Section of Otology

President J P Monkhouse FRCS

Meeting December 2 1960

Discussion on Intracranial Complications of Otogenic Origin

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We have had something over 200 patients in the Department of Neurological Surgery at the Radcliffe Infirmary with intracranial complications of mastoid infection (Table 1).

Table 1
Intracranial complications of mastoid infection

	No. of patients
Temporal lobe abscess	55
Cerebellar abscess	30
Otitic hydrocephalus	28
Cerebral thrombophlebitis	14
Meningitis	13
Subdural abscess (middle and posterior fossa)	10
Lateral sinus thrombosis	8
Extradural abscess (middle and posterior fossa)	5
Osteitis of skull	3
Endaural cerebral hernia	2
Cerebrospinal otorrhœa	1
Subgaleal aerocele	1
Undetermined	35

This is selected material in the sense that all these patients have been referred to a neurosurgical department under suspicion of having intracranial complications which the otologist does not commonly deal with, and this explains the fact that there have been comparatively few cases of lateral sinus thrombosis, extradural abscess and meningitis. It also explains the large number of cases in the undetermined group. These are mostly patients who have headache, giddiness or some other symptom consequent on mastoid infection; all our special investigations are negative and the patients usually recover. Our function with this group is thus largely an excluding one.

But if this is selected material, I should also say that I have seen most of the intracranial complications which have occurred in the Ear, Nose and Throat department at the Radcliffe Infirmary. In fact, there have not been many, and the volume of our material is derived from our being the only neurosurgical centre in our region. The actual incidence of each complication is not of great importance: the fact is that meningitis, brain abscess, sinus thrombosis and otitic hydrocephalus are comparatively common, whereas the other listed complications are comparatively rare.

In the vast majority of cases intracranial complications are the result of direct spread of the infection. On only two occasions, for instance, have I seen a brain abscess due to mastoid disease in an unusual site: one was a child who died with an abscess in the thalamus, and this must have been a hæmatogenous one from a bacteriæmia at some stage in her illness. The other was an abscess in the postero-medial part of the lateral lobe of the cerebellum, which arose by extension of an infective thrombosis of the lateral sinus along one of the tributary veins from the cerebellum.

The locus of the infection is determined by tissue planes, and otologists are familiar with cellulitis of the scalp overlying an infected mastoid, and with subperiosteal abscess. Infection of the mastoid may extend to involve the petrous bone commonly, and the vault of the skull more rarely. Collections of pus in the extradural space, either in the middle or posterior fossa, are common findings at mastoid operations and may mark the limit of the infection or may be associated with infection in deeper planes, e.g. subdural abscess, leptomeningitis, and brain abscess. Proximity of the transverse sinus renders it an easy prey to thrombosis, and this may extend back along tributaries from the cerebrum and cerebellum to disturb the circulation and cause alarming neurological symptoms such as epilepsy, profound paralysis, aphasia and the like. Com-

monly the infection is active in two or three of these planes: thus a patient may have an extradural abscess, a subdural abscess and meningitis at the same time. Or one or more planes may be missed out, or apparently so, as not infrequently a person with a temporal lobe abscess has no evidence of extradural or subdural abscess, and no meningitis.

Diagnosis and Treatment

Most otologists have their own personal standards by which they judge whether a mastoid infection is behaving properly or not. They know how much local pain is allowable, for how long and to what extent the temperature may be elevated, and so on. But undoubtedly the common use of sulphonamides and antibiotics influences the constitutional reaction in such a way that intracranial complications can occur with little evidence from the temperature and pulse chart, and to this extent the problem is more difficult than it once was.

Mr J D K Dawes (p. 315) stresses the importance of headache as a symptom, and rightly so, because headache can be the earliest and most constant feature of all these complications. Its most serious significance is when it is a symptom of increased intracranial pressure, and then it is often accompanied by nausea and vomiting, later by drowsiness going on to stupor, coma and death if unrelieved. Sometimes it is a factor of meningeal irritation, as in meningitis or subdural abscess, and sometimes it is due to no cause that we can determine. Many of the patients in the 'Undetermined' group in Table 1 were referred to us as brain abscess suspects because of headache following a mastoid infection, and our investigations proved entirely negative. It may be very difficult to assess headache in children, and in very ill patients who may be so ill in other respects that they are unable to describe their headache. And so we cannot rely entirely on headache for the detection of intracranial complications, nor can we exclude them by its absence.

Giddiness is another common symptom, but here the neurosurgeon is on less familiar ground than the otologist. Whether it is a feeling of unsteadiness or frank vertigo, I am rarely certain whether it is due to labyrinthine irritation by the disease process (or operative intervention) or whether it has a more central and serious significance. I think all I can usefully say is that it is very common in cerebellar abscess and may indeed be the predominant symptom.

Fortunately in lumbar puncture we have an easy method of getting information about the intracranial pressure, what is happening to the meninges, and whether or not the lateral sinus is patent. This is the first special investigation to be undertaken in cases of mastoid disease which are not going well. It may establish the diagnosis of meningitis at once, and enable treatment to be started, or it may show the increase in pressure and the slight increase in the protein content and cell count of the fluid, which are common in brain abscess and in some cases of subdural abscess. It may reveal the high pressure and the watery or normal fluid which are common findings in otitic hydrocephalus. A normal spinal fluid does not exclude any of these possibilities (except meningitis) as in a few cases of brain abscess (less than 5%) the fluid may be normal, and also in occasional cases of subdural abscess – and this is remarkable when we see that a large collection of pus is separated from the subarachnoid space by the very thin arachnoid membrane.

The dangers of lumbar puncture are well known, but I think they have probably been overstressed, and the value of the information which may be gained outweighs the risks. Lumbar puncture is only dangerous in advanced cases of increased intracranial pressure, when there is violent headache, stupor or coma, and in such there is often papillœdema as well. In these cases, the puncture had better be done in circumstances where an immediate operation can be done if necessary, this usually means neurosurgical facilities.

Ordinary X-rays of the skull may also yield useful information. They may show evidence of osteitis, and in children appearances associated with increased intracranial pressure such as separation of the sutures and convolutional thinning of the vault. In adults the pineal gland may be calcified, and if this is displaced from the middle line it is a considerable argument for an expanding lesion such as an abscess in one cerebral compartment.

Evidence of more localizing value may be obtained from cerebral angiography. Displacement of the normal arterial pattern may reveal a subdural or temporal lobe abscess, or indicate the ventricular dilatation which occurs with cerebellar abscess. The phlebogram may show partial or complete occlusion of one of the large venous sinuses. In skilled hands, angiography is quite safe and rarely upsets the patient or adds to the urgency of his condition. The same cannot be said of ventriculography or air encephalography which, although valuable diagnostic methods, often aggravate matters to the point where immediate operation is imperative. These investigations should only be carried out where neurosurgical facilities are available, but these techniques may reveal lesions not detectable by other means, and they are of particular value in the final exclusion of a space-occupying lesion.

Electroencephalography is also of value in some cases and may reveal an abnormal electrical

discharge in relation to an abscess, which some regard as almost diagnostic, but in our experience the EEG has not been reliable to that extent.

Common Intracranial Complications

Of the complications which have not already been dealt with, brain abscess is the most important. *Abscesses due to mastoid disease* have a very constant situation in the middle third of the temporal lobe or in the anterior part of the lateral lobe of the cerebellum. The temporal ones are adherent to the dura over the tegmen tympani, and the cerebellar ones are adherent to the back of the petrous bone in the region of Trautman's triangle. In either situation – and we have had one case with abscesses in both sites – the abscess produces two groups of effects, those due to increased intracranial pressure, and the focal symptoms and signs due to the part of the brain affected. The general symptoms of increased pressure are due partly to the size of the abscess but also, in the early stages in particular, to considerable œdema of the brain surrounding the abscess. This causes headache, nausea, and vomiting, and if unrelieved there is stupor, coma and death, a picture that the older otologists will remember all too well. As I have said, this general picture may be somewhat modified nowadays by the common practice of administering antibiotics and sulphonamides, and an abscess may develop very subtly. It is for this reason that lumbar puncture is of such great value in the early stages of the development of an abscess because it will often reveal a significant but not dangerous increase in pressure and it will almost always show an increase in the protein content and cell count of the fluid. In the later stages, of violent headaches and stupor, when lumbar puncture may be dangerous, there is often papilloedema, but generally it takes two or three weeks for this to develop, and about the same time for the skull sutures to separate in children.

The focal signs of a temporal lobe abscess may be very slight and inconspicuous or highly eloquent, especially if the abscess is in the speech-dominant hemisphere, because some degree of aphasia is very common. This may be slight in degree, the patient only making an occasional mistake in speaking, or using unusual words, or having difficulty in finding the right word. Or it may be so gross that the disordered speech is taken as evidence of dementia and the patient is sent to a mental hospital. The next most common neurological sign is a defect in the visual fields. Because the lower fibres of the optic radiation sweep down in a loop through the temporal lobe, an upper quadrant hemianopia occurs. It may only be possible to detect this on careful perimetry, but in cases of suspected temporal lobe abscess, it is worth doing this because it is almost diagnostic. As the abscess increases in size the hemianopia may become complete and it can then usually be detected by confrontation tests at the bedside. Gross paralysis and sensory defect down the opposite side of the body are uncommon, but as the face and arm are represented in the lower part of the rolandic area, some weakness of the face and arm on the opposite side are the earliest defects to show themselves. It will be seen, thus, that an abscess in the non-dominant temporal lobe may be relatively silent, as the visual field defect may not be detected and the sensorimotor disturbance may be very slight.

In the cerebellum, on the other hand, the signs are more definite. As noted above, headache may be accompanied by insistent giddiness or vertigo. There is often stiffness of the neck, and the head may be held in an unusual posture, tilted to one side or bent forward almost as though to pull the cerebellar tonsils out of the foramen magnum where they are being pushed by the expanding abscess and surrounding œdema. There is usually nystagmus which is slow and coarse on looking to the side of the lesion and rapid and fine on looking to the opposite side. The limbs on the side of the lesion are ataxic in purposive tests, and if the patient can still walk, his gait is unsteady. In the later stages, the speech may become a little thick and slurred and there may be difficulty in swallowing.

When any of these signs, or any combination of them, suggest the possibility of a brain abscess, steps should be taken to confirm or exclude such a lesion. As noted above, lumbar puncture may provide supporting evidence. In the cases of temporal lobe abscess, ordinary X-rays of the skull may show displacement of the pineal gland to the opposite side, and the EEG may reveal a focal discharge. More certainly, a carotid angiogram will show a characteristic upward displacement of the middle cerebral group of vessels. If facilities are not available for this, a ventriculogram will reveal an expanding lesion in the temporal lobe, but as this method may have to be followed by immediate operation on the abscess, it should be deferred until such an operation is possible. Ventriculography is the only special investigation which gives much help with cerebellar abscesses, and it is usually the immediate precursor of operation on the abscess.

The treatment of a brain abscess nowadays is fairly straightforward. Having diagnosed and localized it, a burr hole is made over it and the pus is aspirated through a blunt brain cannula. We then instil 500,000 units of penicillin and 2 c.c. of a radio-opaque solution (either Thorotrast or Steripaque) so that the exact size and situation of the abscess can be followed in X-rays taken at intervals of two to three days. This initial

aspiration usually brings about a striking improvement in the headache, stupor and focal signs, but when and if the headache recurs and focal signs reappear, or when X-rays show that the abscess is larger, another aspiration is done. For the first three or four days the blunt cannula can be pushed through the wound; thereafter a sharp needle has to be used. As a result of these aspirations, there is usually a steady improvement and this means one of two things, that the abscess is shrivelling up, or that it is developing a thick capsule and the surrounding œdema is lessening. The X-rays will tell which is happening, and if the abscess shrivels up to become a small crenated mass, most surgeons would prefer to leave it alone. But before the patient is allowed to go home, as cured, two conditions should be met; a final lumbar puncture should show that the pressure is normal and that the C.S.F. is normal on analysis; and an air encephalogram should be done to show that the ventricular system is normal, and that no undiscovered loculus of the abscess is present.

On the other hand, if the abscess continues to expand after, say, four or five aspirations, it ought to be excised. By this time, usually four to five weeks after the onset of the infection, it has a thick enough wall to allow it to be dissected out of the brain much like a subcortical tumour.

This method of treatment is applicable to most brain abscesses and it has been evolved over the past twenty-two years, with a progressive improvement in the results as set out in Table 2.

Table 2
Brain abscess due to mastoid infection

	1938–1950			1950–1960		
	No. of cases	*Deaths*	*%*	*No. of cases*	*Deaths*	*%*
Temporal lobe	31	10	32	24	1	4
Cerebellum	19	8	42	11	1	9
Total	50	18	36	35	2	5·7

This account of the diagnosis and treatment of a brain abscess may make both sound almost too easy. I realize that this is very far from the case, and that mastoid disease is being dealt with today in hospitals where the facilities are considerably less elaborate than at the Radcliffe Infirmary. There is the otologist, X-ray equipment, and an operating theatre, but no facilities for perimetry, electroencephalography or angiography, and indeed no neurologist or neurosurgeon. And I suppose that is why we are having this discussion to-day, i.e. to help the ear, nose and throat surgeon who has, or thinks he has, a brain abscess on his hands. My advice is quite simple: as the definitive treatment of a brain abscess is a neurosurgical problem, the patient should pass into neurosurgical hands as soon as possible. Moving the patient to a neurosurgical centre means, of course, that he is fit to travel and that a neurosurgical bed is available. In most cases there is no difficulty in deciding about the former, but if the patient is stuporous or comatose or getting worse rapidly, it is not advisable to send him on a long ambulance journey. In these circumstances, it is worth giving urea intravenously. This has a remarkable effect in reducing intracranial pressure and is quite safe. It is given in doses of 1g/kg of body weight in a solution of invert sugar and the effect is manifest within fifteen to twenty minutes. It persists for several hours, and may thus make it possible to move a seriously ill patient with reasonable safety. If the urea is not effective, i.e. in bringing the patient out of stupor, something must be done on the spot and the right thing is to make a clean burr hole in the squamous temporal bone just above the ear, put in a brain cannula and try to aspirate the abscess. In the cerebellum, the burr hole should be made just below the tranverse sinus and midway between the mastoid process and the external occipital protuberance. This needs only three instruments not commonly included in the otologist's kit, a perforator, burr and a brain cannula, and no modern surgical theatre should be without them, even though they are not much used. If the abscess is found, 500,000 units of penicillin in 1 c.c should be injected, and if Thorotrast or Steripaque is available, 2 c.c. of this should be instilled as well, and the wound closed. This is a life-saving procedure and may provide twenty-four to forty-eight hours' safety in arranging transfer to a neurosurgical centre. If there is difficulty or delay in getting into one centre, another should be tried; most neurosurgeons are ready to co-operate.

The critical situation which I have just described may develop rapidly in a mastoid infection under observation in hospital, or the patient may be sent into hospital in such a state, or it may develop after operation. In the first two possibilities, there may be doubt as to which should be dealt with first, the brain abscess or the mastoid. If the patient is in stupor or coma, his life is at risk from increased intracranial pressure and the abscess should be aspirated first, but the mastoid should be done as soon thereafter as possible, i.e. when the patient has recovered from the pressure effect. This may be within a few hours or the next day. If the patient is not so ill, and the diagnosis is in some doubt, it is usually safe to proceed with the mastoid operation and see what effect this has before going any further.

In any case, I would urge again the importance of seeking for a brain abscess through a clean burr hole and with a brain cannula rather than sinus forceps. If these manipulations are carried out through a mastoid wound there is a considerable risk of a cerebral fungus developing, either behind the ear or in the external auditory meatus, depending on the approach to the mastoid. The endaural fungus may be accompanied by cerebrospinal otorrhœa, and this usually means an intracranial operation to seal off the fistula.

If an abscess is not found neurosurgical help becomes more urgent, and may be unavailing, but occasionally ventricular drainage or a decompressing operation may tide the patient over until the diagnosis is established. In this small group there are occasional cases of coincidental mastoid infection and brain tumours, vascular accidents, or metastatic abscesses – all of which we have seen.

Another complication which may look alarming but is fortunately benign is *otitic hydrocephalus*. This is essentially a disease of children and young adults, but can occur at any age. It is thought to be due to defective absorption of cerebrospinal fluid such as might result from thrombosis of one or more of the large intracranial venous sinuses impairing the efficiency of the arachnoid granulations. The clinical effect is to raise the intracranial pressure and this causes headache, papillœdema and elevation of the spinal fluid pressure. These effects may be apparent during the course of a middle ear or mastoid infection or may come after the infection has apparently subsided. It is probable that many cases are undetected, because the pressure effects are rarely critical; headache may be present for a few days, but is not severe, the patient usually looks and feels well otherwise, and only if the optic fundi are examined and a lumbar puncture is done can the fact of increased pressure be established. In most cases, the appearance of a squint due to paralysis of one or both VI nerves leads to the detection of papillœdema and this immediately raises the question of a brain abscess. Such cases need full investigation: although well-being is characteristic of otitic hydrocephalus, I remember a little boy who was apparently quite well with a chronic abscess in the right temporal lobe which contained 100 c.c of pus. Apart from a VI nerve paralysis, there are usually no focal neurological signs in otitic hydrocephalus. The spinal fluid pressure is raised but usually not to the extent which would be expected from the degree of papillœdema: pressures of 200–250 mm are common, whereas in brain abscess pressures of 300–400 mm are usual. The cerebrospinal fluid moreover, contains no cells and usually less protein than normal, i.e. 15–20 mg%. X-rays of the skull and the EEG may be normal. But to exclude an abscess we usually do a carotid angiogram in the first place, and if this is normal, we finally exclude an abscess by a ventriculogram or air encephalogram. When the diagnosis is established, no treatment is called for, as the symptoms usually subside within two or three weeks. The papillœdema may take two or three months to resolve, and a close watch needs to be kept on the visual acuity as, if there is any evidence of deterioration, a decompression operation may be necessary. One little boy in this series lost his sight despite such an operation, but this is the only case in which this has happened.

Cerebral thrombophlebitis may result from a backward extension of thrombosis in a large venous sinus, or it may occur in the course of a mastoid infection not known to be complicated by sinus thrombosis. It also occurs in association with *subdural abscess* and in practice it has to be differentiated from this condition. The spread of thrombosis to cerebral veins is usually signified by the onset of focal epileptic attacks involving one side of the body, and these may occur at such frequent intervals that there is virtually status epilepticus. When the fits have subsided, the patient will be found to be drowsy and the functions of the affected hemisphere will be largely in abeyance. That is to say that in the non-dominant hemisphere there is hemianopia, hemiplegia, and hemianæsthesia, and to this in the dominant hemisphere is added aphasia. This may all happen within a few hours, too rapidly for a brain abscess, for instance. In some cases, the patient does not seem to be as ill as the neurological picture would suggest, and he will probably have an uncomplicated thrombophlebitis from which he will recover. In others there is a severe mastoid infection with general illness, and in these the thrombophlebitis may be a feature of subdural abscess. In either case, the cerebrospinal fluid is not of great help: it may be normal or it may contain a slight excess of protein and a few cells. The most valuable investigation is angiography which will either be normal, or will reveal a subdural collection. These cases often present as emergencies, and in practice we often make exploratory burr holes to exclude a subdural abscess, and follow this with a deliberate angiogram at a later stage to exclude an intracerebral abscess.

If a subdural abscess is found, a number of burr holes are placed in the skull, up to 6 on one side, and small tubes are placed in the subdural space partly for drainage but chiefly to allow the instillation of penicillin. Although this is one of the most serious complications of mastoid disease, if it can be detected early and is due to organisms sensitive to penicillin, recovery is possible.

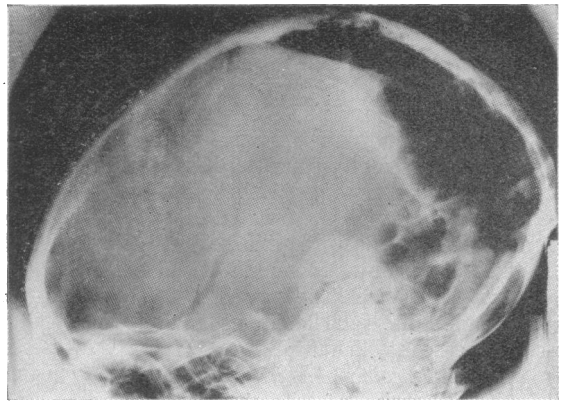

Fig 1A

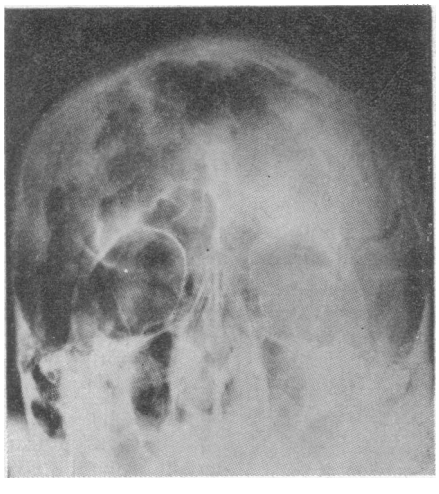

Fig 1B

In conclusion, the following case which appeared unique, may be of interest but whether it was a mastoid infection or injury I am not certain.

Case Report: The patient (R.I.241196) was 33 years old, and came to us in May 1957. In 1948 he had a right-sided middle ear infection with discharge for about three weeks. This resolved and had not recurred. In 1954, he received a blow in the right frontal region which lacerated the scalp but there were no intracranial complications, and the wound healed normally. The only other point in his earlier history was that he had osteomyelitis of the tibia at the age of 11.

In January 1957 he became aware of a soft swelling in the right parietal region which varied in size from time to time and caused few symptoms. He ultimately got into the hands of the late Mr R R Simpson of Hull, who had some X-rays taken and these showed an extensive mottled erosion of the right side of the vault (Fig 1). The Wassermann reaction was negative, and there was no constitutional evidence of infection.

The scalp swellings (Fig 2) were found to contain air and it seemed likely that this was escaping from

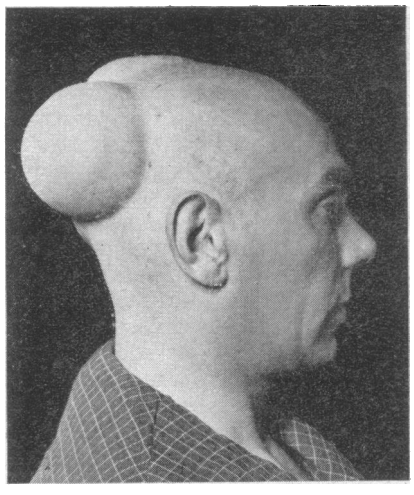

Fig 2A

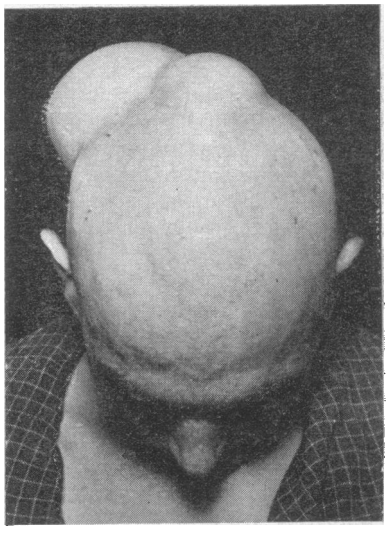

Fig 2B

the mastoid. Mr R G Macbeth and I explored this and found an appearance of the skull vault which I had never seen before: a mottled erosion in places involving the whole thickness and exposing the dura and in other places apparently confined to the inner or outer table. Islands of normal looking bone were seen in the midst of the affected area, and in all it seemed to be neither neoplastic nor inflammatory. Histological examination did not take us much further, except to exclude these possibilities. In any case, Mr Macbeth found a hole in the mastoid which he plugged with a little flap of temporal muscle, and this was effective.

The possibilities are, I suppose, that this was some kind of bony dystrophy which eroded a mastoid cell and allowed the escape of air under the scalp or that the injury fractured a previously

infected cell, and the changes in the bone were due to the pressure of the trapped air.

In recounting these various pathological states, I hope I have made it clear that there is much that the otologist can do alone, but there are some cases in which collaboration with the neurosurgeon is to the general advantage. I have stressed this point on other occasions, but it is more nearly practicable now than it has ever been in the past. There must be few otologists who have not some kind of liaison with a neurosurgical centre, and these I would advise to establish such contact as soon as possible.

Mr J D K Dawes (*Newcastle upon Tyne*)

199 cases of otogenic intracranial complications admitted to the Royal Victoria Infirmary, Newcastle upon Tyne, during the years 1944–1960 are analysed to show the results of treatment, to determine the major remaining problems of clinical practice, and to draw attention to faults in management.

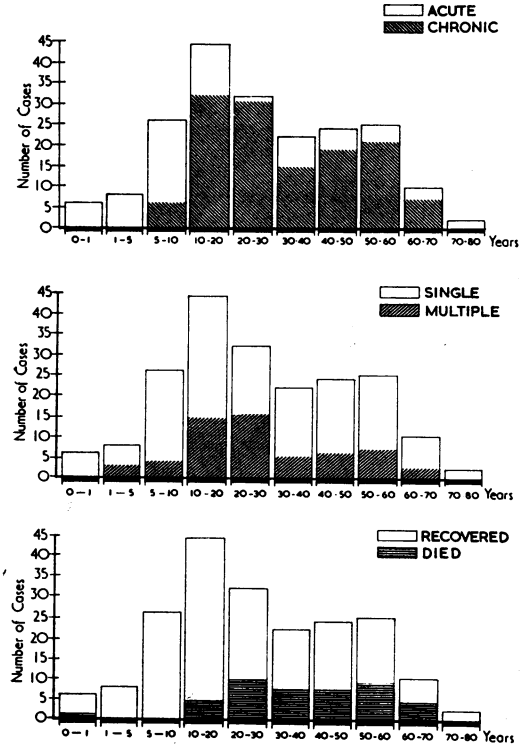

Fig 1 *The age incidence of otogenic intracranial complications*

Fig 1 shows that the greatest incidence of otogenic intracranial suppuration occurs in the 10 – 20 age group, although the lesions occur at all ages. As expected, acute ear disease is the commoner cause in childhood and chronic ear disease in the adolescent and adult. Comparing the three graphs suggests that a multiplicity of intracranial lesions and death more commonly follow chronic suppurative otitis media.

Fig 2 stresses the great improvement in the mortality rate over the years, although the incidence of intracranial suppuration has only recently become slightly less. This improvement is undoubtedly due to the more efficient use of antibiotic therapy. One of the 4 deaths during the past seven years was that of a 1-month-old child who died from an undiagnosed extensive intracranial sinus thrombosis. Throughout this series the highest death-rate has always occurred in those patients with multiple intracranial complications of chronic ear disease (as marked by circles in Figs 2, 3 & 4). Using a similar method I will now consider the major intracranial complications individually (Figs 3 & 4).

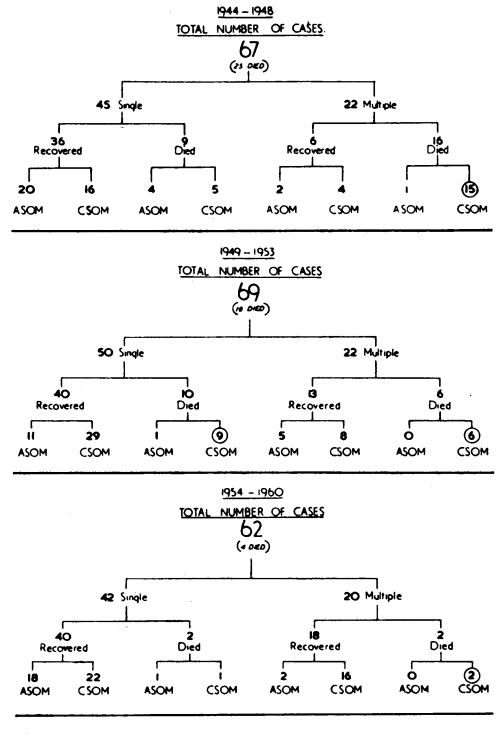

Fig 2 *The incidence of the varied types of otogenic intracranial suppuration and mortality rate grouped according to the years of occurrence*

97 patients had proved intracranial sinus thrombophlebitis, 89 being confined to the lateral and transverse sinuses or jugular bulb, and in three of these the thrombosis had extended into the

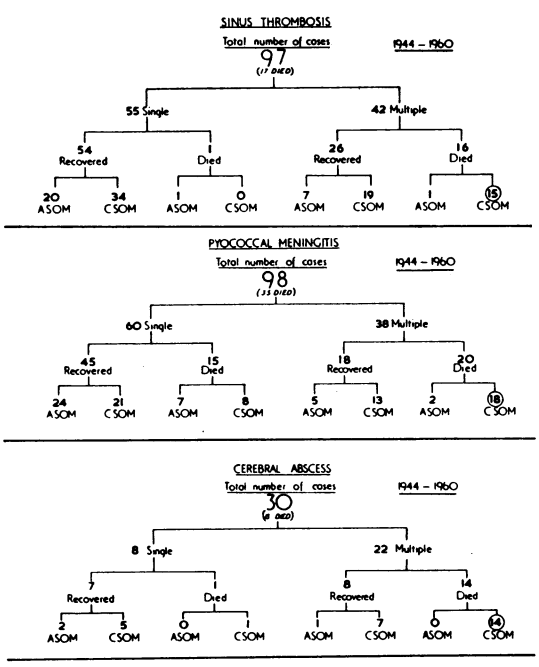

Fig 3

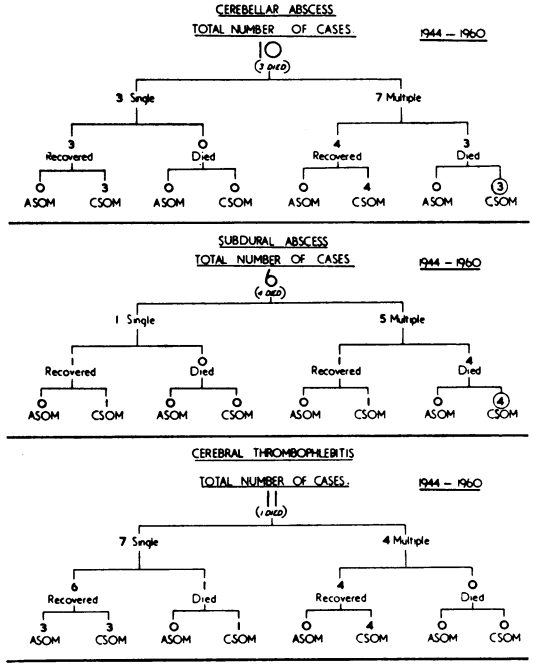

Fig 4

Figs 3 & 4 *The incidence and results of treatment of each major type of intracranial complication*

superior petrosal sinus, and in one beyond this into the cavernous sinus. Included are 7 cases of isolated superior petrosal sinus thrombosis, one of which had extended into the cavernous sinus. 55 patients had involvement of the intracranial venous sinuses alone with or without perisinus abscess, whereas in 42 patients the sinus thrombosis was associated with lesions in the cerebrum, cerebellum, meninges or subdural space. When it is a single complication of ear disease, acute or chronic, the prognosis of sinus thrombophlebitis is excellent, provided that correct antibiotic therapy is given and the ear lesion is brought under control. Rarely is there need for surgery when the thrombophlebitis complicates acute otitis media, since vigorous antibiotic therapy controls the intracranial complication as well as the ear disease. Consequently sinus thrombophlebitis may be suspected but infrequently diagnosed as an isolated complication of acute ear disease. 5 cases of otitic hydrocephalus occurred in the series; all recovered and all were associated with sinus thrombophlebitis. Control of the meningitic and cerebral abscess group has been much less effective and the high mortality requires explanation.

In this series no case of cerebellar abscess or subdural abscess was found to complicate acute ear disease. The mortality rate of subdural abscess was high because the diagnosis was made just before death or at autopsy. It is worth noting that 6 of the 7 cerebellar abscesses in the multiple

Table 1

Causes of death in otogenic meningitis

	Uncontrolled infection	*Late diagnosis*	*Unexplained*	*Mismanagement*
A.S.O.M. 9 deaths	6 (all prior to 1949)	2 moribund	1	
C.S.O.M. 26 deaths	6 (5 Prior to 1947)	15 { 4 moribund; 8 temporal lobe abscesses; 2 cerebellar abscesses; 1 subdural abscess }	3	2

Table 2

Causes of death in otogenic temporal lobe abscess

	Uncontrolled infection	*Late or no diagnosis*
1944–1948 8 deaths (62% mortality)	3 { 2 pyæmias; 1 meningitis }	5 { 1 moribund; 2 meningitis; 1 subdural undiagnosed; 1 undiagnosed }
1949–1953 5 deaths (71% mortality)	1	4 { 1 moribund; 2 meningitis with late diagnosis; 1 pyæmia }
1954–1960 2 deaths (22% mortality)		2 Both meningitis with late diagnosis

group and all cases of cerebellar thrombophlebitis were associated with and presumably were consequent on lateral sinus thrombosis. The prognosis in cerebral and cerebellar thrombophlebitis is excellent; the only death, and this in 1944, was caused by a phlegmonous encephalitis.

Once again these figures stress the importance of the multiple intracranial lesion complicating chronic ear disease.

Seeking to explain the overall high mortality, a more detailed method of analysis has been used, and I have chosen to show the results of this in meningitis and temporal lobe abscess (Tables 1 and 2). All the uncontrolled meningitic infections occurred early in the series when the antibiotics were given in small dosage and their variety and efficiency were limited. Several patients were sent into hospital moribund and on other occasions the presence of a lethal lesion such as cerebral abscess or subdural abscess remained undiagnosed and untreated. Pyococci were found in the cerebrospinal fluid in only 28 cases and in 2 of those who died *B. pyocyaneus* was also cultured. The finding of bacteria in the cerebrospinal fluid did not appear to influence the mortality rate and the low incidence may be because the patients were given chemotherapeutic or antibiotic agents prior to admission. Similarly the deaths of patients having temporal lobe abscess (of whom 50% died) were caused by uncontrollable infections, particularly in the early days, or a failure to make a diagnosis at least till late in the illness. Physical signs of severe meningeal irritation often obscured the diagnosis of a brain abscess until the meningitis was largely under control. Unfortunately the meningeal irritation in some was a leaking brain abscess, particularly when the meningitis had been preceded by more than a week's history of headache and vomiting. The patient whose abscess leaked into the ventricle as illustrated by Thorotrast in Fig 5 had a history of six weeks' earache and four weeks' headache and vomiting prior to the sudden development of a meningitis which brought him to hospital. Only prompt treatment saved his life.

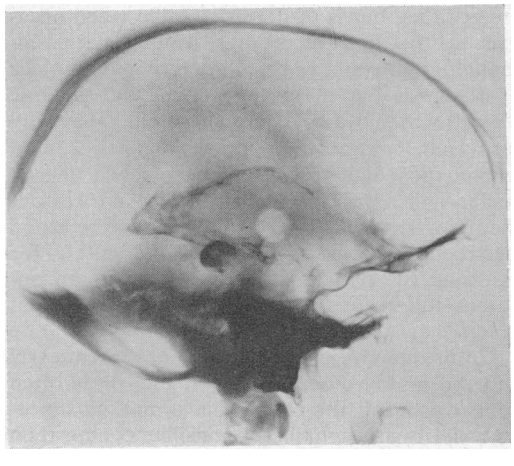

Fig 5 *A radiograph illustrating that Thorotrast has leaked from the abscess into the ventricle. The temporal burr-hole is also seen*

These figures illustrate that sinus thrombophlebitis and meningitis are now controllable by antibiotic therapy and that the main problem lies in the management of cerebral, cerebellar and subdural abscesses. These abscesses have often followed on, or been associated with, other intracranial lesions. Cerebral and cerebellar abscesses are preceded for at least three to four days by a thrombophlebitis, and subdural abscess usually occurs late in the illness. Therefore, if effective treatment is undertaken soon after the onset of an intracranial complication it should be possible to prevent the development of lethal lesions. The earlier the diagnosis, the better is the prognosis.

In most cases a diagnosis can be reached within the first forty-eight hours. Even if the lesion cannot be labelled accurately the suspicion that an intracranial lesion has occurred should provide sufficient knowledge to begin active therapy. What is merely suspicion is often confirmed later.

A meticulous chronological history of events is essential to accurate diagnosis (Table 3). The simple fact that the ear is producing symptoms or signs other than otological ones or even that the patient himself is unwell means that the suppuration has extended beyond the middle ear cleft. In chronic ear disease, earache alone means something has gone wrong and if pus is under pressure within the middle ear cleft an intracranial lesion may be impending. Headache and vomiting, the symptoms of raised intracranial pressure, indicate that an intracranial lesion has already started. A raised temperature with a chronic ear or late in the course of acute ear disease suggests a meningitis or sinus thrombosis; if the temperature swings or rigors develop then

Table 3

Early symptoms and signs of otogenic intracranial complications

Patient is unwell	
Earache	Epileptic fits
Headache	Nominal aphasia
Vomiting	Neck rigidity
Swinging temperature or rigors	Nystagmus
Drowsiness – coma	Temperature and pulse charts

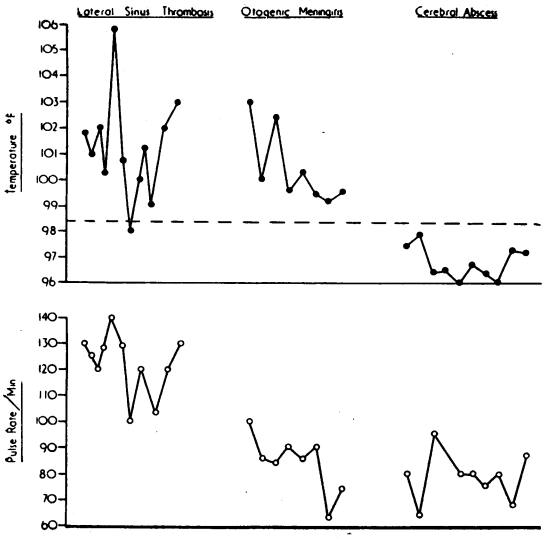

Fig 6 *Portions of temperature charts from three recent cases. The upper charts show temperature in degrees F and the lower chart pulse-rate per minute.*

the patient without doubt has a sinus thrombophlebitis. On the other hand when the temperature remains raised with a relatively slow pulse the patient probably has meningitis, or if the pulse-rate and temperature are subnormal a cerebral or cerebellar abscess has probably formed. Fig 6 illustrates these features from parts of the temperature charts of 3 recent patients. Slight drowsiness means that brain tissue is involved unless proved otherwise. Epileptic fits are certain evidence of cortical irritation and when they occur early probably signify the presence of a cerebral thrombophlebitis; but, if the fits are not controlled fairly quickly by adequate antibiotic therapy the patient should be regarded as having a subdural abscess. Nominal aphasia points to a lesion in the dominant hemisphere. However, the opposite temporosphenoidal lobe is silent and the diagnosis must be suspected on the general picture, and in a co-operative patient by testing the visual fields. Neck rigidity obviously means meningeal irritation but only the time of its development in the history will indicate whether this is a single lesion or a leaking abscess. Nystagmus suggests a labyrinthine erosion or a cerebellar lesion but it is only the accompanying findings which allow the clinician to see the sign in its correct perspective. Other physical signs are late in appearing in the natural history of the intracranial extension, although only a few days may have elapsed in the chronological history. Frequently a tender jugular vein and upper deep cervical nodes and occasionally a subperiosteal abscess over the parietal and occipital bones have been seen in sinus thrombosis. The extent of tenderness over the jugular vein is often a useful sign of the lowest level reached by the thrombophlebitis. A progressing hemiplegia indicates a large abscess in the brain or subdural space, and deep coma with fixed dilated pupils a gross supratentorial hydrocephalus. Papillœdema is usually a late sign, taking two to three weeks to develop in the case of cerebral abscess, but when papillœdema and a raised intracranial pressure appear early and are the only findings the patient probably has the so-called otitic hydrocephalus which in my opinion means no more than that the larger lateral or transverse sinus or the sagittal sinus has become thrombosed.

Lumbar puncture is a useful method of confirming a clinical diagnosis already suspected. Sinus thrombosis occasionally produces raised cerebrospinal fluid pressure, a frank meningitis is obvious enough but even a small number of cells in the fluid is not inconsistent with the presence of brain abscess. Tobey-Ayer and Queckenstedt tests are of no value, for positive or negative findings are not conclusive. When a patient has meningeal irritation, a smear of the cerebrospinal fluid should be examined to determine whether cocci or bacilli are present. If cocci are found, penicillin, streptomycin, or sulphonamides are indicated, whereas if bacilli, chloromycetin or sulphonamides should be used. When no organisms are found penicillin and streptomycin have been used. On two occasions Gram-negative cocci were found but these turned out to be degenerate Gram-positive cocci as determined by culture. To await positive culture of the cerebrospinal fluid or of blood culture in the case of sinus thrombosis before starting treatment is to court disaster.

Electroencephalography is useful when findings are positive, but is of doubtful value when negative at the first recording. Angiography, encephalography and ventriculography have a place in diagnosis but in my experience by the time they are used the patient has already partially recovered from a major intracranial lesion, and in fact most diagnoses have been made or suspected and active treatment undertaken without their aid. Testing of the visual fields and repeated electroencephalography are particularly useful for assessing the patient after he has made an apparent full recovery from a major intracranial lesion.

Unfortunately, all patients do not present early and the timing of operation on the ear is often controversial. If the patient has a marked supratentorial hydrocephalus or tonsillar coning then the cerebral or cerebellar abscess requires urgent treatment and the ear must take second place. In a temporal lobe abscess, a temporal burr is the most direct approach and should be within the

compass of the otolaryngologist. This approach is through a clean field and also avoids the development of cerebral herniation. A cerebellar abscess is usually situated in the antero-superior part of the lobe and I think that the direct approach through Trautmann's triangle or the lateral sinus is as good as, if not better than, the suboccipital approach – a suboccipital tap in the hands of the inexperienced is more likely to miss the abscess. The majority of the recoveries in cerebellar abscess in this small series were obtained by transmastoid tapping. A limited inspection of the middle and posterior fossæ by the neurosurgeon through a burr hole, although important, cannot deal with the ear and the associated extradural disease or sinus thrombosis.

The ear itself is very important, for it is the source of the infection and in uncontrolled acute and chronic ear disease surgical exploration is essential. A persistent nidus for bacteria, such as cholesteatoma, is untouched by antibiotics and the early removal of this source eliminates the possibility of recurrent infection of the intracranial tissue planes and frequently shortens the length of the illness. The bacteria causing the intracranial lesions are most commonly a pyococcus, occasionally *H. influenzæ*, *B. coli* or *pyocyaneus*. I therefore always treat these infections primarily with penicillin and streptomycin or sulphonamides and within forty-eight hours explore the ear if advisable. Dramatic improvement has frequently followed the removal of the septic focus. Time prevents my quoting many of the numerous instances when a patient who is deteriorating or whose lesion has relapsed on antibiotic treatment alone, improves within a few hours after the operation. At operation, the lateral sinus and middle fossa dura are deliberately exposed even if not thought to be involved, for frequently a middle fossa extradural abscess is associated with a lateral sinus thrombosis and many cases of temporal lobe abscess were associated with lateral sinus and superior petrosal sinus thrombosis. Ideally, all otogenic intracranial lesions should be managed in a hospital where an otologist and a neurosurgeon are readily available and work in co-operation. The post-operative care is not easy and frequent examination and careful assessment by skilled clinicians, senior and junior alike, are essential to the well-being of the patient.

Acknowledgments: I would like to thank Mr F McGuckin and Mr Munro Black for allowing me to include the cases admitted under their care and also permitting me to treat many of them during the last seven years. Mr D J Hammersley of the Department of Surgery kindly drew the histograms.

Mr J Angell James (*Bristol*) said that he was impressed by the large number of cases of intracranial complication of otogenic origin which Mr Dawes had collected from the Royal Victoria Infirmary. Did these numbers include all the cases seen in the neurosurgical unit also? In the E.N.T. Department of the United Bristol Hospitals the number was much smaller. He had looked up the number seen in six years, from 1929 to 1935 inclusive, and compared it with the number seen from 1954 to 1959 inclusive. In the presulphonamide period there were 56, and in the last six years only 17, the total number of beds in the Department being 60. He thought the experience in Newcastle must be exceptional. In view of the small number of intracranial complications seen in the average E.N.T. clinic he thought it was important that registrars in training should be encouraged to spend time in either a neurosurgical or neurological unit in order to gather enough experience of these very important problems. He particularly welcomed Mr Pennybacker's suggestion for the first-aid treatment with urea for cases in bad condition in preparation for transport to a distant neurosurgical unit. In the South West Region this type of case might have to travel 200 miles.

Mr J W Dixon (*Glasgow*) wondered how urgent we should regard the common case of chronic otitis media with cholesteatoma and foul discharge, but without any symptoms or signs of intracranial extension. Though very few of these might subsequently develop intracranial complications, some of those that did so still lost their lives even in the antibiotic era. Perhaps these cases should be dealt with as semi-emergencies even if this meant that there was only time to render the ear safe and that possibly tympanoplasty would have to be left for a second-stage operation later.

He was prompted to say this by the case of a young man who developed symptoms of intracranial extension after a short period on the waiting list. Though then admitted immediately and treated with neurosurgical advice right from the start, the patient subsequently died.

Mr J Fulton Christie (*Glasgow*) said the question of timing of the mastoid operation in these cases was important, although not vital. He was old enough to remember the pre-antibiotic days when the first step was to operate on the diseased mastoid. When antibiotics became available and the neurosurgical unit was established in the West of Scotland the intracranial complication, the condition dangerous to life, was naturally dealt with first. The pendulum probably swung too far in the opposite direction and the mastoid operation was often delayed until the meningitis or brain

abscess had been cleared up, and sometimes even until an abscess capsule had been excised. His view at present was that in the first place the infection should be controlled by antibiotics and the intracranial pressure reduced where necessary, by abscess tap, &c. The mastoid operation should then be done as soon as the patient was in a fit condition for the administration of a general anæsthetic.

He was pleased to hear Mr Pennybacker advise ventriculography as he (Fulton Christie) thought that every patient who had been treated for brain abscess or meningitis ought to have a ventriculogram before being dismissed from hospital. He knew of some cases where this had been omitted because the patient appeared to be so well, and the patient had to be readmitted within a very short time, as a second abscess or small loculus had been overlooked and had flared up.

He was surprised that the question of a dead labyrinth had not been mentioned, he wondered what Members' views were on this. Personally he believed that whenever a dead labyrinth was found it should be opened freely above and below the facial nerve.

Mr J D K Dawes, in reply to Mr Angell James, said that 14 intracranial complications of ear disease had been admitted in 1960. The mortality rate was high because many patients were not seen by the otologist or neurosurgeon, and many others arrived for treatment in a moribund state. In defence of transmastoid tapping of a cerebellar abscess, Mr Dawes did not advocate this in the hands of the neurosurgeon but only as a familiar approach to the otologist in an emergency. The 2 cases of cerebellar fungus shown by Mr. Pennybacker appeared to have had a cerebellar exposure posterolateral to the lateral sinus and the dural opening was excessive. Mr Dawes had never seen a cerebellar fungus result from cerebellar tapping through Trautmann's triangle. Although only the major intracranial lesions had been considered, 2 cases of subarachnoid cyst occurred in the series; both settled satisfactorily with tapping by a brain cannula.

Giving due consideration to Mr McGuckin's recent Annual Address at the Institute of Otology (1960, unpublished) on the subject of chronic ear disease and keratosis, Mr Dawes suggested to Mr Dixon that it was essential to examine the ear carefully under a microscope and to discuss fully the indications for surgery with the patient. The hearing might be preserved or improved; a dry ear might be obtained, and there was an excellent chance of preventing the development of an intracranial lesion at a later date. Since his waiting list for an endaural mastoidectomy was almost eighteen months, all patients with potentially dangerous ears were kept under observation in the outpatient department during the waiting period. Even so, the general practitioner must still be responsible for recognizing the emergency between visits.

Penicillin had been used intramuscularly, in a dose of 1,000,000 units six-hourly, and intrathecally in cases of meningitis and brain abscess in a dosage of 5,000 to 10,000 units. The only catastrophe he had seen was due to the accidental injection of 5,000,000 units intrathecally. Mr Dawes had never needed to tie an internal jugular vein for sinus thrombosis, and had not interfered with a solid or mural clot, opening the sinus only when its wall was necrotic or when the lumen contained pus.

He agreed that the killing lesion should be treated first. The labyrinth had only on two occasions been suspected as a cause of intracranial invasion in this series, and as a rule he had dealt with the labyrinth on its own merits.