Chapter 9: Acute suppurative otitis media and acute mastoiditis*

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* This is an abbreviation of the chapter on this subject, in Volume 6.

Definition

Acute suppurative otitis media is an inflammation of the mucous membrane lining of the middle ear cleft (consisting of the eustachian tube, tympanic cavity, mastoid antrum and mastoid air cells) produced by pus-forming organism.

Acute mastoiditis, formerly a common complication of acute suppurative otitis media, is nowadays rare in countries with well-developed primary medical care. While some degree of mastoiditis inevitably occurs early in the course of acute suppurative otitis media, since the middle ear and mastoid mucosa are in continuity, the clinical entity of acute mastoiditis consists of persistence of pain in and behind the ear despite adequate antibiotic therapy or time for natural resolution, together usually with persistence of otorrhoea, fever, and tenderness over the mastoid antrum.

Anatomy

The anatomy of the middle ear cleft is detailed in Volume 1.

Acute suppurative otitis media

Aetiology

Route of infection

(1) Via the eustachian tube.
(2) Via the tympanic membrane.
(3) Blood-borne infection.

Predisposing factors

Age

Acute suppurative otitis media is a common disease of childhood.

Socio-economic factors

The incidence is highest in populations with low hygiene, overcrowding and malnutrition.
Climate

A higher incidence of acute suppurative otitis media is seen in cold climates, especially in winter. The incidence is also higher in urban than in rural areas.

Racial factors

Studies in the USA (Brodley, Brookhouser and Tucker, 1986) have shown a higher incidence of acute suppurative otitis media in white children compared with black; a particularly high incidence is seen in Eskimos and American Indians.

Nasopharyngeal tissue masses

*Adenoids* (see Volume 6)

*Other nasopharyngeal masses*

These act in a similar way to adenoids and include polyps, teratoma, angiofibroma, lymphoma and, in adults, carcinoma (see Volume 4, Chapter 18).

Respiratory disease

Chronic rhinitis and sinusitis produce a constant flow of infected mucus which may enter the eustachian tubes, while the infected sputum of bronchitis, bronchiectasis and pneumonia may also be coughed into the nasopharynx and enter the tubes.

Allergy

The importance of allergy as an aetiological factor in acute suppurative otitis media is still debatable.

Pre-existing middle ear effusion

A pre-existing middle ear effusion may act as a ready culture medium for invading pyococci.

Immunodeficiency syndromes

Immunodeficiency syndromes, including the hypogammaglobulinaemias, are rare but important causes of recurrent upper respiratory infections including acute suppurative otitis media, and should always be excluded when recurrences are frequent. Drug induced immunosuppression may also be contributory.

Chronic systemic disorders

Chronic systemic disorders undoubtedly predispose to acute suppurative otitis media, as they do to other infective disease. Examples occurring in both children and adults are diabetes mellitus, the leukaemias, the anaemias, cystic fibrosis and nephritis.
Cleft palate

Children with cleft palate have a high incidence of middle ear disease, either acute suppurative otitis media or otitis media with effusion, due to eustachian tube dysfunction secondary to the tensor palati anomaly.

Primary ciliary dyskinesia

Primary ciliary dyskinesia, although excessively rare and more usually associated with otitis media with effusion, can also contribute to recurrent acute suppurative otitis media.

Pathology

Microbiology

While acute suppurative otitis media is appropriately considered as a bacterial disease (see Table 3.2), viruses undoubtedly play a role in many cases, paving the way for pyococcal invasion.

Middle ear inflammatory process

This can proceed quite rapidly, and consists of a stage of mucosal oedema with increased secretion, followed by hyperaemia, white cell infiltration and pus formation. This process clearly cannot be limited to the tympanum since the air spaces and mucosa of the entire middle ear cleft are in continuity; hence, tubal occlusion occurs due to mucosal swelling, preventing drainage, and involvement of the mastoid air cells also occur. If pus accumulates under pressure and there is tubal occlusion, the tympanic membrane will rupture in most cases. Destruction of cilia, normally present in the anterior part of the tympanum and in the tube, contributes to the poor drainage of thick secretions through the tube.

Spread of infection

Spread of infection can occur due to retrograde thrombophlebitis, bone necrosis, congenital dehiscences and fracture lines, as follows:

(1) intracranially, giving rise to extradural or subdural abscess, meningitis, brain abscess, lateral sinus thrombosis, and otitic hydrocephalus;

(2) to the labyrinth causing suppurative labyrinthitis;

(3) to the facial nerve canal causing facial paralysis;

(4) to the neck, by breaking through the mastoid tip, producing Bezold's or Citelli's abscess;

(5) to the petrous apex.

Details of (1) and (2) can be found in Chapter 12.
Symptoms

The variation in the clinical picture in any infection is due to the differing virulence of the invading microorganism, varying host defence, and effectiveness of and compliance in treatment.

Acute suppurative otitis media can vary from a relatively minor attack of earache with tympanic membrane hyperaemia lasting a few hours, to a fulminating febrile illness perhaps with complications requiring surgery.

By far the most common presenting symptom of acute suppurative otitis media is pain in the affected ear or ears which is accurately described and well located by older children and adults, who point to the ear canal and say the pain is 'deep inside', and frequently severe and throbbing. Usually the attack will have been preceded by an upper respiratory infection, and symptoms and signs of this may be present. Deafness in the affected ear or ears will soon be noticed by older children and adults; the disease is often bilateral in children, but in unilateral cases the hearing loss may not be apparent.

At this stage the disease may not progress and will gradually resolve, with the pain subsiding and the hearing gradually recovering. In many cases, however, it proceeds to a stage of intense pain, followed by rupture of the tympanic membrane and a complaint of aural discharge. A small percentage of both children and adults also complain of giddiness.

There may be a history of one or more of the predisposing factors described under aetiology.

Signs

Ears

In the early stages, the tympanic membrane will be injected along the handle of the malleus, around the periphery, and sometimes over the pars flaccida. Later, the whole tympanic membrane becomes hyperaemic and opaque. If infection continues and pus begins to accumulate under pressure, the pars tensa starts to bulge, mainly posteriorly, and acquires yellowish colour. Finally, the tympanic membrane ruptures and discharge will be seen in the external canal, which may be serous, serosanguineous, mucopurulent or frankly purulent. It is important to note whether the discharge has the shiny, glossy appearance of mucus - if the discharge contains mucus it can only come from the middle ear. The presence or absence of an offensive odour from the discharge should also be noted - if present it suggest underlying chronic otitis media. (See also Differential diagnosis below.)

At this time the pain usually subsides. After mopping or aspirating discharge, it is usually possible to see a small central perforation, commonly in the posterior segment of the pars tensa. Sometimes, however, the perforation may be seen only with difficulty as the oedematous edges of the middle ear mucosa tend to fill it in. It may be located by aspirating through a Siegle's speculum, or under the operating microscope, when a blood of discharge may be seen passing through the perforation. The perforation may also be anterior, but is only
marginal or in the pars flaccida in acute-on-chronic middle ear disease. If the infection has followed trauma to the tympanic membrane, a jagged perforation may be seen. If a ventilation tube is in situ, pus may be seen pulsating through the lumen.

Mastoid tenderness, elicited by pressure over McEwen’s triangle, is almost invariably present early in the course of acute suppurative otitis media. It assumes significance as a sign of mastoiditis if it persists or increases despite adequate treatment.

During resolution, the hyperaemia fades and the perforation heals, often leaving no trace but sometimes leaving a scar. In older children and adults, the Rinne and Weber tests will indicate conductive deafness. In younger children the hearing for a whispered voice will be impaired.

Nose and throat

As stated, the commonest cause of acute suppurative otitis media is the common cold, and examination of the nose and throat may show inflammation of the mucosa and nasal mucoid discharge.

General signs

Children are frequently febrile, but adults rarely so.

Signs of complications

Acute suppurative otitis media may be a serious illness. Complications may develop rapidly and it is necessary to be alert to the signs of these.

Tenderness and oedema over the mastoid process with protuberance of the pinna indicate mastoiditis, as do sagging of the posterosuperior canal wall, and granulation tissue pouting through the perforation, with discharge persisting for 3-4 weeks from the onset. (See also Differential diagnosis and Acute mastoiditis below.)

Sick children should always be tested for neck stiffness, and in severe cases a thorough examination of the central nervous system should be carried out to exclude intracranial complications. Nystagmus must be looked for in patients complaining of vertigo, and the fistula test carried out.

Investigations

Microbiology

In all cases when otorrhea is present, an ear swab should be taken for culture and sensitivities.
Blood studies

A full blood count, including differential white cell count is mandatory in recurrent cases or very ill patients, or those in whom complications, particularly intracranial ones, are suspected.

Quantitative immunoglobulin electrophoresis to detect varying degrees and types of hypogammaglobulinaemia should also be carried out in patients with frequent attacks of acute suppurative otitis media.

Audiometry

Pure tone audiometry needs to be performed fairly early in the course of acute suppurative otitis media, but need not be done when the patient is in severe pain and febrile.

Tympanometry should not be carried out in the acute stage.

Mastoid X-rays

X-rays are only required if mastoiditis is diagnosed and is not responding to medical treatment (see Chapter 2).

Diagnosis

This is usually straightforward and is based on the history of earache, deafness, and perhaps otorrhoea, probably preceded by a respiratory infection, together with the inflammatory changes found on examination which have been described above. However, there are some pitfalls and these are discussed below.

Differential diagnosis

This is considered later under Acute mastoiditis.

Prognosis

In this antibiotic era, complete resolution is the rule, with absence of complications, healing of the tympanic membrane, and restoration of normal hearing. In a few, a sterile middle ear effusion or a perforation persist, and only a very small percentage proceed to acute mastoiditis. Life-threatening intracranial complications are very rare, and are more often associated with a pre-existing chronic otitis media.
Complications

1. Mastoiditis, which may also lead to postauricular abscess or Bezold's and Citelli's abscess.

2. Facial paralysis.

3. Intracranial complications - extradural abscess, subdural abscess, meningitis, brain abscess, lateral sinus thrombosis and otitic hydrocephalus.

4. Labyrinthitis.

5. Petrositis and Gradenigo's syndrome.

Mastoiditis and petrositis are described below. Intracranial complications and labyrinthitis are rare nowadays, but when they do occur it is more commonly in association with chronic otitis media. These conditions are described in Chapter 12.

Sequelae

(1) Persistence of a sterile middle ear effusion.

(2) High-tone sensorineural deafness.

(3) Persistent perforation of the tympanic membrane; discharge may also persist, and the disease may evolve into chronic suppurative otitis media.

(4) Extensive scarring of the tympanic membrane, middle ear adhesions and resorption of ossicles may occur in recurrent cases (adhesive otitis).

Treatment of acute suppurative otitis media

Treatment is considered under the following headings:

(1) curative
    medical
        general
        analgesics
        topical
        antibiotics
        [decongestants]
    surgical - myringotomy
(2) [prophylactic]
(3) treatment of associated conditions
(4) treatment of complications.

The square brackets indicate treatment modes not generally considered to be appropriate.
Curative

Medical treatment

General

Both children and adults are best managed in bed in the acute phase, in a warm room, with adequate humidity to maintain ciliary function.

Analgesics

These must be given in adequate dosage and with sufficient frequency to control pain.

Topical

When otorrhoea is present the discharge should be gently mopped with dry sterile cotton wool or sucked from the canal, as often as it recurs.

Antibiotics

Most otologists and primary care physicians in the UK would favour early administration of antibiotics in all but the most minor cases, despite the fact that some cases may be viral, and notwithstanding the need to avoid overprescribing.

Route of administration. Oral administration is the route of choice except in very severe cases. In these, one or more antibiotics may be given intravenously.

Duration. Antibiotics should usually be given for 5-10 days, depending on the severity of the case.

Choice of antibiotic. Administration should not be started before an ear swab is taken (or nose and throat swabs if the ear is not discharging), but after this there is no need to wait for the result. Amoxycillin is a useful first-line treatment as it is well tolerated and the common bacteria of acute suppurative otitis media are usually sensitive to it. Other useful antibiotics are erythromycin, trimethoprim, trimethoprim with sulphamethoxazole (co-trimoxazole), and cefaclor. Severe and fulminating cases can be given a combination of ampicillin, flucloxacillin and metronidazole intravenously.

The response to the chosen regimen should be monitored carefully and, if ineffective, it should be altered according to the results of the swab cultures and organism sensitivities.

Decongestants. Both systemic and topical decongestants are often prescribed in the hope that they will improve the patency of the eustachian tube and thus improve middle ear drainage. Unfortunately, however, many trials have now shown that systemic decongestants are no better than a placebo in the management of middle ear disease. Since systemic administration of pseudoephedrine and similar compounds may sometimes cause sleep disturbance, irritability and, occasionally, psychotic symptoms, especially in children, the conclusion is inescapable that their use is unwise. The use of topical decongestants in acute
suppurative otitis media is not recommended as they are unlikely to produce a useful effect on the eustachian tube.

**Surgical treatment**

While the vast majority of ears with acute suppurative otitis media will respond to the above regimen of appropriate antibiotics, bed-rest and analgesia, and while some tympanic membranes will rupture spontaneously with or without treatment, in a very small minority there is persistence of pain and temperature with a red bulging tympanic membrane despite adequate medical management. Myringotomy should then be undertaken with a view to releasing pus accumulating under pressure.

The operation is carried out under general anaesthesia using an operating microscope (see Volume 6), and with full aseptic procedures. The patient is placed supine on the operating table with the head turned to one side. Using an aural speculum and angled myringotome, a radial incision is made in the postero-inferior segment; the maximum bulging is posterior in acute suppurative otitis media, and the inferior incision avoids the risk of damaging the ossicular chain, chorda tympani and facial nerve. Pus then gushes out under pressure, and a swab is taken and sent for culture and sensitivities. Residual pus is gently sucked out. The incision should be about 3-4 mm in length; tiny incisions tend to heal too quickly and allow put to reaccumulate in the middle ear cleft. Ventilation tubes should not be inserted in acute supplicative otitis media.

Postoperatively, on recovering from anaesthesia the patient will usually say that the earache has disappeared, and usually the temperature quickly returns to normal. Antibiotic treatment is continued until resolution is virtually complete, but the regimen is changed if necessary as soon as the results of the swab taken at operation are known.

**Prophylactic treatment**

Long-term prophylaxis with oral antibiotics is not generally recommended.

**Treatment of associated conditions**

The treatment of acute suppurative otitis media, especially when recurrent, should include a search for and management of treatable associated disease. Rhinitis and sinusitis should also be looked for and treated vigorously. The presence of lower respiratory infection requires treatment with the help of a respiratory physician. Other conditions referred to under aetiology must receive the appropriate management.

**Treatment of complications and sequelae**

**Persistence of middle ear effusion**

This is a common sequel to an attack of acute suppurative otitis media and therefore there must be careful follow-up of each case by otoscopy and tympanometry. The treatment is described in Volume 6, Chapter 12.
Persistent perforation of the tympanic membrane

See Chapter 11 on reconstructive surgery of the ear.

Labyrinthitis and intracranial complications

These are described in Chapter 12.

Facial paralysis

The treatment of facial paralysis occurring in the course of middle ear cleft infection is the vigorous treatment of the primary condition - when the infection is controlled the facial nerve recovers.

Mastoiditis and petrositis

These are described below.

Acute mastoiditis

Aetiology and pathology

Acute mastoiditis is a complication of acute suppurative otitis media. In developed countries with effective primary and secondary health care, it is nowadays rare, largely due to the widespread use of antibiotics for acute suppurative otitis media. However, if the preceding attack is untreated, or fails to respond, the inflammatory process will persist and increase in the mastoid air cells. The accumulation of pus in the air cells leads to necrosis of the bony walls of the cells producing the so-called 'coalescent mastoiditis'. For a time the disease may remain walled off within the mastoid bone, but eventually it will spread:

(1) laterally through the lateral outer table of the mastoid bone to give:

(i) a subperiosteal abscess and, if pus ruptures through the periosteum
(ii) subcutaneous abscess

(2) superiorly and posteriorly, giving rise to:

(i) extradural abscess
(ii) subdural abscess
(iii) meningitis
(iv) brain abscess
   (a) the temporal lobe
   (b) the cerebellum
(v) lateral sinus thrombosis
(vi) otitic hydrocephalus

(3) medially causing
   (i) labyrinthitis
   (ii) petrositis and Gradenigo's syndrome (due to direct spread through a pneumatized petrous bone
(4) inferiorly through the mastoid process tip or medial wall causing:
   (i) Bezold's abscess (tracking along the sternomastoid muscle)
   (ii) Citelli's abscess (tracking along the posterior belly of the digastric muscle)
(5) anteriorly to the facial nerve canal causing facial paralysis, and also to the posterosuperior external auditory canal wall, causing the appearance of sagging.

**Predisposing factors**

These are the same as described above for acute suppurative otitis media. The disease can occur at any age.

**History**

The patient will have had an attack of acute suppurative otitis media, with the characteristic symptoms and signs described above, anything from a few days up to 3 or 4 weeks previously.

**Symptoms**

**General**

Mastoiditis is frequently a serious illness with pyrexia and general malaise.

**Local**

Commonly there is persistence of earache, otorrhoea and increasing hearing impairment, from the time of onset of the preceding acute suppurative otitis media. The presence of unilateral headache is a danger sign suggesting the onset of intracranial complications. Similarly a complaint of giddiness is a warning that purulent labyrinthitis is imminent, or developing.

**Signs**

**General**

The patient will frequently appear pale, ill and restless. There may be pyrexia of 40°C or more in children, although in adults pyrexia may be low or absent.

**Local**

*External auditory canal*

On examination of the external auditory canal, there may be:

(1) discharge

(2) sagging of the postero-superior canal wall.
**Tympanic membrane**

(1) Perforation is almost invariably present, and is nearly always postero-central.

(2) Granulations or a polyp, bright red in colour, are sometimes seen pouting through the perforation.

**Signs of complications**

Severe headache, drowsiness, vomiting, and neck stiffness are serious signs of intracranial complications and must prompt an immediate and thorough examination of the central nervous system. Vertigo with nystagmus suggests purulent labyrinthitis.

**Postauricular area**

(1) Mastoid tenderness, elicited by pressure over McEwen's triangle, will invariably be present.

(2) Swelling over the mastoid bone may be present, and if so either the postauricular groove is accentuated indicating that the pus is still subperiosteal, or the postauricular groove is absent, because either the periosteum has given way and the pus is subcutaneous, or there is simple inflammatory oedema over the mastoid. The presence of fluctuation will distinguish the later abscess formation from the earlier simple inflammatory oedema.

(3) Protuberance of the pinna can occur either due to simple inflammatory oedema over the mastoid, or of subcutaneous abscess; subperiosteal abscess with retention of the postauricular groove does not push the pinna forwards unless the abscess is very large.

**Investigations**

These are the same as for acute suppurative otitis media.

**Mastoid X-rays**

While seldom required in simple acute suppurative otitis media, where mastoiditis is present and not responding to treatment, X-rays (see Chapter 2) may show not only clouding of cells (always present in acute suppurative otitis media), but also breaking down of bony air cell walls, indicating progressive disease. The films are also a useful guide if surgery is required, as to the extent of pneumatization which varies greatly.

**Diagnosis**

Diagnosis is made on the history, symptoms and signs, sometimes supported by X-rays, as already described. The principal features can be summarized as follows: an attack of acute suppurative otitis media fails to resolve and is followed by persistent or recurrent earache, pyrexia and otorrhea, increasing deafness, with mastoid tenderness and sometimes a protuberant pinna.
Differential diagnosis of acute suppurative otitis media and acute mastoiditis from other conditions

Acute suppurative otitis media

Acute suppurative otitis media may sometimes have to be distinguished from the following conditions:

Otitis externa

Otitis externa may also give earache and otorrhoea, and the tympanic membrane may appear red as the outer layer is in continuity with the canal epithelium and is frequently involved in the inflammatory process (myringitis). The discharge is frequently watery, but if purulent it never has the shiny, glossy appearance of middle ear discharge due to the presence of mucus. The hearing in otitis externa is normal or only slightly impaired. Itching is a very common feature of otitis externa.

Very severe otitis externa may mimic acute mastoiditis (see below).

Tympanic membrane hyperaemia

The whole tympanic membrane can become quite diffusely red in a child who is crying. Since he may be crying because he has earache, time must be allowed for him to settle down and then the examination is repeated.

Otitis media with effusion

The tympanic membrane may sometimes look pinkish and opaque, but is never as intensely red as in acute suppurative otitis media.

Myringitis haemorrhagica bullosa

This condition frequently occurs during epidemics of respiratory viruses such as influenza, and is characterized by excruciating earache followed by a small quantity of serosanguineous discharge. Inspection of the tympanic membrane shows either the presence of haemorrhagic blebs, or the outlines of ruptured blebs. When uncomplicated the hearing is usually normal. Secondary bacterial invasion of the middle ear may occur, so the two conditions may coexist.

Other conditions

There are conditions which stem from other causes of otalgia (see Chapter 13). In referred pain the tympanic membrane and hearing are not affected.

Acute mastoiditis

This may have to be distinguished from:
**Acute severe otitis externa**

This is usually localized in the form of a furuncle and may lead to really marked postauricular oedema and protuberance of the pinna; this, together with severe earache and some purulent otorrhoea, produces the resemblance to acute mastoiditis. However, in furunculosis, there is severe pain on pushing the tragus gently in and on pulling gently on the pinna. There will be no history of a preceding attack of acute suppurative otitis media. The hearing is usually normal or only slightly impaired. If the postauricular groove is accentuated, this is a sign of subperiosteal pus which has spread from the mastoid. (Note that absence of this sign is not a differentiating factor.) X-rays of the mastoids will show apparent cloudiness of the air cells in either condition (due in external otitis to the overlying oedema), but if breaking down of the bony mastoid air cell walls is shown, this indicates that mastoiditis is present.

**Postauricular lymphadenitis**

Very rarely, suppuration in a postauricular lymph node, due to infection in the skin or scalp, may cause confusion. However the tympanic membrane, external auditory canal and hearing will be found to be normal.

**Erysipelas**

Erysipelas may occasionally affect the skin of the postauricular area, and resemble mastoiditis because of pain, fever and red oedematous skin. However, careful examination will reveal a raised, red spreading edge of the lesion, contrasting sharply with the normal pale adjacent skin. The external canal skin, tympanic membrane, and the hearing, will all be normal.

**Complications of acute mastoiditis**

These have been referred to under pathology for both acute suppurative otitis media and acute mastoiditis and will not be described here, except petrositis. The reader is reminded that labyrinthitis and the intracranial complications are discussed in the chapter on complications of otitis media (Chapter 12).

**Acute petrositis**

The degree of pneumatization of the temporal bone is extremely variable, but may extend right through the petrous bone to its apex. If so, when there is mastoiditis, there is nothing except host defence and timely treatment to prevent infection spreading right to the petrous apex. However, acute petrositis is now excessively rare, and even in the preantibiotic era, it was not common.

**Clinical picture**

The clinical picture is that of the preceding acute suppurative otitis media and acute mastoiditis which fails to respond to treatment, sometimes even if this included cortical mastoidectomy. There is persistence of earache and temperature, then pain is felt in the
distribution of the ipsilateral trigeminal (fifth cranial) nerve. Finally, involvement of the ipsilateral abducent (sixth cranial) nerve gives rise to diplopia, and examination of the eye movements will show paralysis of the external rectus muscle of the eyeball on the affected side (sixth nerve paralysis).

**Gradenigo's syndrome**

The features of this are acute infection of the middle ear cleft associated with pain in the distribution of the trigeminal nerve and sixth nerve paralysis. The syndrome is due to the close anatomical relationship of the fifth and sixth nerves with the petrous apex. Besides acute petrositis it may also be due to an extradural abscess or a patch of meningitis overlying the petrous apex.

**Diagnosis**

Diagnosis depends on the foregoing clinical picture, assisted by polytomography and/or computerized tomographic (CT) scanning of the temporal bone.

**Treatment**

Intensive antibiotic treatment (see above under Treatment of acute suppurative otitis media and below) is begun immediately and in a previously untreated case with a short history, this may well be all that is required. However, if the patient fails to respond in 24-48 hours, or if cortical mastoidectomy has already been performed but the disease nevertheless progresses to petrositis, further surgical exploration will be required. This is considered below following surgical treatment of acute mastoiditis.

**Treatment of acute mastoiditis**

**Medical**

Even when a child or adult presents with an advanced case of acute mastoiditis with postauricular oedema and protuberant pinna, the treatment is initially medical in hospital, and even the majority of these cases will resolve completely. The exceptions are those cases with postauricular fluctuation, previous adequate medical management, or suspected intracranial complications.

**Surgical**

**Cortical mastoidectomy**

Cortical mastoidectomy is indicated:

(1) if subperiosteal fluctuation, suspected intracranial complications, or a neck abscess are present when the case presents
(2) if there is persistence of pain, temperature, and otorrhoea, or even just profuse otorrhoea on its own, after 2-4 weeks of adequate medical management including use of the correct antibiotic based on culture results, and known compliance in the antibiotic regimen.

The aim of the operation is to exenterate the mastoid air-cell system as completely as possible.

**Preoperative investigation**

Besides those investigations previously mentioned, the patient's fitness for general anaesthesia should be assessed, when possible an immediate preoperative audiogram should be carried out, the facial movements examined to exclude preoperative facial paralysis, the eye movements examined to exclude nystagmus, and the central nervous system examined to exclude or assess intracranial complications. Mastoid X-rays not only help to confirm the indications for surgery, but also give guidance to the surgeon on the extent of pneumatization and the positions of the dura of the middle and posterior cranial fossae.

**Preparation**

A postauricular incision is used and, as it is fairly close to the hair-line, the hair should be taped out of the way with Sellotape or other adhesive tape. It is not usually necessary to shave the hair in children.

**The operation**

This is performed under general anaesthesia. A curved incision is made through the skin of the postauricular region a few millimetres behind and parallel to the postauricular groove (see Volume 6).

Care must be taken in the lower half of the incision in infants, in whom the mastoid process is undeveloped, and the facial nerve, as it leaves the stylomastoid foramen, is therefore superficial. The periosteum is elevated forwards as far as the lateral end of the posterior bony meatal wall, backwards for a few millimetres, and upwards (pushing up the temporalis muscle at the same time) to the level of the upper attachment of the pinna.

In exenterating part of the mastoid bone to uncover the antrum it must be remembered that: (a) the antrum is at a depth of 15 mm in the adult, but only a few millimetres in the infant; (b) the surface marking of the antrum is McEwen's triangle; and (c) the position of the middle and posterior fossa dura can be judged by examining the lateral oblique X-ray of the mastoid.

Bearing these landmarks in mind, bone is gradually removed with the drill until the antrum is exposed.

If pus is encountered a further swab is taken and sent for culture. To confirm that the antrum - rather than merely a large cell - has been entered - a small Dundas Grant probe is passed into the aditus. This should be done gently to avoid dislodging the short process of the incus. At the same time the size of the aditus can be judged; if it is very small it may be
enlarged slightly with a fine bone curette to ensure adequate drainage of the middle ear. (Note that the bony posterior meatal wall must be preserved, and the skin not dissected from it.)

The air cells are now followed and removed in every direction. It is particularly important to clear all the cells from the sinodural angle. The smooth bone covering the middle fossa dura above and the lateral sinus posteriorly is usually recognized.

There is frequently a group of cells in relation to the vertical part of the facial nerve which are best removed under the operating microscope. In a well-pneumatized skull, cells may extend anteriorly into the root of the zygoma and posteriorly into the occipital bone; these too must be followed as far as is practicable.

It is not necessary to remove the whole tip of the mastoid; all cells up to the tip should be removed.

The bony cavity thus created has the antrum as its deepest point, and is bounded above by the dural plate, posteriorly by the sinus plate and anteriorly by the bony meatal wall and aditus. In patients with intracranial complications, a small area of both middle fossa dura and lateral sinus should be exposed; if this reveals granulations or an extradural abscess, exposure of dura is continued until healthy dura is found.

A small drain is inserted into the antrum and led out near the mastoid tip. The skin is closed with interrupted sutures, and a dressing pad and bandage should be applied firmly to prevent a subcutaneous haematoma.

**Postoperative care**

As soon as the patient is conscious, the facial movements are examined to exclude operative damage to the facial nerve. Antibiotic therapy is continued.

The patient's temperature should be taken every 4 hours. It usually falls dramatically within the first 24 hours, when the patient can be allowed up.

The drain should be removed when there is no further discharge either through the wound or through the external meatus. In practice this is usually after 2-3 days, but the drain should be left longer if necessary.

**Complications**

Complications of the operation are few and due mainly to errors of technique.

*Persistent deafness.* This may be due to the following:

1. incus dislocation or removal
2. persistent infection due to residual cells.
Complete facial nerve paralysis. If present immediately postoperatively, but not preoperatively, the facial nerve has been damaged at operation, and the mastoid must be reopened and the facial nerve explored.

Meatal stenosis. This may occur if the bony meatal wall is taken down and the skin dissected off the bony wall. It requires excision of the stenosed area and firm packing of the canal until re-epithelialization occurs.

Incision and drainage of a postauricular abscess

As described, this condition occurs when pus spreads beyond the confines of the middle ear cleft and ruptures through the lateral surface of the mastoid process into the subperiosteal space. This then would normally be an indication for cortical mastoidectomy, since incision and drainage alone may not be sufficient to enable the mastoiditis to resolve. However, in two circumstances simple incision of the abscess is indicated:

1) In infants, who may occasionally develop a postauricular abscess from a middle ear infection, but in whom the mastoid is not pneumatized nor the mastoid process developed; particular care must be taken with the incision because of the superficial placing of the facial nerve.

2) In a patient, of any age, judged too ill to sustain even the not very long procedure of cortical mastoidectomy, in whom time is of the essence and rapid evacuation of at least some pus is thought to be adequate for the time being - for such cases an even simpler alternative is needle aspiration.

The procedure consists of a simple postauricular incision over the point of maximum fluctuation. When the pus is found a swab is taken, then as much pus as possible is sucked out. A small drainage tube is stitched in and the incision closed.

Myringotomy

Myringotomy alone is obviously not a sufficient form of surgery for acute mastoiditis, but in those few patients who require surgery, but in whom there has been no spontaneous perforation of the tympanic membrane, a myringotomy should be performed as well as other appropriate procedures.

Surgical treatment of acute petrositis

The indication is the presence of acute petrositis, perhaps with Gradenigo's syndrome, and failure to respond rapidly to medical treatment.

The following account of the various approaches to the petrous cells used in the past, has been given by Mawson (1979). It is emphasized that such surgery would be exceptionally rare nowadays; it is difficult and hazardous, and should only be performed by those with very considerable familiarity with the field.
Extrapetrosal drainage

A cortical mastoidectomy operation is performed or reopened. Any fistulous tracks found must be followed. If necessary surgery must proceed to radical mastoidectomy. Tracks may then be found which lead towards the apex from the hypotympanum or attic.

Various routes for a deep exploration are as follows:

**Eagleton's operation**

A wide exposure of the dura of the middle fossa is made by removal of the tegmen, the base of the zygoma and part of the squamous temporal bone. The dura of the middle fossa is gently elevated towards the petrous apex.

**Almoor's operation**

The petrous apex is approached through a triangle bounded by the tegmen tympani above, the carotid artery anteriorly and the cochlea posteriorly.

**Ramadier's operation**

Here the petrous apex is approached more widely. The tympanic plate of the external auditory canal, posterior to the base of the glenoid fossa suture line, is removed. The carotid artery is lifted forward by a gauze sling. The petrous apex may then be explored through the posterior wall of the bony carotid canal.

**Frenckner's operation**

Sometimes a group of cells runs under the arch of the superior semicircular canal. This is a good approach to the petrous apex, but it would have to be combined with an approach to the hypotympanum.