The Journal of
Laryngology and Otology

(*Founded in* 1887 *by* MORELL MACKENZIE *and* NORRIS WOLFENDEN)

November 1964

MASTOIDITIS IN CHILDREN

By T. PALVA,* I. FRIEDMANN† and A. PALVA*
(Turku and London)

In an earlier study by T. Palva and Pulkkinen (1959) on the frequency of mastoid infection in acute otitis media during a 5-year period from 1956-9, there were, out of a total number of 12,781 patients, 365 patients with acute or subacute mastoiditis admitted to hospital for treatment. Cortical mastoidectomy was performed on 58 of these 365 patients, i.e. 15 per cent. of the admitted patients or 0·3 per cent. of the total number of patients with acute otitis media. It has been concluded that when acute otitis media did not subside within 3 weeks following myringotomy and antibiotic treatment, in some cases mastoidectomy had to be considered, as persistent mastoid infection might cause severe pathological changes in the middle ear associated with loss of hearing.

Among the principal changes noted at operation were the extensively infected mastoid air cells: the mucosa was thickened and there were granulations and thick mucopus or glue-like material in the cells. Only a small number of specimens, however, could be examined histologically showing microscopical evidence of various stages of acute or subacute mastoiditis.

The present clinico-pathological paper is based on the histopathological examination of a greater number of specimens of bone chips and granulation tissue removed at operation.

The diseased mastoid process removed at autopsy or bone chips

* Department of Otolaryngology, University of Turku.
† The Department of Pathology, The Institute of Laryngology and Otology, University of London.

https://doi.org/10.1017/S0022215100063088 Published online by Cambridge University Press

removed at operation on the mastoid process formed the basis of several fundamental histopathological studies of earlier authors, e.g. Krainz (1926), Mayer (1928), Stewart (1928), Ziegler (1936), Mündnich (1939) and others. It was soon noted that osteoclastic and osteoblastic processes proceeded together in the early stages of acute mastoiditis and that new bone formation started early in middle ear infection, occasionally as early as the 5th day of infection (Mayer, 1928 and Lange, 1928). Stewart and Ziegler observed it around the 7th or 8th day. As a rule both lamellar and spongy new bone formation were more common 2 or 3 weeks after the onset of infection and abundant after 4 weeks.

Friedmann (1956 and 1957) has re-examined the pathology of acute and chronic mastoiditis in order to ascertain whether or not, in the antibiotic era, the pathology of infections of the middle ear cleft had changed and concluded that there was no essential change in the pathology of mastoiditis in present-day material when compared with the findings of workers in the pre-sulphonamide period (Stewart, 1928). The investigations underlined the fact that bone was involved early in the course of infection of the middle ear cleft and that new bone formation began early and proceeded, often on a luxuriant scale, to the partial or total obliteration of the air cell system of the mastoid process. Morever, bone chips removed at operation in cases of chronic otitis media and mastoiditis have revealed definite microscopical evidence of repeated absorption and deposition of bone, leading to a reconstruction of the normal bone pattern. It was an obvious conclusion that the adult hypocellular mastoid was mainly a result of repeated attacks of otitis in infancy and not the cause of it (Ojala, 1950 and Tumarkin, 1957).

As regards epithelial changes, it may be pointed out also here that columnar and ciliated epithelium with numerous goblet cells and gland-like structures is frequently present in the infected middle ear cleft and in granulation tissue forming "aural polyps". Cholesterol granuloma and epidermoid cholesteatoma ("epidermoid" of the ear) complete the list of microscopical findings (Singer, 1932; Birrell, 1956; Friedmann, 1959; and 1963).

Own Material

All the patients were treated and operated upon at the Department of Otolaryngology, University of Turku, whereas the histological investigation of the specimens, following preliminary fixation in formalin and embedding in paraffin, was carried out at the Institute of Laryngology and Otology, University of London.

During a period of 5 years (January, 1959 to January, 1964), 191 patients were admitted with a diagnosis of acute mastoiditis, or prolonged acute otitis media, subsequently diagnosed as mastoiditis, which failed

https://doi.org/10.1017/S0022215100063088 Published online by Cambridge University Press

to respond to the usual methods of treatment. These cases included (Table VII) 8 patients with the following complications:

Purulent labyrinthitis	1
Purulent labyrinthitis and meningitis ..	1
Lateral sinus thrombosis	3
Facial palsy	3

The age and sex distribution of the patients is given in Table I: 71 per cent. were children under 5 years of age.

TABLE I

AGE AND SEX CLASSIFICATION.

Age	1	1-2	3-4	5-15	Total
Male ..	31	25	28	25	109
Female ..	24	13	14	31	82

Clinical Observations

Table II shows the number of operated cases annually as well as those who received conservative treatment during the period of observation. A total of 88 patients were operated upon, 23 on both ears. On admission the infection was defined as acute in 55 cases, since the duration of symptoms in a previously healthy ear did not exceed 2 weeks. In the

TABLE II

ANNUAL FREQUENCY OF ADMITTED CASES.

Year	Operative treatment	Conservative treatment
1959	23	29
1960	26	14
1961	14	26
1962	17	19
1963	8	15
Total ..	88	103

remaining 136 cases, the disease was defined as subacute in 61 cases, with symptoms lasting from 2 weeks to 2 months, and as "chronic" in 75 cases of longer duration. These facts are presented in somewhat greater detail in Table II. The table reveals that 60 patients (31 per cent.) were admitted during the first week of treatment (and disease). However, nearly an equal number of patients (58) had been treated for a period of 2 months or more before admission to hospital.

Radiography of the mastoid process of all cases admitted to the clinic

https://doi.org/10.1017/S0022215100063088 Published online by Cambridge University Press

showed arrested pneumatization in almost all patients of the younger age group. In older children, with no previous history of otitis media, there appeared to be definite signs of mastoid disease manifesting itself in cloudiness of the cells associated with necrosis of the bone.

TABLE III
DURATION OF SYMPTOMS, AND OF CONSERVATIVE TREATMENT BEFORE HOSPITALIZATION.

Duration days	No. of cases (symptoms)		No. of cases (conservative treatment)	
	Operated cases	Conservative treatment	Operated cases	Conservative treatment
0- 6	16	18	32	28
7-13	12	9	4	9
14-27	9	27	13	29
28-60	10	15	5	13
61>	41	34	34	24

TABLE IV
LEUCOCYTE COUNT AND SEDIMENTATION RATE.

Leucocyte count	Acute cases		Chronic cases	
	Operative treatment	Conservative treatment	Operative treatment	Conservative treatment
< 8,000	6	3	17	22
8,000-15,000	15	18	31	47
15,000-20,000	5	5	3	4
Not studied	2	1	8	4
Sedimentation rate:				
2- 5	2	1	8	12
6-10	—	5	14	18
11-20	1	5	16	13
21-30	3	2	4	14
31-60	12	9	11	11
61>	8	1	1	2
Not studied	2	4	5	7
Total ..	28	27	59	77

Laboratory Investigations

Hæmatology. Table IV shows the leucocyte counts and the sedimentation rates. The leucocyte counts are arranged according to the scale used by Koskinen (1938) and show that 143 patients (75 per cent.) had white cell counts exceeding 8,000 per c.mm. In view of the age distribution these figures are not unduly high. The erythrocyte sedimentation rate (Leffkowitz, 1937) rose to over 30 mm. per hour in 66 patients.

https://doi.org/10.1017/S0022215100063088 Published online by Cambridge University Press

The hæmoglobin values were determined in all cases so as to exclude iron deficiency, considered to be associated with an increased risk of infection. Only 16 patients (8 per cent.) had Hb values below 10 gm. per cent., the lowest figure being 7 gm. per cent. In 34 patients the eosinophil count exceeded 5 per cent of the total white cell count. The highest value was 33·5 per cent., but on the whole, the figures lay between 5 and 8 per cent. (21 cases).

TABLE V

Results of Electrophoretic Analyses.

Case No.	Total protein	Albumin %	α_1-glob. %	α_2-glob. %	β-glob. %	γ-glob. %
1	7·4	46	6·1	11	15	22
2	7·0	38	5·1	14	14	28
3	6·4	59	4·7	14	13	10
4	6·4	43	5·0	10	15	28
5	7·1	47	5·1	12	17	19
6	6·4	41	5·3	14	17	22
7	6·3	41	5·6	10	16	26
8	7·2	36	7·7	10	15	31
9	5·9	34	8·2	15	19	23
10	6·7	35	7·4	17	21	19
11	6·5	34	9·4	14	18	24
12	6·9	47	4·8	13	16	20
13	6·2	50	7·6	11	13	19
14	6·1	34	7·9	17	19	22
15	7·0	37	7·4	12	18	17
16	6·8	39	3·6	14	18	25
17	5·0	36	7·5	12	17	27
18	6·2	46	5·6	18	15	16
19	6·2	44	7·2	14	17	19
20	7·0	53	5·6	17	11	13
21	6·2	38	5·1	11	13	23
Mean	6·5	42·3	6·3	13.3	16·0	21·6
S.D.	0·5	2·2	1·5	2.4	2·4	5·0
Chronic maxillary sinusitis						
Mean	7·0	41·0	6·4	10·1	14·9	27·7
S.D.	1·0	5·5	1·7	2·3	2·0	4·9
Normal values						
Mean	7·0	71·7	3·7	4·3	7·3	13·1
S.D.	0·4	3·7	0·6	0·9	0·9	2·6

Biochemistry. Table V gives the results of paper electrophoresis in 21 patients using the Biuret reaction for total protein. The average normal values are given at the bottom of the table together with the average values in a series of chronic maxillary sinusitis (T. Palva *et al.*, 1962; see this paper for methods).

Bacteriology. Every effort was made to obtain material for bacteriological examination from all patients admitted. Moreover, specimens were obtained under sterile conditions through myringotomy and aspiration

https://doi.org/10.1017/S0022215100063088 Published online by Cambridge University Press

from all cases of acute otitis media seen as out-patients during a 3-month period at the beginning of 1960 and during another 2-month period at the end of 1963. A proportion of these specimens (141 patients) was obtained from untreated cases, the remainder, after about 1 week's treatment (181 patients). In this way a total of 322 specimens were examined from out-patients in addition to the specimens from 191 patients admitted. The results are given in Table VI.

TABLE VI

Results of Bacterial Analyses in Various Clinical Groups.

Bacterial species	Out-Patients		In-Patients			
	1960 3 mo.	1963 2 mo.	Operative treatment		Conservative treatment	
			acute	chronic	acute	chronic
Streptococcus pneumoniae	38	18	8	10	7	11
Hæmophilus influenzae ..	15	17	3	6	4	13
Streptococcus pyogenes ..	—	—	—	—	—	—
Streptococcus viridans ..	3	1	1	1	1	3
Streptococcus non-hæmolyticus ..	—	5	—	—	2	4
Staphylococcus pyogenes ..	15	30	11	21	4	21
Staphylococcus albus ..	11	21	1	4	2	9
Alkaligenes fæcalis	—	—	1	20	2	17
Pseudomonas pyocyanea ..	—	—	2	5	1	4
Klebsiella pneumoniae ..	—	—	—	4	1	1
Proteus vulgaris	—	—	—	3	—	—
Proteus mirabilis	—	—	—	1	—	1
Neisseria catarrhalis ..	—	—	—	1	—	—
Corynebacterium pseudodiphtheriae ..	—	—	—	1	—	—
Escherichia coli	—	—	—	2	—	3
Aerobacter	—	—	—	3	—	1
Candida albicans	—	—	—	12	—	7
Geotrichium	—	—	—	—	—	1
Sterile	79	72	3	7	4	5

About one half of *out-patients' specimens* proved to be sterile. When positive, the cultures yielded streptococcus pneumoniae (17·4 per cent.). Hæmophilus influenzae (10 per cent.) and streptococcus pyogenes (4·3 per cent.) considered to be primary ætiological agents. A considerable number of yellow and white staphylococci was also isolated mainly from specimens from discharging ears, or from ears with repeated myringotomies when contamination from the external ear canal could not be excluded. It is interesting to note the comparatively high rate of incidence of streptococcus pneumoniae.

The cultures from *in-patients* presented a bacterial flora in accordance with the duration of the disease. Bacteria generally associated with acute otitis media have been frequently isolated but also other bacteria commonly associated with chronic infection of the middle ear cleft.

https://doi.org/10.1017/S0022215100063088 Published online by Cambridge University Press

Pathology

Macroscopical observations of mastoid pathology on 111 ears during mastoidectomy may be divided into five groups.

(1) There was, in 20 ears, definite evidence of mastoiditis with frank pus present subperiosteally or in the air cells.

(2) In 21 ears, the air cells contained some thick glue-like material and there was evidence of osteoporosis.



FIG. 1.
Acute mastoiditis (H. influenzae). Trabecular new bone formation in mastoid cell. Hæmatoxylin and eosin. (×130.)

(3) There was widespread destruction of air cells in 9 ears and the resulting cavities were filled with soft granulation tissues which, in some cases, could be pulled off in large pieces measuring 1 cm. × 2 cm.

(4) In 34 ears there were varying amounts of dense granulation tissue or swollen mucosa present in the otherwise well preserved air cells and some of the peripheral cells contained a clear, straw-coloured fluid.

(5) In the last group comprising 27 ears, only the mucosa and septa appeared to be thickened.

Histopathology

The material submitted for histological study did not always contain bone. The number of satisfactory pathological specimens amounted to 75. The microscopical findings can be summarized as follows:

https://doi.org/10.1017/S0022215100063088 Published online by Cambridge University Press

(1) There was evidence of suppuration with bone absorption, as well as new bone formation or marked osteoblastic activity in 23 cases.

(2) There was purulent granulation tissue with small amounts of normal bone present in 14 cases.

(3) There were varying amounts of granulation tissue present but no bone in 18 cases.

(4) There were only strips of thickened mucosa often covered by fibrinous exudate tissue or bone present in 8 cases.

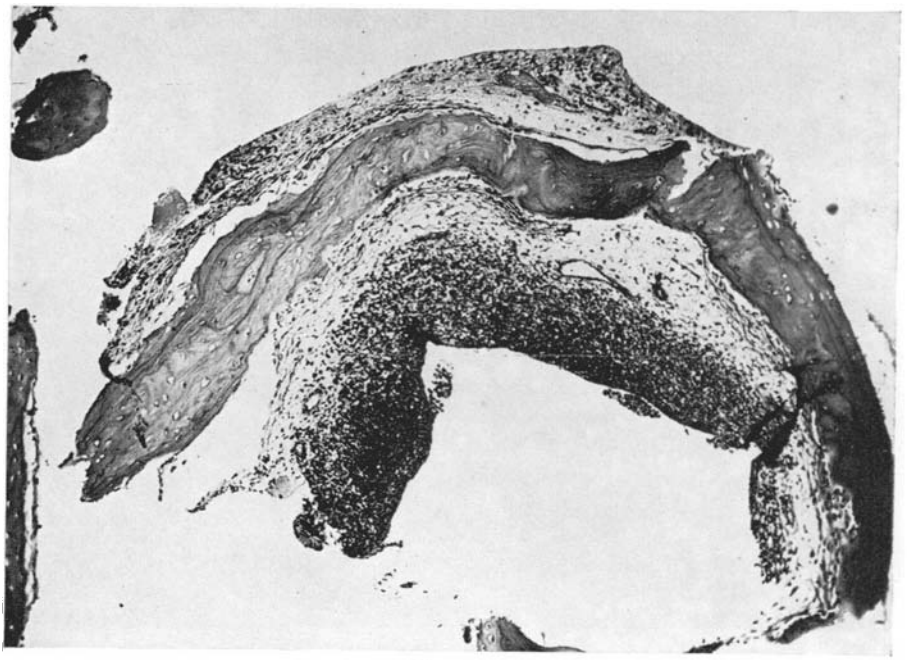


FIG. 2.
Acute mastoiditis (S. pneumoniae). Mastoid cell showing unusual lymphoid infiltration. Hæmatoxylin and eosin. (×75.)

(5) There were only fragments of bone present showing some irregularity of the cement lines in 12 cases.

New bone formation was observed in all cases lasting longer than 14 days (Fig. 1) and in 4 cases of 7 days' duration but not in cases lasting less than 4 days. An unusual feature was the presence of lymph-follicles in 2 cases of acute mastoiditis (Fig. 2).

The mastoid air cells of 32 cases were lined by stratified ciliated columnar epithelium (Fig. 3) and mucous glands were seen in most cases.

Cholesterol deposits surrounded by varying amounts of granulation tissue formed cholesterol granulomas in 8 cases (Fig. 4). There were large numbers of macrophages in several specimens from chronic cases, but there was no evidence of epidermoid "cholesteatoma".

https://doi.org/10.1017/S0022215100063088 Published online by Cambridge University Press

Table VII illustrates that all acute and complicated cases belonged to the first three histological groups, while subacute and chronic cases formed part of all histological groups and the pathological lesions depended upon the duration and/or the individual defence forces and treatment.

A comparison was also made with the bacterial species cultured in the various groups, but this revealed no essential difference of pattern.

TABLE VII

SYNOPSIS OF MASTOID PATHOLOGY

Histological groups	Clinical groups 1 2 3 4 5	Cholesterol granuloma	Pre-operative complications	Remarks
Group I	10 5 3 5 –	1	1 lateral sinus thrombosis 1 facial paralysis 1 purulent labyrinthitis and meningitis	14 acute 9 chronic
Group II	1 3 11 7 2		1 facial paralysis	4 acute 10 chronic
Group III	4 1 4 6 3	4	1 facial paralysis 1 lateral sinus thrombosis and meningitis	10 acute 8 chronic
Group IV	– 2 1 3 2		None	8 chronic
Group V	– 3 – 3 6	3	None	12 chronic

Discussion

The fundamental pathology of acute mastoiditis studied in recent years has not changed when compared with the findings of workers of the pre-sulphonamide and pre-antibiotic era. The present study confirms that there are in this respect no geographical or ethnological differences. It has been confirmed that reconstructive processes play an important role and progressive new bone formation in acute mastoiditis was a common finding in suitable specimens. In subacute mastoiditis, it was noted that the thickened mucosa was often covered by fibrinous exudate which may become organized and form permanent adhesions between the ossicles and the tympanic membrane and lead to tympanosclerosis (Ojala, 1953).

In chronic mastoiditis columnar metaplasia with gland-formation was a common finding and accounted for the mucoid discharge.

Cholesterol was deposited in 8 cases in the infected middle ear cleft leading to the formation of a foreign-body granuloma which has been

https://doi.org/10.1017/S0022215100063088 Published online by Cambridge University Press

called cholesterol granuloma. Cholesterol may be derived from hæmorrhagic or purulent exudate (Birrell, 1956). It may also be released from degenerating macrophages laden with lipid granules (Bryan, 1953; Ojala and T. Palva, 1955).

It cannot be emphasized too often that the so-called cholesterol granuloma is not a clinical entity despite some attempts to elevate this comparatively trivial though interesting microscopical feature to the status of a disease *per se* (Friedmann, 1956 and 1959).

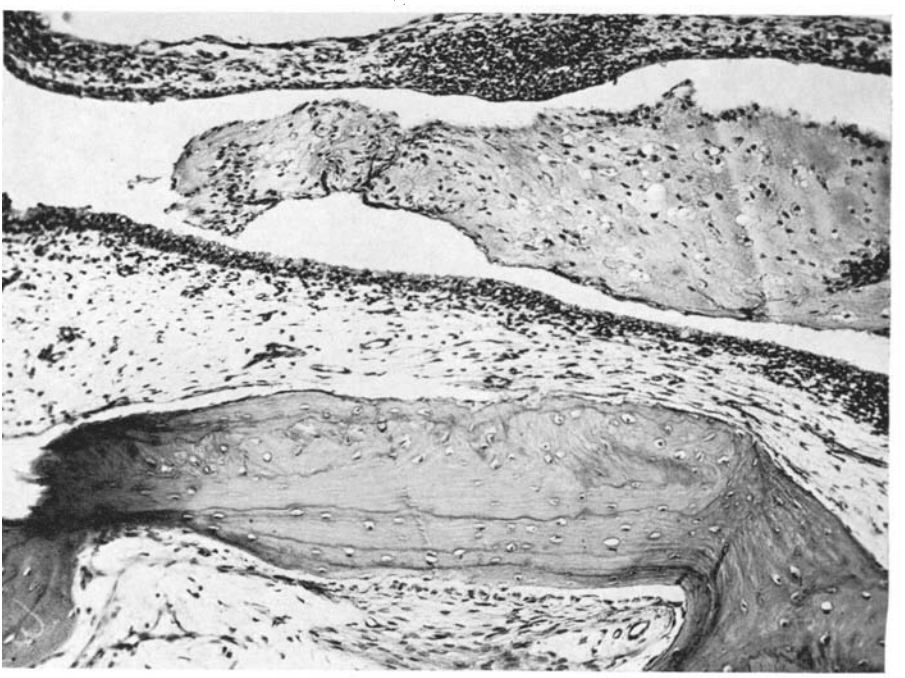


FIG. 3.

Chronic mastoiditis (Staphylococcus pyogenes and Ps. pyocyanea). The mastoid cells are lined by thickened fibrous mucosa covered by pseudo-stratified columnar epithelium and contain some muco-purulent exudate. Note lines of apposition in the bone. Hæmatoxylin and eosin. (×150.)

Routine bacteriological investigation of all patients with acute otitis media is of value but not always feasible owing to the large number of patients with otitis media seen in Finland as out-patients. Nor is it always essential since the common causative organisms, streptococcus pyogenes and streptococcus pneumoniae have remained sensitive to both parenteral penicillin G or oral penicillin V, and the majority of patients are cured with adequate penicillin treatment for one week. Furthermore, patients infected with hæmophilus influenzae considered to be resistant to penicillin V, often respond to parenteral penicillin treatment.

https://doi.org/10.1017/S0022215100063088 Published online by Cambridge University Press

Bacteriological investigations are invaluable in the treatment of patients where the condition does not resolve within a week's time. (All these observations have been borne out by, and are similar to, those experienced at the Royal National Throat, Nose and Ear Hospital in London during the last decade.)

Table VI suggests that the staphylococci do not play an appreciable role in the causation of acute otitis media, and that streptococcus pneumoniae, streptococcus pyogenes and hæmophilus influenzae are the

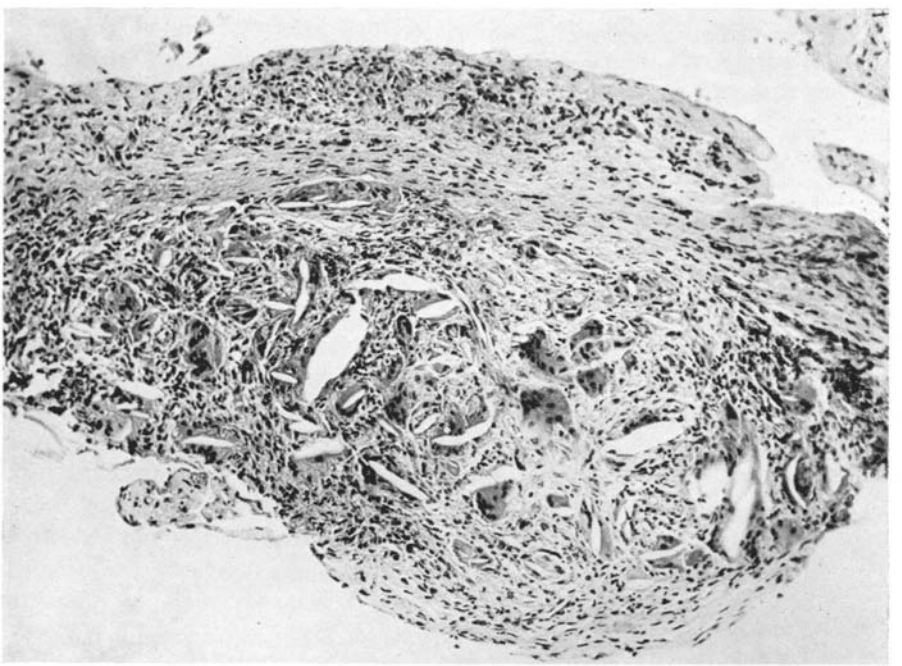


FIG. 4.

Recurrent otitis media (H. influenzae and C. albicans). Cholesterol-granuloma. Hæmatoxylin and eosin. (× 190.)

primary bacterial invaders. This is in line with the previous investigations of Friedmann (1952), Lahikainen (1953) and of Grönroos *et al.* (in the press) from this Clinic. Staphylococci, however, are present in a large number of patients suffering from subacute or chronic purulent otitis media and together with the gram negative flora may contribute to the prolongation of the disease.

It is an interesting, and so far, unexplained fact that some patients do not respond to the usual antibiotic treatment as quickly as others, but there is nothing in the bacteriology of these cases to suggest an explanation. In fact, children are admitted to the Hospital with a history of several weeks' discharge from the ear not responding to the usual treatment

https://doi.org/10.1017/S0022215100063088 Published online by Cambridge University Press

of the general practitioner. Cultures, perhaps surprisingly, yield a pure growth of streptococcus pneumoniae or pyogenes of normal antibiotic spectrum. Friedmann and Jeanes (1961) have drawn attention to the significance of such "failed antibiotic cases" of acute otitis media.

The large number (47 per cent.) of sterile specimens seen in the group of 322 out-patients (Table VI) was almost certainly due to the fact that the patients had received prior anti-microbial treatment. In 35 cases, however, there was no previous antibiotic treatment; these cases might be similar to those reported by Siirala and Lahikainen (1952) and by Siirala and Vuori (1954) under the heading "sterile otitis-media".

During the prolonged course of the disease the middle ear mucosa and the mucosa of the mastoid cells become less resistant to secondary invaders. Moreover, prolonged antibiotic therapy tends to encourage mycotic infection. In the present series both operated and unoperated cases were so affected and their treatment with various mycostatic agents has proved a long and tedious task.

As regards the surgical treatment, the pathological findings confirmed the diagnosis of mastoiditis in every operated case and supported the indication for surgical intervention.

There were 8 cases with serious complications (Table VII). There were no specimens available for histopathological investigations in 2 of these cases. The microscopical findings in the other 6 cases confirmed the underlying progressive inflammatory process. There were, on the other hand, signs of repair in most cases especially of longer duration, suggesting that there exist no exact limits beyond which clinical improvement and/or cure could only be affected by surgical methods.

Indications for Operation and Operative Technique

The indications for mastoidectomy have been outlined in a previous paper (T. Palva and Pulkkinen, 1959) and there has been no major change in our overall practice.

In acute mastoiditis with signs of ear protrusion, subperiosteal abscess or Bezold's abscess, cortical mastoidectomy has to be performed without delay.

In other patients the decision may be more difficult. Surgical treatment may have to be considered if the normal condition of the mucosa of the middle ear cleft could not be restored by conservative methods within 3 weeks. There are also instances when following antibiotic treatment the tympanic membrane, though having healed without perforation, remains thick, lustreless and sluggish: repeated myringotomies and aspirations yield thick muco-purulent exudate or glue-like secretion. During myringotomy it may be noted that the middle ear cavity is smaller or obliterated by the swollen mucosa encroaching upon the inner surface of

https://doi.org/10.1017/S0022215100063088 Published online by Cambridge University Press

the retracted tympanic membrane. Such cases may require surgical treatment so as to prevent the development of adhesions in the middle ear.

The operation is performed via a postauricular incision passing initially through the skin only. A musculoperiosteal flap with the pedicle at the posterior wall of the external auditory canal is formed as described by T. Palva (1962a, 1962b and 1963). The mastoid bone is drilled down to the dural tegmen and sigmoid lamina; in young children if the mastoid tip contains healthy red bone marrow, some of the diploic bone may be left behind.

Special attention has to be paid to Trautmann's triangle and the perilabyrinthine cells; the zygomatic cells are opened and the ossicular chain exposed. This makes it easier to remove all granulations over the facial nerve area and from the lateroposterior epitympanic space. The operation should be carried out in a thorough fashion as described. If there were adhesions around the ossicles and the tympanic mucosa swollen, a part of the posterior bony canal wall over the ossicles should be removed and Prussack's space freed from granulations. The musculoperiosteal flap is then placed into the antrum to occlude permanently the connection between the middle ear and the operative cavity, and the wound is closed with mattress and superficial sutures. If the musculoperiosteal flap did not fill the whole cavity, the remainder could be filled with Gelfoam soaked in neomycin. Nature very soon makes up the defect, and the mattress sutures prevent any post-operative recession of the postauricular skin.

An additional improvement in our experience is the insertion of a polyethylene tube through the drum into the middle ear. This admits air to the middle ear from the meatus and improves drainage; the mucosa shrinks, and the eustachian tube regains its function more easily. The tube may be left in place for several weeks, until the ear remains dry, the drum becomes thin and politzerization succeeds easily.

There were 103 patients in the present series who were not operated, although there were clinical signs of mastoid involvement of a subacute or chronic type. Most of them were small children, and the functional result will only be known and might be compared with the operated group in the future.

On the basis of the material examined in this study it is obvious that some cases of acute mastoiditis may require instant operation. To rely on antibiotics and drainage alone in such patients may lead to prolonged hospitalization and possibly to complications. Immediate operation in skilled hands is safe, and the patient's ear is generally well in a week and hearing restored to normal.

If left unoperated, many cases of subacute mastoiditis undoubtedly heal without damage to hearing but many patients develop adhesions in the middle ear causing some permanent impairment of hearing.

Other patients develop chronic otitis media, with perforation of the

https://doi.org/10.1017/S0022215100063088 Published online by Cambridge University Press

tympanic membrane, chronic osteitis and possibly cholesteatoma originating from Shrapnell's membrane. Therefore, we feel justified in emphasizing —where indicated—the necessity of early mastoid operation and occlusion of the cavity to prevent the risk of permanent hearing disability.

Summary

A series of 191 cases of mastoiditis complicating acute otitis media was studied during the 5-year period, 1959-63. Cortical mastoidectomy was performed in 88 cases (111 ears); the others were treated conservatively.

Histopathological examination of the bone chips and granulations revealed that the changes noted in the mastoid process were essentially similar to those described in the early part of this century with new bone formation and bone reconstruction and epithelial changes the predominant feature.

Similarly, streptococcus pneumoniae, streptococcus pyogenes and hæmophilus influenzae were found to be the most common causative micro-organisms of acute otitis media. Staphylococcus pyogenes probably plays no appreciable ætiological role, but may affect the healing process.

If left unoperated, the middle ear and mastoid process may heal with normal hearing. Frequently, however, adhesions are formed in the middle ear or chronic ear disease develops. Aeration of the tympanum is of utmost importance for the prevention of adhesions. Certain cases of acute purulent mastoiditis should be operated on without undue delay; in subacute and chronic otitis media in children, the otologist's full diagnostic acumen is required to recognize those cases which might suffer permanent loss of hearing where reliance is placed on conservative treatment.

REFERENCES

Birrell, J. F. (1956) *J. Laryng.*, **70**, 260.
Bryan, W. T. K. (1953) *Laryngoscope* (*St. Louis*), **63**, 559.
Friedmann, I. (1952) *J. Laryng.*, **66**, 175.
——— (1956) *J. clin. Path.*, **9**, 229.
——— (1957) *J. Laryng.*, **71**, 313.
——— (1959) *Ann. Otol.* (*St. Louis*), **68**, 57.
——— (1963) *Proc. roy. Soc. Med.*, **56**, 695.
——— and Jeanes, A. L. (1961) *Brit. med. J.*, **2**, 581.
Grönroos, J. A., Kortekangas, A. E., Ojala, L., and Vuori, M., *Acta oto-laryng.* (*Stockh.*) (in press).
Koskinen, P. (1938) *Acta oto-laryng.* (*Stockh.*), Suppl. 25.
Krainz, W. (1926) *Z. Hals-, Nas.- u. Ohrenheilk.*, **13,** 361.
Lahikainen, E. A. (1953) *Acta oto-laryng.* (*Stockh.*) Suppl. 107.
Lange, W. (1928) *Z. Hals-, Nas.- u. Ohrenheilk.*, **20**, 3.
Leffkowitz, M. (1937) Die Blutkörperchensenkung. 4th edition. Berlin.
Mayer, O. (1928) *Z. Hals-, Nas.- u. Ohrenheilk.*, **18**, 628.
Mündnich, K. (1939) *Mschr. Ohrenheilk.*, **73**, 513.

https://doi.org/10.1017/S0022215100063088 Published online by Cambridge University Press

OJALA, K. (1950) *Acta oto-laryng.* (*Stockh.*), Suppl. 86.
——— (1953) *Arch. Otolaryng.*, **57**, 378.
——— and PALVA, T. (1955) *Laryngoscope* (*St. Louis*), **65**, 670.
PALVA, T. (1962a) *Laryngoscope* (*St. Louis*), **72**, 353.
——— (1962b) *Arch. Otolaryng.*, **75**, 329.
——— (1963) *Arch. Otolaryng.*, **77**, 570.
———, GRÖNROOS, J. A., and PALVA, A. (1962) *Acta oto-laryng.* (*Stockh.*), **54**, 159.
——— and PULKKINEN, K. (1959) *J. Laryng.*, **73**, 573.
SIIRALA, U. and LAHIKAINEN, E. A. (1952) *Acta oto-laryng.* (*Stockh.*), Suppl. 100, p. 20.
——— and VUORI, M. (1954) *Acta oto-laryng.* (*Stockh.*), **44**, 197.
SINGER, L. (1932) *Z. Hals-, Nas.- u. Ohrenheilk.*, **32**, 130.
STEWART, J. P. (1928) *J. Laryng.*, **43**, 689.
TUMARKIN, A. (1957) *J. Laryng.*, **71**, 65.
ZIEGLER, E. (1936) *Z. Hals-, Nas.- u. Ohrenheilk.*, **39**, 180.

Institute of Laryngology and Otology,
330 Gray's Inn Road,
London, W.C.1.

https://doi.org/10.1017/S0022215100063088 Published online by Cambridge University Press