Cerebral decompression in ordinary practice : an address / by Charles A. Ballance.

Cerebral Decompression in Ordinary Practice

An Address

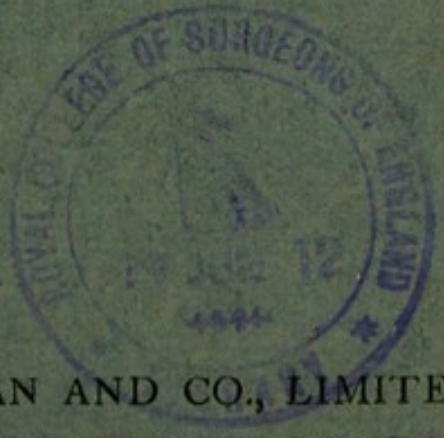
BY

CHARLES A. BALLANCE

M.V.O., M.S., F.R.C.S.

SURGEON TO ST. THOMAS'S HOSPITAL, ETC.

THE SUBSTANCE OF WHICH WAS DELIVERED WITH LANTERN ILLUSTRATIONS AT A MEETING OF THE BATH AND BRISTOL BRANCH OF THE BRITISH MEDICAL ASSOCIATION ON 28TH FEBRUARY 1912



MACMILLAN AND CO., LIMITED
ST. MARTIN'S STREET, LONDON
1912

Two Shillings and Sixpence, net.

Cerebral Decompression in Ordinary Practice

An Address

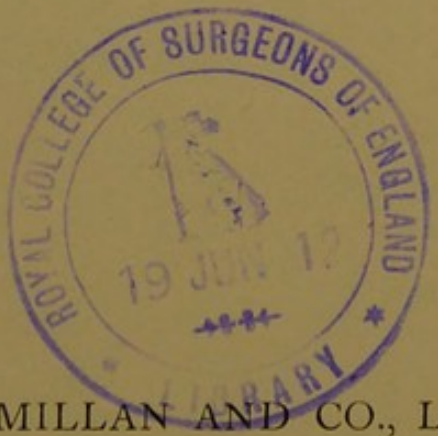
BY

CHARLES A. BALLANCE

M.V.O., M.S., F.R.C.S.

SURGEON TO ST. THOMAS'S HOSPITAL, ETC.

THE SUBSTANCE OF WHICH WAS DELIVERED WITH LANTERN ILLUSTRATIONS AT A MEETING OF THE BATH AND BRISTOL BRANCH OF THE BRITISH MEDICAL ASSOCIATION ON 28TH FEBRUARY 1912



MACMILLAN AND CO., LIMITED
ST. MARTIN'S STREET, LONDON
1912

SYLLABUS

METHODS OF DECOMPRESSION

(*a*) The Methods of Nature.
(*b*) The Methods of Art.

A. DECOMPRESSION FOR INJURY

1. Extradural Hæmorrhage.
2. Intradural Hæmorrhage.

(i) Of the Newly-Born and in Young Children.
(ii) In Later Life.

(*a*) With Fracture of the Base. (*b*) With Injury to a Sinus. (*c*) With Injury to the Vault. (*d*) Remote Effects of Intradural Hæmorrhage. Subdural Blood-Cysts. (*e*) With Laceration of Brain.

B. DECOMPRESSION FOR DISEASE

1. Apoplexy.
2. Hydrocephalus.
3. Meningitis.
4. Abscess of the Brain.
5. Tumour of the Brain.

CEREBRAL DECOMPRESSION IN ORDINARY PRACTICE

MANY morbid conditions occurring within the skull have one feature in common—increased intracranial tension. This constitutes their chief danger, this alone makes them more formidable than similar morbid conditions occurring elsewhere. I propose this evening to show you by examples that this increased intracranial pressure can be effectually relieved by operation. Cerebral decompression has become an established procedure, and can do so much in so many cases that the conditions under which it is called for should be widely appreciated.

We are all familiar with Nature's method of decompression in the case of children with hydrocephalus—the sutures gape and the head enlarges. Cases have been recorded in which separation of the sutures has taken place up to the age of ten years. Occasionally a tumour will cause absorption of bone by local pressure ; this is most often seen in children with cerebellar tumour, and may aid in the localisation of the growth. A case ([1]) has been recorded, too, in which a tumour in the middle fossa destroyed part of the wall of the temporal fossa. The destruction of the inextensile capsule of the brain at this spot was followed by the formation of hernia cerebri,

and then all the symptoms subsided, but the patient became blind from optic atrophy.

The methods at present available for the relief of increased intracranial pressure are: (1) Removal of a moderate quantity of fluid through a cannula from the subarachnoid space, as in lumbar puncture.

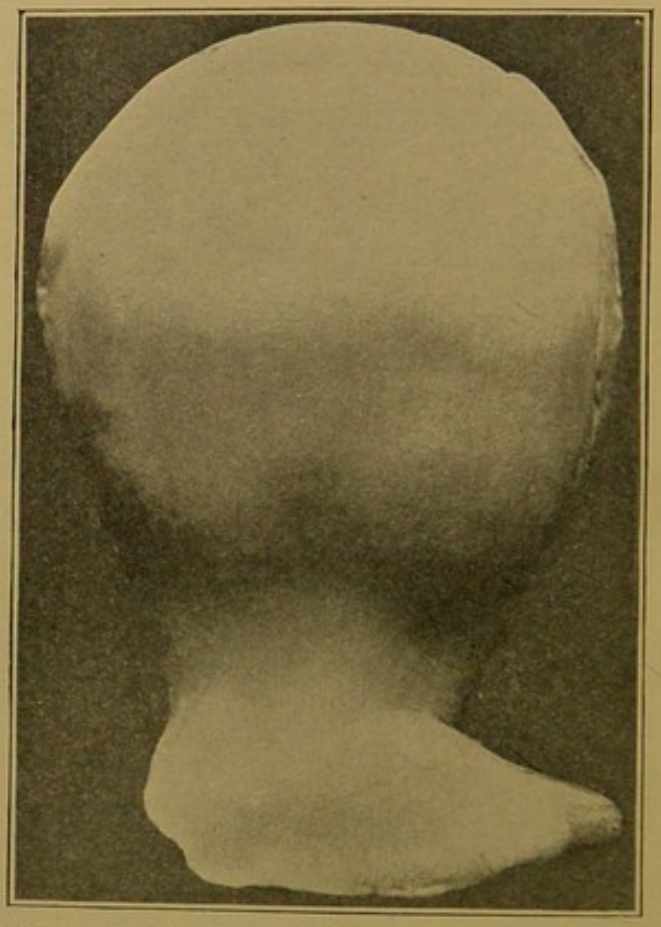


FIG. 1.—Bulging right occipital fossa in a child 3½ years of age, suffering from tumour of the right cerebellar hemisphere. The illustration is a photograph of a cast.

The symptoms were occipital headache, frequent vomiting, ataxic gait, bi-lateral optic neuritis, horizontal nystagmus, and bulging right occipital fossa. The child had been noticed to be ill for two months.

(2) Continuous drainage of cerebro-spinal fluid from a ventricle through a small tube either into another cavity or externally. (3) Removal of part of the inextensile boundary (bone and dura) of the cavity in which the brain is situated, so as to make an opening through which free drainage of fluid can be established, or a hernia may be allowed to occur which will relieve

the general pressure even if the exciting cause cannot be removed.

Decompression by craniectomy is most effectual when made over the actual site of the disease, and hence the problem of accurate localisation is a matter of supreme interest to the surgeon and of vital importance to the patient. It is not the mere bulk of a hæmorrhage, an abscess, or a tumour which is the cause of the increased intracranial pressure, but it is the increase in the amount of cerebro-spinal fluid which is induced by the presence of a foreign body. The acute symptoms, as a rule, arise not from local but from general pressure, as is shown by the frequency of death from failure of respiration. Decompression by craniectomy averts all the dangers of general pressure, as is proved by the many instances in which recovery has ensued though respiration ceased before the operation was begun, or during its performance.

There are two main divisions of the subject: (A) Cerebral decompression for injury, (B) Cerebral decompression for disease.

A. Decompression for Injury

The immediate or the more remote effects of injury may call for a decompressive operation. The remote effects and serious sequelæ of injury ought seldom to occur—they are often evidence of an opportunity lost. Effusion of blood into the cranial cavity may occur from injury in such amount that life is immediately threatened and will be destroyed if the pressure is not promptly relieved. In these cases the increase of intracranial pressure is rapid, and deepening coma is the dominant symptom.

The chief varieties of such hæmorrhage met with in practice are :

(1) *Extradural Hæmorrhage* from rupture of the middle meningeal artery. This is quite classical. It is familiar to us all, and I need not stay to speak of it. The fluid withdrawn by lumbar puncture is clear and not blood-stained.

(2) *Intradural Hæmorrhage.*—(i.) *Of the newly born* occurring from pressure on the skull during birth,

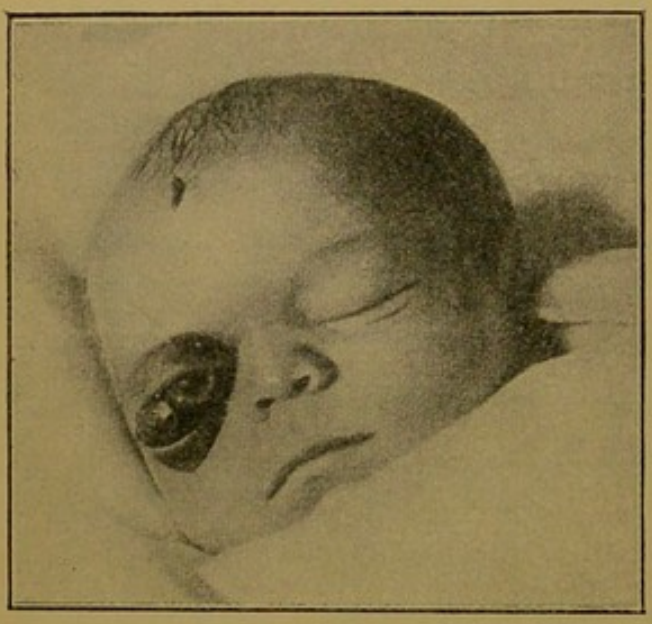


Fig. 2.

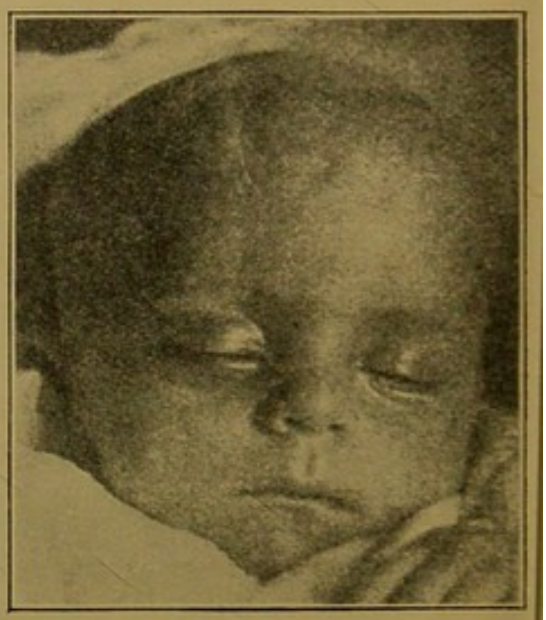


Fig. 3.

Figs. 2 and 3.—Intracranial hæmorrhage of the new-born. (Cushing.)

Fig. 2.—Photograph of 9-day-old comatose female infant. *Note* extreme degree of ocular proptosis and subjunctival hæmorrhage and œdema. Forceps delivery ; inability to suck ; tense fontanelle ; Cheyne-Stokes respiration, and gradual onset of coma.

Fig. 3.—Same patient. Photograph during sleep two months after operation. Complete retrocession of the exophthalmos.

mostly from forceps compression. If not immediately fatal, such hæmorrhages subsequently cause cerebral palsy, epilepsy, and other nervous disorders which may prove a lifelong disablement. The immediate symptoms to which they give rise are : inability to suck, a bulging fontanelle without pulsation, unilateral palsy, a stabile pupil on the side of the

hæmorrhage, irregular respiration, slow pulse, fever, and ocular proptosis. Cushing (2) has drawn attention to the surgical relief of this condition. I have not myself had the opportunity of operating on such cases, but Cushing's paper should prove a stimulant to much good work in this direction in the near future.

Other important results of injury to the head *in young children* are pulsating subcutaneous tumours which may be pulsating hæmatoma, pulsating meningocele, or pulsating encephalocele. These, like the subdural hæmorrhages of the newly born, require craniectomy. Such operations are very successful.

There is an account of a case of a large traumatic encephalocele in my small book on the *Surgery of the Brain*, p. 36. The patient was a boy aged 4 years, and the encephalocele could not return into the cranial cavity till the small opening in the bone and dura was enlarged. No doubt after the stress of the injury had passed the edges of the fracture in the elastic parietal bone sprung back into their original position. In Fig. 4 a large hernia cerebelli which followed the evacuation of a large cerebellar abscess is seen, which resisted all treatment till decompression was performed by enlarging the opening in the bone and dura.

(ii.) *Intradural Hæmorrhage from Injury in later Life.*—Of this the hæmorrhage occurring in fracture of the base of the skull is a common example.

(*a*) *Fracture of Base of Skull.*—A man, aged 29 years, was admitted to hospital on the 14th of October 1908, after having been knocked down by a motor-car. He was semi-conscious and bleeding freely from both ears. Three hours later coma was complete, the pulse was slow and irregular, and the respiration was of the Cheyne-Stokes type. *Opera-*

tion.—Temporal muscle split vertically, a one-inch trephine opening was made, exposing the dura at the lowest part of the middle fossa. A little blood was found between the dura and the bone. Dura dark, not pulsating, and bulging. On incision of the dura about a teaspoonful of clot and some fluid blood was expressed by the intradural pressure. The temporal muscle was allowed to resume its normal position, so that blood-clot and cerebro-spinal fluid could ooze out beneath it. The same operation was repeated on

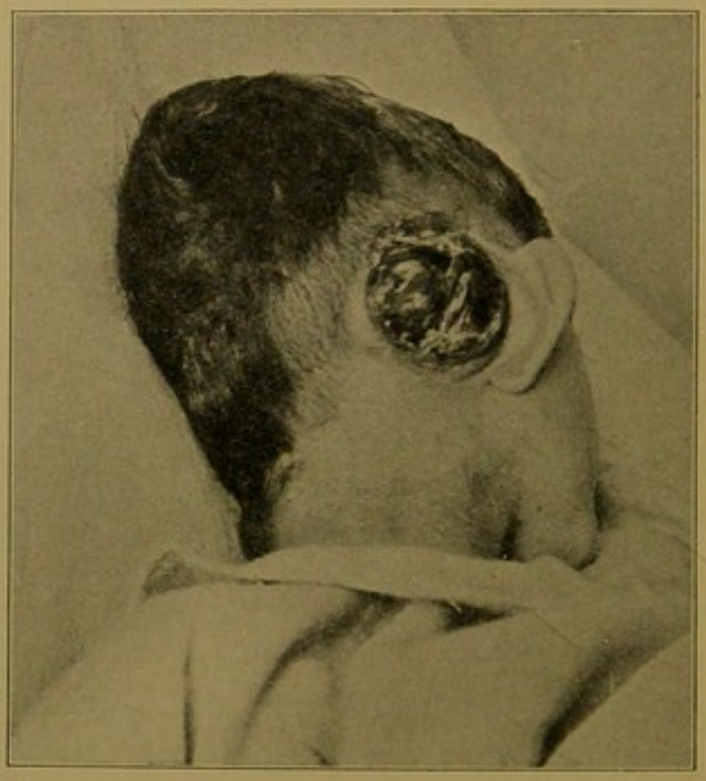


FIG. 4.—Persistent large cerebellar hernia following the evacuation of a cerebellar abscess in a young girl. (See page 5.)

the other side. Next day the patient was conscious. He had incontinence of urine and fæces for three weeks, but made a good convalescence during which he talked incessantly. He resumed his work on the railway in April 1909. In December 1909 he was perfectly well except for some defect of smell. He never had a headache.

(*b*) Another example not very uncommon is *injury to a sinus*. When one of the larger sinuses is injured the effects of the bleeding may be imme-

diately serious, or even fatal, or the sinus may become infected—in either case operation is called for. A case occurred many years ago, in St. George's Hospital, of injury to the lateral sinus, in which death soon took place from ([3]) repeated hæmorrhage. The patient was a powerfully built man aged 51 years.

The following is an example of lateral sinus infection following injury ([4]).

A day labourer, aged 50 years, while intoxicated fell against a stone. At first only a hæmatoma in the occipital region was made out, this suppurated, and the skin over it sloughed, exposing the bone. On the eleventh day the first rigor occurred, and thereafter rigors occurred almost daily. On the fifteenth day the patient was drowsy, and on the twentieth day he died comatose. *Autopsy.*—The bone was infiltrated with pus and much discoloured. An irregular fissure extended through the bone for about two inches and reached the jugular foramen. The dura mater beneath the diseased bone was covered with a thick layer of pus. The lateral sinus contained a suppurating thrombus, and there was a thrombus in the internal jugular vein ; there was an abscess in one lung and an empyæma. This case obviously required a decompressive craniectomy, and also surgical intervention for the arrest of the septic process.

The above case may be compared with cases which have proved fatal after accidental operative injury to the sinus.

(*c*) The following case is an instance of recovery after operation for comminuted simple fracture of the vault, with wound of superior longitudinal sinus, extra- and intra-dural hæmorrhage, and optic neuritis.

Female, aged 15 years, admitted to St. Thomas's Hospital, May 1909. She had fallen on her head from a height of

twenty feet through a skylight. On admission semi-conscious, very irritable, and seemed in pain. Large hæmatoma over left fronto-parietal region, extending across the middle line. There was no palsy, the plantar reflexes were normal, the knee-jerks were not obtained, the other reflexes were normal. Three days later she was more drowsy, and had vomited several times since admission, there was bi-lateral swelling of optic discs measuring 2.5 D. with much engorgement of veins. *Operation.*—Scalp reflected on both sides of the middle line, multiple fractures of left fronto-parietal region, extending by fissures across the middle line. Bone trephined. Some blood outside dura. Dura opened, much clot over hemispheres on both sides of the median line, most of which had come from a wound of the superior longitudinal sinus. The sinus was tied and the blood-clot removed.

Result.—The retinal fields soon became normal and complete recovery ensued.

In discussing a case of intra- and extra-dural hæmorrhage with severe symptoms of compression, Cushing says : "The ophthalmoscope offers a method of determining in clinical cases the presence or absence of intracranial venous stasis, inasmuch as the congestion is transmitted to and is easily observable in the veins of the fundus. Thus the eye grounds become almost equal in diagnostic value to the experimental window."

The principles of treatment in cases of depressed and punctured fractures of the vault by decompression operations are so well understood that reference to them is superfluous.

(*d*) *The remote effects of injury* calling for operation are numerous. The subject is too large for me to deal with now, and I can only enter into one detail of it, viz. *blood-cysts and so-called false membranes* ([5]) *in the meninges* (*subdural space*) *resulting from hæmorrhage.*

In these cases the original effusion of blood may be moderate in amount, or, at any rate, does not give rise to increasing coma or other alarming symptom. The effused blood becomes encysted and, like a blood collection in the tunica vaginalis testis, may continue to increase slowly in size. The signs of pressure on the brain are intermittent. The brain becomes accustomed to the pressure of the cyst and the patient is apparently well. From time to time a small fresh hæmorrhage occurs and then the symptoms recur. Such cysts were described by Richard Bright in

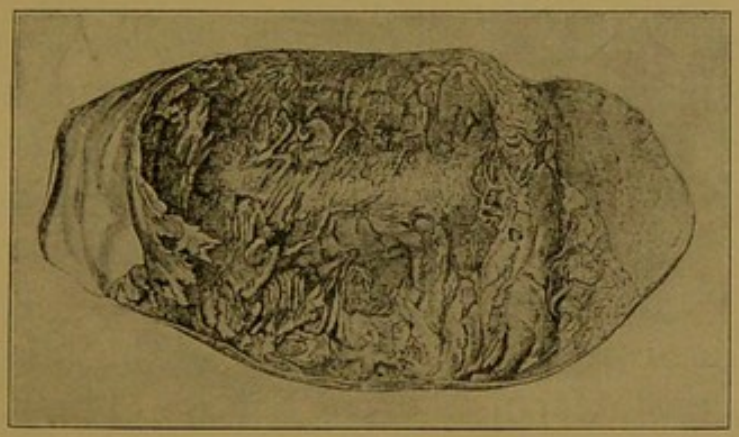


Fig. 5.—Subdural blood-cyst attached to the parietal arachnoid (inner surface of dura). (From Prescott Hewitt and Hulke's article in *Holmes' System of Surgery*, 1883.)

1831 ([6]). I have successfully operated on several such cases.

Prescott Hewitt ([7]) published a striking case in 1846.

A boy, aged 8 years, received a blow on the head from a cricket ball, and shortly afterwards showed symptoms of insanity. He had recurrent attacks of insanity with intervals of health until his death, fifteen years after the injury. The symptoms in the last attack were headache, vomiting, and drowsiness. At the autopsy a large subdural cyst was found.

In 1906, I saw a man, aged 48 years, who the

year previously had received a blow on the right side of the head. His sight was much impaired and he had bi-lateral papillitis with hæmorrhages. He was dull with constant headache, his gait was feeble and slightly unsteady, but there was no paralysis. At the operation a thin layer of clot enclosed in a membrane was found over the right cerebral hemisphere, and was removed. The man made a good recovery.

On another occasion I successfully removed a subdural cyst measuring 7 inches in its long diameter, $4\frac{1}{2}$ inches in its short diameter, and $1\frac{1}{2}$ in thickness. The patient was a man aged 34 years ([8]).

A woman, aged 50 years, had a fracture of the femur and other injuries twenty years before I saw her. Her symptoms when she came under my observation were giddiness and pain in the back of the head extending down the neck. There was some tenderness on deep pressure in the left occipital region, but no deformity of the skull. The symptoms, which had been present for about a year, increased in severity, and a little later optic neuritis became manifest. The left cerebellar hemisphere was exposed, on it was seen a soft brown mass, apparently the remains of old blood extravasation and false membranes, the mass was picked off with forceps. The vertigo was immediately relieved and did not recur. After a protracted convalescence, accompanied by grave mental disturbance, the patient made a good recovery and was able to resume her duties.

Bearing in mind that in almost all serious head injuries blood is effused into the cranial cavity, it may well be that in certain cases of *intermittent headache*, *intermittent paralysis*, *or intermittent insanity subsequent to head injury* the pathological lesion present is

subdural hæmatocele—a condition certainly remediable by operation.

(*e*) Hæmorrhage also occurs *into the subarachnoid space and into the brain itself, in injuries causing laceration of the brain*, even though no fracture of the skull has occurred. The extravasated blood is itself a foreign body, and as such causes increase in the amount of cerebro-spinal fluid, and increased intracranial pressure. In some of these cases, which are common in hospital practice, the patient remains for many days or longer in the state of cerebral irritation, conscious only when roused, curled up in bed, restless, sleepless, and irritable. In such cases great relief is afforded by lumbar puncture, which withdraws cerebro-spinal fluid, reddish in colour and under pressure. These cases are known to every one. A striking example occurred to me some few years ago :

Laceration of brain without evidence of fracture. Cerebral irritation. Lumbar puncture. Recovery.—I was called to see a well-known jockey, who had been thrown the previous day while riding in a race. It was thought that the back of his head had received the impact of the fall, but there was no sign of injury to the head, and no evidence of fracture of the skull. He was quite unconscious and very restless and irritable. There was ptosis of the left eye, and the left pupil was dilated and stabile. The right pupil was small and stabile. The right arm was partly paralysed. On lumbar puncture a large quantity of blood-stained fluid was removed, and five hours quiet sleep was at once obtained. Unconsciousness lasted five days, and the signs of cerebral irritation for another week. The catheter had to be passed for three weeks. The lumbar puncture was repeated on several occasions, and always gave marked relief to the restlessness and irritability. The patient ultimately recovered.

Thus decompression by this simple method

relieved a dangerous condition and allowed time for recovery. Relief might have been still more rapid and perhaps more complete from a frontal decompressive craniectomy. When relief was obtained by lumbar puncture an operation on the skull seemed uncalled for, but I am not sure now that the best course was adopted. The convalescence was prolonged, and the patient has never been able to ride again. In blows on the back of the head the anterior poles of the frontal and temporal lobes receive the most damage. It was thought that in this case the stress of the injury had fallen in chief part on the left frontal lobe.[1]

B. Decompression for Disease

1. *Apoplexy.*—In this emergency acute compression and laceration of the brain occur; the patient does not die from loss of blood but from pressure on the brain. The pressure is both local, from the pressure of the extravasated blood, and general, from the increase of cerebro-spinal fluid caused thereby. We operate for internal hæmorrhage in all other parts of the body except the lung, and when the methods of preventing pneumothorax have been perfected hæmorrhage from the lung will become accessible to direct treatment. Lumbar puncture has been done in cases of acute apoplexy with apparent benefit, as in a case published by me in the *Lancet* for January 20th, 1912. Certain cases of chronic apoplexy (9) have been submitted to operation and the clot removed, but in the *acute stage* only one attempt has, so far as I am aware, been made to relieve the pressure by a craniectomy.

[1] Abscess of the brain sometimes occurs years after injury to the skull. The subject is referred to in the Addendum.

The diagnosis of apoplexy is not difficult, it can seldom be mistaken for thrombosis or embolism, and it is by far the most frequent of these three catastrophes.

It seems to me that the indication to operate is in these cases clear. Dangerous general pressure on the brain, the cause of coma and arrest of respiration, would be relieved, the symptoms produced by local pressure would pass away at once on the evacuation of the clot, and no further local damage would occur.

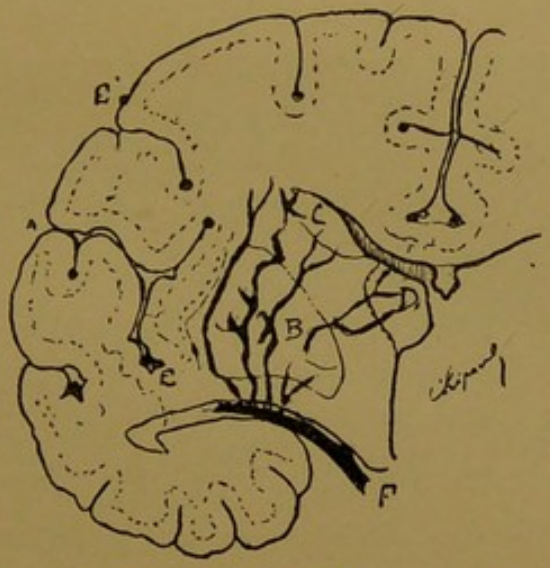


FIG. 6.—Section of brain showing the central grey matter and its arterial supply from the internal carotid. (Chipault.)

A, Fissure of Sylvius.
B, Corpus striatum.
C, Nucleus lenticularis.
D, Optic thalamus.
E, Cortical arteries.
F, Internal carotid artery with its branches, lenticulo-thalamic and lenticulo-striate. One of the latter is termed by Charcot the "artery of cerebral hæmorrhage."

The conductivity of nerve tracts not destroyed but only compressed by the hæmorrhage would be restored. The operation would be simple, the patient need not be disturbed, the preparation of the site of operation is quite easy. The craniectomy need not be large, the incision in the brain should be behind the fissure of Rolando and above the fissure of Sylvius, and should be made at the summit of a convolution. The effused blood would escape

through the incision, and as the ruptured vessel is commonly a small one, no further bleeding would be likely to occur. In any case a strip of ribbon gauze soaked in adrenalin, and passed through the incision in the brain, would arrest it. I trust that this proposal will receive in selected cases the careful consideration of the members of your Society.

Cushing writes: "The majority of cases of apoplexy which are exposed on the post-mortem table show a large clot more or less readily accessible

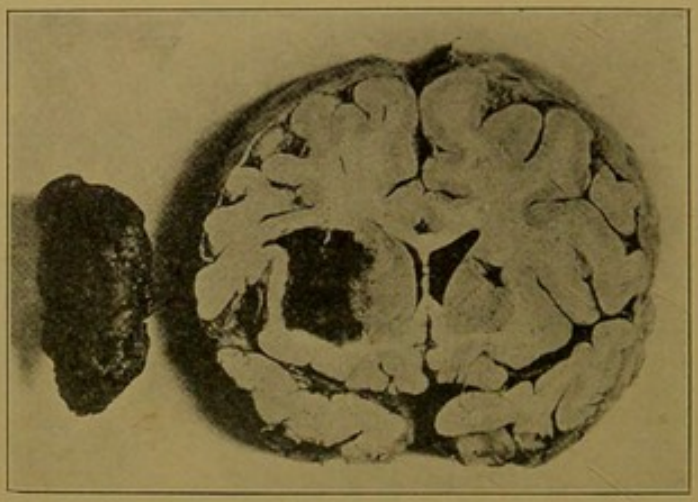


Fig. 7.—Section of brain of an epileptic who died from the effects of capsular hæmorrhage; with clot which extruded itself at the autopsy. (Cushing.)

to operative attack, and one would be much chagrined to have an extradural hæmorrhage of corresponding size brought to light post mortem without an attempt having been made to remove it by operation."

The cerebellum is very rarely the site of apoplexy from disease. In 96 consecutive cases of apoplexy in one only was the cerebellum the site of blood effusion.

My brother, Hamilton Ballance ([10]), has published an unique case of *traumatic cerebellar apoplexy* which was cured by operation.

Male, aged 12 years, had a blow in the right parietal region two months before admission, but his illness only dated back two weeks. The symptoms were headache, giddiness, frequent vomiting, and double optic neuritis. There was marked cerebral irritation, he lay curled up on the left side, and resented being spoken to. On further examination it was found that there was weakness and in-co-ordination of the left arm and left leg, with the eyes shut he fell backwards and to the left, there was marked lateral nystagmus on looking to the left, and weakness of the conjugate movements of the eyes to the left. There was no anæsthesia and the reflexes were normal. On

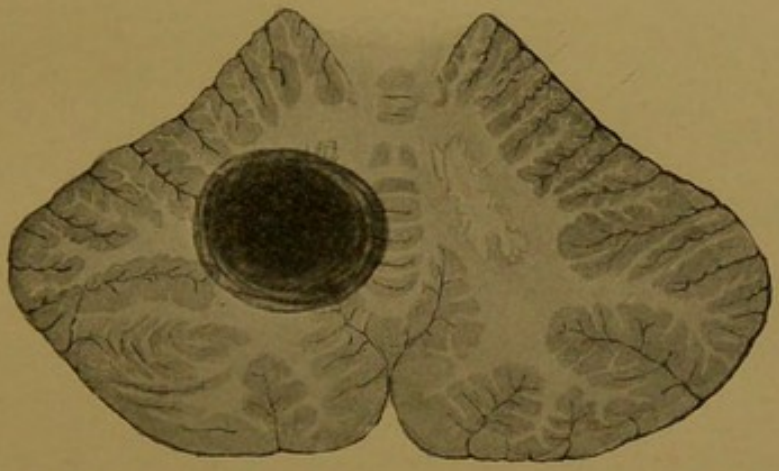


Fig. 8.—Traumatic cerebellar hæmorrhage. (Hamilton Ballance.)
Diagram of site of clot.

walking, which was difficult, he reeled from side to side. On sitting up in bed he had severe frontal headache and the giddiness was pronounced. Objects in the room seemed to move from left to right, and he himself appeared to be going in the same direction, showing that the lesion was intracerebellar.

At the *operation* a large intracerebellar clot was found $1\frac{1}{4}$ inches from the surface of the left cerebellar hemisphere, and removed. Four years later the boy was quite well.

The case is remarkable from the simulation of the signs of intracerebellar tumour by an intra-cerebellar hæmorrhage. Probably, as occurs in

hæmatoceles in other parts of the body, a small fresh hæmorrhage took place six weeks after the primary injury, and determined the onset of urgent and localising symptoms.

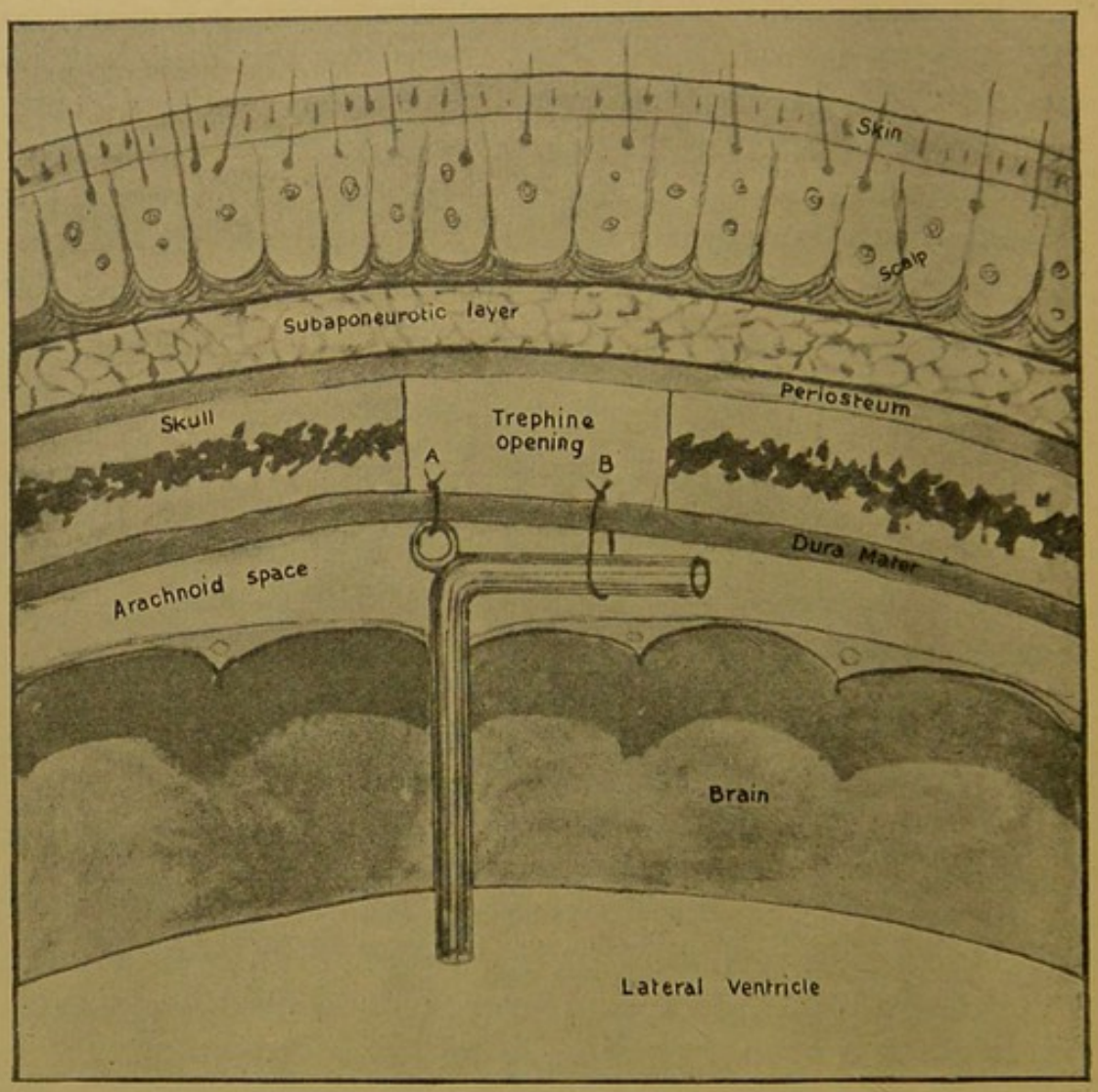


Fig. 9.—Diagram of subdural drainage by an angular metal tube.

The tube is sutured to the dura. The second loose suture prevents the displacement of the tube if the cortex sinks away from the dura. Occasionally the amount of fluid will be in excess of that which can be absorbed by the Pacchionian bodies. The internal hydrocephalus then becomes an external hydrocephalus, and the head may continue to enlarge.

The tube employed is much smaller than that shown in the figure.

2. *Decompression for Hydrocephalus.*—This condition in infants can be relieved by subdural drainage by means of a platinum tube passed into the descending cornu of the lateral ventricle. Lumbar puncture

often fails because the foramina in the roof of the fourth ventricle are blocked by adhesions, which may be due to intra-uterine meningitis. Andrews ([11]) prefers a glass cannula for subdural drainage to a metal one, and Krause ([12]) has had good results from subcutaneous drainage of the lateral ventricle. Decompression of the ventricles by *continuous* drainage is essential, and hence frequent tapping through the anterior fontanelle is of no permanent service.

Drainage of the fourth ventricle necessitates an

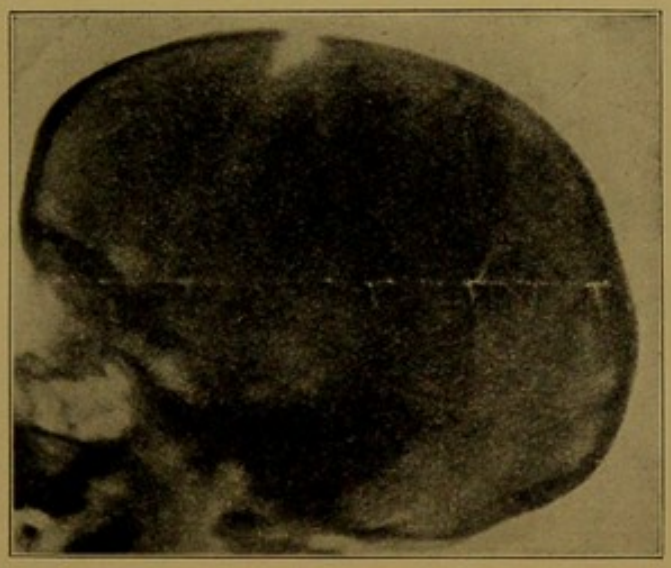


Fig. 10.—X-ray photograph, showing separation of coronal suture in a girl aged 11 years, the subject of acquired hydrocephalus. (Bruce and Cotterill.)

occipital craniectomy extending downwards to the foramen magnum. I have done this operation on several occasions in infants. Last year the late Alexander Bruce ([13]) published a most interesting and instructive case in which this operation was successfully done. A girl, aged 11 years, had an attack of posterior basal meningitis from which she recovered. Soon afterwards she began to show signs of internal hydrocephalus, and later a skiagram showed that the cranial sutures were separating. Finally, an occipital craniectomy was performed, and

a vesicle containing fluid was seen bulging from the back of the fourth ventricle. This was incised, much cerebro-spinal fluid escaped, and the patient made a satisfactory recovery.

An indirect method of decompression is ligation of both common or both internal carotid arteries ([14]). It acts by diminishing the secretion from the choroid plexuses, but as a rule is only to be thought of if

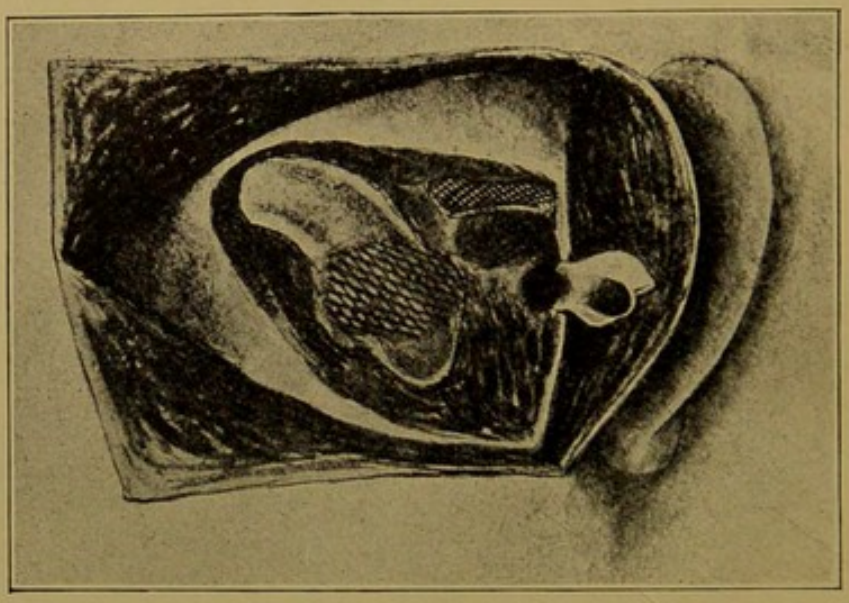


FIG. 11.—Sketch of complete mastoid operation.

In some acute cases the dura when exposed is found of a bright red colour. In the figure the shaded areas over the antrum and attic, and over the sigmoid sinus, indicate the usual sites of inflammation of the dura. (The complete mastoid operation is only very rarely required in acute cases; the figure of the complete mastoid operation is used because it shows clearly the region of the tegmen.) Meningitis serosa may be induced by the inflamed dura, and can be relieved by lumbar puncture.

the head continues to enlarge after subdural drainage has been established. The enlargement of the head after the performance of subdural drainage is caused by the failure of the Pacchionian bodies and cerebral veins to absorb the excess of cerebro-spinal fluid in the subdural space. The internal hydrocephalus becomes an external hydrocephalus, the cerebral cortex collapses, and a deep pond of fluid collects in the subdural space.

3. *Decompression for Meningitis.*—The most useful classification of meningitis is the pathological :— serous, suppurative, and tuberculous.

(*a*) In *meningitis serosa* lumbar puncture, single or repeated, may effect a cure. In cases of acute local cranial bone disease the dura often becomes inflamed and this induces a meningitis serosa. In such cases decompression by lumbar puncture is required in addition to the surgical removal of the causative bone disease ([15]). In the *Lancet* for January 20, 1912, a case of heat apoplexy (meningitis serosa hæmorrhagica) is related. When I saw the patient he was comatose, with Cheyne-Stokes breathing, temperature 104°, and incontinence of fæces and urine. Lumbar puncture on two occasions withdrew blood-stained fluid under pressure and complete recovery ensued.

In localised meningo-cortical œdema of septic origin the symptoms have been known to simulate those of brain abscess ([16]). The appropriate treatment is craniectomy and incision of the œdematous cerebral substance.

The brains of patients dying in the status epilepticus and from some forms of acute delirious mania exhibit at post-mortem examination marked œdema cerebri—such brains, in fact, drip fluid on removal from the skull. Lumbar puncture would be useless, but decompressive craniectomy and incision of the brain substance would appear to offer a hope of recovery.

It is to be noted that a patient with cerebral tumour may pass without warning into the status epilepticus and die in two or three days. I know of one such case in which the presence of tumor cerebri was unsuspected till the autopsy revealed it. The

man had occasionally obtained at St. Thomas's a bromide mixture for headache, but he had worked till thirty hours before death. A decompressive craniectomy in this case would probably have saved life. The tumour, too, though large, of the right frontal region could have been completely removed.

(*b*) In *suppurative meningitis*, certain wonderful recoveries have followed decompressive operations. Hinsberg ([17]), Kummel ([18]), Mygind ([19]) and others have reported such cases. Kummel's patient was a man, aged 33 years, who had suppurative meningitis following fracture of the base of the skull. A bilateral decompressive operation was done low down in the occipital region, sero-purulent fluid escaped. The dura was excised over the whole extent of the apertures in the skull, which were about the size of a five-shilling piece, and large plugs of gauze were inserted as deeply as possible into the posterior fossæ. The patient gradually improved, and in six weeks was discharged well.

Lombard (*Prov. Méd.* 1911, 65) also has recorded a case which shows that when infection has supervened in fracture of the base, life may be saved by operation. A boy of 9 years developed meningitis following head injury, the infection being demonstrated by lumbar puncture. Operation in left temporal region. Much purulent fluid escaped on incising dura. Six weeks later patient had completely recovered.

Mygind had six recoveries out of twenty-six operations for meningitis occurring as a complication of ear disease. He does not admit the diagnosis of meningitis in any case unless turbid fluid is obtained by lumbar puncture. He does not always open the dura, a step which I consider necessary for decompression, but it is noteworthy that of his six re-

coveries the dura was opened in all but one. Mygind observes that meningitis associated with ear disease is most fatal when it commences after operation.

In suppurative meningitis the infection rapidly spreads, in the course of the stream of cerebro-spinal fluid, over the hemispheres and down the spinal theca. It may be that irrigation of the subarachnoid spaces from the lateral ventricle and out through an opening made in the lumbar theca may prove useful in the future in saving some of these desperate cases ([20]).

(*c*) In *tuberculous meningitis* surgery has not yet gained many victories. I ([21]) have reported a case in which the removal of local cranial tuberculous bone disease resulted in complete recovery, though the patient before the operation had paroxysmal headache, vomiting, and squint.

Both in tuberculous and in suppurative meningitis we are no longer justified in regarding the patients as hopelessly lost and in remaining with folded hands, the rather must we attempt to save them by doing the utmost in our power. The future will show, as knowledge increases, better results from surgical intervention.

4. *Decompression for Brain Abscess.*—The successful treatment of cerebral and cerebellar abscess has become current practice. Decompression is as a rule more urgent in cerebellar abscess because of the local pressure on the brain stem, and the danger that hydrocephalus will occur. As I have elsewhere very fully dealt with the treatment of brain abscess, it is not now necessary further to refer to it. Decompression and evacuation of pus from the brain is one of the great triumphs of modern surgery.

The great morbid anatomists of the last century —Auvert, Cruveilhier, Lebert, Bright, Hooper, and

Carswell—all contribute beautiful illustrations of abscess of the brain. How splendid were their labours, and how much we owe to them! On the sure foundation laid by their patient pathological investigations the more perfect clinical diagnosis of the present day has been built up.

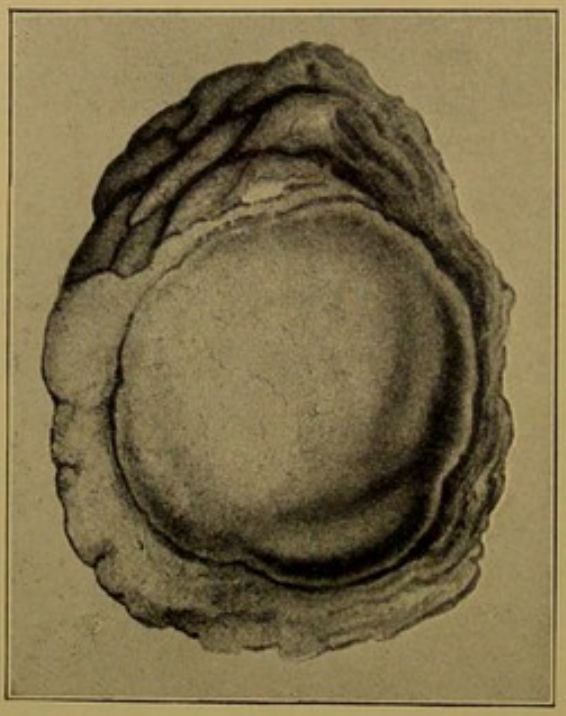


FIG. 12.—Encysted abscess of left frontal lobe. (Hooper, 1826.)

The cyst-wall was as thick as the pericardium. The cyst contained between 2 and 3 oz. of pus. I have known an abscess of the frontal lobe to have so thick a wall that it could be rolled about the floor like a billiard ball.

FIG. 13.—Spreading septic softening of the right frontal lobe. (Hooper, 1826.)

Lebert gives a good illustration of the same condition in the cerebellar hemisphere. I think that this particular result of septic infection occurs more readily and is more dangerous in the cerebellum than in the cerebrum. The brain, just like any other tissue of the body, may be affected by localised or by spreading suppuration.

5. *Decompression for Brain Tumour.*—Von Eiselberg ([22]) has recently published a paper giving the results of 100 operations performed in his clinic for brain tumour. In 10 of these no localisation diagnosis was made, but the decompressive operation gave so much relief that 6 of the 10 left the hospital greatly improved. 13 of the operations were for tumours of the pituitary body. Of the remaining

77 operations 48 were fatal, 32 from the immediate effects of the operation, and 16 from complications occurring later, such as recurrence of growth.

Of the 29 patients who recovered, in 4 the after-result is not known. 12 cases in which a tumour

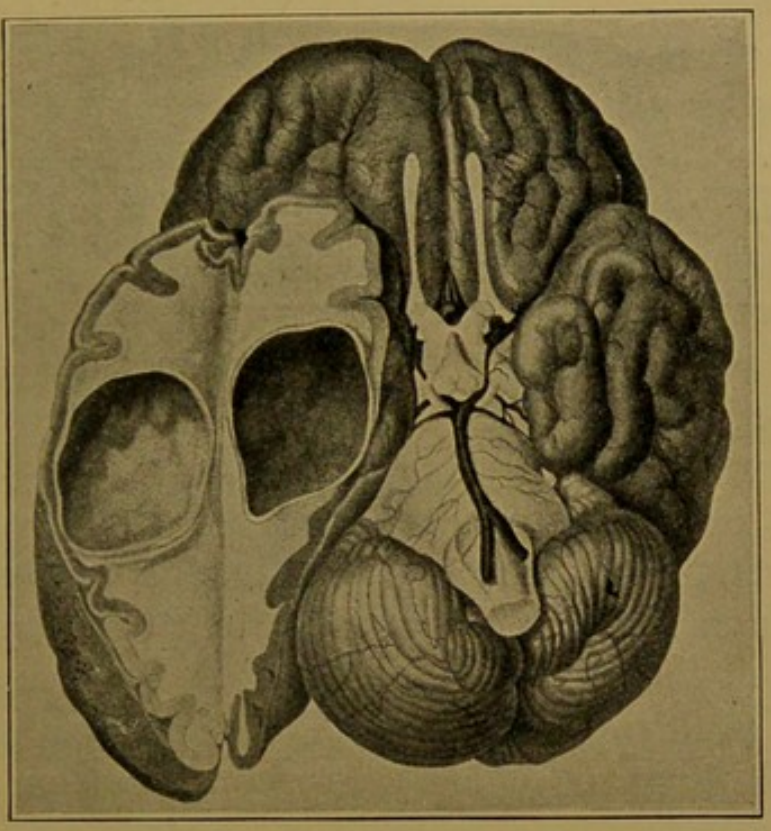


Fig. 14.—Abscess of the right temporo-sphenoidal lobe. (Cruveilhier, 1830.)

Male, aged 32 years. Pain and discharge from right ear for 20 years. *April* 18, 1829. —Taken ill with violent headache and fever. *April* 29.—Seen by Cruveilhier. No affection of sensation, movement, or intelligence. Died suddenly May 11.

Cruveilhier states that the grasp of the two hands the day before death was equal, so there could not have been any gross hemiplegia.

Autopsy.—The ventricles were full of pus, but the encysted abscess had no connection with the ventricles. The last illness was probably meningitis and acute infection of the ependyma of the ventricles—a new infection—from the petrosal disease. The encysted abscess, as we often find, was not the immediate cause of death.

was removed from the cerebrum were known to be alive and well from two months to five years after the operation. Of the other 13 recoveries, 8 were cases in which the tumour was found but was not removed so that simple decompression alone was done (6 of these tumours were in the cerebrum and two in the

cerebellum). In one of the cerebellar cases the patient was free from symptoms eight years after the operation. In 4 of the recoveries a tumour of the auditory nerve had been removed and in one a tumour (cyst) of the cerebellum.

The number of fatalities in this series is depressing,[1] but, as Caesar said of war, "he makes a great mistake who thinks that all his operations will be successful" ([23]). Von Eiselberg has set an admirable example in that all his cases are included in the paper. Such papers have a standard of value far higher than short records of brilliant results.

In Von Eiselberg's opinion "early diagnosis and early operation are the great desiderata in cases of brain tumour. The fate of a patient with brain tumour, condemned as he is to a horrible headache and inevitable blindness, is so terrible that operation should invariably be done, and that before irreparable damage to the eyes has occurred." In a note to the paper Von Eiselberg states that the results he has obtained during the last eighteen months are considerably better than in the time preceding.

A short time ago Cushing ([24]) published a paper in which a series of 35 operations for cerebellar tumour is mentioned. There were only three fatalities, 13 were successful extirpations, 17 palliative decompressions, and 2 were exposures of inoperable growths, the condition remaining practically unimproved despite the decompression.

Decompression relieves the symptoms which depend on increased intracranial pressure. The patient is not, as a rule, *in extremis* from the local effects of the tumour, but from the acute syndrome which supervenes; and is made comfortable and

[1] The same remark applies to the series of cases published in this lecture.

comparatively safe by a decompressive operation. It is the method of choice in all infiltrating tumours. A localised tumour is usually a meningeal tumour and is a fibroma, a fibro-sarcoma, an endothelioma, or a neuro-fibroma. The benign tumours may be present for a long time without giving rise to headache,

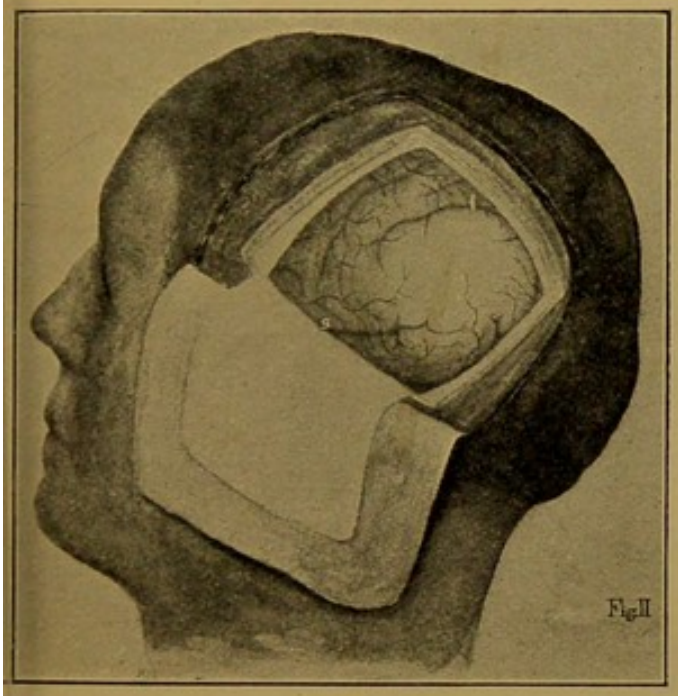


FIG. 15.

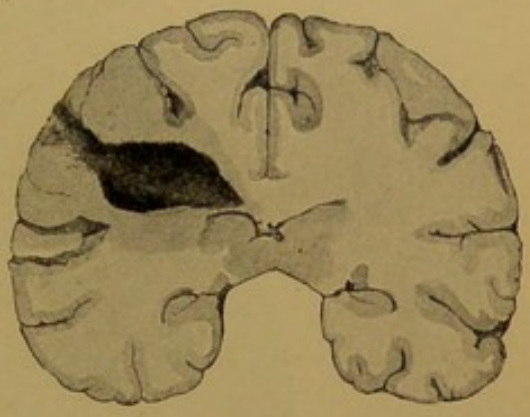


FIG. 16.

FIGS. 15 and 16.—Illustrate decompression and drainage of a malignant cyst of the brain.

FIG. 15.—Second stage of operation. Flap of dura has been turned down. I marks the intra-parietal suscus, S the Sylvian fissure. The cortex of the inferior parietal lobule is thinned, almost translucent towards the centre, and forms the external boundary of the "cyst."

FIG. 16.—Transverse section of brain. The section passes through the centre of the "cyst," and shows the path of drainage from the surface of the cortex. The extent of tumour infiltration is shown by the shading.

Mrs. G., aged 31. Ten months before being seen had a fit. Other symptoms were severe headache, occasional vomiting, optic neuritis, sensory aphasia, alexia, and agraphia, slight right hemiplegia, and hemianæsthesia. There was a remarkable recovery from all symptoms as the result of the operation, but two months afterwards patient died of pneumonia. The tumour proved to be an infiltrating glioma. The plasma filling a malignant cyst is difficult to drain, as it coagulates on cooling.

vomiting, and optic neuritis. The presence of optic neuritis demands immediate operation. We are only too familiar with cases in which a decompressive operation has come too late to save the sight. A

decompressive operation gives the best results when performed over the site of tumour. The operation has a further advantage; it affords an opportunity for exploration ([25]); not in the sense that an exploratory operation allows us to investigate the abdominal cavity; but in a very real sense it does allow an

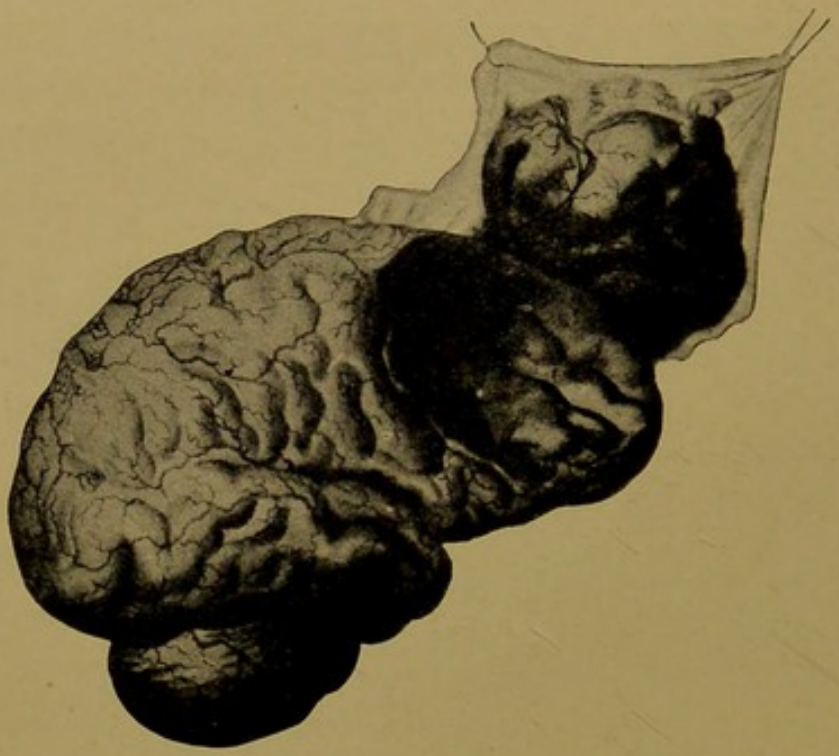


FIG. 17.—Tumour (? endothelioma) of meninges in frontal region. (Cruveilhier, 1830.)

The figure shows the tumour adherent to the inner aspect of the dura, and the depression in the frontal lobe in which it was lodged.

The patient was a woman aged 45 years, a school-mistress, who was seen by Cruveilhier on September 3rd 1829. The symptoms recorded are frontal headache, inability to walk, weakness of left leg, slow speech, mental enfeeblement, and involuntary micturition. She died on October 3rd 1829. Cruveilhier had diagnosed a tumour of the right frontal lobe, and had the satisfaction of demonstrating to his class the tumour in the situation in which he had predicted that it would be found. Nearly seventy years after Cruveilhier my friend, the late Charles Beevor, pointed out that involuntary micturition was an early sign of frontal lobe tumour.

exploration which may lead at the time or later to the removal of the disease.

Are there any symptoms which definitely indicate the position or even the existence of an intracranial tumour? No one symptom alone will give us this information, but the association of certain symptoms

does afford us, if not the certainty, at least a strong probability of the existence of this particular lesion. The time and manner of evolution of symptoms are of great diagnostic importance, as also the absence of certain symptoms. The clinical evolution of cerebral tumour varies greatly. Localising symptoms occur-

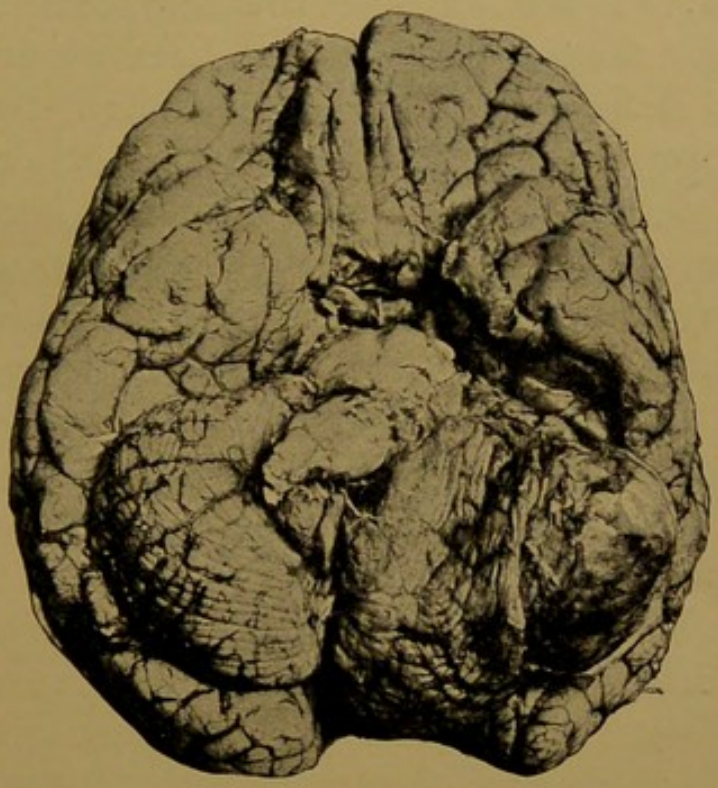


FIG. 18.—Fibro-sarcoma of left cerebellar hemisphere. (Chance and Spiller.)

Male, aged 26. Severe occipital headache, Easter 1904. In July vomiting occurred. In November paralysis of left external rectus, dimness of vision, and photophobia. In December slight left facial palsy and double optic neuritis. Patient gradually deteriorated, and developed a tendency to fall towards the left side. No operation was performed. He died suddenly on April 7, 1905.

Remarks.—The tumour was situated upon the outer portion of the left lobe of the cerebellum, to which it was only loosely attached. It was very favourably situated from the point of view of operation. It measured 4 × 5.5 × 5 cm., was hard in texture, and had deeply indented the cerebellar hemisphere. The third and lateral ventricles were somewhat dilated.

ring late have a less definite localising value than the same symptoms occurring early.

The following are a few recent examples of brain tumour cases from my own practice. Several have been fatal, but I agree with Cushing, who writes: "Surgical knowledge of value is built up more on the

mistakes than on the successes of past experience." In performing a decompressive craniectomy an osteoplastic flap is a mistake. A cranial defect is essential ([26]) and a cerebral protrusion is desirable. It is advisable to select a "silent" area of the brain over which to make the decompression when local diagnosis is impossible. I do not like the temporal method of Cushing for deep-seated tumour, because of the large one-sided deformity that arises when the patient survives for a long time.[1] On the other hand an occipital decompression may produce hemianopsia. Decompression in the cerebellar region usually gives a free escape of cerebro-spinal fluid, which does not always happen when the decompression is made over the cerebral hemispheres. Free escape of cerebro-spinal fluid rapidly relieves the congestion of the optic discs. In tumours beneath the tentorium lumbar puncture is dangerous if done to relieve tension before the dura is opened, for the brain stem may be displaced and sudden death caused ([27]). I have published such a fatality. Krause ([28]) relates a case in which during an operation for tumor cerebelli dextri the respiration ceased, but was re-established by puncture of the descending cornu of the lateral ventricle. Much cerebro-spinal fluid escaped, and the operation was satisfactorily completed.

The salient facts of the following cases alone are mentioned, all others have been omitted for the sake of brevity and precision.

Case 1. Tumour of pons. Decompression impossible.
„ 2. Sarcoma of cerebellum. Fourth ventricle and third ventricle filled with growth. Operation. Death.
„ 3. Large right precentral meningeal tumour. Operation. Hæmorrhage. Death.

[1] See record of a case in the Addendum.

Case 4. Tumour of cerebellum. Dura not opened; no decompression. Arrest of respiration. Death.
„ 5. Cerebellar tumour. Decompression. Tumour irremovable. Death.
„ 6. Five tuberculous tumours in the cerebellum. Decompression.
„ 7. Sarcoma of meninges. Decompression.
„ 8. Glioma of frontal lobe. Failure of localisation diagnosis. Decompression.
„ 9. Meningeal tumour simulating tumour of the pineal body. Decompression.
„ 10. Glioma of cerebellum. Late decompression. Loss of sight.
„ 11. Glioma of cerebellum. Decompression. Recovery.
„ 12. Cerebellar tumour diagnosed but not found. Decompression. Recovery.

Case 1.—*Tumour of pons. Mistaken at first for diphtheritic palsy. Rapid increase of symptoms. Death.*

A doctor, aged 44 years, seen by Sir David Ferrier in North Wales late in November 1910. History of tubercle in family. Pneumonia eighteen months ago. Von Pirquet's test negative. Never syphilis. Was sent to sea in early life in order to get strong; made several voyages round Cape Horn. Has been in practice sixteen years and remained at work until November 11, 1910.

During the last week in October he was attending some cases of diphtheria, on November 1 complained of difficulty in swallowing, and food to some extent regurgitated through the nose, his gait was somewhat unsteady especially in the dark. When seen by Sir D. Ferrier with Dr. Elliott of Chester and Dr. Herbert he had persistent occipital headache, and had remained in bed for some days because of ataxic walk. The knee-jerks were exaggerated, especially the left. Romberg's sign was present, there was slight inco-ordination of the left arm. He had been vomiting at intervals during the preceding four weeks. His speech had become thick since November 1, and he was somewhat drowsy.

On December 27 he came to London in an invalid carriage, and I saw him with Dr. Purves Stewart as Sir David Ferrier was in Cornwall. He had been drowsy for three weeks but understood everything that was said to him. Cerebral vomiting had occurred daily, the urine had been retained for forty-eight hours. Two weeks ago and also a few days ago he had had opisthotonus. There was no optic neuritis, the pupils acted well. There was divergent strabismus, the inward and outward movements of both eyes were impaired, but there was no failure of upward or downward movement or of the movements of the pupil as

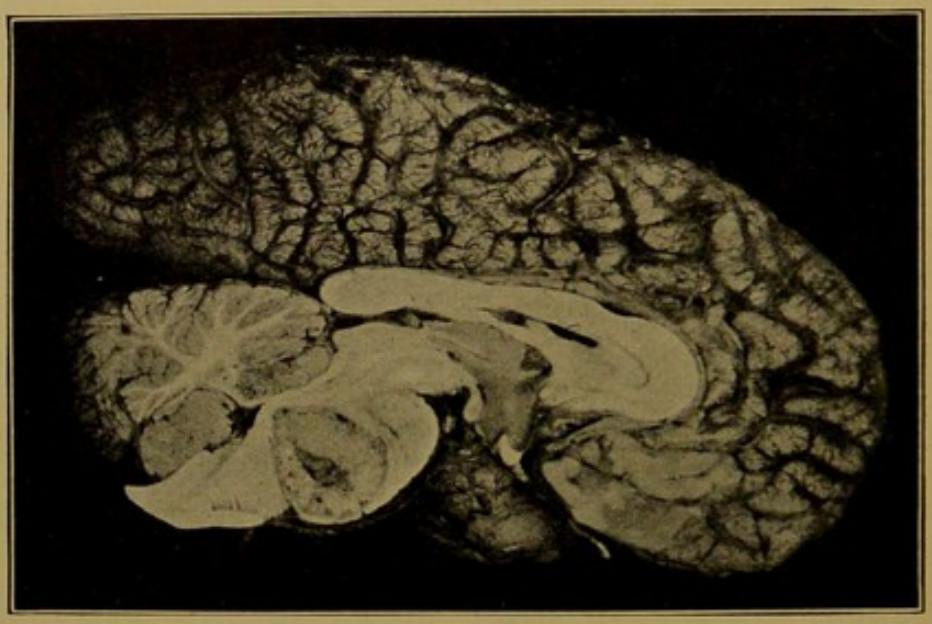


Fig. 19.—Sarcoma of pons varolii.

in nuclear paresis of the 3rd nerve. Hearing slightly impaired on left side, slight peripheral paresis of left face, slight weakness of left arm and leg. Left Babinski reflex and left ankle-clonus, no anæsthesia. Sir David Ferrier wrote from Cornwall: "The tumour affects the medulla oblongata, I think it is in the region of the fourth ventricle. He has signs of conjugate paralysis of eyes (lateral). This occurs in tumours of the fourth ventricle, and with dysphagia is very significant. Make the decompression very low posteriorly so as to expose this region."

Fluid under pressure withdrawn same evening by lumbar puncture. Next day patient gradually became comatose,

and too ill for operation. He had much hiccough all day and died at 10.30 P.M.

Autopsy.—Tumour in substance of the pons, which on histological examination was found to be a sarcoma with large round cells. The central portion of the tumour was fluid.

Remarks.—The condition here described is obviously an "inaidable estate," and for such a case decompression is useless. Even if the pons had been exposed and the tumour tapped, no relief could have followed.

CASE 2.—*Sarcoma of cerebellum. Fourth ventricle and third ventricle filled with growth. Hydrocephalus internus. Operation. Death.*

Male, aged 15 years. Sent to St. Thomas's by Dr. Green of Romford on May 1st, 1911. On September 6th 1910 fell while cycling and struck his forehead, inflicting a small wound. After this he was not well for some days but no doctor was called in. Three months later it was noticed that he began to lose interest and was less able to attend to his lessons. About this time he began to vomit on rising in the morning and he has continued to vomit at intervals since. A little later he complained of right occipital headache. In February 1911 the gait became unsteady. His mother said that he rolled down the street like a drunken man.

On admission. — Patient bright and intelligent but complains of severe headache, vomiting, and very unsteady gait. Pupils somewhat large and react slowly. Horizontal nystagmus on conjugate movement in both directions, slight rotatory nystagmus on convergence and on upward movement. No obvious ocular paralysis. Both discs swollen to 5 D. Veins large, arteries small, no hæmorrhages. Gait very unsteady, can scarcely walk alone. Stands with the feet wide apart. Cannot stand on either leg alone. When eyes shut falls backwards. In walking lurches equally to right and left. Slight incordination of limbs on both sides. Reflexes not abnormal. Right cerebellar region tender on pressure. *Operation.*—May 9th, 1911. Bone removed over right cerebellar hemisphere and over part of left.

Dura very thin and tense. The patient became severely collapsed and in spite of stimulation, infusion, etc., died the same evening.

Autopsy.—Convolutions flattened, cortical veins engorged. The tumour involved the vermis and the adjoining parts of the lateral lobes of the cerebellum. It filled the fourth ventricle, the Sylvian aqueduct (which was dilated), and the third ventricle. The anterior inferior cerebellar arteries crossed on the ventral aspect of the sixth nerves and both nerves showed grooving, but no degeneration was discovered

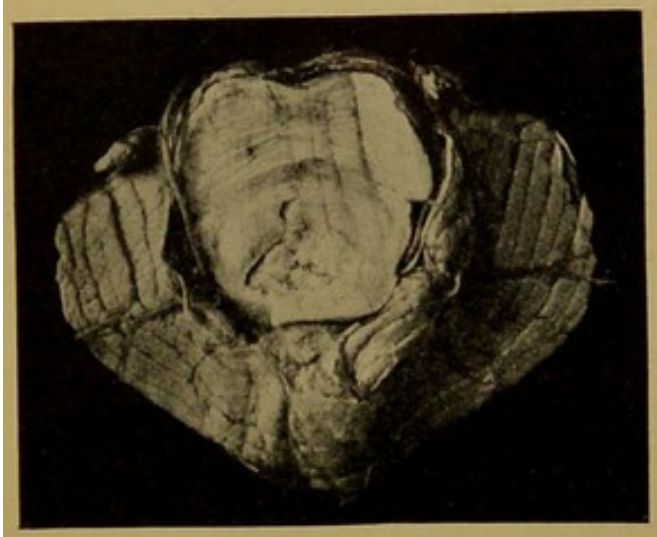


FIG. 20.

FIG. 21.

FIGS. 20 to 24.—Illustrations of Case 2.

FIG. 20.—Section of pons viewed from front. Sylvian aqueduct dilated and filled with tumour.

FIG. 21.—Section viewed from behind. Tumour expanding and filling 4th ventricle.

on microscopical examination after staining by Marchi's method. Histological examination showed that the growth was a sarcoma.

Remarks.—The autopsy showed that the growth was an infiltrating tumour, already widely disseminated by local extension, and irremediable by operation. When the case came under my observation even a simple decompressive operation would have been of no service, but we could not be sure of this before operation. Life and death were so nearly balanced that when decompression was cast into the scale the beam turned to the inevitable result—death.

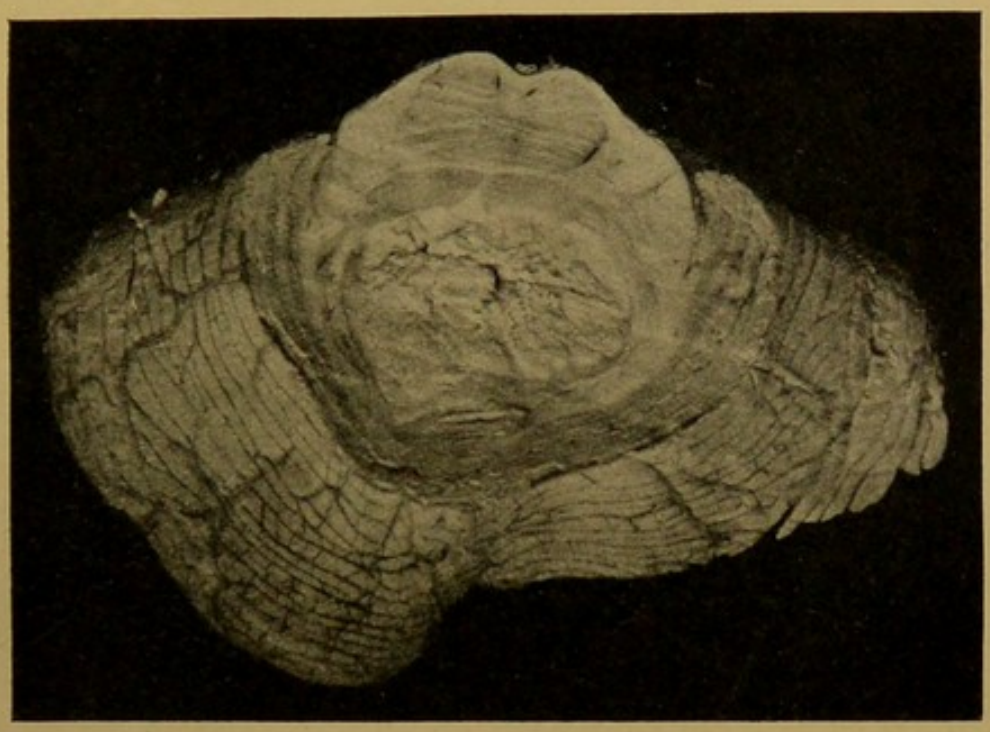


FIG. 22.—View of section seen in Fig. 21, from in front.

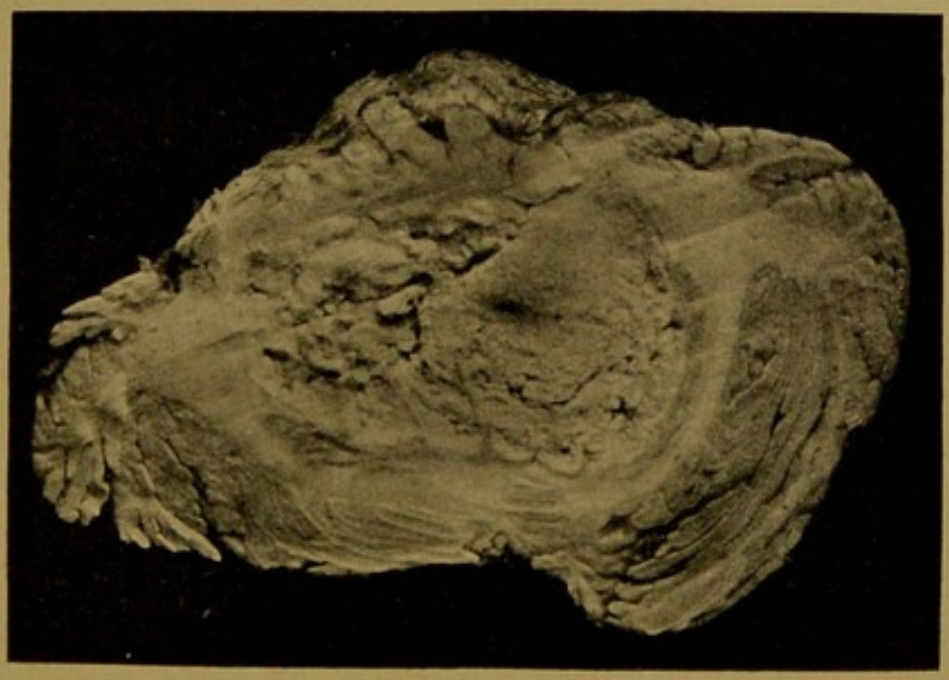


FIG. 23.—Section made more posteriorly and viewed from behind. Tumour destroying vermis, inner portions of lateral lobes of cerebellum, and filling 4th ventricle.

Case 3.—*Large right precentral meningeal tumour. Operation. Hæmorrhage. Death.*

Female, aged 44 years, a patient of Dr. Graham Stewart of Margate. Seen with Dr. Purves Stewart.

History.—Three years ago slight weakness of left foot. Two and a half years ago pain in left foot followed by unconsciousness lasting a few minutes. Ever since has

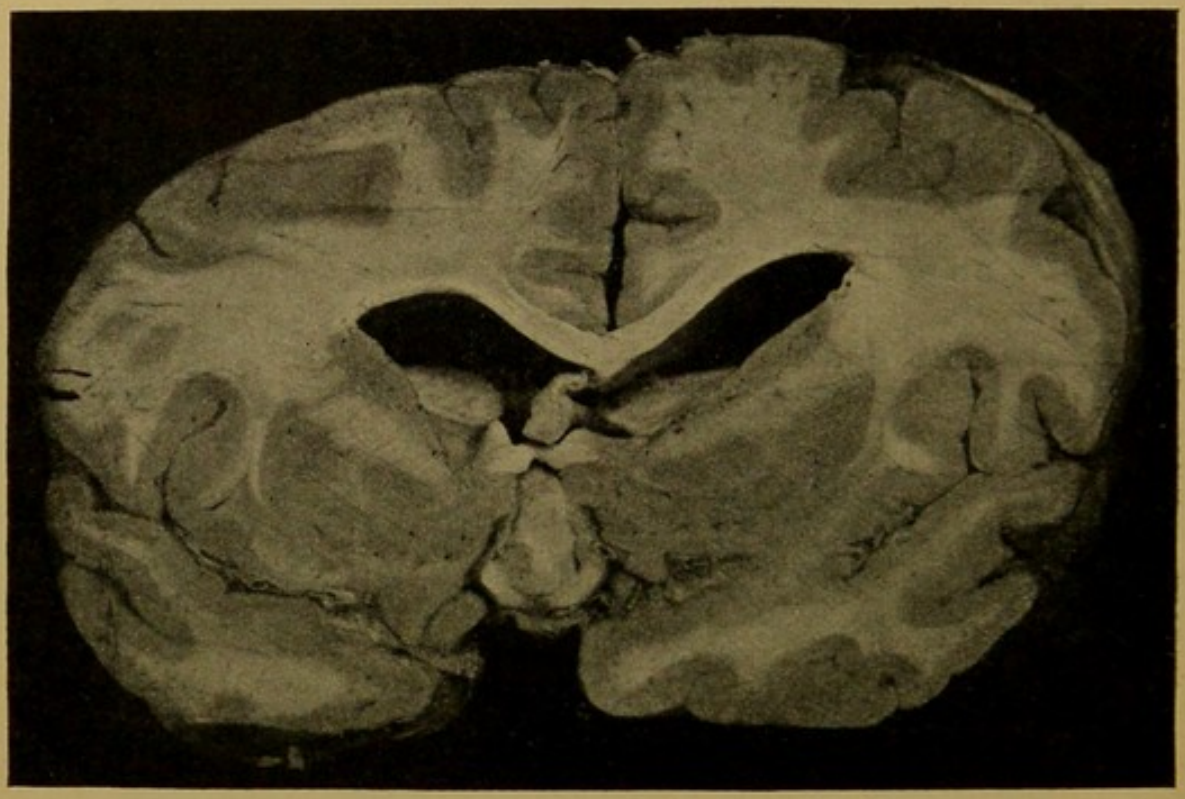


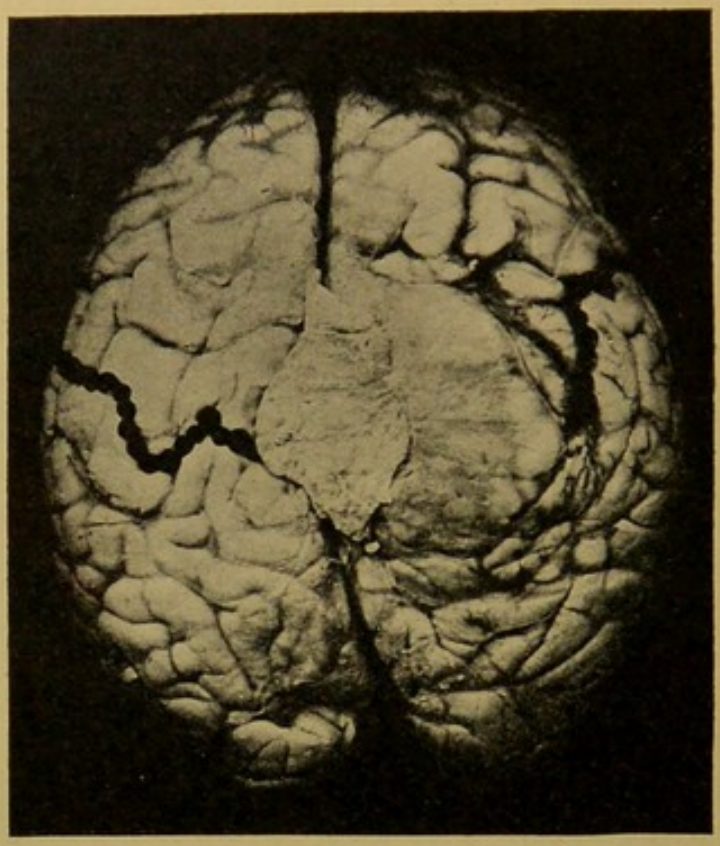
Fig. 24.—Coronal section of brain showing tumour filling 3rd ventricle. Lateral ventricles dilated. (Case 2.)

dragged the left foot and has had numerous fits, commencing in the left foot. The pain preceding the fits was severe, and often extended as high as the knee. The left leg has gradually become weaker, and the left upper limb has also become weak, the patient has had occasional right-sided headache and some dimness of vision. There has been no vomiting. Left Babinski reflex and bi-lateral optic neuritis were observed four months ago; about this time, too, the right foot became weak and the "attacks" seemed to commence in it. *When seen*, January 30, 1912, patient

pale, anæmic, and stout. Speech and articulation normal. Pupils normal. Bi-lateral optic neuritis with commencing atrophy on the left side. No facial weakness. Other cranial nerves normal. No affection of sensation. The left upper limb was weak at all its joints but none of its movements was impossible. The muscles of the *proximal* segment were the most profoundly affected. There was also decided paresis of the left lower limb, but here the muscles of the *distal* segment were the most affected. She could not extend the toes, nor dorsiflex the ankle, nor lift the limb from the bed. She could not walk alone, and when supported dragged the left foot. Reflexes increased, more so on the left than on the right side. Ankleclonus and Babinski reflex on left side. Flaccid paresis of left limbs, right lower limb spastic. Definite tenderness on firm pressure on right side of head, near the median line and just in front of the fissure of Rolando.

Operation February 1, 1912.—Large craniectomy over right precentral area. On exposure of the cranium blood oozed from innumerable openings in the bone. The area chiefly affected by this capillary hæmorrhage was of a bluish colour and about two inches square. The bleeding was uncontrolled by wax, gauze, hot lotions, and muscle grafts. The bone-flap was therefore removed as quickly as possible, but the dura was firmly adherent to it, and during the separation of the vascular dura from the bone-flap more blood was lost. Every means was used to revive the patient, but she died a few hours later.

Remarks.—The photographs show the position of the tumour. It was a large meningeal growth occupying the upper part of the right precentral region, and was lodged in a cup-shaped depression in the cerebral cortex. The transverse section shows that it had at one spot destroyed the falx, had crossed the middle line, and was already affecting, by pressure, the precentral gyrus on the left side. Microscopically the tumour was an endothelioma. The localisation diagnosis was easy. The fits and the localised tenderness pointed to a cortical lesion. The march of the paresis—most intense *distally* in the left lower limb, and



FIGS. 25 and 26.—Right Precentral Meningeal Tumour (Endothelioma). (Case 3.)

FIG. 25.—Shows the brain from above with some dura still adhering to it. The tumour had crossed to the left side, and was pressing on the upper part of the left precentral convolution.

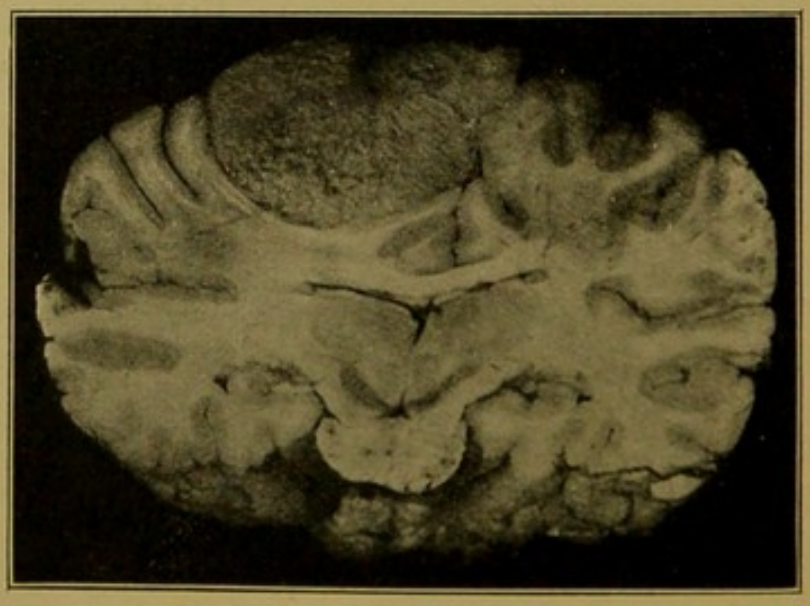


FIG. 26.—Coronal section of brain and tumour. The cerebral substance is not invaded.

proximally in the left upper limb—corresponded to the representation of motion in the cortex of the right pre-central convolution. The more recent affection of the right foot showed that the tumour had crossed the middle line. The long history was incompatible with an infiltrating growth, and made the prospects of operation seem hopeful. Fits followed by permanent paralysis indicate an organic lesion, and therefore, in such cases, it is not desirable to delay operation until the onset of the syndrome. In this

Fig. 27.—Endothelioma and perithelioma of the dura mater. (Case 3.)
(Microscopical section by Dr. Braxton Hicks.)

particular instance of chronic cerebral tumour headache was not a prominent symptom, and vomiting never occurred.

I have had three previous experiences of uncontrollable capillary hæmorrhage from the cranium in cases of brain tumour. (*Royal Med. Chiur. Trans.* lxxix., 1896, p. 195; *Trans. Med. Soc. of London*, vol. xviii., 1895, the final result in this second case is noted in *Some Points in the Surgery of the Brain, etc.*, p. 359; and the third case is related and figured in the same work, pp. 331-36.) In the first case

capillary hæmorrhage from the bone occurred at the final operation, three years after the first operation : the tumour was an angeio-sarcoma. In the second case the hæmorrhage occurred at the final operation, performed six years after the first : the tumour was a sarcoma with both round and spindle-shaped cells. In the third case the hæmorrhage occurred at the primary operation : the tumour was a hæmangeio-endothelioma. The capillary hæmorrhage from the bone was, in each of these three cases, due to tumour-infection of the cranium. I believe that the hæmorrhage in the case I have just related was due to the same cause. The surgeon must be prepared to meet with this condition in tumours which are superficially placed and have long been in contact with the dura. Whether, if on finding the hæmorrhage uncontrollable I had closed the scalp flap, the patient would have survived so that a second operation could have been attempted later, I do not know. In such cases the surgeon's efforts suffer defeat from an "inaidable estate" which cannot be foreseen.

CASE 4.—*Tumour of Cerebellum. First stage of operation. No decompression. Arrest of respiration twenty-four hours later. Death.*

Boy, aged 8 years, seen January 7, 1911. Patient of Dr. Thomson of Leamington and Sir David Ferrier. He had been ailing and losing flesh for about nine months, in October he had severe headache, especially when laughing, and sickness before breakfast. For a month he had had "bilious attacks" for which Dr. Thomson was called in. Twelve days before I saw him he had acute headache and vomiting, and the external rectus on the right side became paretic. When seen, bi-lateral optic neuritis with hæmorrhages. No choroidal tubercle. Pupils normal, frontal and occipital headache. Temperature subnormal, pulse 60. Von Pirquet test positive, Wasserman negative, with Romberg's test fell promptly backwards and to the left. Stands with feet apart, gait zig-zag with lurching to the left. No loss of power of limbs or anæsthesia. Reflexes normal. Enlarged glands in groins and in abdomen.

Diagnosis.—Tuberculous tumour of left cerebellar hemisphere. January 8, dura of left cerebellar fossa and part of right exposed. Much bleeding from foramina in the bone. Dura very tense.

Result.—Twenty-four hours later arrest of respiration and death. No autopsy allowed.

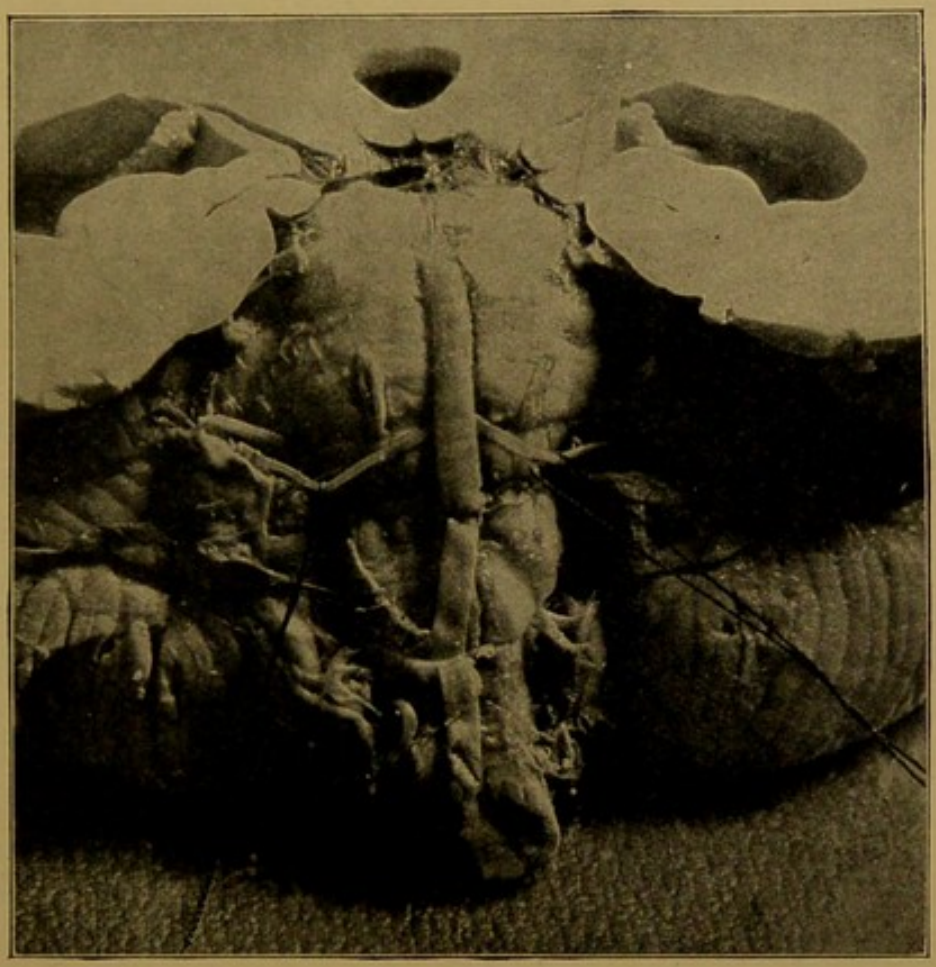


FIG. 28.—Grooving of both 6th nerves by symmetrically placed and superficially disposed anterior inferior cerebellar arteries. (Cushing.)

Patient, aged 9 years, had double abducens palsy and typical cerebellar symptoms with hydrocephalus which had caused diastasis of sutures. *Operation.*—Large median encapsulated, cerebellar tumour. Death.

Remarks.—I have come to the conclusion that in all cerebellar cases the dura should be opened in the first stage of the operation. It is important to note that external rectus palsy is a localising sign, since it has been shown that when the brain stem is displaced downwards the 6th nerve is liable to be constricted and grooved by the arteria auditiva interna or the arteria cerebelli inferior anterior, and further

that a tumour limited to one side of the brain may in this manner cause abducens palsy of both sides ([29]).

CASE 5. — *Cerebellar tumour. Decompression and partial removal. Death.*

Female, aged 27 years. *Seen* September 30, 1910, with Mr. Forbes Fraser of Bath and Dr. Risien Russell. Eighteen months previously she was abroad and could then dance well, but it was noticed that she had slight facial paresis on the right side. During the winter of 1909-10 she had headache, occasionally rather severe. During June, July, and August 1910 she had headache and morning sickness daily. Facial palsy by the time I saw her had become complete ; there had been frequent twitchings of the orbicularis palpebrarum for some months before this occurred. When seen she had pain in right cerebellar region and in right eye and in second division of the right 5th nerve. Complete deafness of right ear but no tinnitus. Lateral nystagmus with wide excursions on looking to the right and with small rapid excursions on looking to the left. Severe bi-lateral optic neuritis. Masseters equal. All reflexes normal. No inco-ordination. Swayed slightly to the left in walking. No Babinski reflex, but left great toe remained still.

October 3rd, 1910.—Bone removed over right lobe of cerebellum and part of left. Dura very tense. The right side of the face twitched for two hours after the operation, perhaps from movement of the tumour. For the next three days she was rather sleepy, respiration was deep and sometimes noisy. The right side of the face, previously paralysed, moved a little.

October 13th.—Has had severe pain in the head for the last two days, with very irregular pulse, and some respiratory difficulty. *Operation.*—The dura, which was under great tension, was opened, and the occipital sinus tied. A layer of tumour tissue of a pinkish colour was found outside the cerebellum, extending from the internal auditory meatus to the foramen magnum and probably further. A good deal

of it was cut away ; while this was being done, spasm of the muscles supplied by the spinal accessory nerve occurred, and the pulse varied from 100 to 200. The cerebellum was incised horizontally ; it contained a mass of similar new growth. Patient died twenty-four hours later.

Remarks.—The tumour was a very vascular glioma. It had probably originated deeply in the cerebellum, burst through the cortex, and spread over the surface. It could not have been removed by operation. It very likely invaded the fourth ventricle and there may have been hydrocephalus.

CASE 6.—*Five tuberculous tumours in cerebellum. Decompression. Three operations. Death.*

A boy, aged 7 years, was sent to me in March 1910 by Dr. Morgan of Seaford. For eight weeks he had had headache, vomiting, and staggering gait.

On admission.—The head was inclined to the right shoulder ; all the reflexes were normal, though slightly exaggerated. Paroxysmal pain in the head which caused him to cry out. Occasional vomiting. Optic neuritis with swelling of 5 D. on both sides. Romberg's sign, fell backwards or to the right. In walking he swayed from side to side. A cast of the occipital region showed enlargement on the right side.

March 20th. First operation.—Bone over cerebellar region exposed by Cushing's incision and removed on both sides. A few days later a rather superficial tuberculous tumour was removed from the right cerebellar hemisphere. The condition of the patient was too bad for further exploration. Free drainage of cerebro-spinal fluid occurred and great improvement resulted, the optic neuritis cleared up and the headache and vomiting ceased. Towards the end of April he was not so well, and headache and vomiting recurred. *May 12th. Second operation.*—Three tuberculous tumours removed from the left cerebellar hemisphere. Much improvement again followed, but toward the end of June, fever, vomiting, nystagmus, and impaired mental state made further exploration desirable. *June 30th. Third*

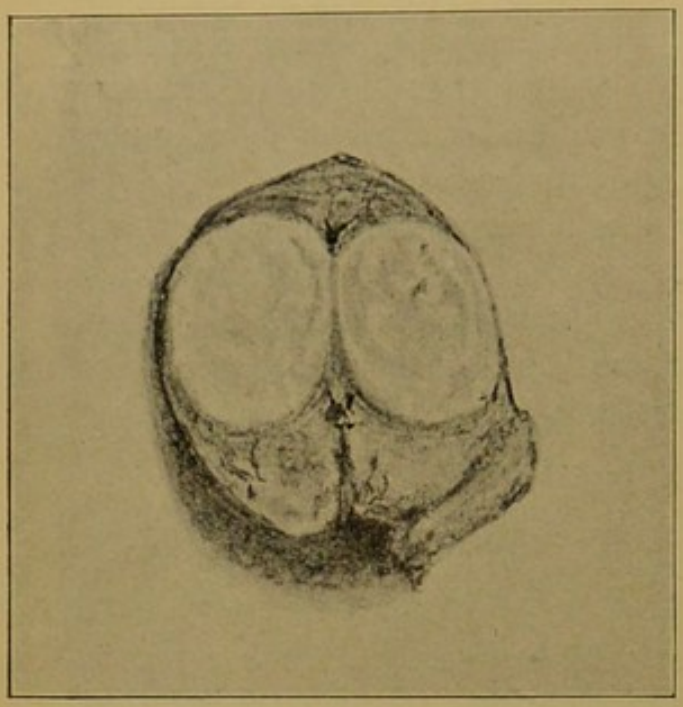


Fig. 29.

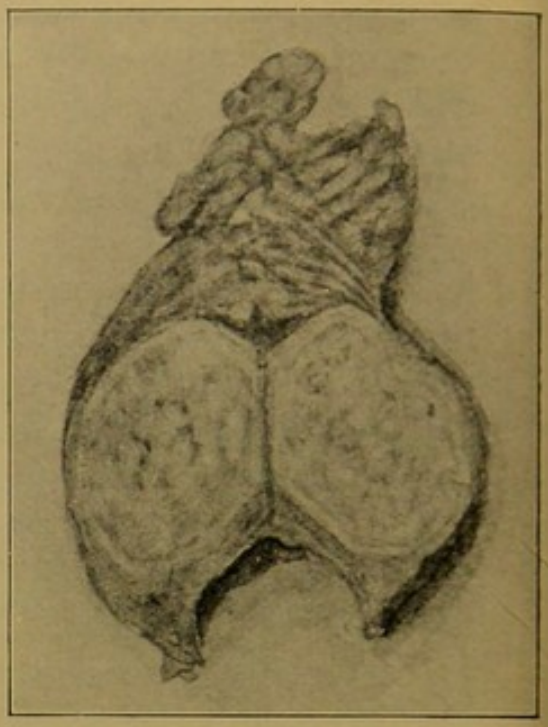


Fig. 30.

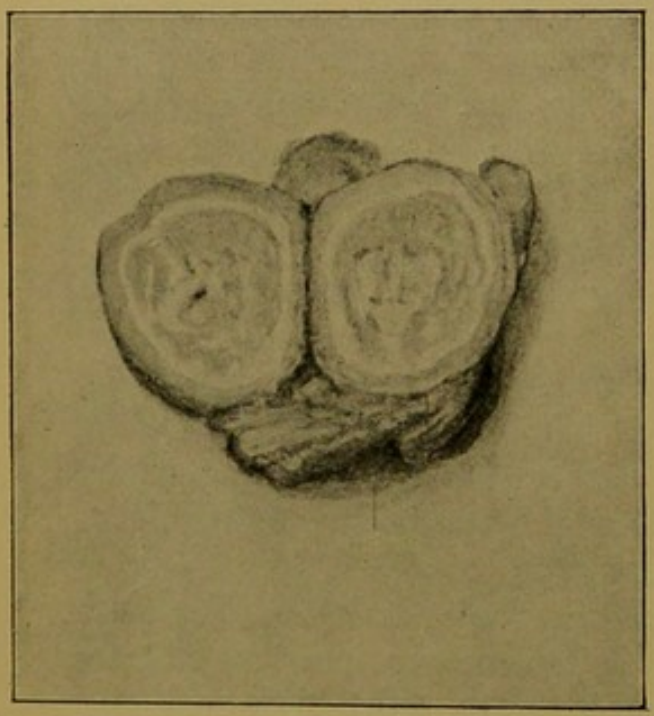


Fig. 31.

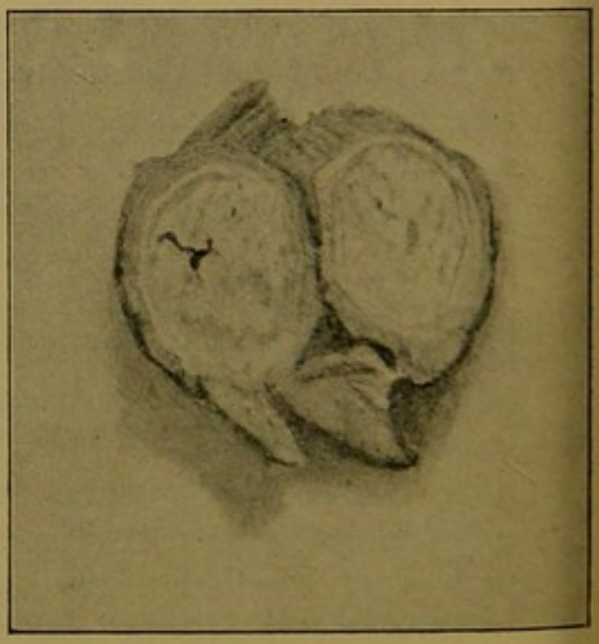


Fig. 32.

Figs. 29-33.—Five tuberculous tumours removed from the cerebellum. Each tumour is natural size and has been bisected and opened out. (Case 6.)

Fig. 29.—Tumour removed on March 20, 1910.
Figs. 30, 31, 32.—Tumours removed on May 11, 1910.
Fig. 33.—Tumour removed on July 2, 1910.

The American Society of Clinical Surgery was present at the third operation.

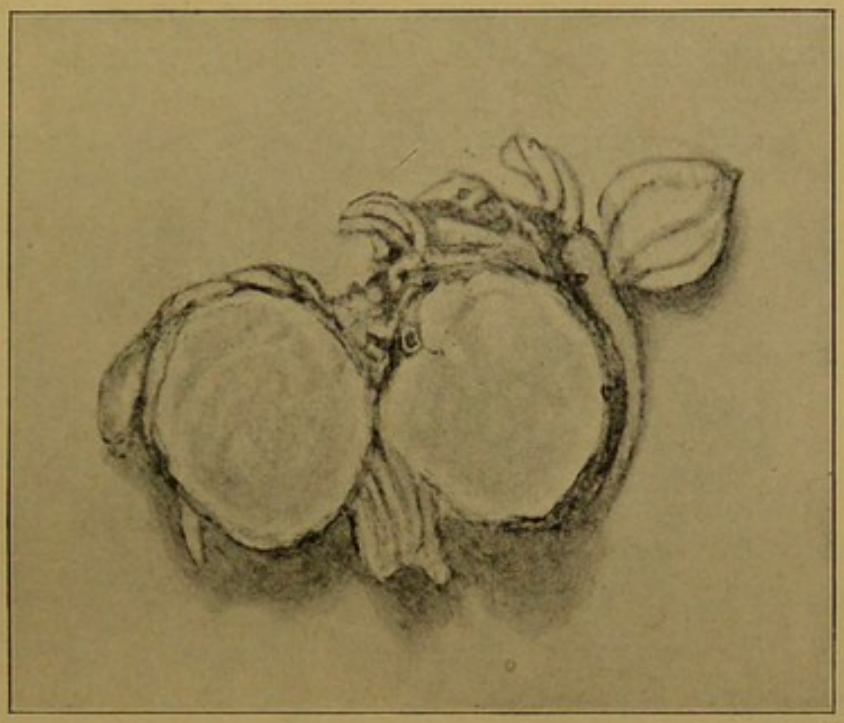


FIG. 33.

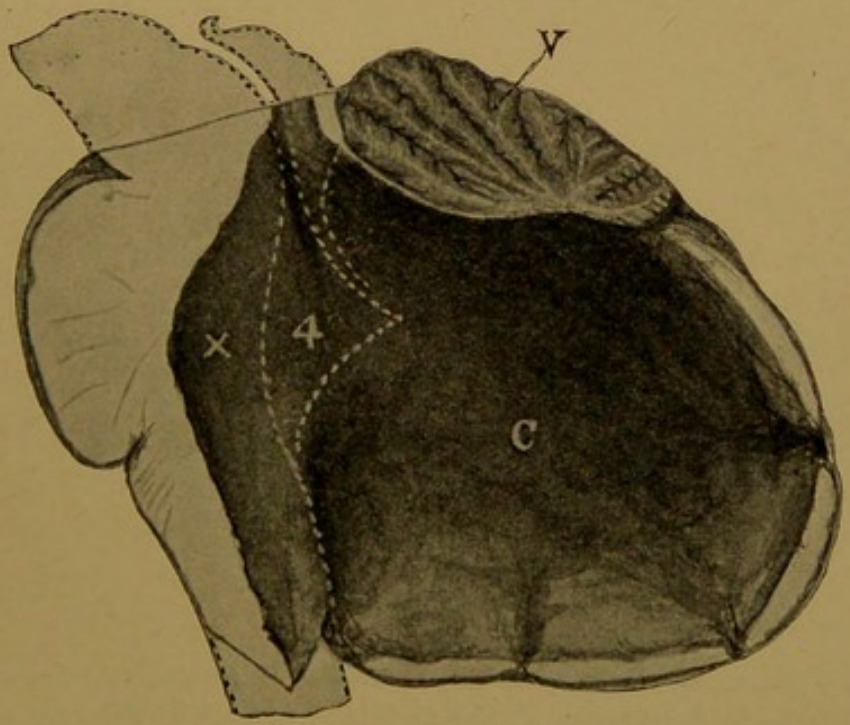


FIG. 34.—Sagittal section through brain-stem and remains of cerebellum. The dotted lines indicate the size and position of the 4th ventricle, Sylvian aqueduct, and brain-stem under normal conditions. (Case 6.)

C, Cerebellum converted into a huge cavity after the removal of five tuberculous tumours. The Sylvian aqueduct is dilated. V, Remains of anterior part of vermis. 4, Outline of normal 4th ventricle. X, This space shows the amount of pressure flattening of the brain-stem.

operation.—A medially-placed tumour pressing on the medulla found and removed. The child, who by this time

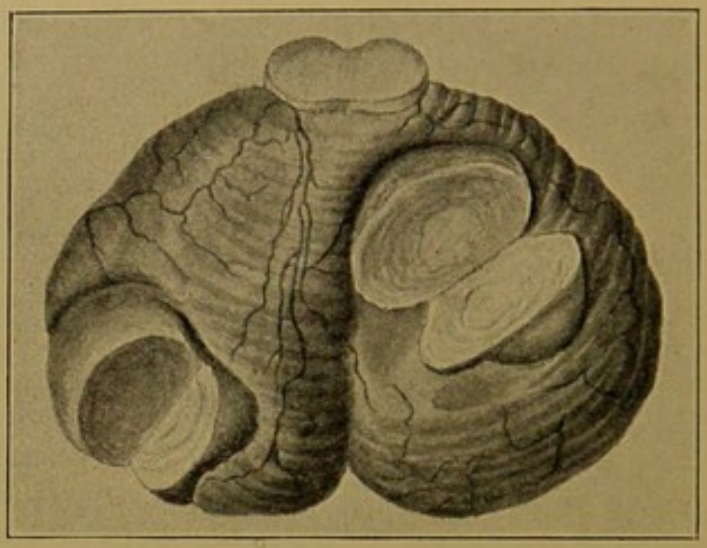


Fig. 35.—Upper surface of cerebellum, showing a solitary tubercle n each hemisphere. (Cruveilhier.)

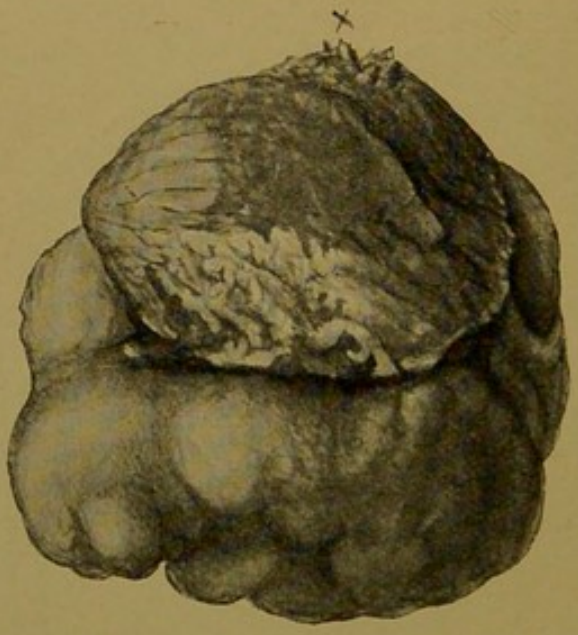


Fig. 36.—Illustration of solitary tubercle successfully removed in 1905 from the left cerebellar fossa. X, Site of attachment to tentorium.

The patient was 4 years and 10 months old. The tumour measured 1.82 × 1.73 × 2.2 inches, and weighed 1¾ oz.

had become greatly emaciated, gradually got worse and died in August.

The tuberculous tumours removed from the cerebellum

were all about the same size. They measured 1 in. × ¾ in. × ¾ in.

Autopsy.—Cerebellum destroyed. Hydrocephalus of the ventricles, and lungs studded with tubercles.

Remarks.—Cruveilhier has in his atlas a beautiful picture of a single tuberculous tumour in each hemisphere of the cerebellum taken from a child aged 9 years (Fig. 35). The failure of operation for tuberculous tumour of the brain may be due to presence of other tuberculous tumours unsuspected during life. This is certainly true of this disease in childhood. I removed ([30]) a large solitary tuberculous tumour from the left cerebellar hemisphere of a child of nearly 5 years of age in June 1905 with complete success, and the patient has been well and strong since (Fig. 36).

CASE 7.—*Round-celled Sarcoma limited to the meninges and ependyma. Decompression. Temporary improvement. Death.*

Male, aged 7 years, very bright and intelligent. Seen on December 10, 1910. Patient of Dr. Deane of Lincoln, seen also by Dr. Risien Russell. Two years previously he was knocked down by a horse and stunned. At Christmas 1909 he had measles. Since then head has been inclined to the right shoulder. For several months he has had pain in the head and vomiting. When seen, zig-zag walk with deviation to right, abduction of right thigh in standing, slight inco-ordination of limbs on right side, cannot stand so securely on right leg as on left. Right occipital region bulging and tender. Lateral nystagmus, with wide excursions in both directions. Bi-lateral optic neuritis with swelling of 7 D. *Operation* in two stages (December 16 and 20). Right cerebellar hemisphere and part of left exposed. On opening the dura several white spots of hard growth about the size of a split pea were found, fusing together the pia and the arachnoid. Some of these were removed, and on histological examination proved to be round-celled sarcoma. X-ray treatment through the scalp was tried, nine treatments being given.

Result.—Convalescence satisfactory. Headache, vomiting, and neuritis disappeared. Child went home, apparently well, towards the end of January 1911. In March I heard the child was dying, and went to see him near Lincoln. He was comatose, and the cerebellar flap was greatly bulging. There had been headache and vomiting. A fine

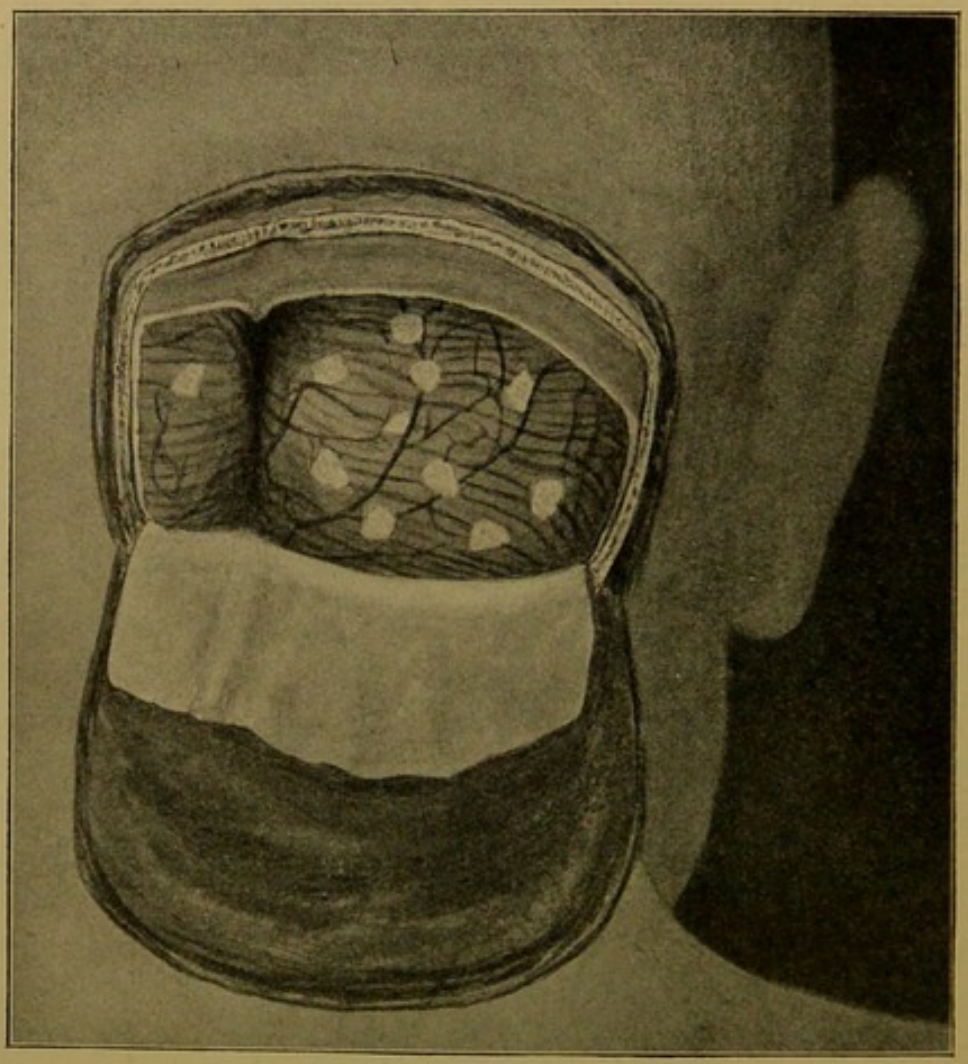
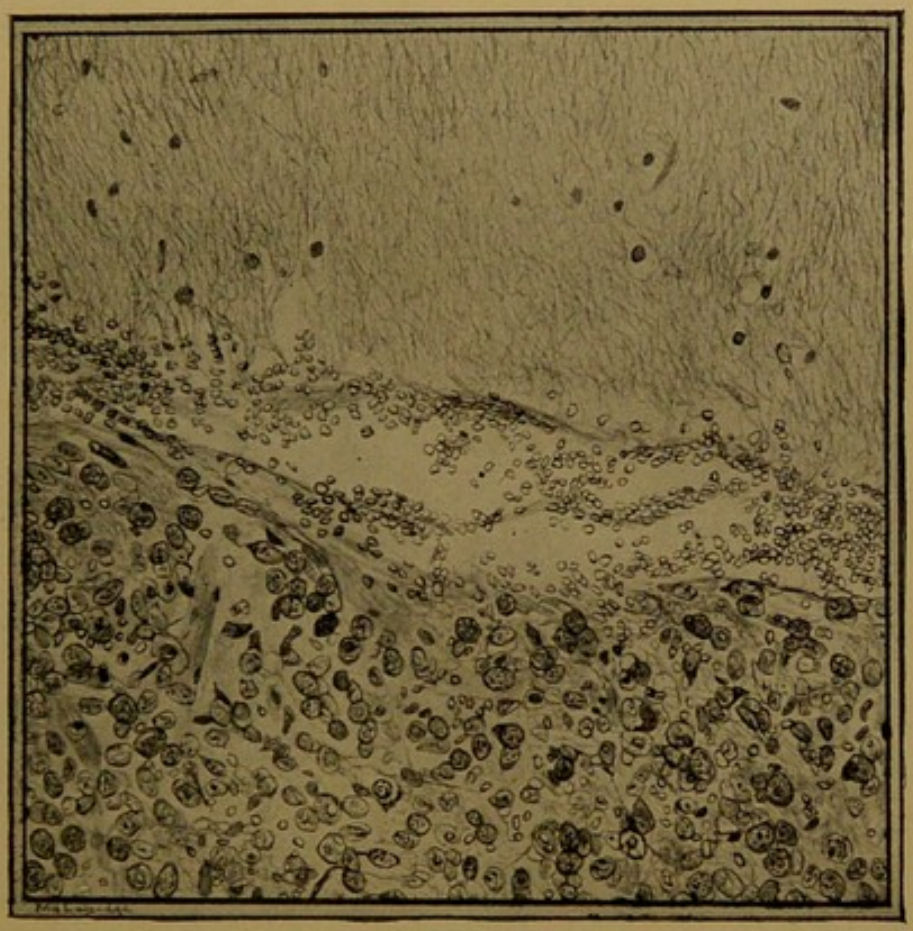


FIG. 37.—Operation for cerebellar tumour disclosing discrete white patches of round-celled sarcoma of pia and arachnoid. (Case 7.)

gold tube was passed through the scalp and cerebro-spinal fluid escaped in quantity. The child again recovered. The drainage proved intermittent and inefficient, so in May I had him up to St. Thomas's Hospital. He was then no longer quite intelligent. Though free drainage of cerebro-spinal fluid was established, he died on June 19.

Autopsy.—Left cerebellar hemisphere encased in a meningeal plaque of white growth ; right also covered with tumour masses, but was so adherent to the craniectomy opening that it became broken up during removal. Plaques of pale growth on meninges of cerebrum, both lateral ventricles dilated, fourth ventricle coated with growth, which had also made its way through the iter.



×225

Fig. 38.—Sarcoma of leptomeninges of cerebellum. (Case 7.)

C, Cerebellum not invaded by growth. T, Tumour tissue.

Remarks.—The limitation of the growth to the arachnoid, the pia, and the ependyma is remarkable. The X-ray treatment entirely failed. In cases of tumour of the cerebellar hemisphere in children the head is often inclined towards the shoulder of the same side. Abduction of the right thigh in standing was noted in this case, and Louis Tollemer ([31]) has published drawings of abduction of the

right thigh in standing and sitting in the case of a child with right cerebellar tumour (solitary tubercle).

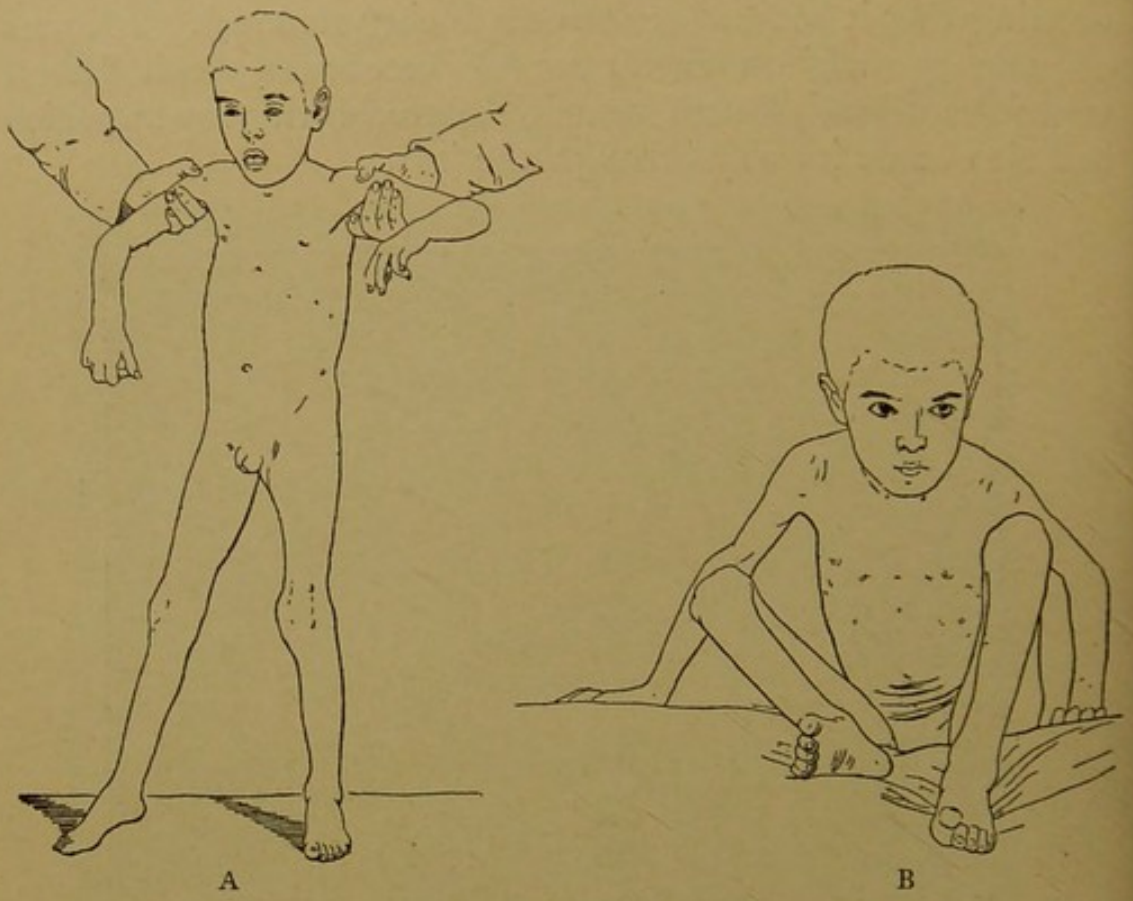


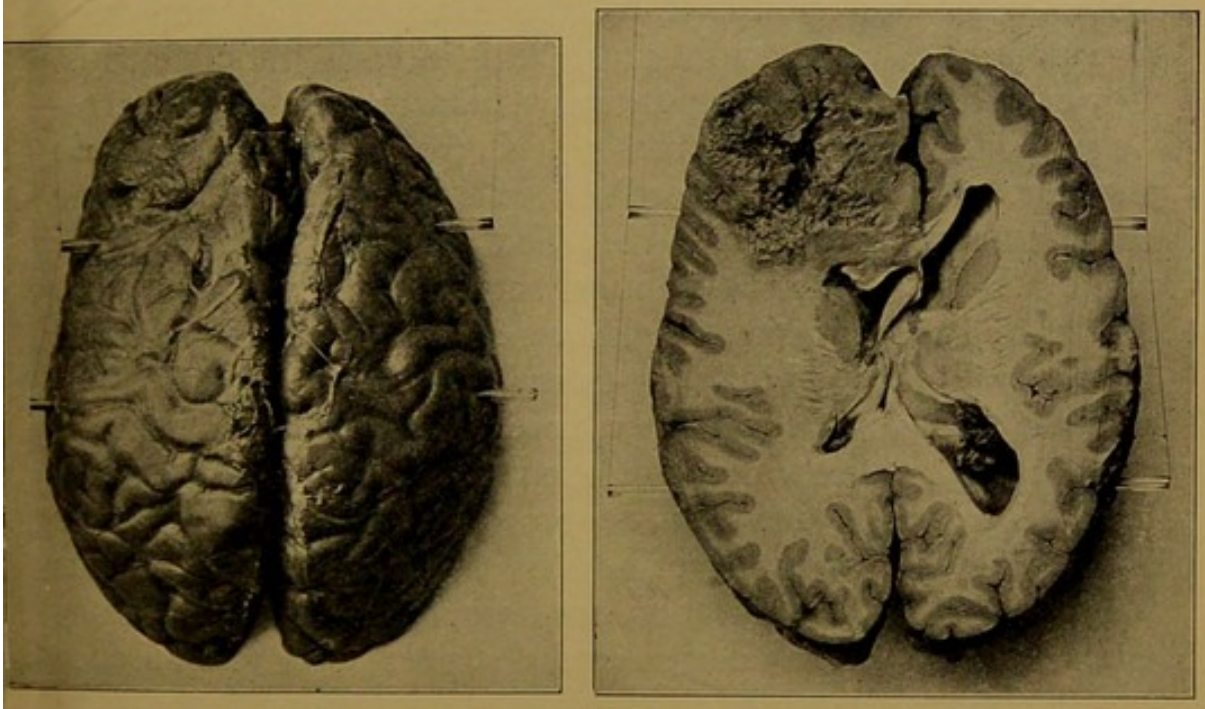
FIG. 39.—Child with right cerebellar tumour (solitary tubercle). (Louis Tollemer.)

A, position when standing, right thigh abducted; B, position when sitting, right thigh abducted.

CASE 8.—*Glioma of frontal lobe. Failure of localisation diagnosis. Delay in operation. Late decompression. Loss of sight. Death.*

Female, aged 30 years. Hospital nurse and daughter of a doctor. Sent to me from Ireland. Seen *February 2nd*, 1911. In August 1910 did not feel well and while away on a holiday vomited. In September vomiting became severe. In October optic neuritis was found and headache commenced. She fell down once and the right arm became weak. On *February 7th*, 1911, optic neuritis was well-marked on both sides, it was thought that the right external rectus was weak, but there was no diplopia. She stood less

securely on the left than on the right foot and there was slight inco-ordination of the left leg (heel-knee test.) In April vomiting began again and she had a fit, with temporary loss of consciousness and rigidity of limbs. She lurched slightly in walking but not more often to one side than to the other. The right side of the face moved less than the left when she smiled, it was stated that this was a congenital peculiarity. There was no alteration of the mental state or character.



FIGS. 40 and 41.—Cystic glioma of left frontal lobe. (Case 8.)

FIG. 40.—Note the swollen condition of the anterior pole of left frontal lobe.
FIG. 41 shows a horizontal section of the brain and tumour.

Operation April 29th.—Right cerebellar region explored, no tumour found. Sir David Ferrier and Dr. Purves Stewart concurred in the view that the right cerebellar region should be explored, and Dr. Purves Stewart gave me much valuable help in the management of this case.

Result.—Patient gradually became blind from optic atrophy and died early in August during the intense heat. At the autopsy a large glioma of the left pre-frontal region was found.

Remarks.—Operation was in this case delayed on the

advice of an ophthalmic surgeon who thought that the optic neuritis was not increasing and also because localisation seemed impossible. The facial weakness which was said to have been congenital was no doubt due to the tumour. Many cases have been recorded in which a tumour of the frontal lobe has been mistaken for one in the cerebellar hemisphere of the opposite side. It is a curious fact that the pain of a cerebellar tumour is sometimes referred to the contra-lateral frontal region, and *vice versa*.

Some years ago I published the case of a man of 40 years who was admitted to the National Hospital with agonising headache, vomiting, and intense optic neuritis. For certain reasons the tumour was located in the left cerebellar hemisphere. I explored this region but found no tumour. The man recovered completely from the syndrome and returned to work. He died eighteen months later when a large tumour was found in the right frontal region. In frontal lobe ataxia the patient sways from side to side, but in walking he does not describe zig-zags and does not stagger like a patient with a cerebellar lesion. Mills ([32]) describes a case in which the ataxia of the fore-limb might have been due to loss of the power of attention. In the above case the signs described by Grainger Stewart ([33]) as being present in frontal lobe tumours, namely, fine vibratory tremor of the extended ipso-lateral upper limb and absence of the contra-lateral abdominal and cremasteric reflexes were not present. I have a case of frontal lobe tumour now under my care which presents fine vibratory movements of both upper limbs when extended, and absence of the contra-lateral cremasteric reflex. Incontinence of urine was noted by the late Charles Beevor ([34]) as an early symptom in tumours of the pre-frontal region, but this sign was not present in this case.

Some ophthalmic surgeons seem to have hardly realised the value of decompression in cases of optic neuritis of intracranial origin. They fear the risk attending decompression. I think these fears are groundless. In abdominal exploration the risk is to all intents and purposes *nil* : danger arises when abdominal exploration leads to interference with organs.

So it is when a window is made in the cranial wall. In decompression the risk is negligible: danger comes when it is necessary to interfere with the cranial contents. In the presence of optic neuritis decompression must not wait for localisation diagnosis, which may never be possible. The calamity of blindness is a too terrible disaster to contemplate in an opportunist manner. Decompression is a sure remedy, and should be promptly at the service of the patient.

CASE 9.—*Encapsuled tumour of meninges simulating tumour in region of pineal body. Right occipital decompression. Drainage of right lateral ventricle for eight months. Distension of left lateral ventricle. Death.*

Male, aged 23 years. Seen in consultation with the late

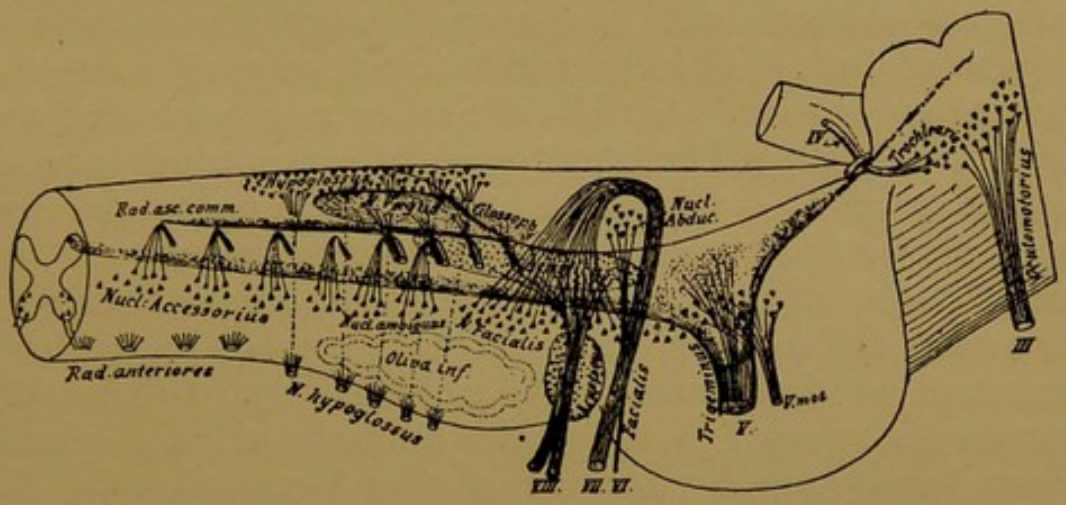


FIG. 42.—The deep origins of some of the cranial nerves. From Hermann's *Topographische Anatomie.* (Case 9.)

Note the origins of the 3rd and 4th nerves beneath the corpora quadrigemina and the origin of the abducens from the floor of the 4th ventricle.

Dr. Stanley Smith and Sir David Ferrier. Patient had also been seen by Dr. Risien Russell and Dr. James Taylor.

Examination December 15th, 1910.—Said to have had a severe blow on the left side of head from a cricket-ball some years ago while at Eton, but no trace of a scar is to be found. For a year he had suffered from headache and diplopia. The pain is always on the left side of the head, and has been very severe, especially on movement. Wassermann reaction negative. No abnormality of motion or of sensation

in face, arms or legs. Both pupils stabile. Lateral conjugate movement of eyes normal. Cannot move the eyes upwards, and convergence is not possible. On making an attempt to look upwards, when the eyes were directed downwards, rapid rotatory nystagmus occurred, both eyes rotating like Catherine-wheels in a direction upwards and inwards. Much swelling of optic disc on both sides with intense engorgement of veins.

Diagnosis.—Mid-brain tumour, possibly of pineal body. After careful consideration it was decided to do a right occipital decompression in order to avoid any chance of damage to the speech centre by doing a decompressive operation on the left side.

December 18.—Bone removed over right occipital region and upper part of right cerebellar region, so that drainage could be effected both above and below the tentorium. Incision of dura. The occipital lobe bulged greatly into the craniectomy wound. Sir David Ferrier suggested that the occipital lobe should be lifted up and the tentorium incised, but the patient's condition was too bad for me to do anything more at the time, and unfortunately this step was not carried out at a later period. The patient passed through a somewhat difficult convalescence, the cerebro-spinal fluid continued to drain away until February 17. The optic neuritis disappeared soon and the headache at once ceased. It was hoped that, by the use of the X-rays through the craniectomy opening, the growth of the tumour would be checked, but the last of the nine treatments, carried out on February 17, produced so much swelling and bulging of the flap that the attempt was abandoned. It was found necessary to introduce a fine tube by the side of the flap to allow fluid to escape and give relief to the bulging and headache which had returned. The tube was removed on February 23. Early in March, while in this patient's house, I injured my knee, and had to go to Bath for treatment. On March 22, hearing that the patient was dying, I left Bath, against the advice of Mr. Forbes Fraser, for London, and found the patient comatose with the flap very tense and greatly bulging. The bulging of the flap had

taken place slowly during the previous two weeks, and had been associated with drowsiness, headache, difficulty in feeding, a left Babinski reflex, and incontinence. A small incision was made in the flap, through which a fine gold trochar and cannula, such as I show you, was passed into the posterior cornu of the right lateral ventricle. A large quantity of fluid immediately escaped and the flap became concave. The next morning the patient expressed himself as feeling quite well. The drainage of the right lateral ventricle was continued until the death of the patient on October 20, 1911. At one time there was a fear of staphylo-

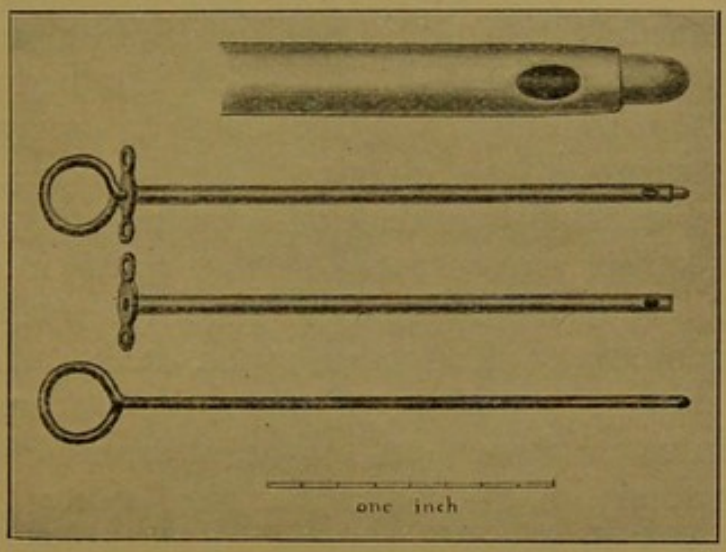


FIG. 43.—Tubes and stylets made of platinum or of an alloy of gold and iridium—materials which are not acted on, as is *e.g.* silver, by the living tissues and fluids. The lower figures are natural size. The upper figure is enlarged. The stylet projects beyond the cannula. (Case 9.)

Cannulas of varying lengths were employed to drain the lateral ventricle on to the scalp for 8 months.

coccic infection, but a few doses of vaccine, prepared by Dr. Dudgeon, made the local condition in a few days again satisfactory. From the time when drainage of the lateral ventricle was established a remarkable improvement occurred, the patient went out walking and driving, and went to the theatre and to church. The movements of the eyes greatly improved, convergence and upward movement again became to some extent possible, the pupils reacted, the vision was 6/12, but there was still intermittent diplopia. The discs showed no sign of the past neuritis. The patient

went into the country early in April and was at that time able to be out all day.

I saw him in Scotland about the middle of September. He was very well, with the exception that of late he had slept much in the afternoon, and complained that the right leg trembled a good deal, and that he was unable to control it. I did not see him again. At the beginning of October there was some difficulty with the drainage, and the patient was not so well. A longer tube was introduced and the drainage of the right ventricle re-established, but the general condition continued to deteriorate. The patient became more and more sleepy, his sight became worse, and he had a tendency to turn towards the right in walking. The walking was sometimes better and sometimes worse. He died on October 20, 1911, ten months from the date of operation. Dr. MacAlister wrote, after the patient's death, to me as follows: "There was a strong tendency in walking to turn to the right, while the movements of the right foot and leg resembled those of a spastic case. About a week before he died he became very sleepy and much weaker. He would go to bed about the middle of the forenoon and often not wake for lunch until well on in the afternoon. He would then take food in a drowsy fashion, quite under the impression that it was his proper lunch time. After this he would sleep again, and wake in the evening, have tea, and again sleep until seven o'clock or so the next morning; he would then be quite bright and fairly alert mentally. In eating it was noticeable that he would cut his food into large mouthfuls, and, after putting a portion in his mouth, would keep it there as long as five minutes before swallowing it, even when he was apparently not drowsy. He complained much of fidgets in his legs, which were only soothed by massage. His waking time became less and less. Three days before he died he was drowsy all day; for the last thirty-six hours he was quite comatose. The drainage of the right ventricle continued to the end. Before death the temperature rose to 102 and the pulse fell to 55 and was irregular. Death occurred from failure of respiration."

Autopsy by Professor Sutherland and Dr. MacAlister.—

A circumscribed ovoid meningeal tumour about the size of a bantam's egg, flattened from above downwards and extending rather more to the left than to the right of the median line, was found lying beneath the tentorium behind and the corpus callosum and fornix in front, which were arched over it. There was a depression on the anterior and internal part of the upper surface of the left cerebellar hemisphere, in which the posterior part of the tumour rested,

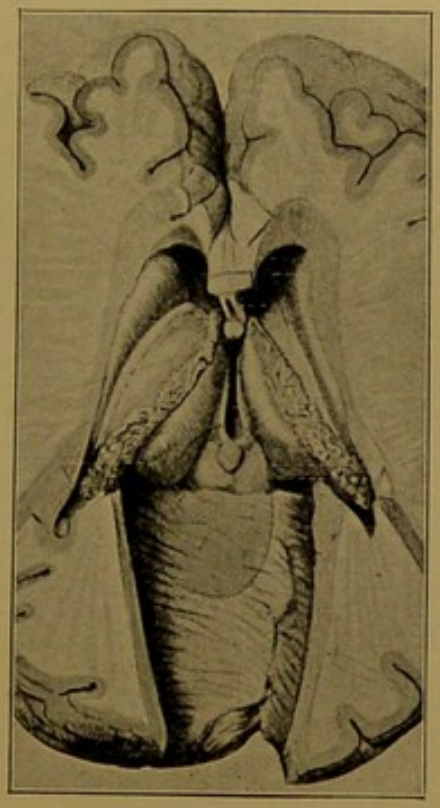


FIG. 44.—The drawing is reproduced from Hermann's *Topographische Anatomie*, and on it is placed an oval outline to indicate the position of the tumour, which lay on the left lobe of the cerebellum, the corpora quadrigemina, and the pineal gland. (Case 9.)

while the remainder extended forwards under the fornix and corpus callosum as far as and beyond the pineal body. The tumour was in part hard and in part soft and vascular. The Sylvian aqueduct was occluded by pressure. The pineal body was not involved in the tumour. The tumour itself had only a meningeal attachment. It was quite separate from the brain substance. The left lateral ventricle and the third ventricle were greatly dilated. The convolutions of the left cerebral hemisphere were much flattened.

The right lateral ventricle had been continuously drained, and the cerebral convolutions on that side were much more nearly normal. Evidently in the later stages of the illness the connection of the left lateral ventricle with the other ventricles had become occluded, and hence death had occurred from internal hydrocephalus affecting the left lateral ventricle. Histologically, the growth was an endothelioma. Professor Sutherland suggests that the tumour probably originated

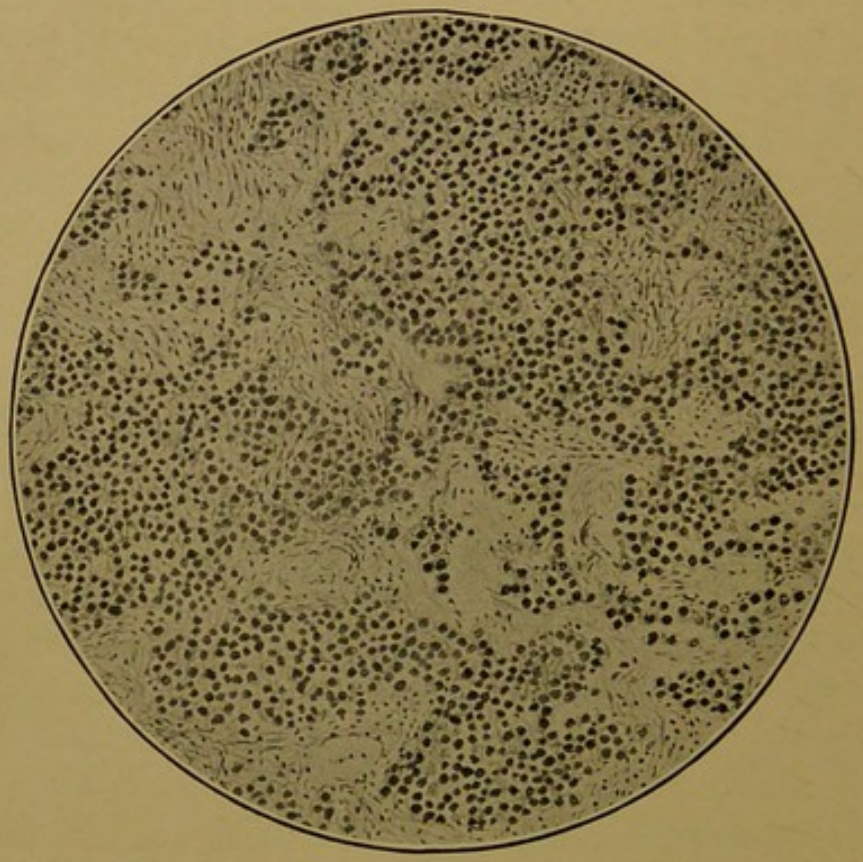


Fig. 45.—Endothelioma of the meninges. (Case 9.)

from the ependyma immediately in front of the pineal gland, but I think it might have arisen from the meninges of the cerebellum.

Remarks.—The tumour might have been removed had Sir David Ferrier's suggestion been carried out. Dr. MacAlister said that the tumour seemed as if it would have shelled out easily.

Decompression had been effected for eight months by means of a tube carried through the scalp into the posterior cornu of the right lateral ventricle. The excellent nursing

(Nurse F. Woods) secured the patient from the dangers of sepsis. It is noteworthy that the left-sided pain, though ignored, was a localising symptom. The recovery of the movements of the pupils and eye-balls proved that the third nerve nuclei were not destroyed by the growth of an infiltrating tumour, and that pressure alone was the cause of the paralysis. Further, if the view that the patient was snffering from an infiltrating tumour of the mid-brain, possibly fungating into the third ventricle, had been correct, drainage of the ventricle for so many months without any difficulty arising would have been very unlikely, as such tumours tend to spread over the surface of the ventricle and

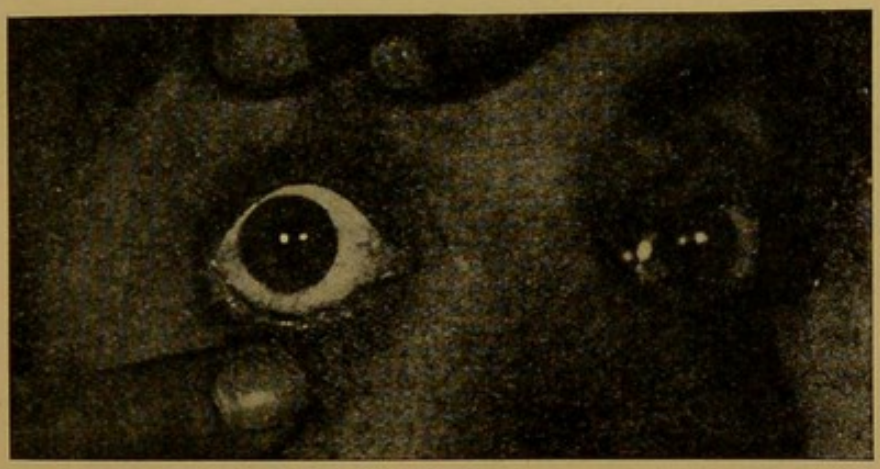


FIG. 46.—*Ectopia pupillæ* in a mesencephalic lesion. (S. A. K. Wilson.)

to produce hæmorrhages, both of which conditions would inevitably interfere with drainage. The Sylvian aqueduct being blocked, the stream of fluid in its way out through the drainage tube would tend to spread an infective growth over the right lateral ventricle, and I always feared that this would occur. The recovery from the ocular palsy and the constant and easy escape of cerebro-spinal fluid ought, in my view, to have suggested to us that the original diagnosis was wrong, that the tumour was encapsuled, and that Sir David Ferrier's suggestion should be acted upon.

S. A. K. Wilson ([35]) has described a curious symptom which sometimes occurs in mesencephalic lesions, namely, *ectopia pupillæ*, a condition in which the pupil is not in the centre of the iris. This sign was not present in the above

case. In Weisenburg's ([36]) paper, entitled "Tumours of the Third Ventricle with the Establishment of a Symptom Complex," much valuable information is given concerning the diagnosis of mid-brain tumours.

CASE 10.—*Cystic glioma in left cerebellar region. Late decompression and partial removal of tumour. Great loss of sight.*

Female, aged 9 years, a patient of Dr. Wright of Coltishall, Norwich, and Dr. Risien Russell. *Seen* June 12, 1911.

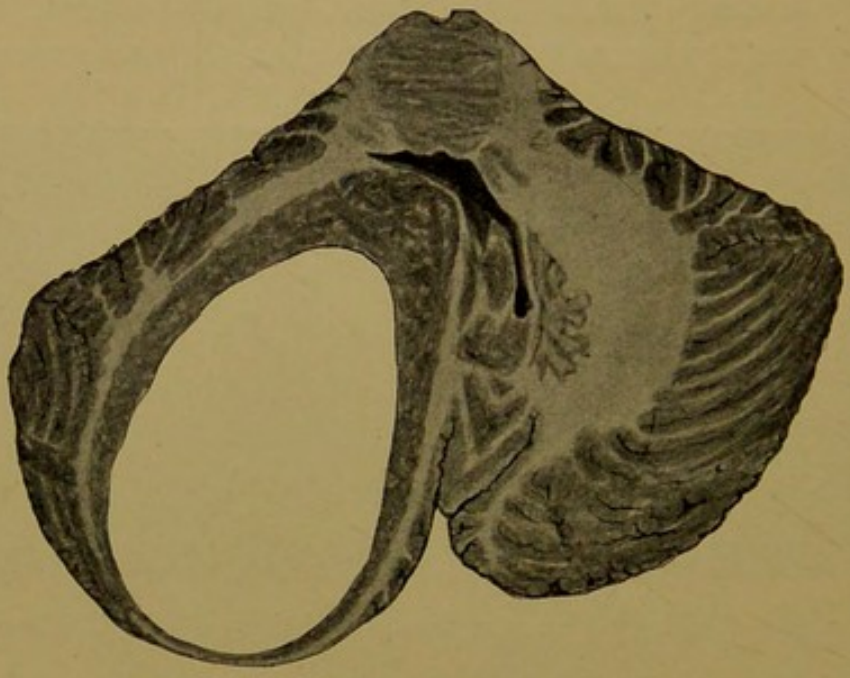
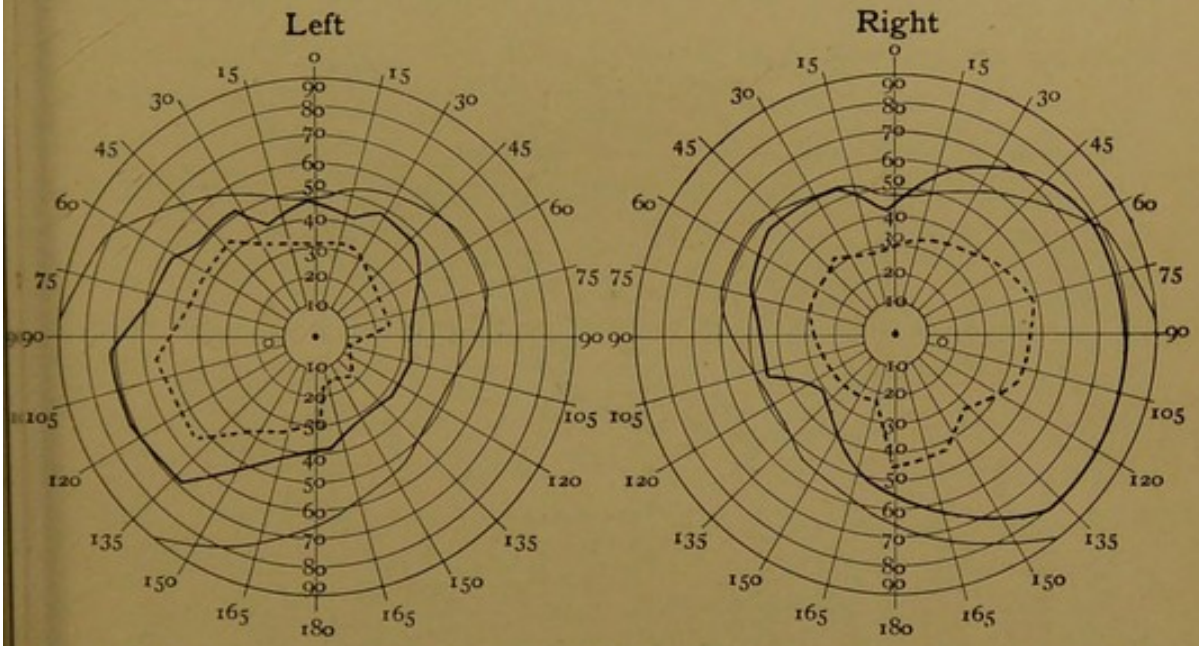


FIG. 47.—Cystic glioma of left cerebellum hemisphere. (Case 10.)
(Drawing of position of tumour made immediately after the operation.)

It was stated that she had been stunned by a fall on the ice two years previously but she had remained quite well until November 1910. It was then noticed, while she was having instruction in dancing, that she was unable to curtsey properly, or to stand on one leg, also it was found that she was unable to learn to ride a bicycle. In March 1911 she began to lurch to the right in walking. Her sight had been notably failing for three weeks. She had neither headache nor vertigo, but had severe attacks of vomiting without apparent cause. Intense optic neuritis had been found ten days before I saw her.

When seen. Standing on left foot much more difficult than on right. Definite inco-ordination of left arm. Romberg's sign decided (fell backwards and to the left). Tenderness over both cerebellar fossæ. Slight impairment of hearing on left side. Slight nystagmus on looking to the left, conjugate movement of the eyes less perfect to the left than to the right. Bi-lateral optic neuritis with swelling of 5 D. The perimeter charts showed considerable contraction of the blue fields. All reflexes minus and difficult to obtain.



FIGS. 48 and 49.—Perimeter charts of a case of cerebellar tumour made in good daylight by Mr. Fisher.

The thick continuous line represents the field of vision for white and the dotted line shows the field of vision for blue.

Diagnosis.—Tumour of vermis and left cerebellar hemisphere with hydrocephalus.

June 16, 1911. *Operation.*—Bone removed over left cerebellar region and over part of right cerebellar region.

June 27. *Second stage.* — Dura opened. Occipital sinus tied, left hemisphere greatly bulging. Incision of the cerebellar hemisphere showed that most of it had become converted into a large gliomatous cyst. The fluid within the cyst was turbid. As much as possible of the cyst and the solid growth around it was removed. The turbid fluid of the cyst yielded a pure culture of *Staphylococcus aureus.*

Convalescence.—Wound did not heal till staphylococcus vaccine was used. Much discharge of cerebro-spinal fluid for three weeks. Left for home July 29 almost blind from optic atrophy. Probably the neuritis had existed since the time of the dancing class the previous November.

February 1912.—Now well but very little improvement in sight. Atrophy of optic discs. There is a good deal of bulging of cerebellar flap.

Remarks.—The delay in diagnosis was the reason why operation was deferred until so late in the evolution of

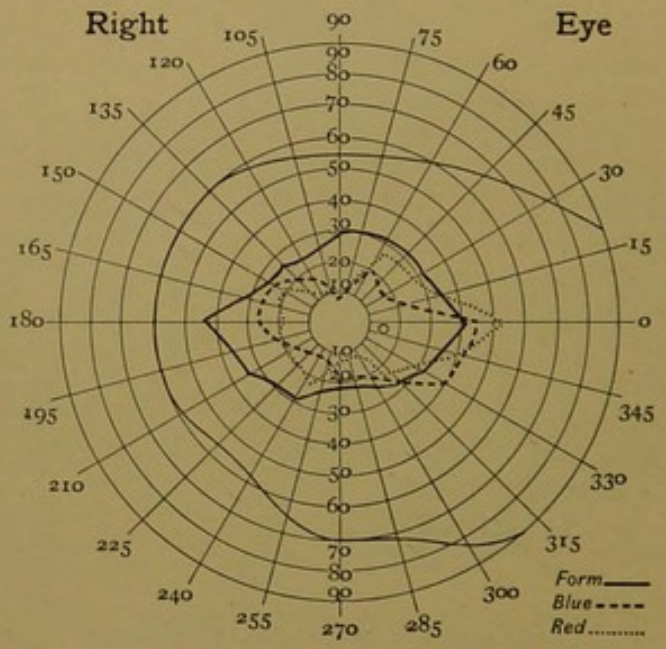


FIG. 50.—Perimeter chart showing alterations in the colour fields in a case of brain tumour. (Bordley and Cushing.)

"The chart shows characteristic contraction, greater in the form than in the colour fields, in addition to colour and form interlacing."

The patient, a young girl, had in addition to the syndrome focal palsy. The swelling of the discs measured 3 D. Complete restoration of normal relations of the colour fields followed a simple decompression which disclosed the brain under tension.

disease. The appalling disaster of almost complete loss of sight was the inevitable result.

The question, arises, Is the cystic degeneration of gliomata due to bacterial infection? This seems to be worthy of future investigation.

Cushing ([37]) has shown in a classical paper that contraction of the blue fields is one of the early signs of brain tumour, that it is not due to hysteria, and that this contraction is part

of the tumour symptom-complex, and that its demonstration is an aid in the diagnosis of an actual organic lesion. The examination of the blue fields, therefore, in a doubtful case of brain tumour should never be omitted.

CASE 11.—*Cystic glioma of cerebellum. Decompression. Removal of tumour. Arrest of optic neuritis. Recovery.*

Female, aged 21 years. *Seen end of July* 1909, brought to me by Dr. Beresford and seen also with Dr. Purves Stewart. In December 1908 began to feel tired and incapable of exertion. In March 1909 severe pain in back of head and vomiting, without apparent cause, especially in the morning. She began about this time to walk unsteadily and lurched to the right. These symptoms persisted. In June optic neuritis had been found by the late Mr. Marcus Gunn. The tuberculin reaction was negative. She became unable to manage the affairs of her father's house, more especially the accounts. When seen her gait was unsteady and she lurched towards the right, she stood less securely on the right than on the left leg, and the finger-nose test showed slight inco-ordination of the right hand, and the heel-knee test showed unsteadiness of the right leg. Giddiness was more intense on rotation towards the right than towards the left in the vertical position. The right occipital region was tender, and right cerebellar region was more prominent than the left. Severe bi-lateral optic neuritis with hæmorrhages, and marked dilatation and tortuosity of veins. All other signs normal.

Operation. First stage July 28.—Bone removed over right and part of left cerebellar region. *Second stage August* 1, as an emergency operation. Severe pain during the previous night, with failure of pulse and respiration, made imperative the immediate opening of the dura. The respiration fell to 6 per minute. The dura, which was very tense, was opened so as to expose both cerebellar hemispheres, and the occipital sinus was tied. The cerebellum bulged through the opening and the cortex of the right cerebellar hemisphere burst, yellowish clear fluid escaped from a cavity in a glioma, and a clot about the size of half-

a-crown, which had probably caused the sudden alarming symptoms, escaped. The posterior half of the right cerebellar hemisphere, in which the tumour was contained, was amputated. Much cerebro-spinal fluid escaped at the operation, and continued to drain for between two and three weeks.

Result.—Complete recovery without impairment of vision. I last saw her on January 31 in the present year, two and a half years after the operation, she was then quite well, and able to do everything as before her illness. The optic discs were normal.

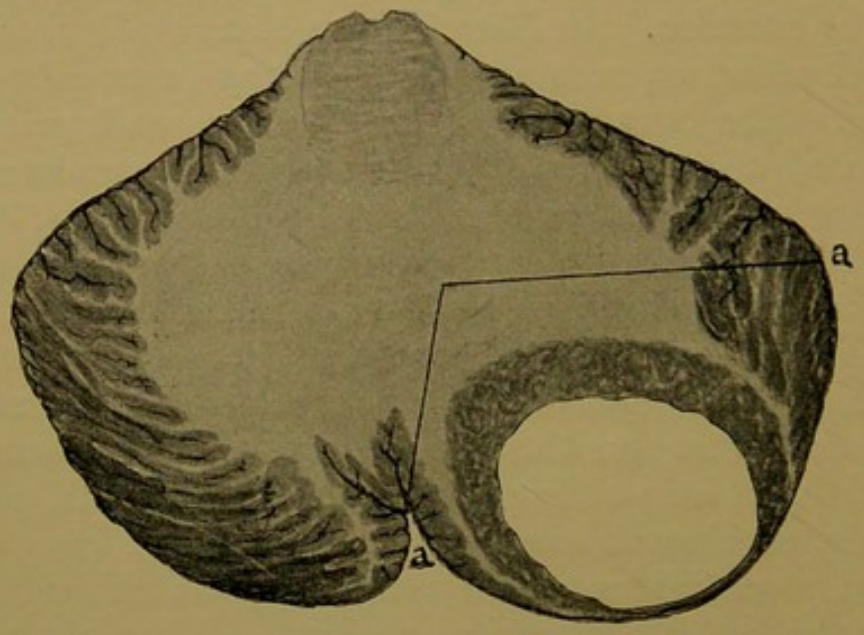


FIG. 51.—Cystic glioma of right cerebellar hemisphere. (Case 11.)
a, a, line of section for removal of tumour.

(The drawing was made immediately after the operation was completed.)

CASE 12.—*Cerebellar tumour diagnosed. Decompression. No tumour found. Recovery.*

Female, aged 32 years, very intelligent, a patient of Sir David Ferrier. In September 1904, six weeks after the birth of her last child, she began to have attacks of violent headache and purposeless vomiting, two or three weeks later acute attacks occurred every few days. The pain then diminished and became limited to the retro-ocular region. In the early part of 1905 the headache became again more frequent and more acute. In April 1905 the headache and retching became associated with mistiness of vision, numb-

ness of the legs, and some difficulty in walking. *When seen in July* 1905 occipital headache, nausea, and mistiness of vision, coming on in what the patient described as "waves." Intense bi-lateral optic neuritis. Left cerebellar region more tender than right. No motor or sensory paralysis of limbs. No ataxy of limbs. Gait feeble, not characteristic. Reflexes normal.

Operation in two stages. Bone removed over left cerebellar hemisphere, and dura, which was under great tension, opened three days later. No tumour was found in the cerebellar fossa. Cerebro-spinal fluid drained for ten days.

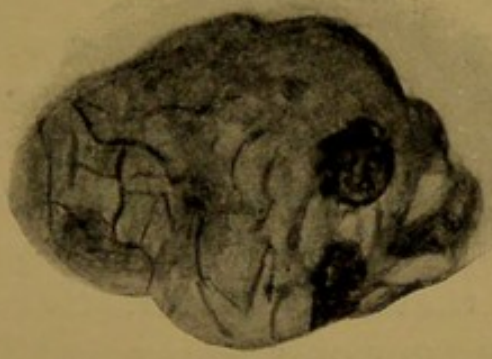


FIG. 52.—Photograph of tumour immediately after removal.

It was a fibro-sarcoma situated in the right cerebello-pontine angle. The operation was done in November 1894. The patient is still alive.

Result.—Rapid disappearance of optic neuritis and other symptoms. I saw this patient quite recently, she was then, seven years after the operation, quite well. As a complement to this case I will just mention one other ([38]). In 1904 I removed an encapsuled extra-cerebellar tumour (Fig. 52) from the cerebello-pontine angle in a woman aged 49. I have heard of her recently and she is quite well.

Sir David Ferrier's address to the Section of Neurology, "On the Treatment of Intracranial Tumours," at the Annual Meeting of the British Medical Association, held in Edinburgh in July 1898, commences with the following striking sentence: "The treatment of intracranial tumours forms rather

a melancholy chapter in therapeutics." I know that this is not his view to-day, but it is still not infrequent to hear pessimistic remarks made concerning the results of operations for tumour of the brain. A careful consideration of the records shows that there is little justification for such views. In malignant disease of an organ, such for example as the breast, the whole organ at least must be amputated, but we cannot do and we never proposed to do a hemi-cerebrectomy for malignant disease say of the frontal lobe. But we can do decompression which relieves completely all the most distressing symptoms of the patient, and I would add that in the whole range of surgical work the operation of decompression in its various applications has given more relief from pain and distress than almost any other surgical procedure. The truth about operations for the removal of malignant disease is that at present, though horribly mutilating, they are our only resource. The time will come when such operations will be replaced by a remedy which will fulfil the ideal of Paracelsus and be antagonistic to the spiritual seed of the disease.

In conclusion let me say that my main object in writing this paper is to urge a decompressive operation:

1. In certain cases of *injury to the head* especially:
   - (*a*) In subdural hæmorrhage of the newly-born.
   - (*b*) In fractured base.
   - (*c*) In those cases in which, after apparent recovery from an injury to the head, there are *intermittent* symptoms such as headache, insanity, or paralysis. In these cases the lesion is often ingravescent subdural hæmorrhage.

2. In selected cases of *apoplexy* in the acute stage.

3. In *tumour of the brain* decompression should be done before vision is lost by optic neuritis, or the mind wrecked by hydrocephalus or constant suffering. The increased intracranial tension should be relieved by a decompressive operation whether or not the tumour can be localised ; and advantage should be taken of the opportunity so afforded for exploration which may determine unexpectedly the possibility of removing the tumour. In all infiltrating tumours of the brain simple decompression is the method of choice. When feasible the tumour should, of course, be removed ; but, as Carlyle says, "It is greatly wise to recognise the impossible, the unreasonably difficult, when it presents itself" ([39]).

Cushing quotes from Leonardo a line of signal warning, "The supreme misfortune is when theory outstrips performance." There is much yet to learn, but we can at least definitely affirm that no patient should be allowed to die from mere increased intracranial tension, and no patient should be allowed to become blind from optic neuritis. The increased intracranial pressure is the one point of the disease which is always *vulnerable to surgical attack*, and whether localisation diagnosis is possible or not decompression should be carried out without delay. Those who remember their schooldays will recollect the great efforts which Caesar always made to give battle in the enemy's country as soon as he knew that a confederation of the Gauls was on foot, and to strike an effective blow before the various tribes could be united against him. On one occasion he passed lightly over the rebellion of one of the nearer tribes, in order that he might not lose the summer season for his more important operations in a more

distant part of Gaul. "The summer," he said," was the time for pressing on the war, not for judicial inquiry" ([40]). So the early or summer stage of increased intracranial tension is the time for pressing on intervention, not for prolonged clinical inquiry. *In recent injury to the head and in apoplexy*, deepening coma, *after apparent recovery from injury*, intermittent symptoms, and in *tumour* optic neuritis should lead the surgeon to take prompt action. By so acting many lives will be saved and much suffering averted, but success will not always be obtained, for to quote once again from Caesar : " Et ad subeundum periculum et ad vitandum multum fortuna valet " ([41]).

ADDENDUM

Fracture of skull. Operation. Apparent recovery. Death from brain abscess ten years later ([42]).

The patient was a woman aged 31 years. Ten years before her admission into hospital she was injured in an explosion. An operation was performed shortly after the accident, and she ultimately returned to her work apparently cured. She remained in excellent health and free from all cerebral symptoms until eight years after the accident, when an attack of acute pain in the left side of the head, with great tenderness at the site of the injury, occurred. She vomited and felt tingling down the right arm, but had no paralysis and no rise of temperature. The attack passed off in three or four days, and, beyond occasional headaches, no further symptoms were noticed until one month before admission; very severe headache now started with frequent vomiting, movements and sensation were apparently perfect, and no other disturbances were noticed. Such was the history given by the medical man who sent her to hospital. When examined in January 1901, ten years after the accident, the patient was looking extremely ill, and complained of intense pain in the left temporal region. Cerebration was accurate but very slow. The left side of the face was marked with bluish scars the result of the gunpowder explosion. Two inches above and one inch and a half behind the external auditory meatus was a surgical scar under which the bone was extremely irregular and depressed—the scar was excessively tender. No paralysis was detected. Bilateral optic neuritis was found, being most intense on the left side. The pupils were normal. The deep reflexes were exaggerated. The pulse was 64 regular and full, and the temperature was

98° F. Examination of the ears showed nothing abnormal, and hearing was good. A cerebral abscess was diagnosed, and operation was performed two days later. The bone was found depressed, and presented tattoo marks from the old injury. An abscess in the temporo-sphenoidal lobe was readily found, and about one ounce of thin green odourless pus was let out. A silver drainage tube was introduced. About a fortnight later she was doing excellently. There had been no return of the headache or vomiting, and the optic neuritis was subsiding. Then suddenly symptoms returned and persisted. The brain was again explored on two occasions, and a reaccumulation of pus found. Larger drainage tubes were inserted. On the thirty-second day after the primary operation very acute symptoms suddenly supervened, the temperature rose to 104, and death rapidly occurred. Unfortunately no necropsy was allowed.

Remarks.—I remember when I was a student there was a fatal case of brain abscess under the care of the late Dr. Murchison which occurred 20 years after an injury to the skull.

Severe syndrome of cerebral tumour. Subtemporal decompression on left side. Return of headache and continuing neuritis. Wider decompression. Recovery but with loss of sight.

A man, aged 23, came under my care in St. Thomas's, in February 1912, with a hernia cerebri the size of a tangerine orange in the left temporal region. This had followed a subtemporal decompression operation performed in the Derby Hospital the previous August. The opening in the skull was subsequently found to be about the size of a two-shilling piece.

History.—Eighteen months ago the patient had a severe fit, and last August he was admitted to the Derby Hospital with severe headache, loss of memory, slowness of speech, dimness of vision, and double optic neuritis. On admission to St. Thomas's the hernia was very tense, the man was drowsy, and complained of constant headache. Vision was very poor, much difficulty in distinguishing fingers at a

distance of one foot, and persons were not distinguished at all. The discs were white and the edges swollen and blurred. Anosmia. Slight right facial palsy. Right Babinski reflex. Tremor of both hands on extension of limbs, and the epigastric and abdominal reflexes on the right side were not obtained.

I thought that the patient was suffering from tumour of the left frontal lobe, and so on February 16 the whole of this lobe was exposed, the craniectomy being extended so that the bone defect was continuous with that made at the previous operation. A large subcortical gliomatous cyst was discovered, occupying the anterior part of the left frontal lobe. After the decompression the scalp wound healed quickly, the mental state became normal, the headache ceased, and the remaining swelling of the discs disappeared, but, alas ! no improvement in sight was possible.

Remarks.—The first craniectomy was too small to relieve the intracranial tension, and too small to arrest the neuritis and save the sight.

REFERENCES

(1) Surgery, Gynæcology, and Obstetrics, October 1905.
(2) American Journal of Medical Sciences, 1905.
(3) Holmes. System of Surgery, 3rd ed., vol. i. p. 585.
(4) Deutsche Zeitschrift für Chirurgie, vol. lvi. p. 129.
(5) See Wigglesworth, Journal of Mental Science, 1888.
(6) Reports of Medical Cases.
(7) R. Med. Chir. Trans., vol. xxviii.
(8) Lancet, August 29, 1903.
(9) Cushing (American Journal of Medical Science, 1903) operated on a negro, aged 40 years, forty-three hours after the onset of symptoms. The blood-pressure was 300 mm. Hg. and medullary paralysis was imminent. The major symptoms were immediately relieved by craniectomy and aspiration of the intracerebral clot.

Sargent's remarkable case (Royal Society of Medicine, 1909) was operated on as soon as he saw the patient, *i.e.* on the eleventh day.

Cushing in a later paper (Johns Hopkins Hosp. Bull., Nov. 1910) makes the somewhat casual remark in reference to operations for apoplexy : "There have been one or two excellent recoveries among one dozen cases operated on."

Milligan of Pittsburg (Journal of the American Association, 1911) reports a wonderful case of complete recovery. The patient was a man aged 42. A pear-shaped clot was removed. The operation was performed on the eleventh day.

Iselin (Deut. med. Woch., May 1911) reports a case of a man of 62 years, in whom a large apoplectiform hæmorrhage into the lateral ventricle was evacuated, but death occurred twenty-four hours later.

(10) Transactions of the American Surgical Association, vol. xxiv., 1906.
(11) Surgery, Gynæcology, and Obstetrics, August 1911.
(12) Surgery of Brain and Spinal Cord, vol. i.
(13) Review of Neurology and Psychiatry, January 1911.
(14) Ballance, Surgery of Brain, p. 81.
(15) Ballance, Surgery of Brain, p. 47.
(16) Zeitschrift für Ohrenheilkunde, vol. xxxviii. p. 122.
(17) Zeitschrift für Ohrenheilkunde, vols. xxxviii. and l.

(18) Archiv für klin. Chirurgie, vol. lxxvii.
(19) Archiv für klin. Chirurgie, vol. xciii.
(20) See Stoddart Barr's case in which this operation was done, Brit. Med. Journal, 1910, vol. ii. p. 1687.
(21) Some Points in the Surgery of the Brain, p. 47.
(22) Wiener medizinische Wochenschrift, January 1912.
(23) De Bello Gallico, liber vii. cap. 29.
(24) Cases of Cerebellar Tumour. Interstate Medical Journal, vol. xvi., 1909.
(25) Combined Exploration and Decompression of Cerebral Tumours. Transactions of the American Surgical Association, 1909.
(26) The Establishment of Cerebral Hernia as a Decompressive Measure. Surgery, Gynæcology, and Obstetrics, 1905.
(27) Weissenburg writes (New York Medical Journal, October 13, 1906): "Sudden deaths recorded as the result of this operation may be due to disturbance of circulation in the bulb."

Further instances of death following lumbar puncture in brain tumour are recorded by Hamburger (Surg. Gyn. and Obstetrics, 1910) and Spiller (Deut. militarärzt. Zeits., Feb. 1911). Last month a small quantity of cerebro-spinal fluid was removed by lumbar puncture for the purpose of the Wassermann test from a patient of mine, aet. 45, with localising signs of chronic tumour in the right cerebello-pontine recess. The syndrome was not present. The lumbar puncture was followed by severe headache, vomiting lasting for three days.

(28) Surgery of Brain and Spinal Cord, vol. i.
(29) Strangulation of the Nervi Abducentes by Lateral Branches of the Basilar Artery. Brain, 1910.
(30) Some Points on the Surgery of the Brain, p. 261.
(31) Traité de Médicine, 2nd ed. vol. ix. pp. 376-377.
(32) University of Pennsylvania Medical Bulletin, April-May 1906.
(33) Lancet, November 3, 1906.
(34) Brain, 1898.
(35) Brain, 1906.
(36) Brain, 1906.
(37) Archives of Ophthalmology, 1909.
(38) Some Points in the Surgery of the Brain, p. 284.
(39) Frederick the Great, Book iv. chap. 8.
(40) De Bello Gallico, liber vi. cap. 4.
(41) De Bello Gallico, liber vi. cap. 30.
(42) English. Lancet, 1904, vol. i. p. 632.

Printed by R. & R. Clark, Limited, *Edinburgh.*