BRAIN ABSCESS IN RELATION TO DISEASES OF THE EAR, NOSE AND THROAT

by

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IN A SERIES of 110 consecutive cases of brain abscess treated in the Nuffield Department of Surgery since 1938 there were 56 due to paranasal sinus and mastoid infections (Table I). Thus diseases with which the ear, nose and throat surgeon are concerned accounted for half of our cases and this experience is in general agreement with other published reports.

TABLE I

SOURCE OF INFECTION IN 110 CASES OF BRAIN ABSCESS

	No. of Cases	Died	Percentage Mortality
Frontal Sinusitis	10	2	20%
Mastoiditis	46	17	37%
Cerebellar	18	8	44%
Temporal Lobe	28	9	32%
Chest Infections (bronchiectasisempyema, lung abscess)	21	13	61%
Metastatic from other sources, e.g., infective endocarditis, boils, carbuncles, &c.	15	3	20%
Direct implantation, e.g., penetrating wounds, infected compound fractures, &c.	10	3	30%
Infection of face, scalp and facial bones ..	8	2	25%
TOTAL	110	40	36%

It will be seen that the mortality is considerable, but if the material is divided into pre-penicillin and post-penicillin groups, the mortality rate is found to have dropped from 46 per cent. to 29 per cent. since penicillin has been freely available (Table II).

TABLE II

ANNUAL INCIDENCE AND MORTALITY OF BRAIN ABSCESS
110 CASES—1938-1949

	Before Penicillin (1938-1943)	After Penicillin (1943-1949)
Annual incidence	8	12
Total number of cases	48	62
Mortality	46%	29%

It will also be seen that the annual incidence of brain abscess in our department has been rather higher in the penicillin era than before, doubtless because many patients' lives are saved by penicillin in the acute stage of a primary infection and they live to develop a brain abscess. But despite continuing improvements in treatment and results, the development of a brain abscess cannot be regarded as anything but a serious complication.

Although infection of the mastoid and paranasal sinuses may cause other intracranial complications such as extradural abscess, subdural abscess, meningitis, and thrombosis of cerebral veins and venous sinuses, this paper deals only with brain abscess, and for the sake of brevity it is convenient that these abscesses generally occur in only three situations: at the frontal pole, in the middle of the temporal lobe, and in the lateral lobe of the cerebellum. In each case, the infection spreads directly from the skull to the adjacent brain, and it is in so doing that the various planes mentioned above may be infected, leading to an extradural or subdural abscess or to a diffuse leptomeningitis. In some cases the brain abscess may be preceded by one or more of these infections. Indeed a short bout of meningitis occurs quite commonly as the infection is traversing the subarachnoid space. With the common use of sulphonamides and antibiotics it may almost go unnoticed, but in some cases the meningitis may be very severe, and the symptoms and signs of a brain abscess only become apparent after the meningeal infection has been brought under control.

Once the infection reaches the brain there is hyperaemia, localised œdema, and later necrosis and liquefaction to form pus. This may all take place within 2-3 days of the onset of the infection. The œdema may be considerable in the early stages, so much so that the infected focus acts like a rapidly growing neoplasm and may cause death simply from increased intracranial pressure. At necropsy, the brain is swollen, tense and pale. When it is cut, there may be a small abscess cavity containing 3-4 ccs. of pus, but more important the affected lobe and often the whole hemisphere is swollen so that the temporal lobes are impacted in the incisura tentorii, or the cerebellar tonsils are jammed in the foramen magnum, obstructing the circulation of the cerebrospinal fluid and leading to a further rise in pressure. These are the common findings in what is called suppurative encephalitis.

In other cases, the abscess develops more slowly and, as pus accumulates, a capsule is formed around it by the deposition of glial and fibroblastic elements. It gets thicker as time goes on, and by the end of 5-6 weeks it may be up to 2-3 mm., and quite tough; in very chronic cases, the abscess may be a hard fibrous capsule 1-2 cm. thick, enclosing only a small amount of inspissated pus. In other cases a fairly thick capsule may enclose large amounts of pus: in one case in this series there was 180 cc. and the author has seen one other case in which 500 cc. of pus were said to have been aspirated from a temporal lobe abscess with

recovery. These very large abscesses usually have only a single loculus, but many of the smaller ones are multilocular, and the different loculi may have capsules of varying thickness suggesting that they develop at different times.

The formation of a capsule is a favourable event because it means that there is a natural attempt to limit the infection, it lessens the risk of rupture and it also improves the chances of whatever kind of treatment is employed. But even after forming a capsule, the abscess is still a hazard because it is a more or less rapidly growing space-occupying lesion and may cause death in virtue of its size—just as a rapidly growing neoplasm—or it may rupture into the subarachnoid space or ventricular system to cause a fulminating meningitis which cannot be controlled.

The symptoms of a brain abscess may be inferred from this brief description of its pathogenesis. Increased intracranial pressure from œdema or the bulk of the abscess accounts for the headache, vomiting, and stupor which precedes the terminal coma. The increase in pressure can be measured by lumbar puncture manometry, although the other objective evidence on which the neurologist relies, i.e., the presence of papillœdema, is often lacking, unless the abscess has been present for some time, and not less than 2-3 weeks. Papillœdema on the whole is uncommon in acute brain abscesses, and the diagnosis should not wait on its appearance.

The focal neurological abnormalities depend on the part of the brain involved and the following is a brief description of the symptomatology of frontal, temporal and cerebellar abscesses.

Abscess of the Frontal Lobe

In our series the frontal abscesses were all associated with an *acute* sinus infection : chronic sinusitis seems more likely to cause extradural and subdural abscess, although surgical intervention for chronic sinusitis may lead to osteitis of the frontal bone and a brain abscess. In each of our cases the usual symptoms of an acute sinus infection were complicated by œdema of the eyelids, redness and swelling of the forehead, local pain and tenderness, signs which we interpret as being due to osteitis of the frontal bone. The swelling of the forehead may subside within a few days or it may persist as an indolent lump—Pott's puffy tumour (Fig. 1).

That all of our cases showed these signs of osteitis of the frontal bone is not to say that every case of osteitis goes on to develop a brain abscess, but the association is frequent enough to make us feel that these cases should not be allowed to escape observation until it is reasonably certain that there is no abscess.

In all of our cases there was an epileptic fit of some kind in the early stages of development of the abscess : sometimes it was a generalised convulsion, sometimes a focal attack involving the opposite side of the body. Focal neurological abnormalities are usually slight, but there is generally some weakness of the face, arm and leg on the opposite side of the body, with increase in the tendon reflexes, and perhaps an extensor

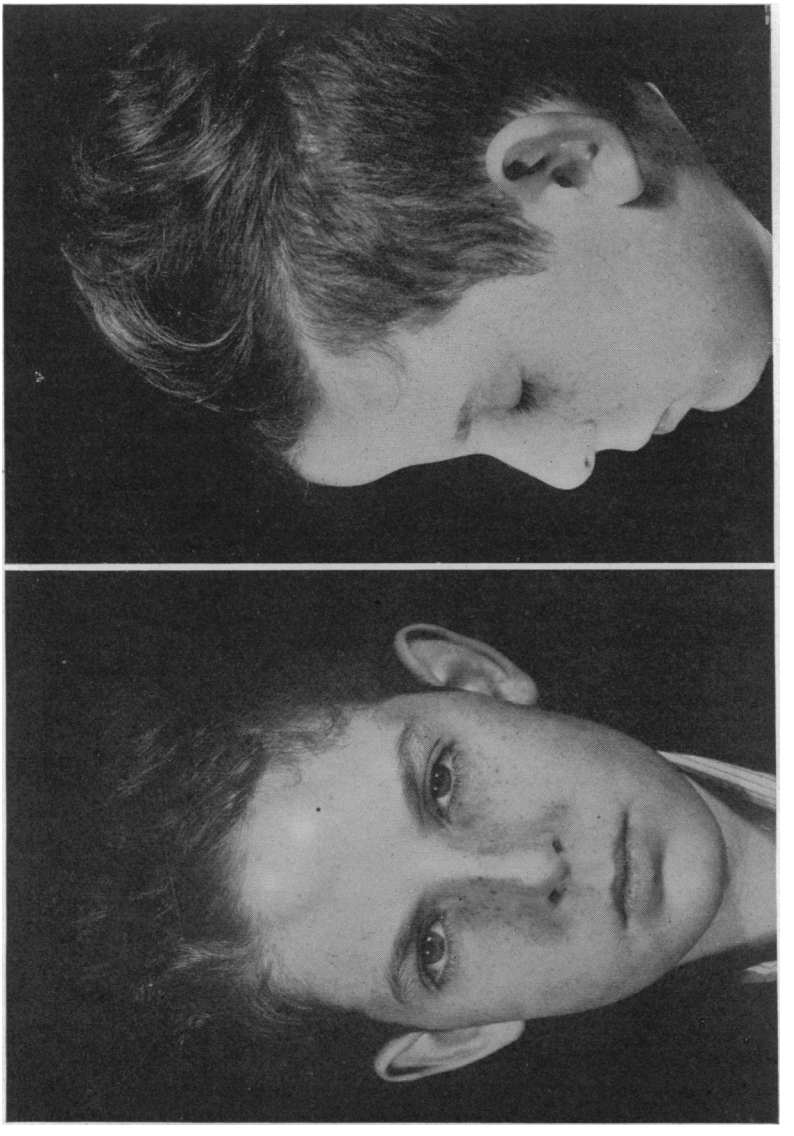

FIG. 1 Pott's Puffy Tumour with osteitis of frontal bone and right frontal abscess.

plantar response. There are usually no sensory abnormalities, and no defects in the visual fields, but if the abscess occurs in the speech-dominant hemisphere, there may be some aphasia, although this is less common than with temporal abscess. Mental symptoms may occur but they are generally overshadowed by the stupor which is due to increased intracranial pressure and is common to expanding lesions in whatever situation.

Temporal abscesses are usually the result of a chronic mastoid infection, but they may develop as a result of an acute otitis media which subsides without operative treatment. The most common symptom is pain in and around the ear extending to the temple, then becoming a generalised headache. They may be remarkably silent, or on the other hand they may produce signs which are unmistakable. If they occur in the speech-dominant hemisphere some degree of aphasia is common, although not invariable. The aphasia may be so slight that it can only be detected by formal tests, e.g., by asking the patient to name a series of common objects, testing his ability to read and write and to understand the spoken word ; or it may be so gross that it is taken by the patient's relatives—or his doctor—as evidence of dementia.

Whichever temporal lobe is involved, some defect in the visual field is common, although again not invariable. It may be gross enough to be detected by confrontation tests, or it may be so slight that it can only be demonstrated by careful perimetry (Fig. 2). It starts as a defect in the upper quadrant and may go on to become a complete homonymous hemianopia. As accurate perimetry demands a good deal of cooperation, it is important that the visual fields be tested in suspected cases before the patient becomes too drowsy or too ill to cooperate.

Neurological abnormalities otherwise are slight in temporal abscesses. Epilepsy is uncommon : only two of our cases have started with fits, and they differ in this important respect from frontal abscesses. Hemiplegia is likewise not common : when it does occur it usually starts in the face, and affects the arm and leg in that order. There may also be slight defects in sensation, demonstrable by careful tests.

Cerebellar Abscesses are usually fairly eloquent, producing signs which need only to be looked for to be found. Characteristically there is nystagmus, slow and coarse on looking to the side of the lesion, rapid and fine to the other side. This is the most constant abnormality but often there is some dysarthria, and usually some hypotonia, ataxy and dysdiadokokinesis of the arm and leg on the side of the lesion. These cerebellar signs are usually more marked in the arm than the leg. Among the less common disorders are squint, slight trigeminal impairment, and slight facial weakness. In several cases of cerebellar abscess we have noticed that the patient tends to keep his head bent forward in contradistinction to the more common opisthotonic attitude seen in cases of cerebellar tumour. Aphasia, hemiplegia, fits, and visual field disturbances do not occur in uncomplicated cerebellar abscesses.

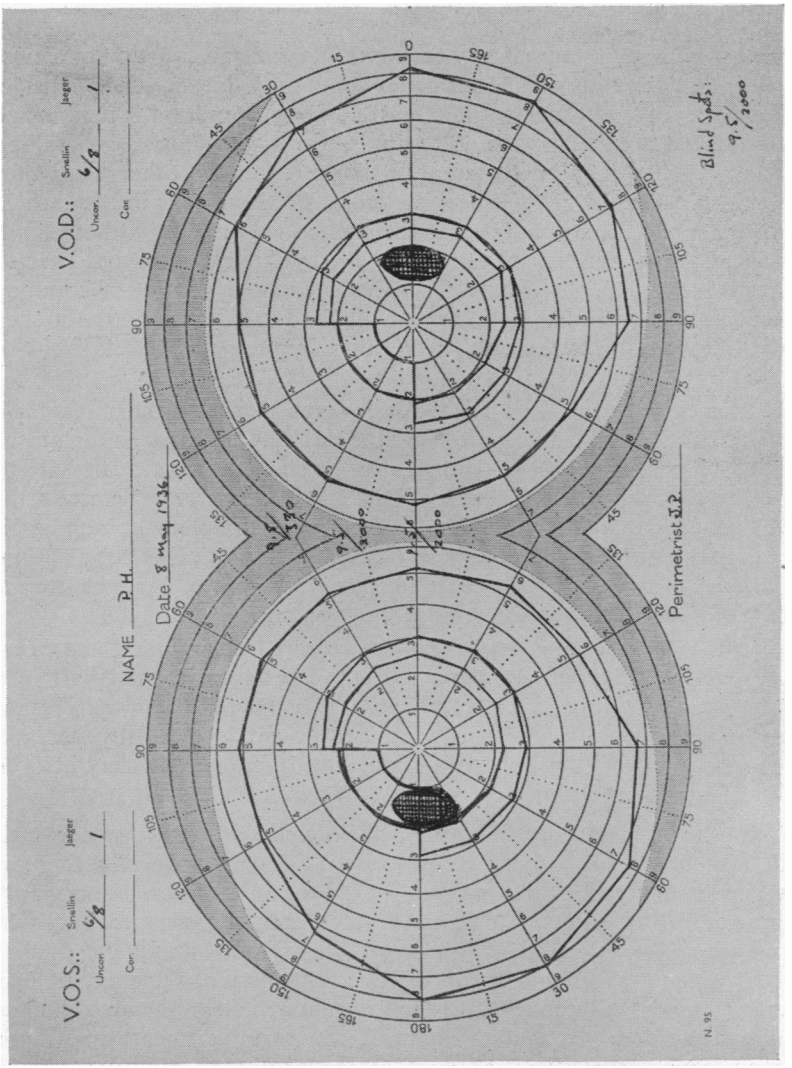

FIG. 2. Visual fields in a case of right temporal abscess. There was no detectable defect on confrontation tests.

Although the clinical features may be sufficient for the diagnosis and localisation of the abscess, there are many cases in which additional evidence is necessary. Examination of the cerebrospinal fluid yields valuable information in almost every case; either the cell count or the protein content is raised, and often both are abnormal. When doing the lumbar puncture the pressure should be measured with a manometer and the opportunity should be taken of doing Queckenstedt's test to determine the patency of each lateral sinus, as this may throw valuable light on an obscure case (see below: Differential Diagnosis). Ordinary X-rays of the skull are generally not very informative. Occasionally a calcified pineal gland may be seen to be shifted in a case of suspected temporal or frontal abscess and this may strengthen the diagnosis. Very rarely a bubble of gas may be seen in the abscess. Rarefaction of the dorsum sella and of the vault of the skull only occurs in those cases of chronic abscess in which there has been increased pressure for several weeks. An area of osteitis in the frontal bone may be visible within 3-4 weeks of the onset of the infection but usually the abscess demands treatment before X-ray changes are evident. We have not been able to detect specific erosion of the petrous bone to suggest an extension into the temporal lobe or cerebellum.

There are special radiographic techniques however which may establish the diagnosis. Arteriography is perhaps the safest and most informative: an injection of pyelosil into the common carotid artery, with skiagrams taken immediately, will show specific displacements and deformities of the normal vascular pattern and may clearly outline a frontal or temporal abscess (Figs. 3-4). In cerebellar abscesses this technique usually does nothing more than to suggest a mild internal hydrocephalus and in such cases the localisation of the lesion can be confirmed by doing a ventriculogram.

Arteriography is preferable to ventriculography in the majority of cases because it does not disturb the pressure relations within the cranium. The patient is usually not at all upset by an arteriogram, whereas a ventriculogram may precipitate a crisis demanding immediate operation. Nevertheless, if there is doubt, and if the facilities for arteriography are not available, there may be no alternative to ventriculography. There are some cases in which the ventricle on the affected side cannot be filled and we then only know that the abscess is in one hemisphere, but not whether it is temporal or frontal. We have encountered such cases in which there has been mastoiditis as well as frontal sinusitis, and the clinical features were not sufficient to tell where the abscess is situated. In such cases, it may only be possible to tell by exploratory trephination and aspiration in various sites.

Differential Diagnosis

The possibility of intracranial complications should be considered in every case of mastoid or sinus infection which is not " doing well."

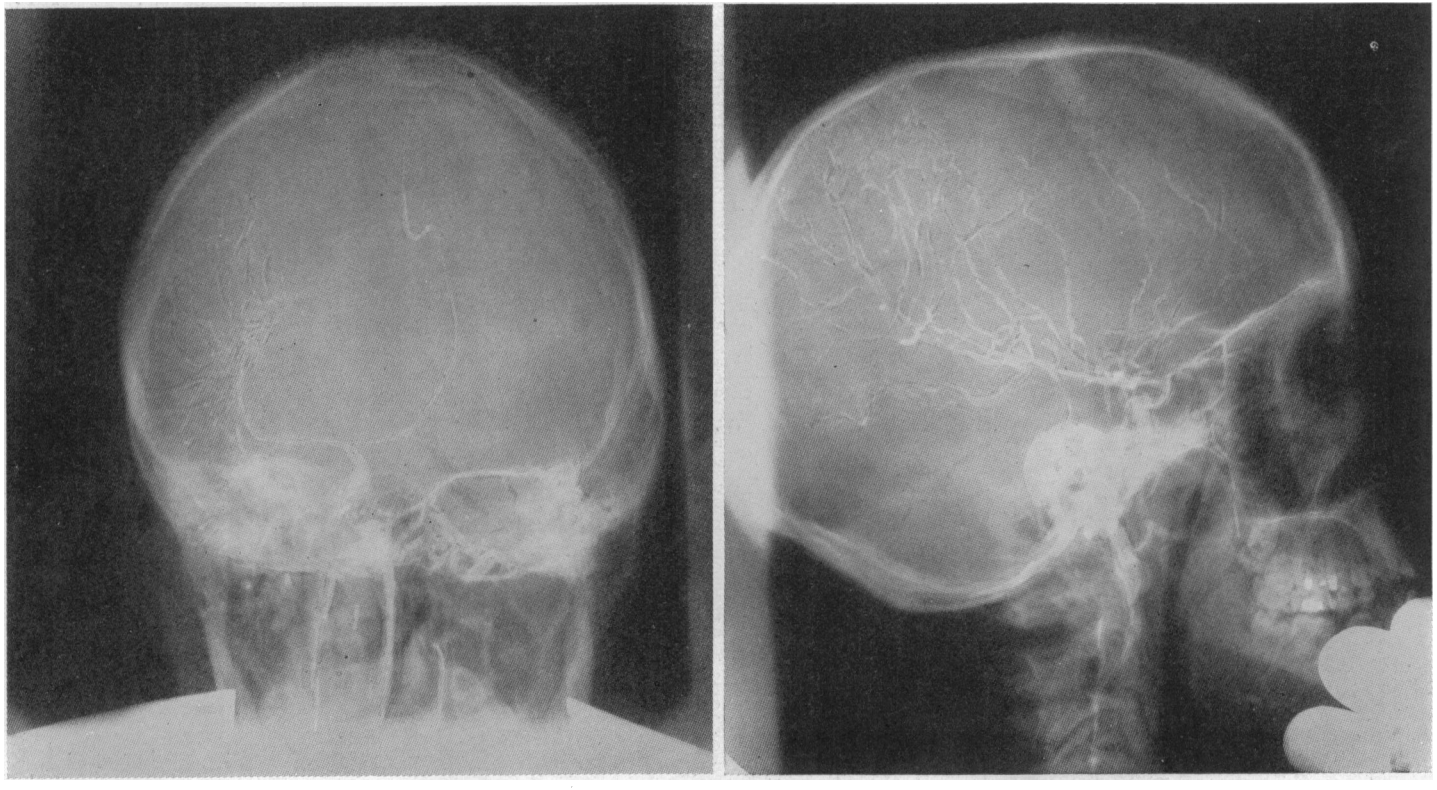

FIG. 3. Arteriogram in right frontal abscess.

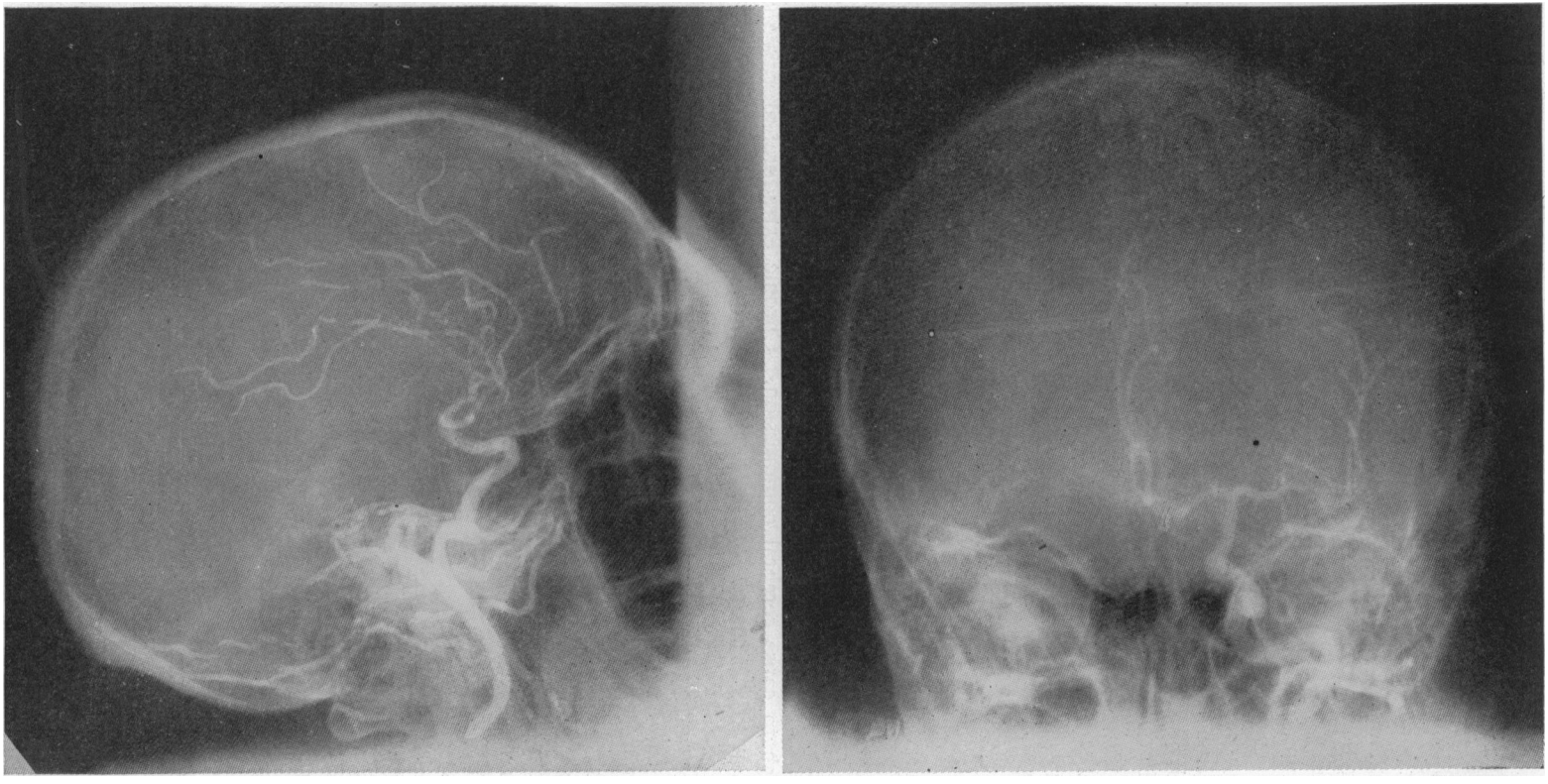

FIG. 4. Arteriogram in left temporal abscess.

Most surgeons have their own private standards for this state, and it may be difficult to describe. But certainly headache, vomiting and drowsiness demand an explanation, and the advent of epilepsy and of focal neurological abnormalities makes it clear that something is amiss within the cranium. A thorough neurological examination should be recorded as soon as possible, as a base-line on which to assess later developments. X-rays of the infected focus and of the whole skull should be taken, as these may show an extension of the bony infection, shift of the pineal gland, or evidence of increased intracranial pressure. A lumbar puncture should then be done to measure the pressure, to determine the patency of each transverse sinus, and for analysis of the fluid. Thus it may be found that headache and vomiting are due to a frank pyogenic meningitis, or that papillœdema is due to thrombosis of one transverse sinus, or that the changes in the fluid described above suggest the possibility of a brain abscess.

If these investigations do not clarify the diagnosis, other procedures may have to be employed. Thus one or more exploratory burrholes in the vicinity of an infected frontal sinus or mastoid may reveal an extradural or subdural abscess, and allow for drainage and instillation of antibiotics. Acute extradural abscesses usually cause a good deal of local pain or circumscribed headache, but little in the way of neurological abnormalities (such as paralysis, hemianopia, etc.) unless they are large enough to cause cerebral compression. Chronic extradural abscess in relation to a focus of chronic osteitis may be almost symptomless and only be discovered during an operation for removal of the infected bone. In both cases, the cerebrospinal fluid is usually normal both as to pressure and analysis.

Subdural abscess (purulent pachymeningitis, subdural empyema) is a more or less diffuse collection of pus between the dura and the arachnoid. It is a common sequel of osteitis of the skull and is often associated with widespread thrombosis of cerebral veins to cause profound disturbances of cerebral function such as hemiplegia, aphasia, hemianopia and sensory impairment. The gross neurological defects develop quickly, e.g., in the course of 24-36 hours—more rapidly than in the case of a brain abscess. Epileptic attacks, focal or generalised, are common, and the patient is usually gravely ill. The neurological picture may closely resemble that of infective cerebral thrombophlebitis, a much more benign condition (v.i.) and the diagnosis can only be made with certainty, or safely excluded, by making exploratory burrholes in the vicinity of the infected bone.

Infective thrombosis of cerebral veins or venous sinuses may produce two or three quite different clinical pictures. The clinical features of infective sinus thrombosis are well known : an infected clot in the sinus often leads to bacteremia and the swinging temperature and general illness associated with it. Pyemic abscesses in the lung are common, and the general illness is usually more impressive than any signs referable

to the cranium. But there is a more subtle variety of this disorder known as otitic hydrocephalus in which, during convalescence from a mastoid infection or operation, the patient complains of a little headache, and is found to have papillœdema and often a squint due to paralysis of one or both abducens nerves. The clinical picture differs from that of a brain abscess in that the patient is usually quite well; headache, vomiting and drowsiness are all slight, if indeed present at all, and the spinal fluid is normal except for increased pressure. The pathology of this condition is not clearly understood, but it is known to be benign, and complete recovery usually occurs. Safe differentiation from a brain abscess demands ventriculography or arteriography, as we have seen cases thought to be suffering from otitic hydrocephalus, and treated by repeated lumbar punctures, which have ultimately been shown to have brain abscesses.

A common problem in differential diagnosis is that between brain abscess and neoplasm. It is not surprising that some patients with chronic mastoid or sinus infection should develop brain tumours, or that a patient with a brain tumour should occasionally develop a mastoid or sinus infection. Careful enquiry into the history may clarify the issue, but there are many cases, especially those in deep stupor or coma, when they first come under observation, in which it may be impossible to decide between a tumour and an abscess without arteriography or biopsy. An arteriogram may reveal the vascular pattern of a tumour or an avascular mass in the brain which might be either an abscess or a cystic neoplasm. In such cases, an exploratory burrhole should be made and a brain cannula inserted into the lesion. Aspiration will then reveal either pus, cystic fluid or neoplastic tissue which can be spread on a glass slide, fixed and stained to establish the diagnosis in the vast majority of cases.

There is also a large group of patients with chronic mastoid or sinus disease who complain of persistent headache, vague giddiness, etc., in whom sooner or later the question of a brain abscess arises, either with the patient, his relatives or his doctor. There are usually no neurological abnormalities, no signs of increased intracranial pressure, and the spinal fluid is normal. In some cases, reassurance may be sufficient, but in some ventriculography or arteriography has to be done to convince everyone that no abscess is present.

TREATMENT

Having diagnosed and localised a brain abscess, how should it be treated? It is apparently still a common practice to incise and drain these abscesses as with abscesses in any other part of the body. We think that this is wrong as a principle of treatment, although there may be times and circumstances which leave no alternative, and the introduction of penicillin has made this a much safer procedure than it ever was in the past. In our own hands, of 17 cases treated by various drainage procedures (prior to 1938) only one recovered.

There are several reasons why incision and drainage cannot be relied upon. The abscess is often quite small, and after the initial gush of pus it may be impossible to place a tube or any kind of drain in the cavity. Even if the cavity is found, its soft walls collapse around the drain or block the end of the tube so that it acts only as a plug. More pus may accumulate and be denied exit, the surgeon meantime feeling secure in the thought that if it wants to come out badly enough it can do so. Furthermore these abscesses are often multilocular: a drain may deal with only one loculus, and another, larger one, may be undetected until it has ruptured or otherwise killed the patient. By punching through the capsule of an abscess a drain may rupture the ventricle or infect deeper structures in which another abscess develops.

Drainage is most effective in cases of large chronic abscesses such as the ones Macewan described 50 years ago; they have a tough capsule, and it may be easy to introduce a tube which acts efficiently. Even so the capsule is left behind and it may cause trouble in the future. In any case, these abscesses are rare enough to make it unwise to apply a method of treatment effective for them to all cases.

In particular we disapprove of drainage through a mastoid wound because this often leads to the formation of a fungus which enormously adds to the difficulties of definitive treatment (Fig. 5).

We believe that the best way of dealing with a brain abscess is to treat it so that it can eventually be extirpated. This means keeping the patient alive until the abscess has formed a capsule sufficiently thick to withstand reasonably careful handling in dissecting it from the surrounding brain. The formation of such a capsule takes 5 to 6 weeks. In the meantime the symptoms due to increased intracranial pressure are dealt with by aspirating the abscess, and repeating the aspirations as often as may be necessary. The aspirations are done through a burrhole (Fig. 6) using a blunt brain needle for the first few days, and a stout lumbar puncture needle after the scalp incision has healed. We make a practice of instilling 2 ccs. thorotrast into the abscess cavity after the first aspiration as this is radio-opaque and is a most useful agent for determining the site and size of the abscess and variations in its size from time to time.

A solution of penicillin (100,000 units in 2-4 cc.) is injected into the cavity after each aspiration if the infection is due to a sensitive organism. If there is a continuing infection of the mastoid or frontal sinus, systemic penicillin is also used.

In many cases aspiration of the abscess and instillation of penicillin will bring about a dramatic improvement, to such an extent that it may be thought that the abscess has resolved. If the abscess has been visualised by thorotrast, it may indeed be seen to have shrivelled up, but there are cases in which, despite the clinical improvement, the pyograms show that the abscess is increasing in size (Figs. 7-8). Moreover, as brain abscesses are often multiple or multilocular, it may be that only one loculus is being aspirated and treated with penicillin while an adjacent

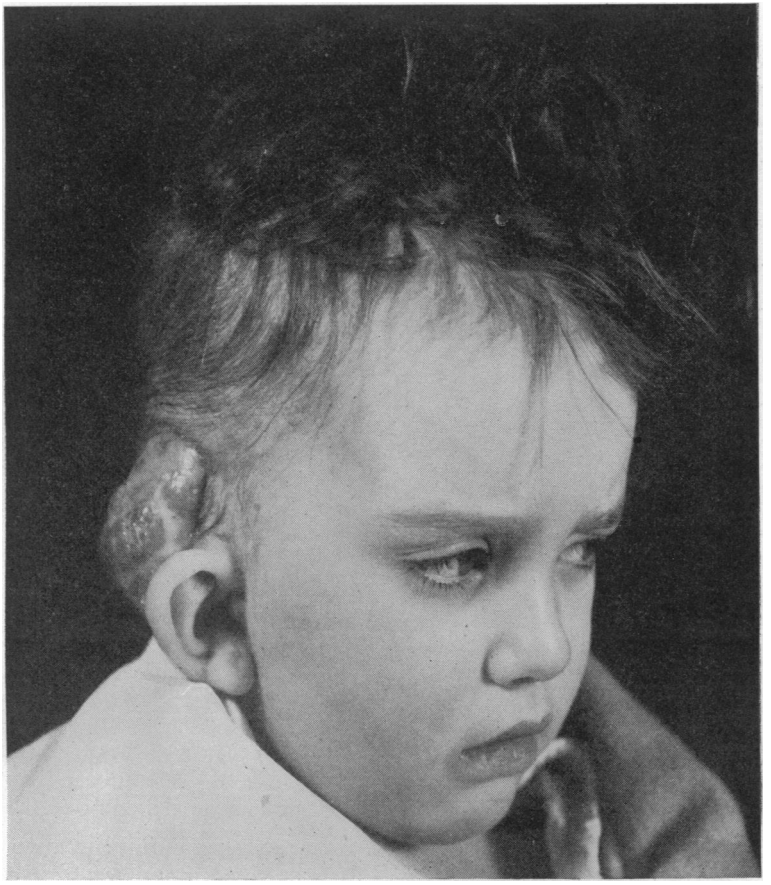

FIG. 5. Brain fungus resulting from drainage of right temporal abscess through a mastoid wound.

loculus is ripening for a fatal rupture into the ventricle. For these reasons we feel that it is generally unsafe to rely on aspiration and penicillin alone, and that only by excision can we be certain that the whole infection has been eradicated.

In some cases, aspiration in the acute stage is not sufficient to deal with a critical increase of intracranial pressure and a decompression operation is necessary as advocated by Vincent(1). An osteoplastic flap is reflected over the abscess, and when it is considered that the abscess wall is tough enough to be dissected out, the flap is re-elevated and the abscess is removed much as a solid tumour. It is preferable to remove it intact, but frequently the field is contaminated by a slight leakage of pus. We have not seen much harm come from this, especially since the

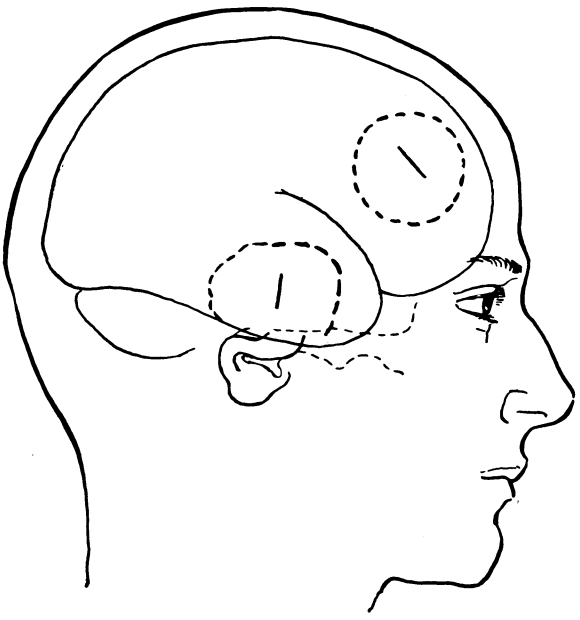

FIG. 6.—Site of incisions for burrholes for aspiration of temporal and frontal abscesses.

advent of penicillin and the sulphonamides. A solution of penicillin (12,000 units) is left in the cavity, and, after dusting with penicillin-sulphonamide powder, the wound is closed in layers without drainage.

As illustrations of the diagnosis and treatment of temporal, frontal and cerebellar abscesses, the following cases are described :—

Case 1.—Left Otitis Media. Spontaneous resolution. Onset of symptoms of left temporal abscess. Repeated aspirations and ultimate extirpation of chronic left temporal abscess. Recovery.

K.C., a boy *æt.* 8 (R.I. No. 20226/41) was admitted on 26th December, 1941. He had been in good health until September, 1941, when he had a left-sided earache and discharge lasting for 4-5 days. This had cleared up and he had no further trouble with the ear, but shortly afterwards he began to complain of headache. It was chiefly in the left side of the head and it became more frequent and persistent, and occasionally he vomited with it. He continued at school but in the week before admission the headache was so severe that he had to go to bed. From that time he became drowsy and incontinent, and when roused he screamed with headache.

On admission he was stuporose but he could be roused and it was possible to say that he had a slight nominal aphasia. There were 3 D. papillœdema and a complete right homonymous hemianopia, right sixth nerve palsy, weakness of the right side of the face and the right

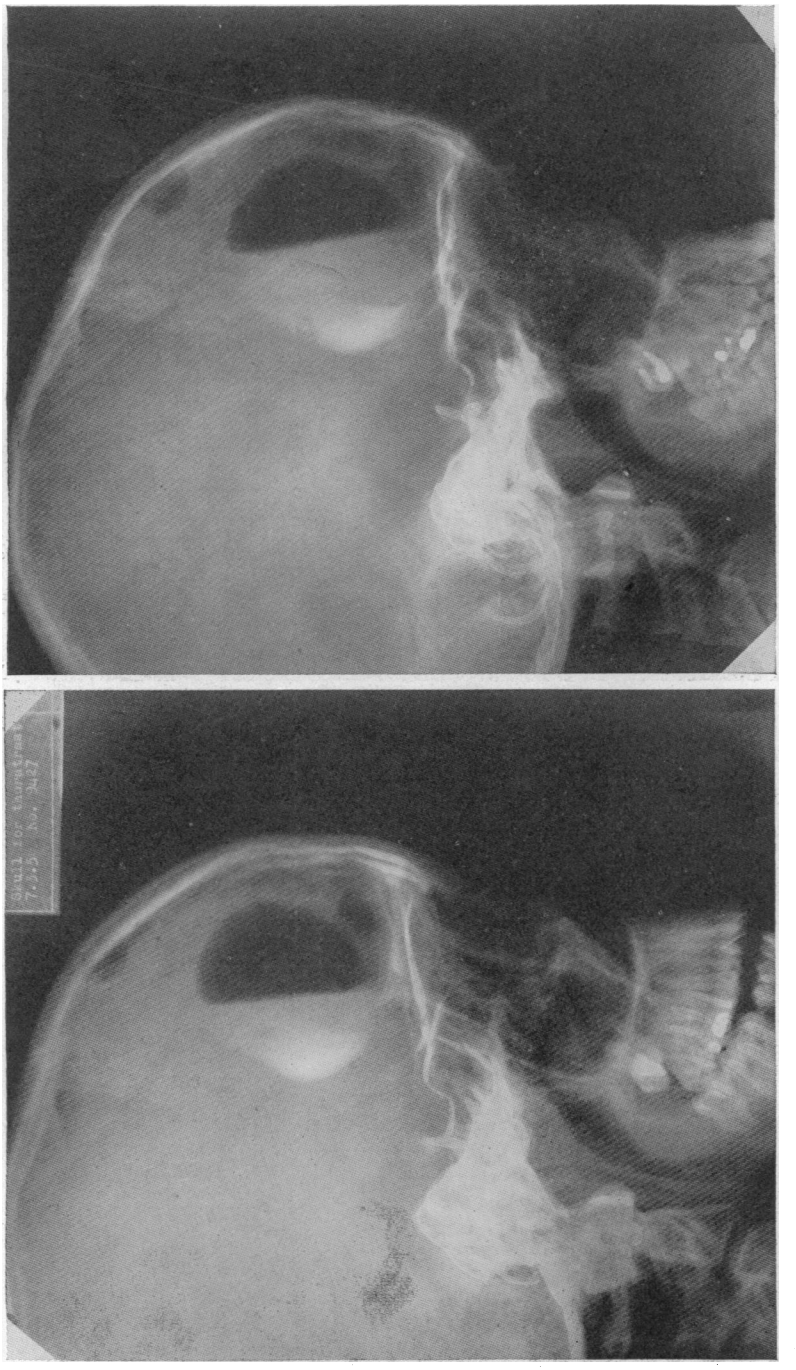

FIG. 7. Pyograms at intervals of 3 days showing increase in size of abscess, despite the patient's well-being and absence of symptoms or signs.

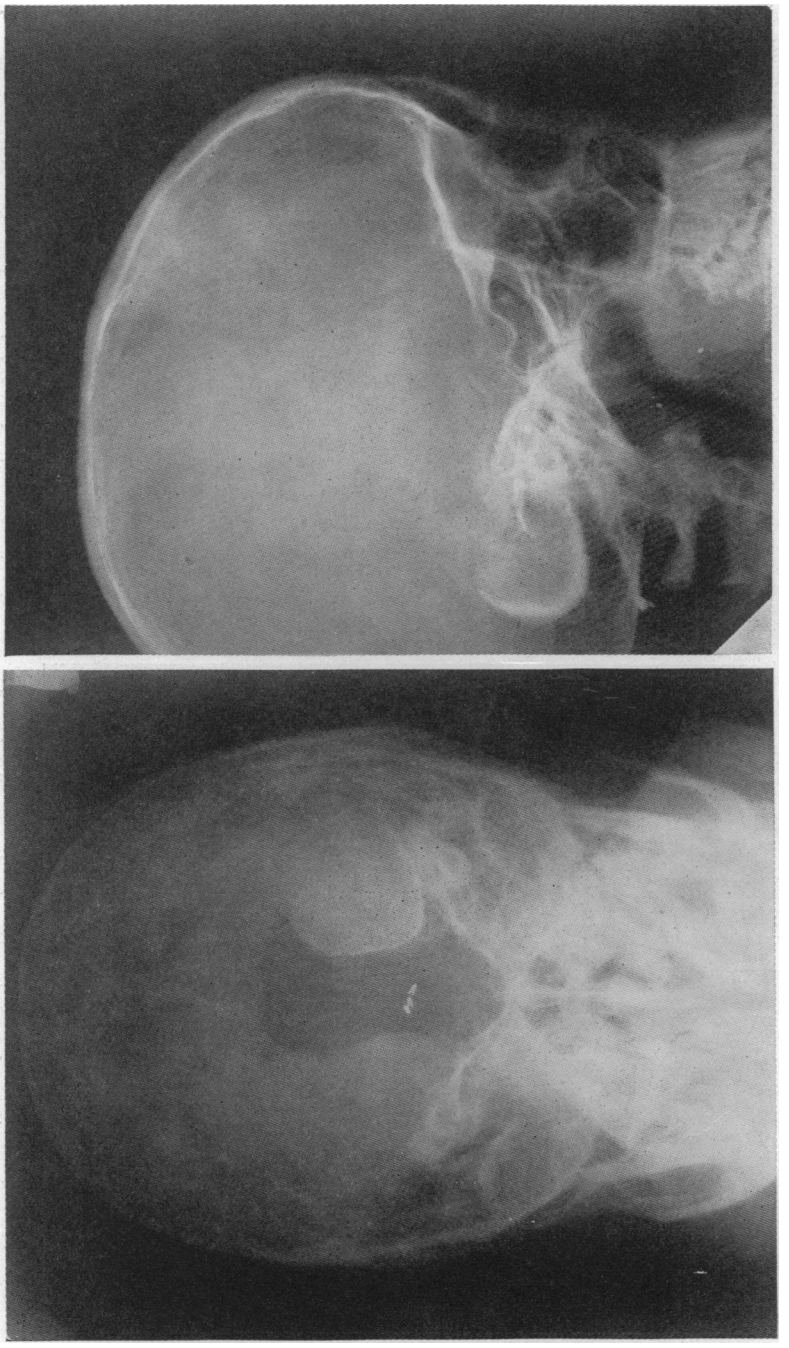

Fig. 8 (*a*). Legend on page 121.

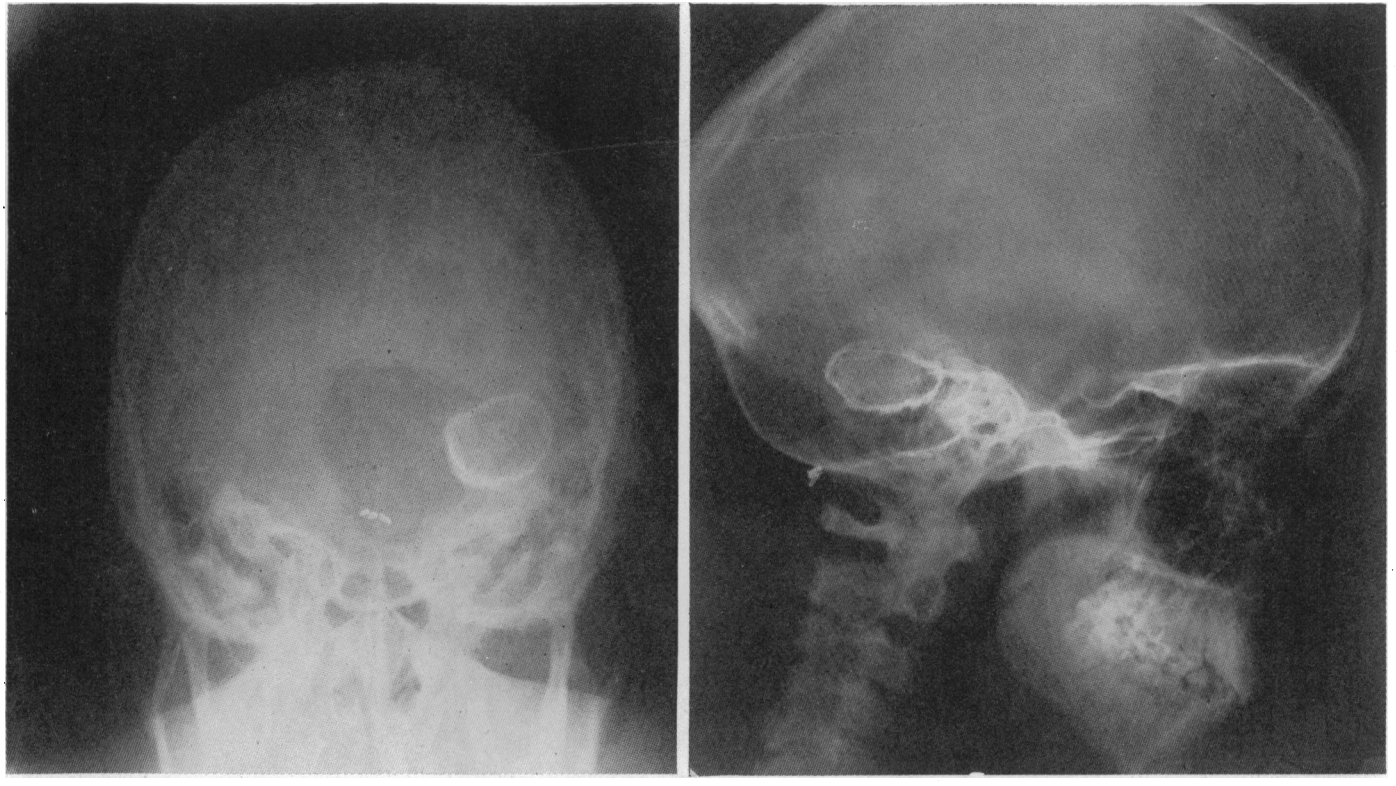

FIG. 8 (*a*) and (*b*). Pyograms of cerebellar abscess at intervals of 2 days, showing increase in size, of which there was no clinical evidence.

arm. X-rays of the skull showed a little separation of the sutures. The C.S.F. pressure was over 300 mm. ; the fluid was clear and colourless, and contained 60 mgm. protein and 15 cells, mostly lymphocytes. Both ears were quite dry and appeared to be normal except for a slight scar in the left tympanic membrane.

There was thus clear evidence of a rapidly expanding lesion in the left temporal lobe, and although the aural infection was relatively inconspicuous, the onset of intracranial symptoms seemed to be immediately

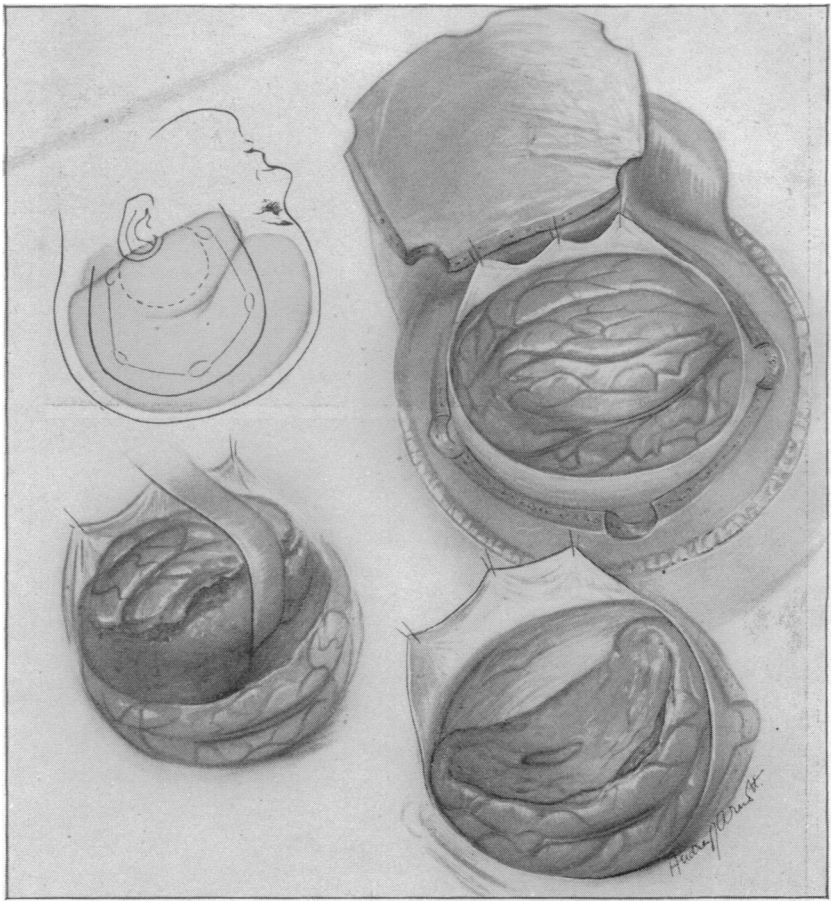

Fig. 9

related to it, and the diagnosis of left temporal abscess was the most likely one. Accordingly, a burrhole was made over the left ear : the scalp, bone and dura looked to be normal, but the brain was under great pressure and began to exude through a small nick in the dura. An

exploring cannula was inserted and, at a depth of less than 1 cm., it was felt to slip into a cavity from which 25cc. of pus were aspirated. 2 cc. of thorotrast were instilled, for subsequent radiological study. There was an immediate improvement: he woke up, asked for a drink, and during the next 24 hours there was considerable improvement in the right hemiparesis. From the pus a pneumococcus was cultured.

On 29th December, 3 days after the first aspiration, he again became very drowsy and X-rays showed that the abscess had increased in size and was tense. Another aspiration was done and 75 cc. of pus withdrawn. Again there was a dramatic improvement, but recurrences of stupor made further aspirations necessary: on 9th January (80 cc.), 13th January (72 cc.) and 7th February (12 cc.). After the last aspiration, the clinical improvement was maintained, and except for a right upper quadrant hemianopia, there were no neurological abnormalities. Clinically he appeared to be cured, but serial X-rays showed that the abscess was still large and was not shrinking. Accordingly an osteoplastic flap was reflected on 7th March and a thick-walled chronic abscess was extirpated from the left temporal lobe (Fig. 9). Convalescence was uneventful and he was discharged from hospital 3 weeks after operation, with no abnormalities except the upper quadrant defect in the right homonymous field of vision which has persisted until his last report in February, 1949.

Case 2.—Frontal Sinusitis. Drainage of Empyema of Left Frontal Sinus.

Symptoms of Intracranial Extension. Repeated aspiration and installation of penicillin into left frontal abscess. Extirpation of abscess. Recovery.

J.W., a prison supervisor, *æt.* 49, was admitted on 21st January, 1942. 17 days before admission he developed a severe head cold in the course of which he complained of acute pain in the left side of his forehead. At the end of a week there was some redness and swelling of the left frontal region and the pain seemed to change its character and to become a severe bifrontal headache. A week before admission he began to have attacks of twitching of the right side of the face and his speech was disordered. There was no loss of consciousness.

On admission, he was obviously in a good deal of pain and there was still some swelling of the left side of the forehead. He was a little confused but in addition there was a considerable aphasia: he had difficulty in naming common objects, so much so that his conversation was at times difficult to follow, and his relatives had naturally spoken of this as confusion and "behaving queerly." There were no other neurological abnormalities. X-rays of the skull showed an opaque left frontal sinus. The cerebro-spinal fluid pressure at lumbar puncture was 180 mm. The fluid contained 50 mgm. protein and 224 cells, mainly polymorphs. The general examination revealed a marked aortic incompetence to the point at which the bed shook noticeably with each heart beat.

On the day following admission Mr. R. G. Macbeth drained an empyema of the left frontal sinus. This eased the local pain, but the aphasia became more marked ; he became drowsy, and gradually during the next 6 days a right hemiplegia developed.

On 29th January, a left frontal burrhole was made, and 30 ccs. of pus were aspirated from the left frontal lobe and 2 ccs. of thorotrast injected. He immediately became more alert, and on the following day there was some increase in the power of the right limbs. Two days later, the return of stupor called for another aspiration and 36 ccs. of pus were aspirated through a sharp needle. Pneumococci were seen on the films and on culture. In the next two weeks three further aspirations were required, and on each occasion penicillin was injected.

With these aspirations there was a dramatic improvement so that the aphasia and hemiplegia cleared up, and the patient felt perfectly well. Clinically he seemed to be cured, but X-rays showed a large bilocular abscess which, although not enlarging to any great extent, was not shrinking. As he lived near the hospital he was allowed to go home on 7th March and remain under out-patient supervision.

He remained well until 12th March, when he again complained of headache and during the course of the day the weakness of the right arm and leg recurred. He was readmitted, in stupor, with a considerable right hemiplegia. The abscess was again aspirated with some improvement, but, as it seemed unlikely to resolve with repeated aspirations and as it was judged to be at least 8 weeks old, and thus to have a reasonably thick capsule, it was felt that the safest plan was to excise it, and this was done on 16th March. The abscess had a tough capsule and was in fact composed of three separate loculi, only one of which had been treated with penicillin, and two outlined by thorotrast. The superficial loculus, which had been treated with penicillin, was sterile, but the two deeper loculi contained gram positive diplococci. Convalescence was uneventful and he remained well until the time of his last report in March, 1949.

Case 3.—Chronic Mastoiditis. Removal of Left Cerebellar Abscess. Recovery.

S.B. male *æt.* 26, was admitted on 1st August, 1946, complaining of severe headache and unsteadiness in walking. He had suffered from chronic otitis media for many years, and six weeks before admission had had a radical mastoidectomy on the right side. His convalescence from this operation was uneventful and there were no intracranial symptoms at all. He returned to his work as a joiner and was quite well until 6 days before admission when he complained of a slight occipital headache and giddiness, and he noticed that he was a little unsteady in walking. The headache rapidly became worse and the unsteadiness was so marked that he had to give up work the next day. On the day before admission he noticed some diplopia and his speech became a little thick and husky.

On admission he did not look acutely ill, but he was a little lethargic and complained of headache. His speech was thick, but intelligible and there was no aphasia. There was no neck stiffness but he seemed to prefer to keep his head bent slightly forward. There was no papillœdema, and the visual fields were full. There was slow coarse nystagmus on looking to the right, and rapid and fine nystagmus on looking to the left. There was no trigeminal impairment or facial weakness. Hearing was grossly reduced on both sides as a result of the chronic aural infection. There was no obvious paralysis of palate, tongue or vocal cords. In the limbs, there was gross ataxy of the right arm in purposive tests, less in the right leg, and the gait was very unsteady. The tendon reflexes were slightly reduced in the right limbs, and both plantar responses were flexor.

X-rays of the skull were normal. The cerebro-spinal fluid pressure was 300 mm. and the fluid contained 150 mgm. protein and 150 cells, mostly lymphocytes.

There seemed no reasonable doubt about the diagnosis of a right cerebellar abscess and operation was advised at once. From the fact that symptoms had been present for only 6 days, it was thought to be an acute one, but when the cerebellar decompression had been made and a needle introduced into the right cerebellar lobe, the abscess was found to have quite a tough capsule. Accordingly a portion of the lateral lobe overlying the abscess was excised, and the capsule dissected from the surrounding white matter. It was found to be firmly attached to the back of the petrous bone and, in freeing it, the capsule ruptured and some pus escaped to contaminate the operation cavity. This was sucked out, and 20,000 units of penicillin left in. The whole wound was dusted with penicillin-sulphamezathine powder and it was closed in the usual way without drainage. Convalescence was uneventful. The wound healed normally; there was no evidence of infection of the cavity or of meningitis and the patient was discharged from hospital 3 weeks after operation. He returned to his work and has remained well. At the time of his last report in April, 1949, no neurological abnormalities could be demonstrated.

RESULTS

Applying this method to frontal abscesses there were 2 deaths in 11 cases. In one of the fatal cases the patient was moribund on admission and succumbed before any treatment could be given. In the other there was a diffuse purulent ventriculitis on admission which did not yield to vigorous penicillin treatment and led to death 3 weeks later.

With temporal abscesses there were 9 deaths in 28 cases, a mortality of 32 per cent. Of the 9 fatal cases some were moribund on admission, some had multiple abscesses or complicated infections, one case was undiagnosed, and one died from meningitis after removal of the abscess (in the pre-penicillin period). These circumstances were such that we do not feel that the present overall results detract from the principles

of treatment outlined above, and there is no doubt that penicillin, and possibly other antibiotics, can do much to improve them. In no group is this shown more clearly than in the cerebellar abscesses: out of the first 9 cases in this series, treated along the lines described above, but without penicillin, only 2 recovered, whereas of the second group of 9 cases treated in the same way, but with penicillin, 8 recovered, and the one fatality in this second group was probably due to a post-operative clot.

As regards the functional results, excision of a frontal abscess usually leaves no detectable neurological or intellectual defect. These patients are prone to epilepsy, however, and should be kept on regular anti-convulsant therapy for several years after operation.

Excision of a temporal abscess usually leaves an upper quadrant or complete homonymous hemianopia as its sequel. Most patients adapt themselves to this very readily and are usually not at all inconvenienced by it. In those cases in which the abscess was in the speech-dominant hemisphere, none has had any permanent aphasia.

Of the ten cases of cerebellar abscesses which have recovered two have had considerable residual disabilities due to ataxy and dysarthria. The others have had no residual disability at all and have been able to return to their former occupations.

Priority of Treatment

In some of these cases, the question of priority of treatment arises: should the brain abscess or the primary focus in the mastoid or accessory sinus be dealt with first? In many this problem does not arise because the brain abscess only declares itself after the primary infection has resolved (as in Case 1) or responded satisfactorily to operative treatment (as in Case 3). In general, the condition which presents the more immediate threat to life demands the most urgent treatment. Thus a patient with severe meningitis and an active mastoid infection needs treatment for the meningitis in the first instance, but the mastoid operation should be done as soon as the general condition allows and in many cases this will be within two or three days of beginning treatment for the meningitis. The same argument applies to subdural abscess (purulent pachymeningitis) which is commonly due to frontal sinus disease. The subdural space must be drained and treated with penicillin as an emergency and definitive treatment of the sinus or the associated osteitis deferred until the patient's general condition improves. In fact, with the general use of sulphonamides and antibiotics, we are usually treating the primary focus all the time, and the question of priority is hardly as urgent as it once was.

A more common situation is that of the patient admitted in stupor due to a temporal or cerebellar abscess with an active mastoid infection. Although it would appear to be more logical to deal with the mastoid first, such patients may die from a sudden increase of intracranial pressure during anæsthesia for a mastoid operation, and the right treatment in the case of a temporal abscess is to make a clean burrhole in the squamous

temporal bone just above the pinna, aspirate the abscess and inject penicillin (250,000 units in 5 cc.) into it. This will usually cause a dramatic improvement and the mastoid can then be dealt with safely within a day or so. The permeatal route is preferable when practicable, because it may be necessary to reflect an osteoplastic flap before a retro-auricular incision has healed. Further aspirations of the abscess may be necessary during the five or six weeks before it is sufficiently encapsulated for extirpation.

If the abscess is in the cerebellum, and the mastoid has not been dealt with, it is usually preferable to do a cerebellar decompression through a middle line skin incision, aspirate the abscess and inject penicillin. If this is not possible, the next best procedure is to make a burrhole in the occipital bone just below the superior nuchal line and midway between the middle line and the posterior border of the mastoid. This provides no decompression, but it affords a route for tapping the abscess and injecting penicillin one or more times as may be required. There is often such prompt improvement that the mastoid operation can be done within a day or so.

In the case of frontal abscesses, the sinus infection has usually resolved or responded to treatment by the time the brain abscess demands treatment. But if, as in Case 2, the brain abscess declares itself during an acute sinus infection, there is no reason why the sinus should not be dealt with first, provided that the brain abscess is not endangering life because of increased intracranial pressure. In some cases it may be practicable to do the two operations at one session, i.e., to drain the frontal sinus and to aspirate the abscess through a clean burrhole.

In conclusion, it is important to say that early diagnosis is the most important factor in the successful management of a brain abscess. Suspected cases should have the benefit of early neurological examination and observation, and more use should be made of lumbar puncture. This procedure carries no risk if there is no intracranial trouble, and a normal fluid may give a great deal of assurance in a doubtful case. If the fluid is at all abnormal as to pressure or content, it means that everyone must be on his toes, and a careful watch kept for new developments as the indication for operation may occur with alarming suddenness. From their nature brain abscesses demand close collaboration between the neurological surgeon and the otologist. Not every hospital can provide a neurosurgical as well as an otological service, but if no neurosurgical service is available and an emergency arises, the otologist should make a clean burrhole and aspirate the abscess one or more times until a transfer can be arranged for definitive neurosurgical treatment.

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