Otogenic intracranial complications

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Introduction

There has been a marked decrease in the incidence of intracranial disease of otogenic origin during the last two decades. Few reports have equalled the large numbers studied by Dawes (1961) and Pennybacker (1961).

Newlands (1965*a*) reported the results of management of eighty cases of brain abscess seen over a ten-year period between 1953 and 1962, with an overall mortality of 27·5 per cent. Yadav and Bhatia (1968) discussed the morbidity and mortality in twenty-one brain abscesses seen at Lucknow Hospital, India, from 1956 to 1964. They reported five deaths in this series.

Our recent experience in finding four silent brain abscesses during mastoid surgery has led us to review the otogenic intracranial complications seen in the Departments of Otolaryngology and Neurosurgery at the Radcliffe Infirmary, Oxford, over an eleven-year period from 1961 to 1971. We believe these figures to be especially interesting as they can be directly compared with data previously given from these departments for periods from 1938 to 1950, and 1950 to 1960 (Pennybacker, 1948 and 1961).

A total of forty-one complications was seen in thirty-six patients. This included patients referred to the Department of Neurosurgery from the region and subsequently operated upon in the Department of Otolaryngology at the Radcliffe Infirmary, in addition to cases which occurred locally (Table I).

Brain abscess was the commonest condition seen: meningitis was second in order of frequency. The other complications were rare. Two deaths occurred in these thirty-six patients. One was due to pneumonia following drainage of a subdural abscess and the other was due to posterior fossa haemorrhage from a ruptured arteriovenous malformation in a patient with meningitis.

Brain abscess

Of the eighteen brain abscesses seen during the period under review, fourteen occurred in the temporal lobe, and four in the cerebellum. This proportion of cerebellar to temporal lobe abscesses has remained constant

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through three decades, although the total number of cases has fallen dramatically (Table II).

TABLE I

ANALYSIS OF 36 INTRACRANIAL COMPLICATIONS OF OTOGENIC ORIGIN SEEN AT THE RADCLIFFE INFIRMARY, OXFORD, BETWEEN 1961 AND 1971

Complication	Number	Deaths
Brain abscess	18	0
Subdural abscess	2	1
Meningitis	9	1
Benign intracranial hypertension (otitic hydrocephalus)	3	0
Lateral sinus thrombosis	2	0
Cerebral thrombophlebitis	1	0
Cerebral hernia	1	0
Total =	36	2

TABLE II.

NUMBER OF CASES AND MORTALITY OF OTOGENIC BRAIN ABSCESSES SEEN AND TREATED AT THE RADCLIFFE INFORMARY, OXFORD, DURING THE LAST THREE DECADES

	1938 to 1949		1950 to 1960		1961 to 1971	
	Number	Deaths	Number	Deaths	Number	Deaths
Temporal lobe	31	10	24	1	14	0
Cerebellar	19	8	11	1	4	0
Total	50	18	35	2	18	0

All patients had a discharging ear on admission to hospital. Two patients had unresolved acute otitis media, and the remainder had had chronic ear discharge for a number of years (Table III). Five patients had been seen previously in ear, nose and throat departments and four had undergone a form of mastoid surgery. No patients were on a waiting list for surgery when their complication developed.

The earliest clinical feature was headache, accompanied by nausea and vomiting, followed by the rapid onset of focal neurological signs (Table III). Lumbar puncture performed in those patients without papilloedema showed a raised cerebrospinal fluid pressure, increased protein, and a raised white cell or lymphocyte count.

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TABLE III.

CLINICAL FEATURES OF 18 CASES OF OTOGENIC BRAIN ABSCESS SEEN DURING THE PERIOD UNDER REVIEW. HEADACHE WAS THE COMMONEST PRESENTING SYMPTOM

Site	Case	Duration of ototthoea	Focal signs or symptoms	Other complications	Residual disability
Temporal lobe	1	15 years	Hemiplegia	Meningitis	Limb weakness
	2	10 years	Hemiplegia	Meningitis	Epilepsy
	3	4 years	Silent	Nil	Nil
	4	12 years	Dysphasia	Nil	Dysphasia and hemiplegia
	5	10 years	Hemiplegia	Nil	Limb weakness
	6	6 months	Hemianopia	Meningitis	Nil
	7	9 years	Silent	Meningitis	Epilepsy
	8	2 years	Silent	Nil	Nil
	9	2 weeks	Dysphasia	Facial palsy	Dysphasia and facial palsy
	10	Unknown	Dysphasia	Nil	Squamous carcinoma in cavity
	11	1 month	Dysphasia	Nil	Nil
	12	Many years	Hemianopia	Meningitis	Nil
	13	18 months	Silent	Nil	Nil
	14	4 weeks	Dysphasia	Nil	Nil
Cerebellar	1	8 years	Ataxia	Meningitis	Ataxia
	2	4 years	Hemianopia	Nil	Nil
	3	20 years	Ataxia	Nil	Nil
	4	7 years	Ataxia	Meningitis	Nil

Radiological investigation included plain skull films, cerebral angiography and air encephalography. Plain films were of value only when the pineal gland was calcified and showed a shift from the midline (Fig. 1), or where infection was due to gas producing organisms and gas shadows could be seen in the brain (Fig. 2). Cerebral angiography frequently showed distortion of the normal pattern of the cerebral vessels (Fig. 3) and a shift from the midline. Air encephalography demonstrated abnormal features in the ventricles of the brain (Fig. 4).

All patients were treated with parenteral antibiotics. Burr holes were created and pus aspirated via a brain cannula. Following aspiration, 100,000 units of soluble Penicillin and 2 ml. of Thorotrast were inserted into the cavity so that shrinkage of the abscess could be monitored on subsequent skull radiographs.

In nine cases radical mastoid surgery directly followed neurosurgery thus obviating the need for a second general anaesthetic. However, in those severely ill patients in whom prolonged anaesthesia was contra-indicated, mastoid surgery was deferred for up to 72 hours.

Some difficulties encountered in diagnosis, and the method of treatment, are well illustrated by the following case report:

B.C., aged 22 years.

This patient was admitted to a hospital medical ward as an emergency with an eight-day history of severe left-sided headache and mild neck stiffness.

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He had had intermittent left ear discharge for one year. On examination there were no focal neurological signs, but an ear swab grew Coliforms and Pseudomonas. Lumbar puncture showed 300 white cells per cu. mm. with 90 per cent lymphocytes and no growth on culture. A diagnosis of viral meningitis was made and he was discharged home to convalesce after five days.

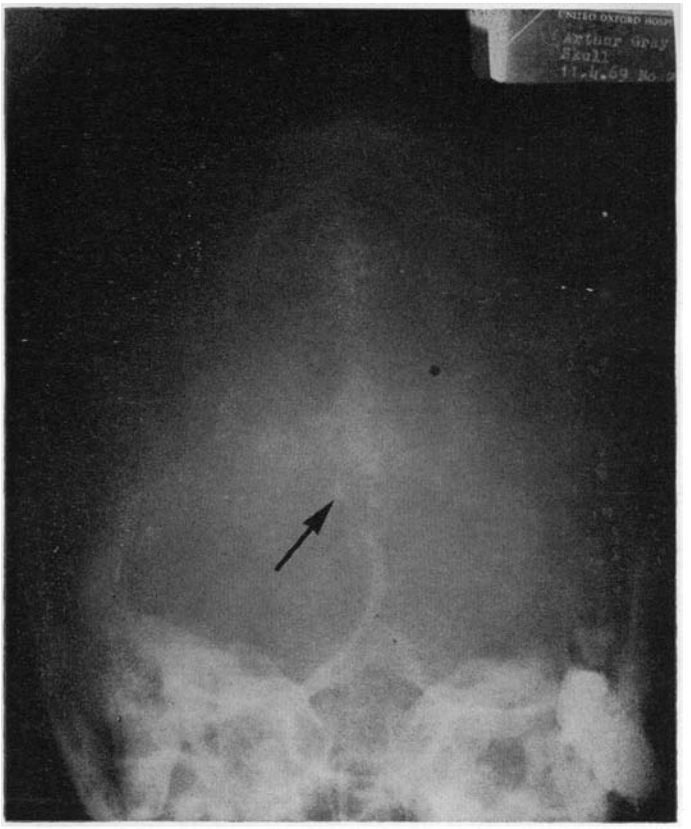


FIG. 1.

Case 10. Brain abscess. Plain skull radiograph showing a calcified pineal gland with an 8 mm. shift to the right side. Radio opaque gauze in the left ear.

One week later he had difficulty in finding words when speaking and asked for a sausage when he wanted an apple. He also started bumping into objects on his right side and became irritable and withdrawn. On re-admission to hospital lumbar puncture was performed and revealed a cerebrospinal fluid pressure of 260 mm. Hg., 40 lymphocytes, and the protein level was 45 mg. per cent. He was given intravenous fluids while under investigation, but became drowsy two days later and was therefore transferred.

On arrival at the Radcliffe Infirmary he was drowsy and dysphasic. There was a complete right homonymous hemianopia. The right pupil was dilated and the right arm and leg were weak. There was profuse foul cholesteatomatous discharge from the left ear.

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A left carotid angiogram was performed and showed elevation of the middle cerebral vessels. Brain needling was then carried out via a left temporal burr hole and 40 ml. of foul pus were aspirated. This was replaced with one mega unit of Penicillin and 2ml. of Micropaque to outline the abscess cavity (Fig. 5). Culture produced a Providence A species of Bacterium associated with an anaerobic streptococcus. A further aspiration was performed 24 hours later.

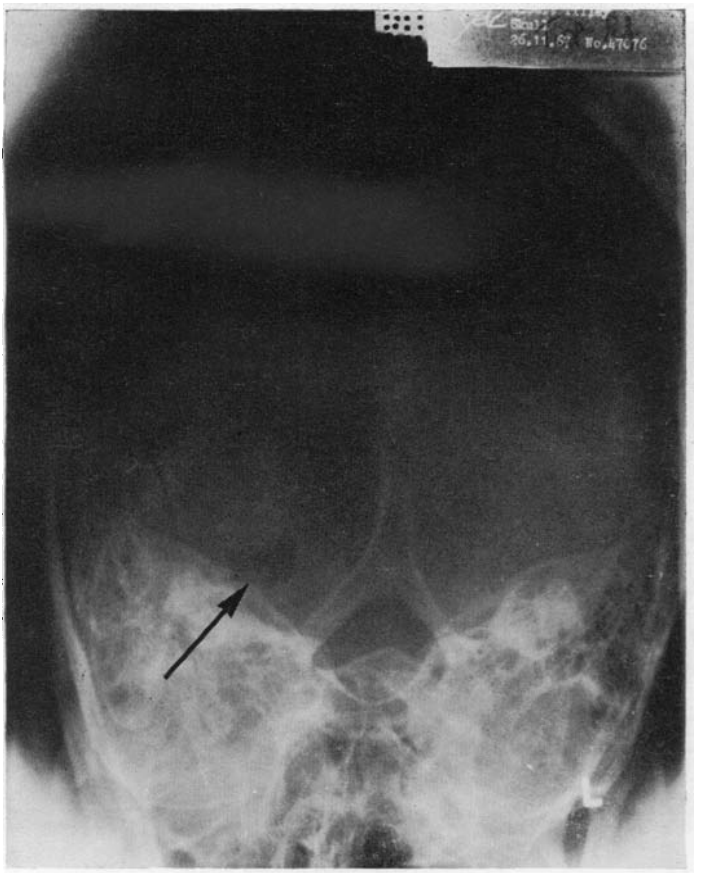


FIG. 2.

Case 2. Cerebellar abscess. Plain skull radiograph showing opaque right mastoid air cells and intracranial gas related to the right petrous apex.

Initial improvement was followed by sudden drowsiness on the third day of treatment, and a left-sided temporal decompression was performed. Under the same general anaesthetic he had a left radical mastoidectomy and a large cholesteatoma was removed. This had eroded the tegmen and exposed the dura over a wide area.

He was treated with Penicillin 1 mega unit and Ampicillin 500 mg. intramuscularly 6 hourly and made a satisfactory recovery with no residual dis-

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ability. Epanutin 50 mg. b.d. and Phenobarbitone 30 mg. b.d. were given for six months as prophylactic anticonvulsants.

Four patients had silent brain abscesses found in the temporal lobe at mastoid surgery. They were asymptomatic apart from otorrhoea of long duration. In each case there was a large defect in the tegmen with a cavity

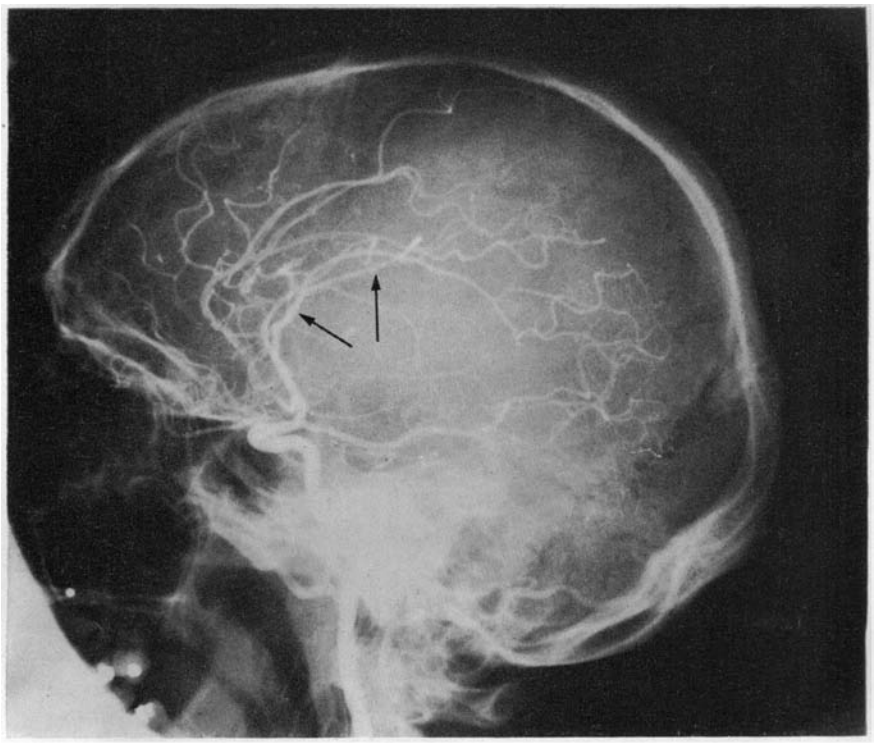


FIG. 3.

Case 6. Brain abscess. Carotid angiogram. There is a large avascular area in the left temporal reion with displacement of vessels upwards and forwards.

above containing granulation tissue, pus, and necrotic brain. Radiographs taken after instillation of contrast medium showed a discrete walled-off cavity in each patient. In one case, however, there was communication with the subarachnoid space and contrast medium spread over the surface of the brain (Fig. 6). This illustrated clearly a possible mode of development of meningitis in these cases. The abscess cavities shrank rapidly following radical mastoid surgery.

There were no deaths due to temporal lobe or cerebellar abscess. However, these conditions were accompanied by considerable morbidity, such as hemiplegia, ataxia and epilepsy (Table III).

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Otogenic intracranial complications

Subdural abscess

This condition is rare as a complication of mastoid disease. Newlands (1965*b*) reported three cases from his series of eighty brain abscesses seen during an earlier decade. Our two patients with this complication had a long history of ear discharge. They were admitted as emergencies with pyrexia, neck stiffness, coma and major convulsions. Both patients had a hemiplegia contralateral to the side of the discharging ear. The diagnosis was made on carotid angiography which demonstrated the characteristic inward displacement of cortical vessels.

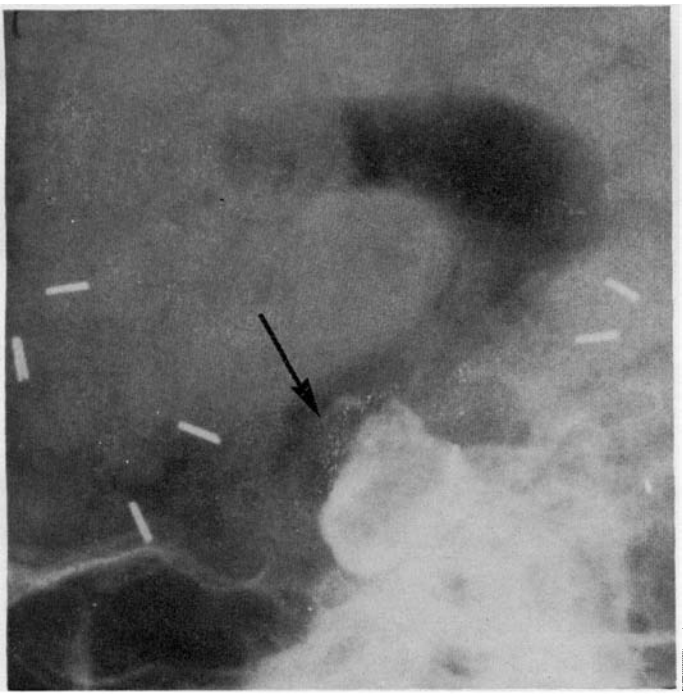


FIG. 4.

Case 6. Brain abscess. Air Encephalogram. There is dilatation of the temporal horn with indentation by the abscess (residual pyogram seen).

These patients were treated by burr hole aspiration of pus from the subdural space and radical mastoidectomy as a combined procedure. A soft rubber catheter was left in the subdural space for irrigation with Penicillin solution. One patient remained deeply comatose and died with bronchopneumonia; the other made a good recovery. Proteus Mirabilis was cultured from the aspirate and ear canal in both cases.

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Meningitis

There were nine cases of meningitis with one death and some patients were left with a residual disability (Table IV). Two cases followed acute otitis media due to a Pneumococcus type 3, and there was no previous history of ear disease. Two patients had undergone mastoid surgery immediately prior to the onset of meningitis. One had a fistula into the

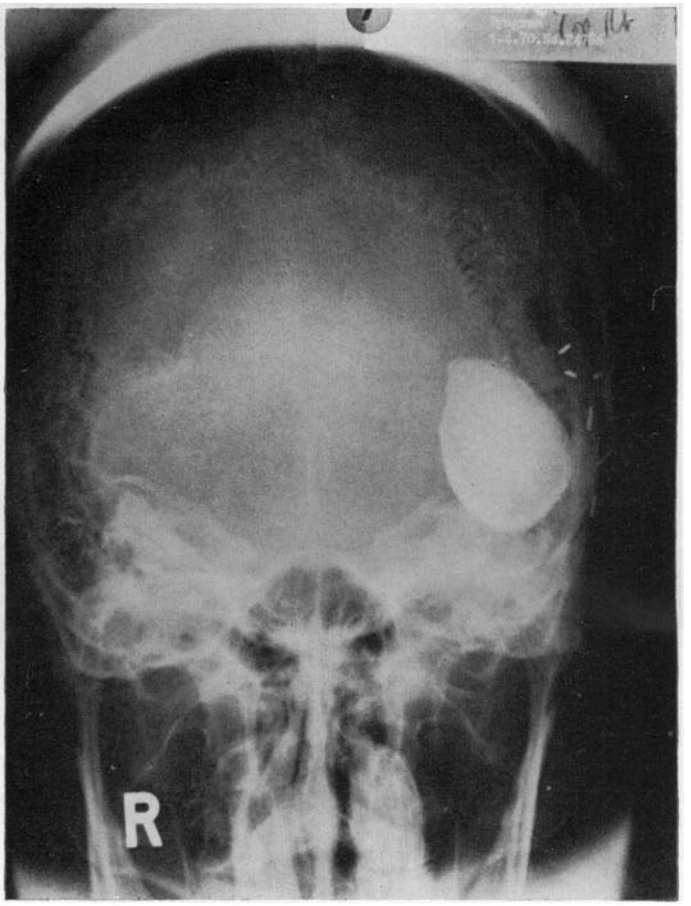


FIG. 5.
Case B.C. Pyogram showing large temporal lobe abscess.

lateral canal and the other had erosion of the promontory. They suffered a total sensorineural hearing loss in the diseased ear, presumably due to a suppurative labyrinthitis. Four of the remaining five patients had been seen in ear, nose and throat departments and the discharge managed conservatively. This shows the possible danger of conservative management of attic cholesteatoma with repeated suction clearance via the

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meatus, as unseen bone erosion may occur. Routine mastoid radiographs may not show a bony deficit due to cholesteatoma and in doubtful cases mastoid tomography may be helpful (Brünner and Sandberg, 1970)

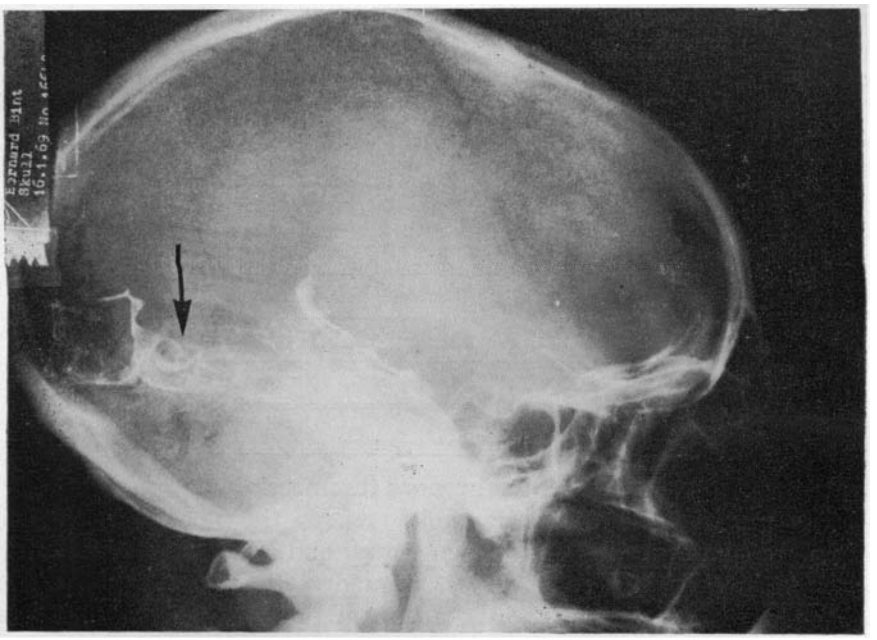


FIG. 6.

Case 7. Silent brain abscess. Contrast injected via the tegmen plate into the abscess cavity has spread into the subarachnoid space around the brain stem and the right temporal lobe.

The presenting symptoms were earache, headache and neck stiffness with ipsilateral ear discharge. One patient with a dry ear had an acutely inflamed tympanic membrane and a conductive hearing loss. Four cases were due to a Pneumococcus, while one was of Streptococcal and another of Staphylococcal origin. In three dases no organisms were isolated from the ear discharge or the cerebrospinal fluid, possibly due to antibiotic treatment from the family doctor before admission to hospital.

All patients received intramuscular or intravenous Penicillin combined with Sulphadiazine or Ampicillin. Daily lumbar punctures were performed initially and intrathecal Penicillin was also given. Mastoid surgery was performed on seven patients with chronic ear discharge as soon as their clinical condition had sufficiently improved, and in all cases there was dural exposure due to mastoid disease (Table IV). Oral antibiotics were continued for one month following discharge from hospital as a prophylaxis against micro-abscess formation and recurrent meningitis. One patient died from a posterior fossa haemorrhage soon after mastoid surgery due to rupture of a brain stem arterio-venous malformation.

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TABLE IV.
CLINICAL FEATURES AND RESIDUAL DISABILITIES IN 9 CASES OF OTOGENIC MENINGITIS SEEN BETWEEN 1961 AND 1971.

Case	Duration of otorrhoea	Residual disability	Surgical findings
1	2 years	Hemiplegia	Dural exposure
2	20 years	Dysarthria	Dural exposure
3	1 month	Facial palsy	Tegmen intact
4	12 years	Dead ear	Fistula
5	4 years	Nil	Tegmen intact
6	20 years	Dead ear	Fistula
7	5 days	Nil	Tegmen intact
8	7 days	Dead ear	No fistula
9	6 years	Died	Dural exposure

Intracranial venous thrombosis

The manifestations of intracranial venous thrombosis of otogenic origin are commoner in younger age groups and all but one of our cases occurred in children aged 10 years or under (Table V). Two patients presented with acute mastoiditis, abducent nerve palsy and unilateral papilloedema with retinal engorgement. Emergency mastoidectomy was performed following angiographic studies and infected thrombus removed from the lumen of the lateral sinus. In one of these patients the underlying disease was acute luekaemia (Wright, 1971).

Three patients aged 6 years were admitted with acute otitis media, headache, vomiting and early papilloedema. There was a raised pressure on lumbar puncture, and a slightly raised cerebrospinal fluid protein in two cases, but no cells were seen and cultures were sterile. Carotid angiography demonstrated occlusion of the ipsilateral lateral sinus. They were treated by craniotomy decompression and antibiotic therapy for the acute otitis media. Ear surgery was not undertaken in these cases and all made a good recovery. One patient showed an avascular area in the temporoparietal region on the angiogram and had a slight contralateral hemiplegia. A diagnosis of cortical thrombophlebitis was therefore made.

The patient, aged 47 years, with benign intracranial hypertension had a long history of tubotympanic chronic otitis media. She presented with headache and papilloedema. Obstruction of the ipsilateral sigmoid sinus was seen on the angiogram. Following craniotomy and decompression she made a good recovery and no ear surgery was performed. This condition rarely occurs later than the third decade and this patient is older than any of the cases reported by Foley (1955) or Newlands (1965*b*).

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TABLE V.

CLINICAL FINDINGS AND OPERATIVE MANAGEMENT IN 6 CASES OF OTOGENIC INTRACRANIAL VENOUS THROMBOSIS

Case	Age in in years	Ear disease	Clinical diagnosis	Angiography	Treatment
1	6	A.S.O.M.	Benign intracranial hypertension (otitic hydrocephalus)	Lateral sinus occlusion	Decompression
2	6	A.S.O.M.	Benign intracranial hypertension (otitic hydrocephalus)	Lateral sinus occlusion	Decompression
3	47	C.S.O.M.	Benign intracranial hypertension (otitic hydrocephalus)	Lateral sinus occlusion	Mastoidectomy
4	6	A.S.O.M.	Cerebral thrombo-phlebitis	Avascular parietal area. Lateral sinus occlusion.	Decompression
5	10	C.S.O.M.	Lateral sinus thrombosis	Lateral sinus occlusion	Mastoidectomy and drainage
6	7	A.S.O.M.	Lateral sinus thrombosis	Lateral sinus occlusion	Mastoidectomy and drainage

Cerebral hernia

Herniation of the temporal lobe into the mastoid cavity may follow mastoid surgery where there has been a wide exposure of the posterior or middle fossa dura. There may be a history of otogenic benign intracranial hypertension, when the raised intracranial pressure is thought to contribute to prolapse of the brain tissue.

The patient referred to the Radcliffe Infirmary with this complication was a boy, aged 15 years, who had had a modified radical mastoidectomy for extensive cholesteatoma and the tegmen plate had been eroded over a wide area. He presented with a soft pulsatile swelling in the mastoid bowl which enlarged rapidly until it filled the ear canal. The prolapsed brain via a right osteoplastic flap was excised and the defect in the tegmen plate repaired with temporalis fascia. Two days post-operatively he had a grand mal epileptic attack. He was treated with Epanutin and Phenobarbitone, but experienced intermittent similar episodes of diminishing severity during the following three years.

Summary

1. Intracranial complications of otogenic origin have decreased markedly during the last three decades due to antibiotic therapy and advances in otological surgical techniques.

2. Thirty-six patients with forty-one complications have been seen at the Radcliffe Infirmary, Oxford, during the past eleven years.

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3. Although the overall mortality is low, the morbidity is high, due to residual neurological deficits and epilepsy.

4. Conservative management of cholesteatoma with repeated suction clearance may not be sufficient to prevent bone erosion and exposure of the dura. Accordingly, such management can be dangerous.

5. Cerebral angiography via the internal carotid artery was the most useful radiological investigation.

6. Silent brain abscesses may be found during mastoid surgery in patients who have received repeated courses of systemic antibiotics for chronic ear discharge. Satisfactory shrinkage of the abscess cavity occurs following drainage via the tegmen defect and radical mastoidectomy. A formal dural repair is not necessary as fibrous obliteration of the tegmen defect takes place.

7. Those patients with clinical manifestations of intracranial venous thrombosis in which a clinical diagnosis of benign intracranial hypotension (otitic hydrocephalus) was made, all showed occlusion of the lateral sinus on cerebral angiography.

8. Headache was the earliest symptom of a developing complication, and a patient with this symptom and a discharging ear must be thoroughly investigated.

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