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68 Bronchiolitis

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SHORT VIEW SUMMARY

Definition
- Bronchiolitis is small airway inflammation/obstruction most frequently caused by infection with respiratory syncytial virus (RSV) in the first years of life.

Epidemiology
- RSV bronchiolitis occurs in winter epidemics in temperate climates and sporadically in the tropics.
- Bronchiolitis is a leading cause of hospitalization in the first year of life in the developed world.

Microbiology
- Many other respiratory viruses may cause bronchiolitis, including human metapneumovirus, influenza, parainfluenza, adenovirus, coronavirus, and bocavirus.

Diagnosis
- Diagnosis is clinically based on presence of RSV in the community, initial episode of wheezing, and evidence of upper respiratory infection.
- Other causes of wheezing in early childhood should be excluded, such as congenital heart disease with failure, foreign body aspiration, dysphagia, and asthma.
- Apnea may occur early in the course of viral bronchiolitis, usually in infants younger than 44 weeks' postconceptional age.

Therapy
- Therapy is supportive and includes hydration, oxygen, and respiratory support as needed.
- Corticosteroids and bronchodilators are not generally beneficial.
- Hypertonic saline aerosols delivered three times daily may hasten recovery but have not been widely adopted.
- Respiratory support by high-flow nasal cannula may prevent or delay intubation in patients with apnea or respiratory failure.

Prevention
- Careful attention to hand sanitation is important in limiting spread of RSV infection during epidemics.
- Monoclonal antibody prophylaxis may prevent or mitigate infection in high risk infants.

With bronchiolitis we have to contend
with illness that's now and disease that comes then;
For many such infants a mold has been cast,
perhaps by their unborn and unknown past,
which destines that they shall in time wheeze again.
For them this disease is the far, boding knell
Of vulnerable lungs to a microbe's dark spell.

—Caroline Breese Hall (1939-2013, the original author of much of this chapter and to whose memory it is dedicated)

Bronchiolitis is the most common acute viral lower respiratory tract illness occurring during the first 2 years of life. Much interest and effort have been aimed at determining the pathogenesis and management of this illness among hospitalized and outpatient children. Despite this, concerns and controversies continue.

Bronchiolitis has acquired during its long lineage a notable number of sobriquets, including "acute catarrhal bronchitis," "interstitial bronchopneumonia," "spastic bronchopneumonia," "capillary or obstructive bronchitis," and, more commonly, "wheezy bronchitis" and "asthmatic bronchiolitis." The diversity of these terms is indicative of the past and ongoing confusion and difficulty in clinical differentiation of bronchiolitis from asthma and infectious asthma. These entities usually refer to repeated episodes of wheezing that may be triggered by infectious agents and tend to occur in children beyond infancy.

The definition of bronchiolitis varies but usually applies to children younger than 2 years of age with a first episode of wheezing commonly associated with fever, cough, rhinorrhea, and tachypnea.[1,2] Consensus does exist, however, that bronchiolitis continues to impose a major and increasing health care burden. Bronchiolitis has been estimated to be the leading cause of all hospitalizations among infants in the United States.[3,4,5]

ETIOLOGY

Bronchiolitis was not recognized as a distinct entity until the 1940s and was initially thought to be caused by bacteria.[6,7] Viruses are now known to be the prime cause of the syndrome and the associated characteristic pathology of the lower respiratory tract. Respiratory syncytial virus (RSV) is the major pathogen identified. The roles played by other viral agents are controversial and depend partly on the population being studied and the laboratory methods used for detection. Correlation with disease is particularly problematic because viruses that commonly infect this young age group can cause high rates of asymptomatic infection or prolonged shedding, including adenoviruses and human bocavirus (hBoV). In addition, some agents, such as rhinoviruses, may trigger asthmatic airway inflammation and bronchospasm without causing the small airway pathology characteristic of infection of the lower respiratory tract with bronchiolitis.

RSV has been identified as the principal agent in two thirds of the cases of bronchiolitis, and in hospitalized patients the proportion is likely higher.[1,8,9] Other viruses that commonly have been identified as single or coinfecting agents among children with bronchiolitis include human metapneumovirus (hMPV), the parainfluenza viruses, influenza viruses, rhinoviruses, human coronaviruses (hCoV), and hBoV (Table 68-1).

Among the parainfluenza viruses, parainfluenza virus types 1 and 3 are more commonly associated with bronchiolitis in hospitalized children than type 2.[10] Illnesses with hMPV and RSV are generally indistinguishable, although lower respiratory tract illness with hMPV is usually less severe.[11-13] Both RSV and hMPV occur from fall to spring, but activity of hMPV in the community is generally less intense.[11-13] Hospitalized children with hMPV infection tend to be slightly older than children with RSV, and almost all children are infected with hMPV by 5 to 10 years of age.[12] Influenza A and B viruses frequently cause lower respiratory tract disease among children younger than 2 years of age, but the proportion manifesting as bronchiolitis is less than that observed with RSV.[13-15]

With the use of sensitive molecular techniques, additional viruses have been identified in young children with bronchiolitis including hCoVs (see Chapter 157) and hBoV (see Chapter 149). hCoVs are composed of four different strains, including the novel strains hCoV-NL63 and hCoV-HKU1, and have been identified in 7% of young children hospitalized with respiratory illness.[16] However, an equal percentage of asymptomatic children younger than 5 years of age have been observed to shed hCoV.[16] The parvovirus hBoV is increasingly being detected by reverse-transcriptase polymerase chain reaction (RT-PCR) in respiratory and fecal specimens from adults and children

KEYWORDS

apnea; bronchiolitis; human metapneumovirus; hypoxemia infant; hypertonic saline aerosol therapy; respiratory failure; respiratory syncytial virus; respiratory viruses; wheezing

TABLE 68-1 Agents That Cause Bronchiolitis

VIRUS	PROPORTION OF CASES WITH VIRUS DETECTED*	SEASONAL OCCURRENCE†	PRIME AGE OF OCCURRENCE (PEAK AGE OF HOSPITALIZED)
Respiratory syncytial virus	50%-80%	Yearly large outbreaks; late fall to spring	<1 yr (<6 mo)
Human metapneumovirus	3%-19%	Yearly occurrence; late fall to spring	3-18 mo (3-12 mo)
Rhinoviruses	3%-30%	Endemic, yearly; most October-April	All ages (<5 yr)
Influenza	6%-24%	Yearly outbreaks; late fall to spring; onset, prevalence vary with year, strain	1-24 mo (<12 mo)
Parainfluenza viruses types 1-3	7%-18%	Type 1: fall outbreak every other year	<5 yr (<12 mo)
		Type 2: sporadic, mostly fall, winter	<5 yr (<2 yr)
		Type 3: yearly, mostly spring to fall	<2 yr (<12 mo)
Human bocavirus	1%-20%	Endemic, yearly	6-24 mo
Human coronaviruses (NL63, HKU1, 229E, OC43)	1%-10%	All year, most in winter	All ages (<12 mo)
Adenoviruses	3%-20%	Endemic, all year	<5 yr (<2 yr)

*Proportion of respiratory samples with virus detected from young children with bronchiolitis or lower respiratory tract illness. Includes samples in which virus was detected along with another virus.
†Seasonal occurrence in temperate climates.

with a spectrum of upper and lower respiratory illnesses, including bronchiolitis. hBoV has been reported as a sole pathogen in 1% to 6% of young children with bronchiolitis and as a coinfection in 6% to 20% of bronchiolitis cases. However, hBoV is shed for prolonged periods and may be detected long after the clinical manifestations associated with acute infection have resolved.[8,17,18,19,20]

Viruses that are primarily agents of upper respiratory tract infections are also commonly identified in specimens obtained from children with bronchiolitis. Notable among these are the picornaviruses (rhinoviruses and enteroviruses) and adenoviruses. The direct role of these viruses in causing bronchiolitis is uncertain because their high prevalence in this age group makes them common agents of dual infection.[8,9] Rhinoviruses, with more than 100 serotypes, are identified in 3% to 30% of children with bronchiolitis, and more than half the time they are present as coinfecting agents, most commonly with RSV.[8,21] Interpreting the role of rhinoviruses in children with bronchiolitis is complicated by the association of rhinovirus with episodes of wheezing from reactive airway or asthma exacerbations.[21] One group that appears to be at particular risk for severe lower respiratory tract infection due to human rhinoviruses are very-low-birth-weight infants.[22] Enteroviruses have been identified in up to 7% of children hospitalized with bronchiolitis and are usually present as coinfecting viruses.[9]

It remains uncertain whether dual viral infections increase the risk of developing more severe illness with bronchiolitis.[8,9] Studies examining whether hMPV coinfection is associated with more severe RSV lower respiratory tract disease suggest an increase in disease severity among hospitalized children, but this has not been consistently demonstrated.[9,23-25]

EPIDEMIOLOGY

Bronchiolitis shows a yearly seasonal pattern that varies according to geography and climate. In temperate climates, the peak occurrence of cases is during the winter to early spring and usually correlates with the prevalence of RSV in the community. Outbreaks of bronchiolitis are less distinctive in warmer and tropical climates where RSV occurs over longer periods. Bronchiolitis cases in these areas may be seen throughout the year, and the prevalence of cases depends on the seasonal patterns of the known and yet unknown agents associated with bronchiolitis (see Table 68-1).

Bronchiolitis is most common during the first year of life, with the peak attack rate occurring between 1 and 10 months of age and among hospitalized cases between 2 and 5 months of age. Each year, 1% to 3% of infants younger than 12 months of age are hospitalized with bronchiolitis; 80% are younger than 6 months of age. For the period 1997 to 2006, age-specific rates of hospitalization for bronchiolitis remained steady with an overall rate of 26 per 1000 children younger than 1 year of age and 48.9 per 1000 for infants younger than 3 months of age.[4,5] For children older than 1 year of age the rate was 1.8 per 1000.[4] Overall, RSV disease accounts for up to 24% of hospitalizations among children younger than 5 years of age with lower respiratory tract illness. In children younger than 1 year of age presenting to the emergency department with bronchiolitis, up to 40% are admitted with an average length of stay of 3.3 days.[26]

Despite the steady rates of hospitalization for bronchiolitis, the mortality rates associated with bronchiolitis have declined in the United States to fewer than 400 deaths per year.[3,27] Most deaths (79%) occur in infants younger than 1 year, primarily during the first several months of life. Children with chronic conditions, especially conditions affecting cardiopulmonary function, are most likely to develop severe or fatal bronchiolitis. Children with prematurity and the associated chronic lung disease have a fivefold increased risk of developing disease requiring hospitalization than children with no comorbid conditions.[3,28,29]

Multiple demographic, environmental, and biologic factors have been associated with increased rates of hospitalization among otherwise normal children. Bronchiolitis is more common in boys, especially among children with more severe illness, with a male-to-female ratio of about 1.5:1.[30] Other factors that have been associated with a greater likelihood of severe illness include young maternal age, lower cord blood antibody titers to RSV, lower socioeconomic status, tobacco smoke exposure, living in crowded surroundings, having older siblings, daycare attendance, lack of breast-feeding, a predisposition to atopy or hyperreactivity of the airway, and illness caused by RSV.[11,31-33] Infants with specific genotypes predicted to modify innate mucosal immunity are at greater risk of severe RSV infections.[34] A similar mechanism might explain the fact that certain ethnic groups of infants have higher rates of hospitalization for bronchiolitis. Native American and Native Alaskan children have hospitalization rates two to three times higher than those of the general population of U.S. children of the same age.[35] Nevertheless, the major independent risk factor for bronchiolitis requiring hospitalization is young age, within the first 6 months of life.[11,33]

PATHOPHYSIOLOGY

In 1940, Engle and Newns[6] carefully described the pathology of a severe and often fatal lower respiratory tract disease they observed in young infants. They called this "proliferate mural bronchiolitis." Their findings of the generalized involvement of the respiratory epithelium of the small airways have been confirmed as being characteristic of infection-induced bronchiolitis among young children.

The virus initially replicates in the epithelium of the upper respiratory tract, with subsequent spread within a few days to the lower tract airways (Fig. 68-1). Early inflammation of the bronchial and bronchiolar epithelium occurs along with peribronchiolar infiltration, mostly with mononuclear cells, and edema of the submucosa and adventitia. The respiratory epithelium becomes necrotic and is sloughed into the lumina of the airways. Subsequently, the epithelium proliferates and shows cuboidal cells without cilia (Fig. 68-2).

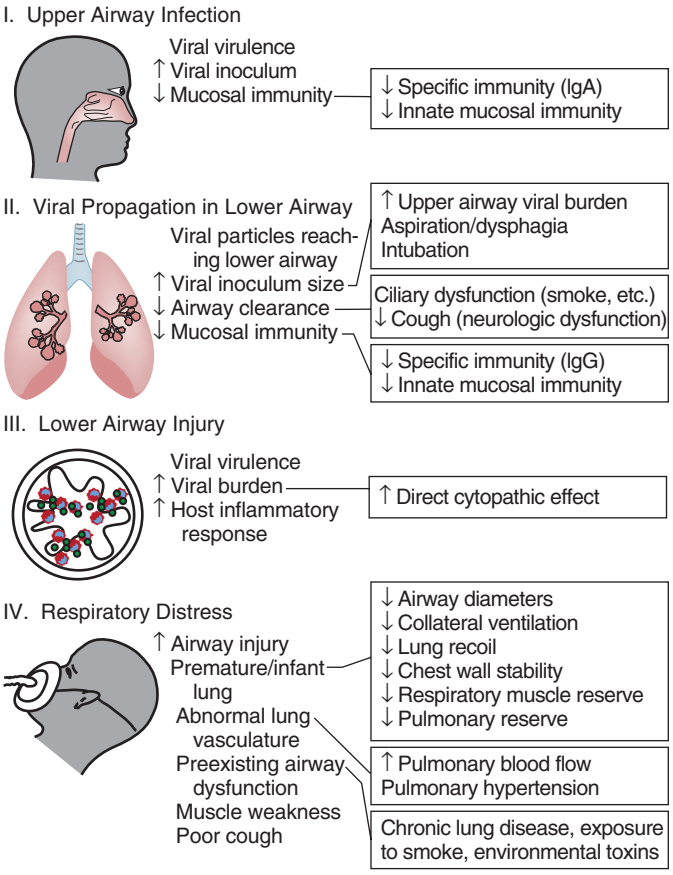

FIGURE 68-1 Factors that contribute to severity of viral bronchiolitis.

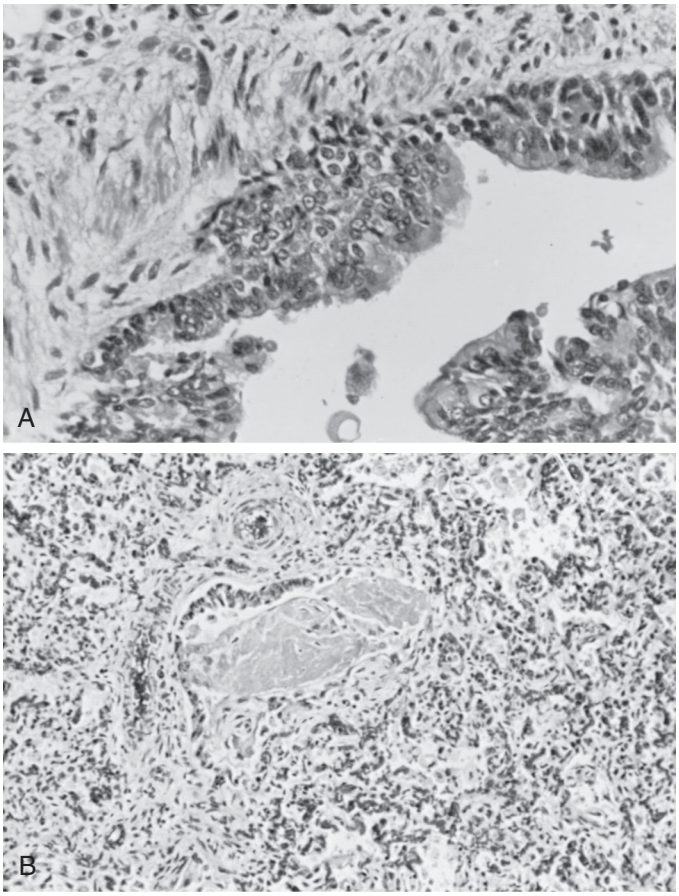

FIGURE 68-2 Bronchiolitis. A, Histopathology of bronchiolitis showing bronchiolar inflammation with regenerating epithelium. **B,** Characteristic inflammation and necroses in bronchiolitis, resulting in obliteration of the bronchiolar lumen.

Inflammatory changes of variable severity are observed in most small bronchi and bronchioles. Because resistance to airflow is related inversely to the cube of the radius of the airway, the inflammation and edema make the lumina of small airways in infants particularly vulnerable to obstruction (see Fig. 68-1). Plugs of necrotic material and fibrin may completely or partially obstruct the small airways. Smooth muscle constriction does not seem to be a major factor in the obstruction. In areas peripheral to sites of partial obstruction, air becomes trapped by a process similar to a "ball-valve" mechanism. Negative intrapleural pressure exerted during inspiration allows air to flow beyond the point of partial obstruction. On expiration, however, the size of the lumen decreases, resulting in obstruction and gas trapping. In areas peripheral to obstruction, trapped air is eventually absorbed, which results in multiple areas of atelectasis. This absorptive atelectasis is accelerated when a child breathes high concentrations of oxygen, which is absorbed into the blood much faster than nitrogen. The degree of atelectasis or hyperinflation that develops is greater in infants than it would be in older children or adults because collateral channels that maintain alveolar expansion in the presence of airway obstruction are not well developed early in life.

The physiologic correlates of airway obstruction are dyspnea, tachypnea, a diminished tidal volume, and a diminished ratio of ventilation to perfusion resulting first in arterial hypoxemia. When an infant is no longer able to compensate for the disordered gas exchange by increasing ventilation, hypercarbia may ensue. The pathologic process may progress to involve the alveolar walls and spaces, producing an interstitial pneumonitis. Recovery tends to be slow, requiring several weeks.

CLINICAL MANIFESTATIONS

Bronchiolitis commonly has a prodrome of several days that is marked by upper respiratory tract signs, especially coryza, cough, and fever, which is usually mild. Lower respiratory tract involvement may be signaled by the development of a prominent cough, followed by an increased respiratory rate, and nonspecific systemic symptoms such as lethargy and poor feeding. Retractions of the chest wall, flaring of the nasal alae, and grunting are evidence of increased work of breathing. The hallmark of bronchiolitis is the rapid variability of the child's respiratory signs. Auscultatory findings may vary from only wheezing or crackles, to both, or to neither. Decreasing lung sounds on auscultation associated with increasing dyspnea and diminished movement of air may indicate progressive obstruction and impending respiratory failure.

Dehydration commonly accompanies bronchiolitis, resulting from paroxysms of coughing, which may trigger vomiting, and from poor oral intake related to the child's respiratory distress and lethargy. Tachypnea increases the fluid requirement further. Of children hospitalized with bronchiolitis in the United Kingdom, 82% on admission had feeding difficulties that lasted an average of 27 hours.[36] Supplemental oxygen was administered at the time of admission to 22% of the children whose mean pulse oxygen saturation was 94%. Within 6 hours of admission, 70% were given supplemental oxygen, although the mean pulse oxygen saturation level decreased an average of 2%. No correlation was observed between the pulse oxygen saturation level obtained at 6 hours and the administration of supplemental oxygen or the length of hospital stay. Infants whose feeding difficulties resolved and who continued to be hospitalized for supplemental oxygen administration only had no evidence of clinical deterioration.

Considering that bronchiolitis is one of the most frequent causes of pediatric ambulatory visits and hospitalization, children at low risk for developing complicated illness have been evaluated to determine which children may be safely discharged home. Among children younger than 2 years of age presenting with bronchiolitis at 30 U.S. emergency departments during 2004 to 2006, 57% were discharged to home.[37] Characteristics of the children whose home discharge was safe included age 2 months or older, a history of eczema, respiratory rates

that were below normal for age, oxygen saturation levels 94% or greater, no or mild chest wall retractions, fewer treatments with bronchodilators during the first hour, and adequate oral intake.

The acute course of bronchiolitis typically lasts 3 to 7 days. A minority of children with RSV lower respiratory infection presents with critical hypoxemia, apnea, or respiratory failure and immediately requires intensive care. Most children admitted to the hospital are less severely affected, and relatively few of them deteriorate dramatically after admission. In one study, less than 2% of previously healthy children admitted to the regular floor for RSV infection subsequently required intensive care.[38] Most infants improve within 3 to 4 days, with a gradual recovery period of 1 to 2 weeks, but cough may persist longer. The median duration of illness in one study of ambulatory children with bronchiolitis was 12 days. After 3 weeks, 18% remained symptomatic, and after 4 weeks, 9% were still ill.[39] Gender, weight, or respiratory rate was not predictive of longer illness.

COMPLICATIONS

Complications associated with bronchiolitis occur most frequently in infants within the first several months of life, in premature infants, and in children with chronic cardiac, pulmonary, and immunodeficiency diseases.[3,27-29] The most serious complication is progression to respiratory failure. Although the risk of respiratory failure is relatively low for most children with RSV bronchiolitis, a small number of severely affected infants will require assisted ventilation in most intensive care units each year. Intubation and ventilation are usually indicated by recurrent severe apnea or hypercapnic/hypoxemic respiratory failure.

Apnea, one of the most frequent acute complications, occurs in 3% to 21% of infants.[40-42] Apnea typically is the presenting manifestation, occurring after several days of respiratory symptoms that may be so mild as to go unnoticed. Infants who present with apnea are at risk of developing severe lower respiratory disease even as the apnea typically resolves within a day or two. Apnea is most likely to occur in premature infants and in infants within the first 2 months of life (e.g., infants who are younger than 44 weeks' postconceptional age). The apnea does not seem to be obstructive, generally has a good prognosis, and is not associated with an increased risk of sudden infant death syndrome subsequently.

Aspiration has been shown to be a frequent complication in infants hospitalized with RSV bronchiolitis.[43,44] It is possible that infants with preexisting dysphagia are at increased risk of severe bronchiolitis with RSV infection, so this may represent association rather than causation. Secondary bacterial infections complicating bronchiolitis are uncommon, and concurrent bacterial infections occur in 0% to 7% of bronchiolitis cases.[28,45-48] Concurrent bacterial infections most frequently are urinary tract infections, unrelated to the bronchiolitis. Bacterial coinfections have been less common in children with bronchiolitis than in control children without bronchiolitis.

The most frequent clinical association observed in infants hospitalized with bronchiolitis is subsequent episodes of recurrent wheezing, estimated to occur in 30% to 50% of infants hospitalized with bronchiolitis. The pathogenesis of this link is unclear (see Chapter 160). Controversy continues over the extent to which this association is explained by a genetic predisposition to both severe RSV disease and subsequent wheezing or by an effect of RSV infection itself.[49] Nevertheless, the prognosis for most children with recurrent episodes of wheezing during early childhood is good. Among most children, the episodes diminish or disappear before reaching the teenage years.[50,51]

DIAGNOSIS

The diagnosis of bronchiolitis may be made for most children on the basis of the characteristic clinical and epidemiologic findings. These include the acute onset of the typical constellation of respiratory tract findings of cough, wheezing, and increasing respiratory effort after an upper respiratory tract prodrome, particularly during the winter respiratory season, in a child younger than 2 years of age.[2] Laboratory and radiologic studies are unnecessary for diagnosis; they do not change the outcome for most children and are not routinely recommended. The assessment of the severity of the bronchiolitis should also be based on the child's history and physical examination according to the American Academy of Pediatrics' guidelines on the diagnosis and management of bronchiolitis.[2] Complete blood cell count values vary in children with bronchiolitis and have not been shown to be helpful in determining the diagnosis or therapy of bronchiolitis.[2] Additional diagnostic procedures should be reserved for children whose history, findings, or clinical course are not as expected.

Although rapid diagnostic testing is generally unnecessary, it may be useful at times for implementing appropriate infection control, monitoring seasonal patterns of respiratory pathogens, restricting antimicrobial use, or providing confirmation of the diagnosis in children with unusual clinical presentations or severe disease. Timely diagnosis of specific viral respiratory pathogens may occasionally be necessary to guide specific antiviral therapy in children with high-risk conditions or severe illness with influenza or RSV.

Rapid diagnostic approaches to identifying the common viral agents of bronchiolitis include tissue culture, antigen detection, and PCR. Nasopharyngeal washes provide the most appropriate specimen. When available, tissue culture by shell vial technique can provide positive culture results within several days. Rapid antigen detection includes direct and indirect immunofluorescent assays, optical immunoassays, and enzyme immunoassays. These rapid viral antigen techniques are most commonly used because of their ease, cost, and availability of results within hours.[52] Rapid real-time PCR testing is becoming increasingly available for the simultaneous diagnosis of multiple respiratory viruses and is capable of high sensitivity and specificity, as well as short turnaround times.[53] The positive predictive value of all these viral assays significantly diminishes when the prevalence of the agent, such as RSV or influenza, is low in the community.

Serologic tests to determine the etiologic agent are rarely helpful in clinical management and may be difficult to interpret because a young infant would have maternally acquired antibody to many of the viral agents of bronchiolitis.

The differentiation of wheezing caused by RSV infection from wheezing caused by many other mechanisms in infants is challenging because RSV occurs in epidemics. During the height of the epidemic, it is tempting to assume that RSV is the culprit in any wheezing infant. The differential diagnosis of wheezing in an infant is broad and requires a careful history and examination.[54] Congestive heart failure is most important to consider because infants with left-to-right shunt are likely to become symptomatic and present with tachypnea and wheezing at around 8 to 10 weeks of age. Gastric reflux and aspiration may produce a picture that is indistinguishable clinically from acute bronchiolitis. An asthma exacerbation precipitated by a viral infection is possible, particularly in infants with a strong family history of asthma. Other considerations include foreign body aspiration, vascular ring, cystic fibrosis, and immunodeficiency.

THERAPY

Supportive care is the mainstay of therapy for outpatient and inpatient children. Guidelines for care have been published and updated.[2] At home, care is aimed primarily at comfort, maintaining adequate hydration, and treating fever if necessary.[2,55] Young children, especially infants, are particularly compromised by a respiratory rate of 60 or greater per minute and by the increased nasal congestion and mucus production in the lower respiratory tract. These may result in diminished fluid intake, inability to sleep, increased work of breathing, and the risk of requiring assisted ventilation. Clearance of secretions by administering chest percussion or deep pharyngeal and tracheal suctioning has been ineffective in the management of bronchiolitis and is not advised.[2]

Among more severely ill children with hypoxemia, supplemental oxygen administration may be of prime importance. The Spo_2 level at which supplemental oxygen should be administered is not well defined, however, and is controversial. Although Spo_2 levels of 90% to 95% on room air have been commonly used, the American Academy of Pediatrics has advised for previously healthy infants that supplemental oxygen should be initiated when persistent measurements of Spo_2 levels less than 90% are obtained.[2] In fact, the use of pulse oximetry in previously healthy children without signs of respiratory distress has not been associated with a better clinical outcome but has led to increased use of medical services and cost and is not routinely recommended.[2] Other factors than just the Spo_2 level should be considered in the

decision to administer supplemental oxygen. Additional risk factors to consider include underlying chronic conditions, poor feeding, clinical respiratory distress, fever, and acidosis, which may shift the oxyhemoglobin association curve such that appreciably lower levels of Pao_2 may occur at Spo_2 levels greater than 90%.

Therapeutic agents most frequently used for RSV bronchiolitis include bronchodilators, corticosteroids, and antibiotics. Of infants hospitalized with RSV infection in North America, Europe, and Australia, 75% to 80% were treated with bronchodilating agents, 10% to 40% were treated with corticosteroids, and 15% to 40% were treated with intravenous antibiotics.[56] Multiple studies have shown these therapies as inconsistently effective, and none is routinely recommended.[2]

A Cochrane Review of the use of bronchodilators for bronchiolitis concluded that the limited transient improvement using various clinical scoring systems observed was associated with questionable clinical benefit.[57] A subsequent review of the evidence by the American Academy of Pediatrics reached a similar conclusion and recommendation against the routine use of bronchodilators for infants with initial episodes of wheezing.[2] The addition of anticholinergic medications to the therapeutic regimen has not been shown to improve the course of viral bronchiolitis. These recommendations may not apply to children who have had recurrent wheezing before the episode of viral bronchiolitis.

Multiple trials have examined the use of nebulized, oral, and parenteral corticosteroid medications among children with bronchiolitis. Most of these trials have not included specific viral identification and are heterogeneous in design and in the populations included. Reviews that analyzed the randomized and controlled trials concluded that the evidence was insufficient to recommend routine use of these medications for bronchiolitis.[58-60]

A subsequent large, placebo-controlled trial of oral dexamethasone therapy was conducted in 20 emergency departments over three RSV seasons among 608 children 2 to 12 months old with their first episode of wheezing. Administration of a single oral dose of 1 mg/kg of dexamethasone had no effect on the subsequent rate of hospitalization or the clinical assessment score, even among children with a family history of asthma.[61] Review of this and the previous studies resulted in the current recommendation that corticosteroid medications should not be used routinely in the management of bronchiolitis.[2]

Nebulized hypertonic saline (most commonly 3 mL of 3% saline combined with a bronchodilator delivered by jet nebulization three times a day) has been shown to result in more rapid clinical improvement and shortened length of stay in infants with RSV bronchiolitis without evidence of side effects.[62] Nevertheless, this therapy has not been universally adopted because several studies have been unable to show benefit and because of the theoretical but unsubstantiated concern that the hypertonic saline aerosol might induce bronchospasm in infants with asthma.

Ribavirin (1-β-D-ribofuranosyl-1,2,4-triazole-3-carboxamide), a synthetic nucleoside, is available for aerosol treatment for RSV bronchiolitis among hospitalized infants. The drug is not recommended routinely, however, and should be considered only for infants with severe disease at high risk of severe illness (see Chapters 44 and 160).[2]

Several approaches are used to provide direct respiratory support for the small number of young infants or those with underlying abnormalities who develop life-threatening apnea or respiratory failure during an episode of bronchiolitis. Noninvasive approaches that have been used to avoid intubation include continuous positive airway pressure, heliox, and high-flow nasal cannula therapy (HFNC).[63-65] The first two have not been consistently shown to be adequately beneficial to justify the challenge of administration. HFNC, however, may be effective in preventing or delaying the need for intubation.[66,67]

PREVENTION

Prevention of the clinical entity of bronchiolitis is a goal unlikely to be reached in the near future because of its multiple etiologies and varying pathogenesis. For prevention of bronchiolitis associated with primary RSV infection, prophylactic administration of humanized monoclonal antibodies directed against the RSV F protein has been effective in reducing the rate of RSV hospitalization among high-risk infants who are premature and have comorbid conditions affecting cardiopulmonary function (see Chapter 160).[29,68]

The mainstay of preventing bronchiolitis remains the interruption of the spread of the infectious agent to infants and to the young age group of children who develop bronchiolitis. Preventing contact of the child with individuals who have signs of illness may be helpful, but many individuals may have infection that is asymptomatic or mild enough that it is unrecognized. Multiple infection-control procedures are recommended for RSV and other agents of bronchiolitis, but among these the most effective, whether in the hospital or home, are good hand hygiene and education of personnel and families (see Chapter 160).

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The complete reference list is available online at Expert Consult.

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