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Acute otitis media in children

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SEARCH STRATEGY

The data in this chapter are supported by searches of Medline and the Cochrane Controlled Trials Register, using the term acute otitis media. Reference lists were reviewed for further articles, and authors of recent presentations contacted personally for their reference lists.

INTRODUCTION

For such a common childhood infection, acute otitis media (AOM) remains something of an enigma. It is hard to diagnose accurately and on existing evidence, as opposed to custom and tradition, there is still a high level of uncertainty over how it should best be treated. This is against a background of increasing bacterial resistance to antibiotics. There is plenty of evidence in the literature of the relative frequencies of viral and bacterial pathogens in AOM, but this is often of little help to the clinician on the spot in an individual case. Some of the epidemiological evidence is also relatively 'soft', since it is based on the flawed premise that AOM can accurately be diagnosed from the history and otoscopy alone, unsupported by tympanometry or tympanocentesis. In this chapter, the authors have tried to thread their way through the often conflicting evidence about the practical management of AOM at the same time as covering what is known of the pathology, epidemiology and complications of this commonest of childhood illnesses.

We have found ourselves uncomfortably often using expressions such as 'uncertainty,' 'insufficient evidence,' or 'limited information,' and quoting [****] and [***] levels of evidence. Even a metanalysis, the supposed gold standard of evidence, is only as good as the studies it covers. It must be remembered that the prevalence of otitis media with effusion (OME), 'glue ear,' only began to be widely appreciated after the development of the twin tools of universal hearing screening and the tympanometer in the 1960s and early 1970s, respectively. The accuracy of correct reporting of cases of AOM lags behind that of OME mainly because in the majority of large population studies of AOM the essential presence of fluid in the middle ear is not confirmed by tympanometry and audiometry.

DEFINITION

AOM may be defined clinicopathologically as inflammation of the middle ear cleft of rapid onset and infective

origin, associated with a middle ear effusion and a varied collection of clinical symptoms and signs. It is synonymous with acute suppurative otitis media. It normally develops behind an intact tympanic membrane, but may include acute infections arising in the presence of ventilation tubes or existing tympanic membrane perforations. The requirement to confirm a middle ear effusion, and the nature of the symptoms and signs, vary between authors.[1, 2]

The literature supports four broadly defined subgroups of AOM.

1. **Sporadic** episodes occurring as infrequent isolated events, typically occurring with upper respiratory tract infections.
2. **Resistant** AOM: persistence of symptoms and signs of middle ear infection beyond three to five days of antibiotic treatment.
3. **Persistent** AOM: persistence or recurrence of symptoms and signs of AOM within six days of finishing a course of antibiotics.
4. **Recurrent** AOM: either three or more episodes of AOM occurring within a six-month period, or at least four or six episodes within a 12 month period (no consensus has been reached on the latter).

Groups two and three appear similar at first glance and this distinction may be questioned. It is included to maintain some consistency with the wider literature.

Grading of the severity of an episode has been attempted and has merit both clinically and for research. Pyrexia from 37.5–39°C, vomiting and severity of otalgia have been used.[3, 4] [*]

DIAGNOSIS

Diagnosis is based on the combination of often nonspecific symptoms, evidence of inflammation of the middle ear cleft and, by some authors, by the additional confirmation of a middle ear effusion. Diagnostic difficulty has affected the quality of research into AOM. There may well not be a clear history of a crescendo of otalgia in a coryzal child, followed by rapid symptomatic relief associated with tympanic membrane perforation and associated blood-stained otorrhoea. The difficulty in establishing clear diagnostic guidelines has been highlighted in an analysis of 80 studies of AOM.[5] In diagnosing AOM, only 52.5 percent of the studies cited middle ear effusions, 32.5 percent included symptoms and signs of inflammation and 2.5 percent considered the rapidity of onset. Clinicians recognize this difficulty. A large multinational study rated clinicians diagnostic certainty in children under one year of age at only 58 percent, rising to 73 percent in those over 31 months.[6]

Symptoms

Diagnosis by symptomatology alone is inaccurate because of the young age of most patients, and the nonspecific nature of the symptoms. One-third of children may have no ear-related symptoms. Two-thirds may be apyrexial.[7] Symptoms suggestive of AOM include rapid onset of otalgia, hearing loss, otorrhoea, fever, excessive crying, irritability, coryzal symptoms, vomiting, poor feeding, ear-pulling and clumsiness (**Table 73.1**). AOM most commonly develops three to four days after the onset of coryzal symptoms. The otalgia will settle within 24 hours in two-thirds of children without treatment.[9] The otorrhoea, if present, is mucopurulent and may be blood-stained. Symptomatic relief is obtained without treatment in 88 percent by day four to seven. The hearing loss, caused by the middle-ear effusion, occurs early in the illness and may persist at greater than 20 dB for one month in over 30 percent, and two months in 20 percent of children. [**/*]

Signs

The child may appear unwell, and may rub his or her ear. The diagnosis is often confirmed, rightly or wrongly, by an attempt at otoscopic assessment of the tympanic membrane. However, a poorly functioning otoscope, the moving target of a child's head, the narrow ear canal of an infant, the natural redness of the tympanic membrane of a screaming child, wax and, above all, the inaccuracy of an untrained (or even trained) eye, straining to interpret a two-dimensional image, all combine to make otoscopy an imprecise art. Since trained observers have been shown to have only an 85 percent accuracy in otoscopic diagnosis,[10] it would not be surprising for a sensible primary care physician to rely more on history and the general aspect of a child than on otoscopic findings. With these reservations, diagnosis may be supported by otoscopic assessment of tympanic membrane colour, position and mobility (**Figure 73.1**). In AOM the tympanic membrane is usually opaque. It is most commonly yellow, or

Table 73.1 Relation of reported symptoms to presence of AOM in 302 children under four years of age.

Symptom	Sensitivity	Specificity	Positive predictive value	Negative predictive value
Earache	60	92	83	78
Restlessness	64	51	46	68
Rhinitis	96	8	41	74
Cough	83	17	40	61
Fever	69	23	38	53

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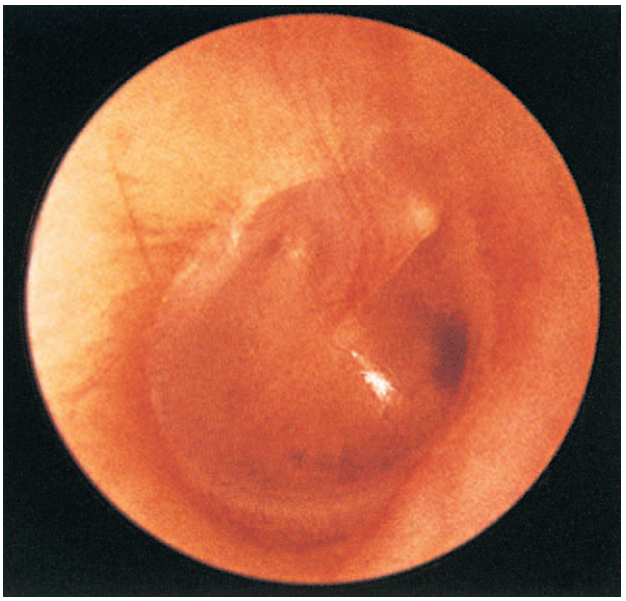

Figure 73.1 Otoscopic findings in early AOM.

yellowish pink in colour, being red in only 18–19 percent.[7, 10] The position of the tympanic membrane reliably predicts AOM only when it is bulging. Hypomobility of the drum demonstrated by pneumatic otoscopy has been shown to aid diagnosis[10] and is felt essential in some countries,[1] although others including the Dutch[2] take a more pragmatic view and do not include this in their diagnostic criteria. Should the drum have perforated, or a ventilation tube be *in situ*, mucopurulent otorrhoea will be seen. [**/*]

Investigations

Tympanometry may be used to establish the presence of a middle ear effusion, but is not usually available. Tympanocentesis and culture of middle ear effusion have been used in a number of studies assessing diagnostic accuracy of clinical signs, and establishing the organisms prevalent in a community. It is rarely required to make the diagnosis, though may be considered in high risk children such as the immunocompromised, an unwell neonate, those that fail to respond to conventional treatment and children who are seriously ill or have complications of AOM. Taking a bacterial swab of persistent otorrhoea following perforation is recommended. Nasopharyngeal swabbing for bacterial culture has been assessed but the correlation with middle ear organisms has been too weak to recommend it clinically.[11]

Specific investigation may be prompted by recurrent AOM not responsive to conventional treatment. Both iron deficiency anaemia and white blood cell disorders have been associated with AOM, so a full blood count is indicated. Immunoglobulin assay may be appropriate: Ig A, G (with subclasses) and M are typically assessed. Children with recurrent infection of ventilation tubes may also merit investigation for primary ciliary dyskinesia, particularly if nasal and pulmonary symptoms coexist. [**/*]

Differential diagnosis

Pain may be referred from tonsillitis, teething, temporomandibular joint disorders or simply be the result of an uncomplicated upper respiratory tract infection. In a screaming child, the tympanic membrane may well appear red. Diagnostic confusion may occur with acute mastoiditis, otitis media with effusion, otitis externa, trauma, Ramsey Hunt syndrome and bullous myringitis. Very rarely, AOM may be the first indication of serious underlying disease, such as Wegener's granulomatosis or leukaemia.

AETIOLOGY

Microbiological, anatomical and environmental factors combine with altered host defence mechanisms to predispose to infection. Genetic predisposition to recurrent AOM is being increasingly cited in the literature.

Infective agents

AOM results from infection of the middle ear cleft. Both bacterial and viral infections are implicated. These infections may occur in isolation or combination.

VIRUSES

Clinically it is apparent that AOM is commonly associated with viral upper respiratory tract infections. As our ability to identify these improves, the role of viruses in the aetiology of AOM is becoming clearer. Increasing use of polymerase chain reaction assays for respiratory viruses suggests 60–90 percent of cases of AOM may be associated with viral infection.[12] In one study, a specific viral cause of upper respiratory tract infection was shown in 41 percent of children with AOM.[13] The viruses most commonly associated with AOM vary between studies, but in decreasing frequency include:

- respiratory syncytial virus (RSV);
- influenza A virus;
- parainfluenza viruses;
- human rhinovirus;
- adenoviruses.

This heterogeneity is important when considering vaccination against viruses as a prophylactic measure.

The mechanism by which they give rise to AOM is likely to vary between viruses. Viral material has been demonstrated in the middle ear aspirates of children with

AOM in 48–71 percent of cases.[12] The viral material may arrive either passively along the Eustachian tube along with other nasopharyngeal secretions or may actively invade the middle ear cleft possibly by haematogenous spread. These alternative routes of entry are suggested by the wide variation in rates of isolation of specific viral strains in the middle ear during systemic infection, ranging from 4 to 74 percent of cases dependent upon the specific virus. If all arrived passively, similar rates of isolation would be expected. This implies some viruses may be actively invading the middle ear cleft, and may be contributing directly to mucosal inflammation. RSV invaded the middle ear most frequently.[13] In contrast, those arriving passively appear to cause AOM by virtue of their action on the Eustachian tube, on bacterial adherence, and on host immunity.

There is good clinical and animal evidence that viral infection affects Eustachian tube function.[12] At a cellular level there is release of multiple inflammatory mediators from cells within the nasopharynx. Ciliated epithelial cells numbers decline, mucus production increases in the Eustachian tube and negative middle ear pressure results. This is likely to predispose to AOM.

Alteration of host immunity has been documented after viral infections, increasing susceptibility to bacterial infections. Cell-mediated immunity has been shown to be affected by RSV infection, and neutrophil function altered by influenza viruses. In a study of children with bronchiolitis caused by RSV, 62 percent developed AOM. Bacteria were isolated from the middle ear in all these children.

The ability of bacteria to colonize and adhere to the nasopharyngeal epithelium appears to be increased by certain viral infections. Increased colonization by pathogenic bacteria may predispose to AOM.

Viral and bacterial infection coexist in the middle ear cleft in AOM in as many as two-thirds of cases where viruses have been identified. This is important as clinical studies show that children who have both viruses and bacteria in their middle ear are very much more likely to have a poor response to antibiotics when compared to those with bacteria only (33 versus 3 percent failure respectively, in one study[14]). Why this should be is unclear, but may be related to the greater concentrations of inflammatory mediators in ears in which both bacteria and viruses are present. [**/*]

BACTERIA

The bacteria isolated from the middle ear in AOM are shown in **Table 73.2**.[15, 16]

In persistent or recurrent bacterial AOM, repeat culture of middle ear aspirates has failed to grow pathogenic bacteria in 30–50 percent of patients, implying that inflammation may persist despite the eradication of the infecting organism. The spectrum of organisms is similar to that in isolated episodes. In the 1980s *H. influenzae* was the most common organism identified in persistent or recurrent AOM, but this has been replaced by drug-resistant *Pneumococcus*. After antibiotic treatment for recurrent AOM it is now estimated that 50 percent of *H. influenzae* are beta-lactamase producing. A similar proportion of pneumococci are penicillin resistant.[16] Penicillin resistance in pneumococci results from decreased penicillin binding protein on the bacterial cell walls, so reducing the affinity for penicillin-related drugs, but means that resistance may often be overcome by increasing drug dosage. This is not the case with beta-lactamase producing organisms. Most *Moraxella catarrhalis* are now beta-lactamase producing.

Studies on HIV-positive children suggest a similar spectrum and prevalence of infecting organisms as occurs in immunocompetent children, except where the child is severely immunosuppressed, when a higher percentage of *Staphylococcus aureus* has been reported. [**]

Table 73.2 Bacteria associated with AOM.

Bacteria	Incidence (%)
Haemophilus influenzae	16–37[a]
Moraxella catarrhalis	11–23
Streptococcus pyogenes	Up to 13
Staphylococcus aureus	Up to 5

[a]There are some 90 serotypes.

Routes of spread of infection

Three potential routes are described: the Eustachian tube, tympanic membrane perforations or grommets, and haematogenous.

The Eustachian tube is traditionally assumed to be the main route by which organisms reach the middle ear, though there are relatively few studies to confirm this. It is speculated that negative middle ear pressure may facilitate the movement of bacteria up the Eustachian tube.[17] Circumstantial evidence is also gained from similarities in organisms cultured from the post-nasal space and the middle ear cleft in AOM. Whether anatomical or physiological differences predispose to AOM is unclear. Studies of Native Americans, who are prone to otitis media, suggest their Eustachian tubes are shorter, straighter and more patulous than in whites, but also that they have a low passive tubal resistance.[18] Research has found no difference in tubal dimensions in otitis prone and non-prone children. However, altered tubal function may play a role. Specifically, otitis-prone children have been shown to have significantly poorer active tubal function (muscular opening function).

Pathogen entry through tympanic membrane perforations or ventilation tubes is most commonly associated with water exposure.

Haematogenous spread is inferred from the evidence provided by studies of viral identification in the blood and middle ear as described previously. It was shown that the wide variation in rates of identification of specific viral strains from the middle ear could not be explained by passive Eustachian tube transport alone.[12] [**/*]

Risk factors

GENETIC FACTORS

There is growing evidence that recurrent AOM is largely genetically determined. It is likely many genes are involved. There are numerous studies suggesting a familial association. A metanalysis of risk factors has shown that when one family member had AOM the risk increased for other family members (relative risk 2.63).[19] Racial differences are well described with increases in American Indians, Eskimos and Australian Aboriginals. However, environmental factors, such as poor economic status, may contribute to the increased risks in these groups. The most powerful evidence comes from twin studies, in particular comparison of monozygotic and dizygotic twins in whom the occurrence of AOM was compared.[18] Many immune related mechanisms, which are likely to have a genetic basis, have been proposed. Certain human leukocyte antigen (HLA) classes have been shown to be significantly associated with increased risk of AOM. Maternal blood group A is reported to an independent risk factor (relative risk 2.82). Atopy has also been associated with increased risk of developing AOM. [****/***/**]

IMMUNE FACTORS

Our understanding of the immune response to AOM remains incomplete. However, a number of specific associations have been identified which suggest that certain defective or immature pathways may predispose to infection. Low levels of IgG2 subclasses have been reported in several studies to be more common in otitis-prone children. Those with IgG2 deficiency were shown to be three times more likely to develop post-ventilation tube insertion otorrhoea for example. Delayed maturation of anti-pneumococcal antibodies (IgG1 and IgG2 were studied) does appear to predispose to AOM. This may explain in part why children grow out of AOM as immunity matures.

Defective complement-dependent opsonization has been associated with recurrent AOM and diarrhoea in infancy.[18] This is caused in some examples by low concentrations of mannose-binding protein which acts as an opsonin. This appears to be a common defect with over 20 percent of children with recurrent AOM affected in some studies. This may be particularly important in infancy when the antibody repertoire is limited.

Aberrant expression of critical cytokines, such as tumour necrosis factor and interleukins, resulting in suboptimal host defence, has been postulated as a cause for persistent infection. Expression of mucin genes, at least nine of which have been identified, may differ in those predisposed to AOM. Middle ear mucosa expresses specifically the MUC5B gene. Mucin genes regulate the production of mucin. Limited evidence is beginning to emerge that over-expression may alter the mucociliary transport system.[18]

A number of studies on children with HIV infection have yielded conflicting results. Advanced disease associated with low CD4 counts does seem to be associated with an increased incidence of AOM. [**/*]

ENVIRONMENTAL FACTORS

There are many reports on the relative contribution of environmental factors. These are important as it may be possible to modify them. The most important is almost invariably stated to be day-care attendance outside the home. The larger the number of children in the group, the greater the risk. Day care outside the home carries a relative risk (rr) for AOM of 2.45, compared to a risk of 1.59 for children cared for in their own home. The incidence of AOM appears to follow that of seasonal upper respiratory tract infections (URTI) in the winter months. Breastfeeding for three months is protective (rr, 0.87). Use of a pacifier (dummy) carries a relative risk of 1.45.[19] Poor socioeconomic status associated with poor housing and overcrowding has been reported to be associated with AOM (overcrowding: rr, 5.55 in a Greenlandic population, for example). Passive smoke exposure from parental smoking is weakly associated (rr, 1.0–1.6). There is more limited evidence to support the role of dietary factors, in particular cow's milk allergy, in predisposing to AOM. [****]

SYNDROMIC ASSOCIATIONS

Syndromes associated with abnormalities of skull base anatomy are well recognized as being associated with chronic middle ear disease, but less is published on associations with AOM. Children with Turner's syndrome do suffer more frequent episodes of AOM. Down syndrome predisposes to middle ear disease, including AOM. In cleft palate there is only a minor increase reported. Certainly Eustachian tube dysfunction in these groups predisposes to middle ear effusion, but it is not clear whether it is this dysfunction or an increase in risk secondary to subtle immunological factors that predisposed to infection. No increase is found in children with primary ciliary dyskinesia if grommets are not inserted, or cystic fibrosis.

A direct association between iron deficiency anaemia, and the degree of anaemia and frequency of AOM has been reported. [**]

EPIDEMIOLOGY

AOM is one of the commonest illnesses of childhood. It accounts for approximately 25 percent of all prescriptions for children under ten years of age in the USA, for example. Its incidence appears highest in the first year of life, more specifically the second six months of life in most studies, and gradually reduces with increasing age. This progression was shown by Strangerup and Tos[20] who reported an incidence of a first episode of AOM in 22 percent in the first year of life, 15 percent in year two and 10 percent in year three, falling to 2 percent by year eight (**Figure 73.2**). Epidemiological studies have been compromised by difficulty in achieving accuracy in diagnosis when large numbers of children are being assessed, hence there are wide variations in reported numbers. Incidences of over 60 percent are stated in some reports of infants up to age one year. By age three years, some 50–70 percent of all children will have had at least one episode of AOM, and at least 75 percent by the age of nine years.

The incidence of AOM certainly varies with the seasonal incidence of viral upper respiratory infections. There are reports that it is increasing over a period of years. Possible reasons include increased day nursery attendance and changes in diagnostic awareness.

Recurrent AOM has been reported in 5 percent of children under two years of age. Others have reported that by age three, half of children will have had at least three episodes. An important indicator of future problems is a first episode before nine months of age: these children have a one in four risk of developing recurrent AOM.

In the first two years of life, AOM occurs bilaterally in 80 percent of cases. After six years of age, it is unilateral in 86 percent.[20]

MANAGEMENT OPTIONS

Most children with AOM will get better quickly and without treatment. Some will not. A very small number may develop potentially serious complications. Current debate questions whether and for whom treatment is required, and the role of prophylactic strategies. As serious complications are rare it can be difficult to obtain high quality evidence of how effective are prophylaxis and the treatment of episodes of AOM in preventing such complications.

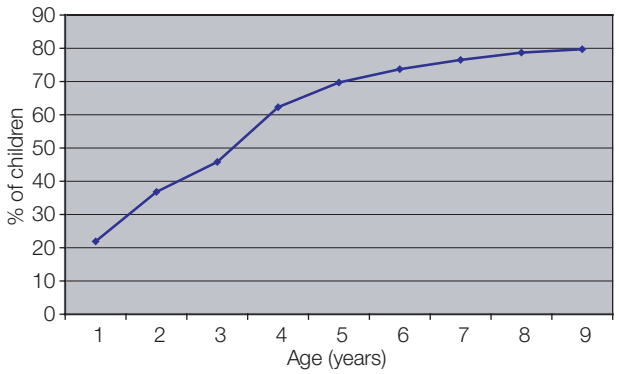

Figure 73.2 Cumulative incidence of acute otitis media (i.e. how many children have had at least one episode in their life). Redrawn from Ref. 20, with permission.

Management of acute episodes

CONSERVATIVE TREATMENT

Most children will benefit from simple analgesics and anti-pyrexials, in a quiet supportive environment. Paracetamol and ibuprofen are most commonly used in the UK. There is limited experimental animal evidence showing that ibuprofen provides additional benefit by reducing mucosal inflammation when taken in combination with amoxicillin.

MEDICAL TREATMENT

Antibiotics

Uncertainty over the use of antibiotics is reflected in wide variations in usage between countries, ranging from 31 percent in the Netherlands to 98 percent in the USA.[9] There is, however, an increasingly good evidence-base for the most appropriate management in children over two years of age. In children under two, the evidence-base is weaker.

Antibiotics, if not prescribed initially, should be given to a child who fails to improve after two to three days of 'watchful waiting', and to all children with an 'irregular' illness course. They should also be given to 'high risk' children, defined by the Dutch as children with craniofacial abnormalities, Down syndrome, immunodeficiencies and those under the age of two years suffering a recurrent episode of AOM.[2] Guidelines must be adapted to suit local experience, for example in regions of the world with a high incidence of complications of AOM where all patients may be regarded as high risk.

A recent metanalysis[9] has addressed the question of whether antibiotics should be given at initial consultation. Two-thirds of children recovered within 24 hours of the start of treatment, and 80 percent by days two to seven, with or without antibiotics. This, of course, raises the question as to whether the children did indeed have AOM in the first place. Antibiotics did lead to 5 percent fewer children overall having pain between days two and seven. That equates to 17 children needing to be treated to prevent one child experiencing pain during days two to seven. Relatively few data were available on hearing loss at one and two months post-infection, but no differences were found between those who received antibiotics and those who did not. No significant differences were found

on progression of disease or relapse of symptoms. Similarly, no differences were found in complications of AOM. However, those taking antibiotics suffer nearly double the side effects, such as diarrhoea, than those who do not, and run a greater risk of developing antibiotic resistant bacteria.

The length of treatment was addressed in a separate metanalysis.[21] Short (five day) and long (ten day) courses of treatment were compared. At 8–19 days the weighted mean failure rate in the short course was 19 percent and 13.7 percent in the long course. By days 20–30, this had converged to 15.7 versus 12.5 percent, which was nonsignificant. It implied that 17 children would need to receive the long course to avoid one treatment failure. The authors concluded that five days of treatment was appropriate in uncomplicated infections in low risk children over two years of age without recurrent AOM or tympanic membrane perforation. Under two years of age evidence is weaker that short course treatment is adequate.

Attempts have been made to identify a subgroup of children who may benefit from antibiotics. Younger age may be an important determinant, but good evidence is lacking because of diagnostic difficulties in this group. However, while published data show only modest benefit from antibiotic treatment between six and 24 months of age[22] (number needed to treat (NNT) = seven for one symptomatic improvement at day four), most would recommend treatment below two years of age. Using short-term symptomatic outcome markers at day three, it has been shown that immediate antibiotics may benefit those children presenting with higher temperatures (>37.5°C) or vomiting (NNT = 3–6).[4]

AOM occurring in the presence of a tympanic membrane perforation or ventilation tubes may be treated equally successfully with oral or topical antibiotics. The potential ototoxicity of topical aminoglycoside ear drops in these cases is well recognized and dose dependent, therefore prolonged topical treatment should be avoided. [****]

Which antibiotic?

This should be determined by national recommendations. Amoxicillin remains the first choice in most centres, but at higher than previously recommended doses (80 mg/kg/day) if drug-resistant pneumococci are common in a particular country or region, or macrolides for penicillin-sensitive patients. For persistent or resistant episodes, national policies should be sought depending on the prevalence of beta-lactamase-producing organisms and culture results if available. Options include amoxicillin-clavulonate or cefuroxime axetil orally, or intra-muscular ceftriaxone (US Centre for Disease Control and Prevention).

Antihistamines and decongestants

A metanalysis of the use of oral or intranasal antihistamines and/or decongestants concluded that their use could not be supported, and that medication side effects were higher when they were used together. While combining the two treatments was shown to slightly reduce persistent AOM at two weeks (NNT = 10.5) the result may have been biased by the design of the studies.[23] [****]

SURGICAL TREATMENT

Surgery has a limited role in the treatment of an uncomplicated episode of AOM. Myringotomy was practised in the pre-antibiotic era, and indeed was continued until the late 1980s in some countries as a first-line treatment for AOM. However, there are now a number of good studies showing that myringotomy plus antibiotics offers no advantage over antibiotics alone. Myringotomy alone has a worse outcome than either of the antibiotic groups.[24] Myringotomy is reserved for severe cases where complication is present or suspected, to relieve severe pain or when microbiology is strongly required. [****]

Management of recurrent acute otitis media

ALTERATION OF RISK FACTORS

It may be possible to alter many of the environmental risk factors discussed previously. Parents should be reassured of the benign natural history of AOM, as these children have been shown to be more demanding than those without recurrent disease, and their mothers more anxious about their care. The most readily modifiable risk factor is exposure to other children. AOM increases with the number of children in day care, the length of time a child spends in day care each week, how young a child is when introduced into day care, the presence of children under two years of age in the day-care setting and having a sibling in day care. Advice should include sitting a child semi-upright if bottle-fed and avoiding passive smoke inhalation. Restricting the use of pacifiers, particularly after infancy, should be recommended for otitis-prone children. The mother may be advised to continue breastfeeding for at least six months after future pregnancies and increasing vitamin C intake and avoiding alcohol in the third trimester, both of which have been weakly associated with AOM. The role of food allergies, in particular cow's milk, is still unclear. No effective role has as yet been shown for homeopathic remedies. [**]

MEDICAL PROPHYLAXIS

Antibiotics

Antibiotic prophylaxis has the potential to cause problems, but should be considered for recurrent AOM. Many organisms need to be covered so a broad-spectrum drug is required. Risks for the development of resistant

organisms increase, adverse drug reactions may occur and active disease may be masked. Studies of prophylaxis of recurrent AOM invariably treat each individual recurrence with additional antibiotics. Trials therefore compare antibiotic prophylaxis versus placebo between acute episodes. The natural history of recurrent AOM is reassuring. Over 50 percent of children having no treatment between attacks will not suffer a further episode in the following six months. Indeed, only one in eight continues to suffer recurrent AOM (i.e. three or more episodes) during the trials, if treated only for acute episodes. However, metanalysis does show a benefit of antibiotic prophylaxis equating to a reduction of approximately 1.5 episodes per 12 months of antibiotic treatment given, above that expected from the natural history. So one child would need eight months of treatment to avoid one episode of AOM. There was a trend for those treated with sulfisoxazole to do better than those treated with amoxicillin, both being used at half the therapeutic dosage.[25] Recommendations are for six months of treatment through the winter months in children who do not have background OME.

Those studies that assessed the length of time a child has OME in association with AOM showed that while antibiotic prophylaxis may reduce the incidence of AOM, it does not reduce the length of time with OME. This is important as antibiotic prophylaxis may therefore be most appropriate for children not prone to OME, while ventilation tubes may be indicated for those prone to OME.

Most trials exclude 'high risk' children, who are often most in need of treatment. On the basis of the metanalysis described above, which shows a modest benefit with the use of antibiotic prophylaxis, there may also be a place for its use in the management of high-risk children with recurrent AOM, despite the absence of specifically targeted studies. [****]

Xylitol

Xylitol is a commonly used sweetener that inhibits pneumococcal growth and the attachment of pneumococci and *Haemophilus* to nasopharyngeal cells. Studies in daycare nurseries using chewing gum or syrup have suggested reductions of 30–40 percent in the occurrence of AOM. However, this translates to 1–1.5 episodes per year.[26] It is ineffective if used in acute upper respiratory infections. Given the very large quantities that must be consumed, and potential concerns over the safety of such consumption, its use cannot yet be recommended. [***]

Vaccination

Vaccines have been used effectively against most common childhood infections caused by single specific organisms such as mumps, measles and rubella. The concept of vaccinating against AOM therefore is an attractive one that is being actively explored. Potential obstacles include the wide range of causative organisms, both bacterial and viral, the varied serotypes, technical difficulties in producing an effective immune response, obtaining an immune response before six months of age, parental resistance to multiple vaccination and the possibility that the successfully targeted pathogens will simply be replaced by others.

Vaccination against viruses

Since 60–90 percent of episodes are initially associated with viral infections (see above under Viruses) viral vaccination seems the most logical first step. AOM secondary to infection by the measles virus is now relatively uncommon in industrialized countries, for example.

Influenza A vaccination is currently the only commercially available preparation for the prophylaxis of viral upper respiratory infections. Three trials of children in daycare have shown its efficacy in preventing AOM, resulting in 30–36 percent fewer episodes during a subsequent influenza epidemic, and reducing influenza-associated AOM by 83–93 percent.[27] This is not an absolute reduction in episodes, and this indication is not yet within the UK Department of Health guidelines.[28] This does show, however, that preventing viral URTI can be an effective method of reducing AOM. [***]

RSV vaccines are undergoing clinical trials for lower respiratory tract infection, but as yet do not seem to be providing significant protection. No studies are under way assessing a role in AOM.

Parainfluenza virus vaccines have been evaluated in animals and need to target types 1, 2 and 3 viruses. Limited human studies demonstrate relative safety and immunogenicity, but efficacy studies are not available.[29]

Vaccination against bacteria

Vaccination against *Streptococcus pneumoniae*, nontypeable *Haemophilus influenzae* and *Moraxella catarrhalis* is made difficult by the low immunogenicity of the polysaccharide capsule of these bacteria in young children and infants. Success against *Haemophilus influenzae* type B (which causes epiglottitis and meningitis) using a polysaccharide-protein conjugated vaccine provides one potential solution.

Streptococcus pneumoniae vaccination is particularly challenging because of the 90 serotypes of the bacteria. However, as only a small number of these cause most pneumococcal AOM, and it has been shown that anti-capsular antibodies can prevent pneumococcal AOM, progress has been made. Early attempts with unconjugated pneumococcal polysaccharide vaccines proved unsuccessful in children under two years of age. However, a heptavalent conjugated vaccine (Prevenar, Wyeth-Lederle Vaccines) has been shown to be highly effective in preventing invasive pneumococcal disease and modestly successful in reducing AOM in two major studies. Immunization occurs at two, four, six and 12, and in one study also 15 months of age. Episodes of AOM from any cause were reduced by 6 and 7 percent, respectively, and pneumococcal AOM by 34 percent.[29] To improve cover

during the critical first six months of life, trials are under way to see if immunizing the mother in the third trimester of pregnancy is effective. Immunization after two years of age is with 23-valent pneumococcal polysaccharide vaccine. Whilst vaccination is recommended in certain 'at risk' children,[28] its place in the management of AOM is not yet clear. It may be, for example, that vaccinating a child presenting with recurrent AOM will be ineffective because colonization of the upper respiratory tract has already occurred. Publications on this are pending. Debate on the quality of systematic reviews on this topic continues and clear guidance cannot yet be given.[30] [***]

Non-typeable *Haemophilus influenzae* vaccines are being developed by several techniques.[27] Phase I clinical trials using a conjugated vaccine are under way. Animal experiments using a chinchilla model have shown some reduction in AOM with this type of vaccine.

Moraxella catarrhalis vaccine research is at a preclinical stage, but products are under development.

Experts in the field hope vaccination against all three of these bacteria may be possible within a decade.[29]

Special attention should be drawn to children with, or awaiting, cochlear implants. Concern has been raised about a number of cases of meningitis. Whether it is implant related or reflects inner ear abnormalities in many of these children is unclear. All such children are recommended to have the heptavalent pneumococcal vaccine before the age of two years and the 23-valent pneumococcal polysaccharide vaccine at the age of two or over (see Chapter 70, Paediatric cochlear implantation). Hib conjugate vaccine is recommended for all children up to four years of age.

Immunoglobulins

The importance of immunological immaturity in the occurrence of recurrent AOM has been emphasized. Intramuscular pooled gamma-globulin in otitis-prone children has been shown not to reduce the incidence of AOM. However, in a Japanese study, intravenous immunoglobulin (GB-0998) in IgG2-deficient infants has been shown to be an effective prophylaxis for AOM, as well as pneumonia in this specific group of children. [**]

Benign commensals

A recent paper considered whether spraying benign commensals (alpha streptococci) into the nose to recolonize the nasopharynx following antibiotics might reduce AOM by inhibiting the growth of pathogenic bacteria. A significant reduction was reported.[31] A separate smaller study, which did not pretreat with antibiotics, showed no difference. [***]

SURGICAL PROPHYLAXIS

In contrast to the large number of trials comparing antibiotic treatments, there are relatively few addressing surgical prophylaxis. Surgery is potentially attractive, however, in that it may reduce problems of antibiotic resistance and also treat subsequent OME.

Ventilation tubes

A recent metanalysis of five trials concluded that the presence of ventilation tubes versus no tubes yielded a relative decrease in episodes of AOM of 56 percent, equivalent to an absolute reduction of 1.0 episode per child per year.[32] The effect occurred mostly in the first year of follow up, presumably as this covered the period when the tubes were in place. Of equal importance is the reduction in the prevalence of OME by 115 days per child-year. Seventy-nine percent were reported to have an improved quality of life. Side effects included recurrent otorrhoea in 7 percent and chronic otorrhoea in 4 percent. Other studies have shown a higher incidence of tympanosclerosis and focal areas of tympanic membrane atrophy of questionable significance in the ventilation tube groups.

These findings need careful interpretation. One of the studies compares antibiotic prophylaxis with amoxicillin to tubes to placebo.[33] The amoxicillin group had a significant reduction in episodes of AOM. The tube and placebo group did not. However, when AOM occurred in the placebo group, it was more distressing than when otorrhoea occurred with AOM in the group with tubes in place. Also, over a two-year period, the surgical group had 26 and 61 days fewer with OME than the antibiotic and placebo groups, respectively.

It is difficult to draw conclusions about the role of ventilation tubes. On the evidence available they may be considered for children with recurrent AOM, but no persistent effusion, in whom medical strategies have failed. There may be a greater role for them in preference to, or following failure of, medical prophylaxis in the child with recurrent AOM and persistent OME. [****]

Adenoidectomy and adenotonsillectomy

The limited evidence-base for best practice is most striking when considering adenoidectomy. Two papers are particularly worth discussing, both with a cohort of children from Pittsburgh. Randomization methods have been questioned in these studies, as has follow up. The first concluded that adenoidectomy may be beneficial in children who had previously had ventilation tube insertion and suffered subsequent AOM. AOM was reduced by 31 percent relative to the control group in a two-year follow up (or 0.32 episodes per child-year), and subjects spent 42 percent less time with OME. Additionally, the need for further tubes was reduced by 50 percent.[34]

Their second trial was of children who had not previously had ventilation tube insertion. Considering children without overt adenotonsillar disease, a modest reduction in the number of episodes of AOM was recorded in the first year after surgery from 2.1 to 1.4

following adenotonsillectomy, but not adenoidectomy. Similarly, OME was reduced from 30 to 19 days in year one in the adenotonsillectomy group, and to 22 days in the adenoidectomy group. The effect was not apparent after the first year. Drop out from the trial was particularly high in the adenoidectomy group, and the results should be viewed cautiously. For children with adenotonsillar symptoms, no AOM benefit was reported from adenotonsillectomy. The authors concluded the risks of surgery were not warranted in children who had not previously had ventilation tube insertion.

In summary, there is little evidence to support adenotonsillectomy. Adenoidectomy may be considered in those children who have failed medical therapy and had further AOM following ventilation tube insertion. The presence of OME increases the benefit of adenoidectomy. [***]

OUTCOMES

An episode of acute otitis media may resolve rapidly with or without antibiotics; it may prove resistant to first-line antibiotics; it may persist or recur shortly after a course of antibiotics has finished; it may subsequently recur; or it may progress to tympanic membrane perforation or other complication of infection. Here we consider the medium- and long-term consequences of infection: the natural history of AOM, middle ear effusions, auditory functioning and speech and language development.

Natural history

Data come from the control arms of randomized controlled trials, and hence usually exclude high-risk children, complicated cases, and those under two years old. Without antibiotic treatment, symptomatic relief from pain and fever occurs in approximately 60 percent of children within 24 hours of diagnosis, in over 80 percent by day two to three, and 88 percent by days four to seven.[9, 25] These data do not equate with complete resolution, for example otorrhoea may still be present without pain or fever, and only 73 percent reach the stage of complete resolution by day seven to fourteen. In all studies, those with resistant or persistent disease will have received antibiotic treatment.

For recurrent AOM the prognosis is also generally favourable. Following study entry, and with only acute episodes treated, recurrence rates fell to 0.13 episodes per child per month in the subsequent 6–24 months – approximately 1.6 episodes per year. Indeed, over half had no further attack in the following six months, and only one in eight continued to satisfy the diagnostic criteria for recurrent AOM.[25] Other work has shown that even in early recurrences of infection three to four weeks after a previous episode, a new organism is usually involved. Caution should be attached to these findings. Though pooled numbers are large, high risk children and those with baseline OME were generally excluded.

MIDDLE EAR EFFUSIONS

Middle ear effusions are an important outcome of AOM. Looking again at those children randomized to placebo, pooled data show rates of OME of 63 percent two weeks after AOM, 40 percent at one month and 26 percent at three months. Antibiotics did not appear to have any effect.[9, 25]

AUDITORY FUNCTIONING

What little work has been carried out on short-term audiometric outcomes suggests that approximately one in three children will have an air–bone gap greater than 20 dB at one month after infection, and one in five at three months. There is limited evidence to suggest that AOM may reduce long-term audiometric thresholds. Several studies following cohorts of children have reported small but significant loss of very high frequency hearing (11–16 kHz) in those with many episodes of AOM. There is a suggestion that this may be more a consequence of disturbed middle ear mechanics than cochlear damage. The significance for auditory functioning as the child grows older is not established.

SPEECH AND LANGUAGE DEVELOPMENT

It is difficult to separate the literature on AOM and OME outcomes. Little is written on speech production or reception. In children with OME, a significant effect seems to occur in the early years of life on expressive language development, but not receptive language. A small number of studies point to persisting effects on expressive language in school-age children. There is little evidence showing different cognitive development in school-age children with a history of otitis media in the first three years of life. There are suggestions that poor behavioural traits may be more common by school age, but more work is required before conclusions are drawn.

COMPLICATIONS

Extracranial

TYMPANIC MEMBRANE

Tympanic membrane perforation is considered a complication of AOM. It is the commonest complication of infection and is reported in 0–10 percent of episodes. Perforation is associated with a purulent or bloody otorrhoea and immediate relief of pain. It typically occurs

in the posterior half of the pars tensa, and is associated with loss of the fibrous middle layer of the drum. This may predispose to future posterior retraction pockets. Four outcomes of perforation may result. In most cases the perforation heals spontaneously and the infection resolves. Second, the infection may resolve, but the perforation persists. This may predispose the ear to future AOM or chronic suppurative otitis media. Third, the perforation and otorrhoea may persist, manifesting as chronic suppurative otitis media. 'Chronicity' is generally deemed to have occurred by three months. Fourth, a further complication may arise.

The long-term outcomes were assessed in a cohort of otitis-prone children followed up from 3 to 14 years of age. By the end of the study 7 percent had collapse of the posterior superior tympanic membrane, chronic suppurative otitis media, or central perforation.[35] Scarring or tympanosclerosis was present in 27 percent, although several studies report that ventilation tubes increase this risk.

ACUTE MASTOIDITIS

Four classes of mastoiditis are defined. During episodes of acute otitis media, infection and inflammation may naturally extend into the mastoid cavity, and be visualized radiologically. This is not associated with the typical signs of acute mastoiditis and is not considered a complication of AOM.

Infection may spread to the mastoid periosteum by emissary veins: acute mastoiditis with periosteitis. At this stage no abscess is present but the post-auricular crease may be full, the pinna may be pushed forward and there may be mild swelling, erythema and tenderness of the post-aural region.

When acute mastoid osteitis develops, the infection has begun to destroy the bone of the mastoid air cells and a subperiosteal abscess may develop. Signs may be similar to those when periosteitis is present. A subperiosteal abscess develops most commonly in the post-auricular region. A zygomatic abscess may develop above and in front of the pinna. A Bezold's abscess may result from perforation of the medial mastoid cortex, tracking down the sternomastoid to the posterior triangle. Pus tracking down peritubal cells may result in a retropharyngeal or parapharyngeal abscess (**Figure 73.3**).

A fourth stage may be reached, subacute ('masked') mastoiditis, in incompletely treated AOM after 10–14 days of infection. Signs may be absent, but otalgia and fever persist. This stage can also progress to serious complications.[3]

In the pre-antibiotic era, mastoiditis was a common and serious complication of AOM. In a study in 1954 the control group was reported to have developed mastoiditis in 17 percent of cases.[9] In some developing countries rates of 5 percent are still quoted. In the 1970s it was estimated that 0.004 percent of cases of AOM resulted in surgery for

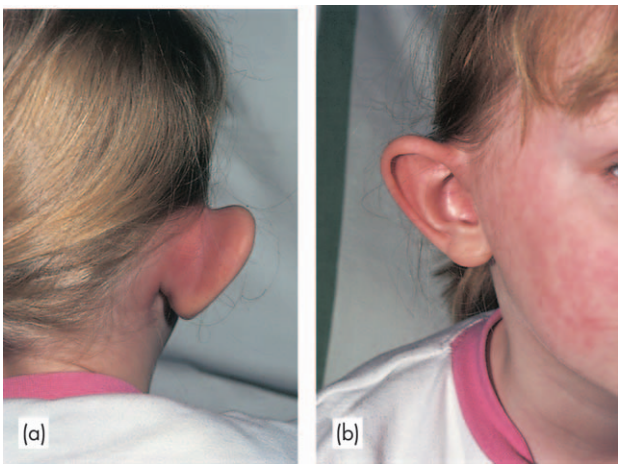

Figure 73.3 (a) and (b) Acute mastoiditis.

mastoiditis. The incidence is reported by several authors to be increasing gradually again. The incidence varies between countries. In the UK, Canada, Australia and the United States, where antibiotic prescription rates are over 96 percent, the incidence ranges from 1.2 to 2.0 per 100,000 population per year. In Norway, Denmark and The Netherlands (prescription rates 67, 76 and 31 percent, respectively) rates are higher at 3.5, 4.2 and 3.8 respectively.[36]

Acute mastoiditis is a disease of childhood. A large multicentre study found 28 percent to be in children less than one year of age, 38 percent in one to four year olds, 21 percent in four to eight year olds, 8 percent in 8–18 year olds and 4 percent in those over 18 years of age.[37] This higher incidence in younger children reflects the peak ages for AOM.

Traditional teaching was that acute mastoiditis is preceded by 10–14 days of middle ear symptoms. However, in many papers the short length of middle ear symptoms prior to presentation is noteworthy. For example, in one large study approximately 32 percent had one to two days symptoms, 34 percent had three to six days, 26 percent seven to fourteen days and 8 percent over 14 days.[37] Prior antibiotic treatment of the infection is common, reported in 22–55 percent of children. Clearly antibiotics do not fully protect against mastoiditis.

Symptoms are of otalgia and irritability in most children. Pyrexia is less common in those treated with antibiotics. Otorrhoea is present in only approximately 30 percent. Clinically, a red or bulging tympanic membrane will often be seen. A normal drum is reported in a very variable proportion of cases, but certainly does not exclude the diagnosis and is believed to result from resolution of the mesotympanic infection following antibiotic treatment, while the osteitis in the mastoid progresses. Retro-auricular swelling is seen in approximately 80 percent and retro-auricular erythema in 50–84 percent (less in previously treated children). Tenderness is typically sited over MacEwen's triangle (on palpation

through the conchal bowl). Pinna protrusion is present in two-thirds of cases. Sagging of the posterior wall of the external auditory canal, resulting from subperiosteal abscess formation, should be looked for, but is quoted as an uncommon finding. Few patients will have a 'full house' of the classic signs.

A somewhat different incidence of organisms has been identified from those gained from culture in AOM. Around 20 percent of samples do not grow bacteria. *Streptococcus pneumoniae, Streptococcus pyogenes, Pseudomonas aeruginosa* and *Staphylococcus aureus* are the most commonly reported. *Haemophilus influenzae* is less commonly reported, and *Moraxella catarrhalis*, *Proteus mirabilis* and Gram-negative anaerobes rarely.

Recommended investigations vary between institutions. A full blood count, C-reactive protein (CRP), and blood cultures are often obtained. A CT scan of the mastoid is recommended when intracranial complications are present or suspected (though MRI may be more helpful in identifying specific intracranial pathology), when mastoidectomy is to be performed and in those not improving on antibiotic treatment. A CT may show evidence of osteitis, abscesses and intracranial complications.

Differential diagnosis includes AOM, otitis externa, furunculosis and reactive lymphadenopathy. Rarely, undiagnosed cholesteatoma, Wegener's granulomatosis, leukaemia and histiocytosis may first present with AOM, hence tissue should be sent for histology if mastoidectomy is performed.

Myringotomy with or without ventilation tube placement, culture of the aspirate and high-dose intravenous antibiotics is the most commonly recommended initial treatment in acute mastoiditis. This is adequate in 75 percent of cases. Failure to improve, subperiosteal abscess formation (occurring in 10–30 percent) or development of complications merits at least abscess drainage with or without cortical mastoidectomy. This can be challenging surgery for the less experienced as the mastoid is often full of granulations and the facial nerve superficial in the young child.

A most important message is that intracranial complications from acute mastoiditis develop in 6–17 percent of cases, and many of these may develop during hospitalization. Although acute mastoiditis may be less common than in the past, its severe complications still occur. [**]

PETROSITIS

Infection may extend to the petrous apex. The classic features of Gradenigo's triad (VI nerve palsy, severe pain in the trigeminal nerve distribution and middle ear infection) are not always present. Patients commonly present with other intracranial complications. Recent papers recommend high-dose broad spectrum antibiotics and a variety of mastoidectomy, from cortical to radical, though drainage of the petrous apex is no longer felt necessary. [*]

FACIAL NERVE PALSY

In the pre-antibiotic era, it was estimated that acute lower motor neurone facial palsy complicated 0.5 percent of episodes of AOM (see Chapter 80, Facial paralysis in childhood). It is now quoted at 0.005 percent.[38] Most are related to bacterial infection but case reports with viral AOM exist. Approximately four out of five children present with a partial paralysis. The case series in the literature report that approximately 80 percent of palsies respond well to ventilation tube insertion and intravenous antibiotics. The remainder undergo cortical mastoidectomy. Advice is conflicting about when and in whom mastoidectomy is required and the role of facial nerve decompression. As recovery is generally so good, a more conservative approach without facial nerve decompression seems appropriate. Most children achieve rapid restoration of normal facial function, with a mean time to complete recovery of four months. Those with a total paralysis at presentation have a recovery stretching over many months.

Sixth nerve palsy in the absence of petrositis has also been reported. It is speculated this may stem from phlebitis spreading along the inferior petrosal sinus from the lateral sinus. [*]

LABYRINTHITIS

Round window permeability changes during acute infection are important as these may allow entry of bacterial toxins. There is some experimental evidence that permeability can be increased by streptococcal toxins. Preformed channels for bacterial entry may also exist, such as surgical or congenital perilymph fistulae. These may allow infection to spread directly to the subarachnoid space causing meningitis. Particular concern arises in children with congenital inner ear abnormalities and those with cochlear implants. Three types of labyrinthitis are recognized. *Perilabyrinthitis* is not associated with AOM. *Serous labyrinthitis* is inflammation of the labyrinth without pus formation, and is characterized by recovery of auditory and vestibular function. *Suppurative labyrinthitis* may result from spread of infection from the mastoid or middle ear. Severe vertigo, nausea, vomiting, nystagmus and permanent hearing loss result. Suppurative labyrinthitis is rare, and the treatment of cases presented in the literature ranges from ventilation tube insertion and aggressive antibiotic use, to tympanomastoidectomy and cochleotomy. [*]

Intracranial

In the pre-antibiotic era, intracranial complications of AOM were more common and mortality rates of over 75 percent are presented. Published mortality rates from intracranial complications now average approximately 5 percent in industrialized countries.

Persistent headache and fever are the most common early symptoms of an intracranial complication. In half of cases there may be signs only of AOM and not mastoiditis. Frequently two or more complications coexist. Early diagnosis is important for improving outcomes.

Seven classical intracranial suppurative complications of AOM are described.[3]

1. **Meningitis** is usually cited as the commonest intracranial complication of AOM, accounting for 54–91 percent of cases. In contrast, studies assessing the aetiology of meningitis are conflicting. One of the largest recent studies found no association between bacterial meningitis and AOM, while another found an antecedent history of AOM in 29 percent,[39] though this does not equate to a causal relationship. Special mention has already been made of possible associations between congenital inner ear malformations such as cochlear dysplasia, cochlear implants and meningitis. Younger children, average age two years, are most commonly infected. Studies focus almost exclusively on bacterial aetiologies. The rate of *Haemophilus influenzae* type B meningitis has dropped dramatically since vaccination was introduced. *Streptococcus pneumoniae* is the causal agent in a greater proportion because of this reduction.[39] A second intracranial complication should be looked for in any infant with meningitis with MR scanning. Myringotomy may help to establish the infective agent if evidence has not been obtained from lumbar puncture. Treatment is medical. If mastoid surgery is required, it is usual to try and wait for an improvement in the medical condition of the child first if possible. [*]
2. **Extradural abscess** is the next commonest intracranial complication. It is more commonly associated with chronic disease. Pus collects between dura and bone, usually after bone erosion. If lying in the posterior fossa medial to the sigmoid sinus, it is termed an extradural (epidural) abscess, if within the split of dura enclosing the sigmoid sinus it is called a peri-sinus abscess. It may be discovered only at mastoidectomy, but may be suspected in the patient with persistent headache and fever or severe otalgia. Treatment is surgical drainage. [*]
3. **Subdural empyema** is a collection of pus between the dura and arachnoid membranes and is termed a subdural empyema. It is rare. It develops by direct extension of infection or thrombophlebitis. In addition to headaches and pyrexia, focal neurological signs, seizures and signs of meningeal irritation may be present. Paranasal sinusitis is reported to be a much commoner cause than AOM. Surgical drainage of the abscess through burr holes or craniectomy may be indicated. Mastoidectomy may sometimes be required, though many cases cited in the literature were treated medically. [*]
4. **Sigmoid sinus thrombosis** most commonly results from erosion of the bone over the sinus from mastoiditis and may also be associated with other complications. However, it occurs in association with otitis media alone in 43 percent of cases. Infected thrombus develops within the sinus and may then extend proximally and distally to the internal jugular vein and superior vena cava, entering the systemic circulation and causing septicaemia. In addition to headache and otorrhoea, a spiking pyrexia may develop. Griesinger's sign is mastoid tenderness and oedema secondary to thrombophlebitis of the mastoid emissary vein. MRI is the imaging of choice showing high signal intensity in the sigmoid sinus on both T_1-and T_2-weighted images and absent flow. If caused only by otitis media, myringotomy and antibiotics may suffice. However, in the presence of mastoid infection, it is more usual to perform a canal wall up mastoidectomy, needle the sinus to assess blood flow and occasionally to remove infected thrombus. As persistent sepsis and distant thrombosis are uncommon, the role of anticoagulation is unclear in the literature.[40] Serial imaging to look for propagation of thrombus has been recommended. [**/*]
5. **Focal otitic encephalitis (cerebritis)**. Focal inflammation and oedema of brain tissue may occur independent of, or in association with, any suppurative complication of AOM. Intensive antibiotic treatment is required. [*]
6. **Brain abscess** is more commonly associated with chronic ear disease but may occur in association with AOM and its complications (**Figure 73.4**). Brain abscess forms a larger proportion of complications in developing countries. It may develop in both the temporal lobe and cerebellum. Persistent headaches are the commonest symptom. Initial symptoms may be of encephalitis, but these often settle as the abscess organizes over days or weeks. Eventually, signs of raised intracranial pressure, focal neurology and infection develop. Invstigations include CT imaging followed by lumbar puncture, if safe. In the early stages of cerebritis, neurosurgical drainage may be avoided but will be required if the abscesses are expanding. Brain abscesses carry a potentially high mortality rate, although in industrialized countries the few large

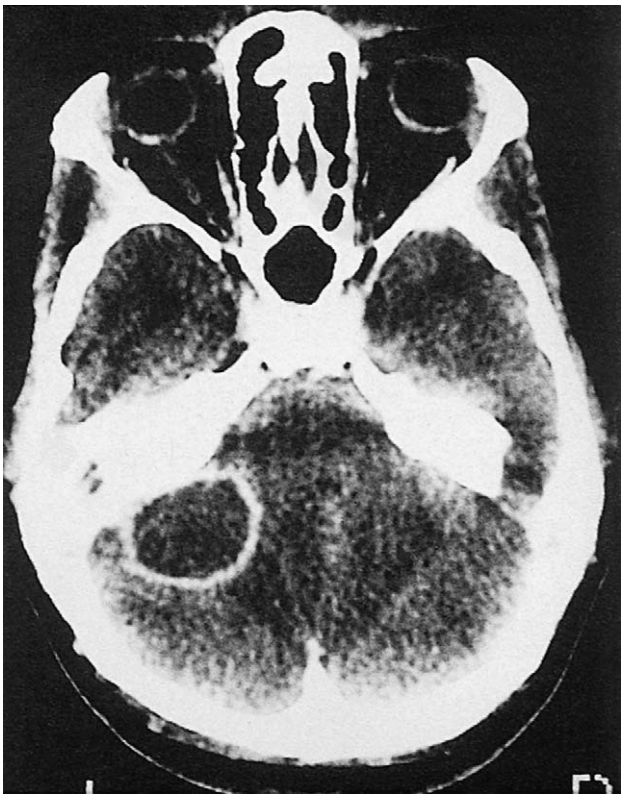

Figure 73.4 Brain abscess secondary to AOM.

series now quote rates of below 10 percent. One large review found the mortality from otogenic causes, at 3.8 percent, was much lower than from other causes. [*].

7. **Otitic hydrocephalus** is a complication of AOM manifesting as raised intracranial pressure in the absence of any space-occupying lesion, and without obstruction to the flow of cerebrospinal fluid (CSF). Benign intracranial hypertension is a synonym. The aetiology is obscure. Headache is the predominant symptom. It is commonly associated with sigmoid or transverse sinus thrombosis and so MRI is an important investigation. Lumbar puncture will show raised CSF pressure, but normal CSF composition. A number of medical treatments may be tried and liaison with a paediatric neurologist is recommended. [*].

CONCLUSION

It can be seen from this chapter that there are deficiencies in our current knowledge of both the diagnosis and aetiology of AOM, and uncertainties in management strategies. However, we have been able to describe a number of potentially exciting developments that have occurred in the past few years.

KEY POINTS

- Acute otitis media is one of the commonest illnesses of childhood.
- Diagnosis can be difficult particularly in very young children.
- Management recommendations vary widely between countries.
- A range of modifiable risk factors should be addressed.
- Evidence is emerging to support new prophylactic strategies.
- Intracranial complications are still seen despite prior antibiotic treatment.

Best clinical practice

- ✓ AOM is one of the commonest illnesses of childhood. Accurate diagnosis is notoriously difficult. A high index of suspicion is required in the unwell child. [Grade B]
- ✓ The clinician should distinguish between sporadic, resistant, persistent or recurrent AOM as management strategies differ. [Grade A]
- ✓ Two-thirds of children recover within 24 hours with or without treatment, so a period of watchful waiting may be reasonable in uncomplicated AOM. [Grade A]
- ✓ Antibiotics should not be withheld in severe or irregular infections, should be given if a child fails to improve within two to three days of the onset of AOM and should be given in sufficiently high doses. [Grade A]
- ✓ Pyrexia (>37.5°C), severe otalgia, vomiting, age under two years and 'high risk' children have all been used as indicators to use antibiotics sooner rather than later. [Grade B/C]
- ✓ Practitioners should be aware of local bacterial antibiotic resistance patterns and prescribing policies. Broad spectrum antibiotics are not generally required as first-line therapy. [Grade A]
- ✓ In otherwise healthy children over two years of age, five days of antibiotics is usually adequate. [Grade A]
- ✓ For persistent or resistant AOMs it should be noted that whilst pneumococcal drug resistance can usually be overcome by increased antibiotic doses, *Haemophilus* may be beta-lactam producing, so broader spectrum antibiotics may be required. [Grade B]
- ✓ Modifiable risk factors should be discussed with parents. These include nursery attendance, parental smoking, breastfeeding and the use of pacifiers. [Grade C]

- ✓ In the management of recurrent AOM, on average eight months of antibiotic prophylaxis would be needed to prevent one episode of AOM. This strategy may be preferred in the absence of effusions between episodes of AOM. [Grade A]
- ✓ Ventilation tube insertion reduces the number of episodes of AOM by over 50 percent. This option may be preferred when effusions persist between episodes of AOM. [Grade A]
- ✓ Additional adenoidectomy may further reduce the number of episodes of AOM. [Grade A]
- ✓ The benefits of tonsillectomy on episodes of AOM are not sufficient to warrant its use in the management of recurrent AOM. [Grade A]
- ✓ Vigilance should be maintained for complications of AOM, the most common symptoms being persistent pyrexia and headache. [Grade C].

Deficiencies in current knowledge and areas for future research

The following list summarizes what we feel this chapter should be able to report on when the next edition is being prepared.

- ➤ Greater standardization and reproducibility of diagnostic criteria is required to compare trials.
- ➤ As most children appear to recover without treatment, better characterization of those who may have initial antibiotic treatment withheld is needed.
- ➤ Trials should be set up to study high risk groups of children who are currently excluded from most studies: those with conditions predisposing them to acute otitis media and children under 18 months of age. These are the children likely to benefit the most from our intervention.
- ➤ We know many important risk factors, but trials are needed to show whether attempts to modify them actually help.
- ➤ The potential benefit of vaccination to older children with recurrent infection needs exploring. As more vaccines are developed, we must know to whom we should be recommending them.
- ➤ The number and quality of trials of surgical intervention does not allow confident guidance to be given as to the long-term benefits or consequences of ventilation tube insertion.
- ➤ More data are required on the long-term consequences of recurrent infection in terms of altered audiometric thresholds, quality of life and language and cognitive development.

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