Initial treatment of otitis externa involves removal of debris from the external ear canal, aggressive pain control, use of topical medications, acidification of the ear canal, and control of predisposing factors. Debridement of the external ear canal allows for removal of infectious material and better penetration of topical medications. In many cases edema of the ear canal will prevent proper penetration of the medicated drop down into the canal. In this situation, placement of a cotton wick directly into the ear canal for several days will facilitate delivery of the medication (1).

Currently, recommended topical preparations consist of antibiotics and steroid combinations. Quinolone antibiotic preparations may have broader microbial coverage and a low risk of contact dermatitis, and are considered first line. Caution should be used when treating with neomycin-containing topical preparations due to a potential for this agent to cause contact sensitivity and in turn lead to worsening of symptoms. Neomycin-containing preparations also have a risk—albeit low—of causing permanent sensorineural hearing loss and should be used with caution in patients with perforated tympanic membranes or tympanostomy tubes. Acetic acid may also be considered as a treatment, but is generally less effective in the long term when compared to antibiotic drops (6). When treating immunocompromised patients, or if otitis externa infection has spread beyond the ear canal, consideration should be given to the use of systemic antibiotics as well, though these are not recommended in otherwise uncomplicated patients. The choice of antibiotics should be based on their antipseudomonal and antistaphylococcal properties (1).

**Chronic Otitis Externa**

COE is a persistent inflammatory disorder of the ear canal usually caused by repeated mechanical debridement or water exposure. Other potential causes are allergic, contact dermatitis, or dermatologic disorders. Chronic inflammation may lead to development of granulation. The treatment of COE involves debridement, avoidance of ear canal manipulation, elimination of the offending agent, and topical corticosteroids. Tacrolimus has also shown promise in the treatment of refractory COE (7). Regular flushing of the ear canal with a mild acidic solution, such as acetic acid or vinegar and distilled water, can also help to eradicate infection and keep the canal free of debris (7,8).

**Otomycosis**

Otomycosis is a fungal infection of the external ear canal. It comprises roughly 10% of all cases of otitis externa, and is more common in geographic locations with a warm and humid climate, in patients following long-term topical antibiotic therapy, and in patients with diabetes, HIV, or other immunocompromising conditions (9). The ear canal will often have cellulitis and edema on otoscopic examination. The canal debris may have a cheese-like or grayish appearance with visualized fungal hyphae. *Aspergillus* species and *Candida* species are the
most common pathogens (10). Treatment consists of debride-
ment, acidification, and drying of the ear canal. For candidal
infections, topical antifungal therapy may also be effective.

Necrotizing Otitis Externa

Malignant or necrotizing otitis externa (MOE) is an aggres-
sive infection that begins as otitis externa but spreads through
surrounding tissues toward the skull base. It is seen predomi-
nantly in the elderly, diabetic, or immunocompromised
patient. P. aeruginosa is the most common causative pathogen;
however, staphylococcal species are also known to cause the
infection (11). Fungal causes of MOE are less common, with
Aspergillus species the predominating pathogen (12).

MOE initially presents with symptoms and signs of AOE.
Subsequently, it may progress to temporal bone osteomyelitis
and affect adjacent cranial nerves (VII to XII), blood vessels,
and soft tissue. If not treated aggressively, the infection can
expand intracranially leading to neurologic symptoms. On
otoscopic examination, granulation tissue is classically seen at
the bony–cartilaginous junction (11–13). A raised erythrocyte
sedimentation rate and abnormal computed tomography (CT)
or magnetic resonance imaging (MRI) scan help to confirm the
clinical diagnosis. Other imaging techniques that assist in
diagnosis include gallium scan, indium-labeled leukocyte scan,
technetium bone scan, and single-photon emission tomographs
(12,14). Patients will require treatment with systemic antibi-
otics that cover pseudomonal and staphylococcal coverage,
including methicillin-resistant Staphylococcus aureus (11,12).

Furunculosis (Ear Canal Abscess)

Furunculosis is a localized infection of the ear canal that is
usually caused by an infected hair follicle. It may present with
otalgia, otorrhea, and localized tenderness. A tender, often
fluctuant nodule within the lateral ear canal can be identified
on the examination. The most common pathogen is S. aureus.
The treatment includes application of heat, incision and drain-
age of the infected area, and systemic antibiotic treatment with
staphylococcal coverage (15).

Acute Otitis Media

Acute otitis media is an inflammation of the middle ear, which
is generally characterized by the rapid onset of otalgia, aural
fullness, and occasionally fever. In the pediatric patient, more
common signs are irritability, sleeplessness, and pulling at the
affected ear. On pneumatic otoscopy, the tympanic membrane
will have a red, opaque, and bulging appearance with decreased
mobility due to the accumulation of purulent fluid in the middle
ear space. Additionally, the tympanic membrane may rupture,
so that patients will present with otorrhea (16,17).

Predominant pathogens in AOM are Streptococcus pneu-
moniae, Haemophilus influenzae, and Moraxella catarrhalis
(18). Observation for 24 to 48 hours in the case of a nonsevere
illness in an otherwise healthy individual greater than 6 months
of age is an initial option, with 60% resolving within 24 hours
without antibiotic treatment. If symptoms persist, antimicro-
bial therapy should be initiated. Amoxicillin is recommended
for initial treatment of acute otitis media, at a recommended
dose of 80 to 90 mg/kg/d. In the case of penicillin allergy,
azithromycin, clarithromycin, erythromycin–sulfisoxazole,
or trimethoprim–sulfamethoxazole could be used. Due to the
increased incidence of β-lactamase–producing organisms, the
bacterial coverage should be expanded if there is no improve-
ment within 48 to 72 hours. In very rare cases, if pain or fever
is excessive, immediate tympanocentesis or myringotomy may
be required, taking care to send purulent fluid for culture in
this case (18,19).

Otitis media with effusion is the presence of fluid in the
middle ear without signs or symptoms of acute ear infection
and should be distinguished from acute otitis media. Otitis
media with effusion often occurs as a result of eustachian tube
dysfunction, or middle ear inflammation following acute infec-
tion. It is most common in the pediatric population between
the ages of 6 months and 4 years, although it may occur at
any age. On pneumatic otoscopy, the tympanic membrane is
often retracted, will have decreased mobility, and an air-fluid
level or bubbles are often visualized. Patients often report a
decrease in hearing. Otitis media with effusion is often self-
limited and is likely to resolve spontaneously within 3 months.
If it persists, hearing testing is recommended, particularly in
children with language delay, learning problems, or suspicion
of significant hearing loss (17,20). In individuals with hear-
ing loss and persistent middle ear effusion for greater than
3 months, myringotomy with tympanostomy tube insertion
should be considered (21). Medical treatment, such as decon-
gestants, has not been shown to be effective in the treatment
of middle ear effusion. In an adult presenting with a unilat-
eral middle ear effusion, an examination of the nasopharynx
should be performed to rule out the possibility of a nasopha-
ryngeal mass causing obstruction of the eustachian tube.

Chronic Otitis Media

Chronic otitis media is diagnosed when infection persists for
more than 1 to 3 months. It may present as chronic suppura-
tive otitis media (CSOM), which is characterized by persistent
bacterial infection and drainage from the ear, or as chronic otitis media with effusion (COME), which results from unresolved inflammation of the middle ear and persistent middle ear secretions with an intact tympanic membrane. Chronic otitis media may be associated with cholesteatoma, which is a keratin cyst that forms from an accumulation of squamous debris in the middle ear with potential for growth and erosion of surrounding structures (22).

Patients with CSOM will present with hearing loss, painless purulent otorrhea, and a chronic tympanic membrane perforation. Evaluation includes visual examination, bacterial culture, and radiographic imaging. Gram-negative and anaerobic organisms are usually seen on cultures, with *P. aeruginosa* being a predominant organism. Temporal bone CT scan allows evaluation of the extent of disease and reveals potential complications. Medical treatment of CSOM consists of topical debridement, along with topical and systemic antibiotics. Topical drops often consist of antibiotic and steroid combinations (23). Ciprofloxacin is recommended for systemic use; however, it cannot be given to children under 17 years of age. Surgical treatment is performed for eradication of the infection and reconstruction of the middle ear (22).

COME is characterized by persistent hearing loss and a middle ear space filled with thick mucus. Chronic inflammation of the middle ear often begins with obstruction of the eustachian tube. The resulting negative pressure in the middle ear leads to collection of transudate. Secondary to chronic inflammation, the middle ear lining becomes hyperplastic and produces further mucous. On examination, the tympanic membrane is intact and has a thickened, opaque appearance. On pneumatic otoscopy, the tympanic membrane does not move. As the disease progresses the tympanic membrane starts to retract and drape over the ossicles. Nasal obstruction and sinus disease may contribute to Eustachian tube insufficiency and lead to middle ear fluid accumulation. Treatment of COME consists of fluid drainage, which is accomplished by myringotomy with ventilation tube insertion. Treating sinus disease and relieving nasal obstruction may improve eustachian tube function (20).

Acute or chronic forms of otitis media, if left untreated, may lead to extracranial or intracranial complications (Table 87.1). Hearing loss, tympanic membrane perforation, atelectasis of the middle ear, mastoiditis, apical petrositis, facial nerve paralysis, labyrinthitis, and ossicular discontinuity are some of the possible intratemporal sequelae of otitis media. Meningitis, extradural abscess, subdural empyema, encephalitis, brain abscess, sigmoid sinus thrombosis, and hydrocephalus are potential intracranial complications. Intracranial complications should be suspected in individuals presenting with changes in mental status (17,24,25).

### Labyrinthitis

Labyrinthitis is an inflammation or infection of the vestibular apparatus. Patients typically present with vertigo, nausea, vomiting, and malaise. The etiology is most often viral or traumatic, but can be bacterial. Bacterial labyrinthitis most often arises as a spread of infection from meningitis or otitis media. It can be serous or suppurative. Viral infections such as mumps, measles, Lassa fever, varicella-zoster, syphilis, and herpes simplex have been associated with labyrinthitis. Labyrinthitis may or may not be associated with a sensorineural hearing loss, which can be temporary or permanent depending on the etiology, patient’s age, and severity of the loss (26).

### Idiopathic Facial Paralysis (Bell Palsy)

Idiopathic facial paralysis is an acute unilateral peripheral facial nerve weakness. It is a diagnosis of exclusion, and only made when other causes of paralysis, such as systemic diseases, infection, trauma, central nervous system disorders, and neoplasm, are ruled out. Patients will usually present with abrupt onset of unilateral facial weakness. Other symptoms may include numbness or pain around the ear, decreased taste, and increased sensitivity to sounds (27). Herpes simplex virus is thought to be an etiologic factor for this disease (28). Bell palsy most commonly occurs in individuals between 10 and 40 years of age. Pregnant women and individuals with diabetes mellitus are at a higher risk of developing Bell palsy. Most cases spontaneously improve within 6 months. Residual facial nerve weakness may persist in about 15% of affected individuals (27). In patients older than 16 years of age, the recommended treatment consists of the early administration (first 72 hours) of high-dose prednisone. Antivirals may be added, but have not shown to have benefit apart from steroids (29). Patients should be educated about using artificial tears and protecting the eye during sleep to prevent corneal abrasion and eye infection.

### Ramsay Hunt Syndrome

Ramsay Hunt syndrome is caused by reactivation of varicella-zoster virus (VZV) in the geniculate ganglion and is associated with eruption of an auricular or oropharyngeal vesicular rash, facial paralysis, and otalgia (30). In addition, tinnitus, hearing loss, nausea, vomiting, vertigo, and nystagmus can be the accompanying symptoms (31). Patients with Ramsay Hunt syndrome present more severe symptoms and have a worse prognosis for recovery of facial nerve function relative to patients with Bell palsy. The timing between onset of facial paralysis and vesicular eruption may vary. Some patients present with facial paralysis, have a rise in VZV antibody, but never develop cutaneous manifestations. Initiation of early treatment with prednisone and acyclovir is currently recommended (32).

### Chondritis/Perichondritis

Chondritis/perichondritis of the ear is an infection of auricular cartilage/perichondrium. It is often caused by penetrating...
injury to the ear, particularly piercing of the pinna (33). Blunt trauma with auricular hematoma can also lead to infection. Cartilage involvement can also be seen in spreading otitis externa. Because of its relative avascularity, cartilage is more susceptible to infection. Infections are more often reported during warm weather, after exposure to water in pools, lakes, or hot tubs. Patients present with a very tender, erythematous, and indurated auricle. It is generally doughy on palpation and is rarely fluctuant. P. aeruginosa has been identified as the most likely cause of the infection, but S. aureus must also be considered (33,34). Treatment consists of removing any foreign body, and drainage of any abscess or hematoma. Patients should be treated aggressively with antibiotics that provide coverage for Pseudomonas. Cartilage necrosis or subperichondrial fibrosis leading to auricular deformity may be seen following the infectious process. Recurrent auricular chondritis should raise suspicion for the diagnosis of relapsing polychondritis (35).

**NASAL INFECTIONS**

**Septal Abscess**

Septal abscesses are collections of pus between the cartilaginous or bony nasal septum and the overlaying mucoperichondrium or mucoperiosteum (36). The leading cause is trauma that leads to a septal hematoma, but can also occur after septoplasty. It has also been shown to occur in association with influenza, sinusitis, nasal furuncle, and dental infection. Immunocompromised patients are at a higher risk of dangerous complications. Patients complain of nasal congestion, nasal pain, fever, and headache. On examination, there is evidence of an anterior intranasal mass, as the septum will appear swollen and fluctuant. Most common causative organisms are S. aureus and group A β-hemolytic Streptococcus (GABHS); however, Staphylococcus epidermidis, S. pneumoniae, H. influenzae, and anaerobes are also possible pathogens. Treatment involves antibiotics and surgical drainage. Complications include ischemic necrosis of the septal cartilage, intracranial infections such as meningitis, brain abscess, and subarachnoid empyema (36,37).

**Rhinosinusitis**

**Acute Bacterial Rhinosinusitis**

Acute bacterial rhinosinusitis most often develops following a viral upper respiratory infection, and is distinguished by a duration of greater than 10 days (38). Some of the presenting diagnostic symptoms include purulent nasal discharge, nasal congestion, maxillary, tooth or facial pain, and worsening of symptoms following initial improvement, but are not specific for bacterial sinusitis as opposed to viral causes. Predisposing physiologic factors include obstruction of sinus ostia, reduction in number or function of sinus cilia, and a change in the quality of secretions (39). The most common pathogens are S. pneumoniae, H. influenzae, M. catarrhalis, and S. aureus. In immunocompromised patients, patients with cystic fibrosis, and in patients with sinusitis of nosocomial origin (on mechanical ventilation, with nasal tubing), P. aeruginosa and other aerobic gram-negative rods are common causative pathogens (40). Anaerobic bacteria are usually associated with sinusitis of dental origin (41). It is often difficult to distinguish between viral and bacterial sinusitis. The diagnosis is usually based on medical history and clinical findings. With bacterial sinusitis, symptoms are usually present for more than 10 days. Sinus puncture with aspiration of sinus contents is the most accurate diagnostic technique; however, since it is invasive it is not commonly used. Radiographic imaging may help confirm the presence of sinus disease. Plain films can be difficult to interpret and should not be ordered. CT findings will include thickened mucosa, sinus opacification, or air–fluid levels, and is the preferred examination although these findings are nonspecific (Fig. 87.2). CT scans are rarely ordered to confirm acute infection unless there is concern about possible complications, such as in the case of frontal or sphenoid sinus infection. Nasal endoscopy will often demonstrate swelling within the middle meatus or sphenoid recess, with purulent discharge. Antimicrobial treatment of acute sinusitis includes amoxicillin (first line), amoxicillin–clavulanic acid, cephalosporins, trimethoprim–sulfamethoxazole, macrolides, doxycycline, and quinolones (42). Treatment can be supplemented with nasal saline irrigation, antihistamines, decongestants, and intranasal steroids (43). If there is minimal improvement after 2 to 3 days, a change in antibiotics may be indicated. Frontal sinus disease in particular may require early surgical management. If not treated, acute bacterial sinusitis may be complicated by the development of a number of orbital and intracranial complications, particularly when the infection involves the ethmoid, frontal, or sphenoid sinuses, and may require additional surgery to treat the complications (Table 87.2) (44–46).

**Chronic Bacterial Rhinosinusitis**

Chronic bacterial rhinosinusitis is diagnosed when the symptoms of sinusitis are present for at least 12 weeks. Symptoms include nasal congestion, purulent discharge, facial pressure, and anesthesia. Nasal endoscopy may reveal nasal polyps, edema, or purulent discharge. CT findings may reveal mucosal thickening, sinus opacification, polyps, or air-fluid levels (47,48). Predisposing factors include smoking, inhalant allergies, obstruction
of the ostiomeatal complex (Fig. 87.3), immune deficiency, and genetic factors (48). Pathogens are similar to those found in acute infections, with a greater predominance of Staphylococcus, Pseudomonas, and possibly anaerobes. The most common anaerobic bacteria include Peptostreptococcus species, Fusobacterium species, Prevotella, and Porphyromonas species. In cases of P. aeruginosa, aminoglycosides, fourth-generation cephalosporins, or fluoroquinolones are used in treatment (40). In chronic bacterial rhinosinusitis (CRS), a prolonged course of antibiotic therapy is often required, ranging from 3 to 6 weeks, with any imaging occurring after the completion of medical therapy. Adjunctive therapy including decongestants, mucolytics, and steroids (both intranasal and oral) may be helpful. In patients with continued symptoms, recent guidelines recommend allergy testing to exclude allergic rhinitis as a contributing factor, as well as considering other immunologic disorders. Testing for cystic fibrosis in younger patients may also be helpful (43,49). If patients do not respond to medical therapy, functional endoscopic sinus surgery may be indicated (50).

**Viral Rhinosinusitis**

Viral rhinosinusitis is more common than bacterial. The most common pathogens are rhinovirus, influenza viruses, adenoviruses, parainfluenza viruses, and respiratory syncytial virus (RSV). Inflammatory symptoms of viral rhinosinusitis are thought to be due to the host response to the virus. Patients may present with symptoms of the common cold such as nasal congestion, nasal discharge, sneezing, cough, fever, malaise, and muscle ache. Viral rhinosinusitis is usually self-limited. Antiviral therapy may be used for specific viruses. Nasal saline irrigation and various anti-inflammatory medications may aid with symptomatic relief (51).

**Fungal Rhinosinusitis**

**Acute Necrotizing Fungal Rhinosinusitis.** Acute invasive necrotizing fungal rhinosinusitis is a fulminating invasive fungal infection that is often life-threatening. It usually affects immunocompromised patients, such as diabetics, patients with immunoodeficiency disorders, and patients undergoing chemotherapy. Patients often present with acute onset of fever, headache, cough, mucosal ulcerations, and epistaxis. On examination, necrotic black turbinates and nasal eschar spreading through mucosa, soft tissue, and bone are seen. Histopathologic evaluation of involved tissue reveals necrosis and inflammatory infiltrate with giant cells, lymphocytes, and neutrophils. Gomori methenamine silver or periodic acid-Schiff histologic fungal stains demonstrate tissue and vascular invasion by fungal hyphae. Most common pathogens are Aspergillus, Rhizopus, and Mucor species. Treatment involves emergent surgical debridement, intravenous antifungal drugs such as Amphotericin B, and treatment of the underlying immunocompromising disorder. If disease is not treated, it may lead to rapid dissemination and death (52,53).

**Chronic Invasive Fungal Rhinosinusitis.** Chronic invasive fungal rhinosinusitis is a chronic (greater than 3 months) and slowly invasive fungal infection. It too usually affects immunocompromised patients, particularly diabetics and patients requiring prolonged corticosteroid treatment, but has also been reported in otherwise healthy individuals. Patients may present with orbital apex syndrome due to the extension of the infection into the orbit. This will result in decreased vision, ocular immobility, and proptosis. Erosion may also occur into the infratemporal fossa, the anterior cranial fossa, or the premaxillary region. Histopathology reveals a dense accumulation of hyphae, with a chronic inflammatory infiltrate of lymphocytes, giant cells, and necrotizing granulomas. If left untreated, the disease may invade cerebral blood vessels leading to ischemic injury, or directly invade the brain. Treatment involves repeated surgical debridement and antifungal drugs (52).

**Mycetoma**

Mycetoma, also described as a fungal ball, is an accumulation of degenerating fungal hyphae within a sinus cavity, most often involving the maxillary sinus. Patients are generally immunocompetent and will present with symptoms of nasal obstruction, postnasal drainage, and localized facial pain. Risk factors include previous sinus surgery, oral–sinus fistula, and chemotherapy treatment. The presence of chronic mucosal
inflammation may be noted on nasal endoscopy, along with green–black concretions within the middle meatus. A CT study will reveal sinus opacification, often with areas of calcification. MRI may be definitive, demonstrating isodense opacification on T1 images, with a ring of enhancement and central attenuation on T2 (Fig. 87.4). This result is from ferromagnetic deposits related to the fungal infection. Aspergillus is the most common organism, although fungal cultures are often found to be negative. Treatment involves surgical removal of the fungal ball with adequate drainage of the affected sinus (52,55).

**Allergic Fungal Sinusitis**

The etiology of allergic fungal sinusitis (AFS) is thought to be in part an allergic response to the presence of noninvasive fungi in the sinus cavity, and has been likened to allergic bronchopulmonary aspergillosis (54,56). Patients will commonly present with hypertrophic sinus disease and nasal polyps. Symptoms of headache, paranasal fullness, and nasal discharge are often reported. Sinus CT often reveals the presence of chronic sinusitis with hyperattenuation present in the opacified sinus, creating an inhomogeneous appearance often with areas of calcification (Fig. 87.5). Serum IgE levels are often elevated, and histologic evaluation reveals the presence of allergic mucin, containing fungal hyphae and elevated eosinophils; there is no evidence of mucosal invasion. Intraorbital and intracranial expansion may occur secondary to pressure resorption of surrounding bone. The most common causative agents are *Bipolaris spicifera* and *Curvularia lunata*. Other causative agents are *Exserohilum rostratum*, *Alternaria* species, and *Aspergillus* species. Treatment consists of sinus surgery to remove the diseased mucosa and allergic mucin, although recurrence is common. Once AFS is diagnosed, if there are no contraindications, treatment with corticosteroids should be initiated. Other treatments include surgical debridement, immunotherapy, antibiotics, and nonsteroidal immunomodulatory medications (57).

**ORAL CAVITY**

**Gingivitis**

Gingivitis, a reversible disease, affects 50% to 90% of the adult population. It has an infectious etiology caused by oral microflora in the accumulating dental plaque, and usually contains both aerobic and anaerobic bacteria. Chronic gingivitis often leads to bleeding of the gums during tooth brushing (58). Gingivitis may progress to periodontitis, which involves inflammation of deeper tissues leading to the loss of supporting connecting tissue and alveolar bone; this disease is nonreversible and may lead to loss of involved teeth (59,60). Treatment involves primarily good oral hygiene along with the mechanical removal of plaque and calculus (58).

**Acute Necrotizing Ulcerative Gingivitis (Trench Mouth)**

Acute necrotizing ulcerative gingivitis (Trench mouth, Vincent stomatitis) is a rare periodontal disease characterized by gingival necrosis, ulceration, pain, and bleeding (61). The disease is most commonly seen in young adults, with patients often presenting with sudden onset of gingival inflammation. Gingival lacerations covered with gray membranes and gingival bleeding are noted on the examination. The causative organisms are
fusospirochetal bacteria (*Borrelia vincentii*), which become pathogenic during periods of compromised immune system function. *Bacteroides* and *Selenomonas* species have also been implicated in the disease (61). Diagnosis is based on clinical findings. Risk factors include dental crowding, physical fatigue, increased stress, low socioeconomic status, immunosuppression, smoking, and poor oral hygiene (62). Treatment includes eliminating precipitating factors, treatment of underlying immunosuppression, oral hygiene, mechanical debridement of affected areas, and antibiotics. Penicillin or metronidazole is recommended for antibiotic treatment (62).

**Herpetic Gingival Stomatitis**

Herpetic gingivostomatitis is an infection due to Herpes simplex virus. Primary infection most commonly manifests in children between the ages of 2 and 5 years. Patients may present with fever and irritability; oropharyngeal pain, mucosal edema, and erythema are often present. Vesicular lesions appear on mobile or nonkeratinized mucosal surfaces (buccal, labial) and attached or keratinized surfaces (gingiva, hard palate); these usually rupture within 24 hours, leaving small ulcers with an elevated margin. Diagnosis is confirmed by viral studies and biopsy. Treatment is usually supportive, although acyclovir may help to shorten the severity and duration of the infection. Once the primary infection resolves, the organism remains dormant, with the reservoir usually being the trigeminal ganglion; periodic reactivation of infection may occur. In most cases, individuals must have an active lesion to be able to transmit the virus (63,64).

**Candidiasis**

Candidiasis is caused by the overgrowth of *Candida albicans*. Often, the patient is predisposed, with a history of immunosuppression, radiation, or altered microflora following long-term broad-spectrum antibiotic use. In the pseudomembranous form, yellow–white plaques are present that have been likened to milk curds (Fig. 87.6A), whereas in the erythematous form, these plaques have disappeared (Fig. 87.6B). Clinical diagnosis may be confirmed with potassium hydroxide staining revealing fungal hyphae. Initial therapy usually consists of oral hygiene and topical treatment; some of the available agents include oral nystatin preparations, amphotericin lozenges, and clotrimazole troches. Ketoconazole, fluconazole, anditraconazole can be used for systemic treatment if indicated (65).

**Odontogenic Infections**

Odontogenic infections often originate from infected pulp and may spread to the fascial spaces of the head and neck where an abscess may form (Fig. 87.7). The potential spaces are found around the face (masticator, buccal, canine, and parotid); in the supraphyoid area (submandibular, sublingual, and parapharyngeal); and in the infrahyoid area (retropharyngeal and paratracheal spaces). The most common causative organisms are *S. aureus*, group A streptococci, and anaerobic bacteria. Treatment with broad-spectrum antibiotics is recommended, and surgical drainage is the primary treatment (66,67).

**Ludwig Angina**

Ludwig angina is an infection that involves the left and right sublingual and submandibular spaces, generally spreading rapidly through fascial planes. It occurs most often in adults with poor dentition, usually from an infection involving the second or third molar. Other sources may include inflammation of the tongue or floor of mouth, and lingual tonsillitis (68,69). Patients will often present with submandibular swelling but not fluctuance, and swelling of the floor of mouth that pushes the tongue upward and backward toward the palate. In the case of advanced disease, patients may present in acute distress with fever, difficulty handling secretions, and dyspnea that favors a seated and head-forward position; infection can be rapidly progressive, leading to airway compromise. Anaerobic organisms and streptococci are the most common cause of Ludwig angina. Treatment requires close airway monitoring, with prophylactic tracheotomy needed for airway protection in most cases, administration of antibiotics, and surgical drainage (68,69).
PHARYNX

**Tonsillopharyngitis**

Tonsillopharyngitis is a common disease characterized by infection of the nasopharynx and oropharynx and its associated lymphoid tissue. Acute tonsillopharyngitis may be caused by viral or bacterial infection, viral being the most common. It is often difficult to distinguish between bacterial and viral causes based on clinical examination. Patients present with fever, malaise, odynophagia, and lymphadenitis. On examination, tonsillar enlargement, erythema, and exudate may be present. Upper respiratory viruses such as rhinovirus, coronavirus, and adenovirus are the most common causes of viral infection (70). The most common cause of bacterial tonsillopharyngitis is a GABHS, which may be diagnosed by performing a group A *Streptococcus* test. Other pathogens have also been associated with the disease such as *M. catarrhalis*, *H. influenzae*, *S. aureus*, and *S. pneumoniae* (71). Diphtheria and gonococcal infections should also be considered. If GABHS infection is suspected, antibiotic treatment should be initiated. Penicillin, amoxicillin, erythromycin, and first-generation cephalosporins are the recommended agents; performance of a group A *Streptococcus* test prior to initiation of antibiotic treatment is appropriate (72). If not treated, bacterial tonsillopharyngitis may lead to complications that can be suppurative and nonsuppurative. Nonsuppurative complications include scarlet fever, acute rheumatic fever, and poststreptococcal glomerulonephritis. Suppurative complications include peritonsillar, parapharyngeal, and retropharyngeal cellulites and/or abscess (73,74). In cases of recurrent streptococcal tonsillopharyngitis or infections unresponsive to antimicrobial therapy, tonsillectomy might be indicated (75,76).

**Herpangina**

Herpangina is a disease that commonly occurs in children (77), with Coxsackie A virus being the most common causative organism (78). Patients will present with fever, malaise, and sore throat. On examination, oropharyngeal erythema is
noted, and vesicles and small ulcers are present on the posterior pharynx, often on the uvula and soft palate. The course of herpangina is usually self-limited.

**Peritonsillar Abscess**

Peritonsillar abscess is the most common deep infection of the head and neck, usually occurring as a complication of bacterial tonsillitis or, less frequently, in cases of infectious mononucleosis, and is most commonly diagnosed in adults or adolescents. Infection may spread through the tonsillar capsule into the space between the tonsil and superior constrictor muscle and sequentially develop into an abscess. Patients will present with increasing pharyngeal pain, dysphagia, trismus, dysarthria, drooling, and a muffled voice. The clinical examination reveals trismus, peritonsillar bulging that displaces the soft palate medially, and uvular deviation toward the opposite side; patients will often have tonsillar exudates and tender cervical lymphadenopathy (79,80). A peritonsillar abscess is usually polymicrobial, with Group A streptococci and anaerobes, the most common pathogens. While the diagnosis is usually made on physical examination, a CT scan of the mid-face and neck may help if there is diagnostic uncertainty, but is usually not necessary. Treatment involves aspiration or incision and drainage of the abscess along with antibiotic therapy, and recent data suggest that steroids may improve outcomes as well (81,82). If the peritonsillar abscess becomes recurrent, a tonsillectomy would be indicated (80). A peritonsillar space infection has the potential for spreading to the parapharyngeal space, the manifestations of which may be delayed.

**Lemierre Syndrome**

Lemierre syndrome is described as the presence of oropharyngeal infection, sepsis, internal jugular vein thrombosis, and septic emboli caused by *Fusobacterium necrophorum* (83,84). This is a gram-negative anaerobic organism which can be part of the normal human oropharyngeal, gastrointestinal, or genitourinary flora. The disease is currently uncommon due to the availability of antibiotics. Lemiere syndrome most often affects young adults with a recent history of oropharyngeal, tonsillar, or peritonsillar infection. Patients will often present with tenderness and swelling of the lateral neck, secondary to thrombophlebitis of the internal jugular vein. Septic emboli may spread and affect other organs, especially the lungs. Mortality rates approach 5%, and thus the disease requires immediate and aggressive antibiotic treatment with agents such as clindamycin, metronidazole, ampicillin–sulbactam, or ticarcillin–clavulanate for a period of at least 6 weeks (84); in the case of abscess formation, surgical drainage might be required (85). Anticoagulation is controversial, but may be indicated in severe disease (86).

**Infectious Mononucleosis**

Infectious mononucleosis is a systemic disease caused by Epstein–Barr virus, transmitted via saliva, and most commonly occurring in teenagers and young adults. Patients will present with fever, fatigue, malaise, sore throat, and generalized nontender lymphadenopathy. On examination, one encounters inflamed tonsils with exudate; hepatosplenomegaly may also be present. Diagnosis is confirmed by the presence of atypical lymphocytes on peripheral smear, a positive monospot test, and positive EBV titers; treatment is supportive (87). Corticosteroids are used to decrease inflammation, particularly in cases where airway obstruction is a concern due to marked tonsillar enlargement; in severe cases, where airway obstruction is a concern, establishing a secure airway may be indicated. Patients will develop a rash if treated with amoxicillin for presumed bacterial tonsillitis, thus administration of amoxicillin should be avoided.

**LARYNX/AIRWAY**

**Supraglottitis/Epiglottitis**

Epiglottitis is an infectious disease of the epiglottis and supraglottis, most commonly bacterial in origin. It usually has a sudden onset, with patients developing high fever, pain with swallowing, drooling due to difficulty handling secretions, and respiratory distress. On presentation the patient is often found sitting in a hunched forward position with an extended neck and open mouth (sniffing position) (88). On a lateral neck film, edema of the epiglottis and a ballooning of the hypopharynx (“thumb” sign) will be noted (Fig. 87.8). On physical examination, the oral cavity and oropharynx will look remarkably benign. However, on direct visualization, the epiglottis will appear erythematous (“cherry-red”) and swollen. Care should be taken with airway manipulation as it may quickly precipitate complete airway obstruction. *H. influenzae* used to be the most common causative agent; however, with the introduction of the vaccine, the incidence of *H. influenzae*–related epiglottitis...
has significantly decreased (89). Currently, *S. pneumoniae* group A and β-hemolytic streptococcus are the most common causative agents (90). Epiglottitis is considered an emergency as it has a potential for rapid complete airway obstruction, particularly in children. When epiglottitis is diagnosed, a secure airway should be established, in most cases, via flexible fiberoptic endotracheal intubation or tracheostomy; the decision to extubate or decannulate the trachea is based upon clinical improvement. Treatment with intravenous antibiotics (ceftriaxone or ampicillin–sublactam) and steroids should be initiated immediately (88,89). Adults may sometimes present with supraglottitis where the epiglottis is not involved. In these cases, the airway can often be managed more conservatively, although in hospital, observation is required.

**Laryngitis**

Acute laryngitis most often occurs as part of an upper respiratory infection, and therefore is usually caused by rhinovirus (91). Laryngoscopy reveals diffuse laryngeal erythema and edema, often producing a cough and hoarseness. The treatment is most often supportive, including voice rest, humidification, and occasionally anti-inflammatory medications.

**Croup**

Croup is an inflammatory disease of the subglottic airway, almost always associated with a viral infection, most commonly parainfluenza and influenza viruses. It is most often seen in children between the ages of 1 and 3 years. Patients usually present with fever, tachypnea, inspiratory stridor, hoarseness, and a barking cough; the history often includes a preceding upper respiratory infection. Radiographic studies reveal subglottic narrowing, the so-called “steeple” sign (Fig. 87.9).

Additional diagnostic information can be provided by flexible bedside endoscopy. Depending on the severity of the symptoms, patients might require close observation and establishment of a secure airway. Administration of glucocorticoids is recommended to decrease airway inflammation, and racemic epinephrine treatments are often helpful. In moderate to severe croup, helium–oxygen (heliox) treatment is also of benefit (92). When croup is recurrent, an airway evaluation with laryngoscopy and bronchoscopy is recommended to assess for anatomic abnormalities such as subglottic stenosis (93,94).

**Diphtheria**

Diphtheria is an infectious disease of the upper airway caused by *Corynebacterium diphtheriae* and is rather rare due to widespread immunization. Patients present with fever and malaise; bloody nasal discharge and pseudomembranes in the nose, oropharynx, and larynx may be noted on examination; the presence of membranes may lead to airway obstruction and respiratory failure. Exotoxins produced by the bacteria may affect the heart, liver, kidney, and brain. Clinical diagnosis is confirmed by bacterial smear and cultures on Löffler or tellurite media. The treatment consists of assuring a secure airway, administration of antitoxin, and antibiotics. Penicillin or erythromycin is recommended for treatment (95).

**Bacterial Tracheitis**

Bacterial tracheitis is a rare, potentially life-threatening respiratory infection. It is characterized by the presence of thick membranous tracheal secretions that do not readily clear with coughing and may lead to occlusion of the airway. Patients present with fever, cough, stridor, and generalized malaise; there is no response to racemic epinephrine treatment. Radiographic findings often reveal irregular tracheal margins with a normal-appearing epiglottis. Diagnosis is made with direct endoscopic visualization of thick membranous tracheal secretions or the presence of purulent tracheal secretions in the glottis and subglottis (96). The most commonly isolated pathogen is *S. aureus*. Other causative bacterial pathogens include *H. influenzae, S. pneumoniae, Streptococcus pyogenes, M. catarrhalis, Klebsiella,* and *Pseudomonas* species (96–98). If the diagnosis of bacterial tracheitis is made, treatment consists of securing the airway, endoscopically removing tracheal membranes, and administration of antibiotics (97).

**NECK**

**Salivary Gland Infections**

**Viral Infections**

Mumps is a viral infection caused by paramyxovirus and is the most common viral infection that involves the salivary glands. Infected patients display signs of fever and malaise; painful parotid swelling occurs within 24 hours of symptom onset and is often bilateral, with pain upon salivation. Ten percent of patients have submandibular gland involvement; 25% of affected adolescent or adult males will develop orchitis, and 5% of females may present with oophoritis. Pancreatitis and central nervous system involvement may also occur in affected individuals; sensorineural hearing loss may also be
seen in mumps. The treatment is usually supportive, although it must be noted that the disease is preventable by vaccination and children have a less severe course than adults (99). Other potential viruses that may infect the salivary glands include Coxackie A, ECHO, choriomeningitis, parainfluenza types 1 and 3, and cytomegalovirus (99).

**Bacterial Infections**

Bacterial infections of the salivary glands often develop following salivary stasis, secondary to ductal obstruction by a stone or mass, or a decrease of salivary flow secondary to dehydration. Any sort of intraoral trauma, such as extensive dental work, may also cause an inflammatory obstruction of the duct. The most common causative pathogens are *S. aureus* and *Streptococcus viridans*. The parotid gland is affected more often, likely due to lower bacteriostatic activity of saliva from this gland as compared to the submandibular or sublingual gland. Patients present with pain, swelling, and erythema in the region of the salivary gland, exacerbated by eating; this disorder is most commonly seen in the elderly. Patients may also have fever, malaise, and an elevated white count. Treatment involves hydration to increase salivary flow, massaging of the effected gland, sialogogues, and administration of antistaphylococcal antibiotics. If there is minimal improvement on conservative treatment, imaging may be considered (CT or ultrasound) to look for abscess. In cases of chronic or recurrent sialadenitis, removal of the obstructing stone via sialoendoscopy or excision of the gland may be indicated (100,101).

**Lymphadenitis**

Lymphadenitis is an inflammatory process involving lymph nodes, most commonly seen in the pediatric group. Upper respiratory viral infections are the most common cause of cervical lymphadenopathy. This condition is self-limited and usually does not require treatment. Bacterial lymphadenitis most often occurs as a complication of skin or respiratory infection. The commonest causative organisms are *S. aureus* and group A streptococci. Patients may present with tender lymphadenopathy, which may progress to formation of an abscess. If bacterial lymphadenitis is suspected, treatment with β-lactamase-resistant antibiotics is recommended. In case of progression to abscess formation, incision and drainage are indicated (102).

**Cat-Scratch Disease**

This disease usually presents with subacute solitary or regional lymphadenopathy in patients with a previous contact with a kitten or cat. It is primarily caused by *Bartonella henselae*; however, cases of *Bartonella clarridgeiae* and *Bartonella elizabethae* have been reported. Small red–brown nontender papules may develop at the site of inoculation 3 to 30 days after contact, and lymphadenopathy is seen 1 to 3 weeks after a scratch, bite, or other contact with an infected kitten or cat. The lymph nodes draining the affected site gradually enlarge and are moderately tender, with the overlying skin appearing warm and erythematous. Up to 10% of lesions may require surgical drainage. Histologic examination shows granulomata with multiple microabscesses. The indirect fluorescent antibody test and enzyme immunoassay are used for the detection of specific serum antibody to *B. henselae* (95).

**Mycobacterium**

Nontuberculous mycobacterial infection is a rare cause of lymphadenitis. It presents as a slowly enlarging, nontender cervical mass. The infection can affect adults; however, it is most commonly found in children less than 5 years of age. It is often diagnosed after the failure to respond to traditional antibiotics. The most common causative pathogen is *Mycobacterium avium–intracellulare*; other reported pathogens are *Mycobacterium scrofulaceum*, *Mycobacterium kansasai*, *Mycobacterium fortuitum*, and *Mycobacterium cobacterium malmoense* (103,104). Nontuberculous mycobacteria are found ubiquitously in the environment—soil, food, water, animals, etc. The infection is usually acquired from the environment, with no evidence of person-to-person transmission. An intradermal purified protein derivative (PPD) test aids in the diagnosis of nontuberculous lymphadenitis; however, positive cultures will be more definitive (105). Complications include fistula tract formation, and thus complete surgical resection of infected tissues has been the gold standard for the treatment of nontuberculous mycobacterial infections. Treatment with antituberculous medications, such as macrolides (clarithromycin) and rifampin, has also been shown to be effective in some cases. Recent studies have suggested an antibiotic therapeutic trial prior to surgical excision or as an adjunct to surgical excision, though this is controversial, and other data suggest complete excision as the treatment with the lowest likelihood of recurrence (106,107).

**Actinomycosis**

Actinomycosis is an infection caused by *Actinomyces israelii*, a gram-positive anaerobic bacterium. The disease has multiple presentations and is often misdiagnosed. The majority (>50%) of these infections occur in the head and neck region, most often entering the tissue through an area of prior trauma. As the infection develops, patients will be noted to have a woody induration that eventually leads to central abscess formation. This abscess will generally track to a mucosal surface or externally to the skin, forming a sinus or fistulous tract. The suppurative drainage will contain so-called sulfur granules, yellow flecks containing the bacterial colonies. The diagnosis is best made by culture, but as anaerobic cultures can be unreliable, diagnosis may have to rely on the clinical picture and histology. The organisms stain best with Gram and Gomori methenamine silver stains. Treatment consists of drainage and debridement of the infected area with administration of penicillin (108).

**Deep Neck Space Infections**

**Retropharyngeal Space Abscess**

The retropharyngeal space is defined by the prevertebral fascia posteriorly, the posterior layer of the deep cervical fascia anteriorly, the skull base superiorly, and the posterior mediastinum inferiorly (see Fig. 87.7). Infection usually develops from infected retropharyngeal lymph nodes, which receive lymphatic drainage from the paranasal sinuses, nasopharynx, and middle ear, and are more common in children; trauma is another common cause. Patients commonly present with fever, pain upon swallowing, decreased oral intake, drooling, malaise, and torticolli; trismus and neck swelling are often present. The most common causative organisms are *S. pyogenes*, *S. aureus*, and anaerobic bacteria (109). Lateral radiographic
images of the neck in extension reveals thickened prevertebral soft tissue. CT aids in determining the presence of an abscess. Therapy involves the administration of intravenous antibiotics and abscess drainage. Transoral drainage is recommended unless there is extension lateral to the great vessels, i.e., the carotid artery (110). Adjunctive treatments include broad-spectrum antibiotics and steroids postoperatively to decrease swelling. If left untreated, spontaneous rupture of the abscess may lead to aspiration of infectious material; additionally, infection may spread to the parapharyngeal and prevertebral spaces, and lead to mediastinitis or involve the great vessels.

**Parapharyngeal Space Abscess**

The parapharyngeal space is a potential space that extends from the skull base to the greater cornu of the hyoid bone (see Fig. 87.7). The pharynx and superior constrictor muscle are the medial boundaries; the internal pterygoid muscle, parotid gland, and mandible are lateral structures; the prevertebral fascia lies posteriorly; and the pterygomandibular ligament is an anterior structure that surrounds the parapharyngeal space. The styloid process divides the space into anterior and posterior divisions. Pharyngeal infections, molar tooth infections, gingivitis, and even mastoiditis may spread to the parapharyngeal space. If untreated, infection has the potential to spread to the retropharyngeal space, to the mediastinum, and to involve the great vessels causing internal jugular thrombosis and erosion of the internal carotid artery; airway compromise may also occur. Patients typically present with a prior history of a sore throat or tooth infection. Initial symptoms are fever and pain upon swallowing. Tender, erythematous swelling at the angle of the mandible and parotid is typically found on clinical examination, but it will not appear fluctuant even if an abscess is present. Examination of the pharynx will often reveal medial displacement of the ipsilateral tonsil. Trismus may develop secondary to inflammation of the medial pterygoid muscle. Torticollis toward the opposite side often results from inflammation of lymph nodes under the sternocleidomastoid muscle; patients may also complain of otalgia (111). The most common causative pathogens are *S. aureus*, *S. pyogenes*, and *S. viridans* and anaerobic bacteria (112). CT with contrast aids in the evaluation of the site and the extent of infection, distinguishing between cellulitis and abscess (Fig. 87.10). Parapharyngeal space infections require immediate treatment with intravenous antibiotics and surgical drainage if a large abscess is present (113). This is usually done through an external approach. Transoral drainage is not recommended.

**Key Points**

- In a patient presenting with an ear infection, pushing on the tragus will elicit pain if it is an outer or external ear infection, but not if it is a middle ear infection.
- Oral antibiotics are rarely helpful in the treatment of external otitis, a condition that is more effectively treated with topical preparations.
- Otitis externa with granulation tissue in a diabetic patient should be considered diagnostic of necrotizing otitis externa and treated aggressively.
- In an adult presenting with a unilateral middle ear effusion, it is essential to evaluate the nasopharynx to rule out the presence of a nasopharyngeal mass obstructing the eustachian tube.
- Auricular perichondritis should be treated early and aggressively to prevent the sequel of auricular deformity. Antibiotics with cartilage penetration, such as fluoroquinolones, are recommended.
- The presence of a nasal septal hematoma or abscess requires immediate drainage to prevent secondary necrosis of the septal cartilage and the subsequent development of a saddle nose deformity.
- A bacterial rather than viral sinus infection should be suspected when symptoms have been present for >7 to 10 days or symptoms are worsening after 5 days. Most sinus infections are of viral etiology
- Ludwig angina can be rapidly progressive and should be considered an airway emergency, with a prophylactic awake tracheotomy recommended in most cases.
- Due to its potential for rapid airway compromise, epiglottitis should be considered an airway emergency.
- Due to its deep location, a parapharyngeal space abscess will cause tender induration of the upper neck, but not fluctuance.

**References**