headache, and said her attack was due to a fall. There was tremor of the tongue, lips, and hands. The pupils reacted to light. The temperature was normal. On the 25th she was drowsier, but she talked occasionally. There was some trouble in swallowing. The arms were rigid. The left arm was moved less than the right. There was no paralysis. The knee-jerks were normal. The tremor still persisted. She had cystitis. On the 31st she was harder to rouse; she lay with her head and eyes turned to the right, and she turned them to the left only with effort and apparent pain. The scalp and back of the neck were very sensitive to pressure. She swallowed very slowly and with great difficulty. April 1st, she was unable to swallow and could not be roused. She lay with her mouth open and the lips drawn. There was divergent strabismus. The left pupil was dilated, the right contracted; neither reacted to light. On the 3d the mouth at times was drawn to the left. It was hardly possible to make her swallow or answer. The night before she had a spasmodic attack lasting for ten minutes, the twitching being confined to the left side, including the face. After this the

mouth was drawn still more to the left. The left side of the forehead was wrinkled, the right side smooth. She was very dull. The arms were rigid; the rigidity was greater on the left side. The knee-jerks were normal. The right leg could be raised, and she gave signs of pain when it was pinched. She would try to draw up the left foot when it was pinched but she could scarcely move it; she would draw up the right. For three days there were no voluntary movements of the hands or eyes. The neck and head were less sensitive. On the 4th the face was not drawn so much to the left. The pulse and respiration varied. The pupils were contracted and equal, dilating upon pinching the arm. She could not be roused. On the 5th she could not swallow, and choked when food was given her. She had not spoken since the 2d. Portions of the face were flushed at times. There was occasional divergent strabismus in the right eye. The head was very much retracted. There was no rigidity on the left side. On the 7th the respiration and pulse became very rapid; respiration 108, pulse 160. The respiration diminished to 60, the pulse increased to 210. She was unable to swallow. The rigidity had disappeared. The pupils were of moderate size. The weakness and coma increased and she died.

Autopsy. — Emphysema, bronchitis, bronchopneumonia, and pericranial ecchymoses. The calvaria was thin; the pia dry; the convolutions were flattened. At the apex of the right fissure of Sylvius, and in the corresponding part of the first temporal convolution were two yellow patches in the pia six millimetres in diameter; on section these proved to be two firm, yellow, opaquen odules in the brain substance, the size of a split pea, both surrounded by gray, translucent tissue two millimetres thick. In the middle of the corpus callosum, extending three centimetres to the right, there was a nodule the size of a large horse-chestnut

partly yellow and firm, partly reddish, involving the right centrum ovale from the roof of the ventricle about two centimetres vertically. In the right parietal lobe, just back of the fissure of Sylvius, there was a dark red nodule, the size of a filbert, like clotted blood, in a cavity whose walls were of brain substance except at the anterior part, where there was a firmer vascular area uniting the clot to the brain substance. In the middle of the right optic thalamus was a circumscribed globular nodule, the size of a filbert, of the same red vascular structure. At the junction of the right parietal and temporal lobes there was a similar nodule in the white matter near the cortex, the size of a filbert meat. The white matter of the right centrum ovale was bluish-white, showed no puncta cruenta, and was much denser than the rest of the brain. Some of these growths were examined and proved to be gummata.

To these cases we ought of course to add Cases XXI and XXIV, but as in these cases the growths were all in one region, I was led to place them elsewhere.

Case XXXVI had general tubercular meningitis; Case XXXVII presented the general symptoms of tumour but hardly any focal symptoms; Case XXXVIII presented still fewer general symptoms and no definite focal symptoms; and Case XXXIX showed also no general symptoms of tumour, and only obscure symptoms of brain disease. Case XL, also, showed symptoms of meningitis rather than of tumour, and was certainly suggestive of a general syphilitic basilar meningitis, involving some of the cranial nerves, but not completely destroying them.

In rare cases, especially where there are only two tumours, they may each present such definite focal symptoms as to render the diagnosis of a multiple growth possible, as happened, for instance, in Case XXIV, where the location of two growths was diagnosticated during life, although the

presence of the third was not suspected. In other cases one tumour gives rise to definite focal symptoms, and the other growths cause no distinctive phenomena, or the two growths may lie close together in the same region, each, perhaps, being accountable for the symptoms. Case VIII and Case XXI came near to answering this requirement. In other cases we have only general symptoms of tumour. In other cases still, as in those just reported, the diagnosis of multiple growths becomes impossible. We may suspect such a condition in certain general dyscrasiæ (tubercle, cancer, and melanotic growths), but it is rarely possible to make a definite diagnosis. As the statistics show that in at least one-seventh of the cases of brain tumour the growths are multiple, and as it is impossible absolutely to exclude multiplicity when the symptoms can all be attributed to the presence of a single growth in one definite region, the importance of multiple growths in the surgical treatment of brain tumours is evident.

VI. DIAGNOSIS.

MUCH has already been said on the question of diagnosis. It will be well to recapitulate briefly the more important features to be borne in mind. The problems to be solved are, first, to decide as to the presence of an intra-cranial growth; second, to decide as to the location of the growth; and, third, to decide as to the nature of the growth.

1. THE DIAGNOSIS OF THE EXISTENCE OF A TUMOUR.

The diagnosis of an intra-cranial growth must be based chiefly upon the general symptoms already discussed; the focal symptoms, while of much value as corroborative evidence, are usually of secondary importance, for they are due to the seat of the lesion rather than to its nature. Given the characteristic general symptoms with the addition of focal symptoms, the diagnosis becomes easy. With general symptoms alone it is often hard to exclude other affections.

Of cerebral affections which may be confused with brain tumour we may mention abscess, meningitis, thrombosis or hæmorrhage, lead encephalopathy, hysteria, migraine, and paretic dementia; of other affections contracted kidney and hypermetropia are those most likely to be mistaken for a tumour.

The distinction between abscess and tumour is often difficult. Optic neuritis is less common in abscess, and chills or rise of temperature may be present. The presence or absence of these symptoms, however, is by no means distinctive. The chief argument against abscess is the

absence of any source for suppuration, especially suppurative disease of the middle ear or the nose.

It is easy to distinguish between typical cases of meningitis and tumour. Localized meningitis of chronic course can often not be distinguished from a tumour. As we have seen in the chapter on pathology, tuberculosis or syphilis may give rise to diffuse meningeal changes or to more discrete lesions which, if of chronic course, become actual neoplasms; and, in the cases recorded, we have found the two conditions of tumour and meningitis frequently co-existing. A chronic course, symptoms of increased pressure, optic neuritis, and certain marked focal symptoms, such as aphasia or monoplegia, are more suggestive of tumour; but the distinction can seldom be made with certainty, except in the cases when we can definitely localize the trouble within the brain substance.

In a few cases a tumour may give rise to symptoms resembling those of hæmorrhage or thrombosis, as in Case XVI. Here the difficulty is due to the absence of the more characteristic signs of a tumour, optic neuritis, headache, etc. In ordinary cases the presence of these symptoms and the nature of their onset will render a diagnosis possible.

Inasmuch as lead may give rise to optic neuritis, headache, vomiting, convulsions, and mental impairment, it is not strange that lead encephalopathy may be mistaken for tumour. The distinction must rest on the history of exposure, the detection of other symptoms of lead poisoning, and the presence of lead in the urine.

Case XXXI was thought to be hysterical; I recall a case of profound hysteria that was for weeks thought to be brain tumour. The mistake has been made by more than one skilled neurologist. Headache, vomiting, partial epilepsy, anæsthesia, amaurosis, monoplegia, may all be seen in hysteria. Optic neuritis and hemianopsia are strong evidence in favour of organic disease. In many cases, however,

the decision can be made only after prolonged and careful observation. The impression which a hysterical patient makes on the observer is often of much value. Facial or oculo-motor paralysis is rarely if ever seen in hysteria, while the presence of the hysterical stigmata is evidence in favour of that affection.

It is usually easy to distinguish between tumour and migraine, but if the headache of tumour be of varying intensity and there be no neuritis or focal symptoms, and if the early history of long-continued attacks of headache be wanting some difficulty may arise.

Case XIV presented a fairly typical picture of paretic dementia, and all who saw him made that diagnosis. Such cases are rare, and ordinarily no confusion would arise. The speech, handwriting, fibrillary tremors and facial expression of paretic dementia on the one hand, and optic neuritis, headache, vomiting, and vertigo of tumour on the other make the distinction easy.

When uræmic symptoms develope in cases of contracted kidney the patient may have headache, vertigo, vomiting, and partial epilepsy. There may be retinitis with white spots and hæmorrhages in the macular region, which may be mistaken for the neuritis of brain tumour, although in the former the changes are more marked in the retina, and in the latter in the nerve. Repeated examinations of the urine, as to quantity as well as quality, determination of the size of the heart, and a study of pulse-tracings may give valuable evidence in favour of contracted kidney. In the forty cases here collected, however, renal disease and a tumour have sometimes both been present. In such cases the problem becomes much more difficult, and its solution, as we have seen, is often impossible.

It sometimes happens that with hypermetropia the patient may have not only severe headache but also a little swelling

and haziness of the discs if the eyes have been much used. Such a case once caused me to be for a day or two somewhat suspicious of a new growth. The absence of other symptoms, and the detection of hypermetropia by careful ophthalmoscopic tests and tests with lenses will usually solve any doubts.

2. The Diagnosis of the Location of a Tumour.

In the preceding section the focal symptoms of a tumour have been gone over sufficiently to render it unnecessary to speak in much detail here as to the focal diagnosis. I will, therefore, cite very briefly the striking focal symptoms of tumours in the various regions of the brain.

Pre-Frontal Region. — Marked mental impairment; symptoms of invasion (partial epilepsy, aphasia); disturbances of smell.

Central Region. — Partial epilepsy; monoplegia; partial anæsthesia; motor aphasia.

Posterior Parietal Region. — Word blindness; disturbance of muscular sense (?); homonymous hemianopsia.

Occipital Region. — Homonymous hemianopsia; soul blindness.

Temporo-Sphenoidal Region. — Latent region. Word-deafness; disturbances of taste, smell, and hearing (?).

Corpus Callosum. — Latent region. Progressive hemiplegia, often bilateral, from invasion.

Optico-Striate Region. — Hemiplegia; contracture. In posterior part, hemianæsthesia, homonymous hemianopsia, post-paralytic chorea, athetosis.

Crus Cerebri. — Crossed paralyses of oculo-motor nerve and limbs.

Corpora Quadrigemina. — Oculo-motor paralyses; reeling gait; blindness (?); deafness (?).

Pons and Medulla. — Crossed paralyses of face and limbs or tongue and limbs. Other cranial nerve lesions.

Cerebellum. — Marked cerebellar ataxia; marked vomiting; often a latent region.

Base, Anterior Fossa. — Mental impairment, disturbance of smell and sight, exophthalmos.

Base, Middle Fossa. — Disturbance of sight; oculo-motor disturbances; hemiplegia.

Base, Posterior Fossa. — Trigeminal neuralgia; neuro-paralytic ophthalmia; paralyses of face and tongue; disturbance of hearing; crossed paralyses.

Hypophysis. — Disturbances of vision; oculo-motor disturbances.

In addition to determining the seat of a tumour, it often becomes of some importance, in tumours of the cerebrum, to determine whether they are in or near the cortex or deeper down in the white matter. The symptoms are often much the same, but Seguin[17] has formulated certain rules which are of some value, although, as he states, the distinction is often only conjectural. "In favour of a strictly cortical or epicortical lesion are these symptoms, none of them having specific or independent value: localized clonic spasm, epileptic attacks beginning by local spasm, followed by paralysis; early appearance of local cranial pain and tenderness; increased local cranial temperature. In favour of subcortical location of a tumour: local or hemiparesis followed by spasm; predominance of tonic spasm; absence, small degree, or very late appearance of local headache and of tenderness on percussion; normal cranial temperature."

3. The Diagnosis of the Nature of a Tumour.

The diagnosis of the nature of an intra-cranial growth is, in the present state of our knowledge, only conjectural. The determination of the seat renders it possible to suspect

certain forms of growth; thus, in the cerebellum, growths are more apt to be tubercular, and in the white substance of the cerebrum, sarcomatous or gliomatous; multiple growths are more commonly tubercular or syphilitic.

Certain other symptoms may give us some help. The discovery of cancer or sarcoma in other organs may lead us to suspect a metastatic deposit in the brain. The presence of a marked cachexia strengthens this suspicion. Evidence of tuberculosis elsewhere, or of a tubercular diathesis, or symptoms of syphilis may help us in our diagnosis. A denial of previous syphilis is, of course, of little value. The age of the patient may also be a guide, tubercle being especially common in young subjects. Heredity may throw a little light, especially with cancer and tubercle.

So far as the actual symptoms of tumour go, they give comparatively little help. Frequent apoplectic attacks, and a moderately slow progress are said to point to a glioma. Sudden changes in the symptoms, as in Case IV, may be due to a varying amount of blood in very vascular growths. If, with a tumour localized at the base, we get a distinct bruit, or if we can localize the growth distinctly in the neighbourhood of a vessel, we may suspect an aneurysm, but Gowers[4] thinks the bruit is rare in such cases. In very few cases, however, can we be at all positive as to the nature of a growth.

VII. COURSE, DURATION, AND PROGNOSIS.

Course. — Some of the cases reported show that a limited number of intra-cranial growths, especially tubercular growths, may be situated in a so-called latent region or may become encapsulated and calcareous and remain in the brain for years giving rise to no symptoms. Other growths may give rise to a few symptoms, but they cause little disturbance and the patient may die of some other disease. The cases collected show that the percentage of tumours which apparently cause no symptoms worth mentioning is much larger than we are ordinarily led to suppose. In many cases, however, the symptoms steadily progress to a fatal termination. In such cases we note first, as a rule, symptoms of irritation, headache, vertigo, vomiting, or spasm; and later symptoms of deficit, paralysis, neuritis, mental failure and anæsthesia. The cases reported show, however, that such a course is by no means constant. Fluctuations in the severity of the symptoms are not uncommon. The patient may rally after rather marked symptoms and go on for a number of months in comparative comfort. Where, however, the symptoms of tumour are well developed, the course is, in the main, pretty steadily progressive.

Duration. — The duration of tumours is very uncertain. In the first place we can never say how long the tumour has been growing before it gives rise to symptoms; in the second place, the statement as to the first onset of symptoms is seldom trustworthy. Following the symptoms of these cases as recorded, I find that in thirty-two cases the shortest

duration of the symptoms was twenty-five days, the longest twelve years, with an average duration of about sixteen months. One of Seguin's cases of cerebellar tumour had definite symptoms for eighteen years.

In regard to the termination of the disease eleven died of intercurrent disease, generally Bright's disease, general tuberculosis or tubercular meningitis. Twelve died of exhaustion, in one or two cases from bed-sores and cystitis, and twelve died comatose. Two died from the effects of operation. In several cases I have known patients suddenly to become comatose and to die in a few hours or days, without any special symptoms to account for the sudden change in their condition. Such danger of sudden death should always be borne in mind.

Prognosis. — The prognosis of intra-cranial growths is usually regarded as hopeless. This I think to be an extreme view. We have seen in some cases that the growth, if tubercular, may take on a capsule which becomes calcareous, and that it may exist for years without giving rise to any definite symptoms. I was led several years ago to give a distinctly unfavourable prognosis as to life in a case of probable tubercle of the cerebellum, where the patient has apparently made a fair recovery. The optic neuritis has left him blind. All other symptoms, however, have ceased. He is in good health, bright and intelligent. He was for some time unable to walk, but he has now regained the power to do so. He has learned to read with the blind alphabet, and is doing very well. The prognosis in children, where there is a probable tubercle of the brain, is generally better than with any other form of new growth. Other cases, as has been seen, may go on for a number of years with few, if any, signs of cerebral trouble and the patient may finally die of some intercurrent disease. In some cases of syphilitic new growths proper treatment may bring about resolu-

tion; but, in the chapter on pathology, it has been pointed out that the gumma is composed partly of round cells and partly of cicatricial tissue. In many cases remedies may bring about partial resolution; the round cells may disappear, but I doubt if treatment can ever succeed in removing the connective tissue, and, if much of that has formed, there is left, in spite of all our treatment, a hard, dense mass, somewhat smaller than the original growth. In fact the more I see of cerebral syphilis the more doubtful I feel as to the permanent benefit of treatment in the average case. In the majority of cases, however, where the symptoms are sufficiently clear to enable us to make a diagnosis of an intra-cranial growth, the prognosis is bad. The symptoms gradually progress, and the patient finally succumbs.

VIII. TREATMENT.

1. Medical Treatment.

A consideration of the efficacy of the medical treatment of tumours of the brain naturally leads one into a state of pessimism. It is obvious that our only hope of a radical cure by means of drugs is in those cases where the new growth is of a syphilitic character. A glance at the preceding tables, however, will show that such growths form only a small percentage of the whole number, even if, as in Table VI, cases of syphilitic meningitis be included. Furthermore, in the preceding chapter I have called attention to the anatomical reasons which render it probable that in a part even of the syphilitic cases treatment will be of little avail. If the syphilitic process be taken early, while there is as yet only a round cell infiltration, it may yield to treatment; but, when fibrous cicatricial tissue is formed, or when portions of tissue become cheesy and necrosed, treatment cannot remove the cicatrix or replace the destroyed cells.

Nevertheless, small as the chances are of the success of anti-syphilitic treatment, we must bear in mind Hebra's dictum, "Jeder Mensch kann syphilitisch sein," and give the patient the benefit of the doubt. To this end both mercury and iodide should be given. The former may be given by inunction, by the mouth, or by subcutaneous injection. Of the last method I have had no experience; of the other two I prefer inunction as less likely to disturb the stomach. In whatever form mercury is given it should be pushed rapidly until slight salivation is produced. Iodide

of potassium should be given freely. If small doses be given they do little good and there is greater danger of producing iodism. We should begin with doses of at least two grammes three times a day given in a large amount of water; Vichy or Giesshubler water is better than plain water. The dose should be pushed as rapidly as possible, unless the stomach will not tolerate it, until the patient takes at least six grammes three times a day. If possible, even larger doses, ten to twenty grammes or more, should be given, and these doses are often well borne, especially if the patient be syphilitic. If, in four weeks, no signs of improvement are seen, the treatment may as well be abandoned. Seguin[55] is of the opinion that iodide is also of some benefit in tubercle.

Failing this our treatment must be largely symptomatic. The most distressing symptom to combat is undoubtedly the headache. This may be met at first by milder remedies, evaporating lotions, cold, massage, or a mild galvanic current; but these will soon prove unavailing. The newer analgesics, phenacetine, antipyrine, antifebrine, and the rest, often give admirable results. For a year phenacetine, in doses of one or two grammes two or three times a day, greatly relieved the headache in Case XXIX and kept the patient comfortable without any bad effect. A bromide salt, either alone or combined with iodide of potassium or chloral, is sometimes, although less frequently, of benefit. Later on, however, all these drugs fail, and we are forced to employ morphine, the dose of which must gradually be increased. In some cases even this proves useless against the intense headache.

Against convulsions our main resource is in the bromides, which are to be used as in idiopathic epilepsy. One or two grammes of bromide, preferably the sodium salt, will often hold the convulsions in check. It is seldom, in fact, that the convulsions prove a very serious complication, but in some cases they are so frequent as to demand interference. In

several cases I have known the bromides to work admirably, almost wholly checking the convulsions. Even though the preliminary treatment with large doses of iodide of potassium has failed, it is often of benefit to give it in small doses, about half a gramme, in addition to the bromides.

Where there is severe vomiting the diet must first be regulated. This is, indeed, an important element in the treatment of these cases. Sometimes the vomiting is of rare occurrence, the digestion is good, and convulsions are seldom seen; in such cases it is often better to let the patient gratify his fancy in regard to food than to lay down strict and annoying rules. In other cases, it becomes necessary to follow a pretty strict regimen. Here I should advise such a diet as would be suitable for an epileptic; an avoidance of alcohol, tea and coffee, and highly seasoned foods, a very limited indulgence in meat or fish, and as near an approach as possible to a milk diet. In cases of severe and persistent vomiting the diet must be still more limited. Here cracked ice, peptonized food, milk and lime water, milk and soda water, koumyss, matzoon, or champagne frappé may be all that can be borne. In extreme cases the stomach should be given complete rest and nutriment should be given by the rectum. In addition to a regulation of the diet it may be necessary in the prevention of vomiting to precede the administration of food by cocaine given by the mouth or by hypodermic injections of morphine.

In other respects the ordinary precepts are to be observed. The bowels, bladder, and skin require close attention, especially in bed-ridden cases. In addition to this our efforts should be directed to promoting the patient's comfort, aiding nutrition, procuring sleep, and relieving pain. In many cases such a task is, unfortunately, beyond our powers.

2. Surgical Treatment.

When on the 25th of November, 1884, Mr. Godlee removed a tumour from the brain of one of Hughes Bennett's patients a new hope dawned for the victims of intra-cranial growths. The patient, to be sure, died; but it was shown that he died from preventable causes, and subsequent operations have proven that it is possible to remove an intra-cranial growth and that the patient may recover from the operation.

Subsequent investigation, however, has shown that this hope is limited. It is a self-evident proposition that those tumours alone are accessible to operation which are near the surface of the brain. It is not probable that any advance in cerebral surgery can make it possible to cut into the optic thalamus, the corpora quadrigemina, or the pons with safety. The cases thus far operated on, however, indicate that it is possible to remove cortical tumours, and even tolerably deep-seated sub-cortical tumours, from any part of the external surface of the cerebrum, especially in the Rolandic region, and the percentage of success thus far obtained proves that the operation is a justifiable one. Of course, tumours in the convolutions at the base of the brain, and on the median aspect of the hemispheres, except those near the surface, must remain inaccessible.

Although the external aspect of the cerebrum is accessible to the surgeon, it unfortunately happens that our present knowledge of cerebral localization does not enable us to determine the situation of neoplasms in every part of the cortex. At present we can diagnosticate with some accuracy lesions involving the ascending convolutions, and the parts of the frontal and parietal convolutions immediately adjacent, lesions of the posterior part of the first temporo-sphenoidal convolution on the left, lesions of the occipital lobes involv-

ing the cuneus, lesions of the cerebellum, and, perhaps, lesions in the prefrontal region, the inferior parietal convolution and the angular gyrus, — rather a limited portion of the region accessible.

Unfortunately it only too often happens that tumours are situated, not in these regions, but in the inaccessible parts. Out of four hundred and eighty-five cases collected by Bernhardt,[1] only fifty-seven were in the cortex; and on examining the summaries of the reports of these cases, it seems as if twenty-seven might have been localized. Out of these twenty-seven cases, in three there were growths elsewhere; in two there was also tubercular meningitis; and in one the growth was a cancer, — leaving twenty-one possible cases. In one hundred and twenty-four cases of tumours of the white substance of the cerebrum I find fifteen more which might probably have been localized; but in one of these, again, the growth was a cancer, and in one the tumours were multiple, leaving thirteen cases. Thus, out of four hundred and eighty-five cases, thirty-four, that is, seven per cent., might possibly have been localized successfully, and have been more or less accessible to the knife; but it is not easy to say, from the tables of Bernhardt, just how deeply seated the cases in the second category were. The estimate that I have made errs rather through being too liberal. The examination of Mills' and Lloyd's[11] table of one hundred selected cases is rather more favourable, for ten of these seem to have been cases that could have been operated on.

Hale White[56] in an elaborate analysis of one hundred autopsies of cases of tumour of the brain at Guy's Hospital, thinks that, including cerebellar growths, ten might well have been operated on, but six of these were cerebellar. In four more, one of which was cerebellar, an operation was possible. On the one hand, however, he excludes very large tumours which can, perhaps, be successfully removed, and

also infiltrated growths, which, as is shown by a number of cases, may sometimes be in part removed with temporary benefit; but, on the other hand, in the absence of clinical histories, we do not know in how many of these cases symptoms existed which would permit of localization.

Starr[57] has made an admirable study of three hundred cases of tumour in children, with special reference to the question of surgical interference. Out of these three hundred cases he finds that fifty-six involved the cortex or the centrum ovale; of these he found that forty cases gave sufficient data to draw some conclusion from. Of these forty cases an operation was indicated in nineteen; but it would have succeeded in only sixteen (five per cent.), two being infiltrated, and one multiple. In adults, however, Starr finds that cortical and subcortical tumours are relatively more frequent than in children, in whom, on the contrary, cerebellar growths are commoner.

In the forty cases here collected only fourteen (I, III, IV, VI, VIII, IX, X, XI, XII, XIII, XXV, XXVII, XXVIII, XXIX) could, by the most liberal interpretation, be regarded as situated in regions accessible to the surgeon, and of these only four (III, IV, VIII, XXVIII) presented symptoms which could have made the local diagnosis possible. Of these four one (IV) was deeply seated and infiltrated, and probably could not have been successfully removed; and another (XXVIII) involved almost the whole of the cerebellum, leaving only two cases or five per cent. in which an operation was likely to have been successful.

If we add cerebellar growths, our percentage of operable cases becomes larger. Hale White gives seven cerebellar tumours which might have been operated on, out of a total of one hundred. Starr finds ninety-six cerebellar tumours out of three hundred in children, about one-third of which could have been removed. Mills and Lloyd give nine cases

out of one hundred, four of which could have been localized, and three removed. Bernhardt gives ninety out of four hundred and eighty-five, of which forty-one, making a very liberal estimate, might have been localized, and nineteen removed. Thus, about three or four per cent. of all cases are cerebellar growths which can be localized and removed.

The chance for operative treatment of cerebral tumour, therefore, is not great, the percentage of cases situated in accessible regions, and giving rise to definite focal symptoms, varying from five to seven per cent., if we exclude tumours of the cerebellum. Including these growths the percentage rises from eight to fifteen at the most.

It has been a matter of some discussion whether it were justifiable to attempt to remove a growth which was believed to be in the cerebellum. The first three cases operated on died, and it was questioned whether a portion of the cerebellum could be removed without fatal results. Although the cases are still very few there is fortunately no need of further *a priori* reasoning upon the subject, for two cases at least are now on record where a growth has been removed from this region and the patient has recovered.

Further limitations of the field of operation for intra-cranial growths are set by the nature of the growth. The successful removal of tubercles and gummata shows that granulomata may be operated on. Von Bergmann [58] shows that with tubercle there is a much greater chance of multiplicity, that there is probably tuberculosis elsewhere, and that, if the growth be not encapsulated, there is difficulty in removing it completely and risk of further infection of the brain. These arguments must, therefore, be weighed carefully. The same arguments, however, may be urged against operating for tubercular disease elsewhere, tubercular glands or joints, and in some cases they may be strong enough to decide against operating; but, unless the patient be in an advanced stage of tuber-

culosis, or unless we can be quite sure that there are two growths or a co-existing meningitis, we should not be deterred from trephining.

I have already spoken of the possibilities of success in the treatment of gummata by drugs, and argued that drugs will not remove cicatricial tissue. Von Bergmann doubts whether the artificial cicatrix which follows the knife be less injurious than the cicatrix of a syphilitic process after thorough anti-syphilitic treatment. To decide this point more time is required, but a clean, surgical cicatrix is probably smaller and less disturbing than the hard irregular cicatrix of an old syphilitic process, and the successful removal of some syphilitic growths should lead us to venture upon further operations.

The accompanying tables (Tables VIII and IX) give the most complete list yet published of operations for removal of cerebral tumours. They give us further information in regard to the limitation set by the nature of the growth. Infiltrating growths, unless the growth be small, cannot be completely removed, for, to remove them properly we must cut out the healthy tissue which surrounds them. Therefore, as the cases show, the operation is often incomplete, and the growth may recur. This is, of course, especially true of malignant growths, particularly sarcoma. When the growth is surrounded by softened brain tissue, moreover, the possibility of a successful operation is further limited. Nevertheless partial removal often affords great although temporary relief.

From what has been said as to the diagnosis of the nature of the growth it is evident that we can only rarely know much as to the nature of the growth that we have to deal with in any given case. In some cases we can decide as to the advisability of removal only after the brain has been exposed; in others, perhaps, we can have some grounds for believing

that the growth is highly vascular and probably infiltrating, and that therefore an operation will have slight chances of permanent success.

Case IV shows another limitation to the field for operation, which is apparent to every one, but which has not been made prominent. In this case I was consulted because the family were anxious for an operation. The condition of the heart, however, rendered it almost certain that the patient would die from any attempt at operation, and perhaps even from the ether. In addition the growth was believed to be quite vascular, infiltrated, and deep seated, but the condition of the heart led me to decide against operation. Nevertheless Horsley has operated successfully on a patient *in extremis*, who had been comatose for ten days, and similar successful operations are on record.

Table IX shows another limitation. The growth may be so large or so deeply seated as to render it hazardous if not impossible to remove it. This again can rarely, if ever, be determined before the skull is opened; hence we must expect that in a certain percentage of cases the operation will have to be abandoned.

Such are the limitations upon the operation presented by the situation, size and nature of the growth; and from them we see that the percentages given above, small at the best, must be still farther reduced. In only a very small number of cases of intra-cranial growth, therefore, can we hope for a cure by surgical interference; yet this is probably a larger number than we can hope to cure by medical treatment, and thus the operation becomes justified.

TABLE VIII. Cases of Removal of an Intra-cranial Growth.

Reporter and Reference.	Region.	History of Case.	Operation.	Result.
1. Bennett and Godlee. Med. Chir. Trans. lxviii, 243, 1885.	Central.	Male, 28. Three years previously twitching of left side of face. Convulsions; sensora aura and spasm beginning in face. Paralysis of left arm. Headache. Optic neuritis.	Glioma, size of walnut, removed from middle and upper thirds of right ascending convolutions, 25 Nov., 1884.	Death from septic meningitis one month later.
2. Durante. Lancet, 1 Oct., 1887.	Prefrontal.	Female, 40. Left eye lower than right. Poor memory. Sense of vacuity. Uncertain movements. Mental depression.	Fibro-sarcoma weighing seventy grammes removed from anterior fossa, May, 1884.	Recovery.
3. Hirschfelder and Morse. Pacific Med. and Surg. Journal, April, 1886.	Central.	Male, 33. Headache and vertigo for two years. Spasm of arm and face. Left hemiparesis. Contracture. Slight anæsthesia of face.	2½ cub. cm. of soft, infiltrating glioma removed from right ascending parietal, 15 Feb., 1886.	Death eight days later from septic encephalitis.
4. Horsley. British Med. Journal, 23 April, 1887.	Central.	Male, 20. Epileptic seizures beginning in thumb. Paresis of arm.	Tubercular tumour removed from thumb centre, 21 June, 1886.	Recovery.
5. Ibid.	Central.	Male, 38. Left hemiplegia. Convulsions beginning in shoulder. Comatose for ten days.	4½ ounces of infiltrating glioma removed from right Rolandic region, 23 Sept., 1886.	Recovery. Death six mos. later from recurrence of growth.
6. Ibid.	Central.	Male, 37. Right hemiplegia. Epilepsy beginning in finger. Constant headache. Slight aphasia.	Tumour weighing 4¼ ounces removed from left Rolandic region, 7 Dec., 1886.	Recovery.

Table VIII. — *Continued.*

Reporter and Reference.	Region.	History of Case.	Operation.	Result.
7. Ibid.	Cerebellum.	Male, 18. Headache, vomiting, optic neuritis, general weakness. Head and eyes turned to right.	Tubercle weighing seven drachms removed from right lobe of cerebellum, 17 Dec., 1886.	Death. Shock; chronic tuberculosis.
8. Horsley. British Med. Journal, 6 Dec., 1890.	No record.	No record.	No record.	Death. Shock.
9. Ibid.	No record.	No record.	No record.	Death. Shock.
10. Ibid.	No record.	No record.	No record.	Death. Shock.
11. Ibid.	No record.	No record.	No record.	Recovery.
12. Macewen. Lancet, 11 Aug., 1888.	Central.	Female, 7. Pain and spasms in right great toe. Convulsions. Paresis right leg.	Tubercle size of hazel-nut removed from top of left ascending parietal. Tubercles in meninges.	Recovery.
13. Ibid.	Central.	Male, 22. Injury to head. Paresis of right arm. Spasm of tongue, face, and platysma.	Cyst, size of filbert, removed from base of left ascending frontal.	Recovery.
14. Ibid.	Central.	Female, 25. Syphilis. Left hemiplegia.	Trephined over right parietal. Yellow mass over upper part of ascending convolutions removed.	Recovery.
15. Macewen. Lancet, 16 May, 1885.	Central.	Male, 35. Left hemiparesis. Right facial paralysis.	Trephined over right ascending frontal. Tubercular (?) membranous mass removed.	Recovery.

16. May. Lancet, 16 April, 1887.	Cerebellum.	Male, 7. Frontal headache, vomiting, poor vision, optic neuritis, gait reeling backwards and to the left. Nystagmus. No knee-jerk.	Tubercle size of pigeon's egg removed from cerebellum, 19 July, 1886.	Death. Shock.
17. Suckling. Lancet, 1 Oct., 1887.	Cerebellum.	Female, 12. Headache, vomiting, vertigo, dim vision, right hemiparesis, tremor of right arm, staggering gait, optic neuritis, left facial paralysis with reaction of degeneration. Knee-jerk diminished.	Part of soft, infiltrating glioma removed from cerebellum, March, 1887.	Death. Shock.
18. Seguin and Weir. Am. Journ. Med. Sci., July, Aug., Sept., 1888.	Central.	Male, 39. Headache. Spasm right cheek for six years. Three years later spasm of right arm and face. Anæsthesia, hand. No neuritis.	Sarcoma size of end of finger removed from left precentral sulcus, 17 Nov., 1887.	Recovery.
19. Keen. Am. Journ. Med. Sci., Oct., 1888.	Central.	Male, 26. Tender scar on head. Headache. Convulsions. Right hemiplegia. Aphasia. Left hemianopsia. Optic atrophy. Paralysis right internal rectus, left inferior and superior recti.	Fibroma weighing 3 ounces removed from lower two-thirds of left ascending frontal, 15 Dec., 1887.	Recovery.
20. Birdsall and Weir. Phila. Med. News, 16 April, 1887.	Occipital.	Male, 42. Unsteady gait, vomiting, diplopia, right hemianæsthesia. Headache. Tendency to go to right. Left hemianopsia. Neuritis. Awkwardness of left side.	Sarcoma weighing 5¼ ounces removed from right occipital lobe.	Death. Hæmorrhage.
21. Ballet, Gelineau, Pean. Gaz. des hôpitaux, 21 Feb., 1889.	Central.	Male, 28. Epilepsy for six years. Spasm right great toe, rigidity of leg. Paresis of right leg. Status epilepticus.	Fibro-lipoma of pia removed from upper part of left ascending convolutions, 7 Dec., 1888.	Recovery.

Table VIII. — *Continued.*

Reporter and Reference.	Region.	History of Case.	Operation.	Result.
22. Pean. Bull. soc. anat. de Paris. May, 1888.	No record.	No record.	Cholesteatoma removed. Second operation. An apparently fatty tumour had been previously removed and this recurred. It seems probable that this is a second operation on the preceding case.	Recovery?
23. Knapp and Bradford. Boston Med. and Surg. Journal, 4, 11, 18 April, 1890.	Central.	Case VIII of this essay. Male, 32. Convulsions beginning in left arm. Left hemiplegia with contracture. Left hemianæsthesia. Headache, vertigo, vomiting, optic neuritis.	Tubercle weighing 35¾ grammes removed from middle of right ascending convolutions.	Death. Shock.
24. Fischer. Verhandl. d. deutsch. Gesell. f. Chirurg. 18te Kongress, p. 42.	Central.	Male, 37. Severe vertigo. Convulsions. Right arm weak; sensibility diminished. Paralysis of arm later, with paresis of leg, and slight aphasia.	Trephined and nothing found. Symptoms increased. Trephined again and sarcoma removed, which pressed on central convolutions from one side.	Recovery. Death two months later from recurrence of growth.
25. Thomas. Trans. Am. Inst. Homoeop., 1889, p. 464.	Central.	Female. Spasm of left hand with loss of consciousness. Left arm became paralyzed and rigid; left leg and face paretic. Headache. Thick speech. No optic neuritis.	Tumour weighing 3 ounces and attached to dura removed from middle of Rolandic region, pressing on but not involving convolutions.	Death in three days. Softening?
26. Parker. Brit. Med. Journ., 30 Nov., 1889.	Parietal.	Male, 38. Headache, dullness. Paralysis left arm, paresis left leg. No optic neuritis. Thickening of pericranium over right parietal eminence.	Gumma size of walnut removed, which was attached to dura beneath spot of thickening.	Slow recovery.

27. Markoe. Phila. Med. News, 5 Nov., 1887.	Parietal.	Male, 25. Injury to head. Convulsions. Dull, localized headache. Depression over left parietal.	Cyst removed containing mass of brain substance, 27 Jan., 1887.	Recovery.
28. Fitzgerald. Sajou's Annual Med. Sci., Vol. II, p. 36, 1888.	Central?	Female, 16. Loss of sight right eye. Partial paralysis. Convulsions.	Left temple trephined, Jan., 1887. Trocar inserted and contents of echinococcus cyst evacuated.	Recovery.
29. Limont and Page. Brit. Med. Journ., 26 Oct., 1889.	Central.	Female, 32. Became unconscious 14 days after fourth confinement. Regained consciousness. Spasms in right arm. Speech affected.	Portion of a large glioma removed.	Recovery. Growth recurred.
30. Maunsell. New Zealand Med. Journal, ii, 151. 1889.	Cerebellum.	Male, 18. Headache and vertigo, becoming intense. Vomiting. Reeling gait, staggering to the right. Eyes became prominent. Head and neck much swollen. Blind left eye, almost blind right. Deaf left ear. Later wholly blind and deaf. No smell. Later passed urine and fæces in bed. Head retracted. Pupils dilated. Head greatly swollen. Convulsions, beginning with retraction of head, involving right arm and leg.	Trephined, 12 Feb., 1889. Enormous sub-tentorial hydatid cyst, four inches long and three broad, evacuated.	Recovery complete, except that he is still blind.
31. Lucas-Championnière. Journ. de med. et de chir., lix, 298. 1888.	Central.	Male, 29. Right hemiplegia and aphasia.	Trephined over motor centres. Large hyperostosis removed.	Recovery. Still partly paralyzed. Secondary degeneration?

Table VIII. — *Continued.*

Reporter and Reference.	Region.	History of Case.	Operation.	Result.
32. Mercauton and Combe. Revue med. de la Suisse Romande. 20 Aug., 1889.	Central.	Female, 12. Neurotic. Injury to head. Pain in head. Convulsions affecting right arm. Paresis of arm after fit. Choked discs. Later fits involved whole right side. Vision failed. Right hemiparesis, intention tremor. Reflexes exaggerated on right. Sensibility to touch and position diminished. Slight aphasia of transmission. Defective visual fields. Tender spot near bregma on right.	History of operation not published in May, 1890, and I have been unable to obtain it.	No record.
33. Rannie. Brit. Med. Journ., 19 May, 1888.	Central.	Female, 35. Syphilis. Local headache. Mental disturbance. Spasms of left face and arm, followed by paresis.	Trephined 9 June, 1887, over right ascending frontal. Thickened dura containing small gumma removed.	Recovery.
34. Maudsley and Fitzgerald. Trans. Intercolon. Med. Cong. ii, 113. 1889. Ref. in Lond. Med. Recorder, June, 1890.	Cerebellum.	Male, 28. Headache. Vomiting. Vertigo. Staggering gait. Dim vision. Later blindness, deafness, loss of smell. Facial paresis. Optic atrophy. Awkwardness of left side.	Trephined 20 April, 1888, over left lobe of cerebellum. Much of cerebellum ran away. Solid nodule found fixed to bone, not removed.	Recovery. Blindness and deafness in one ear persist.
35. Clarke. Lancet, 1 March, 1890.	Central.	Male, 47. Syphilis. Localized headache. Convulsion followed by weakness of right leg. Knee-jerk exaggerated. Right arm grew weak. No optic neuritis; field contracted.	Trephined over leg centre on left. Mass of thickened dura half an inch thick removed.	Death in nineteen days. Septicæmia.

36. Church and Frank. Am. Journ. Med. Sci., July, 1890.	Central.	Male, 39. Sudden attack of unconsciousness followed by convulsion, beginning in right index finger. Right hemiplegia. Frontal headache. Straddling gait.	Trephined. Radiating sarcoma removed from middle and lower thirds of fissure of Rolando. Some improvement. Grew worse. Rise of temperature. Wound re-opened, pus removed.	Recovery. Sarcoma recurring.
37. Oppenheim and Koehler. Berliner kl. Wochenschr. 28 July, 1890, 20 April, 1891.	Central.	Female, 36. Apoplectic seizure eight months before. Headache, psychical disturbances, spasm beginning in left face, left facio-brachial monoplegia with contracture, anæsthesia of left face and arm, vomiting, dullness, beginning coma. No optic neuritis. Six months pregnant.	Cyst evacuated in right temporo-parietal region, 26 April, 1890, and gliomatous tissue removed.	Complete recovery. No headache, paralysis, spasms or contracture. Anæsthesia diminished. In July safely delivered of a healthy child. After this the symptoms gradually returned. Total left hemiplegia. Died 7 Dec., 1890. Glio-sarcoma size of apple in lower part of right ascending convolutions. Encephalitis.
38. Dunin. Neurol. Centralbl. 15 Aug., 1890.	Central.	Hemiplegia. Aphasia. Jacksonian epilepsy.	Tumour removed from anterior central convolutions.	Recovery. Four months later sudden death. Hæmorrhagic cysts.

Table VIII. — *Continued.*

Reporter and Reference.	Region.	History of Case.	Operation.	Result.
39. Lampiasi. Ref. Colella, La Psichiatria. viii, 261, 1890.	Central.	Convulsions, and paresis of right side. Right optic atrophy.	Trephined. Syphilitic products removed.	Recovery.
40. Bremer and Carson. Am. Journ. Med. Sci., Sept., 1890.	Central.	Male, 23. Spasm of left arm and neck. Later, feet involved. Speech temporarily disturbed. Morning vomiting. Lack of initiative. Left hemiparesis, with temporary contractures. Peculiar gait. Spasm beginning in neck. No headache or vertigo. Good vision. Beginning choked disc. Spasm produced by constant current to platysma centre.	Trephined 26 March, 1890, over right fissure of Rolando. Cavernous angioma removed.	Recovery.
41. Graham and Chubbe. Australas. Med. Gaz., 15 July, 1890.	Central.	Male, 16. Poor memory, dull, depressed. Frontal headache. Blindness. Vomiting. Legs weak. Right hemiparesis. Post-neuritic optic atrophy. Convulsions.	Trephined over upper part of fissure of Rolando. Hydatid cyst removed, four inches in diameter, holding nineteen ounces.	Recovery.
42. Verco. Trans. Intercolon. Med. Cong. ii, 377, 1889.		Record not examined.	Hydatid of brain removed.	Death four days later.

43. Castro. Anales del circulo medico argentino xiii, 125, May, 1890. Ref. in Neurol. Centralblatt, 15 Oct., 1890.	Central.	Male, 14. Headache, vomiting, right hemiparesis. Atrophy of arm and leg. Pain in left side of head.	Trephined over left fissure of Rolando. Tumour removed. 8 days later tumour found in wound, which broke on the 9th day, and was a hydatid cyst, 7 cm. in diameter. Some days later a second cyst broke.	Death 17 days later. Basilar meningitis.
44. Barton. Annals of Surgery, Jan., 1889.	Prefrontal.	Female, 30. Syphilis. Abscess of forehead. Pain, lethargy, confusion, right hemiparesis.	Abscess opened, necrosed bone beneath. Trephined 7 May, 1888. Necrosed bone removed. Yellow cheesy gumma scooped out, size of egg. Hernia cerebri.	Death. Encephalitis.
45. Macewen. Brit. Med. Journal, 11 Aug., 1888.	Prefrontal.	Female. Headache, dullness, spasm of right face and arm, myosis left pupil. Orbital growth. Nodule on skull half way between ascending frontal and anterior aspect.	Orbital growth removed. Trephined in 1879 over nodule. Tumour of dura half an inch thick removed.	Recovery. Died eight years later of nephritis. No trace of further growth at autopsy.
46. Von Bergmann. Die chirurgische Behandlung von Hirnkrankheiten. 2te Aufl. p. 137.	Central.	Male, 25. Slight headache. Right hemiplegia and contracture. Motor aphasia. Loss of muscle sense. Epileptic attacks.	Trephined over Rolandic region. Cyst evacuated which communicated with the ventricle.	Death four weeks later. Meningitis.

Table IX. — Cases of Trephining where a Growth was not Removed.

Reporter and Reference.	Region.	History of Case.	Operation.	Result.
1. Sands. Phila. Med. News, 28 April, 1883.	Central.	Female, 39. Injury to head. Spasm of right hand. Right frontal headache. Right face and arm weaker. Slight aphasia. Optic neuritis.	Trephined over lower ascending parietal for possible depressed fracture. Dura not opened. Gumma found beneath opening at autopsy.	Death. Shock.
2. Amidon and Weir. Annals of Surg., June, 1887.	Cerebellum.	Female, 26. Sarcoma of neck. Left brachial paralysis. Cramps on left side. Frontal headache. Right arm weak. Left optic neuritis.	Sarcoma of neck previously removed. Trephined, 11 Oct., 1886, over upper part of left fissure of Rolando. Cortex removed. Sarcoma of cerebellum found at autopsy.	Recovery. Died ten weeks later.
3. Dana and Pilcher. N. Y. Med. Rec., 9 Feb., 1889.	Prefrontal.	Male. Injury to skull. Epilepsy and mental impairment. No focal symptoms.	Trephined over seat of injury (angular gyrus). At autopsy glioma of left frontal lobe, extending into corpus callosum.	Death. Respiratory failure.
4. Ross and Heath. Lancet, 7 April, 1888.	Central.	Male, 20. Headache, spasm of left arm and face, left hemiparesis. Optic atrophy. Tender over lower part of right ascending frontal.	Trephined 13 Oct., 1887, over tender spot. Large adherent growth found which could not be removed.	Recovery. Relief of headache.
5. Sciamanna. Bull. d. R. Accad. med. di Roma. xi, 75, 1885–6.	Central.	Male, 46. Injury to head. Headache, vomiting, fainting. Convulsions. Slow pulse. Left arm and leg paralyzed. Somnolent. Later right oculo-motor and facial paralysis.	Trephined over seat of injury (right fronto-parietal region). Nothing found. At autopsy glioma of right centrum ovale, from right inferior cornu and temporal cortex to corpora quadrigemina.	Death two days later.

6. Wood and Agnew. University Med. Mag., April, 1889.	Temporal.	Male. Vertigo, loss of consciousness, loss of mental power, optic neuritis, left hemianopsia, loss of equilibrium, severe headache.	Trephined, 11 Feb., 1889, over right cuneus. Hæmorrhagic cyst found. At autopsy tumour of right temporo-sphenoidal lobe.	Death. Hæmorrhage.
7. G. M. Hammond. Journ. Nerv. and Ment. Dis., June, 1887.	Central.	Female, 29. Pain over right ear. Spasm of left side. Left hemiplegia. Intense headache. Optic neuritis.	Trephined, 30 March, 1887, over right Rolandic region. Nothing found. Cysts found at autopsy in same region below cortex.	Death. Shock.
8. Fraser. Lancet, 27 Feb., 1886.	Temporal.	Male, 44. Injury to head. Headache, vertigo, vomiting, indistinct speech, agraphia, loss of power to recall words. Stupid. Right hemiplegia with contracture.	Trephined over left third frontal. Nothing found. At autopsy glioma of left temporal lobe, involving ascending convolutions. Tumour in middle of right ascending parietal.	Death.
9. Wyman. Phila. Med. News, 8 Feb., 1890.	Cerebellum.	Male. Severe headache, failure of vision, vomiting, frontal headache, epilepsy, divergent strabismus, loss of smell, doubtful rotatory movements.	Trephined over right supra-orbital region. Nothing found. Cyst and tumour of left lobe of cerebellum.	Death.
10. Kerr. Occid. Med. Times, Feb., 1890.	Optico-striate.	Male, 35. Injury to head. Scar across upper part of fissure of Rolando. Right hemiparesis and spasm.	Trephined over scar. Nothing found. Glioma of left corpus striatum and optic thalamus.	Death.
11. Putnam and Beach. Boston Med. and Surg. Journal, 3, 10 April, 1890.	Parietal.	Male, 51. Tremor of right hand. Sudden temporary loss of speech. Similar attacks later. Convulsions. Memory failed. Headache. Staggering gait. Optic neuritis. Spasm right hand, paresis of extensors.	Trephined over left second frontal. Nothing found. Tumour in left inferior parietal lobule at end of fissure of Sylvius.	Death. Shock.

Table IX. — *Continued.*

Reporter and Reference.	Region.	History of Case.	Operation.	Result.
12–17. Horsley. Brit. Med. Journ., 6 Dec., 1890.	No record.	No record.	Six cases trephined to relieve pressure	Recovery in all.
18. Springthorpe and Fitzgerald. Australian Med. Journ., 15 Nov., 1890.	Cerebellum.	Male, 13. Blow on right forehead. Frontal headache, vomiting, poor vision, convulsions, head and eyes turning to right, uncertain gait. Optic atrophy. Knee-jerk lost. Straddling gait.	Trephined over right lobe of cerebellum. Serous fluid escaped. Glioma of middle lobe. Erosion through occiput.	Death.
19. Seguin. Boston Med. & Surg. Journ., 5 Feb., 1891.	Central.	No record.	Trephined. Nothing found. Glioma half an inch in diameter found in same region at autopsy.	Death.
20. Ibid.	No record.	No record.	Nothing found. No autopsy. Seguin thinks there was probably a soft glioma.	Death.
21. Stoker and Nugent. Dublin Journ. Med. Sci., Oct., 1890.	Parietal.	Male, 42. Tonic spasm left side, beginning in leg, followed by paralysis. No headache, vomiting, or neuritis.	Trephined over leg centre. Nothing found. Small spindle-cell sarcoma farther back, back of right parietal.	Recovery. Relief. Died 3 weeks later.
22. Lampiasi. Wien. Med. Wochenschr., 19 May, 1889.	Cerebellum.	Child, 2. Optic neuritis, exophthalmos, convulsions. Signs of pressure.	Exploratory trephining. Tubercle, size of egg, in left lobe of cerebellum.	Death in four days.

23. Keetley, Hood, Ball and Colman. Lancet, 21 Sept., 1889.	Pons.	Female, 7. Injury to head. Two days later vertigo and staggering gait. Vomiting. Left internal strabismus. Diplopia. Increased knee-jerk. Otorrhœa left ear. No optic neuritis. Drowsy. Collapse on twelfth day.	Trephined over left temporal region, and exploratory punctures made. Pons three times natural size, of a uniform yellow character.	Death in two days.
24. Eskridge. Unpublished case. Personal communication.	Prefrontal.	Female, 32. Loss of strength, memory, and mental power. Inability to continue train of thought. Paresis of right arm and leg. Headache. Later hemiplegia.	Trephined 25 Feb., 1890. Large mass, probably glioma, measuring $3\frac{3}{4}$ by 4 inches, found in left frontal lobe, extending back to beyond fissure of Rolando. This was too large to be removed.	Death in 8 days.
25. Bullard and Bradford. Boston Med. and Surg. Journal, 30 April, 1891.	Cerebellum.	Female, $6\frac{1}{2}$. Vomiting, vertigo. Staggering gait. Weakness of right leg, later of all four limbs. Incoordination. Optic neuritis. Blindness. Headache. Dilated pupils. Exaggerated reflexes.	Hole in bone over torcular, opening into sinus, torn open in laying bare skull. No autopsy. Trephined over cerebellum after death. Cheesy tubercular matter removed after death through trephine opening.	Death. Hæmorrhage.
26. Knapp and Bradford. Unpublished case.	Cerebellum.	Case XXIX of this essay. Male, 28. Headache, vomiting, optic neuritis, blindness. Tenderness in right temporal region. Deafness. Paræsthesia of mouth and hands.	Trephined over right temporal region. Nothing found. Considerable relief of headache. Cerebral hernia, left hemiplegia and hemianæsthesia.	Recovery. Death later from hernia and encephalitis.

Summary of Tables VIII and IX.

Tumours of	Recovered.	Died.	Result Unknown.	Total.
Prefrontal region	2	1	0	3
Central region	22	6	1	29
Parietal region	2	0	0	2
Occipital region	0	1	0	1
Cerebellum	2	3	0	5
Unknown location	2	4	0	6
Totals	30	15	1	46
Tumour not found at the point of operation	2	13	0	15
Tumours which could not be removed	1	3	0	4
Trephining to relieve increased intra-cranial pressure	7	0	0	7
Totals	10	16	0	26
Grand Totals	40	31	1	72

NOTE. — These tables are based on an investigation made for me by Dr. Lorini, at the Library of the Surgeon-General's Office in Washington. This investigation, however, extended only to May, 1890. I have endeavoured to add the cases since, but, owing to the impossibility of getting certain journals or of obtaining further assistance at Washington, owing to Dr. Lorini's removal, the record is incomplete. I have not included cases of perforating tumours of the skull where external manifestations were the sole guide for operation.

A study of these tables will make apparent certain other factors, independent of the seat or nature of the growth, which tend to make the benefits of operation still more limited. Out of 73* trephinings therein collected 27, or over one-third of the whole number, were ineffectual in that the growth was not removed. In sixteen cases this was due to errors in diagnosis, in three the growth was found but from its size and extent it could not be removed, in one the patient died before the skull was opened, and in seven the trephining was not done with the expectation or intention of removing the growth, but merely to relieve pressure.

Turning now to the table of cases where a new growth has actually been removed we find that the results have been distinctly favourable. Out of 46 cases we note 30 recoveries and 15 deaths. This is, of course, a shade too favourable, for in four cases the growth recurred, with fatal consequences. Even then, however, we have a mortality of less than half the number of cases. The cases in Table IX show a much greater mortality. Here, out of twenty cases when the tumour was not found or could not be removed, only four recovered from the operation. It would almost seem as if the operation had some bad influence upon the processes in the brain when the new growth was left in it, were it not that all the cases operated on to relieve pressure have recovered.

In a very small percentage of cases, then, we can make a correct diagnosis, the tumour is of such a nature that it can be completely removed, and in the majority of these cases the patient will survive the operation. With advancing knowledge of cerebral localization and greater skill in diagnosis this percentage may be increased, but it will always be small. Nevertheless, in view of the gloomy prognosis without surgical interference, it is something to give the victims of intra-cranial growths this small hope of recovery. Even

* Fischer's case, No. 24 of Table VIII, was twice operated on.

in the successful cases, however, the patient is not always restored to his former condition. Blindness or paralysis, the result of the morbid process or of the knife, only too often leave him crippled.

At the International Medical Congress at Berlin in 1890 Horsley suggested a new application of surgical procedure for the relief of intra-cranial growths. In cases of inaccessible or malignant growths, or in cases where there are no focal symptoms, yet where the patients suffer from intense headache or from other symptoms of increased intra-cranial pressure, he suggests the propriety of trephining to relieve pressure. This beneficial result had been obtained in some of the cases of Table IX, although the object of the operation had been primarily to remove the tumour or to explore. In six cases Horsley has operated to relieve pressure, in every case with success. Unfortunately he does not give the details of his cases. In Case XXIX there were no symptoms which warranted a focal diagnosis, but the headache was so severe that I advised an operation simply to relieve pressure. The benefit was somewhat questionable. The operation probably hastened the patient's death, but while he lived he had very little of his former headache. Two one-inch trephine holes were made close together, and these were connected by rongeur forceps. Were I to advise the operation again I should certainly have a larger opening made. It would probably be well to tap the lateral ventricles in addition. At any rate the escape of cerebro-spinal fluid seemed to cause great relief. Of course such an operation is simply palliative, but its results in these seven cases certainly warrant a further trial.

Surgical interference, then, is warranted in all cases where there is a reasonable probability that there is a new growth in an accessible situation. In such cases trephining is necessarily exploratory, because errors in focal diagnosis are pos-

sible and because we are unable to tell whether the growth be infiltrated or whether it may not extend into deeper parts of the brain. In cases where the focal diagnosis is uncertain, and where there are symptoms due to increased intra-cranial pressure, especially where there is intense headache, relief may be obtained by the removal of a large piece of the skull.

The details of the operation require a little consideration. In the first place I hold that it should be done early. Seguin [59] has suggested that it may be done too early, before the tumour is large enough to be found. This suggestion is not without value, but, on the other hand, if we wait too long the optic neuritis may progress so as to cause incurable blindness, and the patient's strength may be so much reduced as to diminish his chances of surviving the operation. The contra-indications to an operation, beside those relating to the seat and nature of the growth to which I have already referred, are those of any severe surgical operation. Optic neuritis is, of course, not a contra-indication, as it was present in many of the successful cases.

On the day before the operation the patient's head should be shaved, which will afford an opportunity to observe any scars or depressions in the skull. The important fissures and the probable seat of the growth should be marked out with a dilute solution of nitrate of silver. This is the only thing that will resist the antiseptic solutions, but too strong a solution will destroy the epidermis.

The most important fissure is the fissure of Rolando, for the majority of operations are done for growths in this region. I have elsewhere shown,* from measurements of heads and skulls, the inaccuracy of Broca's method. The Thane-Horsley method [60] is the one to be employed, and the cyrtometer devised by Wilson renders this easy. The prin-

* Boston Medical and Surgical Journal, 4, 11, 18 April, 1890.

ciple is to measure backwards from the glabella to the inion (occipital protuberance) 55.7 per cent. of the whole distance. Then draw a line forwards and downwards at an angle of sixty-seven degrees for 5.6 centimetres. This will give very accurately the upper two-thirds of the fissure. The lower third runs a trifle more vertically for two centimetres more. For the fissure of Sylvius draw a vertical line from the stephanion to the middle of the zygoma. Draw a horizontal line from the external angular process to the highest part of the squamous suture, curving it up until it reaches the parietal eminence. The two lines will join at the beginning of the fissure. The horizontal line will correspond with the horizontal branch of the fissure. The vertical branch is about 2.5 centimetres long and is inclined a little forwards from the vertical line. For the parieto-occipital fissure find the lambda, or, measuring from the inion, take 22.8 per cent. of the distance from the inion to the glabella, which will give the lambda. Then take a point three millimetres in front of the lambda, and draw a line 2.25 centimetres long at right angles to the median line. The superior occipital curved line corresponds fairly to the transverse fissure. These rules will enable us to determine the main fissures, which are the chief landmarks in operating. For further details I must refer to Dana's[61] careful study of cranio-cerebral topography.

Having drawn the lines desired the head should be carefully scrubbed with alcohol and a corrosive solution, or with whatever other antiseptic solution is desired, and protected by a bandage. The next day the bowels should be moved, the head again washed, and a subcutaneous injection of morphine ($\frac{1}{4}$ grain) given. The operation must be absolutely aseptic. It is still a question whether chloroform is preferable in such an operation. Most American surgeons would prefer to avoid the risk of chloroform and use ether,

but this probably causes greater congestion of the head. A rubber tube tied tightly around the head is sometimes efficacious in controlling hæmorrhage from the scalp, although I have found comparatively little benefit from it in one or two of my cases. The precise point of operation can best be determined by driving a small tack into the skull, which will keep in position after the flap is laid back.[62] After the flap is once laid back it is difficult to determine with accuracy any part on the skull by the aid of skin markings. The skull should be laid bare by a curved incision, with the convexity pointing upwards and backwards. This incision should be made straight to the bone, and the flap reflected. The hæmorrhage is often great and it must be controlled before proceeding farther.

In Case XXIX the operation was done with the patient in a sitting position, which Bradford advises, as affording an easy method of combatting beginning collapse; since, should this occur, by putting the patient in a horizontal position, more blood can at once be brought to the brain. By operating in this position there is likely to be less bleeding. One of the cerebellar cases was operated on with the patient lying on his belly and the head bent downwards over the edge of the table.

Our methods of opening the skull are still somewhat defective. A dental engine, or an electric motor, if at hand, affords the best device. A large opening must be made, and a large trephine will not act well on a curved surface, while to make repeated openings with a small trephine, connecting them by a saw or rongeur forceps, prolongs the operation and adds to the shock. If there be oozing from the diploë, it should be checked by plugging the bone with the following wax, which is recommended by Horsley: —

℞.

Ceræ flavæ	1 part.
Vaselini	4 parts.
Acidi Carbolici $\frac{1}{40}$ . . .	
Ceræ albæ, ana	q. s.

℔. Boil before using.

There seems to be an objection raised by many against opening the dura, if nothing appears on removing the bone. If the operation be aseptic I cannot see that it adds materially to the risk; if the operation be not aseptic nothing should be done. To fail to open the dura seems to me like hiring a locksmith to open a trunk in search of an article, and then to fail to lift up the tray within the trunk. On removal of the bone the dura will probably bulge. It should be opened, not by the old crucial incision, but by a curved incision near the edge of the opening of the skull.

Horsley[60] lays some stress upon a discolouration of the brain over a new growth. If this be not seen the brain should be very gently palpated. If nothing is felt, and the focal diagnosis is reasonably certain an incision may be made in the cortex, vertically to its surface, and the finger inserted. Palpation, if the opening be large, can be practised under the edge of the opening, and thus be extended over a considerable area. Prolonged manipulation of the brain should be avoided. If a tumour be found it can be removed by the finger or by means of a scoop. All bleeding vessels should then be tied. A ten per cent. cocaine solution may check any troublesome oozing from the brain.

The question of drainage is still in dispute. It is perhaps better to drain for twenty-four hours. Keen and Park are in favour of drainage, but Horsley has abandoned it.

Having removed the growth the dura should be sewn up, the bone, which should be kept warm and aseptic, should

be cut up into fragments and replaced upon it, the skin sewn up, and an antiseptic dressing applied.

The after treatment requires little discussion, as it differs in no way from the after treatment in any severe operation. In favourable cases patients do surprisingly well. After trephining for epilepsy, when a bit of brain tissue was removed, I have known a patient to sit up and read in two days, and to be walking about the ward in a week.

The risks of operation are, first of all, shock. This is to be prevented by an early and rapid operation, and by the ordinary methods at our disposal, such as stimulants, heat, etc. The second risk is sepsis, which, of course, is to be avoided by the most rigid adherence to the principles of antiseptic surgery. Hæmorrhage has proved fatal in a few cases. The risk of œdema of the brain from the sudden removal of the growth, on which von Bergmann [58] lays stress, seems to be exceptional.

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