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Abscess of the Brain.

By Professor HEINRICH NEUMANN (Vienna).

ABOUT five decades have passed since Macewen's fundamental work on the pyogenic diseases of the brain and spinal cord. The question which now arises is: What results have the last fifty years of research on brain abscesses brought to us? Firstly, we will consider some statistics and compare Macewen's results with those of later times. I will begin with statistics of temporal lobe abscess :—

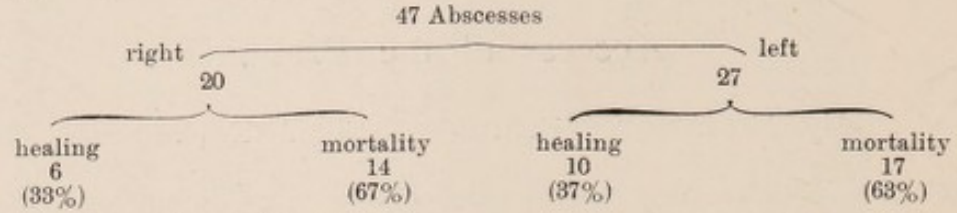
Author	Summary	Healing	Mortality
Röpke ...	142	59 (41 per cent.)	83 (59 per cent.)
Hammerschlag	53	27 (53 ,,)	26 (47 ,,)
Schmiegelow	13	5 (38 ,,)	8 (62 ,,)
Körner ...	7	2 (29 ,,)	5 (71 ,,)
Hegener	24	5 (21 ,,)	19 (79 ,,)
Henke ...	16	5 (35 ,,)	11 (65 ,,)
Heine ...	31	10 (32 ,,)	21 (68 ,,)
Michaelsen	8	5 (62 ,,)	3 (28 ,,)
Maier ...	23	9 (39 ,,)	14 (61 ,,)
Nühsmann	18	6 (33 ,,)	12 (67 ,,)
Behlau ...	5	1 (20 ,,)	4 (80 ,,)
Neumann	47	16 (34 ,,)	31 (66 ,,)
Summary	387	150 (39 per cent.)	237 (61 per cent.)

The first two, being collective figures, and not taken from one special clinic, lose in real value. The results of other figures, even the smaller ones, do not differ greatly from my own personal statistics of forty-seven cases treated at my clinic during the last ten years. And the average of them all agrees approximately (34-39%) with my figures. Comparing these statistics with Macewen's, we are surprised to find that even the best, with 62% of successes, is not nearly so good as Macewen's results, which I will indicate later, when discussing cerebellar abscess.

This difference in the successful results is the more astonishing because general medicine, and with it otology, has made great progress both in diagnosis and operative treatment. Brain surgery and diagnosis of endocranial complications especially, have improved considerably. It is an interesting question why, in spite of these facts, we are so far behind Macewen's results with his 80% of successes. Probably it is due to difference in the material. Macewen operated on selected cases, excluding all those combined with meningitis or sinus thrombosis, and he operated personally. But even when I select cases from my material, excluding all cases in which there are bacteria in the spinal fluid, and all cases of sinus thrombosis, I get only 47% of healing, which is far below Macewen's percentage. There is, perhaps, another factor that helps to explain the difference between the present day successes (47%) and Macewen's (80%). Even after the diagnosis of brain abscess was made, Macewen, at a preliminary operation, exposed the dura and painted on it a 20% solution of carbolic acid to get protective adhesions before actually opening the brain; thus, we are justified in thinking that he waited

longer than we do before operating. Then, if we consider that some pathologists think that the prognosis of otogenic brain-abscess would be better if operation were performed in the so-called "cold stage," it is possible that this "delayed operation" is one of the reasons why Macewen's results are better than ours.

Comparison of results in right- and left-handed brain-abscesses.—Since brain-abscesses on the right side have not the typical localizing symptoms of aphasia, they are generally operated on later than those on the left side. We have examined our figures with a view to discovering whether abscesses on the right have a better prognosis than those on the left. Here is the result :—



Now in reality this slight difference cannot serve as an explanation. Study of the history of the cases showed that in cases of left-sided brain-abscess bacteria are found in the cerebrospinal fluid twice as often as in cases of right-sided abscess: this means that they are probably operated on even later than right-sided abscesses. The explanation of this fact is probably that the diagnosis of brain-abscess on the basis of general symptoms is always uncertain; we are inclined to diagnose a brain-abscess with certainty only when the characteristic focal symptoms show themselves. Therefore, in a case of left-sided otitis, we feel sure of our diagnosis of "brain-abscess" only when there is aphasia, and, in the case of a cerebellar abscess, only when there are cerebellar symptoms. But, as I can show on the basis of pathologico-anatomical findings, and, by the history of cases, aphasia is not really an early, but a late symptom. Thus, the prognosis for right-sided abscesses is better, since we have not to wait for the aphasia, the characteristic symptom of left-sided abscess. On the contrary we are obliged to make our diagnosis on the basis of general symptoms, and therefore make an exploratory brain puncture much earlier than in cases of left-sided abscess without asphasia. In other words, abscess on the right side, in spite of causing fewer symptoms, is likely to be operated upon earlier than one on the left side.

Not only with regard to brain-abscess, but also in cerebellar abscesses, the great difference between the percentage of successes obtained by Macewen and that of our own results is an unpleasant fact, as, in all his four cases of cerebellar abscess, the patients recovered.

Now, if we compare the published statistics of the last ten years, in nearly all there are similar bad results: some have even 100% mortality; only a single one (Denker) shows a large proportion of recoveries (45%), and even this is far below Macewen's outstanding percentage (100%).

CEREBELLAR ABSCESSES.

Author	Summary	Healing	Mortality
Schmiegelow ...	5	0	5 (100 per cent.)
Körner	5	1 (20 per cent.)	4 (80 ,,)
Hegener ...	6	1 (16 ,,)	5 (84 ,,)
Rejtö	6	1 (16 ,,)	5 (84 ,,)
Henke	7	0	7 (100 ,,)
Michaelsen ...	8	3 (37 ,,)	5 (63 ,,)
Nühsmann ...	8	0	8 (100 ,,)
Denker	9	4 (45 ,,)	5 (55 ,,)
Behlau	10	0)	10 (100 ,,)
Maier	14	2 (14 ,,)	12 (86 ,,)
Heine	19	1 (5·2 ,,)	18 (94·8 ,,)
Neumann ...	27	2 (7·4 ,,)	25 (92·6 ,,)
Summary ...	124	15 (12 per cent.)	109 (88 per cent.)

To explain this difference we may employ again the same arguments that we used with regard to brain abscess; and in addition we must remember the possibility of confounding a cerebellar abscess with an intradural suppuration, a cloudy flaky exudation in the lateral cisterna, or an empyema of the saccus endolymphaticus. Further, the progress made in the diagnosis of the symptoms of cerebellar abscess brings us to a similar conclusion to that which was indicated by the symptom of aphasia in the case of brain abscess. Macewen operated in his cases of abscess without waiting for the characteristic cerebellar symptoms (nystagmus, past pointing, etc.), with which modern neurology has made us familiar. We, on the other hand, while valuing these characteristic special signs, have under-estimated and neglected the general symptoms. The results of operations on cerebellar abscesses are not, however, so much influenced by early or late operations as by the kind of abscess and its pathological conditions. Therefore the pathological findings are of the first importance—as, for instance, whether the abscess is deep or on the surface—and must be taken into account in passing judgment on statistics. The shape of the abscess is not apparently influenced either by the presence of a capsule, or by its being the result of an acute or chronic otitis. The capsule does not influence the extent of the abscess, for it may be broken through and encephalitis may progress outside it. The direction of the growth and the consequent position of the abscess are probably dependent upon the blood-vessels and the structure of the nerve fibres. But in spite of the uniformity of the pathological findings (Freml. Hofmann) the clinical picture does not seem to conform with them, but is influenced to a great extent by the surroundings of the abscess.

I should now like to speak about exploratory puncture of the brain. As abscesses nearly always have a typical localization and extent, it is remarkable that in the literature, as also in my own experience, a number of cases are found in which abscesses have been diagnosed, yet in which the puncture produced a negative result. Whether a thick abscess capsule or the consistence of the contents of the abscess is responsible for this has not been proved, but it seems that in the cases of brain abscess following acute otitis, the puncture was more often without result than in those following chronic otitis (Eisinger).

In cerebellar abscess the topographical position of the abscess may also explain an unsuccessfnl puncture. As the majority are flat, the abscess may be missed by the needle, even when the diagnosis is clinically certain, as shown by typical cerebellar symptoms, or by pus actually coming out of a fistula in the dura exposed at operation. Just because the abscess is slit-shaped, it is very possible to go through it with the needle without knowing it, even when we aspirate while withdrawing the needle.

As I have said, the experience gained from my cases of brain abscess led me to the conclusion that in cerebral abscess as well as in cerebellar abscess, the characteristic focal symptoms appear relatively late. Therefore it is wiser, instead of waiting for these symptoms, to puncture as soon as the general state of the patient suggests that an abscess is present.

I have spoken of aphasia in connection with cerebral abscesses. My study of cerebellar abscess has shown that it nearly always begins with symptoms which, as we see them in retrospect, might have led us to suppose from the first that an abscess was present, and this even in the absence of the specific symptoms which appear later, or which occasionally, but rarely, may not appear at all. In my collection, for instance, there was one single abscess, 4 × 5 cm. in extent, which from the onset produced no symptom referable to the posterior fossa except paralysis of the abducent nerve. We know that cerebellar abscess is a relatively rare complication; we also know that the general symptoms of a cerebellar abscess may be produced by other cranial complications and that cerebellar abscesses are often accompanied by such complications.

The study of our cases has taught us to pay great attention to the general symptoms in the diagnosis of an abscess; repeated vomiting, for instance, is not so frequent in other complications, as in affection of the posterior fossa. As all the patients suffering from cerebellar abscess complained about dizziness from the very beginning, special notice should be taken of this symptom, especially in the case of children, who seldom complain of dizziness spontaneously. The constant occipital headache found in nearly all our cases should also receive special attention.

The course of development of symptoms cannot be explained from the localization or extension of the abscesses. It seems that for the appearance of the characteristic focal symptoms, not only the decomposition in the abscess itself, but the changes around it, are of importance. This may be explained by the fact, proved in my clinic, that the cerebellar abscess affects the white matter very late, and at first tends simply to spread between the grey and the white matter. Only when collateral processes touch the corresponding fibre-systems do the special symptoms appear; and it should be noted that in all our cases of cerebellar abscesses, except one, the specific symptoms appeared after an operation on the middle ear for the relief of some other complication.

In temporal lobe abscesses the course of the characteristic symptoms, especially aphasia, was similar.

While dealing with diagnosis, I must again emphasize the value of exploratory puncture. The diagnosis of brain-abscess is reached, clinically, only in the presence of characteristic symptoms, but from a combined study of the pathological and clinical data we found that practically the most reliable diagnostic aid was the discovery of pus by exploratory puncture. Some cases, had they been diagnosed earlier on the basis of general symptoms, and operated upon earlier, would have been saved. Therefore, in the future, when we are in doubt clinically, we should puncture more often than we did before. The possible dangers of puncture, especially of the posterior fossa, in case of other complications (sinus-thrombosis, ligature of the jugular vein, or after lumbar puncture) has up to the present deterred us from doing so, especially in cases in which the specific cerebellar symptoms have not yet developed (Fremel).

There is still another question. Should the dura be incised before the puncture? Although there are advantages in an incision, the following objections must carry weight: the circulation and the tension of the spinal fluid in the subarachnoid spaces may be adversely influenced by it, and hernia of the brain may develop.

The surgical treatment of brain and cerebellar abscesses must vary with each individual case. My study of the morbid anatomy of the condition has convinced me that each abscess needs its special method of operation, and that neither for cerebral nor for cerebellar abscesses can a uniform treatment ever be successful.

I have seen deep abscesses covered with a thick layer of brain tissue, and I have seen others lying on the surface. For the former any sort of drainage seems useless, and only removal or amputation of this part of the brain ("unroofing"—King) and packing the abscess cavity appears to be of use; the abscesses near the surface can be healed by more delicate methods such as drainage with filiform drainage tubes, as suggested by Lemaitre, or simply by opening widely with brain forceps.

As every operator finds that he succeeds in curing cases by his own method, he is naturally inclined to keep to this method so long as his results do not differ greatly from those of others: indeed all statistics show more or less the same percentage of successes.

Recently the method proposed by Lemaitre was shown by himself and by workers in certain clinics to give infinitely better results than others. I tried to treat a few cases by this method, but in some of these I had to fall back on my old method of stretching, packing, or making a counter incision. The filiform drain of Lemaitre inserted into the abscess after the puncture, worked well in the beginning:

but in the course of treatment, when thicker drains were used, we found that drainage was insufficient, and on the removal of the drain, the pus gushed out under pressure. Lemaitre's form of drainage works very satisfactorily during the first few days, and we expected much from this method, but later it failed. This failure is probably a consequence of the changed conditions of circulation and tension in the brain and spinal fluid, affecting the abscess. The change for the worse in the general state of the patient due to insufficient drainage induced us in such cases to change the treatment.

When the question of a *lasting* cure of cerebral abscess is considered, it must be recognized that the healing of an abscess cannot be looked upon as a permanent accomplishment, for brain abscesses have not sufficient tissue-material to form a solid scar over a great defect in the brain substance: consequently it is possible that one part of the resulting cavity heals as a solid mass, whilst another part forms a cyst. This cyst, sooner or later, sometimes even years later, may be reinfected by an acute exacerbation, and thus give rise to an abscess which must be considered as a recurrence of the cerebral abscess.

When dealing with cerebral and other intracranial complications of middle-ear disease, we must operate to save the patient's life, but at the same time we must not act so drastically that he may be crippled for life. On the other hand, in such a dangerous disease as brain abscess, it would be a mistake to risk the patient's life by operating timidly.

Brain Abscess from the Point of View of the Neurological Surgeon.

By Hugh Cairns, F.R.C.S.

In taking stock of my own experience I find that I have personal records of forty-eight cases in which brain abscess was either suspected or overlooked. In twenty-three of these the presence of an intra-cerebral abscess was subsequently verified by operation or necropsy. Seven never came to operation; in some others operation was performed too late. Thus, in one case, that of a frontal abscess secondary to frontal sinusitis, I arrived at the bedside of a patient only to find that she had stopped breathing about one minute before. A hasty examination while artificial respiration was being performed showed severe papillœdema, slight right facial weakness, and X-ray evidence of increased density of left frontal sinus. The left frontal lobe was hastily tapped and 2 oz. of pus were withdrawn. The patient improved temporarily, but died eight hours later from pyocephalus and leptomeningitis.

I cite this example that you may have less cause to disbelieve me when I say that, in my own practice at least, the bad results are almost entirely due to delays or mistakes in diagnosis, and it is to the difficulty of diagnosis that I would particularly like to direct attention. I had originally hoped to say something about treatment, but in the course of preparation of this paper I found that there was so much to say about diagnosis that it seemed best to limit myself on the present occasion to this aspect of the subject.

There is one aspect of treatment, however, that I am prompted to bring up by listening to Professor Neumann's interesting paper. Professor Neumann has made a careful comparison between the recent results in treatment of otogenic brain abscess and the results obtained by Macewen over thirty years ago, and he has sought an explanation for the fact that the results of otologists of the present day are very much worse than those of Macewen. This fact is one which has puzzled me also, and I have read through Macewen's book several times in the hope of finding some

adequate explanation. Probably there are a number of causes for this divergence, but there is one possible cause that has not, so far as I am aware, received adequate consideration. It is, I believe, the usual practice among otologists of the present day to use the mallet and gouge or chisel in clearing out the mastoid process. As far as I have been able to ascertain from his book, Macewen never used a mallet and gouge, but always opened the mastoid process with hand burrs. I would like to learn from Professor Neumann and the Members of this Section whether the more modern method, involving as it does, intermittent percussion of the skull, may not produce in the underlying brain a disturbance that tends to promote the spread of the infective process. The success of any method of treatment depends in the first instance on the ability of the brain to localize the infection. No form of operation avails if an abscess spreads rapidly and ruptures into the ventricle within a few days of its inception. But this is, fortunately, a rare event. The brain is richly endowed with connective tissue cells, in addition to its own supporting glia, and these cells are rapidly mobilized when any focus of infection occurs. That they are often successful in limiting the spread of infection is shown by histological examination of the wall of an abscess, and by the not infrequent observation of cases in which an abscess is found to be completely surrounded by a dense fibrous capsule. The barrier against the spread of infection from a recently-formed abscess into the ventricles is always rather precarious, and transmission of any force through the brain—such as might conceivably occur from the use of a mallet and gouge on the skull overlying a temporo-sphenoidal abscess—might diminish the chances of successful limitation of the abscess by natural methods. The importance of avoiding anything but the slightest injury to the abscess wall and the overlying brain is well recognized as the basis of modern methods of treating brain abscess.

In twelve of my twenty-three verified cases—just over half—the abscess had followed infection through the nose or ear. This represents fairly accurately the relative frequency of these varieties of brain abscess. For the opportunity of seeing most of these cases, I have to thank my otological colleagues at the London Hospital.

I propose first to make some general remarks about the diagnosis of brain abscess and then to consider particularly the diagnosis of the temporo-sphenoidal abscess.

Errors in the diagnosis of brain abscess.—How does it come about that so many cases are never given the benefit of surgical exploration—as, for example, seven out of twenty-three cases in my own series, and a considerable percentage of the two hundred and more cases that have come to necropsy at the London Hospital since Professor Turnbull first began keeping accurate records in the year 1908? You will, perhaps, be thinking that this is not very much the concern of the otologist, but many of the unexplored cases in the London Hospital series proved to be cases of abscess following infections of the ear and nose.

The reason for all these mistakes is that the symptomatology of brain abscess is not, as yet, clearly defined. The localizing signs of abscesses in various parts of the brain have been well established by such admirable papers as that delivered before this Section by Dr. C. P. Symonds a few years ago.[1] A temporo-sphenoidal abscess is rarely missed if it is examined for in the ways laid down by Dr. Symonds, but it is not so easy to consider the possibility of temporo-sphenoidal abscess in every case of mastoiditis, and to make the requisite full neurological examination in every case before mastoidectomy is performed. It is even more difficult to become alive to the possibility of brain abscess in a patient who has lapsed into a state of stupor after a relatively short and insignificant period of shivering, vomiting and headache. Such a case is often sent to hospital with a diagnosis of encephalitis or of cerebral thrombosis, and is admitted to the medical wards. It is clear to me from the variety of cases which I am asked to see, that my medical colleagues at the London Hospital

1 "Some points in the Diagnosis and Localization of Cerebral Abscess," by C. P. Symonds, M.D., *Proceedings*, 1927, xx, 1139 (Sect. Otol., 41).

are now fully alive to the varied symptomatology of brain abscess. But it is not many years since these cases often lay in the medical wards until they finally came to the necropsy room, where the tradition grew up among the pathologists that cases coming to them with the clinical diagnosis of cerebral thrombosis usually turned out to be cases of brain abscess!

Some mistakes in diagnosis may be assigned to neglect of ophthalmoscopy and perimetry, but it is well to emphasize the fact that many brain abscesses produce little or no papillœdema. A more frequent source of error lies in the common misconception that a brain abscess, like abscesses in other parts of the body, must produce a rise of temperature. It cannot be too strongly stressed that the temperature in a case of brain abscess that has not been operated on is usually subnormal.

In some cases the patients die because, although it may be realized that there is a brain abscess, the condition is not considered so urgent as it actually is. These, I believe, are cases in which an abscess has formed during some previous illness. A patient may have a mastoidectomy performed and the post-operative period is not entirely smooth: there is some headache and vomiting, and the temperature is not even, but shows falls to 96° and rises to 100°. However, the symptoms clear up within two or three weeks and the patient is discharged from hospital apparently well. He is walking about months later when suddenly he has violent headache, goes into coma, and dies within a few days. At necropsy an abscess is found which is surrounded by a firm capsule, that has obviously taken many weeks or even months to form. In this type of case it is probable that the abscess has formed during the disturbed convalescence from operation. The patient's resistance at that time has been good, and the abscess (call it purulent encephalitis if you wish) has become well localized and even partially absorbed; in this condition he is able to go about his work, apparently in good health. When convalescence after mastoidectomy is disturbed in this way, the patient should be kept under close observation for a long period, because these partially subsiding abscesses are as dangerous as volcanoes, and they erupt rapidly and without warning. It is not only after mastoidectomy that such an abscess may form, but also after mild attacks of mastoiditis and frontal sinusitis that go untreated.

The localizing signs of temporo-sphenoidal abscess.—I have practically nothing to add to Dr. Symonds' paper on this subject, but I would urge the importance of repeated daily perimetry in these cases. There is no investigation so valuable in the diagnosis of temporo-sphenoidal abscess. The finding of a homonymous quadrantic defect not only clinches the diagnosis of abscess, but it also gives valuable information as to the situation of the lesion. The usual type of defect is in the superior quadrants, but a homonymous inferior quadrantic defect may also be produced by a cerebral abscess secondary to mastoiditis. The discovery of this type of defect indicates clearly that it is useless to attempt to drain the abscess through the mastoidectomy wound. The exploration in such cases must be made at a higher level.

By choosing a period of temporary improvement I have been able to take on the Bjerrum screen, the fields of patients who were acutely ill with brain abscess. A portable perimeter would perhaps be better, but, whatever the method employed, it is important not to rely on confrontation tests if there is the slightest chance of obtaining more accurate records. The condition of the fields in a case of abscess of a cerebral hemisphere after mastoiditis, not only indicates the level at which the abscess should be sought, but it is also a valuable guide as to when to operate. Extension of a quadrantic hemianopia, into a complete hemianopia calls for immediate operation. If the defect is not expanding, it is safe to wait, if waiting is indicated.

There is sometimes, however, a grave difficulty about examining for field defects and the other signs that Dr. Symonds has described, and that is when the patient is so drowsy that he does not coöperate sufficiently. It is the important ocular tests,

especially tests of ocular movements and of the visual fields, that cannot be made. Much may be discovered in these cases by patient observation at frequent intervals, but, in some cases, and particularly in children, the diagnosis and localization of the abscess cannot be made with certainty by clinical methods. X-ray examination of the skull is particularly valuable in this type of case. Abscesses of the brain very often contain gas. How often, when the brain needle enters the abscess cavity, do we not first observe a little puff of offensive gas before the pus wells out? This gas will show in the skiagram and it indicates exactly the position of the abscess.

Differential diagnosis.—In this series of cases confusion in diagnosis has arisen between brain abscess and the following conditions: Encephalitis, cerebral thrombosis, cerebral aneurysm, tuberculous meningitis, purulent pachymeningitis, purulent leptomeningitis, arachnoiditis (serous meningitis), and intracranial tumour. I do not propose to go into the clinical differentiation of these groups. Indeed, this is often impossible in the present state of our knowledge, especially when the intracranial disturbance affects an individual who incidentally suffers from a chronic running ear. In many cases a diagnosis is only reached by doing an exploratory puncture of the brain. The following case may be cited as an example:—

Case I.—Meningo-encephalitis simulating brain abscess. Otitis media and chronic mastoiditis. Mastoidectomy. Exploratory puncture of the brain, with findings that excluded brain abscess. Recovery.

History.—A boy, aged 8, was admitted to the London Hospital in a semi-conscious condition on December 26, 1928. Six days before he had come home in the evening complaining of feeling tired. On the following afternoon he had complained of frontal headache, and had vomited several times without warning. The next day he gradually became unconscious.

Past History.—There were several points that were likely to be of importance in the diagnosis. He had had bronchitis and pneumonia at the age of five months, and from that time onwards he had had much chest trouble, and for four years before admission had attended a tuberculosis dispensary. At the age of two he had had discharge from both ears, and this had continued for twelve months. A week before the onset of the present illness the left ear had begun to discharge again, and then the right ear had begun to discharge. Those who saw him at the time of his admission were thus prepared to consider a diagnosis of tuberculous meningitis or of brain abscess.

Examination.—The boy was semi-conscious and had some head retraction. Temperature 100·6°. Pulse-rate 112. Internal squint of the left eye. Slight right facial weakness. Bilateral extensor plantar responses. On clinical examination there was no evidence of any disease of the lungs. Examination of the right ear showed no abnormality. There was profuse offensive discharge from the left ear, and the left drum was perforated anteriorly and elsewhere reddened. Lumbar puncture showed clear fluid under raised pressure. The fluid contained twenty white cells per cm., 0·035% protein, no tubercle bacilli.

Progress.—After admission the patient became more unconscious and gradually developed a right hemiparesis. The pulse-rate became slower.

First Operation.—December 28, 1928. An incomplete left mastoidectomy was performed. In the region of the antrum there was a polypoid mass of soft, red tissue, and this contained a little mucopurulent fluid, which was subsequently shown to be sterile. No pus was found. The dura of the middle fossa was exposed over an area about one-half inch in diameter. It did not bulge unduly. Histological examination of the fragments of bone removed at operation showed fibrosis of the marrow.

Post-operative Notes.—On the following day there was slight papilœdema and the right hemiparesis was rather more pronounced. The boy continued in a semi-conscious condition, and the pulse-rate slowed to 44, the respiration rate to 16.

Second Operation.—January 2, 1929. Through a separate incision above and in front of the mastoidectomy incision, the left temporal lobe was tapped for abscess, but none was found. When the subarachnoid space was opened a free escape of cerebrospinal fluid occurred. This observation was considered to be strongly against the presence of an abscess. In addition the small fragment of brain tissue that was removed in the brain needle after tapping of the temporal lobe looked to the naked eye normal—quite unlike the soft, glistening

yellowish white tissue that is recovered when the needle has passed near an abscess. It was felt as a result of the operation that abscess of the left temporal lobe could be excluded.

Subsequent Course.—Two days after this operation the boy began to recover consciousness, and from that time onward his condition steadily improved. The right hemiparesis cleared up almost completely within a week, though traces of it could still be detected for a further three weeks. The incision for puncture of the brain healed by first intention, notwithstanding its proximity to an open mastoidectomy wound. The boy recovered completely and was discharged from hospital on February 12, 1929. He was last seen on January 9, 1930, when it was found that his complete recovery had been maintained and that he was entirely free from symptoms of any sort.

Comment.—No more definite diagnosis than one of meningo-encephalitis can be made in this case. The patient would probably have recovered without either of these operations, but, in view of the difficulty of excluding brain abscess on clinical grounds, it would be hazardous at the present day not to explore such a case when explorations of the type described above can be shown to be free from danger. There would be risk of purulent leptomeningitis if such intracranial explorations were made through an open mastoidectomy wound, but no such risk attends a small exploratory incision through an adjacent region, provided the field of operation is carefully prepared. I use gutta-percha tissue for sealing off the mastoidectomy wound from the field of exploration. It is laid over the mastoid wound and it sticks firmly to the shaved scalp when a hot moist swab is pressed against it for a second or two. The brain is then explored through a small perforation, or a series of perforations, above the level of the zygomatic process. This method should be used in all cases in which, after mastoidectomy, a temporo-sphenoidal abscess is suspected, but is not clinically certain. Should an abscess be encountered in such a position that it cannot be satisfactorily drained through the same perforation, then it is easy, either at once or, preferably, at a subsequent stage, to drain the abscess through the mastoidectomy incision or through an appropriately placed burr hole. By this method the risk of producing meningitis is avoided, and one is not over-reluctant to explore the brain in a doubtful case.

The evidence of a negative character that can be obtained by this method of exploratory puncture is not limited to failure to encounter pus within the brain. There are several other signs which strongly suggest that there is no cerebral abscess.

(1) The finding of free pus in the subdural space, after a tiny incision has been made in the dura, practically excludes the presence of an intracerebral abscess. It indicates rather that the condition is one of purulent pachymeningitis, of which more will be said later.

(2) An abundant flow of cerebrospinal fluid from the subarachnoid space, as in the case of meningo-encephalitis just described, is also strongly against the presence of an abscess in the underlying brain. It is a common observation, in cases both of abscess and of tumour of the brain, that the surface of the brain over the lesion is swollen and that its subarachnoid space is almost empty of cerebrospinal fluid.

(3) If during exploration for a temporo-sphenoidal abscess the temporal horn of the lateral ventricle is entered by the brain needle and more than 1 c.c. of cerebrospinal fluid is obtained from it, then it is almost certain that there is no abscess in the temporal lobe. Examination of post-mortem specimens shows that the temporal horn of the lateral ventricle is almost completely obliterated when there is an abscess in the corresponding lobe of the brain.

(4) Additional evidence may sometimes be obtained by examining the small fragment of brain tissue that is found in the needle after puncture of the brain. It is sometimes possible to distinguish by naked-eye, fragments of brain tissue that are œdematous from those that are normal. The œdematous brain tissue is very soft, yellowish-white and unduly glistening, in contrast to the moderately soft, pure white, slightly glistening appearance of normal white matter. If the tissue obtained from the brain needle is obviously normal to the naked eye it is unlikely that there is an abscess near the site of puncture, for an abscess of the brain is always surrounded by a very wide area of œdema.

Exploratory puncture of the brain for abscess is thus not merely a matter of

finding pus, or of failing to find it. There are numerous observations to be made, and from them one can get a fairly accurate idea, even at the stage when the needle has been passed only once into the substance of the brain, whether subsequent explorations are likely to reveal an abscess. A small abscess may be difficult to find, even though the operator is fully armed with an exact clinical diagnosis and with an accurate knowledge of the topography of the brain. But skill and care in use of the brain needle will usually lead him to the abscess, though it may be necessary for him to complete his exploratory punctures at a second operation. As a rule, it is advisable not to do too much at one session. When the abscess is entered, it is important to observe whether there is any resistance to the passage of the needle, for on the thickness and firmness of the abscess wall depends to a considerable extent the variety of surgical treatment that should be undertaken.

Purulent pachymeningitis.—Two of the conditions that I have mentioned as simulating brain abscess deserve more consideration. The first of these is purulent pachymeningitis. This condition may sometimes follow mastoiditis, and it is characterized by the pressure of free pus in the subdural space. Its importance in connection with the present subject is that it may produce symptoms and focal signs identical with those of temporo-sphenoidal abscess—aphasia, hemiparesis, hemianopia, headache, papillœdema, bradycardia, and so forth. The fact that the temperature is high, instead of being low as in abscess, is scarcely a distinction on which differential diagnosis can be based, and the only certain way in which the diagnosis can be made is by careful observation at operation. Even at operation the condition may be overlooked, for it is the uppermost part of the subdural space that is opened, whereas the pus, being free, tends to sink to the lowest level of the space. Whenever purulent pachymeningitis is suspected, the head should be tilted after a small opening has been made in the dura, so that any pus that is present in the subdural space may gravitate towards the opening. If pus is found at this stage of the operation it is most unlikely that there is also an intra-cerebral abscess. This condition is not common. Mr. Patterson and I have had two examples in recent years, one associated with a large cholesteatoma, and the other after acute mastoiditis and osteomyelitis of the skull.

Arachnoiditis (*serous meningitis*).—The second condition that I wish to describe more fully is that known as arachnoiditis, or serous meningitis. This condition simulates cerebellar abscess rather than temporo-sphenoidal abscess. It comes on after acute mastoiditis and chronic mastoiditis, especially when the labyrinth has been infected. The main clinical feature is papillœdema, often of extreme degree, and the headache and other general symptoms may be so slight that the condition is not recognized until sight is seriously impaired. The following is an example, and I think it is so important that I will describe it in detail:

Case II.—Left mastoiditis. Two mastoid operations. Lateral sinus thrombosis. Arachnoiditis. Blindness.

J. K., a boy, aged 5 years, first came under my notice on October 29, 1929. His illness had begun in January, 1929, when a sore throat had developed, followed two weeks later by left-sided earache. On January 31, 1929, an incomplete mastoidectomy was performed on the left side. The lateral sinus and a large area of dura were exposed, but were not opened. During convalescence there was slight fever and occasional vomiting, but the boy was discharged from hospital five weeks after the operation, quite free from symptoms.

He remained well until his tonsils were removed on June 27, 1929. Within three days the left ear began to ache and to discharge, and vomiting also occurred. On June 30, 1929, lumbar puncture revealed clear fluid under pressure, and subsequent analysis of the fluid showed no white cells, protein 0·02%, sterile cultures. Immediately after the lumbar puncture, the left mastoid was reopened, revealing much pus and granulation tissue over the dura and the lateral sinus. A needle was passed into the left temporal lobe in four directions, but no pus was found. The lateral sinus was then opened and was found to contain no clot. The wound was packed tightly with gauze. At the first dressing two days later there was sharp hæmorrhage, which was stopped by further tight packing of the wound.

The post-operative course was this time rather stormy: the child was very drowsy for several days, and suffered from severe headaches and frequent vomiting. However, he was sufficiently recovered to leave hospital on August 1, 1929, one month after operation. Headache recurred during the first two weeks in August, but then disappeared. The first indication of any failure of vision occurred on August 21, when the mother noticed that the boy tripped over curb-stones. In September he began to fall over objects and to feel his way about as he walked. Ophthalmological examination was carried out on October 14, apparently for the first time. The boy could not count fingers. The discs showed severe papillœdema and optic atrophy, and there was exudate around each macular region. Two teeth were removed on October 15.

Condition on examination, October 29, 1929: Left eye completely blind; right eye perception of light only. No reaction of left pupil to direct light, moderate reaction of right pupil. Severe secondary optic atrophy, with 2·5 D. swelling on right side, but no swelling on left side. Exudate around each macula. No other physical signs except generalized hypotonia and diminished tendon-jerks, and slight enlargement of the head. The boy was cheerful and, apart from his blindness, free from symptoms. Lumbar puncture: pressure 230 mm. In unilateral Queckenstedt tests, the usual rise of pressure occurred when the right jugular vein was compressed, but there was no rise of pressure when the left jugular vein was compressed. In this case a thrombosis of the left lateral sinus had thus evidently developed since the lateral sinus was opened at the second operation on the left mastoid process. The cerebrospinal fluid was clear. It contained one cell per c.mm.; 0·02% protein. Cultures sterile.

Comment.—As far as it is possible to judge from the available evidence, this appears to be a case of the so-called arachnoiditis or serous meningitis. The pathology of "arachnoiditis" is at present quite obscure. In the only case of the condition in which I have been able to make a histological examination, the arachnoidea showed no evidence of inflammation, and I think the term "arachnoiditis" is probably an incorrect one. All that we know about the pathology of the condition is that there is hydrocephalus, but whether this is due to increased formation of cerebrospinal fluid, or to decreased absorption of cerebrospinal fluid, or to a combination of both these factors, is at present unknown. The condition follows middle-ear and mastoid infection, but it may also occur without any demonstrable focus of infection, either in the ears or elsewhere.

This is the first case of arachnoiditis that I have seen in which thrombosis of the lateral sinus has been demonstrated. Such an occurrence would tend to interfere with the absorption of cerebrospinal fluid, and thus to aggravate the rise of intracranial pressure. This may account for the extreme degree of destruction of the optic discs by papillœdema and atrophy. The case is typical in the absence of severe headaches, except for a short period at the beginning of the illness. This absence of headache is not to be explained solely by the fact that in this instance the patient was a child whose head could expand in response to increased intracranial pressure, for absence of severe headache is a noticeable feature also in adults.

The condition tends to recover spontaneously. I have seen two cases—both in children—in which perfect recovery occurred without any treatment at all. In one of these the papillœdema reached 4 diopters before it began to subside; in the other the swelling was never higher than 2 diopters. In both cases vision eventually became normal. In other cases, however, although all the symptoms clear up, they do not do so until vision is seriously affected. The little boy whose case I have just described is now permanently blind, though perfectly well in other ways. From my experiences with this condition I believe that a cerebellar decompression, performed before the destruction of the optic discs has become far advanced, is a sure method of preserving vision. It should be carried out in every case in which the papillœdema becomes at all severe, or in which there is any sign of lowering of visual acuity. In one case of this condition in which the patient was practically blind, vision returned in a most dramatic manner after a cerebellar decompression had been done. The operation, however, cannot be expected to effect a return of vision after blindness has been present for some weeks. Nevertheless, I would have been prepared to operate

in the case described, in the faint hope of getting some recovery of sight in the right eye, if it had not been that my colleagues expressed a desire to treat the boy by repeated lumbar punctures. This course of treatment was carried out. That no return of vision occurred in a case so advanced as this cannot be counted against the method employed, but the intracranial pressure, as measured by the spinal manometer, rose instead of falling, and this suggests that treatment by repeated lumbar punctures is not a satisfactory method in severe cases.

The pressing need in this type of case is early diagnosis and, as the neurological signs are scanty, this depends on the employment of ophthalmoscopy in all cases of ear disease that show any signs of intracranial disturbance. The case described shows how it is possible for the condition to be overlooked through neglect of ophthalmoscopy. Even if the headaches and vomiting are severe in the early stages, they are always very much better at the time when the patient is going blind. Treatment, therefore, should be entirely governed by the state of the eyes.

This condition of "arachnoiditis" comprises a part of that mixed group of cases known as "pseudo-brain abscess." Mild cases of purulent pachymeningitis are probably also included in the same category.

Conclusion.—Finally, diagnosis must be carried on during treatment. Operation should often be carried out in stages. There is a right and wrong time to operate in each case, and this can only be decided after accurate observation of the clinical condition. Additional symptoms often arise in the first few days after drainage of the abscess, due to œdema, to a slight degree of meningitis, or to faulty drainage of the abscess. Is the tube to be moved, or is it wiser to leave it undisturbed? These and many other problems depend for their solution on careful day-to-day examinations. In my experience there is no disease in which repeated clinical investigations are so important as in abscess of the brain.

Temporal Lobe Abscess. (Notes on seventeen consecutive cases operated upon at the Royal Infirmary, Edinburgh, from 1908 to 1929.)

By J. S. Fraser, F.R.C.S.Ed., and B. B. Blomfield, F.R.C.S.Ed.

In the first case, that of a female child aged five years, suffering from chronic middle-ear suppuration on the left side, the temporal lobe abscess was complicated by a peri-sinus abscess, labyrinthitis and meningitis, which were all present on admission. The patient died.

The second case was that of a male, aged 17 years, suffering from chronic middle-ear suppuration on both sides. The symptoms of brain abscess only came on after the radical mastoid operation had been performed on the left side; a skin graft had been applied. It is noteworthy that the posterior wound healed well, and that the radical mastoid cavity and skin graft appeared satisfactory. Elevated temperature, headache and vomiting were followed by subnormal temperature and dilatation of the pupil on the same side, and twitchings of the opposite side of the body. After drainage there was retention of pus in the abscess; this was relieved by improving the drainage. The patient's mental condition changed with the development of the abscess; from a shy, quiet youth he became violent and even maniacal. Recovery took place.

In the third case, that of a female aged 11 years, suffering from chronic suppurative otitis media on both sides, with temporal lobe abscess on the left side, the drainage of the abscess was evidently insufficient, although a piece of brain was removed at the time of operation. Leakage occurred into the lateral ventricle, and thereafter meningitis developed. Death occurred only seventeen days after the onset of discharge of purulent cerebrospinal fluid from the abscess.

In the fourth case, in a male aged 29 years, with chronic suppurative otitis media

on the right side, there was some doubt as to whether the abscess was present on admission. There had been a rigor before admission, but no headache. Operation showed an extradural abscess on the right side. There was no dilatation of the pupil on this side to guide one. When the brain abscess was opened, cerebrospinal fluid escaped along with the pus, so that it was practically certain that the abscess had already ruptured into the lateral ventricle. The patient died.

The fifth case was in a male, aged 23 years, suffering from chronic suppurative otitis media (bilateral) with temporal lobe access on the right side. Owing to the presence of meningitis and of repeated rigors, and the fact that the lesion was on the right side, the abscess was not diagnosed. The patient had been treated by his own doctor as a case of biliousness! Autopsy showed that the abscess had ruptured into the lateral ventricle, and it is probable that leakage into the lateral ventricle was present on admission. The patient died. It is interesting that an uncle of this patient had also died of brain abscess.

The sixth case, in a male aged 27 years, was one of acute suppurative otitis media on the right side. Before admission there had been sudden stoppage of the discharge, followed by severe headache and vomiting, with irritability and also sudden loss of speech. The lesion was on the right side and the patient was right-handed. There was no dilatation of the pupil, but there was tenderness on tapping over the temporal lobe. It is interesting to note that at the operation, though the case was an acute one, the mastoid was dense and an extradural abscess was present in the middle fossa. The temporal lobe was not investigated at this stage. The patient did well till he was allowed to get up ten days after the first operation. Immediately after this there appears to have been a rupture of the abscess into the lateral ventricle, because when the abscess was opened pus and cerebrospinal fluid at once escaped. The patient died.

The seventh case in a female, aged 43, was again an acute one (left side). Owing to the intense frontal headache the patient was sent in by her doctor as a case of acute frontal sinus suppuration. There was, however, no pus in the nose. The first operation was performed by another surgeon, but, unfortunately, though the sinus was exposed, the dura of the middle fossa was not uncovered. The frontal pain persisted and a herpetic eruption appeared on the left side of the face. The onset of drowsiness, subnormal temperature and optic aphasia, with weakness of the grasp on the contralateral side, led to the diagnosis of temporal lobe abscess. Drainage in this case was insufficient until a comparatively large piece of dura and brain were cut away; after that the patient did well.

The eighth case in a female, aged 55, was also an acute one (left side), but it is interesting to note that the patient had had previous attacks of acute middle-ear suppuration in 1917 and 1921 before the last attack in 1922. Further, the perforation of the drumhead was an anterior one. Radiograms showed a dense mastoid on the left (diseased) side and a cellular mastoid on the right. In addition to the general symptoms of brain abscess the patient had an epileptiform attack. There was no paresis of the third nerve and no dilatation of the pupil. A large extradural abscess was opened at the first operation. In spite of the opening of this abscess, vomiting continued, accompanied by subnormal temperature and sensory aphasia. A second operation was performed, evacuating the temporal lobe abscess. The patient recovered.

In the ninth case a male, aged 21, suffered from chronic suppurative otitis media on the right side. He had been operated upon by one of us (J.S.F.) for septic thrombosis of the lateral sinus on the right side, with a metastatic abscess in the ilium, five years before his second admission. Though this patient was left-handed, the grasp of his right hand was stronger than that of the left. A temporal lobe abscess on the right side and purulent meningitis were diagnosed. The abscess was opened, but later ruptured into the lateral ventricle. The patient died.

The tenth case, in a male, aged 19, was one of bilateral chronic suppurative otitis media, with abscess of the right temporal lobe. Most of the general symptoms of brain abscess, except vomiting, were present. The patient constantly clawed the right side of his head. During the operation the respiration stopped for a time. An extradural abscess was present in the middle fossa. The patient recovered.

The eleventh case, in a female, aged 9, was also due to chronic middle-ear suppuration (right). A peri-sinus abscess was present. The temporal lobe abscess was opened at the first operation, but a hernia cerebri formed. Rigors and hectic temperature developed after operation, and therefore the sigmoid sinus was opened, but no clot was found. The patient died. Autopsy revealed meningitis, with pus in the lateral venticle.

The twelfth case was in a female, aged 3 years, with chronic suppurative otitis media (left). There were right-sided convulsions and the right pupil was large. Later there was paralysis of the right side. A temporal lobe abscess was evacuated at the first operation. Later, cerebrospinal fluid came away from the brain abscess and death followed from meningitis.

In the thirteenth case, the patient, a male, aged 23, had chronic suppurative otitis media (left). He had headache and pain in the back, but no vomiting and no optic aphasia. The dura of the middle fossa was greyish-green in colour. The abscess was not opened at the first operation. Later, optic aphasia was noted and nystagmus to both sides. The brain abscess was then opened and the patient recovered.

The fourteenth case in a male patient, aged 54, was one of chronic suppurative otitis media (right). There was severe pain in the right side of the head. A radical mastoid operation was performed and a skin graft applied. Later the patient had temporal headache and vomiting. The temporal lobe was explored with a negative result. Meningitis developed. At the second operation an acute temporal lobe abscess, situated higher up and further forward than usual, was evacuated. The patient died. It is probable that the abscess had only supervened after the radical operation.

In the fifteenth case, the patient, a male, aged 28, suffered from chronic middle-ear suppuration (left) with giddiness. He refused operation. Seven years later he was admitted as an urgent case, with mastoid tenderness and a history of vomiting, headache, rigor and drowsiness. At operation an extradural abscess was found in the middle fossa. The temporal lobe abscess was opened at the first operation. Lumbar puncture evacuated purulent cerebrospinal fluid. The drainage of the abscess was not sufficient till a large piece of dura mater and brain had been removed. After that the patient did well and recovered.

In the sixteenth case a male patient, aged 21, with chronic suppurative otitis media (right) was admitted in a moribund condition. The temperature was 103°; Kernig's sign was present, the neck was stiff, and the right pupil dilated. There was paresis of the left arm. At operation a peri-sinus abscess was found. The cerebellum was therefore investigated but with negative result. At the same operation a large temporal lobe abscess was evacuated. Later, rigors occurred, and therefore the lateral sinus was investigated but no clot was found. The patient died, and at the autopsy numerous abscesses were noted in the brain; meningitis was also present.

The seventeenth case in a female, aged 56, was one of chronic suppurative otitis media (left). The left Gasserian ganglion had been removed by a general surgeon, on account of trigeminal neuralgia, three years before the patient's admission to the Ear and Throat Department. The case record stated that the wound became infected, but the patient recovered. On admission to our department the patient had pain in the left mastoid and occipital regions. The left ear was quite deaf. At the operation a cavity was found above the cochlea, leading to the dura mater

of the middle fossa (this was, of course, due to the previous Gasserian ganglion operation). Later there was severe pain in the left temporal region and epileptiform fits occurred followed by coma and neck stiffness. The cerebrospinal fluid was cloudy and contained streptococci. Translabyrinthine drainage was performed and the left temporal lobe investigated, but with negative result. Two days later a large temporal lobe abscess was opened, but the patient died. Post-mortem examination showed purulent meningitis.

Summary.

Of the seventeen patients, ten were males and seven females.

Age.—Thirteen were under 30 years. The youngest was aged 3 years, and the oldest 55.

Side.—The right side was affected in nine and the left side in eight cases. In only three cases was the otitis media acute, whereas in fourteen it was chronic.

Examination of the inner ear.—The labyrinth was found to be functionating in thirteen cases and inactive in two; in the remaining two the inner ear could not be tested.

General symptoms.—Headache was a marked feature in fifteen and vomiting in thirteen cases. The temperature on admission is interesting, for while only seven cases showed subnormal temperature, fever was present in eight and in two the temperature was normal. The pulse was slow in five cases and rapid in eight; in the remaining four it was normal. Ten of the patients were drowsy or even comatose on admission, making neurological examination difficult and in some impossible. In four cases, slight delirium alternated with drowsiness.

Localizing symptoms.—As is well known, the localizing symptoms in cases of temporal lobe abscess are often far from definite, but tenderness on percussion of the temporal region was noted in four, dilatation of the pupil on the affected side in three and sensory aphasia in only four of the eight patients with left-sided abscess. One patient with a right-sided temporal lobe abscess suffered from sudden loss of speech. In two cases there was twitching of limbs, or paresis, on the opposite side; one case had slight paresis of the opposite side of the face; three showed contractions of the face on the same side; three patients had epileptiform or convulsive attacks. Of the eight cases in which the eyes were examined by an ophthalmologist, double optic neuritis was noted in two, congestion and swelling of the disc on the affected side in one, and slight œdema of both discs, but most marked on the diseased side in one.

Findings at operation.—An extradural abscess was found in the middle cranial fossa in six; the dura mater showed granulations in one; the dura mater of the middle fossa was tense and bulging in six; the dura mater was greyish-green and necrotic in two.

Drainage.—A cigarette drain was first employed in five cases; iodoform gauze drainage in six; tubes were employed in two cases; in one case the iodoform gauze drainage was found to be unsatisfactory till a fairly large piece of dura and brain was removed.

Complications.—In six cases meningitis was present on admission and, of these, one patient recovered and five died. In other six cases meningitis developed after the first operation and all these patients died. In the remaining five cases there was no meningitis and all recovered.

Results.—Six patients recovered and eleven died.

Pathology of Adjacent Brain Abscess.

By E. Miles Atkinson, F.R.C.S.

I PROPOSE to confine my attention entirely to adjacent brain abscess of aural origin. The essential point at issue is the route of passage of infection into the brain tissue. In order to get a clear conception of how a brain abscess originates, it is necessary to bear in mind certain points of anatomy and physiology.

(1) In the first place, the vascular supply of the brain. This comes from two sources, central and cortical vessels, branches of the circle of Willis. The central vessels pass at the base of the brain directly into its substance and then outwards through the white matter towards, but not as far as, the cortex. The cortical vessels supply the grey matter of the cortex by means of a large number of small vessels,

and also a layer of white matter immediately subjacent to the cortex, by a smaller number of larger vessels, these latter in the white matter passing towards, but not anastomosing with, the terminal branches of the central vessels—not anastomosing with them, because the cerebral arteries are end-arteries. Therefore between these two groups of vessels is a zone of white matter with a poor blood supply which we may call the avascular zone. In the cerebrum this zone is situated just below the grey matter of the cortex, but in the cerebellum, by reason of the greater convolution, it likewise becomes folded and is found therefore to lie down the centre of each folium. I will return to this point later in speaking of the direction of spread of these abscesses.

(2) The second point to be borne in mind is that whenever a cortical vessel passes from the surface down into a fissure, it takes with it a prolongation of the subarachnoid space in which the cerebrospinal fluid circulates, so that both arteries and veins as they lie together in a fissure, are surrounded by the perivascular or Virchow-Robin space.

(3) The third point to note is that the cerebrospinal fluid normally flows outwards to the surface in these perivascular spaces, but Weed has shown that any increase in pressure in the subarachnoid space will cause a cessation—and then a reversal—of the flow.

Now we are in a position to sketch briefly the mode of onset of a brain abscess. The infection in the ear passes through the bone of the skull and reaches the dura. If the organism is of high virulence, the dura fails to withstand the passage, an area of necrosis appears, and the subarachnoid space is infected before there is time for adhesions to form—a generalized meningitis has resulted. If, however, the degree of virulence is less and the tissue resistance greater, an area of localized meningitis occurs, and subjacent to the point of attack adhesions form to wall off the main part of the subarachnoid space from infection, just as adhesions form round an area of infection in the peritoneal cavity. This area of localized meningitis—the point of entry of the infection—is signalized by a thickening of the dura mater, more or less obvious according to the extent and nature of the adhesions beneath. Such an area of thickened dura is to be found in the great majority of cases. In those I have been able to examine, it was found in eight out of fourteen (57%) at operation and in thirteen out of fifteen (87%) museum or post-mortem specimens. Most authorities are agreed on this. It is therefore worth while at operation to make an adequate exposure of the dura and to hunt carefully for such a point of thickening, for, if found, it will lead one straight to the underlying abscess, and, further, drainage through such an area surrounded by adhesions, is more free from risk.

This area of localized meningitis involves an interference with the cerebrospinal fluid circulation in its neighbourhood—a stoppage or reversal of flow—and the infecting organisms are thus provided with an easy passage along the perivascular sheaths into the brain substance. This is the first, and in my experience the most common, of the three possible routes of entry; it occurs in 80% of cases.

The organisms having reached the brain by way of the perivascular space, proceed with their pus-forming activities at the point of least resistance, that is, in the avascular zone beneath the cortex. This slide, [shown] from a cerebellar case, illustrates some of the points, the extremely localized nature of the attack upon the brain (one lobule) and the intense perivasculitis. The next one [slide shown] from the same case shows a small abscess situated accurately in the centre of the white core of a lobule, in the position which we have been led to expect. It is next to impossible to obtain a similar specimen from a temporal lobe case, because the abscess in extending, does so circumferentially and destroys the site of origin, but by analogy one may presume, without undue stretching of the imagination, that an exactly similar process occurs there also.

The venous route is much less common (12%), although Eagleton claims that it is

the usual one. It arises by the coincidence of a pial vein happening to lie across the point of infection, when thrombosis occurs and spreads backwards into the brain substance. It is most liable to happen in connection with a lateral sinus thrombosis. The histological appearances are quite distinct. Here we see a vein filled with breaking-down clot [slide shown], the accompanying artery being patent, and the vessels in the fissure, which we cannot see here, show similar appearances. The degree of surrounding perivasculitis is slight, due merely to the infected thrombus. The area of brain drained by this vessel begins to degenerate owing to interference with its blood supply, and again the part least able to stand up to this is that in which the blood supply is least good, i.e., the avascular zone. Another slide from the same case shows an abscess, again occupying the position of the white core of a folium, with several thrombosed veins on the surface. The cases in this group are more likely to show multiple abscesses and to cause death by meningitis, as they tend to spread towards the surface along the course of the thrombosis and to rupture into the subarachnoid space.

The third route requires but brief mention. If an artery is caught in the area of infection, it may become partially or completely occluded by a thrombus, and from this thrombus a small embolus can be shot off. This slide shows a thrombosed artery in the wall of an abscess which was probably occasioned by the thrombosis [slide shown]. The onset of such abscesses due to embolism is typically apoplectiform, and their situation is not necessarily adjacent to the focus of infection.

Spread.—Once an abscess has started in the brain substance it tends to increase in size in a fairly constant manner. The grey matter of the cortex is more resistant, by reason of its better blood supply, than the central white matter, and extension therefore occurs at the expense of the latter. In the cerebrum the abscess tends to spread along the course of the vessels towards the lateral ventricle, perforation into which is a not unusual terminal event. Exploration, therefore, should be towards the ventricle, and the operator must have a very clear conception of the position of this structure inside the brain.

In the cerebellum extension occurs again at the expense of the white matter, but, in this case, of the white matter forming the centre of a lobule. This central core is gradually destroyed, inwards as far as the central mass of white matter and backwards through the lobule. The central white mass is respected until quite late in the course of the disease. In other words, the ordinary cerebellar abscess, as distinct from the commonly multiple abscesses secondary to a sinus thrombosis, is a single abscess and is nearly always confined to one lobule. That lobule is usually the one immediately below, and sometimes the one immediately above, the great horizontal fissure, which is covered by the horizontal part of the lateral sinus. One of these two lobules is the most likely place in which to find a cerebellar abscess in default of any guiding sign, such as a patch of dural thickening. It is, moreover, quite frequently a smooth-walled regular cavity, and not, as often stated, full of nooks and crannies and hence difficult to drain.

Capsule.—A few words about the capsule of the abscess. I use the word because it is part of the usual brain abscess jargon, but it is a complete misnomer. All we mean by it is the wall of the abscess cavity, and it differs in no essential respect from the wall of an abscess in any other part of the body; it is simply the expression of the attempt on the part of the body to wall off and limit the spread of an infection. It occurs more slowly and with more difficulty in the brain, because the neuroglia, being a specialized connective tissue, does not enter into reparative and protective processes with the same zeal as connective tissue in other parts. Thus we have, with a very virulent infection, no evidence of a protective mechanism, but simply an acute spreading encephalitis similar to an acute spreading cellulitis in the arm. At the other end of the scale we have a very chronic abscess with a thick wall, and between the two, all possible gradations. The point of practical importance is

that the greater the resistance, the more obvious the so-called capsule will be, the greater will be the chance of recovery from operation, but the fewer and more equivocal are the signs. The cases prolific of signs, general and localizing, are those which approach nearest to the acute spreading encephalitis and are the ones of worst prognosis. I believe that if we are to diminish the mortality of this condition we must be prepared to explore the brain on much less evidence than we usually demand at present, bearing in mind that brain exploration involves little risk, while on the other hand, failure to find an abscess which is present in the brain, of necessity involves the death of the patient.

CASES.

Actinomycosis of Brain.—W. H. JEWELL, M.D.

E. M., spinster, clerk, aged 29.

History.—Onset: Sore throat and stiffness of jaw five weeks before admission to hospital. Several carious teeth extracted three weeks previously. Owing to the patient becoming collapsed remainder of teeth could not be removed. Vomiting lasting three days, fourteen days before admission. Frontal and occipital headache ten days; photophobia and pain in eyes, seven days. So severe and fixed was the brow pain that the case was sent to hospital as one of frontal sinus trouble.

On admission.—Patient apathetic, and cerebration slow, complaining chiefly of brow pain. Pupils unequal; active to light, the left dilating more than right. No evidence of any infiltration in mouth, jaws or neck. Temperature 100·6°; pulse 88; respiration 24. Ten days later anæsthesia of left side supervened, followed by paresis of leg, arm and lower half of face, the orbicularis palpebrarum escaping. Babinski, left.

Lumbar puncture.—Small excess of cells, some of these being polymorphonuclears—no organisms; no excess of globulin; glucose content slightly subnormal; chlorides 0·78%.

The skull was trephined over the right Rolandic area and an extradural abscess, and also a larger subdural abscess, were evacuated and drained with split rubber tubes. Dr. Southgate examined the pus and reported the presence of the ray fungus.

Following the operation the patient's condition greatly improved; the headache disappeared, temperature became normal, and the mentality almost natural, but the paralysis had only slightly diminished. Fourteen days afterwards the patient began to go downhill, and she died seventeen days after the operation.

[Slide and brain shown.]

Postscript.—It has been ascertained that the patient had worked in premises adjoining a seed warehouse from which seeds were frequently blown in, but she denied ever having had any in her mouth. (W. H. J.)

Ossiculectomy for Vertigo, due to Attic Cholesteatoma. Recovery. (Previously shown November 5, 1927).[1]—Sir JAMES DUNDAS-GRANT, K.B.E., M.D.

A young man, first seen September 15, 1926. Suppuration in the ear since childhood. Headache and especially giddiness (had been in bed for six weeks with vomiting and vertigo). Cholesteatomatous masses pent up by ossicles. Ossiculectomy, May, 1927. Free from vertigo ever since. Hearing better than before.

A Mastoid Process shown by X-rays to be Abnormal, but not found to be so Clinically.—C. HAMBLEN THOMAS, F.R.C.S.

M. A., a girl, aged 11. *History*: Earache in both ears for two years intermittently, but no aural discharge, no headache, giddiness, or vomiting. When examined, complained of pain in mastoid regions, especially on left side.

Condition on admission.—Pale-faced, but nothing found wrong in general

[1] See *Proceedings*, xxi, 1928, 392 (Sect. Otol., 6).