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Disorders of the ear in pediatric patients include infections of the middle ear, infections of the external ear canal, congenital and acquired masses, and complications related to blunt or direct head trauma. Early and correct recognition of these conditions is essential.

Otitis media (OM) is an acute or chronic inflammation of the middle ear. Acute OM (AOM) is an acute infection of the middle ear with middle ear effusion (MEE) by otoscopic examination. The tympanic membrane (TM) often is red, bulging, and opaque. AOM usually is manifested by ear pain or tugging or rubbing of the ear. Fever and irritability may be present. OM with effusion (OME) is a painless collection of fluid in the middle ear without any signs of acute inflammation or infection. Most cases evolve from AOM, but a preceding infection is not necessary.

Recurrent AOM is defined as three new episodes of AOM within 6 months or four episodes within 1 year. Resolution of the infection should be documented between each episode of AOM, but this may be difficult if OME develops. Chronic OM is the presence of MEE for more than 3 months. Recurrent and chronic disease can occur simultaneously because the presence of one condition often predisposes to the other.

Complications of OM can be divided into intratemporal and intracranial causes (Table 61-1). Children differ from adults in that complications are more likely to occur from AOM than chronic ear disease and often are the result of delayed treatment. Hearing loss, the most prevalent complication, can be conductive, sensorineural, or mixed. The magnitude of the conductive hearing loss ranges from 15 to 40 dB and seems to result from the quantity, rather than the quality, of fluid in the middle ear. This type of hearing loss disappears when the MEE resolves. Infectious or inflammatory mediators in the middle ear or in the labyrinth can cause sensorineural hearing loss.

Children with AOM usually have fluid present in the mastoid air cells because of the direct connection with the middle ear (Fig. 61-1). Mastoiditis, an inflammatory process that accompanies OM, develops when there is destruction of bone or formation of a subperiosteal abscess within the cavity. Early in its course, mastoiditis may resolve spontaneously. When the infection persists for more than 1 week, inflammatory granulation tissue forms, and a series of changes occurs within the cavity, resulting in either acute or chronic mastoiditis. Acute mastoiditis is subdivided into pathologic stages depending on the progression of the disease within the mastoid air cells. Chronic mastoiditis almost always is associated with chronic suppurative OM, a chronic (≥2 months) drainage from the middle ear and the mastoid through either a perforation of the TM or a tympanostomy tube.

The most common suppurative intracranial complication of OM and mastoiditis is bacterial meningitis, although its incidence has decreased dramatically since the advent of antibiotic therapy for acute infections and routine immunization against Haemophilus influenzae type B. Other
intracranial complications, such as subdural empyema and brain abscess, occur rarely in developed countries and are associated more often with other chronic diseases, such as sinusitis, rather than AOM.

Otitis externa (OE) refers to inflammation and infection of the external ear canal or the auricle. It is commonly known as swimmer’s ear, an acute diffuse inflammation of the external auditory epithelium extending from the pinna all the way to the TM. Although inflammation of the TM also may occur, it usually can be differentiated from AOM by pneumatic otoscopy.

Perforations of the TM may be temporary or permanent depending on whether they are associated with AOM, a chronic infection, a surgical procedure, or trauma. Permanent perforations are classified as central or marginal. In central perforations, the fibrous annulus is unaffected, and the TM circumscribes the entire intact ring. Marginal perforations involve the fibrous annulus, and the defect is seen posteriorly on the TM. Abnormal growth of squamous epithelium into the middle ear as a result of destruction of the margin of the TM may lead to formation of a secondary cholesteatoma.

The most common ear mass in children is a cholesteatoma, a histologically benign lesion in the middle ear, mastoid space, or petrous bone containing keratinizing squamous epithelium. Occasionally a cholesteatoma may be found in the external auditory canal. Congenital, or primary, cholesteatoma is defined as the presence of squamous epithelium medial to an intact TM without a significant past history of AOM or eustachian tube dysfunction. Acquired, or secondary, cholesteatoma occurs more commonly as a complication of chronic OM. Cholesteatomas are locally invasive and can destroy important structures, such as the ossicles, cochlea, or semicircular canals.

**FUNDAMENTALS**

Figure 61-1 illustrates the normal anatomy of the ear and the relationship of the external, middle, and inner ear to one another. The location of the eustachian tube acts as a conduit connecting the middle ear to the nasopharynx. The development of OM in children is the direct result of abnormal function of this tube. Anatomic features, such as its length and position relative to the posterior nasopharyngeal wall, contribute to increased frequency of OM. The eustachian tube in young infants is shorter and more horizontal than in older children (Fig. 61-2).

The eustachian tube has three major physiologic functions: (1) ventilation of the middle ear, (2) drainage of middle ear fluid, and (3) protection of the middle ear from nasopharyngeal secretions. These functions occur via active dilation of the tube by contraction of the tensor veli palatini...
muscle (Fig. 61-3). Normal function of this muscle is essential in preventing MEE and acute infections.

Eustachian tube dysfunction is the result of abnormal patulence of the tube, mechanical obstruction (Fig. 61-4), or both. Functional obstruction results from poor tensor veli palatini function. Mechanical obstruction occurs from either an extrinsic or an intrinsic cause. Extrinsic causes include enlarged adenoids or the presence of a nasopharyngeal tumor. Intrinsic causes often are related to injury or inflammation of distal tubal epithelium by upper respiratory infections with viruses such as influenza or respiratory syncytial virus. Epithelial damage leads to increased mucus secretion and cellular debris, which then obstructs the lumen. If the eustachian tube is occluded, negative middle ear pressure develops as oxygen is absorbed from this space. Transudative serous capillary fluid accumulates in the middle ear and the mastoid air cells and can be infected with nasopharyngeal bacteria that enter through the eustachian tube as it opens intermittently. Replication of the bacteria within the serous fluid leads to a series of inflammatory processes with the release of bacterial and host cell products into the middle ear. The clinical manifestation of these events is AOM.

Some infants and young children are at increased risk for developing OM. Well-documented environmental risk factors include passive tobacco smoke exposure, lack of breast-feeding, bottle-propping, poor socioeconomic status, and day care attendance. Children with craniofacial anomalies or congenital or acquired immunodeficiency syndromes have a higher incidence of OM and recurrent disease, as do certain ethnic groups, including whites, Alaskan Eskimos, and Native Americans.

The peak incidence of AOM occurs during the first 2 years of life, particularly between 6 and 12 months of age. Another peak occurs after the time of school entry. In temperate areas of the world, most infections occur during the fall, winter, and early spring, seasons when most viral upper respiratory infections occur in young children. Recurrent disease after age 5 years should prompt a more extensive evaluation to exclude other predisposing factors, such as allergies or an underlying immunologic condition.

Streptococcus pneumoniae, nontypable H. influenzae, and Moraxella catarrhalis account for approximately 85% of acute infections. Gram-negative enteric bacilli account for about one fifth of middle ear effusions in infants younger than 6 weeks old. Other, less common bacteria reported to cause AOM include group A, C, and G streptococci; Staphylococcus aureus; Streptococcus epidermidis; and mixed flora. The role of viral pathogens has become much more apparent with more sensitive laboratory detection methods. Common virus isolates from middle ear fluid in acute infections include respiratory syncytial virus, influenza A and B, rhinovirus, adenovirus, coronavirus, and parainfluenza (types 1, 2, and 3). Some studies report that 25% of middle ear effusions are "sterile." Approximately 70% of isolates from chronic MEE yield bacterial growth. In MEE Pseudomonas aeruginosa and coagulase-negative staphylococci play a prominent role.

Mastoiditis is an extension of the acute inflammatory process of OM. At birth, the mastoid air cell system com-
prises a single cell, the antrum, which is connected to the middle ear by a small channel called the *aditus ad antrum*. Pneumatization of the mastoid bone begins soon after birth and continues throughout life. By age 2, this process is usually extensive. All mastoid air cells end up interconnected with the antrum, which sometimes is referred to as the bottleneck when an acute infection cannot drain from the mastoid into the middle ear.

Clinically significant disease develops when an infection within the mastoid cavity spreads to the periosteum covering the mastoid process. With further progression, the bony trabeculae that separate the mastoid air cells are destroyed, resulting in a mastoid empyema. Invasion of adjacent structures leads to formation of a soft tissue, subperiosteal abscess, or chronic mastoiditis. Rarely, further extension into the petrous portion of the temporal bone may occur, resulting in petrositis. A classic triad of retrobulbar pain, persistent otorrhea, and ipsilateral abducens nerve palsy (Gradenigo’s syndrome) is seen in patients with this complication. Infection also can spread to the labyrinth and facial nerve or into the intracranial cavity causing more serious morbidity and mortality. Acute mastoiditis occurs most commonly as a complication of chronic suppurative OM but can develop after an acute infection. Acute TM perforation and the presence of a cholesteatoma are predisposing factors.

Currently the incidence of mastoiditis is reportedly less than 0.1%. The incidence may change with recommendations to use oral antibiotics only in children with unequivocal acute infections of the middle ear. Countries in which acute OM is observed initially, rather than treated with antimicrobials, do not have a higher incidence of mastoiditis. Most cases of acute mastoiditis are caused by the same organisms that cause AOM. Other pathogens include *Streptococcus pyogenes* and *S. aureus*. In subacute or chronic cases, *S. aureus* and gram-negative enteric bacilli, such as *Escherichia coli*, Proteus, and *P. aeruginosa*, are common isolates from persistent and indolent infections.

The external ear consists of the pinna and the external auditory canal, which consists of a lateral cartilaginous portion (approximately one third of the canal) and a medial osseous portion. Before development of the osseous portion during infancy, the external auditory meatus is predominantly cartilaginous. The squamous epithelium is thicker over the lateral cartilaginous portion of the canal compared with the osseous portion from subcutaneous tissue containing hair follicles and sebaceous and ceruminous glands. The sebaceous glands, located superficially in the dermis, secrete an oily substance called *sebum*, whereas the apocrine (ceruminous) glands secrete a milky opaque fatty fluid. Cerumen is a mixture of these two secretions and desquamated epithelial cells. Its purpose is to form a protective acidic lipid layer that limits pathogenic bacterial overgrowth and inhibits maceration from water or sweat.

OE develops from alterations in the pH of the external auditory canal, local trauma, or secondary maceration of the skin. High humidity, increased environmental temperature, and water contamination increase the pH of the canal and subsequent risk for acute OE. Moisture in the canal raises the pH and removes the protective lipid layer of the skin,
leading to edema and maceration of the epithelial lining. Excessive sweating, absence of cerumen, hearing aid irritation or improper fit, earplug usage, and the insertion of a foreign body to scratch the ear canal also can play a role in the development of infection. Water that has high bacterial counts and the pH of pool water play an important role in OE rather than the number of bacteria in the water. Hot tubs, whirlpools, and pressurized ear irrigation are other sources of water contamination.

OE is seen most often in the summer months in temperate climates and year round in warmer areas. Most cases are unilateral, with mild-to-moderate diffuse inflammation. Rarely, in severe cases, infection may extend to the surrounding soft tissue and lymph nodes. The organisms primarily responsible for acute OE are *P. aeruginosa* and *S. aureus*. Other organisms include *S. epidermidis*, *Proteus*, *Enterobacter*, *Klebsiella*, and streptococci. Many acute infections are polymicrobial, with aerobic and anaerobic bacteria. Acute otomycosis (fungal OE) accounts for 10% of cases of OE and is caused most often by *Aspergillus niger*.

Acute tympanic perforations are secondary to trauma or AOM. Certain racial groups may experience perforations with AOM at a higher rate than the general population. Spontaneous eardrum perforation has been reported with almost every episode of AOM in Eskimos and Native Americans. Chronic perforations occur under a variety of circumstances, including (1) when an acute perforation fails to heal after an episode of AOM, (2) after spontaneous extrusion (or removal) of a tympanostomy tube, (3) after long-standing atelectasis of the TM, and (4) when an acute traumatic perforation fails to close. Small chronic perforations, regardless of their location, have little impact on hearing in the absence of other abnormalities. Large perforations can be associated with a 20- to 30-dB conductive hearing deficit.

Traumatic perforations of the TM occur from rapid changes in ambient pressure that occur with certain sports, such as diving, water-skiing, and surfing, or with activities such as flying in an unpressurized airplane. Blunt trauma with child abuse (e.g., the slap of an open hand against a child’s ear by an angry parent) can rupture the TM. TM lacerations may occur by the accidental or intentional placement of a foreign body, such as a cotton-tip applicator or hairpin, into the external auditory canal. Some injuries are severe enough to damage the ossicular chain or cause a perilymphatic fluid leak or fistula.

Congenital cholesteatoma is an asymptomatic keratotic white mass located behind an intact membrane in a patient with no significant history of recurrent middle ear disease or previous ear surgery. Although the origin of the lesion is not well understood, it is presumably present since birth. The most widely accepted theory is that congenital cholesteatoma results from a persistent epidermoid formation in the developing middle ear that, under normal circumstances, disappears after 33 weeks’ gestation. The most common age of diagnosis is 4 years, but this also can range from infancy to adolescence. Boys are affected more commonly than girls in a ratio of 3:1. Most acquired cholesteatomas occur as a complication of chronic OM and arise as a focal area of retraction of the TM. Three main theories exist to explain their pathogenesis: (1) implantation and invasion of squamous epithelium into the middle ear secondary to ear surgery or temporal bone fracture, (2) migration and invasion of squamous epithelium through a perforation of or retraction of the TM, and (3) metaplasia of low cuboidal epithelium of middle ear mucosa caused by infection. Regardless of the etiology, certain children are at increased risk for the development of secondary cholesteatoma, including children with cleft palate, trisomy 21, and aural atresia or stenosis. In the case of aural atresia, occult lesions can develop in the remnant of the external auditory canal or in the middle ear cleft.

### DIAGNOSIS

In the verbal child, the diagnosis of AOM is usually straightforward, with the patient complaining of ear pain (otalgia), fever, and an antecedent upper respiratory infection. The affected TM appears markedly erythematous or injected and often is opaque and thickened, obscuring visualization of the bones (ossicles) in the middle ear. The TM is “full” or bulging. An air-fluid level also may be seen.

Preverbal children and infants are a greater diagnostic challenge. Symptoms often are nonspecific, such as irritability, poor sleeping, and decreased appetite. Unaccustomed ear tugging or rubbing can be seen, but is inconsistently predictive. The cartilaginous external ear canal of an infant can be tortuous, making visualization of the TM difficult. Accurate assessment of the color and opacity of the TM is particularly hard in an apprehensive, febrile, crying toddler, sometimes requiring the use of additional diagnostic procedures, such as tympanometry.

Tympanometry is an objective test that measures the mobility, or compliance, of the TM and the middle ear. It does not evaluate hearing but is sensitive in detecting MEE. Compliance is measured via a probe tone presented to the sealed canal. Compliance is reported as high (≥0.5 mL), intermediate (<0.5 mL but >0.2 mL), or low (<0.2 mL). A tympanogram also measures middle ear pressure, which is categorized as normal, negative, or positive and represented as a curve, or peak, on a graph (reported in mm H2O). A peak between –100 mm H2O and +50 mm H2O is normal. A peak less than –100 mm H2O signifies high negative pressure, and a peak at greater than +50 mm H2O is consistent with high positive pressure. A sharp tympanometric peak suggests a low likelihood of MEE, a rounded one suggests a greater likelihood of fluid. A flat one is highly suggestive of MEE. Tympanometry alone cannot distinguish between an acute infection of the middle ear and an uninfamed effusion (OME). The highly compliant cartilaginous walls of the external canal of infants younger than 6 months old can expand when air pressure is increased in the canal in this age group and result in a falsely normal reading.

All examinations of the middle ear should include a determination of the mobility of the TM via pneumatic otoscopy. Normal mobility is shown when positive pressure is applied in the external auditory canal and the TM moves rapidly inward (away from the examiner). When the bulb is released, negative pressure is created, and the TM moves outward (toward the examiner). Mobility of the TM is reduced greatly by fluid or pus in the middle ear.
Tympanocentesis, needle aspiration of the middle air space, is a diagnostic and therapeutic procedure. It may be indicated to determine the causative organism of a middle ear effusion in infants younger than 6 weeks old, children who are immunocompromised, or children who are toxic or have signs of invasive bacterial disease. Presence of supplicative complications also may warrant this procedure. Tympanocentesis may be indicated if the diagnosis of AOM is unclear or if the patient is not improving after appropriate therapy. Potential complications include chronic perforation of the TM, facial nerve paralysis, dislocation of the incudostapedial joint, and bleeding from an exposed jugular bulb. The risks and benefits must be weighed carefully when considering the procedure.

Myringotomy is an incision into the TM to drain middle ear fluid acutely and usually is preceded by tympanocentesis. Indications for myringotomy include treatment for complications of purulent OM, such as mastoiditis, labyrinthitis, or facial nerve paralysis. Severe otalgia may be relieved by myringotomy.

Most cases of OME are identified when children return for follow-up after a recent AOM. OME also may be an incidental finding. Residual fluid in the middle ear may represent incomplete resolution of the acute infection or the natural course of an uncomplicated treated infection. Distinguishing between these two entities can be particularly challenging (Table 61-2). Figure 61-5 shows the persistence of MEE after a diagnosis of AOM. Approximately 40% of children continue to have an effusion at 4 weeks, 20% at 8 weeks, and 10% at 12 weeks. Frequently these patients are asymptomatic (e.g., no fever, normal hearing), but occasionally children continue to complain of unilateral hearing loss.

The TM of OME is retracted (as indicated by a prominent short process of the malleus) with minimal or no signs of inflammation. Pneumatic otoscopy reveals diminished-to-absent mobility of the TM. In equivocal cases, tympanometry can be a useful adjunct. The positive predictive value of an abnormal or flat tympanogram is 49% to 99%, which means that half the number of ears with abnormal tests may have OME. A normal tympanogram more accurately predicts absence of an effusion.

The development of an intratemporal complication of acute or chronic ear disease should be suspected by clinical signs and symptoms. Acute mastoiditis usually presents with fever and postauricular swelling, erythema, and tenderness to palpation. Common symptoms associated with AOM also may be present, such as otalgia and hearing loss. The pinna is displaced downward and outward, with swelling or sagging of the posterosuperior canal wall (Fig. 61-6). Acute otitis media may be seen as the result of a perforation in the TM. The canal is filled with purulent material and debris. Visualization of the TM can be difficult, but it is important to differentiate between AOM with a spontaneous perforation and mastoiditis. In such cases, assistance from an otolaryngologist may be needed. Rarely a postauricular fistula may develop from the mastoid area, or a fluctuant subperiosteal abscess may be palpated in the same region. Children who have persistent retroauricular pain and a history of recurrent OM may not have other signs of an acute infection but may have clinically significant disease.

The diagnosis of acute and chronic mastoiditis is confirmed by computed tomography of the temporal bone. Even in the absence of fluid in the middle ear by otoscopy, any haziness, distortion, or destruction of the bony trabeculae in the mastoid cavity by computed tomography indicates the presence of mastoiditis. These findings should prompt the generalist to seek consultation from an otolaryngologist for assistance in the evaluation and treatment of this condition, especially because a surgical procedure may be indicated. Other indications for consultation include a history of recurrent or chronic OM, especially if the condition is bilateral, and a concern that the child’s hearing, speech, or language development is abnormal.

The presence of otorhea or blood in the external canal suggests the possibility of an acute perforation of the TM or chronic suppurative OM with or without cholesteatoma. The challenge for the clinician is to distinguish these two entities from simple OE. Specific features of the history and physical examination associated with each condition are summarized in Table 61-3.

Patients with acute OE complain of pruritus, the acute onset of unilateral ear pain that is worsened by applying pressure to the tragus or with movement of the pinna, and sometimes a sense of aural stuffiness or fullness. Significant

| Table 61-2. Clinical Characteristics of Acute Otitis Media (AOM) and Otitis Media with Effusion (OME) |
| Signs and symptoms                      | AOM | OME |
|----------------------------------------|-----|-----|
| Upper respiratory infection            | +   | +/− |
| Otitalgia, irritability                | −   | −   |
| Fever                                  | +   | −   |
| Otorrhea                               | +/− | +   |
| Hearing impairment                     | +/− | + (usually) |
| Middle ear effusion                    | +   | +   |
| Opaque TM                              | +   | − (air-fluid level) |
| Bulging TM                             | +   | −   |
| Retracted TM                           | −   | +/− |
| Decreased mobility of TM               | +   | +   |

TM, tympanic membrane.
edema of the external canal can cause hearing loss. Otalgia may range from mild discomfort to severe, unrelenting pain requiring narcotic analgesics. Occasionally, normal jaw movements (chewing) exacerbate the pain.

In mild acute disease, the external canal is minimally edematous and erythematous, with or without serous or purulent drainage filling the lumen (Box 61-1). As the infection progresses, the exudate becomes more profuse and malodorous. Often the pinna and tragus are exquisitely tender to palpation. Sagging of the superior canal, periauricular edema, and preauricular and postauricular adenopathy develop with more severe disease. Extension of the infection to surrounding soft tissues results in obstruction of the ear canal with or without a circumscribed cellulitis. Involvement of the parotid gland, mastoid bone, and infratemporal fossa are other rare complications.

Traumatic perforations of the TM usually are accompanied by considerable pain and bleeding from the ear that stops spontaneously. A mild conductive hearing loss may be observed on audiometric testing (10 to 35 dB), although young children rarely complain of this consequence of injury. An isolated accidental injury to the TM must be differentiated from injury caused by physical abuse. A suspicion of nonaccidental trauma or the presence of other stigmata suggesting child abuse mandates an immediate referral to child protective services and the performance of long bone radiographs. Regardless of the etiology, significant complications can accompany traumatic perforations. The presence of a significant sensory hearing loss or severe vertigo indicates a perilymphatic fluid leak into the middle ear space secondary to a transient subluxation of the stapes into the inner ear vestibule. These injuries require a referral to an otolaryngologist for further evaluation and treatment.

Congenital cholesteatoma usually is discovered incidentally on routine otoscopic examination of an asymptomatic child by the general pediatrician. It appears as a pearly white mass medial to an intact TM, in the anterior-superior quadrant (Fig. 61-7). A cholesteatoma can be diagnosed during the routine evaluation of a hearing deficit; unilateral

| Feature                  | Otitis Externa | Acute Otitis Media with Perforation | Chronic Suppurative Otitis Media |
|--------------------------|----------------|------------------------------------|---------------------------------|
| **Historical Features**  |                |                                    |                                 |
| Pain                     | Present        | Present before perforation          | Rare                            |
| Fever                    | Uncommon       | Usually present                     | Rare                            |
| Season                   | Summer         | Winter/spring                       | Variable                        |
| Associations             | Swimming       | URI, history of otitis media        | Perforation of TM, recurrent OM |
| **Physical Examination** |                |                                    |                                 |
| External canal           | Edema, erythematous, tender, tragus/pinna tender | Debris in canal, edema/erythema depend on degree of secondary OE Perforation; red, opaque/purulent drainage behind TM | Chronic inflammatory changes, debris in canal Perforation secondary to OM or tympanostomy tubes |
| Tympanic membrane        | Usually normal |                                    |                                 |
| **Microbiology**         |                |                                    |                                 |
| *Pseudomonas aeruginosa* |                |                                    |                                 |
| *Staphylococcus aureus*  |                |                                    |                                 |
| *Haemophilus influenzae* |                |                                    |                                 |

OE, otitis externa; OM, otitis media; TM, tympanic membrane; URI, upper respiratory infection.
hearing loss is detected by audiometric testing. Examination with an otomicroscope by a pediatric otolaryngologist confirms the diagnosis.

Children with acquired cholesteatoma usually have a long history of AOM or chronic suppurative OM (see Fig. 61-7). Occasionally the child is asymptomatic and is noted only to have a retraction pocket in the posterior-superior portion of the TM. More commonly, acquired cholesteatomas are associated with a chronically draining ear and hearing loss or vertigo. Mild hearing loss and other subtle symptoms, such as tinnitus, may be missed in children. Fever is not a sign of cholesteatoma and if present requires search for another intratemporal or intracranial suppurative complication of middle ear disease, especially when accompanied by otalgia.

Pneumatic otoscopy is essential to distinguish between tympanosclerosis and a middle ear congenital cholesteatoma. The TM should move independently of a middle ear mass when pressure is applied and released in the external canal. A white tympanosclerotic plaque moves with the rest of the TM.

Because direct visualization of either type of cholesteatoma is often difficult, general pediatricians should maintain a high index of suspicion when a TM appears abnormal or if a defect in the TM is associated with pearly white squamous debris. Adjunctive tests, such as tympanograms and audiograms, are not diagnostic and may be normal even when a lesion is present. All children suspected of having a cholesteatoma should be referred to a pediatric otolaryngologist. High-resolution computed tomography of the temporal bone is helpful when the condition is suspected and often is necessary to delineate the extent of the lesion. The differential diagnosis of hearing loss and indicated diagnostic studies for each condition are discussed in Chapter 67.

**MANAGEMENT**

Therapeutic goals for infants and young children with OM are to treat acute infections appropriately and prevent recurrent and chronic middle ear disease, reducing the morbidity and mortality associated with intratemporal and intracranial complications. Another goal is to prevent associated speech and language delay.

Most cases of AOM and OME can be expected to resolve spontaneously. Short-term resolution of AOM most likely reflects the host immune response and local inflammatory reaction. The primary benefit of antibiotic treatment is reduction of the incidence of delayed suppurative complications. Selection of appropriate empirical therapy should be based on its effectiveness against the most predominant organisms.

Despite the emergence of resistant *S. pneumoniae*, amoxicillin continues to be standard first-line therapy for uncomplicated AOM. This agent is well tolerated by most children, is inexpensive, and maintains an excellent safety record. A dose of 80 to 90 mg/kg/day three times a day for 5 to 10 days is now recommended as empirical initial therapy. The 5-day course can be considered for children 6 years old and older. High-dose amoxicillin is recommended, since the likelihood of highly resistant bacteria is increased in children younger than 24 months of age, children who attend day care, and children who have been treated recently with β-lactam drugs. Erythromycin-sulfisoxazole acetyl, clarithromycin, and azithromycin dehydrate are alternatives for children who are allergic to penicillin (Type I reaction).

Failure while on therapy for at least 3 days or relapse within 2 weeks of treatment requires coverage against
**SECTION 3**

Disorders of the Head and Neck

β-lactamase–producing *H. influenzae* and resistant strains of *S. pneumoniae*. Antibiotics currently recommended for second-line therapy are those that are effective against these organisms while achieving adequate concentrations in the middle ear. High-dose amoxicillin-clavulanate (90 mg/kg/day of amoxicillin component, with 6.4 mg/kg/day of clavulanate in two divided doses) and many of the oral cephalosporins (cefuroxime axetil, cefpodoxime, and cefdinir) are second-line therapies. According to a Centers for Disease Control and Prevention working group on drug-resistant *S. pneumoniae*, clindamycin might be effective against pneumococci that are resistant to β-lactam antibiotics. A 3-day course of ceftriaxone (50 mg/kg/day), either intravenously or intramuscularly, can be used in children vomiting or in other clinical situations that preclude administration of oral antibiotics. A substantial number of pneumococcal isolates are resistant to erythromycin. Therefore, erythromycin-sulfisoxazole is not optimal second-line therapy for AOM. The use of trimethoprim-sulfamethoxazole as an alternative agent is controversial because pneumococcal surveillance studies indicate that approximately 25% of isolates are resistant.

Failure of second-line therapy is an indication for tympanocentesis to confirm the diagnosis of OM and to obtain a specimen for culture. Before this procedure, patient compliance must be ascertained. If good compliance can be confirmed, the infection is most likely due to a resistant strain of pneumococci. The role of intramuscular ceftriaxone in this setting has been investigated and is advocated by most experts. Others recommend referral to an otolaryngologist, however, in the case of recurrent disease for placement of tympanostomy tubes. Adenoidectomy is reserved for children older than 4 years of age who need tympanostomy tubes a second time.

Children with acute inflammation of the middle ear may benefit from symptomatic therapy to reduce pain. Oral acetaminophen and ibuprofen are acceptable analgesics for most cases of mild-to-moderate pain. Topical anesthetics include benzocaine formulations (Auralgan and Americaine). Some families may use home remedies for the treatment of ear discomfort, such as placing a clove of garlic or warm olive oil in the external auditory canal. The role of antihistamines and decongestants in the treatment of acute infections has not been well supported. Although these medications may improve symptoms associated with a viral upper respiratory tract infection, studies have not shown that they eradicate middle ear fluid any faster. Corticosteroids also have no role in the treatment of AOM, although their use remains controversial for long-standing MEE.

Longitudinal studies of OME show that most effusions resolve spontaneously within 3 months. Specific recommendations have been developed for young children age 1 through 3 years who are otherwise healthy (e.g., with no craniofacial or neurologic abnormalities or sensory deficits). Highlights from a U.S. consensus panel in 1994 include a short period of observation for most children, a hearing evaluation if the effusion is bilateral and present for more than 3 months, control of environmental risk factors such as exposure to tobacco smoke, and antimicrobial therapy for the few children whose effusions persist. Because most middle ear effusions resolve without treatment, the general pediatrician should reexamine the asymptomatic child 6 weeks from initial diagnosis with pneumatic otoscopy. If the effusion persists at follow-up, its presence should be confirmed by tympanometry, followed by oral antibiotic therapy for 10 days. For young children that continue to have OME 3 months after the initial diagnosis, a hearing evaluation should be performed and a referral made for possible myringotomy with tympanostomy tube placement, especially if the patient is found to have a hearing deficit. Steroid medications, antihistamine/decongestant therapy, and adenoidectomy with or without tonsillectomy are not recommended for the treatment of OME in otherwise healthy children 1 through 3 years old. The reader is referred to the practice guideline published by the American Academy of Pediatrics for further details regarding the management of OME in young children. Adenoidectomy has been shown to be of value in children older than 4 years of age with bilateral effusion.

Antimicrobial prophylaxis should be reserved for patients with a history of recurrent OM and administered during the months when upper respiratory infections are the most prevalent. Current treatment options are a single daily dose of amoxicillin, 20 mg/kg, and sulfisoxazole, 75 mg/kg/day divided in two doses. Children who continue to have episodes of AOM despite medical prophylaxis should be referred to an otolaryngologist for further evaluation and consideration for tympanostomy tube placement. The administration of prophylactic antibiotics for the prevention of MEE is not advisable because of bacterial resistance.

Parental education and the administration of specific immunizations can play a major role in the prevention of AOM. As a part of anticipatory guidance, the generalist always should review the role of passive tobacco smoke exposure, bottle-feeding instead of breast-feeding, and pacifier use in the development of AOM. Additional interventions include providing the influenza vaccine for children who are particularly susceptible to an acute infection during the winter months and administering the heptavalent conjugate pneumococcal vaccine routinely at well-child visits. Data report that the pneumococcal vaccine reduces the incidence of AOM by 6% to 8%.

The management of acute mastoiditis may be medical or surgical, depending on the extent of bony destruction. Mastoiditis is an indication for referral to an otolaryngologist. For uncomplicated mastoiditis, intravenous antibiotic therapy is the mainstay of treatment. A myringotomy also may be necessary to decompress the middle ear and is important to provide a specimen for culture and sensitivity. If there is evidence of significant destruction of the bone or no improvement while on intravenous medication, a mastoidectomy is indicated. The goal is to clean out and drain the mastoid air cell system into the middle ear. In addition, a tympanostomy tube is inserted to allow further drainage and ventilation of the middle ear.

Mild-to-moderate OE can be managed by the general pediatrician with the primary goals of therapy aimed at controlling the inflammation and infection. Although difficult and occasionally painful for the patient, the ear canal should be cleansed gently of the debris from the entire length of the external auditory canal. This cleansing
may be accomplished by gentle irrigation with warmed saline or 2.5% acetic acid solution. Gentle suctioning adequately clears the canal. Most cases also require the installation of ototopical drops into the ear canal or onto a wick if there is significant edema. Classes of ototopical medications include steroids, acidifying agents, antiseptics (alcohol), and antibiotics. Many of the most commonly used products contain polymyxin B or E, neomycin, and hydrocortisone. The usual dosage schedule is three to four times a day for 7 to 10 days. If a wick is placed, it should be removed within 24 to 72 hours. Newer fluoroquinolone topical antibiotics contain ciprofloxacin/hydrocortisone and ofloxacin. Patients who do not respond well to initial treatment should be switched to drops containing ciprofloxacin or tobramycin. Otomycosis requires treatment with antifungal drops, such as clotrimazole.

Swimming should be prohibited during the course of treatment. Further episodes of OE can be prevented through the use of earplugs while swimming. Other recommendations include the use of acidic solutions in the ear canal after exposure to water, drying the ear with a hair dryer set on low heat from 1 foot away for 60 seconds, and avoiding manipulation of the ear canal with cotton swabs and other objects to avoid irritation and maceration of the skin. Cases of OE that are persistent or recurrent warrant a culture of the ear discharge and the assistance of a specialist to evaluate the child for cholesteatoma or another undiagnosed condition.

Traumatic perforations of the TM should be managed in consultation with an otolaryngologist. Although small perforations often heal spontaneously within a few weeks after the injury, larger perforations may persist. Consultation with a specialist is essential to evaluate the extent of the defect, any associated complications such as involvement of the ossicular chain, and the timing of any surgical procedures.

The management of cholesteatoma is surgical and requires the expertise of a pediatric otolaryngologist. The goals of surgery are twofold: (1) to eradicate the squamous epithelium from the middle ear, mastoid, or both and (2) to preserve or restore hearing. The initial surgical procedure is aimed at direct removal of the squamous epithelium. Removal is done either through the ear canal or by a postauricular approach, depending on the size of the external auditory canal and location of the lesion. Commonly, with acquired cholesteatoma, mastoidectomy also is necessary to remove the entire lesion. Other surgical procedures may include ossicular chain reconstruction. Hearing aids are often necessary for children who are awaiting elective surgery and have significant bilateral hearing loss or who are not candidates for ossicular chain reconstruction.

OUTCOME

Children with uncomplicated AOM generally have a favorable outcome depending on their age at diagnosis, frequency of infections, and length of time for fluid in the middle ear to resolve. Severe adverse sequelae of treated OM are rare. The most common adverse outcomes include hearing impairment, speech delay, and significant TM perforation.

Various studies indicate that recurrent disease or prolonged MEE in young infants may affect hearing and normal speech and language development. Referral to a pediatric otolaryngologist for tympanostomy tube placement is essential for children with severe-to-profound hearing loss or children with abnormal speech development. Other children should be evaluated on a case-by-case basis to assess whether medical prophylaxis (antibiotics) should be initiated before the referral. For children who require a second placement of tympanostomy tubes, adenoidectomy without a tonsillectomy has been shown to be an effective additional procedure to prevent recurrent disease.

The outcome of intracranial complications of AOM, such as meningitis, is variable, ranging from full recovery with no long-term sequelae, to mild hearing loss with minimal associated neurologic deficits, to recurrent seizures, to developmental delay. Reported mortality rates in severely affected children range from 0% to 20%. The outcome of intratemporal complications depends on the particular condition being considered. Mastoiditis usually has a favorable outcome if the infection is controlled acutely.

The outcome of TM perforation depends on the size and location of the defect and any associated complications. Most lesions heal spontaneously, however. After an uncomplicated TM perforation, a mild conductive hearing loss may be observed on audiometric testing. Destruction of the margin of the TM may lead to additional complications, such as secondary cholesteatoma formation.

In children, cholesteatoma is thought to be an aggressive disease that, if not discovered early, may have a poor outcome. Because extensive disease usually is found at the time of surgery and there are higher rates of residual and recurrent disease after surgery, the outcome is variable.

FOLLOW-UP

General recommendations for follow-up of AOM are based on several factors, the most important being the amount of time for the accompanying MEE to resolve. Because approximately 80% of effusions resolve by 8 weeks after the acute infection, most practice guidelines recommend reexamination of the TM within 6 to 8 weeks of the initial diagnosis. Audiologic testing usually is reserved for children with a history of recurrent OM, chronic MEE, or suspected speech delay. An evaluation by a speech therapist is warranted for abnormal language development, especially during the first 4 years of life.

A child diagnosed and treated for mastoiditis should continue to be screened for further episodes of OM in consultation with an otolaryngologist. Children with traumatic perforations should be screened initially for a hearing deficit and then followed until complete resolution of the defect has been documented. All children with cholesteatoma should continue to be followed by a subspecialist so that recurrent lesions can be prevented and controlled.
Disorders of the Eye
Michael C. Struck

The ocular system is subclassified by the ocular structures. External disorders are conditions that affect the orbit, lids, or lacrimal system. Disorders of the anterior segment are conditions that affect the cornea, iris, or lens or lead to glaucoma. Posterior segment disorders involve the retina, retinal vasculature, vitreous humor, and optic nerves. Neuroophthalmologic disorders, inflammatory conditions, and trauma often involve multiple structures. Vision development, nystagmus, amblyopia, and ocular torticollis are special ocular conditions that affect pediatric patients.

DEFINITION
Disorders of the eye may be congenital, developmental, hereditary, infectious, immune-mediated, traumatic, neoplastic, and neurologic conditions. Ophthalmic manifestations may be the heralding sign in a spectrum of systemic conditions, such as Stevens-Johnson syndrome, Crouzon’s disease, or albinism, in which the eye may be the most significantly involved structure. An understanding of both neurologic function and the basic function and anatomy of the eye, periocular structures, and orbit is essential when developing an assessment and treatment plan.

Conditions that affect the eye or its function often are subdivided by the particular ocular structure involved. Different disorders can lead to a final common pathway of dysfunction. Treatment of the presenting condition requires evaluation of a broad differential diagnosis and the effect of the condition on the developing visual system.