TUMOURS OF THE FRONTAL LOBE

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General Considerations

The difficulties in diagnosing frontal tumours have been well recognized, not only by all writers on the subject, but by all clinicians whether they have contributed to the written word or not. The reasons for this are not hard to find, and centre chiefly round the fact that large though the frontal lobes are they contain no structure whose destruction must necessarily cause an obvious alteration in the patient, an alteration that could not have been produced by a lesion elsewhere. Objections to this statement come to mind, for it will be remembered that the motor area and the pre-motor area both lie within the frontal lobe. They demarcate, however, its extreme posterior border and it is clear that unless a tumour is so far back in the frontal lobe that it almost ceases to be a pure frontal tumour no disturbance of motion will result. On the left side the so-called motor speech

centre, or Broca's area, comes within the anatomist's definition of the frontal lobe, so that difficulties in the exteriorization of speech might well be found with a frontal tumour far enough back to involve these mechanisms. We must not be unduly impressed with the importance of the grey matter of the cortex at the posterior end of the left third frontal convolution. Tumours, and for that matter other lesions as well, more commonly produce their effects by interference with the white matter, by cutting deep association and projection fibres, than by destruction of the layers of cells in the cortex. It is more probable that defects of speech are due to damage to these deep fibres than to loss of cells with a special function in the cortex. This belief has led in more recent years to speech defects being referred to damage of 'the region of the insula' (island of Reil), a brain area that belongs as much to the temporal lobe as

Sketch of a right frontal lobectomy showing the bare floor of the anterior fossa falx and left anterior cerebral artery.

to the frontal. Tumours have a way of transgressing the anatomical boundaries of tradition; a tumour may be temporal as well as frontal, temporal as well as parietal, occipital as well as both temporal and parietal. When, therefore, one wishes to speak of the signs of a tumour in any area it is best to confine the examples to lesions which are entirely local. Hence frontal tumours of classical description will only slightly, if at all, encroach on the motor area, nor will all of those even on the left side lead to deficiency in speech. Is there then within the frontal lobe no other structure, is it an unbroken mass of white matter with the usual grey matter covering it? There lies within it the caudate nucleus and the front end of the lateral ventricle. It may be that the tremor to which Grainger Stewart first drew attention in 1906 and has since been noticed by many as a peculiarity of frontal tumours, is due to partial involvement of the caudate nucleus and basal ganglia. The anterior horn of the lateral ventricle could have no neurological significance, but it is important that the foramen of Monro lies within the frontal lobe and if a tumour is so situated that it could block it, a great rise in intracranial pressure must follow. Otherwise the pressure within the skull tends to be low with anteriorly placed tumours, as Stopford first pointed out though not for the reason that he adduced (absence of pressure on the vein of Galen).

In this review of the neural structures of the frontal lobe, in the search for areas whose destruction might lead to outspoken signs or symptoms, no mention has been made as yet of intellectual processes. Are not the frontal lobes the 'organs of the mind,' the organizers of personality, the censors of conduct, almost one might say the seat of the soul? Is there no recognizable anatomical cortical area which houses the mind? Although the frontal lobes are concerned in these things, and probably more concerned than any other part of the brain, all the evidence points to these qualities being more diffusely spread throughout the brain than was thought as recently as 20 years ago. It is more possible for a patient to have a large frontal lesion without alteration in his memory or personality or moral sense. Sometimes of course these things are disturbed, but much less commonly than would be the case if the frontal cortex was an exceedingly highly specialized mosaic where the whole of our intellectual and (for want of a better word) spiritual potentialities or achievement were lodged. This fascinating subject will be dealt with a little more fully below.

Symptoms and Signs of Frontal Lobe Tumours

As the result of the analysis of frontal lobe

tumours which have been made during the past 30 years a certain uniformity of description has emerged. Writers differ in the emphasis which they place on this or that symptom or sign; such emphasis tends to be individualistic and what has helped one observer may be thought unimportant by another, even though he agrees that the observation is correct. However desirable it may be for teaching purposes to produce a syndrome, a formula, something which shall represent the photographic likeness of a disease entity, we know very well that in practice the resemblance will be a family rather than an exact one. It is exceedingly rare for any case to show every sign which the closest scrutiny of a large number of cases has proved to be a possibility. Only too often but one or two of the theoretically possible diagnostic critical points are all there is to work on.

We will begin with a list of symptoms and signs, and first amongst them we must place those of that rise in intracranial pressure which is the general rule in intracranial tumours.

1. *Signs of raised intracranial pressure:* headache, choking of the optic nerve heads, vomiting, paralysis of cranial nerves at a distance, usually the abducens, and (in later stages) drowsiness.

2. Signs and symptoms due to *interference with the brain mass* of the frontal lobes:

(a) Epileptic fits.

(b) Memory defects, personality changes, incontinence, inertia, loss of powers of concentration and of introspection, facetiousness, unwarranted good humour (Witzelsucht, euphoria).

(c) Weakness of the contralateral limbs, perhaps a 'grasp reflex.'

(d) 'Motor' aphasia, if the lesion is left-sided in a right-handed person.

(e) Tremor of the arms, often ipsilateral.

(f) Pseudo-cerebellar signs—dizziness, ataxia, nystagmus.

(g) Electro-encephalographic evidence of a focal or general disturbance of cortical activity.

3. *Neighbourhood signs*, due to compression of nearby structures:

(a) Anosmia, uni- or bilateral.

(b) Optic atrophy, especially when it is unilateral. Central scotomata. Foster Kennedy—Paton syndrome.

4. *X-ray evidence of:*

(a) Calcification in the benign frontal gliomas.

(b) Hyperostosis of the floor of the anterior fossa or of the vault, associated with some, but not all, meningiomas.

(c) Increased local diploic vascularity.

Discussion

If all the signs mentioned above occurred always we should have a very clear picture indeed, unless

ANALYSIS OF THE SYMPTOMS OF 50 FRONTAL LOBE TUMOURS

	20 Cases of Meningioma Per cent.	30 Cases of Glioma Per cent.
1. Pressure signs:		
Headache, vomiting, visual disturbances, etc.	90	77
2. Symptoms and signs attributable to the frontal lobe:		
(a) Epileptic attacks:		
Generalized	20	50
Localized	10	27
Minor	15	13
(b) Contralateral pareses	20	27
(c) Other pyramidal signs	25	23
(d) Early apathy, loss of concentration, memory defects, euphoria, personality changes	30	20
(e) Grasp reflex	5	3
(f) Aphasia	25	10
(g) Hypersomnia	15	3
3. Pseudo-cerebellar signs:		
(a) Ataxia	25	0
(b) Tremor	10	7
(c) Vertigo	25	20
(d) Nystagmus	5	3
4. Neighbourhood signs:		
(a) Anosmia	25	7
(b) Optic atrophy, and Foster Kennedy-Paton syndrome	25	7
5. X-ray evidences:		
Calcification, hyperostosis	40	33

the cerebellar signs were also outspoken. There might then be a conflict in the observer's mind as to which signs were the more reliable. It is better not to get involved in that problem at this stage, so we will go back to the signs of raised pressure. Unless these are insistent the idea of a tumour being present is scarcely likely to arise. This is, alas, only too true and many a patient with a frontal tumour has been thought to be a hysteric or to be at fault in some system other than the nervous. It is a good working rule to attribute nervous symptoms to the nervous system. The practitioner is not always so ready to do so as he should be. Headaches are often dismissed as bilious or migrainous, vomiting as due to gastric or other sub-diaphragmatic disorder, and the symptoms otherwise generally attributed, if sex and age make that possible, to menopausal disturbances. Unless the patient has a fit or the doctor an ophthalmoscope things may drag on indefinitely in this way until the tumour is inoperable. A fatality at this late stage may confirm the doctor's impression that brain tumours are very risky things and that most patients die of them. Although there must of course be a substratum of truth in such an opinion, otherwise it would not have arisen, the more conscientious would more properly wonder whether the picture would not be different if tumours were diagnosed when they were small. They never will be recognized at that stage unless the practitioners to whom the patients first go with their complaints are

'tumour conscious' and ready to suspect a brain lesion on little evidence. The last ten years has shown a remarkable improvement in this direction in this country, for which praise must be given to him to whom it is due, the family doctor.

1. *General pressure signs.* There is nothing very noteworthy about the headaches from which these patients suffer. They are often not very severe, but they are continuous and progressive. They do not have the paroxysmal qualities of the pain produced by some tumours in the posterior fossa. The pain may be unilateral, and then it is generally on the same side as the tumour. It has to be differentiated from migraine in which the pain is commonly unilateral, may be accompanied by fleeting visual disturbances (teichopsia) and by vomiting, but it dates back commonly over years, is well spaced in time, and periodical. It does not have the continuously worrying character of tumour headache. Some patients with tumour, and even with very high pressure, have no headache at all, but thanks to the work of Pickering in England and of Harold Wolff and his collaborators in New York it appears probable that headache is caused by distortion of brain-meningeal relationships. The only structures within the skull in which pain can be experimentally induced are the basal dura, the great vessels and the cranial nerves; pain cannot be caused by stimulating the brain itself. It appears therefore that a tumour causes headache by slight rotations or drags such as distort the relationship of the brain to the pain-

sensitive structures mentioned. When a patient has no headache and yet has a tumour we assume that insufficient disturbance of intradural relationships has occurred.

Changes in the optic discs, papilloedema and congestion of the nerve-heads are present in most cases. It is undoubtedly possible for a tumour in the anterior fossa to attain some size before any pressure signs arise. The brain does not so tightly fill its membranes and bony capsule that the slightest encroachment on the intracranial space leads to clearly recognizable cerebral embarrassment. A small tumour in the posterior fossa will cause trouble much earlier than one in the prefrontal region because it will interfere with the circulation of the cerebrospinal fluid. In that case the mass of fluid in the dilated ventricles must be added to that of the tumour itself if we wish to compare tumour sizes with tumour effects. Equally a small meningioma springing from the sphenoidal wing, burying itself in the brain and hindering the outflow of cerebrospinal fluid from the lateral ventricle will, in the end, cause more disturbance than a much larger tumour at the frontal pole. The outflow of cerebrospinal fluid may be interfered with by the cerebral oedema which is so common an accompaniment of the gliomas, and which itself adds to the bulk of the tumour proper. It is less common in meningiomas (though it can occur with these as well) and thus it comes about that a frontal tumour springing from the meninges of the convexity may attain a considerable size over a number of years before its bulk is comparable with a glioma. Some of the gliomas, to be sure, are very slow growing and it is not so rare as might be imagined to have a five-year, a ten-year or even longer history with tumours of this sort. Nearly all of these are astrocytomas. And since this tumour tends to weave itself amongst the normal neural structures, and even to push them on one side, disturbances of function are not nearly so obvious or so early as they are with the malignant types of glioma (especially the dreaded spongioblastoma multiforme, still, in spite of radiotherapy, an incurable lesion). Vomiting is a rare and always a late symptom, as will be deduced from the foregoing considerations, for it requires for its production involvement of centres either in the region of the third ventricle, the hypothalamus or the posterior fossa.

2. *Signs and symptoms due to interference with local brain mechanism.* (a) *Tremor* has already been mentioned and is rarely an outstanding sign or complaint. Much more important are *epileptic fits*. These occur in about 50 per cent. of frontal tumours, and they are often almost, or even quite, indistinguishable from the idiopathic variety. They are commoner in glioma than in meningioma

cases. If the tumour spreads far back towards the motor area the myoclonic movements may preponderate in the contralateral limbs and may rarely be Jacksonian. Most often the convulsions come without warning or with an uninformative aura. The importance of this apparently idiopathic characteristic is that the fits may be the only sign of a tumour for months or years. How are we to distinguish fits of this kind from the idiopathic? Only by general inference and by auxiliary tests. The vast majority of idiopathic epilepsies begin before the patient is 20 years old. As age advances true idiopathic epilepsy becomes rarer, and it is our duty to search most carefully for a cause in all patients in whom it starts during the third, fourth, fifth and later decades of life. The fact that the fit begins with turning of the head to the opposite side is not diagnostic, because a great many youthful epileptics do that. James Collier (1929) thought that point important, but something else that he observed seems to the writer much more so—the tendency of these fits to be multiple, to approach or to attain ‘status epilepticus,’ if there is a tumour in the frontal lobe. This has certainly been so in some of my own cases. If, therefore, a man or woman in the forties, let us say, begins to have generalized fits and at the same time headache and some unaccountable falling off in working powers we ought certainly to suspect that a frontal tumour is present. If there is as yet no papilloedema the best plan will be to examine the cerebro-spinal fluid for pressure and for raised albumen content, and to make pneumograms or electro-encephalograms.

(b) *Loss of memory* for recent events, some subtle alteration in personality, loss of application at work and failure to recognize that duties must be completed, loss of reticence and decency, are frequently encountered in patients with frontal tumours. Complete moral breakdown so that the patient becomes delinquent or liable to imprisonment for his acts is in the writer's experience very uncommon (one case only). Much more common is it to find the patient good humoured and docile, though given at times to periods of unreasonableness impenetrable to persuasion or argument. Few of my frontal tumour series have realized the seriousness of their condition, few have been worried at the prospects of an operation, which to the lay mind is particularly dangerous, few have been bothered about money matters, the expense or duration of their illness has brought no anxieties or questionings. They have a certain amount of insight, for they may be brought to understand perfectly what is the matter with them, but even then they seem to be spared anxieties of a really worrying kind.

There is no doubt whatever that all of the mental

troubles so firmly linked in our minds until recently with tumours of the frontal lobes also occur with tumours elsewhere. As the result of experiences in the unilateral removal of the frontal lobes in man the writer has been driven to the conclusion that although the frontal lobes have much to do with the workings of the intellect, and of all the faculties subsidiary to it, that they are not its one and only seat. Nor can any differences be detected whether it be the right lobe or the left that is excised. If a frontal lobe is removed the patient is left quantitatively with less neural tissue than he had before to carry on his life and thought, to direct his conduct. So much is obvious. But what could not have been foretold is that the deprivation makes remarkably little difference to him. It is clear that thought processes are not so intensively cultivated in the frontal cortex that removal of a part leads to permanent loss of such and such a faculty of the mind. It would have been very interesting if it were so, for a careful correlation study of pathological material and of the observed clinical facts would have led to the establishment of individual human faculties in different areas of the cortex. Some might have been in the right lobe, some in the left. Such an arrangement would have completely justified the phrenologists, Gall and Spurzheim, the first localizers of function.

The facts are, of course, quite otherwise. Function is diffusely sown in the brain, though no one would deny that it is heavily concentrated in certain parts. The greatest mental defects are caused by bilateral frontal lesions or by tumours of the corpus callosum, which by cutting commissural fibres impair the activities of both lobes. Mesially placed frontal tumours are very apt to invade the corpus callosum. Severe symptoms will always, therefore, suggest callosal involvement. Mental power, in Foster Kennedy's (1911) words, depends on the exquisite integration of the entire brain. Disturbances of commissural fibres and tracts leads to an upset in this integration far more than does cortical damage, which is in human pathology rarely diffuse. It is worth making the generalization that the kind of mental alteration which brain tumours bring are dementias, the detailed patterns of which depend on the previous education and personality of the sufferers. But generally they take the form of loss of previous abilities. Schizophrenic characters do not occur—and would not except in those who are already schizophrenes. Noisiness and restlessness are most seen in folk who are noisy and restless by nature and this is true of head injuries as well.

Remarkable disturbances of personality sometimes follow left temporal lesions, neoplastic or otherwise. High intracranial pressure, wherever

the lesion, interfering as it does with the actions of the brain as a whole, is capable of causing great changes in alertness and in thought, and examples could be brought forward of a clear frontal lobe syndrome when the tumour was later proved to be elsewhere. We must therefore be wary of accepting mental peculiarities as incontrovertible evidence of a frontal lesion. If these changes are present in a person with no signs of high pressure that would be better evidence.

Incontinence. In the highly compressed, drowsy or stuporous patient incontinence is common wherever the lesion is. Normally few individuals past infancy micturate at the first feeling of desire to do so, they have acquired inhibition. The drowsy patient reverts to childhood's state and may wet the bed like the sleepy child. But for an adult to do so whilst awake is always a positive sign, usually of a frontal lesion—it is as positive a piece of evidence as papilloedema or a positive Babinski. Some patients are greatly disturbed by such events but explain that the bladder empties before they can prevent it, as if their powers of inhibition have vanished. We believe that the cortical inhibitory pathways to the bladder are chiefly subfrontal, linking the frontal lobes to centres in the hypothalamus. Incontinence is common happening after the deep bifrontal transactions of complete leucotomy, rare after limited dorsal frontal cuts. But although there is probably an anatomical basis in fibre-tract disconnections in frontal tumours, the thoughtful may be interested to pursue their own reflections on the subject of inhibition of micturition and the effects that sleepiness alone might exercise upon it. But it cannot too often be repeated that incontinence during wakefulness is a sign with localizing value. In the highly compressed, drowsy or stuporous patient this calls for no special comment. But it is another matter if it occurs when the patient is conscious. It is then strongly suggestive of a frontal lesion. In the analytical table of 50 frontal tumours it will be observed that mental and memory defects occur as an early sign in less than one-third of the patients. As a late sign there are few patients who do not show some alteration for they must all eventually become drowsy and incontinent. These alterations are due to high pressure and are very similar whatever part of the brain the tumour affects.

(c, d) Better still would be the additional presence of an aphasia, a difficulty in saying what the patient wishes though he declares that he is perfectly clear headed. This can only occur when the tumour is on the left side (with the usual reservation of right handedness) and far back. Similarly slight pyramidal signs, absent abdominal reflexes, may suggest the side of the tumour if not

its site. Sachs has stressed particularly weakness of the face on the opposite side; this certainly occurs but it is as frequent with temporal tumours. None the less Sachs' studies of frontal tumours are important; he regards fits, mental changes, and nystagmus as most important signs. A grasp reflex may be present, that reflex action by which the patient will always clench hold of anything which so touches his palm. It has been admirably described by Adie and Macdonald Critchley (1927). Weakness of the leg is rare in frontal tumour patients, because the motor area slants forward offering the face area most readily to the tumour.

(e) The cerebellar signs shown by some frontal tumours will best be dealt with in the differential diagnosis.

3. *Neighbourhood signs.* The olfactory tracts are so closely applied to the under surface of the frontal lobes that their compression by sub-frontal tumours is well imaginable. Complete anosmia, on one or both sides, is rare unless the olfactory bulbs are disorganized, as happens chiefly when there is an olfactory groove meningioma. Elsberg has introduced more delicate tests for estimating depression of the sense of smell short of complete loss. Special, even though simple apparatus, is necessary and outside hospital it will suffice if the clinician tests the sense of smell with whatever easily identifiable odours he can find to hand. These tests must be remembered, for especially if the loss is unilateral, the patient may be unaware and does not mention it. When it is bilateral (in the absence of nasal disease) it is always important, always organic, though it can be a false localizing sign (Cushing, 1916).

Unilateral optic atrophy caused by the pressure of a frontal tumour bearing heavily on the nerve of the same side is an extremely important sign. It was first given diagnostic significance by Leslie Paton, and worked up with illustrative cases in an important paper by Foster Kennedy, whose name is usually attached to the syndrome of atrophy of the optic nerve on the side of the tumour, papilloedema in the uncompressed nerve of the opposite side. Of recent years the significance of unilateral optic atrophy has more and more impressed itself on ophthalmologists and neurologists. It is an organic sign of the first order, and never more so than when the opposite disc is oedematous. Its presence means exactly what the facts indicate. (The present writer has published a study of compression of the optic chiasma by gliomas and discussed this syndrome fully, Doyné Lecture, *Trans. Ophth. Soc.*, 45, 1945.) The optic atrophy can be as well produced by a temporal tumour as by a frontal (see Fig. 124 in W. R. Henderson's essay on the

anterior basal meningiomas, 1938). Also one must beware of being deceived by an atrophy consecutive to severe choking of the discs which has become more complete on one side than the other.

4. No special comment is required by the *X-ray evidences* except to say that a number of the benign gliomas calcify and that the patterns of these calcifications need an expert eye to recognize. They may be very faint and easily overlooked. Of recent years angiography has proved of great use in the final diagnosis of brain tumours, its advantage being that it will often enough give evidence not only whether a tumour is present and where it is, but will also indicate its nature. It is particularly successful with the meningiomas because their self-contained fine capillary shadow makes a well-defined mass in the X-ray film. Equally it may demonstrate an unsuspected angioma or the wild vascular pattern of a malignant glioma.

Differential Diagnosis

We will assume that the fits and mental changes from which our hypothetical patient could suffer have been proved non-syphilitic by a negative Wassermann reaction. The chief differentiation necessary will be between either temporal or cerebellar tumours. Temporal tumours equally produce raised pressure (only more early because they tend to compress the third ventricle), facial weakness and fits. There are two points peculiar to temporal lobe tumours—uncinate fits and homonymous visual field defects. If neither of these is present, differential diagnosis can only be arrived at by mechanical means.

The possibility of cerebellar signs in frontal tumours has already been mentioned once or twice. This possibility of confusion is brought about by the important pathways which leave the frontal lobes and run to the pontine nuclei, there to be relayed to the cerebellum and labyrinthine apparatus. But it can be said at once that a full-fledged cerebellar picture is never given by frontal tumours. The symptoms and signs are suggestive and no more. Why then should difficulties ever arise? The reason is that cerebellar tumours themselves frequently give rise to symptoms and signs which are scarcely more obtrusive or classical. The clinical picture which Gordon Holmes (1922) has made so well known is best seen in injuries or vascular insults of the cerebellum. It can, of course, occur in patients with tumours, too, but not so often as might be thought. Even nystagmus itself may, if rarely, be minimal or absent in cerebellar lesions. It is quite certain that the coarse, unmistakable nystagmus of some cerebellar tumours never occurs with frontal.

Before having recourse to angiography or ventriculography the cortical potentials should be studied electronically. Sometimes extremely accurate localization can be given by these means by alterations of potentials, phase reversals and so forth as Grey Walter, Denis Williams and George Dawson have shown. Electro-encephalography has become an essential exploration of organic cerebral lesions. Further information can be obtained by angiography or ventriculography.

Operations on Frontal Tumours

In general the attack on a frontal tumour follows the lines generally adopted in neurosurgery. It is assumed that the aim is the removal of the tumour. This can sometimes be effected by enucleation, in other cases the extent and position of the tumour make the sacrifice of the lobe unavoidable. The lobe may have to be excised to give access to a tumour beneath it. The steps of the operation are as follows. An incision is made in the mid-line backwards from a point just above the glabella to one inside the hair line. The cosmetic result is superior if the incision is very accurately placed in the mid-line. Having reached the higher point the incision curves over the scalp to fall vertically towards the ear which it should reach. The scalp flap should be first marked out with iodine and then with a scratch. Novocaine-adrenalin solution (1 per cent.) is then injected along the proposed line of section. The whole scalp flap is dissected downwards and forwards, free from the bone. Great care must be taken by finger compression to keep the blood-loss during the cutting of the scalp flap down to a few cubic centimetres. Suitable points are now chosen for making drill holes, avoiding the frontal air sinus. The bone dust obtained in making the holes is saved for replacement at the close of the operation. The holes are most safely made by brace and perforator and burr worked by hand. After separation of the dura mater from the bone the holes are connected with the Gigli saw. The base of the bone flap in the temporal fossa may be narrowed with de Vilbiss forceps; it is then broken down, hinged on the temporal muscle. The bleeding from the exposed dura is usually negligible, and is well controllable with wet cotton or lintine. If bleeding is severe either there is a meningioma beneath the dura with a wide attachment to it, or the anaesthetic is wrong or the airway imperfect. It is a great handicap to good surgery to have a high intracranial pressure, hence the need for early diagnosis. The patient's chances of recovery are greatly endangered if the brain tends to bulge irresistibly through a dural incision. If the pressure cannot be lowered by puncture of an intracerebral cyst or

of the ventricle it will be wiser to break off the operation before more harm is done and try again under local anaesthesia alone later. Supposing that the dura can be safely opened this is next done, cutting it below, in front and behind. The cortex is now inspected for evidence of tumour, which may be very clear. If not it will be found by puncture. The recognition of the presence of a tumour, of its extent and especially of its nature needs considerable culture. It is on this vital point that the general surgeon was so constantly defeated. If excision of the frontal lobe is decided upon the cortical veins entering the superior longitudinal sinus are first coagulated so that the falx is clear. This necessitates a bone incision exactly on the mid-line. A half- or one-inch overhang will greatly hamper the securing of these veins. When clearly seen they are easily diathermized and divided. The line of cortical incision is next planned on the surface of the brain, the veins to be cut coagulated and the arteries occluded with silver clips. The line of this section will commence below, as a rule, at the junction of the anterior and middle fossae. The incision into the brain is best made with a narrow blunt dissector, which allows the recognition of deep vessels. The anterior horn of the lateral ventricle may be cut across if the tumour has not pushed it too far back. This does not matter. The only difficulty may be the clipping of the anterior cerebral artery on the mesial surface deep in. After the orbital surface of the brain has been cut through, the whole lobe is lifted out. All bleeding points are secured, the large cavity filled with Ringer's solution and the dura closed. A small drain can be left in between bone and dura for 24 hours, but it is certain that daily aspirations of fluid will have to be made during the next week or ten days. The dead space eventually settles down, it may permanently communicate with the ventricle.

Lobectomy gives the patient the best chance of long survival and obeys the surgical maxim of block removal of non-enucleable tumours. In none of the writer's cases has there been any important mental, moral or other disability as the result of it.

The prognosis depends on the nature of the tumour. It is excellent in the meningiomas and angiomas, hopeless in the malignant gliomas and intermediate in the more benign gliomas (astrocytomas). The writer has had several patients with astrocytomas who have survived five years and a few who lived usefully for over ten years after frontal lobectomy. The great advantage of the operation is that it leaves so large an 'internal decompression' that given a very slowly growing tumour the space serves them well.

FIG. 11.—Calcification in Rathke pouch tumour in a child. Note also convolucional thinning of vault and separation of sutures.