common bacterial causes of acute otitis media, and concern is increasing with MRSA as an emerging etiologic agent. Viruses, such as those mentioned above, have been recovered either alone or with bacteria in 17–40% of cases.

CLINICAL MANIFESTATIONS Fluid in the middle ear is typically demonstrated or confirmed with pneumatic otoscopy. In the absence of fluid, the TM moves visibly with the application of positive and negative pressure, but this movement is dampened when fluid is present. With bacterial infection, the TM can also be erythematous, bulging, or retracted and occasionally can perforate spontaneously. The signs and symptoms accompanying infection can be local or systemic, including otalgia, otorrhea, diminished hearing, and fever. Erythema of the TM is often evident but is nonspecific as it frequently is seen in association with inflammation of the upper respiratory mucosa. Other signs and symptoms occasionally reported include vertigo, nystagmus, and tinnitus.

TREATMENT ACUTE OTITIS MEDIA

There has been considerable debate on the usefulness of antibiotics for the treatment of acute otitis media. A higher proportion of treated than untreated patients are free of illness 3-5 days after diagnosis. The difficulty of predicting which patients will benefit from antibiotic therapy has led to different approaches. In the Netherlands, for instance, physicians typically manage acute otitis media with initial observation, administering anti-inflammatory agents for aggressive pain management and reserving antibiotics for high-risk patients, patients with complicated disease, or patients whose condition does not improve after 48-72 h. In contrast, many experts in the United States continue to recommend antibiotic therapy for children <6 months old in light of the higher frequency of secondary complications in this young and functionally immunocompromised population. However, observation without antimicrobial therapy is now the recommended option in the United States for acute otitis media in children >2 years of age and for mild to moderate disease without middle-ear effusion in children 6 months to 2 years of age. Treatment is typically indicated for patients <6 months old; for children 6 months to 2 years old who have middle-ear effusion and signs/symptoms of middle-ear inflammation; for all patients >2 years old who have bilateral disease, TM perforation, immunocompromise, or emesis; and for any patient who has severe symptoms, including a fever ≥39°C or moderate to severe otalgia (Table 44-2).

Because most studies of the etiologic agents of acute otitis media consistently document similar pathogen profiles, therapy is generally empirical except in those few cases in which tympanocentesis is warranted—e.g., cases refractory to therapy and cases in patients who are severely ill or immunodeficient. Despite resistance to penicillin and amoxicillin in roughly one-quarter of *S. pneumoniae* isolates, one-third of *H. influenzae* isolates, and nearly all *M. catarrha-lis* isolates, outcome studies continue to find that amoxicillin is as successful as any other agent, and it remains the drug of first choice in recommendations from multiple sources (Table 44-2). Therapy for uncomplicated acute otitis media typically is administered for 5–7 days to patients ≥6 years old; longer courses (e.g., 10 days) should be reserved for patients with severe disease, in whom short-course therapy may be inadequate.

A switch in regimen is recommended if there is no clinical improvement by the third day of therapy, given the possibility of infection with a β -lactamase-producing strain of H. influenzae or M. catarrhalis or with a strain of penicillin-resistant S. pneumoniae. Decongestants and antihistamines are frequently used as adjunctive agents to reduce congestion and relieve obstruction of the eustachian tube, but clinical trials have yielded no significant evidence of benefit with either class of agents.

Recurrent Acute Otitis Media Recurrent acute otitis media (more than three episodes within 6 months or four episodes within 12 months)

generally is due to relapse or reinfection, although data indicate that the majority of early recurrences are new infections. In general, the same pathogens responsible for acute otitis media cause recurrent disease; even so, the recommended treatment consists of antibiotics active against β -lactamase-producing organisms. Antibiotic prophylaxis (e.g., with trimethoprim-sulfamethoxazole [TMP-SMX] or amoxicillin) can reduce recurrences in patients with recurrent acute otitis media by an average of one episode per year, but this benefit is small compared with the high likelihood of colonization with antibiotic-resistant pathogens. Other approaches, including placement of tympanostomy tubes, adenoidectomy, and tonsillectomy plus adenoidectomy, are of questionable overall value in light of the relatively small benefit compared with the potential for complications.

Serous Otitis Media In serous otitis media (otitis media with effusion), fluid is present in the middle ear for an extended period in the absence of signs and symptoms of infection. In general, acute effusions are self-limited; most resolve in 2-4 weeks. In some cases, however (in particular after an episode of acute otitis media), effusions can persist for months. These chronic effusions are often associated with significant hearing loss in the affected ear. The great majority of cases of otitis media with effusion resolve spontaneously within 3 months without antibiotic therapy. Antibiotic therapy or myringotomy with insertion of tympanostomy tubes typically is reserved for patients in whom bilateral effusion (1) has persisted for at least 3 months and (2) is associated with significant bilateral hearing loss. With this conservative approach and the application of strict diagnostic criteria for acute otitis media and otitis media with effusion, it is estimated that 6-8 million courses of antibiotics could be avoided each year in the United States.

Chronic Otitis Media Chronic suppurative otitis media is characterized by persistent or recurrent purulent otorrhea in the setting of TM perforation. Usually, there is also some degree of conductive hearing loss. This condition can be categorized as active or inactive. Inactive disease is characterized by a central perforation of the TM, which allows drainage of purulent fluid from the middle ear. When the perforation is more peripheral, squamous epithelium from the auditory canal may invade the middle ear through the perforation, forming a mass of keratinaceous debris (cholesteatoma) at the site of invasion. This mass can enlarge and has the potential to erode bone and promote further infection, which can lead to meningitis, brain abscess, or paralysis of cranial nerve VII. Treatment of chronic active otitis media is surgical; mastoidectomy, myringoplasty, and tympanoplasty can be performed as outpatient surgical procedures, with an overall success rate of ~80%. Chronic inactive otitis media is more difficult to cure, usually requiring repeated courses of topical antibiotic drops during periods of drainage. Systemic antibiotics may offer better cure rates, but their role in the treatment of this condition remains unclear.

Mastoiditis Acute mastoiditis was relatively common among children before the introduction of antibiotics. Because the mastoid air cells connect with the middle ear, the process of fluid collection and infection is usually the same in the mastoid as in the middle ear. Early and frequent treatment of acute otitis media is most likely the reason that the incidence of acute mastoiditis has declined to only 1.2–2.0 cases per 100,000 person-years in countries with high prescribing rates for acute otitis media.

In countries such as the Netherlands, where antibiotics are used sparingly for acute otitis media, the incidence rate of acute mastoiditis is roughly twice that in countries like the United States. However, neighboring Denmark has a rate of acute mastoiditis similar to that in the Netherlands but an antibiotic-prescribing rate for acute otitis media more similar to that in the United States.

In typical acute mastoiditis, purulent exudate collects in the mastoid air cells (Fig. 44-1), producing pressure that may result in erosion of the surrounding bone and formation of abscess-like cavities that are