

Chapter 156: Acute Otitis Media and Otitis Media with Effusion

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Otitis media (OM) is one of the most common diseases of children, the leading cause of hearing loss in children, and the most frequent indication for antimicrobial or surgical therapy in children. The morbidity associated with OM is substantial in the aggregate, and the costs of therapy are not trivial, with estimates of over \$1 billion expended each year in the USA for medical/surgical treatment (Gates, 1983) and more than 1 million operative procedures being performed annually in the USA (Paradise, 1977). Given that prevention of OM by immunization is not feasible today and that 85% of children experience at least one episode of OM, it is fair to say that this disorder is one of the most important unsolved pediatric problems in otolaryngology.

The causes of OM are multiple and, in spite of extensive research, are defined only in general terms. Known predisposing factors are young age, maleness, bottle feeding, crowded living conditions (including day-care centers), heredity, and a variety of associated conditions including cleft palate, immunodeficiency, ciliary dyskinesia, Down syndrome, and cystic fibrosis. The genetic factors of otitis media have yet to be delineated, even though it is well known that OM occurs more frequently in certain families. Treatment of OM varies worldwide with divergent results from numerous studies and clinical trials. The indications for therapy, in particular surgical therapy, remain controversial and, often, empiric. Although the widespread use of antimicrobial agents for OM has reduced the number of acute complications, most importantly intracranial extension of infection, the number of chronic complications, particularly hearing loss due to persistent middle ear effusion, appears to be increasing.

This chapter reviews current knowledge about OM based on contemporary clinical research. Emphasis is on treatment in the ambulatory setting within the context of the opposing forces of societal attitudes about cost-containment on the one hand and the continuing parental pressure for optimal care on the other hand. As the chief providers of surgical care for children with OM, otolaryngologists frequently find their recommendations constrained by the policies of third parties and their advisors who seek to control health care costs by limiting surgical treatment at a time when such therapy has been shown effective in reducing OM morbidity. The welfare of the patient being our primary concern, surgical therapy is selected from a rational, research-based, conservative protocol for patients for whom medical therapy fails and who have no other choice for remediation of symptoms and correction of hearing loss.

Background

Definitions

OM is used herein as a generic term for inflammation within the middle ear cleft beginning behind an intact tympanic membrane. Specifically excluded from this chapter are the various types of chronic suppurative otitis media associated with a permanent perforation of the tympanic membrane or with cholesteatoma. Acute otitis media (AOM) is typified by the symptoms and signs of acute infection (fever; pain; a red, bulging tympanic membrane; and middle ear effusion). Chronic otitis media with effusion (OME) indicates a middle ear effusion without pain, redness, or bulging of the tympanic membrane. Equivalent terms are chronic secretory otitis media, chronic serous otitis media, and "glue ear". Middle ear effusion (MEE) denotes a liquid in the middle ear cleft regardless of etiology.

Clinical and functional anatomy

The middle ear cleft is a continuous space that extends from the nasopharyngeal orifice of the eustachian tube to the furthestmost mastoid air cells. The three main segments are the eustachian tube; middle ear (tympanum); and the air cells of the mastoid, petrosa, and related areas. The middle ear cleft is normally gas-containing and is highly variable in dimension, with each of the main segments delineated or constricted by narrow isthmi and subdivided by mucosal folds or by discrete, communicating cells. The physical characteristics of the tympanic membrane that facilitate sound transfer also allow it to serve as a clinical window into the middle ear cleft, one that permits inferences about the condition of the tympanum based on visible changes in the color, mobility, or position of the tympanic membrane or from changes in the acoustic compliance of the middle ear mechanism, as noted on immittance testing.

The mucosal lining of the middle ear cleft varies from the thick, ciliated, respiratory epithelium of the eustachian tube and anterior tympanum to the thin, relatively featureless cuboidal epithelium in the mastoid cells. In the middle ears of patients with OM, hyperplasia and an increase in the number of goblet cells are common findings (Sade, 1966), which are thought to predispose to the formation of effusion. As the tympanic mucous blanket is swept toward the nasopharynx by the coordinated action of the ciliated epithelium, secretions and particulate matter are cleared from the middle ear into the nasopharynx via the eustachian tube.

The eustachian tube, which is normally closed, opens briefly with swallowing and other maneuvers in which the tensor veli palatini muscle contracts (for example, yawning). Equilibration of pressure in the middle ear to ambient pressure occurs during opening, provided the tubal lumen is unobstructed by inflammatory edema, secretions, or, rarely, by neoplasm or trauma. The closed eustachian tube protects the middle ear from the entry of unwanted materials, except during swallowing, when reflux of nasopharyngeal secretions may occur if the tube is patent and nasopharyngeal pressure rises substantially above that of the middle ear cleft. The detailed morphology of the middle ear cleft is described elsewhere.

Epidemiology of Otitis Media

In a private pediatric practice in the southern USA, 84% of children had one episode of OM, 50% had three or more episodes, and 25% had six or more episodes; 9% of the children had 31% of the total episodes (Brownlee et al, 1969). In the Greater Boston Otitis Media Study (Teele et al, 1989), 93% of children had at least one episode and 74% had three or more episodes of OM. The greatest number of episodes occurred in the first year of life, with decreasing prevalence thereafter. OM is also most common in the first year of life in Finland, with 50% of first episodes occurring by the third year of life (Ingvarsson et al, 1982). Howie et al (1975) described the otitis-prone condition, noting that children with 6 or more episodes of AOM had in common: onset of AOM before their first birthday and infection with *Streptococcus pneumoniae*.

OM is more frequent in the winter months regardless of climate, presumably due to crowding in schools during the upper respiratory infection (URI) season. Henderson et al (1982) confirmed the chronologic proximity of OM to URI in a day-care setting, which held regardless of time of year. Among the multiple risk factors for OM found to be statistically significant by Pukander et al (1982), attendance at a day-care center had the highest correlation.

Risk factors for OM identified by Teele et al (1989) were male gender, bottle feeding, a sibling with OM, and early occurrence of OM. In addition to day care, Pukander et al (1982) found allergy, socioeconomic status, smoking by the mother, a parenteral history of otitis media, and viral infections in the home to be significant risk factors; but birth weight was not significant and breast feeding was protective only until discontinued. The risk factors for AOM and OME are discussed together because they represent two stages of the same disorder, namely OM, and because in the studies where the two are discussed separately the risk factors appear to be the same.

OM is less common in blacks and more common in Native Americans than in whites. Differences in the size and angulation of the eustachian tube appear to be an important factor in understanding racial predilection for or protection from OM (Doyle, 1977). However, submucous cleft palate and sinusitis are also more prevalent in Native Americans (Wiet, 1979) and cannot be excluded as contributing factors to OM. Although impoverished living conditions have long been cited as a possible factor in OM, Shaw et al (1981) found no variation in rates of OM in Arizona Native American children based on living conditions, number of persons in the home, sanitary conditions, method of infant feeding, distance to a health care facility, or mother's education.

The epidemiology of OME has been studied extensively in Denmark through several large-scale tympanometric serial screenings of children in different age groups (Tos and Poulsen, 1979; Tos et al, 1979; Tos et al, 1982). These surveys showed that tympanometric evidence of OME was uncommon in newborns, that by 1 year of age 24% of ears had either a type B or C2 tympanogram, and that the tympanometric patterns change spontaneously in the majority of examinations repeated at 3-month intervals. Improvement was noted more in the spring and summer months, and worsening was more prevalent in the winter months. The point prevalence of type B tympanograms was greatest in 2- to 4-year-olds and declined after the age of 6 to 7 years.

Pathophysiology of Otitis Media

The pathophysiology of OM has been historically linked with abnormalities of eustachian tube function. The three classic functions of the eustachian tube are aeration, clearance, and protection of the middle ear. Early studies of OM in children suggested that obstruction of the tube, that is, underaeration, was the underlying problem. Newer work, however, has refined that concept to suggest that AOM is the result of bacterial entry into the middle ear, that is, failure of protection. This entry is due to an abnormally patent (or compliant) tube, rather than an obstructed one. Tubal obstruction along with failure of clearance, which are common findings in children with OME, may be secondary rather than primary processes. The evidence for these hypotheses is presented below.

Acute otitis media

That AOM is a bacterial disorder is beyond question. Worldwide, the same pathogens are cultured with remarkably similar frequency from the middle ears of children with AOM. Table 156-1 illustrates the results of cultures from children with AOM (Bluestone et al, 1990). Viruses have been recovered from the middle ear in 20% of early cases, in some cases as the sole agent but more often with pathogenic bacteria (Klein et al, 1982). Thus, there is now evidence that respiratory viruses are important in AOM, both directly in the middle ear and indirectly as a cause of antecedent URI.

Because the majority of cases of AOM have a bacterial etiology, it is necessary to account for how the bacteria reach the middle ear. First, in the average case of AOM viral nasal infection precedes the ear infection (Henderson et al, 1982). Pathogenic bacteria subsequently appear and are found in the nasopharynxes of 97% of patients with AOM, with correspondence to the organisms in the middle ear effusion in 69% (Howie and Ploussard, 1972). The adenoid of children with recurrent AOM contains pathogenic bacteria in clinically significant amounts (Brook, 1981; DeDio et al, 1988; Pillsbury et al, 1981). Stenfors and Raisanen (1990) noted a significant age correlation with middle-ear pathogens in the nasopharynxes of clinically disease-free children; 57% of the under-2-year-old group were culture positive compared with 40% of the 11- to 15-year-old children. Thus, it appears that one role of the adenoid in AOM is that of a bacterial reservoir in the nasopharynx.

The most likely route of entry of nasopharyngeal microorganisms into the middle ear cleft is via the eustachian tube. Reflux from the nasopharynx into the middle ear during swallowing has been demonstrated radiographically in OM-prone children by Bluestone et al (1972). Reflux is probably facilitated by nose blowing and closed-nose swallowing, that is, the Toynbee maneuver (Jorgenssen et al, 1988), or aspiration into the middle ear as a result of negative middle ear pressure. One source of negative middle ear pressure is sniffing (Aschan et al, 1980). Children with patulous eustachian tubes, in whom free flow of radiographic material into the middle ear occurs, have been shown by Bluestone et al (1972) to be at high risk for OM. Bluestone et al (1974) demonstrated that young children have shorter, straighter, and more compliant eustachian tubes than adults, which is an important factor in the pathogenesis of OM in children. Sade (1966) was among the first to identify the open eustachian tube as a factor in AOM.

During swallowing, the adenoid is elevated by the soft palate and, when large, may obstruct the posterior choanae and contribute directly to increased nasopharyngeal pressure and, thus, indirectly to reflux. Adenoid enlargement has long been postulated as a factor in otitis media (Meyer, 1870), but if reflux is indeed a sequel of adenoid enlargement, one would expect a greater prevalence of adenoidal enlargement in children with otitis media. However, the evidence does not support this assumption. Adenoidal size in children with otitis media did not differ from that of control children either radiographically (Hibbert and Stell, 1982) or by weight (Gerwat, 1975). No difference was noted in the recurrence rate of effusion in children with large versus small adenoids, nor did the effect of adenoidectomy depend on adenoid size (Gates et al, 1988; Maw, 1985; Paradise et al, 1990).

The close proximity of the adenoid to the mouth of the eustachian tube has led many to presume the existence of a cause-and-effect relationship of the adenoid to eustachian tube function. Under the working hypothesis that bacteria from the adenoid and nasopharynx enter the middle ear via the eustachian tube, it is unnecessary to postulate eustachian tube obstruction as a necessary precedent for AOM to occur, as has been held since the time of Politzer (1883). Indeed, in the early stages of AOM, bulging of the tympanic membrane and positive middle ear pressure are common. Furthermore, it has never been shown that the adenoid physically obstructs the eustachian tube; in fact, it has now been shown that it does not. Honjo (1988) studied 52 children with OME and compared eustachian tube function in those with a large adenoid, which appeared on fiberoptic endoscopy to obstruct the pharyngeal end of the eustachian tube, to those with a clearly open tube. No difference in the opening pressure or in positive pressure equalization was noted between the two groups. Further, there was no difference in eustachian tube ventilation function before and after adenoidectomy. Therefore, Honjo concluded that the adenoid does not mechanically obstruct the eustachian tube.

Takahashi et al (1987) studied 10 adult patients with OME using a thin-pressure catheter and identified the site of eustachian tube obstruction in the distal part of the cartilaginous portion, 5 to 15 mm from the orifice, rather than at the orifice proper. Thus, the role of eustachian tube obstruction as a precursor to AOM is called into question, and the locus of obstruction in chronic OME is likely to be within the eustachian tube rather than at its nasopharyngeal ostium.

The preceding findings suggest that eustachian tube dysfunction, which is clearly demonstrable in these children, may be the result of AOM rather than the cause of it. Thus, the classic theories that the adenoid causes mechanical obstruction of the eustachian tube and that adenoidal enlargement is a factor in the pathogenesis of otitis media are not supported by current research. Improvement in eustachian tube function, however, does occur after adenoidectomy (Bluestone et al, 1972; Honjo, 1988). The exact mechanism for this effect is not clear, although relief from infectious stimulation may play a role (see Adenoidectomy).

Otitis media with effusion

Although obstruction of the eustachian tube does not necessarily precede AOM, eustachian tube dysfunction is a nearly universal finding in children with OME, and, further, ligation of the eustachian tube in animal models invariably produces MEE. However, in humans the obstruction is probably secondary to the inflammatory process rather than the cause of it. The obstruction is usually functional in nature and due to edema, viscous secretions, or both.

There appear to be two broad categories of OME: (1) persistent MEE following an acute effusion and (2) secretory otitis media. However, it is generally not possible to distinguish them on clinical grounds, except when the patient has been examined before onset.

In younger children, at least, OME is often the unresolved stage of an AOM. In a long-term study of otitis media in infants and children, Teele et al (1980) found persistent effusion (> 30 days) in 40% of children after their first episode of AOM and continued effusion in 10% up to 3 months. Pathophysiologically this represents a failure of the middle ear clearance mechanism. Factors that may be involved include ciliary dysfunction, mucosal edema and hyperplasia, viscosity of secretion, and, possibly, a middle ear/nasopharyngeal pressure gradient. Bacterial exotoxin causes a reversible paralysis of middle ear cilia (Bakaletz, 1989), and inflammatory edema can obstruct middle ear drainage. The high viscosity of MEE is well known to every otolaryngologist who has attempted to aspirate a middle ear. Any or all of these pathologic changes may result in a functional impediment to clearance of the secretion. Negative middle ear pressure may result from ciliary clearance of viscous secretions through the eustachian tube: as the bolus of secretion becomes impacted into the tube, the pressure decreases behind it, thereby drawing the tympanic membrane inward. Fenestration of the tympanic membrane, as with placement of a tympanostomy tube, relieves this negative pressure and aids clearance.

A great many cases of OME, especially in older children, have a silent onset without a clinically evident antecedent AOM, although most patients have a history of OM in earlier childhood. It is necessary, therefore, to postulate a second mechanism for the MEE in these cases. Secretion of fluid by the abnormal middle ear mucosa (that is, secretory otitis media) is held by many to be the origin (see, for example, Sade, 1966). Antigenic stimulation in an ear sensitized by prior infection may result in recurrent effusion in the absence of clinically identifiable acute infection (Ryan et al, 1985). Thus the fluid in these cases is produced by the middle ear mucosa in response to subclinical antigenic stimulation rather than by an over acute inflammatory process. Many investigations have confirmed the finding of Senturia et al (1958) that chronic middle ear effusion is not sterile, as previously hypothesized, and contains the same spectrum of microorganisms (Table 156-2) as is found in acute effusions. Thus, the available evidence links chronic middle ear effusion, regardless of its clinical presentation, to bacterial infection.

Barotrauma is a known cause of MEE. When middle ear pressure becomes rapidly lower than atmospheric pressure, a clear, watery transudate may form in the middle ear, which is known as serous OM. Serous OM is also seen sporadically, due perhaps to viral infection and certainly to URI and acute eustachian tube dysfunction. The presence of negative middle ear pressure in many people with URI and the subsequent formation of MEE led Politzer to

develop his hydrops ex vacuo theory as the source of MEE (see Politzer, 1883). Although the theory is attractive in its simplicity, the experimental data do not support ex vacuo theory as the cause of OME (see Treatment, Middle ear infection).

OM is nearly a universal finding in children with cleft palate, and the mechanisms and treatment have been extensively studied (Paradise et al, 1969). The principal defect is related to the function of the tensor veli palatini muscle, which, lacking its usual insertion into the soft palate, is unable to open the tube properly on swallowing. This creates a functional obstruction of the tube.

Histopathology of the temporal bone in cases of childhood OM shows vascular dilatation and proliferation, mononuclear cell infiltration, thickening and metaplasia of the epithelium, gland formation, edema, and exudation. Sando and Takahashi (1990) found eustachian tube malformation and inflammation in their cases with cleft palate and Down syndrome and a striking relationship of inflammatory changes in the middle ear to the degree of eustachian tube malformation.

Sequelae

The adverse effects of OME on hearing and the development of cognitive, linguistic, auditive, and communicative skills are of concern to parents and physicians alike. Although it is clear that severe hearing loss produces severe impairments, there is less agreement that mild hearing loss invariably produces mild impairment.

A number of studies have demonstrated an adverse effect of OM on development. Holm and Kunze (1969) were among the first to discuss this problem. Klein et al (1983) studied 205 3-year-old children in Boston and found that in higher socioeconomic groups, over 130 days of OME in early life is associated with significant language delays and lower IQ scores as compared with children with <30 days of OME. Thus, there is evidence that OME has a retardive effect on development. What remains to be established, however, is the most effective strategy to counter this effect.

The differential effect of hearing loss on development as opposed to just being chronically ill needs to be established. Certainly, in many cases compensatory actions by the parents and teachers and, to an unknown degree, by the children themselves, may provide some remedy.

Frequent or chronic illness of any type often has deleterious effect on the child and family. Disruption of family life by an irritable, painful, or inattentive child plus the extra costs for physicians' services, time lost from work, and medications may place a financial and emotional strain on the family.

The otologic sequelae of OM are permanent perforation of the tympanic membrane, chronic suppurative otitis media, tympanosclerosis, adhesive OM, ossicular necrosis, retraction pockets in the tympanic membrane, cholesteatoma, and sensorineural hearing loss (Tos et al, 1984). In social groups receiving prompt and effective medical therapy these sequelae are uncommon. In areas where health care is not readily available, however, these problems are common. With improved access to care the number of new cases of otologic sequelae

decreased (Wiet et al, 1980). A detailed classification of the otologic sequelae is provided elsewhere.

Diagnosis

The first step in diagnosis is recognition of a problem by the child's care giver. Older children will complain of earache, but infants become fussy, sleep poorly, and pull at the affected ear. In infants, a fever often heralds the onset of AOM, but this sign may be absent in older children. Many parents are alerted by the URI that often precedes the AOM. OME, on the other hand, may be completely asymptomatic. Often the child is so accustomed to reduced hearing sensitivity that parents become aware of the problem only after the child turns up the volume of the radio or television or becomes inattentive to normal conversation.

Otoscopy

The normal tympanic membrane is gray, concave, and translucent and moves briskly on pneumatic otoscopy. The classic signs of AOM are redness and bulging of the tympanic membrane. In the beginning stages of AOM, the drum bulges outward due to positive pressure in the middle ear and it often moves normally. As effusion develops, drum mobility is decreased. In the severe case none of the usual landmarks may be visible. If the process continues to worsen, necrosis of the tympanic membrane occurs and the effusion passes into the ear canal through a typically pinpoint perforation. Massive necrosis of the drumhead is now rare, although necrotizing streptococcal infection is a known cause of permanent perforation.

In early or mild cases of AOM, considerable judgment must be used to differentiate subtle redness of the drum head in the crying child, whose struggling efforts can result in vascular congestion of the tympanic membrane and a simulated otitis media. For this reason, assessment of tympanic membrane mobility is useful to confirm the presence of MEE.

The pneumatic otoscope can be used to increase and decrease the pressure in the external auditory canal, and the tympanic membrane should move inward and outward in synchrony with the pressure changes. In the normal ear, pressures as low as 1 to 2 mm H₂O result in detectable motion of the drumhead (Gates, 1986). Use of the operating microscope improves one's ability to make these subtle judgments. The best method to train one's eye is to examine the ears just before myringotomy in the preoperative holding area. Comparing one's preoperatively recorded findings with the amount of MEE found at surgery is the surest way to develop good otoscopic discrimination for MEE. Most examiners quickly develop good sensitivity for the presence of effusion, but being confident about the absence of effusion (specificity) takes experience and practice.

A number of clinical variants of AOM are known and detectable otoscopically. Myringitis is an inflammation of the tympanic membrane without MEE. The etiology and pathogenesis of myringitis are not well documented. In some cases, myringitis may represent the initial stages of bacterial AOM; in others, it may be due to a viral agent; in still others it may represent the effects of nose-blowing or sniffing. Because of the uncertainty of the clinical course of myringitis, most practitioners is seen in both adults and children. Early reports suggested an association with *Mycoplasma pneumoniae* infection. However, in most

cases bullae on the tympanic membrane are associated with the same pathogenic bacteria as AOM and should be treated similarly. Pain is an outstanding feature of bullous myringitis and is not relieved by opening the bullae.

The classic findings of OME are a retracted, hypomobile or immobile tympanic membrane and a dark, fluid-filled tympanum that obscures visualization of the long process of the incus. Negative middle ear pressure is also associated with a retracted tympanic membrane, but the membrane moves outward briskly when the pressure in the ear canal is sharply reduced with the pneumatic otoscope. Judging the absence of MEE in the face of a retracted tympanic membrane is often difficult.

Tympanometry

Tympanometry has an important place in the evaluation of children with middle ear disorders. The ease of use, acceptance by patients, reproducibility of results, and availability of low-cost machines have led to wide use of tympanometry in physicians' offices. By plotting the amount of energy reflected from the tympanic membrane as the pressure in the external auditory canal is varied from -400 mm H₂O to 100 mm H₂O a tympanogram is obtained, the shape of which provides considerable information about the status of the middle ear. When the tympanum is completely filled or impacted with effusion, the compliance is low and the tympanogram is flat, that is, does not vary with the pressure change. This shape was labeled type B by Jerger (1970). In air-containing ears the shape of the tympanogram is usually peaked with the peak at between 100 mm H₂O (type A) or below -100 mmH₂O (type C). Variants of these tympanogram types have been described for research purposes by Paradise et al (1976) who pointed out that the ear canals of infants (< 7 months) are hypercompliant, and normal tympanograms are possible in the presence of MEE. In these cases, the walls of the canals move with pressure, producing a "canalogram" rather than a tympanogram.

The type A tympanogram has a sharp compliance peak between \pm 100 cm H₂O and suggests an aerated tympanum. Type B curves may be completely flat or have a very rounded compliance peak, which is often below -300 cm H₂O. The type C tympanogram usually has a sharp peak between -100 and -200 cm H₂O. Such tracings reflect only negative middle ear pressure. When the pressure is below -200 cm H₂O, the likelihood of MEE increases. Fig. 156-1 depicts the most common types of tympanometric curves seen in children with OM.

The acoustic reflex is often absent in children. As such, acoustic reflex testing has little usefulness in screening for OM or for making treatment decisions. However, if the tympanogram is normal, an absent reflex may indicate a sensorineural hearing loss (Cooper et al, 1975).

Tympanometric screening should be considered for children in the early school grades when OM is a frequent problem. The high sensitivity of tympanometry in screening programs often leads to overdiagnosis. Therefore, a modified protocol that restricts referrals to those with bilateral low-compliance tympanograms on two occasions 2 months apart appears to be a reasonable plan (Gates et al, 1989).

Audiometry

MEE that fills the middle ear usually results in a moderate conductive hearing loss. Small amounts of MEE may not alter hearing sensitivity. Many studies have shown the variable range of hearing loss in OM, with air conduction thresholds averaging 27.5 dB (see, for example, Fria et al, 1985). Because of excellent cochlear sensitivity, as evidenced by bone conduction thresholds less than audiometric zero in many children, even a significant MEE may not be detectable by audiometric screening methods. Therefore, school hearing screening programs are of limited value in detecting middle ear disease.

Because hearing loss due to MEE is a principal indication for surgical treatment of children with OME, audiometric testing is useful for validating the need for surgery in older children. In young children and infants with classic otoscopic and tympanometric findings, audiometry is unlikely to yield important new information that might alter clinical decisions.

Treatment

Acute otitis media

Antimicrobial therapy

Antimicrobial therapy is the mainstay of treatment in the USA, but not worldwide (Thomsen et al, 1980). Modern agents are effective by the oral route, widely available, safe, and generally well tolerated. Due to the emergence of beta-lactamase-producing strains of *Haemophilus influenzae* and the increasing role of *Branhamella catarrhalis* in otitis media (Shurin et al, 1983), it is important to consider using beta-lactamase-resistant agents. In the past, only a minority of cases of AOM have been due to beta-lactamase-producing organisms, and it has not been cost-effective to use beta-lactamase-resistant drugs, which are generally more expensive, as the first line of treatment. In many cities, however, the incidence of beta-lactamase-producing organisms in AOM has risen to the point that amoxicillin may not be the best initial therapy (Nelson, 1990).

Duration of therapy is an important, unresolved issue. A 10-day treatment course is usually prescribed as a compromise between cost and adequacy of therapy. In children with AOM, comparable results were found by Meistrup-Larsen et al (1983) after 2 to 7 days of penicillin therapy and by Hendrickse et al (1988) after 5 vs 10 days of cefaclor. The latter study noted a significantly higher failure rate with the 5-day treatment in children whose AOM resulted in a perforation and recommended a full 10-day course for these more severe cases.

In communities where beta-lactamase organisms are uncommon, amoxicillin is the first-line drug of choice in non-allergic patients. Trimethoprim-sulfa and the combination of erythromycin estolate and sulfisoxazole (Pediazole) are appropriate alternatives except that the sulfas are not effective against the rare case of beta-hemolytic streptococci. If the inflammation does not show signs of response, the initial agent is discontinued and a second-line drug, such as cefaclor, or the amoxicillin-clavulanate combination is used. Judgment and caution must be exercised if a short treatment course is chosen to be sure that the middle ear has been sterilized and that the infection is controlled. A 10-day course of treatment is

generally used to assure adequate therapy. Many cases would undoubtedly do well with a shorter course, but assuring adequacy would increase costs because of the need for an interim physician visit. It is not possible with current technology to predict which cases would do well with a shorter course of treatment. However, those patients with severe AOM, especially with spontaneous perforation, require full, if not prolonged, treatment.

Adjunct medical therapy

Because of the high frequency of OM and URI, use of a nasal decongestant to open the airway is helpful, but antihistamines should be avoided because their atropine-like effect leads to drying of secretions. Topical decongestants may be used for short periods, but prolonged use may actually worsen rather than improve the nasal airway because of rebound.

Tympanocentesis

Knowledge of the specific organism is important for selection of therapy in cases of AOM occurring in (1) premature newborns, (2) immunocompromised patients, (3) patients with progression of symptoms and signs while receiving an appropriate antimicrobial, (4) cases with intracranial infection, and (5) research subjects. The procedure is performed with an 18-gauge spinal needle attached to a 1-mL tuberculin syringe. No anesthetic is necessary. The needle is inserted into the anteroinferior quadrant of the tympanic membrane and the fluid aspirated into the needle (Fig. 156-7). The fluid is then flushed out of the needle onto an agar plate containing appropriate media for middle ear organisms. The needle hole closes too quickly for any significant drainage to occur, although the aspiration does temporarily decompress the middle ear.

Myringotomy

Although severe AOM has many clinical features of a closed-space abscess, incision and drainage, that is, myringotomy, has proven to be of limited value. Myringotomy promptly relieves severe pain due to AOM in patients with severe pain (Roddey et al, 1966) but adds little to either remission of infection or clearance of MEE in cases of AOM treated with amoxicillin/clavulanate (Englehard et al, 1989). In some European countries, myringotomy has been the mainstay of treatment of AOM (Diamant and Diamant, 1974), with antibiotics being reserved for cases with persistent drainage. The report of van Buchem et al (1981) has been cited as showing no difference in outcome of children with AOM whether antibiotic, myringotomy, both, or neither were used. However, the large number of methodologic flaws and the small number of subjects in each treatment group create substantial problems in interpretation. Although it appears that myringotomy does not offer sufficient benefit in the average case of AOM to justify its costs or risks, in complicated cases, the procedure should be performed without delay to provide prompt drainage and material for culture.

Follow-up

Follow-up examination is important for two main reasons: to assure that the infection is responding to the antimicrobial and to determine that the MEE has resolved. In infants and young children, follow-up is also important to exclude meningitis. In most of these cases, AOM and meningitis coexist, rather than have a cause-and-effect relationship, but the signs of meningitis in infants may be subtle at first. Therefore, a 3-day check is often performed in infants for this reason and to determine that the child is responding to the drug. A 2-week check is often performed to determine if the MEE has cleared. The natural history of AOM indicates that only half the ears will have cleared at this point. Unfortunately, this is often taken as an indication for a second round of antimicrobial therapy, and it is not unusual to see asymptomatic children who have received four different antimicrobials for a single episode of AOM. There is little evidence to suggest that prolonged or repeated therapy is efficacious in the routine case of AOM. If the tympanic membrane has ruptured, indicating a severe episode, it is prudent to continue the antimicrobial agent until drainage has ceased and the tympanic membrane has sealed.

Recurrent acute otitis media

Prophylaxis

Perrin et al (1974) and, later, Varsano et al (1985) demonstrated the effectiveness of sulfisoxazole chemoprevention of AOM in children with frequently recurring ear infections. Because of the risk of hypersensitivity reactions to the long-term use of sulfonamides, many practitioners use a single daily dose of amoxicillin, 20 mg/kg, for 3 to 6 months to prevent recurrent AOM. Casselbrant and her colleagues have compared this form of prophylaxis with placebo and confirmed its effectiveness in preventing AOM (Casselbrant, 1990). Many use the fixed combination of trimethoprim/sulfa even though the manufacturer's recommendations specifically proscribe against prolonged use for AOM prophylaxis. The indications for prophylaxis have not been established scientifically. An empirical indication for prophylaxis is three or more episodes of AOM in a 6-month period.

Children who develop recurrent AOM while on prophylaxis are candidates for surgical management. Using a randomized protocol, Gebhart (1981) demonstrated the effectiveness of tympanostomy tube insertion in preventing recurrent AOM.

Adenoidectomy should logically also be an effective preventive against recurrent AOM, but this hypothesis has not been tested. In the San Antonio trial, the number of episodes of AOM in the two adenoidectomy groups did not differ from the two nonadenoidectomy groups (Gates et al, 1987). In the Pittsburgh study, there was a reduction in the number of episodes of AOM in each of the 2 years after adenoidectomy, but the reduction was significant only in the second year (Paradise et al, 1990). These reports provide only limited insight into whether adenoidectomy can protect against AOM because this was not the primary research question. A clinical study of this question would be both appropriate and timely.

Complications

The suppurative complications of AOM were not trivial in the preantibiotic era. Extension of the infection to involve the bone of the mastoid, soft tissues of the neck, meninges, and cerebral/cerebellar cortex were not uncommon. Today such problems are seldom seen except in areas where access to modern medical care is limited. The most likely problem to be seen by the otolaryngologist today is the patient with postauricular tenderness and fullness in whom the diagnosis of mastoiditis is suspected. Because all severe cases of AOM have mucosal involvement of the mastoid, clouding of air cells on plain radiographs or CT scans is not unusual. These patients deserve prolonged antimicrobial therapy. The key sign of mastoid osteitis, namely demineralization of the air cell septae, lags behind the clinical findings. Once osteitis is diagnosed, mastoidectomy is generally warranted to remove the infected, often necrotic bone. In many of these cases, a subperiosteal postauricular abscess is present and surgery is performed for drainage of this pus and removal of the infected bone.

Intracranial complications are more common now in cases of chronic suppurative otitis media than in acute cases. Nonetheless, awareness of the possibility of meningitis, extradural abscess, or even brain abscess is an important part of the evaluation of patients with severe or neglected AOM.

Otitis media with effusion

Antimicrobial therapy

OME, as AOM, is also a bacterial disease, and the MEE is known to contain viable, pathogenic bacteria (Liu et al, 1975). Antimicrobial therapy is a logical choice, and the efficacy of this therapy has been determined in several reports. Gates et al (1986a) used the combination of erythromycin ethylsuccinate and sulfisoxazole (Pediazole) and found that 45% of cases cleared by 1 month and 60% by 2 months. In placebo-controlled trials by Healy (1984) using trimethoprim-sulfisoxazole, Mandel et al (1987) using amoxicillin, and Thomsen et al (1989) using amoxicillin/clavulanate, the clear rates in the treated cases in each study were significantly greater than in the control groups. For children with asymptomatic OME discovered by screening or interval examination, a course of an appropriate antimicrobial is recommended, followed by at least a 1-month observation period. If signs of improvement are noted by otoscopy or tympanometry, a second course or additional observation may be warranted. If no change is seen at 1 month, surgical treatment may be considered.

Antihistamines and decongestants

To determine whether systemic medical therapy is of value in patients with OME, Cantekin et al (1983) randomly assigned 533 infants and children with OME to receive either a decongestant-antihistamine or placebo. The clearance of OME did not differ between the two groups. As a consequence of this study, the routine use of decongestants in children with OME has been abandoned. However, specific nasal symptoms in OME patients should be sought and treated as indicated.

Middle ear inflation

Because negative middle ear pressure and retraction of the tympanic membrane are common findings in OME, middle ear inflation has been used historically as a treatment option (Gottschalk, 1962). However, inflation, either by eustachian tube catheterization (Politzer's maneuver) or by autoinflation (Toynbee's maneuver), is not generally used. Although one can achieve transient aeration, the long-term net effect is often to worsen the negative middle ear pressure because the excess air injected into the middle ear passes out the eustachian tube, and the oxygen remaining is quickly absorbed through the middle ear mucosa (Shinkawa et al, 1990). It is important to remember that the gas in the middle ear differs substantially from air, being hypercapnic and hypoxic, with gas tension values similar to arterial blood (Segal et al, 1983). Therefore, middle ear inflation with a different gas such as room air disturbs the equilibrium of the system and produces little lasting benefit. Transtympanic inflation of the middle ear with an inert gas or injection of mucolytic agents has been used, but the need for a general anesthetic for instillation in children and the limited duration of effect have negated their usefulness.

The ex-vacuo theory does not adequately explain negative middle ear pressure without MEE. It has long been presumed that the oxygen in the middle ear is continually absorbed, until replenished by the ingress of nasopharyngeal air with swallowing. This effect would mean that middle ear pressure would fall during sleep or other states in which swallowing does not occur. However, it has been shown that resting middle ear pressure in the morning is often slightly positive due to CO₂ retention (Magnuson and Falk, 1984). Sniffing is a common practice in patients with a URI that may lead to negative middle ear pressure (Holmquist, 1985). Although the ex-vacuo theory is probably valid in cases of barotrauma, where there is a sudden drop in relative middle ear pressure and formation of MEE from transudation due to capillary damage, it does not appear to play a role in AOM or OME.

Corticosteroid

The use of a short course of prednisone (1 mg/kg maximum dose) as a possible alternative to surgical therapy for treatment of persisting OME was studied by Schwartz et al (1980), who demonstrated a benefit, and by Lambert (1986) who did not. A randomized, double-blind, crossover experimental design was used in both studies with concomitant use of sulfisoxazole in the former and amoxicillin in the latter. Explanation and reconciliation of these contradictory results must await further study. Although the risks of a short course of corticosteroid is low in a child receiving an antimicrobial agent, one should keep in mind the hazards of such therapy in the presence of a systemic viral infection. For the moment, then, corticosteroid treatment of OME must be considered experimental but worthy of continued study.

Surgical therapy

Considerable controversy continues about the indications for surgical therapy. The debate centers primarily on duration of effusion and whether persistent effusion, with its attendant mild conductive hearing loss, is harmful to the child's development. It is clear from the work of Klein et al (1983) that even mild conductive hearing loss is a risk factor for impaired development. It is undoubtedly true that this risk may be reduced through compensatory measures of parents, teachers, and the children themselves; but this reduction is difficult to identify and quantify. Therefore, reduction of morbidity by surgical therapy remains an important option for parents of affected children, who often elect surgical treatment for prompt remediation of the hearing loss and avoidance of long-term sequelae. Surgical therapy does not cure patients with OME, but it does substantially reduce morbidity when medical therapy has failed.

For OME persisting more than 90 days in spite of adequate antimicrobial therapy, surgical treatment may be recommended for those children with persisting OME and hearing loss. Some physicians advise waiting 120 or more days, hoping that if one delays long enough the effusion may resolve. However, Maw (1988) has noted persistent effusion for up to 3 years in 40% of cases and up to 5 years in 25% of cases. Thus a balance must be reached between the probability of spontaneous resolution versus the sequelae of prolonged morbidity and the risk of intercurrent infection. The time criterion is tempered by season. In the fall surgery is recommended more often because of the high likelihood of exacerbation of the condition during the winter months; whereas in the spring, when otitis media and upper respiratory infections are less prevalent, the effusion has a higher likelihood of spontaneous resolution and longer observation is warranted. Other, more urgent indications for surgical intervention, regardless of time of year, relate to structural abnormalities of the tympanic membrane that may lead to ossicular erosion or cholesteatoma. These indications are retraction pockets in contact with the long process of the incus or the stapes or pockets in which epithelial debris accumulates, thus heralding an incipient cholesteatoma.

After a decision is made to treat the child surgically, a second decision about the type of procedure must be made. Historically, myringotomy, adenoidectomy, tympanostomy tubes, and even tonsillectomy have been advocated. Because tonsillectomy appears to have no additional effect on the middle ear effusion over adenoidectomy alone in cases of OME (Maw, 1983), it will not be discussed further.

Myringotomy. Evacuation of the MEE by myringotomy and suction aspiration has been studied as one type of therapy for OME. The results from this simple procedure have been disappointing (Gates et al, 1985; Mandel et al, 1989). Most investigators and clinicians agree that if a child is to receive an anesthetic for such treatment, then tympanostomy tubes should be inserted or an adenoidectomy performed (or both) because the cost-benefit ratio for myringotomy and aspiration is too low to justify myringotomy as an independent procedure.

Tympanostomy tubes. Modern tympanostomy tubes (TT) were introduced by Armstrong in 1954 and have become the therapeutic gold standard and the most widely used treatment option for OME. Improved hearing and a decreased rate of recurrent AOM have resulted from their use in large number of cases. However, the complications of tympanostomy tubes - purulent otorrhea, permanent perforation, recurrent effusion - are not

clinically insignificant. Most parents are willing to accept these small risks for the benefits accrued by improved hearing and freedom from OM while the tubes are in place. Nonetheless, the search continues for the most effective treatment strategy for children with OME.

TT are known by many different terms - pressure-equalizing tubes, grommets, bobbins, ventilating tubes - and are manufactured in a variety of shapes, sizes, and materials. No convincing evidence exists to suggest that one material has better results than another. The major classes of TT are the short, generally grommet-shaped tubes and the long T-shaped tubes introduced by Goode (1973). The former have retention times of a year or less, whereas the T-shaped tubes may stay in place for several years. The risk of permanent perforation from the short-term tubes is about 1% and increases to a maximum of about 5% depending on the length of time the tube remains in place and the diameter of the tube. The factors that influence perforation have not been determined.

The rationale for TT is prolonged ventilation of the tympanum. Replacement of the MEE by an aerated tympanum results in prompt return of hearing to preinfection levels in the vast majority of cases. Experimental evidence suggests that the mucosal hyperplasia of the tympanum will revert to a more normal condition with aeration (Sade, 1966). Once the tubes are extruded, however, the clinical benefit appears to end (Gates et al, 1987).

Many investigators have compared the effects of TT in one ear and myringotomy in the opposite ear in children undergoing an adenoidectomy. The conclusions of the studies varied: No difference was noted by Leek (1979) or Lildoholdt (1979), but Shah (1971) found better results in the ear with a TT. Kilby (1972) used the same protocol, except that adenoidectomy was not performed, and found no difference in outcome between TT in one ear and myringotomy in the other. Using a different design in which patients, not ears, were compared, Gates et al (1987) found a significantly better outcome in terms of hearing, less time with MEE, longer time to recurrence, and fewer repeat operations in the children with TT as compared with myringotomy and aspiration. Similar conclusions were reached by Mandel et al (1989).

Paradise et al (1988) argue that tympanostomy tubes should be used as the initial procedure of choice for persistent OME, as it is less involved and less expensive than adenoidectomy. They reserve adenoidectomy for cases of recurrent OME. However, owing to the substantially higher rate of reoperation after tubes-only procedures, the greater initial expense of adenoidectomy may be negated by the cost of repeat intubation procedures and the overall cost may be equivalent, depending on hospital and physician charges (Gates et al, 1988b). In making such a decision, risks versus effectiveness must be considered in light of the needs of the child and family.

Technical considerations. Otological medications containing topical antimicrobial agents are often used after TT insertion or, later, if purulent otorrhea develops. These medications have not been associated with sensorineural hearing loss in humans as in experimental animals, presumably because of differences in the anatomy of the round window niche and membrane (Morizono, 1990). However, caution should be exercised in the indiscriminate use of these medications in noninflamed ears because of the risk of round window absorption and possible sensorineural loss.

Adenoidectomy. Adenoidectomy is being used again for the treatment of OM because recent studies have confirmed its effectiveness (Gates et al, 1987; Maw, 1983; Paradise et al, 1990). Adenoidectomy was once the principal surgical treatment for OM; with the widespread use of TT, it was used far less often in USA, but not in the parts of the world (Muenker, 1980). Probable reasons for this difference were the following: adenoidectomy takes longer than TT insertion, adenoidectomy carries a risk of hemorrhage and other complications such as hypernasality, the mechanism of its effect on the middle ear is not well understood, and several studies failed to demonstrate effectiveness (Fiellau-Nikolajsen et al, 1983; Roydhouse, 1980; Widemar et al, 1985).

The adenoid (pharyngeal tonsil) forms the uppermost part of the ring of lymphoid tissue surrounding the oropharyngeal isthmus, described in 1884 by von Waldeyer. Historically the term *adenoids* has been used loosely to indicate enlargement of the pharyngeal tonsil and does not mean that the adenoid is a bilateral structure. The adenoid is covered by respiratory epithelium that is rich in goblet cells and is plicated into numerous surface folds. Abundant lymphocytes are found within, especially on the crests of the folds. The adenoid develops as a midline structure by the fusion of two lateral primordia during early fetal life (Slipka, 1981), is fully developed during the seventh month, and increases in size until the fifth year of life, often causing some degree of airway obstruction. Thereafter, the adenoid gradually atrophies and with growth of the nasopharynx the airway improves (Jeans et al, 1981).

The adenoid is an important site of contact of inhaled microorganisms and other antigens with immunoreactive cells. The nasal mucociliary blanket carries material posteriorly across the adenoid. With the other components of Waldeyer's ring, the adenoid is important in the mucosal immunity of the upper aerodigestive tract, and like the palatine tonsil, the adenoid is regarded as a B-cell organ.

Rationale for adenoidectomy. The chief rationale for removal of the adenoid in children with OM has been enlargement, particularly that causing nasal obstruction and mouth breathing. Removal of a large adenoid that occludes the nasopharynx will open the airway and relieve the nasopharyngeal overpressure that occurs with closed-nose swallowing (Bluestone et al, 1975). Removal should also lessen eustachian tube reflux. Although the proportion of children with OM due to reflux is unknown, this rationale for adenoidectomy is logical. The association of enlargement with abnormality has been called into question, however, because adenoid and tonsil enlargement results from clonal expansion of immunocompetent cells (Fujiyoshi et al, 1989). Thus the large adenoid may be more immunocompetent than the small adenoid because chronic infection is associated with cellular depletion (Bernstein, 1990). Brandtzaeg and Berdal (1978) demonstrated a general decline in immunocytes in the tonsils of patients with recurrent tonsillitis. Thus current knowledge suggests that adenoid enlargement, a common phenomenon in the 4- to 7-year age group (Fujioka et al, 1979), does not necessarily indicate abnormality. Basing the rationale for adenoidectomy in children with OM on size alone, therefore, has little scientific basis. Further, clinical evidence from three separate studies (Gates et al, 1987; Maw, 1985; and Paradise et al, 1990) indicates that the effect of adenoidectomy is independent of size. Removal of a large adenoid is a clinically attractive option because it offers symptomatic relief to a compromised airway; removing a small, chronically infected adenoid offers no obvious benefit except in controlling OM.

The other classic rationale for adenoidectomy is improvement in eustachian tube function. Honjo (1988) showed improvement in equilibration of positive middle pressure postadenoidectomy but no change in the ability to equilibrate negative pressure and no change in the static opening pressure of the tube. Where there is obstruction of the eustachian tube, either anatomic or functional, this is a logical rationale for the procedure. Bluestone et al (1972) showed that obstruction of the eustachian tube is unusual in most children with OME. The obstruction is functional in most cases. Eustachian function tests are not available except in research centers and, thus, eustachian tube function is not routinely tested preoperatively. In children with hypercompliant eustachian tubes, adenoidectomy may increase reflux, particularly if tissue in the Rosenmüller's fossa is removed (Bluestone et al, 1972).

The third and most current rationale is removal of the chronically infected adenoid to eliminate a nasopharyngeal source of infection (Gates et al, 1988a). Adenoidectomy should result in a smooth lining of the nasopharynx, which should decrease bacterial colonization of the nasopharynx and, indirectly, the middle ear. Further research is needed to develop the methodology to identify subgroups of children who might benefit the most from adenoidectomy for control of infection.

Efficacy. Studies of the efficacy of adenoidectomy have yielded conflicting results. The wide variation in experimental design and methods used in studies of adenoidectomy make direct comparisons of efficacy difficult. Important variations include sample size, observer validation, entry criteria, randomization strategies, and follow-up procedures. The principal studies cited as showing a lack of effect of adenoidectomy are those by Roydhouse (1980), Fiellau-Nikolajsen et al (1983), and Widemar et al (1985). In these studies, comparisons of outcome were made at one or a few widely spaced observation times rather than sequentially at regular intervals, which prevented the determination of recurrent interval disease. Thus it is likely that these studies were not sensitive enough to detect differences among the treatment groups that may have been clinically significant. Studies demonstrating a significant effect from adenoidectomy are those by Maw (1983), Gates et al (1987), and Paradise et al (1990). Other earlier studies of adenoidectomy (Mawson et al, 1967; McKee, 1963a, b) are not reviewed here because of severe flaws in experimental design (Paradise, 1976).

Studies failing to demonstrate efficacy. Roydhouse (1980) randomly assigned 100 children aged 3 to 14 years with OME to receive TT with or without adenoidectomy. Otoscopy was performed annually and showed a slightly higher disease-free rate in the adenoidectomy group: 82% vs 77% at 1 year, 85% vs 82% at 2 years, and 94% vs 84% at 3 years, but he concluded that adenoidectomy was not effective.

Fiellau-Nikolajsen et al (1983) observed 42 3-year-old children for 4 years, who had been randomly assigned to receive bilateral myringotomy with or without adenoidectomy. No significant difference in outcome at any assessment interval was noted, even though nearly half the children had normal tympanograms preoperatively. The inclusion of a large proportion of patients destined to do well regardless of therapy may have decreased an already small sample size to the point that a clinically significant difference could not have been detected.

Widemar et al (1985) nonrandomly assigned 54 children with mucoid effusion, selected from 78 patients with chronic otitis media undergoing myringotomy, to receive adenoidectomy or no adenoidectomy. After 2 years, otoscopy was normal in 24 patients (54%) who had adenoidectomy vs 13 of 35 patients (37%) without adenoidectomy. They found this difference to be statistically insignificant but acknowledged that their limited sample size could not detect a difference of 30% or less between the groups.

Studies demonstrating efficacy. Maw (1983) studied 103 children from 2 to 12 years old (mean age, 5.25 years) with bilateral OME and randomly assigned them to three groups: adenotonsillectomy (N = 34), adenoidectomy (N = 36), or neither (N = 33). At operation, one ear was assigned randomly to receive a tympanostomy tube. The rate of clearance of MEE in the unoperated ear (as judged by otoscopy only) was the criterion of success. At 3, 6, 9, and 12 months after surgery the clearance rates were: 56%, 64%, 58%, and 72%, respectively, for adenoidectomy; 50%, 59%, 62%, and 62%, respectively, for adenotonsillectomy; 22%, 26%, 19%, and 26%, respectively, for the control group. The difference between the two surgical groups and the control group at 12 months (71% vs 26%) was significant, but the difference between the adenoidectomy and the adenotonsillectomy was not.

Gates et al (1987) randomly assigned 491 4- to 9-year-old children with OME to surgical control, TT, adenoidectomy, or adenoidectomy plus TT. All children had failed antimicrobial therapy and none had prior tonsil or adenoid surgery. Bilateral myringotomy and suction aspiration of the middle ears were performed in all cases. The fossae of Rosenmüller were not disturbed. The children in the two adenoidectomy groups experienced significantly less time with effusion and fewer repeat surgical treatments over the 2-year follow-up period than those who did not undergo adenoidectomy. There was no difference in time with abnormal hearing in the three surgical groups, regardless of whether TTs were placed. The reduction in morbidity (from control levels) in time spent with recurrent effusion was 29% with TT only, 38% with adenoidectomy and myringotomy, and 47% with adenoidectomy and TT.

Paradise et al (1990) examined the effect of adenoidectomy in two groups of children with OME recurring after prior TT. One group (N = 99) was randomly assigned; the other (N = 114) group was determined by parental choice for the procedure. In both groups, the outcomes for the adenoidectomized children were statistically better than for the control children for both of the follow-up years, with greater differences in the first than the second year. Adenoidectomy included routine curettage of Rosenmüller's fossa. In addition to significant reduction in time with effusion, they noted clinically significant reductions in AOM (28% and 35% in years 1 and 2, respectively) in the adenoidectomy group, which was statistically significant only in the second year.

These three recent randomized clinical trials, with adequate sample size and frequent postoperative follow-up, have demonstrated statistically significant and clinically important effects from adenoidectomy in reducing morbidity in children with persistent OME. Thus the evidence supports the conclusion that adenoidectomy is effective in improving the natural history of OME in severely affected children 4 years of age and older.

Technical considerations. The goal of adenoidectomy is complete removal of the midline adenoid pad to achieve smooth reepithelialization of the nasopharynx. Curettage of the tissue in Rosenmüller's fossa is performed by many who strive for complete removal of all upper pharyngeal tissue and not by others for fear of scar tissue formation and contracture, which may contribute to a patulous eustachian tube and reflux. In either case, care must be taken to avoid direct injury to the torus tubarius that might result in stenosis. From our experience the latter complication is rare, whereas the incidence of the former is not known but is probably not uncommon.

Although the most common complication of adenoidectomy is postoperative bleeding, the incidence is low. Of 250 cases performed by 13 surgeons, only one child required operative treatment for bleeding (0.4%) and none needed or received blood transfusion (Gates et al, 1987). Helmus et al (1990) noted that only four patients in 1000 (0.4%) bled after outpatient adenoidectomy, and all instances occurred in the first 6 postoperative hours and were without managed transfusion.

Transient velopharyngeal insufficiency (VPI) may occur after removal of a large adenoid but resolves quickly in the majority of cases. Persistent VPI is the most feared complication because it requires either a prosthesis or a secondary procedure, such as a pharyngeal flap, for correction. Most cases of postoperative VPI are due to an undetected submucous cleft palate. Preoperative evaluation with fiberoptic nasopharyngoscopy is helpful in detecting an occult submucous cleft, which is often more obvious on the posterior than on the anterior surface of the palate (Croft et al, 1978).

Reports regarding the immunologic consequences of tonsillectomy and adenoidectomy are conflicting, yet it is clear that no major systemic immunologic deficiencies result from these procedures (Siegel, 1984). Ogra (1971) showed a threefold to fourfold drop in titers after tonsillectomy and adenoidectomy in children previously immunized with live poliovirus vaccine. Because adenoidectomy alone does not disturb the remainder of Waldeyer's ring, likelihood of a major immunologic sequel developing from adenoidectomy is remote.