OTOGENIC MENINGITIS

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As a result of the successful use of the sulphonamides and antibiotics in the treatment of otitis media, otogenic intracranial infection is now not commonly encountered. Thus Jeanes (1962) reports a current incidence of intracranial infection of less than 0·15 per cent. among patients attending the Royal National Throat, Nose and Ear Hospitals with acute or chronic suppurative otitis media, compared with other reports from the pre-sulphonamide era of around 2 per cent.

The fact, however, that otogenic intracranial infection is not yet extinct and may still cause loss of life, warrants its periodic survey, and in this paper 41 patients with otogenic meningitis are reviewed and used as a basis for discussion of this particular condition. The patients were seen at the otology clinic associated with the West of Scotland Neurosurgery Unit at Killearn Hospital, near Glasgow, during the 10-year period 1953-62.

Incidence

Twenty-two patients were admitted during the first 5 years with five deaths, and 19 in the second 5 years, with five deaths.

Diagnosis

Headache, neck stiffness and persisting pyrexia are the pathognomonic triad of meningitis and diagnosis is confirmed by examination of the cerebrospinal fluid obtained at lumbar puncture. Frequently the patient displays restlessness and anxiety before the onset of headache, and early diagnosis will only be made by carefully testing such patients for neck stiffness and performing lumbar puncture where this is found. If a full bacterial diagnosis is to be made, lumbar puncture should be performed before antibacterial drugs are given as the cerebrospinal fluid may become sterile within 24 hours of commencing therapy.

The nomenclature proposed by Cawthorne (1939) has been adopted; thus in "serous meningitis" only the pressure is raised as is found in the earliest stage, in "cellular meningitis" there is also an increase, almost always of polymorphonuclears, in the cell content of the spinal fluid, and

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in "bacterial meningitis" organisms are demonstrated as well. Microscopic examination of a Gram stained film of the centrifuged deposit gives the quickest guide to the causative organisms and should always be carried out in addition to bacteriological culture. Additional findings in the examination of the spinal fluid are a raising of the protein level, while bacterial activity will lower the glucose level, and there is often a reduction in the chloride content.

It must be remembered that the presence together of an otitis media and meningitis does not necessarily incriminate the ear as the primary focus. When there is an associated upper respiratory tract infection, an acute sinusitis may be the cause, and in cases where otitis media and meningitis arise almost simultaneously, both may be the result of a bacteræmia.

In this series ventriculography was performed early in the illness in 37 patients in an endeavour to exclude associated brain abscess.

Bacteriology

Bacteriological examination was not proceeded with in two patients who died shortly after admission. Organisms were obtained in pure culture in nine and in mixed culture in three, and Gram-positive cocci were seen on stained films in four where no growth was obtained. There are no cases of serous meningitis in this series, 23 of cellular meningitis and 16 of bacterial meningitis.

Nature of the Otitis Media

A. *Acute otitis media*

None of the ten patients with acute otitis media had clinical evidence of associated mastoiditis. In two cases meningitis developed within 24 hours of the onset of otitis media, which was then likely to be in the pre-suppurative stage. The three cases of pneumococcal otitis media had acute tympanic changes, but pneumococcal otitis media can be notoriously lacking in prominent signs and symptoms. In six out of nine cases of pneumococcal meningitis with mastoiditis reported by Bastrup-Madsen and Nørby (1955) the otoscopic changes were minimal and none of the classical indications for cortical mastoidectomy were present.

In three patients in this series the otitis media proceeded to perforation of the drumhead.

B. *Chronic otitis media*

Earache due to an acute exacerbation was present in the majority. In four patients meningitis developed within the 24 hours following mastoidectomy (three radical and one cortical).

In only one chronic ear was the perforation central, the remainder being postero-superior or attic.

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Clinical Features

A. *Uncomplicated meningitis*

Twenty-three patients (14 C.S.O.M. and nine A.S.O.M.) survived after an uncomplicated course. This represents 45 per cent. of the C.S.O.M. and 90 per cent. of the A.S.O.M. cases. The average duration of fever was 5 days and in three it was 9 days before the temperature was normal. There were eight cases of bacterial meningitis and 15 of cellular meningitis. The cultures were Staph. pyogenes + *E. coli*; Staph. pyogenes + Strep. pyogenes; and in pure culture two with pneumococcus, three with Staph. pyogenes and one with *E. coli*. The cell counts ranged from 380 to 6,000 per c.mm., and polymorphonuclears predominated in all. In addition to the meningitis triad the main features were: depression of consciousness in 12; nystagmus in five; giddiness in four; papillœdema in three; focal signs in two; contralateral extensor plantar response in one.

The nystagmus consisted of first degree paralytic in three and first degree irritative in two. (The hearing in the affected ear was preserved in all five.)

The focal signs consisted in one patient with left A.S.O.M. of twitching and weakness of the right side of the body and aphasia. Speech and motor power were completely normal 4 weeks later. The other patient with right A.S.O.M. had similar motor signs and likewise recovered fully. Cerebral thrombophlebitis is the likeliest cause of such symptoms.

B. *Meningitis developing brain abscess*

Eight patients, all with underlying C.S.O.M., developed brain abscess (six temporal lobe and two cerebellar) and seven survived.

In all instances ventriculography was performed shortly after admission with normal findings.

Suspicion of the presence of further intracranial pathology was aroused either by a failure of the patient's general condition to improve, or by the appearance of additional clinical signs. The commonest features associated with abscess formation were persisting headache (seven cases) and persisting pyrexia (five cases). A lowering of the consciousness level occurred in four cases and papillœdema developed in four cases. Focal signs associated with temporal abscess formation appeared in three (contralateral facial weakness in two and contralateral hemiparesis, which was preceded by focal epileptic attacks, in one). Neither cerebellar case had apparent cerebellar signs prior to the detection of the abscess. All cases presented at least two of the above features, any one of which warrants exclusion of abscess formation by ventriculography or carotid angiography. The presence of an abscess must also be suspected if serial cell counts demonstrate an increasing percentage of lymphocytes in the spinal fluid. Alteration in the pulse rate, generally a slowing, may occur but this was not a feature in the above cases. It is noteworthy that in

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six patients, while abscess formation was occurring, the meningitis was responding to treatment as judged by a fall in the spinal fluid cell count, the disappearance of any organisms from the spinal fluid, and an increase in the glucose content.

In seven cases abscess formation was confirmed by repeated ventriculography. Case 8 developed signs of posterior fossa compression (unconsciousness and slow respiration) on the sixth day. Immediate decompression was performed and 5 c.c. of pus drained from a right cerebellar abscess. Case 7 was clinically normal by the end of the first week. Pyrexia and headache returned on the twelfth day; repeat ventriculography was normal. Drowsiness developed on the twenty-fifth day and ventriculography showed the development of a left cerebellar abscess. Case 5 died on the thirteenth day. An internal decompression by excision of the brain abscess was undertaken on the eleventh day because aspiration on the ninth and tenth days had produced no improvement, but the patient continued to deteriorate.

One must concede the possibility that in any of the above patients a small abscess, undetected by ventriculography, existed before the onset of meningitis. However, as is seen from the case summaries, in all except Case 7 abscess formation was detected about a week after the onset of meningitis and it seems likely that in those cases abscess formation was initiated about the time of onset of meningitis, and we are dealing from the start with the entity "Meningitis + Abscess Formation". Case 7 appears to be a true example of "Brain Abscess complicating Meningitis".

C. *Fatal meningitis*

Nine complicated chronic otitis and one acute otitis media.

Cases 9, 10 and 11 became comatose on the day of onset, were moribund at the time of transfer, and died without regaining consciousness. In Case 12 meningitis ensued within 24 hours of radical mastoidectomy. Case 13 became drowsy several hours after the onset of meningitis and developed papillœdema and a left hemiplegia on the second day. Case 14 apparently progressed well and was afebrile on the fourth day, but sudden coma and death ensued on the sixth day. Case 15 relapsed on the seventh day. Confused on admission, she died 2 days later. Case 16 became drowsy and developed a right upper motor neurone facial palsy and papillœdema on the fifth day. Ventriculography was then normal. A right hemiplegia developed on the twelfth day; repeat ventriculography was normal. Deep coma and death ensued on the sixteenth day. Case 17 died 17 days after the onset of pneumococcal meningitis complicating A.S.O.M. Death was due to a combination of broncho-pneumonia and heart failure. Post-mortem examination showed the meninges to be normal.

Summaries of the relevant features of the other post-mortem examinations are as follows:

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Case 9—pus in the right internal auditory meatus, leptomeningitis of the right half of the posterior fossa and right lateral sinus thrombosis with a small subdural abscess deep to the sinus. Case 11—leptomeningitis of the posterior fossa, left lateral sinus thrombosis. Case 12—erosion of the right tegmen, purulent leptomeningitis more on the vertex than the base, tentorial and cerebellar coning. Case 13—leptomeningitis mainly on the vertex, septic thrombosis of the superior longitudinal sinus and some associated right cerebral thrombophlebitis, tentorial and cerebellar coning. Case 14—basal leptomeningitis, right lateral sinus thrombosis with a subdural abscess on the deep aspect of the sinus, tentorial and cerebellar coning. Case 15—right tegmen erosion, basal leptomeningitis, cerebellar coning. Case 5—purulent leptomeningitis over the right vertex, much abscess formation in the remainder of the right temporal lobe, right lateral sinus thrombosis and associated cerebral thrombophlebitis.

Thus, of eight post-mortem examinations, death was due to intracranial sepsis in seven and in five of these the meningitis was associated with further intracranial pathology. It is noteworthy that lateral sinus thrombosis figures prominently in the associated pathology. In the two with tegmen erosion there was no further pathology present—in Case 12 the meningitis was fulminating, while in Case 15 there was overwhelming reinfection.

From a clinical aspect those who quickly become unconscious have a poor prognosis—the only three patients in this series who became unconscious the same day that the meningitis started all died. Considering the brevity of the history of Case 9, the degree of pathology present is surprising. This patient alone in the series was never pyrexial, presumably due to a very poor resistance. The presence of a bacterial meningitis with a low spinal fluid cell count as in Case 10 may also indicate a poor resistance, especially with an associated low spinal fluid glucose level of 8 mg. per cent.

Case 14 provides a demonstration of how antibacterial therapy may render the patient apyrexial despite the presence of frank intracranial suppuration.

Bronchopneumonia was present in Case 5, Case 15, and Case 17.

Treatment

In addition to the treatment of the meningitis and the primary focus of infection in the ear, any fluid and electrolyte disturbance must be corrected and repeated neurological examination and constant vigilance are required to detect any developing intracranial abscess.

Treatment of the meningitis

In this series sulphonamides and penicillin have been used to initiate treatment and are changed if the spinal fluid culture demonstrates

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Otogenic Meningitis

TABLE I.

BASIC STATISTICS OF PATIENTS UNDER REVIEW.

Underlying otitis media	No.	Sex		Side		Mortality	
		M.	F.	R.	L.	No.	Rate %
Acute (A.S.O.M.)	10	5	5	5	5	1	10
Chronic (C.S.O.M.)	31	18	13	16	15	9	29
Total	41	23	18	21	20	10	24·4

TABLE II.

INCIDENCE OF OTOGENIC MENINGITIS BY AGE GROUP.

Age group	0-10	11-20	21-30	31-40	41-50	51-60	61-70
A.S.O.M.	2	2	0	1	1	2	2
C.S.O.M.	0	9	6	5	5	6	0
Total	2	11	6	6	6	8	2

TABLE III.

SEASONAL INCIDENCE.

	Jan.	Feb.	Mar.	Apr.	May	June	July	Aug.	Sept.	Oct.	Nov.	Dec.
A.S.O.M. ..	—	1	2	4	1	1	—	—	—	—	—	1
C.S.O.M. ..	1	4	3	1	2	5	2	1	3	5	—	4
Total ..	1	5	5	5	3	6	2	1	3	5	—	5

TABLE IV.

BACTERIOLOGICAL FINDINGS IN CEREBROSPINAL FLUID.

Underlying otitis media	Streptococcus pyogenes	Staphylococcus pyogenes	Pneumococcus	*E. coli*	Sterile
A.S.O.M. ..	0	3	3	0	4
C.S.O.M. ..	2*	3*	2*	2*	23†
Total	2	6	5	2	27

* Includes mixed cultures. † Includes four where Gram + cocci seen.

Mixed cultures were obtained in three: Staph. pyogenes + Strep. pyogenes; Staph. pyogenes + Strep. pyogenes + Pneumococcus; Staph. pyogenes + *E. coli*.

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TABLE V.

NATURE AND INCIDENCE OF OTITIS MEDIA AT ONSET OF MENINGITIS.

Underlying ear condition	No.	% of total
A.S.O.M.	10*	24·4
C.S.O.M. with acute exacerbation	19	46·3
C.S.O.M. (no apparent change in behaviour)	7	17
C.S.O.M. (cessation of usual otorrhœa) ..	1	2·4
C.S.O.M. (post mastoidectomy)	4	9·8

* Two cases probably in pre-suppurative stage.

resistant organisms, or if there is no satisfactory response after 2 days' therapy. Sulphadiazine is the sulphonamide of choice and the sodium salt is given intravenously if vomiting is troublesome. Attention must be paid to the maintenance of an adequate urinary output and the urine should be rendered alkaline.

In addition to frequent intramuscular injections of large doses of benzylpenicillin, this drug is given intrathecally by lumbar puncture to ensure a rapid bactericidal level in the cerebrospinal fluid. The intrathecal dosage is 10,000 units daily, and in this series it has been given for 2 to 12 days. The complications of intrathecal penicillin therapy—irritation and sometimes permanent damage to the spinal cord and nerve roots, spinal block, cerebral damage, and sometimes death—have been reviewed by Wyss (1959) and the outstanding feature is the large dosage employed, up to 500,000 units having been given in one single injection. Untoward reactions are very rare if a dosage of 10,000 units per day is not exceeded.

With the diminution of penicillin-sensitive organisms and the relative increase of Gram-negative organisms, the use of other antibiotics may be indicated.

Chloramphenicol crosses the blood-brain barrier to attain a concentration of 30-50 per cent. that of the blood in the cerebrospinal fluid (Ross *et al.*, 1949) and was employed alone in three of the uncomplicated cases in this series (in two where *E. coli* was cultured from the spinal fluid and in one case where no organisms were demonstrated that was responding slowly to penicillin and sulphonamides), and also in several of the cases with associated abscess. Up to 4 g. a day may be given in adults with severe infection, but in view of its liability to cause blood dyscrasias, chloramphenicol should not be used if there are other suitable drugs, and the total adult dose should not exceed 25 g. unless its further use is considered life saving. Frequent blood counts may show when agranulocytosis or thrombocytopenia is imminent, but are of little value in anticipating aplastic anæmia which may present after treatment has been stopped.

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Of the tetracycline group, tetracycline itself was shown by Wood and Kipnis (1953) to diffuse the most readily over the blood-brain barrier and in the treatment of meningitis Dowling (1955) recommends tetracycline 500 mg. intravenously at 8-hourly intervals, and for children Koch and Hansen (1957) recommend a total daily dose of 75 mg./kg./day, given intravenously for 3 days and then orally. Intravenous tetracycline alone was employed in Case 8 who had transient aphagia after coning.

The broad spectrum of chloramphenicol or tetracycline usually covers organisms resistant to penicillin.

Either cloxacillin or methicillin is indicated where the meningitis is caused by penicillin-resistant staphylococci. Colomycin is active against Gram-negative organisms and streptomycin may be useful in mixed infections containing *E. coli*. When any of the latter three drugs are used they should be given intrathecally as well as by intramuscular injection and streptomycin therapy should not exceed 5 days because of the great rapidity with which resistant strains may develop.

The use of systemic and intrathecal ampicillin in a case of *E. coli* meningitis is reported by Spittle and Phillips (1962) and they observe that ampicillin with its apparent lack of toxicity and its prompt action merits further trial in the treatment of meningitis caused by Gram-negative organisms.

The principal limitation to the use of systemic polymyxin B is the narrow margin between therapeutic efficacy and toxic concentration. It is of interest, however, that polymyxin B can be given intrathecally, and may be thus employed in combination with the systemic administration of any other appropriate drug when sensitive Gram-negative organisms are present. No serious side effects have been observed when the intrathecal dosage is limited to 10 mg. on alternate days in adults (and half this dose in children), and antagonism to polymyxin B has not been demonstrated (Jawetz, 1956), while in *E. coli* infections it occasionally displays a synergistic effect with streptomycin, the tetracyclines or chloramphenicol (Jawetz *et al.*, 1954).

Generally speaking, the simultaneous administration of penicillin with either the tetracyclines or chloramphenicol should be avoided (Jawetz and Gunnison, 1952).

Antibacterial therapy should always be continued until the temperature and cerebrospinal fluid have been normal for at least 5 days, but intrathecal administration should be stopped when organisms have not been demonstrated in the spinal fluid on three successive examinations and the cell count drops below 1,000/mm.^{3} (Teng, 1953).

In cases of meningitis complicated by brain abscess, it is inadvisable to continue treatment by the lumbar route as the pressure of the abscess distorts the subarachnoid channels at the tentorial opening and elsewhere, and this may prevent the penicillin from reaching the ventricles or

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spreading upwards over the hemispheres. There is also always the risk of lumbar puncture inducing a fatal pressure cone if intracranial pressure is markedly raised. Consequently the intraventricular administration of antibiotics should be substituted, employing the same dosage that was given intrathecally.

Treatment of the primary focus

(a) Acute otitis media

The antibacterial therapy directed at the meningitis usually cures the otitis media without recourse to surgery, and none of the cases in this series required operation.

It is possible, especially if the patient received inadequate treatment for the acute otitis media before the meningitis developed, that a subacute mastoiditis may exist with few symptoms. Thus, if the meningitis does not respond satisfactorily to treatment and the ear not return to normal as rapidly as one might expect, exploratory cortical mastoidectomy should be carried out about a week after the onset of aural symptoms. Persisting deafness despite an improved otoscopic appearance should also lead one to suspect the presence of a subacute mastoiditis.

(b) Chronic otitis media

An underlying chronic otitis media is generally of the unsafe type with a superior marginal perforation of the drumhead, and associated bone disease and cholesteatoma which form a persistent nidus for bacteria untouched by antibacterial therapy. Surgical removal of the diseased tissue is then indicated, but operation on the ear can usually be safely delayed until the meningitis is under control and the patient more fit for operation.

Of the 31 cases in the series, eight died without operation, and one patient with a safe perforation was not operated upon. The findings at operation in the remaining 22 are as follows: sclerotic mastoid in eight, diploic with varying cell development in 14; cholesteatoma in 17, granulations in the remaining five, and also in seven of those with cholesteatoma; polypi in two; pathological exposure in the middle fossa dura in seven and the sigmoid sinus in four others; erosion of the lateral semi-circular canal in five (hearing preserved in all). Radical mastoidectomy was performed in 18, and modified radical mastoidectomy in four.

As regards the timing of operation, which varied in this series from the second day of meningitis until 2 months later, medical treatment for the meningitis should always be initiated before mastoid surgery is undertaken. If organisms resistant to all drugs are cultured from the spinal fluid or if there is no response after 2 days' therapy with drugs to which the organisms are reported sensitive, mastoidectomy should be seriously considered before the patient's condition deteriorates further. Operation

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and post-mortem findings in this series show that pathological exposure of the dura had occurred in 12 cases (39 per cent.), and in view of the possible presence of such an inviting avenue of reinfection, operation must not be delayed too long. Another reason for avoiding undue delay in operating is the possible presence of an unsuspected lateral sinus thrombosis requiring treatment. As emphasized by Smith (1950), the diagnosis of lateral sinus thrombosis is generally more difficult when the patient is receiving antibacterial therapy, and rigors were not a feature of any such cases in this series. Each case must thus be treated on its merits and the balance struck between operating too soon on a patient whose general condition could have been improved further, and delaying operation too long with the risk of reinfection, or of further intracranial pathology existing undetected. Certainly no patient should ever be discharged from hospital without the necessary operation having been performed.

The deliberate exposure of the middle and posterior fossa dura should always be undertaken in order that any unsuspected extradural abscess may be dealt with and also the state of the lateral sinus can be assessed. A healthy sinus containing a firm clot should probably be left alone, but the presence of soft infected clot or a frank sinus abscess demands opening of the sinus and removal of all infected material until there is either healthy clot or free bleeding at each end. Bleeding is controlled by packing inserted between the bone and the sinus wall, although in some instances additional packing into the sinus lumen is required. The lateral sinus was not opened in any of the patients in this series.

Pathology

Operation and post-mortem findings showed erosion of the tegmen in eight and exposure of the sigmoid sinus in four of the C.S.O.M. cases, and those preformed spaces were the likely avenues of infection, while in another there was purulent labyrinthitis with pus in the internal auditory meatus. In this and three other cases, lateral sinus thrombosis was also demonstrated at post mortem. In the remaining C.S.O.M. cases and the A.S.O.M. cases, infection of the meninges probably occurred by a spreading thrombophlebitis from the small veins in the middle-ear mucosa.

Of the C.S.O.M. cases, eight developed brain abscess, four others at post-mortem examination had multiple intracranial pathology, and Case 16 had the features of cerebral thrombophlebitis. Thus 13 (42 per cent.) of the C.S.O.M. cases had multiple intracranial pathology and six of those 13 (46 per cent.) were fatal. Of the remaining 18 C.S.O.M. cases, three (17 per cent.) were fatal, illustrating the prognostic importance of multiple intracranial pathology.

The two (20 per cent.) A.S.O.M. cases with features of cerebral

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thrombophlebitis both survived. The fatal A.S.O.M. case died of intercurrent disease.

The histopathological changes in the ear in cases of otogenic intracranial infection have been described in detail by Jeanes (1962). He found the features of chronic inflammation in the mucous membrane and bone of ten specimens obtained at operation, and five of these had superadded features of acute inflammation.

Discussion

The antibacterial drugs have in particular reduced the incidence and improved the prognosis of meningitis complicating acute otitis media. Thus 54 of 119 cases (45 per cent.) reported by Neumann (1925) complicated acute otitis media, compared with 24 per cent. of this series. Only one of 35 cases (2·9 per cent.) complicating acute otitis media in Stewart's (1929) series survived, as opposed to seven of 79 (8·9 per cent.) of his cases with chronic otitis media. The reduction in the incidence of associated intracranial pathology is less striking. In Stewart's series there was associated intracranial pathology in 23 per cent. of cases complicating acute otitis media (20 per cent. in this series) and in 68 per cent. of the chronic otitis media cases (42 per cent. of this series).

The preponderance of males is usually attributed to the greater susceptibility of the male to infection. There is not believed to be any significant preponderance of side of affected ear. The peak of incidence in the second decade and the tendency to increased mortality rate with advancing years have also been recorded by Dawes (1961) in otogenic intracranial infection generally. Meningitis complicating acute otitis media appears to be less common in summer and autumn, but this may be a chance finding due to the small number of such cases in this review.

Outstanding feature of the bacteriological examination is the large number in which no organisms were demonstrated in the spinal fluid and the prior administration of antibacterial drugs for the otitis media no doubt contributes to this. Five of 16 (31 per cent.) of cases of bacterial meningitis were fatal as opposed to three of 23 (13 per cent.) of cases of cellular meningitis. A less striking difference in mortality was recorded by McLay (1954), and Dawes (1961) does not regard the finding of bacteria in the spinal fluid as appearing to influence the mortality rate. The demise of the streptococcus as a causative organism—cultured in 5 per cent. of this series and 59 per cent. of Stewart's series is likely to be another reflection of the successful use of antibacterial drugs in the treatment of acute otitis media. This organism has the reputation of causing brain abscess in association with meningitis (Smith, Schiller and Cairns, 1946), and although the streptococcus was isolated from the abscess pus in only one of the eight with associated brain abscess, it is interesting to note that Gram-positive cocci were demonstrated in the

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abscess pus of three others (Table VI). Pneumococcus Type III has the particular reputation of pursuing an insidious course, and if there is no evident primary focus outside the ear pneumococcal meningitis should be regarded as being secondary to otitis media even although otological examination discloses little abnormality.

This series illustrates in particular the potential danger of an acute exacerbation of chronic suppurative otitis media of the unsafe type.

TABLE VI.

CEREBROSPINAL FLUID AND ABSCESS FINDINGS IN CASES OF MENINGITIS DEVELOPING BRAIN ABSCESS

Case	Sex	Age	Side	Initial C.S.F. examination		Abscess bacteriology	Day
				cells/mm.^{3}	bacteriology		
1	M	33	R.	212*	Sterile	*E. coli* P. pyocyaneus	7th
2	F	29	L.	860	Sterile	Gram + cocci	7th
3	M	49	R.	1,136	Sterile	B. proteus	8th
4	M	37	R.	1,088	Sterile	*E. coli*	6th
5	M	43	R.	2,000	Gram + cocci	Gram + cocci	9th
6	F	18	R.	700	Gram + cocci	Strep. pyogenes	10th
7	M	55	L.	1,020	Sterile	Gram + cocci	25th
8	M	18	R.	2,200	Staph. pyogenes Strep. pyogenes Pneumococcus	Pneumococcus	6th

* Lumbar puncture on the third day of antibacterial therapy.

The right-hand column (Day) refers to the time of the initial abscess aspiration with reference to the onset of meningitis.
Polymorphonuclears predominated in all patients' cell counts.

TABLE VII.

BASIC DETAILS OF FATAL CASES OF MENINGITIS.

Case	Sex	Age	Side	Initial C.S.F. Examination		Time of death*
				cells/mm.^{3}	bacteriology	
5	M	43	R.	2,000	Gram + cocci	13th day
9	M	59	R.	—		Within 24 hours
10	F	20	R.	420	Gram + cocci	4th day
11	F	48	L.	2,800	Pneumococcus	5th day
12	M	13	R.	—		Within 24 hours
13	M	56	R.	1,300	Sterile	3rd day
14	M	18	R.	1,900	Sterile	6th day
15	F	45	L.	5,540	Gram − cocci	9th day
16	F	29	L.	493	Sterile	16th day
17	M	55	R.	4,280	Pneumococcus	17th day

* With reference to onset of meningitis.

Polymorphonuclears predominated in all counts.

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Such patients should not be managed at home, but referred without delay to an otologist, and treatment will almost certainly be surgical.

The need is stressed for repeated neurological examination in every case of otogenic meningitis in order that the early recognition of additional intracranial pathology may be achieved.

The sudden appearance of paralyses, especially when preceded by focal epileptic attacks, is generally due to cerebral thrombophlebitis. A cerebral abscess *per se* will not usually produce such gross neurological changes with such rapidity. However, a cerebral abscess may co-exist with cerebral thrombophlebitis, and always requires exclusion. Cerebral thrombophlebitis sometimes occurs on the contralateral hemisphere as in the case report by Benciolini (1962) in which aphasia and a right hemiplegia were associated with a right acute otitis media.

The abrupt appearance of focal signs may also be due to the development of a subdural abscess, as in the case report by Jackson (1962). In this condition there is usually an associated diminution of consciousness, and in one of the case reports by Wood (1952b), ipsilateral hemiplegia occurred due to the brain stem being forced against the sharp edge of the opposite side of the tentorium with interruption of the contralateral pyramidal impulses at midbrain level. The presence of focal signs thus warrants cerebral angiography or ventriculography, and if the possibility of a subdural effusion is still entertained despite negative radiological findings, burr-hole inspection of the subdural space is indicated.

We must rely on antibacterial therapy to control cerebral thrombophlebitis. A subdural abscess requires drainage through suitably placed burr-holes via indwelling tubes, through which penicillin is injected daily, while a cerebellar or temporal lobe abscess is treated by aspiration and the instillation of antibiotics, repeated if necessary.

In view of the possibility of concomitant or impending abscess formation, the co-operation of the neurosurgeon is essential, and although the treatment of brain abscess is now generally in the hands of the neurosurgeon, the otologist should be acquainted with the procedures involved, such as those described by Wood (1952a), Pennybacker (1961), Dawes (1961). Finally, it should always be remembered that the transfer of a patient to a neurosurgical unit for further investigation or treatment does not lessen the responsibility of the otologist to the patient, as the otologist is in the best position to decide if and when mastoid exploration is indicated.

Summary

Forty-one patients with otogenic meningitis provide a basis for the general discussion of this condition. Its management and also the features associated with further intracranial pathology receive emphasis. The

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co-operation of the neurosurgeon is regarded as being essential for successful treatment.

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REFERENCES

Bastrup-Madsen, P., and Nørby, G. (1955) *Acta med. scand.*, **151**, 135.
Benciolini, F. (1962) *Arch. Otolaryng.*, **76**, 398.
Cawthorne, T. (1939) *J. Laryng.*, **54**, 444.
Dawes, J. D. K. (1961) *Proc. roy. Soc. Med.*, **54**, 315.
Dowling, H. F. (1955) *Practitioner*, **174**, 611.
Jackson, J. McG. (1962) *J. Laryng.*, **76**, 641.
Jawetz, E. (1956) Polymixin, neomycin, bacitracin. New York, p. 18.
———, Coleman, V., and Gunnison, J. B. (1954) *Ann. intern. Med.*, **41**, 79.
———, and Gunnison, J. B. (1952) *Antibiot. and Chemother.*, **2**, 243.
Jeanes, A. (1962) *J. Laryng.*, **76**, 388.
Koch, R., and Hansen, H. (1957) *J. Pediat.*, **51**, 65.
McLay, K. (1954) *J. Laryng.*, **68**, 140.
Neumann, H. (1925) *Z. Hals-, Nas.-u. Ohrenheilk.*, **12**, 165.
Pennybacker, J. (1961) *Proc. roy. Soc. Med.*, **54**, 309.
Ross, S., Bischoff, H., Preisser, W., and Orr, W. (1949) *J. clin. Invest.*, **28**, 1050.
Smith, A. Brownlie (1950) *J. Laryng.*, **64**, 12.
Smith, H. V., Schiller, F., and Cairns, H. (1946) *Proc. roy. Soc. Med.*, **39**, 613.
Spittle, C. R., and Phillips, B. M. (1962) *Postgrad. med. J.*, **38**, 168.
Stewart, J. P. (1929) *J. Laryng.*, **44**, 225.
Teng, P. (1953) *Antibiot. Ann.*, p. 244.
Wood, P. H. (1952a) *J. Laryng.*, **66**, 71.
——— (1952b) *J. Laryng.*, **66**, 496.
Wood, W. S., and Kipnis, G. P. (1953) *Antibiot. Ann.*, p. 98.
Wyss, E. (1959) *Pract. oto-rhino-laryng. (Basel)*, **21**, 150.

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