ACUTE AND ACUTE-ON-CHRONIC (CHOLESTEATOMA) MASTOIDITIS: COMPLICATIONS AND MANAGEMENT

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ABSTRACT

A review of all cases of acute and acute-on-chronic mastoiditis managed by the ENT Department at Groote Schuur Hospital between 1980 and 1984 inclusive is presented. Emphasis is placed on the high incidence of intracranial and especially intradural extension of infection when cholesteatomatous ears present in the acute phase. Aggressive medical and surgical management is described, often in conjunction with the neurosurgeons. Special attention is also directed at lateral sinus pathology and the management of internal jugular vein thrombophlebitis with aseptic embolization. The bacteriology of these patients is examined to determine a standard antibiotic regime before definitive bacteriological analysis is available.

The literature pertaining to this subject is reviewed but it is clear that this is the largest series analysed in recent times and the prognosis with the protocol developed and practised in our unit is better than previously accepted.

INTRODUCTION

Sir William Wilde's famous statement in 1853; "So long as otorrhoea is present, we never can tell how, when or where it may lead to", may have lost much of its impact in many parts of the world. It is true that there have been dramatic advances in the management and outcome of patients with acute and acute-on-chronic mastoiditis, but a review of the patients managed at Groote Schuur Hospital reveals there is no room for complacency and the dangers of this disease are as real today in Cape Town as they were in Sir William's time.

The potentially lethal complications of otitis media and acute mastoiditis have been recognised for many centuries. In about 460 BC, Hippocrates noted that "acute pain of the ear with continued high fever is to be dreaded for the patient may become delirious and die" (Cawthorne, 1953). In later years it was believed that purulent otorrhoea was caused by brain abscesses but Morgagni (1682-1771) documented that brain abscesses were often secondary to ear suppuration (Heine et al, 1927).

Before the introduction of Prontosil (Sulfanilamide) in 1935, figures taken from a number of studies (Sellars, 1974), reveal 566 deaths from otogenic intracranial sepsis in 101,897 postmortems - i.e. 0.55%.

Surgery of brain abscesses was undertaken by otologists in the 18th and 19th centuries and in 1893 MacEwen reported a remarkable survival of 18 and 25 cases of otogenic brain abscess (MacEwen, 1893),

an achievement not to be equalled until the advent of antibiotics. Before antibiotics, the outcome of purulent meningitis secondary to otitis media was invariably fatal. This led otologists to direct their therapy to surgery but early attempts at trephination of the mastoid in the mid-17th century were unsuccessful and the procedure fell into disrepute.

Sir William Wilde (1815-1876) later described a postaural incision, 2,5 cm long and 2 cm posterior and parallel to the outer ear in patients with mastoid sepsis. Although bringing symptomatic relief, the procedure was destined to be abandoned as it failed to cure the basic pathology. By twist of historical irony, Oscar Wilde, the celebrated poet and satirist and Sir William Wilde's son, died in November 1900 at the age of 46 from otogenic intracranial sepsis.

The first real major advance came in 1873 when Herman Schwartze published details of his work on the mastoidectomy operation (Schwartze, 1973). Using a hammer and chisel he performed a cortical mastoidectomy and cleared away diseased tissue. Although crude by today's standards, the procedure proved highly effective in patients with mastoiditis without cholesteatoma.

The presence of cholesteatoma evaded effective control until Zaufal in 1890 described in detail the radical mastoidectomy operation by removing the superior and posterior osseous meatal walls and all remnants of the malleus, incus and tympanic membrane. In 1891 Stacke described the addition of a meatoplasty to increase access

to and aeration of the mastoid cavity. In 1899 Körner suggested in some cases the tympanic membrane and ossicles could be left to preserve hearing, and in 1906 Heath described a modified radical mastoidectomy in which the osseus posterior meatal wall was partially removed, leaving a "bridge" at the level of the annulus. This operation proved unsuitable for cholesteatoma and failed to become universally accepted.

A more effective modification of the radical technique was described by Bondy in 1910 for attic cholesteatomas with a perforation confined to the pars flaccida and the presence of intact ossicles. After the bone- work of a radical mastoidectomy is completed, the cholesteatoma matrix is exteriorized but care is taken not to enter the tympanic cavity, and the ossicles and pars tensa are left intact. This procedure was regarded with suspicion and only widely accepted when the one-stage fenestration operation was introduced by Lempert in 1938.

In an attempt to avoid the radical cavity and improve postoperative hearing, the combined approach tympanoplasty operation with a posterior tympanotomy (CAT) was devised by Wullstein in 1952 and popularized by Smyth and by Jansen from 1972. With a documented recurrence rate of 40%, this procedure is now accepted as having limited application except in expert hands with excellent follow-up. In the context of the population served by Groote Schuur Hospital, the operation is generally contra-indicated, especially in the acute phase.

With advances being made in the control of the primary mastoid pathology, attempts to surgically manage the complications of brain abscesses, extradural abscess and lateral sinus pathology were becoming more successful. Antibiotic therapy has proved a major advance with improved prognosis in patients with complicated mastoiditis.

With improved socio-economic conditions, early radical mastoid surgery in cholesteatomatous ears, antibiotics and aggressive medical and surgical treatment of established acute and acute-on-chronic mastoiditis, extradural spread of the infection has dramatically declined in most privileged parts of the world. The potential for spread, however, remains ever present and the clinical index of suspicion should remain high, especially as previous antibiotic use may mask the classical symptoms and signs.

PATHOLOGY

A complete description of the pathogenesis of cholesteatomas is beyond this thesis. A concise summary was published by Beales (1978) in which he describes the basic pathology as being the presence of desquamating stratified squamous epithelium within the middle ear cleft, but the exact origin of the epithelium is the basis of controversy. Cawthorne (1963) and Derlacki and Clemis (1965) believed congenital epidermoid cysts in the temporal bone to be the basic pathology. These certainly occur as in the conditions of cholesteatoma vera tympanicum and cerebrospinal epidermoids, but are probably very rare. The migration or immigration theory of Habermann (1885) proposed an ingrowth of epidermis from the external meatus and this was modified by Bezold in 1908 who suggested the attic retraction pocket and subsequent accumulation of keratin was a result of negative middle ear pressure from eustachian tube dysfunction. This theory has been enlarged by Tumarkin (1961) who believes that hypocellularity of the mastoid, secretory otitis media, cholesterol granuloma and cholesteatoma all have their origins in childhood upper respiratory tract infections which may have caused Eustachian tube obstruction and subsequent middle ear pressure changes.

Sadé (1978) disputes this migration of epithelium from the external canal and gives his support to the metaplasia theory. He finds difficulty with the migration theory because many cholesteatomas are medial to the ossicular chain and also because of the phenomenon of contact inhibition of epithelium, and the natural outward migration of epithelium from the tympanic membrane.

There is probably some truth in all these theories, but the concept of Eustachian tube dysfunction and negative middle ear pressure is fundamental to understanding middle ear pathology and management. What is true, however, is that the incidence and severity of cholesteatomas is decreasing in affluent communities as the general health of the population improves.

The locally invasive and destructive potential of cholesteatomas remains another source of intrigue and controversy. Certainly pressure plays an important role but the fact that destruction often does not follow the plane of least resistence (as in acute mastoiditis) suggests that other factors are operational. The osteolytic properties of the cholesteatoma matrix have given rise to many explanatory theories. Iino et al (1983) feel anaerobic microorganisms and organic acids may be involved and this activity would be curtailed if the matrix was exteriorized and exposed to the atmosphere. This is supported by clinical experience.

Collagenase activity has been demonstrated by Aramson and Huang (1977) and then the collagen may be further broken down by Cathpepsin, a collagenolytic enzyme. Ganz (1984) suggested an immunologic factor when he demonstrated a high number of Langerhan's cells within the cholesteatoma matrix which probably exhibit an "immunological alerting function"

This destructive activity, however, is apparent only when the matrix is moist and infection stimulates the formation of invasive

granulation tissue. Once the matrix is exteriorized and exposed to oxygen and the infection controlled, the epithelium becomes quiescent and benign.

EXTENSION OF SEPSIS

The potential for spread of infection from the middle ear cleft is great and may proceed in all directions; via the mastoid cortex forming a sub-periosteal abscess, superiorly through the tegmen, posteriorly through the sinus plate to involve the lateral sinus, inferiorly through the mastoid tip to the sternomastoid muscle and medially to involve the facial nerve and inner ear.

The dura mater initially resists spread, localizing the disease to form an extradural abscess, but thrombophebitis of the dural veins provides the next route of spread. Another route is the perivascular Virchow-Robin space which is a cuff of arachnoid covering a cortical artery and in direct continuity with the subarachnoid space.

Suppuration of the inner ear may spread along various routes to involve the intracranial cavity. These include the perineural spaces of the internal auditory canal, the cochlear duct to the subarachnoid space and the endolymphatic duct to the extradural space of the posterior cranial fossa.

Surrounding an area of cerebritis and abscess formation, microglial and granulation tissue formation takes about two weeks to develop a capsule with varying degrees of surrounding cerebral oedema.

Progression of the abscess may result in rupture into the lateral ventricle from a temporal lobe abscess or into the fourth ventricle from a cerebellar abscess.

Intracranial pressure may arise from hydrocephalus and, if limited to the supratentorial compartment, an uncal herniation may result. This, in turn, may further restrict CSF flow with a vicious cycle being set up and inevitable death occurring if not treated. Below the tent, a cerebellar abscess may cause herniation of the flocculus into the foramen magnum with failure of the cardiovascular and respiratory centres.

With this description of the progress of pathology in mind, it is easy to understand how combinations result - e.g. perisinus abscess with lateral sinus thrombosis and cerebellar abscess, or middle fossa extradural abscess associated with subdural empyaema and temporal lobe abscess. These associations were clearly apparent on analyzing our own patients' records.

LATERAL SINUS PATHOLOGY

The spectrum of the pathology includes perisinus granulations and pus, mural thrombus formation, occlusive thrombophlebitis, intrasinus empyaema and, finally, organization of the thrombus with recanalization or obliteration. The thrombophlebitic process may extend to the superior sagittal sinus, via the superior petrosal sinus to the cavernous sinus and inferiorly to the internal jugular

vein. Showering of septic emboli may produce multiple pyaemic abscesses, peritonitis or suppurative arthritis. Extension of thrombophlebitis to the mastoid emissary vein may cause a localized painful mass, called Griesinger's sign.

Aseptic and septic forms of lateral sinus pathology occur with different clinical presentations and require different treatment. Aseptic thrombosis is often found incidentally or may present insidiously with signs and symptoms of raised intracranial pressure. Headaches become more severe and may be associated with visual disturbances, papilloedema and unilateral or bilateral sixth nerve palsies. The exact mechanism of raised intracranial pressure is still unclear (O'Connor and Moffat, 1978) and more sinister pathologies such as brain abscesses and intracranial tumours must be excluded. It is in this respect that computerized tomographic scanning is most helpful (Schonsted-Madsen et al 1984). Symonds, (1931) first used the term "otitic hydrocephalus" to describe the well-known clinical picture of raised intracranial pressure complicating otitis media in the presence of a normal cerebrospinal fluid. However, the ventricular system is normal and there is no evidence of hydrocephalus. This condition is a varient of benign intracranial hypertension or pseudotumour cerebri and led Horowitz in 1949 to introduce the more accurate term of "otogenic intracranial hypertension".

LABYRINTHINE FISTULAE AND LABYRINTHITIS

Labyrinthitis secondary to chronic suppurative otitis media with cholesteatoma is a well-recognized complication. Initially, a perilabyrinthitis (circumscribed labyrinthitis) occurs with erosion of the bony labyrinth, resulting in a fistula and exposure of the inner ear endosteum. This may be asymptomatic except on increasing and decreasing pressure gradients in the external ear (fistula test) or there may be varying degrees of vertigo, often aggravated by head movements.

With progression of the inflammation, a serous (irritative) labyrinthitis occurs with symptoms ranging from intermittent unsteadiness and stumbling to frank continuous incapacitating vertigo. There is usually associated nausea and vomiting with the patient falling towards the side of the affected ear. The unsteadiness is worse in the dark with loss of visual fixation and, on examination, the nystagmus is noted to be towards the diseased ear.

Suppurative labyrinthitis occurs with the presence of inflammatory cells within the labyrinth itself with rapid destruction of the inner ear. These patients are usually extremely ill with vertigo, vomiting and deafness. Spread of the infection is usually via a lateral semicircular canal fistula, but may also occur via the footplate of the stapes or by erosion of the promontory. With inadequate or delayed management, infection from suppurative labyrinthitis may spread along several routes - via perilymph in the cochlear aqueduct to the subarachnoid space, via perineural and peri-

vascular channels in the internal auditory meatus and via the endolymphatic sac in the vestibular aqueduct to the posterior fossa extradural space.

With complete destruction of the labyrinth by suppuration, the nystagmus changes direction with the fast component away from the diseased ear. Compensation usually occurs in 4 to 6 weeks, although this may be delayed in elderly patients.

Initially it is not possible to distinguish absolutely between diffuse serous labyrinthitis which resolves without loss of auditory or vestibular function and diffuse suppurative labyrinthitis which leaves a dead inner ear.

CLINICAL MATERIAL

The records of all patients with acute and acute-on-chronic mastoiditis managed by the Otolaryngology Department at Groote Schuur Hospital between 1980 and 1984 inclusive were analysed. Acute-on-chronic mastoiditis was diagnosed in patients with symptoms and signs of acute infection in cholesteatomatous ears. Patient details were obtained from the records in the ENT Department and from the Neurosurgical records of patients operated on by the ENT Department in the Neurosurgical theatre and subsequently managed in the Neurosurgical wards.

130 patients were identified in this 5-year period of whom 74 had cholesteatomas. There was an overall male predominance of 77 to 53 females and the age incidence shows an interesting pattern with the majority of acute mastoiditis patients being under 5 years of age. The majority of patients with cholesteatomas presenting in the acute phase, however, was between 5 and 20 years old (Figure 1).

There were 56 patients in the non-cholesteatoma group; 46 (82.1%) and 37 (66.1%) having subperiosteal pus and cortical dehiscence respectively. 26 (46.4%) patients were recorded as having intact tympanic membranes and mucopus was obtained in 25 patients on myringotomy. It is interesting to note that one patient had a thickened tympanic membrane but a clear middle ear cavity. 9 patients had spread of sepsis to the extradural space, 3 had sigmoid sinus pathology and 2 had temporal lobe abscesses. One patient had meningitis and there were no cases of cerebellar abscesses. In this group (Table 1), the patient with a large subdural empyaema died.

In contrast, there were 74 patients with cholesteatomas, of whom 39 (52.7%) and 22 (29.7%) had subperiosteal pus and cortical dehiscence respectively. The aggressive and dangerous nature of this disease is demonstrated by 58 patients (78.4%) having intracranial spread of infection and 33 patients (44.6%) having intradural extension. There were 19 patients with sigmoid sinus pathology, 6 with subdural empyaemas, 14 with temporal lobe abscesses and 13 with cerebellar abscesses. 11 patients had lateral semicircular canal fistulae (Table 1).

In total, 22 patients (16.9%) had pathology of the lateral sinus. 19 of these patients (86.4%) had cholesteatomas and Table 2 illustrates that 25,7% of patients with cholesteatomas operated on in the acute phase had lateral sinus pathology.

The sinus was thrombosed in 12 patients, contained frank pus in 9 patients and was fibrosed and obliterated in one patient. There was a male predominance of 15 to 7 females and the ages ranged from 2 to 40 years (Figure 2). In this series there was a peak incidence in the 11 to 15 year age group, all of whom had cholesteatomas. The 3 patients without cholesteatomas were all under 10 years old.

There was perisinus granulation tissue and perisinus pus in 4 and 14 patients respectively. Lateral sinus pathology was present in 4 patients without documented perisinus disease.

All patients presented with pyrexia and otalgia and 30 had neck stiffness. Of these latter, 9 had definite unilateral neck tenderness. The ages of patients with intradural sepsis is demonstrated in Figure 3 and shows the majority to lie within the 6 - 20 age group, shadowing the overall pattern of patients with acute-on-chronic mastoiditis. 31 patients had documented depressed level of consciousness with 19 presenting with vertigo, vomiting or nystagmus. 5 patients were grossly ataxic, 6 had seizures and 8 patients had hemipareses. 4 patients had seventh nerve palsies. 3 patients had sixth nerve palsies, probably due to apical petrositis (Gradenigo's Syndrome) or raised intracranial pressure.

Positive evidence of embolic phenomena was evident on Chest X-ray in 4 patients and definite hydrocephalus was demonstrated by computerized tomography in another 4 patients.

Lumbar punctures are not done as a routine but were performed in 6 patients, of whom 3 had active meningitis (2 of these had an associated cerebral abscess), 1 had a neighbourhood syndrome with 11 lymphocytes and no polymorphonucleorcytes and 2 patients had normal cerebrospinal fluid.

The high association of lateral sinus thrombosis with other intracranial sepsis is well documented and in this series, apart from those with perisinus disease, there were 3 patients with middle cranial fossa extradural pus, 1 with supratentorial subdural empyaema, 4 with temporal lobe abscesses and 4 with cerebellar abscesses - i.e. 9 patients (40,9%) had associated intradural sepsis.

The internal jugular vein was ligated in 3 patients and incised and drained in another 2 patients.

Overall, there were 5 deaths and a brief case report of each patient is presented.

CASE 1

A 26 year old male was seen in August 1980 with a 4 day history of severe headaches followed by a generalized seizure. On examination he had a depressed level of consciousness, neck stiffness, cyanosis and a left hemiparesis. He required urgent intubation. A lumbar puncture revealed purulent cerebrospinal fluid (CSF) and intravenous antibiotics were started. A computerized tomographic (CT) scan showed a right temporal abscess and urgent burrhole yielded 20cc of pus. Four days later a right radical mastoidectomy was performed and a large cholesteatoma in continuity with the temporal lobe abscess via an eroded tegmen was found. Postoperatively he deteriorated and died two days later.

CASE 2

In November 1982 a 3 year old female was treated at a peripheral hospital for meningitis but, because of gradual deterioration, an ENT opinion was sought and a radical mastoidectomy was performed. This revealed a large cholesteatoma with lateral sinus thrombosis and, after a stormy postoperative course, she was referred to Groote Schuur Hospital. A CT scan showed a right temporal lobe abscess which was drained. Postoperatively she developed multiple pulmonary abscesses and after one week the radical mastoidectomy

was revised and a lateral sinus empyaema was drained. The internal jugular vein was ligated and noted to be thrombosed to its junction with the subclavian vein. She died soon after surgery.

CASE 3

In 1981 a 15 year old male was referred to Groote Schuur Hospital from a peripheral hospital after 4 days of treatment for meningitis. He became progressively more toxic and drowsy. A CT scan revealed a posterior fossa abscess which was drained via a burrhole and this was followed by a radical mastoidectomy. There was a large cholesteatoma, a perisinus abscess and a thrombosed lateral sinus which was opened. Postoperatively the patient deteriorated and died the next day.

CASE 4

In 1981 a postauricular abscess was incised and drained in a 23 year old male who had a history of chronic suppurative otitis media. The following day he was noted to be drowsy, pyrexial and to have neck stiffness. On arrival at Groote Schuur Hospital a CT scan demonstrated a large temporal lobe abscess which was drained. A cortical mastoidectomy exposed infected granulation tissue and a posterior fossa extradural abscess. There was no cholesteatoma. He deteriorated postoperatively and died the following day.

CASE 5

A 13 year old female, seen in 1982 with progressive drowsiness, had a respiratory arrest in the Casualty Department. She was intubated and ventilated but both pupils were fixed and dilated. A CT scan showed a left temporal lobe abscess which yielded 60 ml of pus on drainage. A radical mastoidectomy was performed for a large cholesteatoma which had eroded the tegmen. She died the next day.

BACTERIOLOGY

Knowledge of the common pathogenic bacteria in the different forms of mastoiditis is essential in instituting the correct first line antibiotic therapy before patient response or bacterial analysis may indicate the need to change. The spectra of bacteria isolated may vary in different parts of the world and it is important to note that Groote Schhur Hospital serves a large population, mainly from the lower socio-economic group with limited exposure to previous antibiotics. The simple first line antibiotic policy evolved by analysing the cultures and sensitivities of bacteria isolated in our community has proved effective in the majority of patients.

Culture results were available on 112 of the 130 patients (86,2%). Of those with bacteriology available, 42 were diagnosed as acute mastoiditis and 70 as acute-on-chronic mastoiditis.

17 of the 112 specimens yielded no growth on bacteriological culture. 53 yielded pure cultures and 42 mixed organisms. There were proportionately more pure culture results in the acute mastoiditis group (57,2%) whereas, in the cholesteatoma group, mixed cultures made up 48,6% of the total (Table 3).

The predominant organism isolated in the acute group was the group A strepococcus (Strep pyogenes), followed by Streptococcus pneumoniae and Staphylococcus aureus (Table 4). Anaerobes did not play a major role in this group of patients. In the chronic mastoiditis with cholesteatoma group, however, anaerobes made up almost 40% of

the organisms isolated, with Bateroides fragilis being second only to Proteus Mirabilis in numbers (Table 5).

All Enterobacteriaceae isolated were sensitive to the aminoglycosides tested and most were sensitive to chloramphenicol. Streptococci, other than Enterococci, were sensitive to Penicillin. There were no cloxacillin resistant staphylococci isolated and all anaerobic organisms were Metronidazole sensitive. Both Haemophilus isolates were Ampicillin sensitive. All the Proteus Mirabilis strains and most of the Proteus sp strains were sensitive to Amoxycillin/ Ampicillin.

As expected, the high association of acute mastoiditis in cholesteatomatous ears and intradural sepsis yielded a close correlation between the bacteriological spectra isolated in these conditions. With otogenic intradural sepsis, however, chloramphenicol is added because of its wide bacteriostatic activity and its excellent cerebral penetration.

With the knowledge of probable pathogens, the current first line intravenous antibiotic policy at Groote Schuur Hospital is as follows :

(i) Simple acute mastoiditis - Penicillin

(ii) Acute-on-chronic (cholesteatoma) Mastoiditis - Ampicillin and Metronidazole

(iii) Otogenic intradural sepsis - Ampicillin, Chloramphenical and Metronidazole.

MANAGEMENT

In all patients presenting with acute and acute-on-chronic mastoiditis, management is directed at the primary pathology (i.e. mastoiditis) and any extension of the sepsis by both medical and surgical means. After a full clinical examination, intravenous fluids and antibiotics are started and the patient prepared for theatre. Haemoglobin estimation and white cell differential counts are done routinely and, although mastoid X-rays are useful in determining the presence and extent of a cholesteatoma and the size of the mastoid system, the decision to operate is made purely on clinical grounds. Chest X-ray and Mantoux tests (with Candida) should be done on debilitated patients and children under 5 years of age.

In this series, three patients aged 5 months, 1 and 4 years old had aural tuberculosis presenting with acute mastoiditis. The 5 month old baby was so malnourished and anergic that both the Mantoux and Candida tests were negative. All three had postauricular abscesses and "pale hypertrophic" mucosa in the antrum and, although a cortical mastoidectomy and medical treatment is usually adequate, a radical mastoidectomy had to be performed in the 4 year old due to the severity of local bone destruction and sequestrum formation.

Because of the high incidence of intracranial pathology found in patients with cholesteatoma presenting in the acute phase (44,6% in this series), computed tomography of the brain should be done routinely in these patients.

Although mastoidectomy is not done routinely in some units, (Hawkins et al, 1983 and Rubin and Wei, 1985), the nature of the pathology in the Western Cape renders early surgery mandatory in these patients.

Confusion in the diagnosis may arise with acute otitis externa, diffuse or local (furunculosis), and postauricular lymphadenitis secondary to scalp sepsis. Examination under anaesthetic with microtoilette of the ear should be performed if there is any doubt in the diagnosis, at which time a cholesteatoma should be obvious and a myringotomy should be done if the tympanic membrane is intact. Although rare, ENT surgeons should be aware that mastoiditis can exist without middle ear suppuration, especially in patients with mastoiditis masked by prior treatment with antibiotics.

MASTOIDECTOMY

In the absence of a cholesteatoma, a full cortical mastoidectomy with exposure of the middle and posterior fossa dura is performed. When a cholesteatoma is present, a radical mastoidectomy with a wide meatoplasty should be performed routinely. Canal up techniques are dangerous and require conscientious follow-up. In this series, two patients had a Combined Approach Tympanoplasty (CAT) performed in the acute stage, both of whom required conversion to radical mastoidectomies later, and 5 patients who had CAT performed previously for uncomplicated cholesteatoma presented later with an acute infection. The greater safety of the radical operation is an established fact. Lund (1978) quotes reported incidences of post-operative cholesteatomas with canal up techniques to be as high as 40%.

Although there is no place for canal up techniques in these patients, an initial drainage procedure in very ill patients may be followed by a definitive radical mastoidectomy once the patient is well enough to withstand a longer operation.

The middle and posterior fossa dura should be exposed and any extradural pus removed and the signoid sinus examined for patency.

LATERAL SINUS PATHOLOGY AND INTERNAL JUGULAR VEIN THROMBOPHLEBITIS

The management of aseptic lateral sinus thrombosis is controversial, with some authors (Pennybacker 1961, Goldenberg 1985) proposing a "wait and see" approach for those with minimal symptoms. If therapy is required, this is essentially medical with surgery reserved for rapidly failing vision, deterioration on medical treatment and the presence of mastoid pathology. The sinus is initially needled and, if a thrombus is demonstrated, the sinus is incised but obtaining free flow of blood is not necessary. Once otogenic intracranial hypertension is established and vision becomes jeopardized, various manoeuvres aimed at lowering the pressure can be utilized and include repeated lumbar puncture, steroids, acetazolamide, glycerol dehydration and, finally, subtemporal decompression or orbital optic nerve decompression (O'Connor and Moffat, 1978).

Septic lateral sinus thrombophlebitis is a more florid condition requiring urgent mastoid surgery and antibiotics. The symptoms and signs are related to the infection, the raised intracranial pressure and the possible effects of septic embolization. In these

cases, the sinus should be opened widely, as much of the contents evacuated as possible and a drain inserted. It may be possible to "milk" the pus out by massage over the internal jugular vein. An empyaema of the internal jugular vein should be treated by early incision and drainage, but further thrombophlebitic extension need only be treated by ligation with uncontrolled pathology. For example, the presence of septic emboli after definitive mastoid and lateral sinus surgery and high dose intravenous antibiotics warrants ligation of the internal jugular vein. This procedure was indicated in 3 of the patients with dramatic results (2 others required incision and drainage of the internal jugular vein because it contained frank pus).

The exact place of ligation of the internal jugular vein has been controversial. Some authors claimed worse results (Meltzer 1935), and others cited the danger of air embolism and damage to local structures (Jensen 1962). In the pre-antibiotic era, some surgeons ligated the vein routinely, above the level of the common facial vein, to prevent propagation along this vessel. An excellent description of this historical controversy is given by Teichgraeber et al (1982), but reasonable consensus has now been reached. There is no place for ligation as a routine but its value in some selected patients with uncontrolled internal jugular vein thrombophebitis should not be underestimated.

Anticoagulant therapy to prevent further propagation of the thrombus in patients with septic embolization is advocated by Shambaugh and Glasscock, but its use is highly controversial. Heparin was used in one patient with septic pulmonary embolization from lateral

sinus thrombosis reported by Hawkins (1985). He states that anticoagulants may be beneficial with extending thrombus, especially with impending cavernous sinus thrombosis. In the Groote Schuur Hospital series, anticoagulants were not used at all.

LABYRINTHINE FISTULAE AND LABYRINTHITIS

Fistulae may be suspected clinically before surgery but many are identified "incidentally" while lifting the cholesteatoma matrix. A safe approach is to suspect a fistula in all cholesteatomatous ears. Radiology of the mastoid is useful; an erosion of the lateral semicircular canal is often seen in the Stenver's view and this sign should be looked for routinely. Differential diagnosis from a cerebellar abscess may be very difficult because the conditions may co-exist and both present with vertigo, nystagmus and ataxia. Although surgery is indicated in both conditions, a preoperative CT Scan is recommended.

In the Groote Schuur Hospital series, there were 11 cases of lateral semicircular canal fistulae in 74 patients with cholesteatomas operated on in the acute phase, i e 14.9%. There were no fistulae in patients without cholesteatomas.

A radical mastoidectomy should be performed on all these patients, although some otologists advocate more limited surgery. Sheehy in 1983 reported on 1024 cases of cholesteatoma in whom 97 had labyrinthine fistulae. Only 25% of these patients had an open cavity, 16,5% had the cavity obliterated and 57,5% had a combined approach tympanoplasty.

Probably the safest approach is to leave matrix over the fistula undisturbed and perform a radical mastoidectomy. When matrix is lifted inadvertently, a fascial graft should be placed over the defect.

In the rare case of suppurative labyrinthitis and a dead ear, however, the full labyrinthine operation should be performed with removal of the bony promontory between the oval and round windows, allowing drainage of the basal turn of the cochlea as well as widely opening the semicircular canals with preservation of the facial nerve. The pars superior and pars inferior should be widely opened above, below and medial to the nerve. One of the patients in this series, a 7 year old male, actually had a sequestrum lying within the labyrinth which accounted for recurrent subacute mastoiditis. He had had an abscess of the cerebellopontine angle excised 3 years previously and subsequently had two cortical mastoidectomies before the sequestrum was discovered in November 1984 and the definitive labyrinthine operation performed (Mathews, 1986).

Palva outlined his technique for handling the fistula problem in 1983. Up to the late 1960s, he left the cholesteatoma matrix in place over the fistula. He stressed a high level of clinical suspicion for this condition and warned against sharp instrumental palpation to determine the condition of the underlying bone since this could result in infection or direct damage to the membranous labyrinth. After carefully isolating the fistula and using gelfoam soaked in ampicillin, he then removed the matrix and covered the fistula with fascia. In an attempt to stimulate new bone

he currently uses periosteum covered with bone patē and bone chips. Fibrin glue has been found to be a useful adjunct. Palva then proceeds to obliterate the mastoid cavity and perform any ossicular reconstruction and tympanic membrane repair he deems necessary.

Gormley (1986) reported on 684 patients having surgery for cholesteatomas by G Smyth over 19 years; 35 (5,1%) had labyrinthine fistulae. Initially a combined approach tympanoplasty (CAT) with a staged relook 6 to 9 months later was performed but this was later changed to an open cavity technique because of the high incidence of recurrences of cholesteatoma with the canal-up procedure (Smyth 1985). The current policy in Belfast is to leave the matrix over the fistula undisturbed and to date they have not recorded any progression of the disease process.

INTRADURAL SEPSIS

These patients are aggressively managed jointly by the Neurosurgical and Otolaryngology Departments from the beginning. CT scanning has proved the most effective diagnostic investigation for intradural complications and lumbar puncture is generally contra-indicated. The diagnosis of a cholesteatoma is made clinically and although radiology is useful, it may be misleading as a cholesteatoma is often not apparent on X-ray.

In general, the neurosurgical management takes precedence and is directed at:

(i) Lowering the raised intracranial pressure. This is directed at:

(a) The mass effect of the abscess. The pus is aspirated via a burrhole with a blunt brain cannula. The injection of antibiotics into the abscess cavity is controversial and its value has never been proven. Injection of radio-opaque material such as sterile barium or Thorotrast is no longer practised at Groote Schuur Hospital as serial CT scans have replaced serial pyograms. Repeated aspirations of temporal lobe abscesses are performed until resolution occurs. Formal excision is reserved for resistent cases. This policy is even more conservative when the dominant temporal lobe is involved. With cerebellar abscesses, however, routine formal excision is undertaken after an initial tap because of potential fatal consequences. Subdural empyaemas are drained via multiple burrholes or by craniotomy, depending on the extent of the pathology.

(b) Surrounding Oedema. Steroids (Dexamethasone) are used routinely with good effect, even with the theoretical disadvantages of decreasing the inflammatory response and limiting antibiotic penetration. This takes 6 to 12 hours to be effective so, in case of suspected cerebral herniation, osmotic diuretics (Mannitol) are used.

(c) Hydrocephalus. This is exceptionally rare with temporal lobe abscesses, but is a common complication of cerebellar abscesses. In these cases, a ventricular drain is inserted and is left in until normal cerebrospinal fluid circulation is re-established, usually in about one week. Rarely however there is irreversible CSF pathway obstruction and a ventriculo-peritoneal shunt needs to be inserted after all the infection is controlled.

(ii) Treating abnormal neurological function

Anticonvulsants (Phenytoin Sodium) are used routinely in supratentorial abscesses. If no seizures occur by the time the abscess is considered cured, medication is stopped. In the event of seizures occurring, anticonvulsants are retained until a two-year seizure-free period has elapsed. Neurological deficits must be managed by rehabilitation using physiotherapy, occupational therapy and speech therapy services.

(iii) Treating infection

Control is obtained from aggressive surgical and medical approaches. Intravenous Ampicillin, Chloramphenicol and Metronidazole are used initially until the bacterial culture and sensitivity result together with the patient's condition indicate a change.

POSTOPERATIVE MANAGEMENT

Essential in the management is the close monitoring of these patients following surgery, using clinical, haematological and radiological (CT Scan) parameters. Regular full neurological examinations are imperative as intracranial pathology may manifest up to three weeks after the mastoid infection has been controlled. Repeated CT scanning may be indicated and a persistently raised ESR or white cell count should alert the clincian to residual pathology. Antibiotics are maintained for a further two weeks after all signs of infection have been controlled, a period which is often 4 to 6 weeks long depending on the case.

DISCUSSION

It is essential that the dangers of mastoiditis are appreciated by clinicians in all fields so that early diagnosis can facilitate appropriate therapy. Patients with resistent meningitis in the Medical wards or with intracerebral abscesses in the Neurosurgical wards often have otogenic disease and therapy only becomes effective once definitive mastoid surgery has been performed.

A marked improvement in the prognosis in the post antibiotic era has been recorded and, in 1955, Courville and Nielsen reported 25 deaths from ear infections in 9,737 postmortems which was a 10-fold reduction from the figures published by the same authors in 1934. Computerized tomography (CT) and better surgical techniques have also contributed to improving the management and prognosis so that today, even with advanced disease, an excellent outcome can be expected.

Cholesteatomas complicated by infection are especially lethal even when frank otorrhoea is absent. It appears that with simple acute mastoiditis, the infection takes the path of least resistance and in this series 82,1% had subperiosteal pus and only 23,2% had intracranial sepsis. In those patients with cholesteatomas, however, the disease actively erodes compact osseous barriers in all directions and only 52,7% had any subperiosteal pus, but 78,4% had some intracranial evidence of infection.

The lethal potential of this condition needs to be re- emphasized as 44.6% (33/74) of patients with cholesteatomas who presented with local and constitutional effects of acute aural sepsis at Groote Schuur Hospital already had intradural extension of the infection. This contrasted with only 7,1% (4/56) of patients with acute non-cholesteatomatous mastoiditis who had concomitant intradural sepsis. It was interesting to note that there were equal numbers of temporal lobe and cerebellar abscesses in the cholesteatomatous group, whereas it was previously recorded that in the Oxford experience temporal lobe abscesses were five times more common than cerebellar abscesses (Lund, 1978).

Keet and de Villiers (1985) reviewed 557 patients seen with brain abscesses or subdural empyaemas in Groote Schuur Hospital between 1952 and 1983 and found that 195 brain abscesses and 21 subdural empyaemas were of otogenic origin, an incidence of 39%. Overall there was a mortality of 38% for otogenic intracerebral abscesses, but these figures include terminal patients who died before any mastoid surgery could be performed and patients who had had mastoidectomies at other hospitals and, therefore, are not included in this series. They also showed that the outcome of intracranial abscesses varied directly with the level of consciousness on admission. A good result could be expected in a good grade patient assessed on the Glasgow Coma Scale (Table 6) and treated promptly, while a high mortality was found in patients presenting with a de-

pressed level of consciousness and features of insipient or established transtentorial herniation.

In Pennybacker's (1961) series of over 200 patients with otogenic intracranial complications treated by the Department of Neurological Surgery at the Radcliffe Infirmary, Oxford, 55 patients had abscesses in the temporal lobe and 30 in the cerebellum. He showed a dramatic improvement in mortality from 35% between 1938 and 1950 to 5,7% between 1950 and 1960. Wright and Grimaldi (1973), in the same hospital, reported on 36 patients with otogenic intracranial complications seen between 1961 and 1971. Of the 18 patients with brain abscesses, 14 had temporal lobe and 4 had cerebellar abscesses, reflecting the same proportions as found during the previous two decades (Pennybacker, 1961). There were no deaths but they did report a considerable morbidity, consisting of hemiplegia, ataxia and epilepsy.

Wolfowitz (1972) reported on 28 cases of otogenic intracranial complications treated at Baragwanath Hospital, Johannesburg, between 1965 and 1971 with an overall mortality rate of 25%.

Dawes (1961) reported on 199 patients with otogenic intracranial complications treated at the Royal Victoria Infirmary, Newcastle-upon-Tyne between 1944 and 1960. 30 had temporal lobe abscesses and 10 cerebellar abscesses, with a mortality of 50% and 30% res-

pectively. Dividing the study into approximately 5-year periods, he demonstrated a progressive fall in the mortality from 37,3% to 23,2% to 6,5%, and emphasized the increased mortality associated with multiple intracranial lesions.

Proctor (1966) compared patients with otogenic intracranial sepsis in the pre-antibiotic and post-antibiotic eras treated at the University Hospital in Ann Arbor, Michigan. In the 10 years before antibiotics, 129 patients were treated and there were 39 deaths (mortality 30,2%). Of these, there were 9 patients with temporal lobe abscesses, 6 with cerebellar abscesses and 3 with subdural abscesses. In the 10 year antiobiotic period up to 1963, 27 patients with otogenic intracranial sepsis were treated, of whom 5 had temporal lobe abscess and 3 had cerebellar abscesses, and there was only one death.

This dramatic improvement in management is further illustrated by Gower and McGuirt (1983) who reported 100 patients with otogenic intracranial sepsis and only 6 had brain abscesses. The overall mortality was 10%.

Lateral sinus pathology is rare, and delayed recognition and treatment is common. A recent survey of North American Otolaryngology Clinics (Goldenburg 1985), of which 99 responded, showed only 34% as having encountered the condition in the preceding 5 years.

The widespread use of antibiotics has not only dramatically lowered the incidence of this disease but, in many cases, has changed the clinical presentation to a subacute form, often with vague symptoms and signs. The clinical picture of an ill patient with a high spiking temperature, anaemia, headache, otalgia, neck stiffness and tenderness under the sternocleidomastoid muscle is rarely seen and most cases present with an intermittent low grade temperature, vague headaches and possibly mild discomfort on moving the head.

If lumbar puncture is done, a Queckenstedt or Toby Ayer test is often helpful, but possible false results and the dangers of a sudden increase in the intracranial pressure must be appreciated.

The mortality of lateral sinus thrombosis is quoted as being between 18 and 36% (Jackson and Dickens, 1979; Teichgraeber et al, 1982). Meltzer (1935) reported 45 deaths in 161 patients (27,9%) and the statement is often made that, although antibiotics have decreased the incidence of this disease, the mortality remains similar to the pre-antiobiotic era. It is important to appreciate that lateral sinus pathology has a high association with intracranial sepsis and it is really in these patients that the high mortality applies. Jensen (1962) reported 3 deaths in 12 patients (25%), occurring in Denmark in 5 years; 2 had cerebellar abscesses and 1 had a temporal lobe abscess. In Seid and Sellars review of 13 cases in 1973, there were 3 deaths (23,97%); 1 had a temporal lobe abscess, another had cavernous sinus thrombosis and the third an overwhelming septicaemia.

There were 2 deaths in the current Groote Schuur Hospital series in patients with lateral sinus pathology - a mortality of 9.1%. A 3 year old girl and a 15 year old boy, both with cholesteatomas, had been treated at peripheral hospitals for meningitis before deterioration prompted referral to Groote Schuur Hospital. The girl had an associated temporal lobe abscess and an internal jugular vein empyaema, and the boy had a cerebellar abscess. Prompt surgery was performed on both patients but they succumbed to overwhelming septicaemia.

Both of these patients presented late and had intracerebral abscesses but with early diagnosis and prompt aggressive management the prognosis for patients with lateral sinus pathology, even with internal jugular vein thrombophlebitis and septic embolization, should be much better than occurred previously.

A study of the bacteria responsible for acute and acute-on-chronic mastoiditis has been useful in evolving a first line intravenous antibiotic policy. As previously reported (Mawson), there is a marked difference in the bacteriological spectra of these two conditions, with a predominance of Gram negative bacillae and anaerobic organisms in the cholesteatomatous ears.

In acute mastoiditis, there was a high proportion of Group A Streptococci, although the most common organisms in acute otitis media are Streptococcus pneumoniae and Haemophilis influenza (Klein, 1981). Hawkins et al (1983), also observed this variation and ascribed it to the population they serve.

Other organisms isolated in this group are as expected; however the presence of Staphylococcus aureus in 6 cases is important as anti-staphylococcal agents are not routinely used as first line therapy. Other authors have found even higher incidences of Staph. aureus (Pfaltz and Griesemer, 1984; Rubin, 1985), and the proper collection of material for bacteriological culture, therefore, becomes important for appropriate antibiotic therapy.

The correct choice of therapy in the rare cases of gram negative bacillary (other than Haemophilus) acute mastoiditis is also important as these have been shown to have a higher rate of complications (Ostfeld and Rubinstein, 1980). The incidence of anaerobic organisms in this group was low, as has been previously reported (Ginsberg et al, 1980; Hawkins et al, 1983). At Groote Schuur Hospital, patients presenting with uncomplicated acute mastoiditis are initially treated with intravenous penicillin.

In patients with acute-on-chronic mastoiditis (cholesteatoma), Proteus mirabilis and Bacteroides fragilis, often in mixed culture, were the most common bacteria isolated. The combination of intravenous ampicillin and metronidazole have proved the most effective first line agents. While ampicillin is adequate for most Proteus mirabilis strains and also covers Enterococci and Streptococcus milleri, Enterobacteria other than Proteus mirabilis are rarely sensitive and Pseudomonas species are resistant. The importance of isolating Pseudomonas is highlighted by Fairbanks (1981) and, in these patients, the inclusion of an aminoglycoside has proved the most effective.

The virulence of Pseudomonas should be emphasised as it was isolated in 7 patients with cholesteatomas, but never in acute simple mastoiditis. 4 of these patients had temporal lobe abscesses, 2 had cerebella abscesses, and 1 had a posterior fossa intradural abscess together with an empyaema of the lateral sinus. In this latter patient, ligation of the internal jugular vein was also performed.

This study highlights the importance of mastoiditis, especially in those patients with cholesteatoma. General Practitioners, Paediatricians, Physicians and Neurosurgeons should have a special knowledge of this disease in order to avoid delay of appropriate surgery and development of potentially fatal complications.

By following the guidelines as laid down by the Department of Otolaryngology at Groote Schuur Hospital, effective management can be readily instituted and the mortality and morbidity kept lower than previously reported.

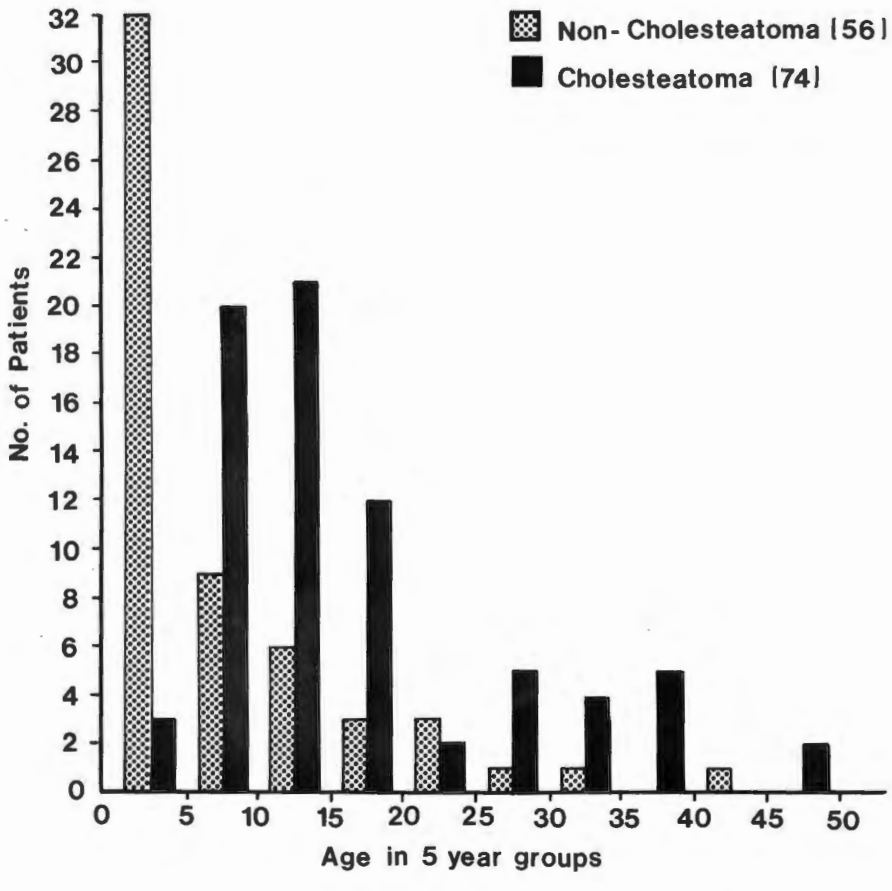
Non- Cholesteatoma [56]
Cholesteatoma [74]
No. of Patients
0
2
4
6
8
10
12
14
16
18
20
22
24
26
28
30
32
0
5
10
15
20
25
30
35
40
45
50
Age in 5 year groups

FIGURE I

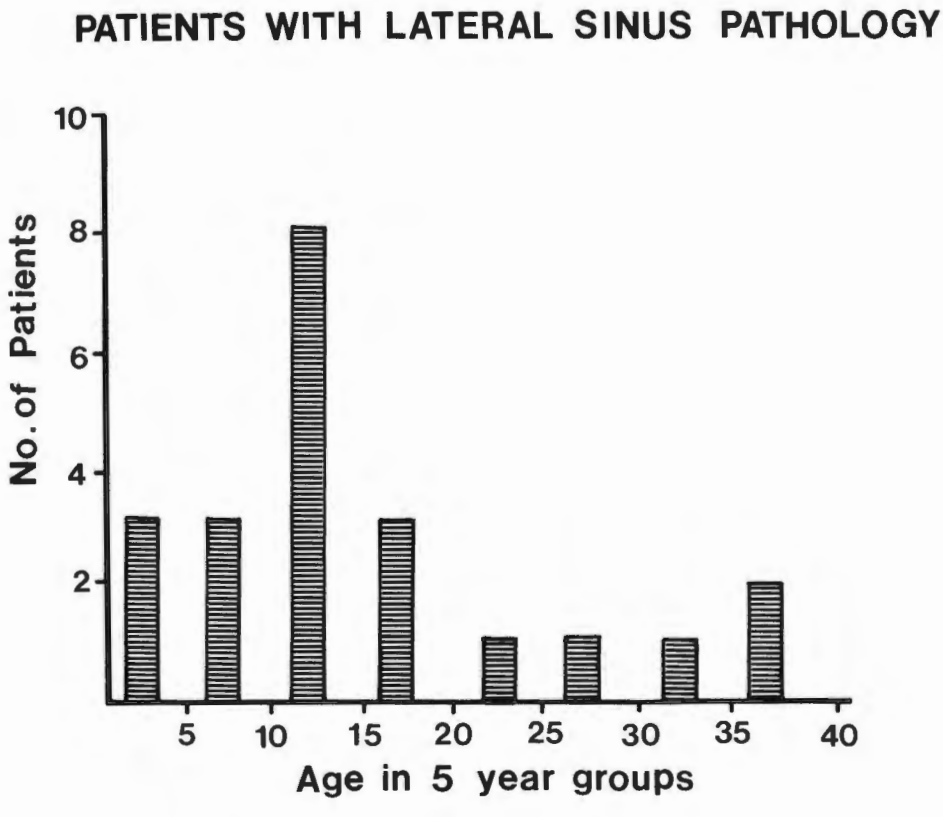
PATIENTS WITH LATERAL SINUS PATHOLOGY
No. of Patients
10
8
6
4
2
5
10
15
20
25
30
35
40
Age in 5 year groups

FIGURE 2

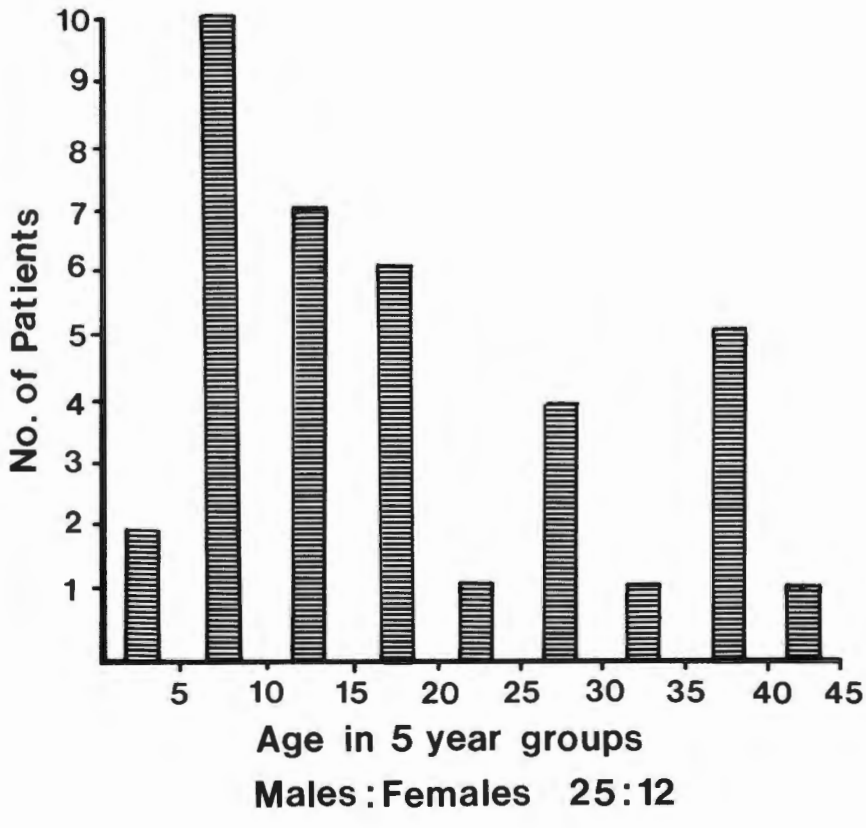
No. of Patients
10
9
8
7
6
5
4
3
2
1
5
10
15
20
25
30
35
40
45
Age in 5 year groups
Males : Females 25 : 12

FIGURE 3

TABLE 1

	Non-Cholesteatoma Patients	Cholesteatoma Patients
TOTAL NUMBER	56	74
SUBPERIOSTEAL PUS	46 (82,1%)	39 (52,7%)
CORTICAL DEHISCENCE	37 (66,1%)	22 (29,7%)
EXTRADURAL DISEASE		
Granulations	2	21
Post Fossa Pus	2	11
Middle Fossa Pus	5	22
SIGMOID SINUS PATHOLOGY	3 (5,4%)	19 (25,7%)
L S C FISTULAE	-	11 (14,9%)
TEMPORAL LOBE ABSCESS	2	14
	+ 1 Meningitis Only	
CEREBELLAR ABSCESS	-	13
SUBDURAL EMPYAEMA	1	6
MORTALITY	1	4
INTRACRANIAL EXTENSION	13 (23,2%)	58 (78,4%)
INTRADURAL EXTENSION	4 (7,1%)	33 (44,6%)

L S C LATERAL SEMICIRCULAR CANAL

TABLE 2

	Number of Patients	Lateral Sinus Pathology	%
Non-Cholesteatoma Patients	56	3	5,4
Cholesteatoma Patients	74	19	25,7
TOTAL	130	22	16,9

TABLE 3

	No of Patients	No Growth	Pure	Mixed
ACUTE	42	10 (23,8%)	24 (57,2%)	8 (19%)
ACUTE ON CHRONIC (Cholesteatoma)	70	7 (10%)	29 (41,4%)	34 (48,6%)
TOTAL	112	17 (15,2%)	53 (47,3%)	42 (37,5%)

TABLE 4

ORGANISMS ISOLATED FROM CASES OF ACUTE MASTOIDITIS

Organism	No of Isolates
AEROBES	
Grp A Streptococci	15
Strep. pneumoniae	7
Staph. aureus	6
Proteus mirabilis	4
Strep. viridans	1
Proteus vulgaris	1
Enterobacter	1
ANAEROBES	
Bacteroides sp	4

TABLE 5

ORGANISMS ISOLATED FROM CASES OF ACUTE-ON-CHRONIC MASTOIDITIS

Organism	No of Isolates
AEROBES	
Proteus mirabilis	37
Proteus sp	9
Pseudomonas aeruginosa	7
Streptococcus milleri	6
Enterococci	6
Staph. aureus	4
Esch. coli	4
Non-enterococcal grp D Streptococci	3
haemolytic strep (not grp A)	3
Proteus vulgaris	2
Haemophilus influenzae	2
Enterobacter sp	1
Serratia sp	1
Strep. pneumoniae	1
ANAEROBES	
Bacteroides fragilis	21
Bact. melaninogenicus	4
Bacteroides sp	3
Anaerobic cocci	3
"Mixed anaerobes"	3

TABLE 6

GLASGOW COMA SCALE

EYES	Sponteaneously	4
	To speech	3
	To pain	2
	None	1
BEST VERBAL RESPONSE	Orientated	5
	Confused	4
	Inappropriate words	3
	Incomprehensible sounds	2
	None	1
BEST MOTOR RESPONSE	Obeys commands	6
	Localised pain	5
	Flexion to pain withdrawal	4
	Decorticate	3
	Extension to pain	2
	None	1

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