Airway Infectious Disease Emergencies

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Upper and lower respiratory infections are commonly encountered in the emergency department (ED). Visits for respiratory disease account for 10% of pediatric emergency department visits and 20% of all pediatric hospital admissions [1]. The causes of upper airway infections include croup, epiglottitis, and retropharyngeal abscess-cellulitis (pharyngitis and peritonsillar abscess are described separately). Lower airway infections arise from bacterial and viral infections and cause illnesses such as pneumonia and bronchiolitis. Signs and symptoms overlap with upper and lower airway infections but differentiation is important for the appropriate treatment of these conditions. This article reviews the various clinical characteristics of upper and lower airway infections.

Upper airway infections

Upper airway infections in children include a variety of common and uncommon conditions that can pose significant diagnostic and therapeutic challenges. These difficulties tend to be augmented by the potential for rapid airway compromise and limited evaluations in smaller, apprehensive children. As with many infections, the primary challenge in these conditions lies in identifying the causative pathogen and determining the extent of disease progression. In this discussion, upper airway infections are grouped into the three categories of pharyngotonsillar, laryngotracheobronchial, and deep neck space infections, with an emphasis on recent advances in diagnostic and management strategies.

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Pharyngotonsillar infections

Pharyngotonsillar infections are a group of commonly encountered upper airway problems that include pharyngitis, tonsillitis, and peritonsillar infections. Pharyngitis refers to infections of the pharynx and may also include tonsillitis, in which case the complex is referred to as pharyngotonsillitis. The varied causes but overlapping clinical presentations of these infections have made them the focus of several recent practice guidelines that promote selective and targeted antibacterial therapy in an attempt to reduce the number of unnecessary antibiotic prescriptions [2,3].

Viruses are the most common cause of pharyngitis and tonsillitis in all age groups. Common viral pathogens include respiratory viruses such as influenza virus, parainfluenza virus, adenovirus, and rhinoviruses as well as others, such as coxsackievirus, echoviruses, and Epstein-Barr virus. Group A streptococci (GAS) is the most common bacterial cause of pharyngitis, but a number of other bacteria such as *Mycoplasma pneumoniae*, *Chlamydia pneumoniae*, *Neisseria gonorrhoeae*, and *Arcanobacterium haemolyticum* are also implicated, although less commonly [2,4,5].

GAS pharyngitis is the only commonly occurring form of bacterial pharyngitis that definitely requires antibiotic therapy [2]. The significance of GAS infection is related to its association with both suppurative complications such as otitis media, sinusitis, peritonsillar and retropharyngeal abscesses and non-suppurative sequelae, including acute rheumatic fever and acute glomerulonephritis [6]. In light of this, the primary challenge in the diagnosis of pharyngitis lies in distinguishing between streptococcal and nonstreptococcal infections.

Clinical symptoms suggestive of GAS infection include the acute onset of sore throat, fever, headache, pain on swallowing, abdominal pain, nausea, vomiting, scarletiniform rash, and enlarged tender anterior cervical lymph nodes [6,7]. Symptoms more suggestive of nonstreptococcal pharyngitis include concurrent viral respiratory or gastrointestinal infection and associated cough, coryza, conjunctivitis, and diarrhea [6]. A number of decision rules have been proposed to assist with the clinical diagnosis of GAS pharyngitis [8–10]. Of these rules, the Centor criteria, which include tonsillar exudates, swollen and tender anterior cervical lymph nodes, lack of cough, and a history of fever, are used most commonly [9]. However, despite their value in stratifying the risk of GAS infection, the sensitivity and specificity of these criteria are too low to forgo the use of diagnostic tests [2,6,11].

Diagnostic tests for the detection of GAS pharyngitis include rapid antigen detection testing (RADT) and blood agar plate cultures of throat swab specimens with adequate pharyngeal and tonsillar secretions. Several authors have shown that the sensitivity of RADT is not fixed but variable and is proportionately related to the pretest clinical likelihood of GAS pharyngitis [12,13]. The higher sensitivity of RADT in adults with a high pretest clinical likelihood of GAS pharyngitis has been used as a justification to forgo culture confirmation in those with negative RADT results [2]. However, unlike adults, the sensitivity of RADT...
in children, even in those with a higher clinical likelihood of GAS pharyngitis, has been shown to be too low to forgo culture confirmation of negative RADT results [2,12,14].

The use of RADT in patients who are clinically suspected of having GAS pharyngitis has the advantage of rapid availability of results which allows for early treatment, reduction in risk of spread, sooner return to school or work, and reduction in acute morbidity [15]. Additionally, negative RADT with pending culture confirmation allows physicians to withhold antibiotics from the majority of patients who may have an infection from a viral cause [16]. Interestingly, the use of two throat swabs versus one did not increase the sensitivity of a specific RADT, in one study [17]. Laboratory studies such as serum C-reactive protein, peripheral white blood cell (WBC) counts, and erythrocyte sedimentation rates have not been shown to help in differentiating between viral streptococcal sources of infection in acute suppurative tonsillitis [8,18].

In patients who have GAS pharyngitis, antibiotic treatment is indicated for eradicating GAS from the throat, shortening clinical course, decreasing the risk of transmission, and reducing the risk of suppurative sequelae. Furthermore, if started within 9 days after the onset of acute illness, antibiotic therapy has been shown to prevent acute rheumatic fever [6]. Despite the wide variety of antibiotics that have been shown to be effective against GAS, penicillin continues to be the drug of choice. The advantages of oral penicillin V include its proven efficacy, safety, narrow spectrum, and low cost [2,19]. Although the clinical resistance of GAS to penicillins has never been documented, the treatment failure with these agents does occur and are likely related to inadequate compliance with the 3 times daily dosing and 10-day course of therapy [6]. In light of this limitation, some authors have advocated a 10-day single daily dosing regimen of amoxicillin as an equally effective but more convenient alternative for the treatment of GAS pharyngitis [20,21].

Other antibiotic regimens for the treatment of GAS pharyngitis include a single dose of intramuscular penicillin G benzathine as well as various orally administered macrolide and cephalosporin antibiotics. The administration of intramuscular penicillin G avoids the problem of poor compliance but is painful [6]. Erythromycin is recommended for patients who have penicillin allergy, and clindamycin has been shown to be most effective in the elimination of chronic streptococcal carriage [6]. Other antibiotics, most notably azithromycin, have been studied and advocated as 3- and 5-day short-course alternatives to standard penicillin therapy [16,22–24]. The emerging problem of increasing macrolide resistance among GAS bacteria, however, makes this alternative inadvisable [25,26].

The pain associated with pharyngitis can be reduced by the use of standard nonsteroidal anti-inflammatory drugs and acetaminophen [27]. Additionally, children with moderate to severe pharyngitis have been shown to have an earlier onset of pain relief and a shorter duration of sore throat when given a single dose of oral dexamethasone suspension at a dose of 0.6 mg/kg, with a maximum of 10 mg [28].
Laryngotracheobronchial infections

Laryngotracheobronchial infections include a spectrum of common seasonal upper respiratory infections that result from varying degrees of subglottal airway inflammation and obstruction. Laryngotracheobronchitis or croup is most commonly encountered in the second year of life but is also seen frequently in children from the age of 6 months to 6 years. The overall incidence of croup is estimated at 1.5% to 6% and is noted in boys 1.4 to 2 times more commonly than in girls. Admission rates for croup have ranged from 1.5% to 31% and vary greatly with differing practice patterns [29–31].

Parainfluenza virus types 1 and 3 are associated most commonly with croup across all age groups. Other important but less common pathogens include respiratory syncytial virus (RSV), which is noted more commonly in children less than 5 years of age, influenza virus, and M pneumoniae, which is more prominent in children older than 5 to 6 years of age. Corresponding to the seasonal prevalence of these pathogens, croup is most predominant in late fall and early winter [6,29].

Characteristic clinical findings of croup include a hoarse voice, inspiratory stridor, and a barking cough, which tends to be worse at night [31]. The severity of these symptoms is related to the degree of narrowing of the larynx and trachea as a result of infection-induced mucosal inflammation and edema [32]. Children with mild croup tend to have inflammation limited to the larynx and present frequently with symptoms of hoarseness, intermittent barky cough, and inspiratory stridor that may be noticeable only with agitation. More severe cases of croup are associated with the extension of inflammation to the trachea and bronchi and present with inspiratory stridor that is audible at rest and is associated with signs of respiratory distress, including nasal flaring and intercostal retractions [32]. Although, croup is diagnosed primarily on clinical grounds, the finding of the classical “steeple sign” in the subglottal area on anteroposterior neck radiographs may be used to confirm the diagnosis [32].

Because of the self-limited nature of croup, the treatment is directed primarily at relieving symptoms of airway obstruction. Standard treatment approaches include the use of humidified air, nebulized racemic epinephrine, and systemic corticosteroids [32]. Although traditional treatment has included a mist of humidified air, a recent randomized controlled study did not show this approach to deliver any incremental benefit in relieving the clinical symptoms in children with moderate croup [32]. The use of nebulized racemic epinephrine, on the other hand, has been well established as an effective, albeit temporary, means of relieving upper airway obstruction by means of local vasoconstriction and decreasing mucosal edema [32–35].

Although the use of racemic epinephrine generally has been reserved for patients who have more severe respiratory distress, the use of systemic oral or intramuscular corticosteroids in the form of dexamethasone has been adopted more commonly and has resulted in a significant reduction in croup-related hospitalizations. Numerous studies have shown that the use of corticosteroids results
in a significant reduction of croup-related respiratory symptoms within 6 hours of administration [30,36]. Furthermore, patients treated with corticosteroids have been shown to require fewer doses of racemic epinephrine and have a significant reduction in the duration of emergency department and inpatient stay [30,36]. The addition of inhaled corticosteroids in the form of budesonide to systemic dexamethasone has not been shown to add any incremental benefit to treating children with croup [37]. Other studies have shown that children who have responded to emergency department treatment with nebulized racemic epinephrine and corticosteroids can be safely discharged home after a 2- to 3-hour period of observation [38,39].

**Epiglottitis**

Epiglottitis, also known as supraglottitis, is an inflammatory condition of the epiglottis and its adjacent structures that can progress rapidly to life-threatening airway obstruction. Compared with adult epiglottitis, the now rare childhood form of this condition presents with several distinctive clinical features that further add to the diagnostic and management challenges of this potentially fatal condition [40,41].

Historically, epiglottitis has been closely associated with invasive *Haemophilus influenzae* type b (Hib) infection. Before the initiation of childhood vaccination programs with Hib-conjugated vaccines in 1998, epiglottitis was second only to meningitis as the most common presentation of Hib disease. Since the early 1990s, a dramatic decline in the number of cases of childhood epiglottitis has been noted [42–47]. By contrast and for uncertain reasons, during this same period, the incidence of epiglottitis in adults has risen significantly [48–50].

In the post-vaccine era, most cases of childhood epiglottitis are caused by pathogens other than *H influenzae*. Among these, *Streptococci* and *Staphylococci* organisms and *Candida albicans* are the most common bacteria, although the relative frequency of epiglottitis caused by these pathogens has not increased [43,45,46]. Despite the widespread use of Hib vaccination, a number of cases of Hib-related epiglottitis still have been reported in both immunized and non-immunized children [46,51,52]. For these reasons, an up to date immunization history should not exclude the possibility of epiglottitis in a child with a clinically consistent presentation.

In light of the now infrequent nature of childhood epiglottitis and the potential for rapid clinical deterioration, the diagnosis of this condition requires a high index of suspicion and careful attention to subtle clues in the patient’s history and physical examination. Classically, childhood epiglottitis presents as a rapidly progressing illness in a previously healthy individual. Presenting symptoms may include fever, irritability, sore throat, drooling, stridor, and a “hot potato” voice. Additionally, patients frequently appear toxic and exhibit evidence of difficult breathing [46,48,53]. The classic “tripod position” refers to the preferential forward-leaning posture with bracing arms and extension of the neck that allows
for maximal air entry. The clinical presentation of childhood epiglottitis differs from that of adults in that the latter tends to have a more gradual course, is less likely to present with airway compromise, and frequently has symptoms that are limited to sore throat and odynophagia [40,41].

Clinical features associated with a higher likelihood of airway obstruction in children include evident respiratory distress, stridor, drooling, and a shorter duration of symptoms [49]. Whenever a child’s clinical presentation suggests epiglottitis, priority should be given to protecting the airway. In light of the child’s smaller airway, particular care must be taken to avoid advancing a partial airway obstruction to a complete one. For this reason, the clinician should avoid attempts at direct visualization or interventions, such as venopuncture, that may further agitate the patient.

The definitive diagnosis of epiglottitis requires direct visualization of a red swollen epiglottis under laryngoscopy [48,51,53]. Because of the higher likelihood of airway obstruction in children, this examination should be attempted only in an interdisciplinary collaboration with an anesthesiologist and an otolaryngologist in a controlled setting, such as the operating room, which allows for the establishment of an artificial airway.

Once a secure airway has been assured, additional diagnostic and therapeutic interventions can be initiated. Diagnostic studies that can aid in the management of patients who have epiglottitis include a complete blood count (CBC), blood culture, and soft tissue lateral neck radiographs. Patients with epiglottitis have elevated leukocyte counts on complete blood counts and frequently have positive blood cultures for the offending bacterial pathogen [48,51]. Soft tissue lateral neck radiographs may reveal an enlarged epiglottis with a classic “thumbprint” appearance that can confirm the diagnosis in uncertain cases [48].

Broad-spectrum intravenous antibiotics against β-lactamase-producing pathogens should be initiated as soon as a secure airway has been established. The antibiotics most commonly used include ceftriaxone and ampicillin-sulbactam [48]. Although intravenous steroids are frequently administered for the management of airway inflammation, no controlled studies exist to justify this approach in childhood epiglottitis [48].

Differential diagnostic considerations in childhood epiglottitis are extensive and further add to the diagnostic challenges of this condition. Among these, foreign body aspiration or ingestion as well as anaphylactic reactions and laryngotracheobronchial or retropharyngeal infections are most notable. Complications of childhood epiglottitis can include the progression of infection to deep neck tissue as well as respiratory failure and death [48,49,53].

**Deep neck space infections**

Peritonsillar, retropharyngeal, and parapharyngeal infections are among a group of potentially life-threatening deep neck infections in children that share common clinical features and can present significant diagnostic challenges.
Prompt diagnosis and management of these conditions are essential to ensure successful recovery and prevention of complications [54]. Approaches to the diagnosis and management of these infections are evolving and are the focus of this discussion.

Parapharyngeal and retropharyngeal infections

Parapharyngeal or lateral pharyngeal infections develop in a funnel-shaped space lateral to the pharynx that posteriorly contains the carotid sheath and cranial nerves [55]. In children, these infections may be related to complications of pharyngotonsillar, dental, or adjacent deep neck space infections [55]. Retropharyngeal infections develop in the potential space located between the posterior pharyngeal wall and the prevertebral fascia and may be medical (45%), traumatic (27%), or idiopathic in origin [54,56,57]. Infections secondary to traumatic injuries can be seen in children and adults and may be associated with accidental trauma, foreign body ingestion, or complication of medical procedures [58].

Retropharyngeal infections of medical causes are noted most commonly in children younger than 6 years old, with a peak incidence at 3 years of age [56,58,59]. These infections are generally secondary to contiguous spreading along a lymphatic chain that originates from the nasopharynx, adenoids, and paranasal sinuses and extends to the adjacent pharyngeal tissues. Accordingly, retropharyngeal infections in children tend to be preceded by upper respiratory tract infection such as pharyngitis, tonsillitis, sinusitis, and cervical lymphadenitis. The reduced incidence of retropharyngeal infections in older children has been attributed to the atrophy of these lymphatic structures with age [54,56].

Offending pathogens in these infections tend to vary with the source of origin and frequently include multiple aerobic and anaerobic organisms. Common isolates include *S viridans* and *pyogenes*, *Staph aureus* and *epidermidis*, as well as *Bacteroides*, *Peptostreptococcus*, *Fusobacterium*, *Haemophilus*, and *Klebsiella* organisms [54,56,60]. The extent of tissue involvement can range from cellulitis to frank abscess formation and frequently contributes to the varying clinical presentation.

Because of the relatively infrequent nature of these infections, a high index of clinical suspicion is required to ensure a correct diagnosis and early intervention. The most important clinical findings include fever, neck swelling, pain, and torticollis. A limitation of neck movement associated with decreased oral intake or drooling is an especially important clue in the diagnosis of retropharyngeal abscesses. Other important associated findings include cervical lymphadenopathy and trismus. Interestingly, the signs and symptoms of respiratory distress such as stridor or wheezing are not common initial findings. Important differential diagnostic considerations include epiglottitis, laryngotracheobronchitis, and meningitis [56,58–60].

Once clinical findings raise the possibility of deep neck infections, prompt hematologic and radiologic studies can establish the presence, extent, and location of the infection. Leukocytosis on complete blood count is a common finding
and can provide an initial evidence of infection [56,59]. The definitive diagnosis, however, is established with radiologic studies, which most commonly include lateral soft tissue neck radiographs, neck CT, and neck ultrasonography.

Lateral soft tissue neck radiographs can serve as a simple screening study for retropharyngeal infections. Prevertebral soft tissue swelling of greater than 7 mm at the level of the second cervical vertebra or greater than 14 mm at the level of the sixth cervical vertebra is concerning for retropharyngeal pathology. However, unless gas is noted in the area of tissue swelling, distinguishing between cellulitis and abscess formation with this modality is not possible. Additionally, because prevertebral space dimensions can change independently with crying, swallowing, expiration, and neck flexion, best diagnostic results depend on attention to proper technique, which includes imaging during inspiration and with adequate extension [56,59,61]. This requirement can significantly limit the value of neck radiographs in an apprehensive child with limited neck movement.

Because of the limitations associated with radiographs, CT scanning of the neck continues to be the method most commonly used in the diagnosis of deep neck infections. However, although CT scans can be very helpful in assessing the location and extent of infection, the distinction between cellulitis and abscess also may not always be possible. Various studies have reported sensitivities of 43% to 100% and specificities of 57% to 88% for the detection of retropharyngeal abscesses by CT scanning [56,57,59,62]. False-positive reports have been noted with necrotic lymph nodes. In one series, 25% of patients whose condition elicited initial suspicion for abscess formation were at the time of surgical drainage found not to have a pus collection [56,57]. To address this shortcoming, some authors have advocated considering areas of hypodensity greater than 2 cm³ as more suggestive of abscess formation [63]. Additionally, “scalloping” or the irregularity of the abscess wall on CT scans has been suggested as a late indicator of impending rupture and the need for surgical intervention [64].

Ultrasonography of the neck has been advocated by some authors as a sensitive nonradiating alternative to CT for evaluating deep neck infections as well as monitoring their progression. This modality, especially when combined with color Doppler, can diagnose infections in the early nonsuppurative stage and thereby allow for earlier antibacterial treatment and a reduced number of unnecessary surgeries [56,65,66]. Ultrasonography also has the added benefit of being able to distinguish between adenitis and abscess as well as serve as a tool for guided intraoperative aspiration and drainage [67].

Although the traditional management of deep neck infections had relied more heavily on surgical intervention, more recent evidence suggests that the majority of patients can be managed successfully with early the administration of intravenous antibiotics alone [54,56,58,62,68]. Success rates with a trial of conservative medical management have been reported to be as high as 75% to 90% [56,62]. The selection of the initial antibiotic regimen should be directed by regional bacterial sensitivity patterns and the need for the coverage of multiple mixed aerobic and anaerobic pathogens [56]. Surgical incision and drainage should be reserved for cases that do not respond to medical therapy or those in
whom persistent or large abscesses have been noted [54,56,68]. In addition to early antibiotic therapy, careful monitoring and supportive therapy in an institution prepared for airway support can help to ensure successful recovery [54,58,59,68].

Although rare, complications of deep neck infections in children can pose a serious risk of morbidity and mortality. The complications most commonly reported include airway compromise, aspiration pneumonia, extension of infection to adjacent structures or compartments, and the recurrence of abscesses. Accordingly, most cases of complications are related to inadequately treated abscesses that may result in spontaneous rupture [54,56,58].

Peritonsillar infections

Peritonsillar infection, noted most commonly in patients who have chronic or recurrent tonsillitis, represents an extension of infections from the tonsils [32]. Unlike pharyngitis and tonsillitis, which are noted frequently in all age groups, peritonsillar infections are more common in adolescents and adults [54]. The cause of these infections tends to be polymicrobial and frequently includes both aerobic and anaerobic bacteria [32,54]. Management of these infections is based on their classification into the more common peritonsillar cellulites (PTC) and the less frequent peritonsillar abscesses (PTA) or quinsy [32,69,70].

Typical signs and symptoms of peritonsillar infections include fever, trismus, poor oral intake, drooling, and uvular deviation [32,54,71]. Among these, uvular deviation combined with trismus can aid in differentiating between PTA and PTC [70]. However, because the clinical presentations of PTC and PTA are very similar, the imaging studies are frequently needed to further delineate the degree and extension of infection. CT with contrast enhancement has been the traditional choice for confirmation of abscess formation in children [32,71]. However, some authors have advocated ultrasonography of the neck as a highly sensitive, inexpensive, and nonradiating alternative modality for differentiation between PTC and PTA [72].

The treatment of peritonsillar infections includes antibiotic therapy with or without incision and drainage. Because of their polymicrobial nature and the frequent implication of β-lactamase-producing bacteria, antibiotics with activity against this group of organisms should be selected [54]. Although early antibiotic therapy can abort the formation of an abscess in patients who have peritonsillar cellulites, once pus has formed, an incision and drainage procedure is mandatory [32,54,69,71]. Several studies have found that the incision and drainage of PTAs under conscious sedation in a pediatric emergency department with skilled personnel can be safe and effective [73–75]. Complications of peritonsillar infections include airway obstruction, mediastinitis, and Lemierre syndrome [54,76,77]. The latter is a potentially fatal condition that is usually caused by F necrophorum and is characterized by thrombophlebitis of head and neck veins and systemic dissemination of septic emboli [76,78].
Lower airway infections

Definition

Pneumonia has been defined as pulmonary infiltrates as observed on a chest radiograph or by clinical signs and symptoms [79,80]. The World Health Organization considers a diagnosis of pneumonia using clinical signs such as tachypnea (respiratory rate ≥ 50 breaths/min in infants less than 1 year of age and ≥ 40 breaths/min in children more than 1 year of age), retractions, or cyanosis [81,82]. Tachypnea may also be seen in conditions such as asthma and bronchiolitis [83]. Bronchiolitis is defined as an acute lower respiratory tract infection usually in children less than 2 years of age that results in inflammation and obstruction of the peripheral airways [84].

Pathophysiology

Bacterial pneumonia is seen after the inhalation or aspiration of pathogens. Less commonly, it can also occur after hematogenous spread. An inflammatory reaction follows, with the release of fluid and polymorphonuclear white blood cells into the alveoli, followed by fibrin and macrophage deposition over days. Viral pneumonia occurs mainly after the inhalation into the lung of infected droplets from upper airway epithelium. RSV, the major cause of bronchiolitis, is transmitted by contact with infected nasal secretions and more unusually by aerosol spread [84]. In both viral pneumonia and bronchiolitis, the resulting inflammatory response causes epithelial cells to slough into airways, thereby causing bronchial obstruction and hyperinflation. Inflammation mostly affects the smaller caliber peripheral airways, essentially sparing the alveoli in bronchiolitis. Lymphocytes infiltrate in the peribronchial and peribronchiolar epithelium, promoting submucosal and adventitial edema in bronchiolitis. Mucous plugs and cellular debris accumulate because of impaired mucociliary clearance, leading to ball-valve obstruction and subsequent hyperinflation [85]. Viral pneumonia may also predispose infected children to bacterial pneumonia because of damage to mucosal barriers.

Pneumonia in the neonatal period may occur as a result of infection or colonization of the nasopharynx or conjunctiva by organisms found in the mother’s vaginal tract. Lung injury from aspiration or host immunologic factors such as in cystic fibrosis [86] may also predispose the child to pneumonia.

Epidemiology

Pneumonia is diagnosed in approximately 4% of children in the United States per year, but the attack rate varies by age. The annual rate of pneumonia is 35 to 40 cases per 1000 children younger than 1 year of age, 30 to 35 cases per 1000 children 2 to 4 years of age, 15 cases per 1000 children aged 5 to 9 years, and less than 10 per 1000 for children older than 9 years of age [79,80,87,88].
Compared with developing nations, most cases of pneumonia in the United States have lower mortality rates and are treated on an outpatient basis [89]. Some populations are at a higher risk for pneumonia, including children who have cystic fibrosis, aspiration syndromes, immunodeficiencies, neurologic impairments, or congenital or acquired pulmonary malformations [90–93]. Bronchiolitis affects nearly all children by the age of 2 years and is the leading cause of hospitalization for infants less than 1 year old. Between 1992 and 2000, bronchiolitis accounted for approximately 1,868,000 ED visits for children less than 2 years of age. For this population in the United States, the overall rate was 26 per 1000 children (95% CI, 22%-31%); the rate of ED visits was 31 per 1000 (95% CI, 26%-36%); and the overall admission rate was 19% [94].

**Causes**

Many microbiologic agents cause childhood pneumonia, but given the difficulty of establishing the definitive cause, the most likely pathogens are usually inferred from factors such as age, season, and clinical characteristics. Radiographs, blood tests, and cultures are of limited value to the emergency department physician in determining the cause of pneumonia. Depending on the specific laboratory testing used, such as culture, antigen detection, or serology, the microbial cause of pneumonia was found only in 20% to 60% of cases in a European review [95].

*S pneumoniae* has been found to be the most common cause of bacterial pneumonia and RSV the most common viral cause. In children hospitalized with pneumonia, viral infections become less common with increasing age, whereas the age-specific incidence of bacterial infections remains relatively constant [96]. In both hospitalized and ambulatory children, *S pneumoniae* is the most common bacterial pathogen identified in children less than 4 years of age [97]. RSV infection is most often the cause of bronchiolitis, but contributing pathogens include *Mycoplasma* organisms and other viruses, including parainfluenza virus, influenza virus, rhinovirus, adenovirus, and paramyxovirus (measles) [84].

*M pneumoniae* and *C pneumoniae* have been isolated more frequently in children 5 to 9 years of age and 10 to 16 years of age, overall [98,99]. Estimates of the percentage of pneumonias caused by *M pneumoniae* vary from 7% to 30% for children 5 to 9 years old to 14% to 51% in children 10 to 16 years old. *C pneumoniae* is implicated less frequently, ranging from 9% to 13% for children 5 to 9 years old to 14% to 35% for children 10 to 16 years old [98,99]. Studies in other areas of the United States have confirmed that *M pneumoniae* and *C pneumoniae* are more common causes of pneumonia in children over 5 years old [100,101].

Overall, *S pneumoniae* causes most cases of bacterial pneumonia in infants and children, and viruses become less prevalent with age, whereas infection from *Mycoplasma* and *Chlamydia* organisms are more commonly found with increasing age, particularly in adolescents. Mixed viral and bacterial infection has been reported in 16% to 34% of children with pneumonia [95,96]. Depending
on the clinical picture, the emergency physician should also consider the rarer causes of bacterial pneumonia such as \textit{Staph aureus}, \textit{Moraxella catarrhalis}, \textit{H influenzae} (type b, encapsulated types other than b, and nontypable), group A and B streptococci, \textit{Mycobacterium tuberculosis}, and \textit{Bordetella pertussis} \cite{102}.

\section*{Clinical characteristics}

\textbf{Streptococcus pneumoniae}

The clinical spectrum of signs and symptoms can be broad with pneumonia secondary to \textit{S pneumoniae} infection, ranging from mild, nonspecific symptoms of emesis, cough, and abdominal pain to severe respiratory distress. Tan and colleagues \cite{103} have found the most common presenting symptoms to be fever and nonproductive cough, followed by tachypnea, malaise, lethargy, and rhinorrhea. The most common findings were decreased breath sounds, crackles, or rales. Although the classic finding of radiographic lobar consolidation is often seen, its absence does not eliminate the possibility of pneumococcal pneumonia. In this study, more than half the patients had lobar consolidation, and 38\% of patients had effusions. Empyema, a known complication of pneumonia caused by \textit{S pneumoniae}, was present in 14\% of the patients.

Antibiotic resistance to \textit{S pneumoniae}, particularly penicillin and cephalosporin resistance, has been noted because the early 1990s. It is an important factor when considering invasive disease and antibiotic selection. As with crude rates of colonization and rates of invasive infection, exposure to antibiotics, young age, and day-care attendance are associated with a greater likelihood of colonization or infection with a penicillin-resistant \textit{S pneumoniae} isolate \cite{104,105}. Serious complications and treatment failures are rare with pneumonia compared with other invasive infections and otitis media \cite{106}. The outcomes of therapy for pneumonia were not found to be different between patients who had penicillin-susceptible and penicillin-nonsusceptible isolates, who were treated with traditional antibiotics \cite{103}.

The American Academy of Pediatrics recommends standard antibiotic therapy for noncritically ill, immunocompetent patients who have possible invasive pneumococcal infections other than meningitis. Additional initial broader antibiotic coverage for potential penicillin-nonsusceptible strains could be considered for patients who have lower airway disease who are critically ill, including those with severe multilobar pneumonia with hypoxia. The coverage for possible penicillin- and cefotaxime- or ceftriaxone-nonsusceptible strains could be considered with vancomycin. If vancomycin is used, it should be stopped as soon as antibiotic susceptibilities demonstrate effective alternative agents \cite{106}. Generally, oral therapy with low- or high-dose amoxicillin or second-generation cephalosporins such as cefuroxime should be effective for the initial management of outpatient pneumococcal pneumonia \cite{107} in children less than 5 years of age, excluding the neonatal period. Macrolide antibiotics, including azithromycin, are
also appropriate but usually are not necessary given their broad-spectrum coverage [83]. Hospitalized children who are suspected of having pneumococcal pneumonia can be treated with intravenous penicillin, ampicillin, or cefuroxime, unless they are critically ill. Cefotaxime, ceftriaxone, and clindamycin can also be considered when a penicillin-resistant organism is suspected. When a pneumococcal isolate is resistant to cefotaxime or ceftriaxone, clindamycin or vancomycin is recommended [108].

Bronchiolitis

Bronchiolitis is a common, usually self-limited, lower respiratory tract infection caused by RSV that is observed in all geographic areas and usually seen between the months of October through April. There are two strains of RSV, A and B, with numerous genotypes and serotypes. The incubation period varies from 2 to 8 days and, after a prodrome of several days, there is an acute illness characterized by rhinorrhea, cough, and low-grade fever. Young children may be restless or lethargic and drink less than normal. The physical examination is marked by tachypnea, accessory muscle use, wheezes, or crackles. Hypoxemia may be seen secondary to ventilation-perfusion mismatch. The complications of apnea and respiratory failure are seen most frequently in young infants and those with underlying conditions such as prematurity, bronchopulmonary dysplasia, chronic lung disease, congenital heart disease, or immunodeficiencies [84]. In typical cases, laboratory testing or chest radiographs are generally not useful [109] but should be considered if the diagnosis is unclear because viral myocarditis, congenital heart disease, and pneumonia may have similar clinical presentations. Because the diagnosis of bronchiolitis is a clinical one, routine RSV antigen testing has little value in management. Respiratory viral antigen testing may be helpful for infection control if patients are admitted to inpatient units [110]. Oxygen saturation should be performed routinely because cyanosis is difficult to detect and an oxygen saturation of less than 95% was found to be the single best predictor of severe illness in a study of outpatients who had bronchiolitis [111]. The treatment for bronchiolitis is supportive, including intravenous hydration, supplemental oxygen, nasal suction, and mechanical ventilation for respiratory failure [112]. Although many therapies have been attempted for bronchiolitis, including ribavirin, interferon alfa, vitamin A, montelukast, β agonists, epinephrine, and corticosteroids, the optimal therapy is still controversial [113–118]. Although some studies, including a meta-analysis and a systematic review, all failed to show significant clinical improvement with β agonists [116,119], other studies have reported a positive effect [120,121]. Racemic epinephrine may be more effective in the treatment of bronchiolitis because of its additional vasoconstrictor effects in reducing microvascular leakage and mucosal edema. Infants with bronchiolitis, who are treated with nebulized racemic epinephrine, showed more improvement than those treated with nebulized albuterol without an increase in side effects [122,123]. Nebulized ipratropium bromide has not been shown to be of added benefit in treating
bronchiolitis [124]. Confounding results have also been seen with corticoste-
roids. Although the majority of studies have not demonstrated a benefit with oral,
nebulized, or parenteral steroid therapy [125–127], some studies suggest that
steroid therapy may be effective in improving recovery [128,129]. In actual
hospital practice, short-acting β agonists have been shown to be used 53% to 73%
in various studies [94,130]. Steroids are used 8% to 13% [94,130–132]. Many
physicians will attempt a trial of nebulized albuterol for children with bron-
chiolitis with mild respiratory distress; if there is moderate or severe distress,
then nebulized racemic epinephrine and possibly steroids are used. The main-
stay of treatment remains supportive care with oxygen and intravenous fluids
as needed.

Atypical pneumonia

The term “atypical pneumonia” has referred, for the most part, to pneumonia
caided by organisms other than the historically common and more easily cultured
bacteria, including most often M pneumonia, C pneumoniae, Chlamydia species
(eg, burnetti, trachomatis, and psittaci), Legionella pneumophila, Bordetella
pertussis, and viral pathogens [102,133,134]. M pneumoniae and C pneumoniae
have been reported to be the most frequent causes of community-acquired
pneumonia in children age 5 years or older [98,99,135].

Pneumonia caused by C pneumoniae and M pneumoniae has been reported as
relatively mild and rarely resulting in hospitalization; however, Legionella spp,
which are the exception and are often classified as the causative organisms in
atypical pneumonia, usually cause more acute and severe symptoms [134]. The
pneumonias caused by M pneumoniae and C pneumoniae can be further dif-
ferentiated clinically by wheezing on presentation [136]. Chest radiographic
findings are less likely to be lobar for atypical pneumonias than those caused by
S pneumoniae. Radiographic abnormalities in M pneumoniae vary, but bilateral,
diffuse infiltrates are common [137]. Pleural effusions are also less likely to be
seen with M pneumoniae and C pneumoniae [136]. The treatment for atypical
pneumonia, covering M pneumoniae and C pneumoniae, includes macrolide
agents in any age group and tetracyclines in children older than 8 years of age.
Fluquinolones (including levofloxacin and oflaxacin but not ciprofloxacin) also
are appropriate in children older than 16 years of age [138,139].

Chlamydia trachomatis

C trachomatis can be transmitted from the genital tract of infected mothers
to their newborn infants. Following vaginal delivery, 50% of infants will acquire
the organism. The nasopharynx and the conjunctivae are the most commonly
infected sites, but not all colonized newborns progress to overt infection. Pneu-
monia occurs in 5% to 20% of infected infants. Chlamydia pneumonia typically
presents between 2 and 19 weeks after birth, making C trachomatis the most
common cause of infection in the 4- to 11-week-old group. A staccato cough,
tachypnea, and crackles are often present, but fever is not usually present. Infants who have pneumonia caused by *C trachomatis* often present with bilateral diffuse infiltrates on radiography and peripheral blood eosinophilia. Treatment is initiated based on clinical suspicion; a culture of the nasopharynx or by the detection of bacterial antigens or DNA is confirmatory. A 14-day course of oral erythromycin is recommended but is known to have an efficacy of only 80%. Oral sulfonamides are appropriate in children 2 months old or older [140].

**Pertussis**

Infection by *B pertussis*, an important respiratory pathogen, may lead to pneumonia. *B pertussis* remains endemic in the United States and continues to cause epidemics, despite widespread vaccination of the population in childhood. However, most cases occur in individuals who have not been adequately vaccinated [141], and infection in vaccinated individuals is usually mild [142]. However the incidence of pertussis among adolescents is rising, and this group forms a reservoir of infection for young infants [143]. A case is defined clinically as an acute cough illness lasting at least 14 days and accompanied by paroxysms of coughing, inspiratory whoop, or post-tussive emesis. If there is exposure to a confirmed case, 14 days of cough is the only requirement for diagnosis [144]. Paroxysmal cough and post-tussive vomiting are the most common presenting symptoms, but fever is rare. In one study, the median age on presentation was 4.1 years; and the overall complication rate was 5.8%, with pneumonia being the most common complication [141]. The complication rate was higher (23.8%) in infants less than 6 months of age. Of all infants less than 6 months of age, the most common complication was apnea (15.9%) [141]. The Red Book reports a fatality rate of 1.3% in children younger than 1 month and 0.3% in children 2 to 11 months of age. Other complications described in a German study include pneumonia (29%) and vomiting (50%) [141]. New seizures occurred in 2% of cases of children less than 1 year of age with pertussis reported to the US Centers for Disease Control and Prevention between 1990 and 1996 [145]. The classic presentation described for pertussis after an incubation period of 7 to 10 days consists of a catarrhal, paroxysmal, and convalescent phase. Young and recently vaccinated children may present atypically. In the catarrhal phase, coryza is present with mild cough lasting 1 to 2 weeks. Fever is absent or low grade, but cough worsens and becomes paroxysmal. These series of coughs may be associated with gagging and cyanosis and last for 2 to 3 weeks before becoming less severe. In the convalescent phase, the cough decreases over weeks to months. Infants with pertussis present atypically, with a short or absent catarrhal phase. Cough with a characteristic whoop is uncommon compared with older children [146]. Children who have been vaccinated and have developed pertussis had cough for a shorter period and less apnea and cyanosis [147]. The clinician should be alerted to the possibility of pertussis based on the typical staccato cough in which multiple forceful coughs proceed in rapid progression followed by a deep inspiration (and possible whooping sound and post-tussive emesis).
These paroxysms may be followed by periods of calm, when the child appears relatively well. Support for the diagnosis of pertussis is a CBC with a high proportion and absolute number ($\geq 10,000$ lymphocytes/$\mu$L) [148]. Young infants should be treated promptly and possibly admitted (given the risk of apnea), based on clinical suspicion, while confirmatory nasopharyngeal cultures are ordered (this will guide prophylaxis of household contacts). Treatment is supportive and includes hydration, nutrition, oxygen, and cardiorespiratory monitoring for complications. Antibiotic treatment during the early often-unrecognized catarrhal stage is required to ameliorate disease, but treatment initiated after paroxysms have been established is still recommended to limit disease spread. The treatment of choice is erythromycin estolate therapy for 14 days, whereas the macrolide agents clarithromycin for 7 days and azithromycin for 5 to 7 days are likely to be effective and better tolerated [149]. Trimethoprim-sulfamethoxazole/SMX is also considered an alternative in children older than 2 months of age [142]. The hospital infection control team and the local public health authorities should be notified of all cases of suspected and confirmed pertussis, and recommendations for the care of exposed people should be followed carefully.

**Neonatal pneumonia**

Neonatal pneumonia and neonatal sepsis are very different entities than community-acquired pneumonia in older children. These neonates typically present with tachypnea, grunting, and retractions. Nonspecific symptoms such as irritability and poor feeding may also be seen. They may have a fever, or they may present with hypothermia. The most common bacterial agent is group B streptococci, but *Listeria monocytogenes* and other bacteria that cause pneumonia in older infants can also be seen. Gram-negative enteric bacteria can also cause pneumonia in neonates older than 1 week, usually from nosocomial infection [102]. The treatment is supportive and includes broad-spectrum antibiotic coverage such as parenteral ampicillin and gentamicin or ampicillin and cefotaxime.

**Diagnosis**

**Clinical presentation**

Pneumonia can have different clinical presentations, depending on the cause and the patient’s age. In most cases of bacterial community-acquired pneumonia, children have a sudden onset of fever, tachypnea, and cough. This constellation may be preceded by symptoms of a minor upper respiratory tract infection.

Because neonates may be discharged from the newborn nursery within 24 hours of delivery, the emergency physician may need to identify the neonate who has neonatal pneumonia or neonatal sepsis. These neonates typically present
with tachypnea (respiratory rate greater than 60 breaths/min), grunting, and retractions. Nonspecific symptoms such as irritability and poor feeding may also be seen. Hypothermia rather than fever may be seen in this population.

Infants greater than 1 month of age with pneumonia may have similar symptoms, but cough is a more prominent symptom. Unlike newborns, infants who are infected with bacterial pneumonia are more often febrile. Infants with pneumonia caused by viruses or atypical organisms may be afebrile and have wheezing respirations.

Toddlers and preschool children who have pneumonia will usually present with cough, and vomiting, chest pain, and abdominal pain may also be seen. Sometimes, fever and tachypnea may be seen with few other respiratory symptoms [150]. Older children and adolescents may present with symptoms similar to younger children and, in addition, may have generalized symptoms such as headache and abdominal pain. In a study of a series of patients who had bacteremic pneumococcal pneumonia, 28% of patients had no respiratory symptoms, 6% of patients presented with only gastrointestinal symptoms in addition to fever, and 4% of patients had only fever. Tachypnea was recorded in 19% and crackles in 14% of patients [150]. Older children and adolescents may also complain of chest pain, which is often pleuritic in nature and localized [151,152].

In 1997, a Canadian task force [153] reviewed the literature and published evidence-based guidelines for diagnosing pediatric pneumonia. They concluded that the absence of each of four signs (ie, respiratory distress, tachypnea, crackles, and decreased breath sounds) accurately excludes pneumonia (level-2 evidence). When an attempt was made to validate these guidelines in pediatric patients in an urban emergency department, the guidelines were only 45% sensitive (95% CI, 35%-58%) [81] and 66% specific (95% CI, 18%-34%) for diagnosing pneumonia. Positive and negative predictive values were 25% (95% CI, 18%-34%) and 82% (95% CI, 77%-87%), respectively [154]. A recent study has found that patients were more likely to have focal infiltrates on chest radiography if there was a history of fever, tachypnea, increased heart rate, retractions, grunting, crackles, or decreased breath sounds [155]. However, this finding still needs to be validated, and a reliable diagnosis of pneumonia is still difficult and requires careful attention to the patient’s individual clinical characteristics.

**Radiologic and laboratory testing**

Chest radiography remains the diagnostic test of choice in tertiary care hospitals, and although they cannot be used to discriminate reliably between bacterial and viral pneumonia [154,156,157], typical patterns are seen. Most radiologists support findings of an alveolar or lobar infiltrate with air bronchograms to be an insensitive but fairly specific indication of bacterial pneumonia [158,159]. Unilobar or round infiltrates may be seen with pneumococcal pneumonia [150,160]. Pneumatoceles may be present with severe necrotizing pneumonia, such as that caused by *Staph aureus* [160]. Viral pneumonia infections
usually are characterized by diffuse interstitial infiltrates, hyperinflation, or atelectasis. Peribronchial thickening or hilar adenopathy may also be seen [160,161]. Chest radiographs are often normal in pertussis, but perihilar infiltrates of the right middle lobe or lingual region can also be seen [160]. Chlamydia pneumonia is characterized by diffuse interstitial markings and hyperinflation [160,162]. Radiographic findings in patients who are infected with *M pneumoniae* may be normal or have characteristics of viral or bacterial pneumonia as described above. Pleural effusions can also be seen with *M pneumoniae* [160,163] but are more common with *S pneumoniae* and *Staph aureus*. Pediatric tuberculosis can also produce radiographic appearances that vary, which is caused in part by the time point at which the radiograph is taken during the disease’s progression. The most common finding is mediastinal or hilar adenopathy. Lobar consolidation as well as pleural effusions can also be seen. In later stages, the classic findings of calcification, focal fibrosis, and cavitory lesions in the upper lobes may be seen, even in children. Miliary tuberculosis is characterized by a diffuse mottling on chest radiographs [164]. Infiltrates may be seen in the right upper lobes for infants and in the posterior or bases of the lung for older children in aspiration syndromes [160].

Other laboratory tests are marginally helpful in distinguishing bacterial from nonbacterial pneumonia. Clinical and epidemiologic factors are most important in reaching a diagnosis; whereas radiographs and selected blood tests can clarify the diagnosis in certain instances [165]. The C-reactive protein level may be elevated in bacterial pneumonia [166,167], and likewise, elevated absolute neutrophil count and elevated white blood cell count are seen frequently with bacterial pneumonia, especially pneumococcal pneumonia [150,168].

Blood cultures are rarely positive in pneumonia because ≤ 10% of children who have pneumonia will have bacteremia, but blood culture results may be helpful in the management of children who are suspected of having pneumococcal pneumonia who do not respond to initial treatment [96,169]. Serologic blood tests are of limited clinical value to the emergency physician. One exception is the cold agglutinin test, which can be performed at the bedside for children over the age of 3 with suspected pneumonia caused by *Mycoplasma*. Although there are false-positive results with viral disease, the test is positive in 70% to 90% of cases of *Mycoplasma* infection [170]. Sputum cultures should be obtained for suspected bacterial pneumonia in preadolescent and adolescent children, but adequate specimens are difficult to obtain in younger children. Many tests performed on nasopharyngeal samples, such as enzyme-linked immunosorbent assays or direct fluorescent antibody assays, are sensitive and specific for detecting viral causes of lower respiratory disease such as respiratory syncytial virus and influenza. Finally, a purified protein derivative skin test should be ordered and a follow-up for reading arranged, if tuberculosis is suspected.

Pulse oximetry measurements of oxygen saturation can correlate with clinical signs of pneumonia (tachypnea and crepitations) in undeveloped countries [171,172] and in areas of high altitude [173]. In developed countries, measure-
ments of oxygen saturation should be useful to the emergency physician in supporting the diagnosis of pneumonia, and influence management decisions such as admission or outpatient treatment.

Treatment

In the ED, the decision to treat a patient for pneumonia is based usually on epidemiologic, clinical, and radiographic findings and other laboratory data as adjuncts. It is unusual for the exact cause to be known at the time of initial treatment. Empiric antibiotic treatment should be based on the likelihood of bacterial disease. For instance, if a virus is detected, usually by rapid antigen detection techniques from a nasopharyngeal aspirate, during a seasonal peak for that virus in a mildly ill child, then withholding antibiotic therapy would be appropriate, despite the presence of a streaky infiltrate on a chest radiograph. However, it is important to recognize that there may be infection with bacterial and viral co-pathogens. Table 1 summarizes the most appropriate first- and second-line therapies for hospitalized and ambulatory pediatric patients who have presumed community-acquired and neonatal pneumonia.

The treatment for lower respiratory illness is supportive and should include supplemental oxygen titrated to an oxygen saturation greater than 95% and albuterol administration by nebulization if wheezing is heard. Intubation and positive pressure ventilation is required for respiratory failure and apnea, which is seen with RSV pneumonia or pertussis. Intravenous fluids should be given to patients who have tachypnea and signs of increased work of breathing or moderate to severe dehydration.

Although many cases of pneumonia can be treated on an ambulatory basis, admission should be considered for patients who have pneumonia who are not responding to outpatient therapy. Empyema may be the cause of prolonged or secondary fever, despite appropriate therapy in a child with pneumonia, and is an important complication of *Staph aureus*, *S pneumoniae*, *H influenzae*, group A *Streptococcus*, *Legionella* organisms, *Mycob tuberculosis*, and other pathogens [174]. All infants younger than 1 month of age should be admitted, and infants under the age of 6 months or children whose caretakers may be poorly compliant with therapy should also be seriously considered for admission. Patients should be admitted to the hospital if there are significant clinical signs such as respiratory distress, dehydration, or hypoxia, but an observation unit in the emergency department can be used if clinical improvement is expected to occur rapidly [175]. Children with other chronic diseases such as congenital heart disease, chronic lung disease, immunodeficiency, or neurologic impairment who present with a new onset respiratory disease are also strong candidates for admission. Finally, patients who have other complications (eg, pneumatoceles) or potential complications (paroxysmal phase of pertussis with apnea) should be admitted for further therapy and observation.
<table>
<thead>
<tr>
<th>Causes and treatment</th>
<th>0–4 wk</th>
<th>4–8 wk</th>
<th>8–12 wk</th>
<th>12 wk–4 y</th>
<th>5 y–adolescence</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Causes (in order of prevalence)</strong></td>
<td>Group B streptococci</td>
<td>\textit{C. trachomatis}</td>
<td>\textit{C. trachomatis}</td>
<td>Viruses (RSV, parainfluenza, influenza, adenovirus, rhinovirus)</td>
<td>\textit{M. pneumoniae}</td>
</tr>
<tr>
<td></td>
<td>Gram-negative enteric bacterial</td>
<td>Viruses (RSV, parainfluenza)</td>
<td>Viruses (RSV, parainfluenza)</td>
<td>\textit{S. pneumoniae}</td>
<td>\textit{C. pneumoniae}</td>
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<td></td>
<td>\textit{L. monocytogenes}</td>
<td>\textit{S. pneumoniae}</td>
<td>\textit{S. pneumoniae}</td>
<td>\textit{H. influenzae} (non-b type)</td>
<td>\textit{S. pneumoniae}</td>
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<tr>
<td></td>
<td>\textit{B. pertussis}</td>
<td>\textit{B. pertussis}</td>
<td></td>
<td>\textit{M. catarrhalis}</td>
<td>Viruses (RSV, parainfluenza, influenzavirus, adenovirus, rhinovirus)</td>
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<td></td>
<td>Group B streptococci</td>
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<td>Group A streptococci</td>
<td>\textit{M. pneumoniae}</td>
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<td></td>
<td>Gram-negative enteric bacteria</td>
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<td>\textit{Mycobacterium tuberculosis}</td>
<td>\textit{Mycobacterium tuberculosis}</td>
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<td></td>
<td>\textit{L. monocytogenes}</td>
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<tr>
<td><strong>Outpatient (in order of initial choice)</strong></td>
<td></td>
<td>For \textit{B. pertussis} or \textit{Chlamydia} erythromycin or other macrolides</td>
<td>For \textit{B. pertussis} or \textit{Chlamydia}, erythromycin or other macrolides, sulfonamides</td>
<td>Amoxicillin or amoxicillin/clavulanate or cefuroxime</td>
<td>Macrolides or tetracyclines (( \geq 8 ) y old)</td>
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<td></td>
<td></td>
<td></td>
<td>For \textit{S. pneumoniae} see next column</td>
<td>Macrolides</td>
<td>Fluoquinolones (( \geq 16 ) y old)</td>
</tr>
<tr>
<td><strong>Inpatient</strong></td>
<td>Neonatal pneumonia or sepsis, ceftriaxone or cefotaxime plus ampicillin</td>
<td>Neonatal pneumonia or sepsis, ceftriaxone or cefotaxime plus ampicillin</td>
<td>For \textit{S. pneumoniae} see next column</td>
<td>Penicillin, ampicillin, or cefuroxime</td>
<td>Macrolides</td>
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<td></td>
<td>Cefotaxime or ceftriaxone</td>
<td>Cefuroxime plus macrolides</td>
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<td>Clindamycin</td>
<td>Macrolides plus cefotaxime</td>
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<td></td>
<td></td>
<td>Vancomycin until alternative susceptible agents are identified</td>
<td>or ceftriaxone or clindamycin</td>
</tr>
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<td></td>
<td>Vancomycin</td>
<td>Vancomycin</td>
</tr>
</tbody>
</table>
Summary

Upper and lower airway infections are common in pediatrics and are usually diagnosed clinically based on the history, physical examination, and specific epidemiologic characteristics. Changes in pneumococcal resistance and immunization practices with pneumococcal and influenza vaccines will continue to change the incidence rate and causative findings of pneumonia. The treatment of airway infections is always supportive, but specific management strategies for certain pathogens, including the selection of antibiotics, bronchodilators, steroids, and inpatient or outpatient disposition, depend on the disease, the age, and the clinical characteristics of the host.

References


[65] Ben-Ami T, Yousefzadeh DK, Aramburo MJ. Pre-suppurative phase of retropharyngeal infec-


