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Respiratory Syncytial Virus Infection in Children: Multidisciplinary Respiratory Care, Radiologic Assessment, Public Health Surveillance, Laboratory Diagnostics, and Healthcare Professionals.

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Abstract

Background: Respiratory syncytial virus (RSV) is a leading cause of acute lower respiratory tract infections in infants and young children, contributing to significant global morbidity and mortality. Despite advances in prevention, RSV remains a major pediatric health challenge.
Aim: To review the multidisciplinary aspects of RSV infection in children, including pathophysiology, clinical evaluation, management strategies, and emerging preventive measures.
Methods: A comprehensive synthesis of current evidence and guidelines was conducted, focusing on epidemiology, diagnostic approaches, supportive care, and immunoprophylaxis innovations.
Results: RSV infects nearly all children by age two, with severe disease concentrated in infants under one year and those with comorbidities. Clinical management remains primarily supportive, emphasizing hydration, oxygen therapy, and escalation to high-flow or mechanical ventilation when needed. Routine use of bronchodilators, corticosteroids, and antibiotics is not recommended. Preventive strategies have evolved with maternal vaccination and long-acting monoclonal antibodies (nirsevimab, clesrovimab), offering season-long protection and reducing hospitalization rates.
Conclusion: RSV continues to impose a substantial global burden, particularly in resource-limited settings. Integration of maternal immunization and infant prophylaxis into public health programs, combined with vigilant clinical care and infection control, is essential to reduce morbidity and mortality.
Keywords: Respiratory syncytial virus, bronchiolitis, pediatric infection, maternal vaccination, monoclonal antibodies, supportive care.

Introduction

Respiratory syncytial virus (RSV) is one of the most significant viral pathogens affecting pediatric populations worldwide, representing a leading cause of acute lower respiratory tract infections in infants and young children. RSV infection contributes to substantial global morbidity and mortality, accounting for the majority of hospital admissions related to viral respiratory disease in children under five years of age. In particular, RSV is responsible for an estimated 58,000 hospitalizations and hundreds of deaths annually in the United States among children younger than five years, with the highest disease burden occurring in infants below one year of age.[1][2] Globally, the World Health Organization estimates that RSV is responsible for millions of hospitalizations and tens of thousands of deaths annually, especially in low- and middle-income countries where access to advanced respiratory care is limited. RSV primarily infects the epithelial cells of the respiratory tract, spreading via respiratory droplets and direct contact with

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contaminated surfaces. The virus initially colonizes the upper respiratory tract, presenting as a mild cold-like illness, but in vulnerable populations—particularly neonates, premature infants, and those with underlying cardiopulmonary disease—it can progress to the lower respiratory tract. This progression leads to bronchiolitis, a clinical syndrome characterized by inflammation, edema, mucus plugging, and necrosis of small airway epithelial cells. The resultant small airway obstruction can lead to air trapping, hypoxemia, and, in severe cases, respiratory failure. Infants may also present with apnea, dehydration, or cyanosis, and those with severe infection often require hospitalization for oxygen therapy, mechanical ventilation, or other forms of intensive care [1][2].

RSV exhibits distinct seasonal patterns, with peak incidence typically occurring during the late fall through early spring in temperate climates, though seasonality may vary geographically. Virtually all children are infected with RSV by two years of age, and reinfections are common throughout life. While most infections are mild, the public health burden is considerable due to the large number of cases requiring medical attention and the potential for severe disease in high-risk populations, including preterm infants, those with congenital heart disease, chronic lung disease, and immunodeficiency disorders. Until recently, RSV prevention relied heavily on passive immunization through monoclonal antibodies such as palivizumab, which offered partial protection but required monthly dosing and was reserved for select high-risk infants due to cost and logistical constraints. However, the landscape of RSV prevention has changed dramatically with the development of maternal vaccines and long-acting monoclonal antibodies. The approval of the bivalent prefusion F protein-based maternal vaccine allows transplacental antibody transfer, providing passive immunity to infants during their most vulnerable early months of life. Similarly, the introduction of nirsevimab and clesrovimab, long-acting monoclonal antibodies with extended half-lives, provides season-long protection with a single dose, offering a feasible strategy for widespread prophylaxis.[3][4]

These advances represent a major paradigm shift in global RSV prevention, especially for healthcare systems aiming to reduce hospitalization rates and severe disease in early infancy. Integration of maternal immunization and infant prophylaxis programs is now a key component of public health policy, with the potential to significantly lower the global burden of RSV-related morbidity and mortality. Despite these preventive breakthroughs, the mainstay of RSV management remains supportive care, as there are currently no antiviral therapies with proven safety and efficacy for routine use. Supportive measures include maintenance of adequate hydration, airway clearance, oxygen supplementation, and, in severe cases, mechanical ventilation. Corticosteroids and bronchodilators have not demonstrated consistent benefit in clinical trials and are not routinely recommended. The focus of clinical management therefore lies in early recognition of respiratory distress, timely escalation of care, and implementation of infection control measures to prevent nosocomial transmission. RSV remains a major global pediatric health challenge due to its ubiquity, potential severity, and lack of specific antiviral treatment. However, the emergence of maternal vaccination and next-generation monoclonal antibody prophylaxis marks a transformative era in the fight against RSV. Early diagnosis, preventive immunization, and high-quality supportive care remain the cornerstones of management, requiring ongoing collaboration between pediatricians, public health professionals, and researchers to optimize outcomes and minimize the global burden of RSV disease [1][2][3][4].

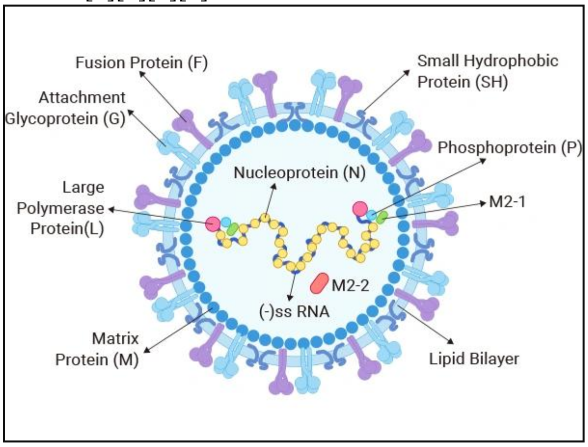

Fig. 1: Respiratory syncytial virus (RSV) structure.

Etiology

Respiratory syncytial virus (RSV) is an enveloped, nonsegmented, negative-sense, single-stranded RNA virus classified within the genus *Orthopneumovirus* of the family *Pneumoviridae*. This family also includes other clinically important respiratory viruses, notably human metapneumovirus (hMPV), which can produce a similar spectrum of upper and lower respiratory tract disease in children. RSV was first identified in 1955 in chimpanzees and was subsequently recognized as a human pathogen of major pediatric significance. Since that time, it has been established as a ubiquitous agent of seasonal respiratory illness, with particular pathogenic importance in infants and young children due to their narrower airways, developing immune responses, and increased vulnerability to inflammation-driven airflow limitation. The etiologic basis of RSV disease is closely linked to its structural and functional viral proteins, which coordinate attachment, entry, replication, and cell-to-cell spread within the respiratory epithelium. RSV virions contain multiple structural proteins that support infection, including nucleocapsid-associated proteins required for genomic packaging and replication, as well as surface glycoproteins that mediate host cell interactions. Among these, the two principal surface glycoproteins—G (attachment) and F (fusion)—are central to viral pathogenicity, tissue tropism, and host immune recognition. The G glycoprotein primarily facilitates viral attachment to respiratory epithelial cells and is notable for its genetic variability. This antigenic diversity contributes to the classification of RSV into two major antigenic subgroups, RSV-A and RSV-B, which co-circulate and may alternate in predominance across seasons and geographic regions. The variability of the G protein is also clinically relevant because it can influence immune escape and reinfection patterns, helping explain why RSV infections can recur throughout life despite prior exposure [3][4].

In contrast to the variable G protein, the F protein is more conserved and is essential for viral fusion and propagation within the host. The F protein mediates fusion of the viral envelope with the host cell membrane, allowing entry of the viral genome into the cytoplasm. It also promotes fusion between infected cells and adjacent uninfected cells, producing multinucleated syncytia—an eponymous hallmark of RSV infection that enhances direct cell-to-cell spread and may facilitate viral dissemination even in the presence of neutralizing antibodies. From a pathobiological perspective, this mechanism contributes to epithelial injury, inflammatory amplification, and mucus production in the small airways, thereby linking molecular viral behavior to the clinical syndrome of bronchiolitis and hypoxemic respiratory disease in infants. Because of their indispensable roles in infection, both the G and F glycoproteins represent high-value targets for preventive and therapeutic innovation. The F protein, in particular, has become a focal point for modern vaccine development because its prefusion conformation exposes potent neutralizing epitopes. Likewise, monoclonal antibodies and candidate antivirals often seek to block attachment or fusion processes mediated by these glycoproteins, thereby interrupting the earliest steps of viral pathogenesis. Consequently, RSV etiology cannot be understood solely as viral classification; it is more accurately defined by a tight mechanistic relationship between viral surface proteins, host epithelial entry, immune evasion, and the distinctive propensity for lower airway disease in young children.[5]

Epidemiology

Respiratory syncytial virus (RSV) is a ubiquitous pathogen with near-universal exposure early in life and a well-recognized capacity to cause recurrent infection across the lifespan. Epidemiologic studies indicate that approximately 90% of children acquire RSV infection within the first two years of life, and reinfections are common throughout adulthood because naturally acquired immunity is incomplete and does not confer durable, sterilizing protection.[6] This pattern of repeated infection reflects both the virus's ability to circulate efficiently within communities and the limitations of long-term mucosal and systemic immune memory following primary infection, particularly against antigenically diverse strains. At the global level, RSV constitutes one of the most important causes of acute lower respiratory tract illness in young children. In 2015, worldwide estimates attributed 33.1 million episodes of RSV-associated acute lower respiratory infection to children, with approximately 3.2 million hospital admissions linked to these infections and 59,600 deaths occurring in those younger than five years of age. More recent summaries describe a continuing and substantial annual burden, with roughly 3.6 million hospitalizations and close to 100,000 deaths each year in children under five, underscoring that RSV remains a leading contributor to preventable pediatric morbidity and mortality.[1] Importantly, the majority of fatal outcomes occur in low- and middle-income countries, where barriers to timely care, limited access to oxygen therapy and intensive respiratory support, and higher prevalence of comorbid malnutrition or chronic disease amplify clinical risk and worsen outcomes.[1]

RSV transmission displays a characteristic seasonal rhythm that varies by climate and geography. In temperate regions, epidemics typically peak during winter and early spring, and in the Northern Hemisphere the highest activity often spans October through March.[7] These seasonal surges are clinically relevant because they drive predictable increases in pediatric emergency visits, hospital occupancy, and demand for oxygen and respiratory support services. The COVID-19 pandemic introduced a notable, temporary disruption to usual RSV circulation patterns, likely driven by nonpharmaceutical interventions and altered population mixing; however, accumulating observations indicate that more typical seasonality has largely reemerged in many regions as public health restrictions eased and respiratory virus ecology normalized.[7][8] Disease severity is not evenly distributed across populations. Elevated morbidity and mortality are consistently observed among premature infants—particularly those born before 29 weeks' gestation—as well as children with chronic cardiac disease, chronic pulmonary conditions, and immunocompromising disorders, in whom limited physiologic reserve and impaired immune responses increase the likelihood of lower airway involvement and respiratory failure.[9][10] Additionally, recent evidence emphasizes that RSV is not exclusively a pediatric threat: adults older than 60 years' experience a substantial disease burden, with mortality estimates approaching those reported in young children, reinforcing the need for age-inclusive surveillance and prevention strategies across health systems.[9][10]

Pathophysiology

Respiratory syncytial virus (RSV) is transmitted primarily via respiratory droplets and direct contact with contaminated secretions, with inoculation commonly occurring through the nasopharyngeal mucosa or, less frequently, the conjunctiva. Following exposure, RSV undergoes an incubation period that typically ranges from 2 to 8 days, after which symptomatic infection may manifest and viral shedding increases, facilitating further spread within households and community settings. After entry, the virus exhibits tropism for the respiratory epithelium, with a particular predilection for ciliated epithelial cells lining the upper and lower airways. Attachment and fusion mechanisms enable infection of epithelial cells, and subsequent viral replication initiates a cascade of cytopathic injury and host inflammatory responses that together drive the clinical syndrome. The core pathophysiologic event underlying RSV bronchiolitis is small-airway obstruction produced by the combined effects of direct epithelial damage and immune-mediated inflammation. Viral replication within airway epithelial cells leads to epithelial necrosis and sloughing, disrupting mucociliary clearance and exposing submucosal tissues to inflammatory mediators. In parallel, activation of innate and adaptive immune pathways generates inflammatory edema and increased mucus production, while exfoliated epithelial cells and leukocytes contribute to intraluminal debris.[11][12] In infants, these processes are particularly consequential because bronchiolar caliber is small and the airway wall is relatively compliant; therefore, modest degrees of swelling and mucus accumulation can substantially increase airway resistance. The resulting obstruction is often patchy and dynamic, producing heterogeneous ventilation across lung regions. This physiologic derangement manifests clinically as wheezing and increased work of breathing, while mechanically it promotes air trapping due to impaired expiratory flow, especially when bronchioles collapse during expiration.

As obstruction progresses, downstream consequences include ventilation–perfusion mismatch, atelectasis in poorly ventilated segments, and areas of hyperinflation where air trapping predominates. These changes contribute to hypoxemia, which is a defining feature of more severe bronchiolitis and a key reason for hospitalization. In some infants, the combination of increased respiratory effort, fatigue, and impaired gas exchange can culminate in respiratory failure, while younger infants may present with apnea as a prominent early manifestation. The extent of mucus plugging and edema, alongside individual variation in immune response and baseline airway anatomy, influences clinical severity and the need for supportive interventions such as oxygen therapy or ventilatory support. Beyond acute illness, RSV infection has been linked to longer-term respiratory morbidity. Immunologically, RSV responses in some children demonstrate a type 2 (T helper 2)-skewed inflammatory profile with relatively diminished interferon activity, a pattern that may reduce effective antiviral clearance and amplify airway inflammation.[13] This immunologic milieu has been implicated in post-bronchiolitis airway hyperreactivity and may contribute to the later development of recurrent wheezing phenotypes and asthma in susceptible individuals, although the causal pathways likely involve both viral effects and underlying host predisposition.[13]

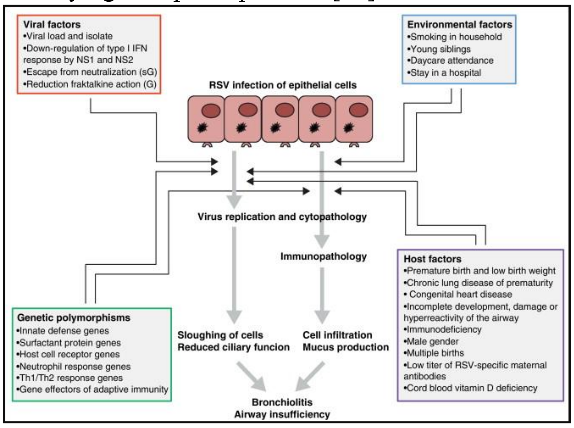

Fig. 2: Pathogenesis of RSV.

Histopathology

Histopathologic examination plays a limited role in the routine diagnosis of respiratory syncytial virus (RSV) infection, largely because clinical presentation and virologic testing provide more timely and practical confirmation in most healthcare settings. Tissue sampling from the lower respiratory tract is not typically indicated in uncomplicated bronchiolitis, and invasive procedures would rarely be justified given the self-limited course in many patients. Nonetheless, histopathology has been valuable in elucidating RSV disease mechanisms, clarifying why infants and high-risk patients develop small-airway obstruction and impaired gas exchange, and describing the cellular injury patterns that correspond to severe clinical phenotypes. A distinctive pathologic feature of RSV is its capacity to induce cell-to-cell fusion among infected respiratory epithelial cells. This effect is mediated primarily by the viral fusion (F) protein, which facilitates the merging of adjacent cell membranes and results in the formation of multinucleated syncytial giant cells. The presence of these syncytia—while not universally demonstrable in all clinical specimens—represents a characteristic hallmark that historically contributed to RSV naming and underscores an important mechanism of local viral spread. Infected epithelium is often damaged both directly by viral replication and indirectly through host inflammatory responses, leading to cytopathic changes and disruption of normal mucociliary function. Typical histopathologic findings in RSV bronchiolitis reflect a combination of epithelial injury, luminal obstruction, and peribronchiolar inflammation. Microscopic examination commonly reveals sloughed or exfoliated epithelial cells within bronchiolar lumens, accompanied by abundant mucus that contributes to plugging. This intraluminal mixture of mucus, cellular debris, and inflammatory exudate narrows or occludes small airways and provides the structural basis for clinical wheezing, air trapping, and patchy atelectasis. Necrosis of the bronchiolar epithelium is frequently observed, reflecting extensive epithelial cell death and impaired barrier integrity. Surrounding these airways, peribronchiolar lymphocytic inflammation is often present, indicating activation of adaptive immune pathways and ongoing host response to viral antigens. In severe RSV disease, the histopathologic picture is typically more pronounced and diffuse. Extensive epithelial denudation and heightened airway wall edema are common, further narrowing already small-caliber bronchioles in infants. Immune cell infiltration becomes more conspicuous, with temporal variation in the dominant cell populations. Early in the illness, polymorphonuclear leukocytes frequently predominate, consistent with an acute innate inflammatory response, while later phases more often demonstrate lymphomononuclear cell infiltration, reflecting evolving adaptive immune activity. These shifts help explain why clinical severity can change over time, with some patients worsening after initial symptoms as inflammation and airway obstruction intensify. Although histopathology is seldom necessary for diagnosis, it remains an important explanatory framework for understanding the structural and inflammatory mechanisms that underpin RSV bronchiolitis and its more severe manifestations [9][10][11][12].

History and Physical

The clinical evaluation of respiratory syncytial virus (RSV) infection in children hinges on careful history-taking and a structured physical examination that distinguishes uncomplicated upper respiratory involvement from evolving lower respiratory tract disease and identifies early markers of severe illness. RSV occupies a unique place among pediatric respiratory pathogens because its initial presentation may be indistinguishable from benign viral “colds,” yet a subset of infants—especially those with physiologic vulnerability—can deteriorate rapidly as infection extends into the small airways. For this reason, the clinician’s goal is not simply to label the illness as RSV-like but to determine the current anatomic level of involvement, quantify respiratory compromise, and anticipate trajectories that warrant close observation, supportive escalation, or hospitalization. When RSV remains confined to the upper respiratory tract, children typically present with nonspecific symptoms that overlap substantially with other viral infections. Caregivers commonly report rhinorrhea, nasal congestion, sneezing, and cough, sometimes accompanied by low-grade fever and mild irritability. These symptoms reflect mucosal inflammation and increased secretions in the nasopharynx and upper airways, leading to obstructive nasal breathing that can be particularly problematic in young infants who are obligate nasal breathers. Parents may describe difficulty feeding, shorter feeding sessions, or disrupted sleep due to congestion. Importantly, because upper airway RSV disease can appear clinically routine, the history should specifically address duration of symptoms, change over time, and early warning signs of progression, including increasing cough frequency, reduced oral intake, signs of dehydration, or increased breathing effort. Mild upper respiratory illness may precede lower respiratory involvement, and this is especially relevant in infants, in whom airway caliber is small and inflammatory edema can quickly translate into clinically meaningful obstruction.[14] Therefore, even apparently minor symptoms warrant contextual interpretation, particularly when the patient is very young or has known risk factors.

Physical examination in upper respiratory disease may reveal nasal discharge, erythematous nasal mucosa, mild pharyngeal erythema, and transmitted upper airway sounds on auscultation. The clinician should assess hydration status through mucous membrane moisture, tear production, capillary refill, and diaper counts, since feeding difficulties can develop early. Baseline vital signs are essential, including temperature, respiratory rate, and oxygen saturation. While oxygenation is often preserved in isolated upper airway disease, documenting a normal baseline is important because it provides a reference point if symptoms evolve. In addition, the clinician should evaluate for alternative or concurrent diagnoses, such as otitis media, which can accompany viral upper respiratory infections and may influence management. Progression to lower respiratory involvement—classically bronchiolitis—occurs most frequently in infants younger than two years of age and is most clinically significant in those with prematurity, chronic lung disease, congenital heart disease, or other conditions that reduce respiratory reserve. Bronchiolitis represents inflammation and obstruction at the level of the small airways, and it often presents after a brief prodrome of upper respiratory symptoms. The history in suspected bronchiolitis should focus on the onset and progression of breathing difficulty, changes in feeding and activity level, and any episodes suggestive of apnea or cyanosis. Caregivers may describe fast breathing, audible wheezing, persistent cough, or increased fussiness. They may also report that the child tires during feeding or cannot complete usual volumes, reflecting the high metabolic cost of breathing and impaired coordination between suck, swallow, and breathing. Because infants can deteriorate rapidly, questions about urine output, lethargy, and responsiveness are essential markers of systemic impact [13][14].

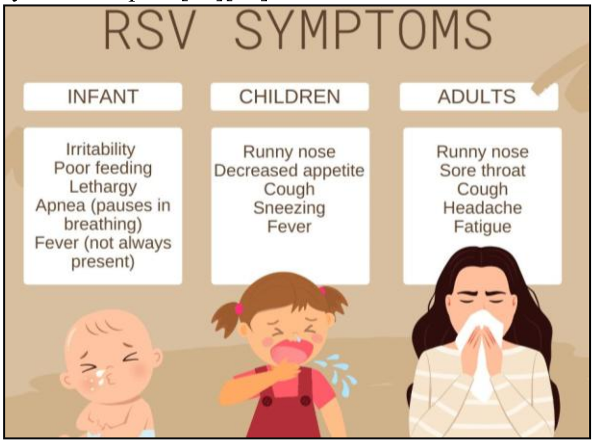

Fig. 3: RSV symptoms.

On examination, bronchiolitis is characterized by tachypnea and increased breathing. Clinicians often observe subcostal, intercostal, or suprasternal retractions, nasal flaring, and the use of accessory muscles, all reflecting increased respiratory effort needed to overcome airway resistance. Wheezing and prolonged expiration are common, though auscultation may also reveal diffuse crackles or diminished air entry when obstruction is severe. Importantly, wheezing may not be prominent in very young infants or in those with significant mucus plugging, and the absence of wheeze does not exclude bronchiolitis. The clinician should assess the child's ability to maintain adequate ventilation by noting respiratory rate relative to age norms, the depth and regularity of breaths, and signs of fatigue such as head bobbing, grunting, or decreasing retractions as the child tires. Oxygen saturation measurement is critical, as hypoxemia is a key indicator of clinically significant lower respiratory involvement and can guide decisions about escalation of care.[15] In addition, auscultation findings should be interpreted in the context of overall work of breathing; a child with quiet breath sounds and poor air movement may be more critically ill than one with loud wheezing and preserved ventilation. The evaluation should also include a general assessment of perfusion and neurologic status. Tachycardia may accompany fever, dehydration, or respiratory distress; conversely, bradycardia in an ill infant may indicate impending decompensation. Mental status, tone, and interaction with caregivers provide important clues: an alert infant with robust crying and good tone may tolerate moderate bronchiolitis, whereas lethargy or diminished responsiveness suggests systemic compromise. Feeding assessment is equally central, because poor intake may be both a consequence and a contributor to severity through dehydration and reduced energy reserves [15].

Severe RSV infection represents the point at which respiratory compromise threatens oxygenation or ventilation, or where systemic effects such as apnea and poor feeding pose immediate safety risks. Apnea is a particularly concerning manifestation and is more common in very young infants, premature neonates, and those with immature respiratory control.[9] Caregivers may report pauses in breathing, color change, limpness, or episodes requiring stimulation. In some cases, apnea may be the presenting complaint even before prominent lower respiratory signs develop, underscoring the importance of age-based risk assessment. Severe disease is also suggested by marked respiratory distress, persistent hypoxemia, cyanosis, or signs of impending respiratory failure, including progressive fatigue, decreased respiratory effort despite ongoing tachypnea, altered mental status, and inability to feed. Lethargy and poor feeding are not merely nonspecific symptoms; in bronchiolitis they often signal that the work of breathing has become unsustainable and that the child's physiologic reserves are exhausted. On physical examination, children with severe disease may display profound tachypnea, significant retractions, nasal flaring, grunting, and hypoxemia, sometimes with diminished breath sounds if airflow is critically reduced. Signs of dehydration may be evident, including dry mucous membranes, delayed capillary refill, and reduced urine output, often compounded by feeding refusal. In advanced cases, respiratory failure may develop, requiring prompt escalation to supplemental oxygen, high-flow respiratory support, noninvasive ventilation, or mechanical ventilation depending on institutional resources and the child's response to initial measures.[9] In this context, the clinician's assessment must be continuous rather than episodic, since children can transition from moderate distress to decompensation over a short time interval. Overall, history and physical examination in pediatric RSV infection should be framed around trajectory and risk. Upper respiratory features are common and nonspecific,[14] but the clinician must maintain vigilance for progression to bronchiolitis, particularly in infants under two years and in those with high-risk conditions.[15] Recognition of severe manifestations—especially apnea, lethargy, and feeding failure—should prompt urgent intervention and appropriate disposition planning, because timely supportive escalation is the primary determinant of safe outcomes in RSV disease.[9][14][15]

Evaluation

Evaluation of RSV bronchiolitis in children is centered on clinical diagnosis, with investigations used selectively rather than routinely. This approach reflects two realities of pediatric bronchiolitis: first, the syndrome has a recognizable clinical pattern in most cases; and second, management is largely supportive, so confirmatory testing rarely changes immediate treatment decisions. Consequently, the primary diagnostic task is not simply identifying RSV as the causative virus but determining the severity of lower respiratory involvement, identifying risk factors for deterioration, and excluding alternative diagnoses when the presentation is atypical. In practice, clinicians emphasize careful assessment of respiratory rate, work of breathing, hydration status, and oxygenation, while incorporating age and comorbidity profiles to guide disposition. Clinical assessment should begin with structured triage and vital sign measurement, including temperature, heart rate, respiratory rate, and pulse oximetry. Tachypnea and increased work of breathing are hallmark features of bronchiolitis and should be interpreted relative to age-based norms. Physical examination focuses on signs of respiratory effort—subcostal and intercostal retractions, nasal flaring, grunting, head bobbing—and on auscultatory findings such as wheezing, crackles, and prolonged expiration. Importantly, auscultation should be integrated with the overall clinical picture, because severe obstruction may produce diminished breath sounds and poor air entry even in the absence of prominent wheeze. Hypoxemia is a key marker of clinically significant disease and is particularly relevant in infants, preterm children, and those with chronic cardiopulmonary disorders, in whom physiologic reserve is limited and thresholds for escalation are lower. Assessment of feeding tolerance, urine output, and signs of dehydration is also essential, as bronchiolitis frequently compromises oral intake and can produce clinically relevant volume depletion, which may itself worsen respiratory fatigue. The clinician should also screen for apnea risk, especially in young infants and those born prematurely, because apnea may be an early or predominant manifestation in vulnerable patients [14][15][16].

Confirmatory virologic testing is generally reserved for circumstances in which results would affect clinical management or operational decisions. A common example is infection prevention and control, where identifying RSV can support cohorting of hospitalized patients, optimize isolation practices, and reduce nosocomial transmission. Testing may also be considered when diagnosis is uncertain, when the patient is immunocompromised, or when institutional protocols require pathogen identification for outbreak tracking or public health reporting. When testing is pursued, specimen collection is typically performed from the nasopharynx using swabs or aspirates, with the understanding that timing relative to symptom onset and sampling technique influence yield. Rapid antigen detection tests are widely used in pediatric settings because they are inexpensive, provide results in less than one hour, and can support real-time cohorting decisions. During outbreak periods and in infants—who often have higher viral loads—these assays may demonstrate approximately 80% to 90% sensitivity. However, sensitivity declines in older children and adults, in whom viral loads tend to be lower and shedding patterns differ, increasing the risk of false-negative results.[16] For this reason, a negative rapid antigen test does not necessarily exclude RSV in older age groups or in later phases of illness, and clinical judgment remains essential when interpreting results. Reverse transcription polymerase chain reaction (RT-PCR) testing offers substantially higher sensitivity and is increasingly used as a preferred diagnostic method when precise viral identification is required. RT-PCR can detect RSV even at lower viral loads and often provides multiplex panels that simultaneously identify other respiratory pathogens, which can be useful for differential diagnosis and infection control planning. This higher analytic sensitivity makes RT-PCR particularly valuable in older children and adult patients, as well as in settings where co-infections or alternative viral etiologies are plausible and might influence cohorting or broader clinical decision-making.[16] Nevertheless, even RT-PCR results should be interpreted within the clinical context, since detection of viral nucleic acid does not always distinguish between active infection and residual shedding, particularly when symptoms are mild or evolving [15][16].

Imaging is another area where evaluation must be judicious. Routine chest radiography is not recommended in uncomplicated bronchiolitis because it rarely changes management and may yield nonspecific findings that prompt unnecessary antibiotic use or additional investigations. When obtained, chest radiographs commonly show hyperinflation, patchy atelectasis, and peribronchial thickening—features that reflect small-airway obstruction and inflammation but do not reliably differentiate RSV from other viral causes or from noninfectious processes.[17] However, radiography may be appropriate in atypical or severe presentations, such as when focal findings raise concern for bacterial pneumonia, when there is unexpected clinical deterioration, when the child has significant underlying cardiopulmonary disease, or when alternative diagnoses (e.g., aspiration, congenital anomalies, foreign body) are being considered. In these situations, imaging is used not to "confirm bronchiolitis," but to evaluate complications, clarify diagnostic uncertainty, and guide escalation of care. Overall, evaluation of RSV bronchiolitis is best conceptualized as severity stratification rather than pathogen confirmation. Clinical examination remains the diagnostic cornerstone, while targeted use of rapid antigen testing or RT-PCR can support infection control and selected clinical decisions.[16] Imaging is reserved for presentations in which findings would meaningfully alter management, recognizing that routine radiography in typical bronchiolitis offers limited benefit and may inadvertently increase low-value interventions [16][17].

Treatment / Management

Management of respiratory syncytial virus (RSV) infection in children is fundamentally supportive, reflecting the fact that no broadly safe and consistently effective antiviral therapy is available for routine clinical use in otherwise healthy pediatric patients.[15][16] The central objectives are to maintain adequate oxygenation and ventilation, preserve hydration and energy balance, reduce respiratory effort, and prevent secondary complications while the child's immune system clears the infection. Because RSV bronchiolitis exists on a continuum—from mild upper respiratory symptoms to severe lower airway obstruction with respiratory failure—treatment must be individualized according to age, baseline risk factors, and dynamic markers of severity. Clinical teams should adopt a trajectory-focused approach: identify children likely to remain stable with home care, recognize early signs of progression requiring observation, and rapidly escalate respiratory support in those showing decompensation. Supportive care begins with careful physiologic assessment and ongoing monitoring. In mild cases, the priority is comfort and maintaining oral intake. Caregivers are typically advised to ensure adequate fluids, monitor feeding and urine output, control fever when present, and address nasal congestion to improve feeding and sleep. For infants, particularly those with obligate nasal breathing, congestion can markedly increase work of breathing and decrease feeding efficiency, so simple measures such as saline drops and gentle nasal suctioning before feeds may be highly beneficial. Supportive management also includes anticipatory guidance: caregivers should be instructed to watch for signs of worsening respiratory distress (increasing retractions, rapid breathing, grunting, cyanosis), apnea, reduced responsiveness, and decreased intake. Even when home management is appropriate, clinicians should emphasize that disease severity may peak several days into the course, making vigilance critical during the expected window of deterioration. A key element of evidence-based RSV care is avoiding interventions that do not improve meaningful outcomes. Multiple guidelines and clinical trials have shown that routine use of bronchodilators, corticosteroids, and antibiotics in otherwise healthy infants with bronchiolitis does not reliably reduce hospitalization rates, shorten length of stay, or improve clinically significant endpoints, and may introduce harm through side effects, unnecessary costs, and downstream over-treatment. Consequently, contemporary practice emphasizes "less is more" when the child's presentation is consistent with typical bronchiolitis and there is no evidence of alternative pathology. This is particularly important in busy acute care settings, where nonspecific wheeze may prompt reflex bronchodilator use; in bronchiolitis, wheeze arises primarily from small-airway inflammation and mucus plugging rather than bronchospasm, explaining the limited benefit observed in most patients [16][17][18].

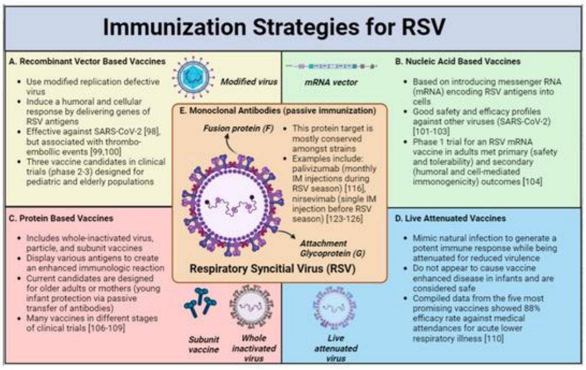

Fig. 4: Immunization of RSV.

Hydration

Hydration support is one of the most consequential components of pediatric RSV management because feeding difficulty is common and dehydration can develop quickly, especially in infants with tachypnea and increased breathing. A stepwise strategy is typically employed. When respiratory distress is mild and the child can feed safely, clinicians encourage continued oral intake, often in smaller, more frequent volumes to reduce fatigue. If oral feeding is compromised but the child is otherwise stable, nasogastric tube feeding can provide an effective intermediate option, maintain caloric intake and hydration while avoid some of the risks associated with intravenous fluids and repeated cannulation attempts in small infants. Intravenous fluids are reserved for children with moderate to severe dehydration, persistent vomiting, or significant respiratory distress that makes oral or nasogastric feeding unsafe. Adequate hydration supports mucociliary clearance, reduces secretion viscosity, and helps prevent metabolic complications that may arise when intake is markedly reduced. Importantly, fluid administration should be carefully titrated to the patient's clinical status; overly aggressive fluids can worsen respiratory status in some children by increasing pulmonary interstitial fluid, whereas inadequate fluids can exacerbate fatigue and compromise recovery [18]

Oxygen therapy and respiratory support

Oxygen therapy is indicated when hypoxemia is present, and it represents a primary driver of hospitalization decisions. Supplemental oxygen is typically titrated to maintain oxygen saturation in a clinically acceptable range, commonly around 90% to 92%, recognizing that brief, self-limited desaturations may occur during sleep or feeding in bronchiolitis. The choice of oxygen delivery method depends on severity. Low-flow nasal cannula oxygen may be sufficient for children with mild hypoxemia and preserved work of breathing.

When oxygen needs rise or when work of breathing becomes more pronounced, high-flow nasal cannula (HFNC) therapy is often used because it can deliver heated, humidified oxygen at higher flow rates, reduce inspiratory resistance, improve secretion clearance, and provide a degree of distending airway pressure that may decrease effort and improve ventilation. HFNC has become a common escalation step in many pediatric units due to its tolerability and ability to stabilize patients who would otherwise progress to more invasive support. If the child demonstrates worsening respiratory failure—manifested by persistent hypoxemia despite escalating oxygen delivery, rising carbon dioxide levels, exhaustion, decreasing respiratory drive, or recurrent apnea—noninvasive positive pressure ventilation such as continuous positive airway pressure (CPAP) may be indicated, particularly when airway collapse and severe small-airway obstruction are prominent contributors. Mechanical ventilation is reserved for children who cannot maintain adequate gas exchange or airway protection, or in whom apnea and fatigue pose immediate risk. The decision to intubate is clinical and must weigh the child's trajectory and reserve; delayed intubation after prolonged struggle can increase morbidity, whereas appropriately timed ventilation can stabilize oxygenation and reduce metabolic demand [18][19].

Airway clearance

Airway clearance in bronchiolitis should be effective but conservative. Gentle nasal suctioning with saline drops can improve airflow and feeding, particularly in young infants with significant nasal congestion. However, deep suctioning is generally discouraged because it can cause mucosal trauma, increase airway edema, and has been associated with longer hospitalization in some studies. The goal is therefore to clear the nares and improve comfort without provoking additional inflammation. Humidified oxygen and adequate hydration can also support secretion mobilization. Chest physiotherapy is not routinely recommended for typical bronchiolitis because it has not shown consistent benefit in improving key outcomes and may increase distress [19].

Hospitalization criteria

Determining whether a child requires inpatient care is a central decision point in RSV management. Hospitalization is generally indicated when the child demonstrates hypoxemia requiring supplemental oxygen, when oral intake is insufficient to maintain hydration, or when there are signs of moderate to severe respiratory distress that require close monitoring and potential escalation. Young age—often operationalized as less than three months—warrants a lower threshold for admission when significant symptoms are present because very young infants have a higher risk of apnea, rapid deterioration, and feeding failure. Likewise, children with high-risk comorbidities such as prematurity, congenital heart disease, chronic lung disease, or immunocompromise may require inpatient observation even with less dramatic symptoms because their physiologic reserve is limited and the consequences of deterioration are more severe. Admission decisions should also consider social determinants of health and caregiver capacity, as safe home monitoring depends on reliable access to follow-up and the ability to recognize worsening signs [17][18].

Nonroutine management approaches and therapies to avoid

Evidence-based guidelines advise against routine bronchodilator therapy (such as albuterol) in typical bronchiolitis because it does not consistently improve clinically meaningful outcomes such as hospitalization rates, length of stay, or overall disease course in otherwise healthy infants. While a carefully monitored trial may be considered in select older children with a strong history suggestive of reactive airway disease, routine administration in classic bronchiolitis is generally discouraged. Similarly, nebulized epinephrine is not routinely recommended because any observed clinical improvement is typically transient and has not translated into durable outcome benefits. Corticosteroids have likewise demonstrated lack of efficacy in typical bronchiolitis and are not recommended as routine therapy, reflecting the primarily viral and inflammatory—not steroid-responsive—mechanisms driving disease in most infants. Hypertonic saline has been studied as a strategy to improve mucociliary clearance and reduce airway edema, but guideline recommendations generally advise against routine use in otherwise healthy infants because consistent improvements in meaningful outcomes have not been demonstrated across settings, and effects—when present—may be modest and context-dependent. Antibiotics are also not routinely indicated because RSV is a viral illness and the risk of concurrent bacterial infection in otherwise healthy infants with RSV bronchiolitis is low, reported at less than 2%. Antibiotic use should therefore be reserved for situations where there is clear evidence of bacterial infection, such as focal pneumonia, otitis media requiring treatment, urinary tract infection, or sepsis concerns supported by clinical and laboratory findings. Restricting antibiotics to appropriate indications reduces adverse drug effects and supports antimicrobial stewardship. Finally, antiviral therapy remains limited. Aerosolized ribavirin, introduced and approved decades ago for severe RSV, is rarely used in contemporary practice due to limited evidence of clinical benefit in most populations, practical challenges of administration, and safety concerns related to aerosol exposure for healthcare personnel. Its use is generally reserved for select immunocompromised patients in whom RSV can cause prolonged, severe disease and where specialized teams judge that potential benefits outweigh risks.[18][19] As a result, for the vast majority of pediatric cases, effective management remains anchored in careful supportive care, timely escalation of respiratory support, and avoidance of nonbeneficial interventions.[15][16]

Prevention Strategies

Prevention of respiratory syncytial virus (RSV) infection in children is now best understood as a layered framework that integrates hospital-based infection control with population-level immunization strategies designed to protect infants during their highest-risk months. In healthcare facilities, nosocomial RSV prevention depends on structured infection-control programs that emphasize early recognition of symptomatic patients, prompt implementation of transmission-based precautions, and cohorting or isolation to reduce hospital-acquired spread. These measures are particularly important in neonatal and pediatric units because RSV can cause severe disease in premature infants and children with cardiopulmonary comorbidities, and because hospital transmission can amplify outbreaks in settings where vulnerable patients are concentrated. Early identification of suspected cases and rapid isolation remain central operational tools for protecting high-risk patients, minimizing staff exposure, and reducing downstream morbidity associated with in-hospital transmission.[20] Beyond the hospital environment, RSV prevention has entered a transformative era through maternal immunization and long-acting monoclonal antibody prophylaxis for infants. Maternal vaccination with the bivalent prefusion F (preF) vaccine—commercially available in the United States as Pfizer's Abrysvo—has been incorporated into seasonal guidance to provide passive protection for newborns through transplacental antibody transfer. Current CDC clinical guidance recommends a single maternal RSV vaccine dose during weeks 32 through 36 of gestation, administered seasonally (typically September through January in most of the United States) to align infant protection with peak RSV circulation. This strategy is intended to reduce severe RSV lower respiratory tract disease during early infancy, when bronchiolitis-related hospitalization risk is highest. Real-world and post-implementation monitoring have supported meaningful reductions in infant RSV-associated hospitalization without identifying an unexpected increase in preterm birth beyond background rates, consistent with the emphasis on administering the vaccine within the 32–36 week window.[3][21] In practical prevention planning, maternal vaccination also offers a systems-level advantage: it provides protection beginning at birth, which is especially relevant for infants who may not access outpatient prophylaxis promptly after delivery.

Long-acting monoclonal antibodies have further expanded RSV prevention capacity by offering direct passive immunization to infants. Nirsevimab (Beyfortus) is recommended for infants born during or entering their first RSV season and for certain high-risk children entering their second RSV season (typically 8–19 months of age at increased risk of severe disease), based on ACIP recommendations and supporting evidence reviews. These products are designed to provide approximately a full RSV season of protection (about five months) following a single dose, which improves feasibility compared with earlier monthly prophylaxis models. Clesrovimab (Enflonsia), a newer long-acting monoclonal antibody, received FDA approval in 2025 for prevention of RSV lower respiratory tract disease in neonates and infants during their first RSV season, with expected protection lasting roughly five months. Importantly, current CDC guidance indicates that clesrovimab is not recommended for children over 8 months of age and does not have FDA approval for children entering their second RSV season, which matters when designing protocols for older high-risk toddlers.[4] These long-acting products have also reshaped the role of palivizumab (Synagis), the old monthly monoclonal antibody historically used for select high-risk infants. Rather than being "replaced" purely by preference, palivizumab availability is changing structurally: CDC clinical guidance notes that palivizumab will no longer be available starting December 31, 2025, and the American Academy of Pediatrics has reported discontinuation timelines aligned with this date. This shift reflects both the broader eligibility and operational convenience of long-acting agents and the evolving preventive ecosystem in which most infants can be protected either via maternal vaccination or a single-dose monoclonal antibody strategy. Despite these major advances, implementation gaps remain a persistent challenge. Population-level protection depends not only on product availability but also on awareness, timely access, supply logistics, and integration into obstetric, newborn, and pediatric workflows. CDC analyses of early rollout have demonstrated that overall infant RSV immunization coverage has been suboptimal in some settings, with substantial variability by state—highlighting the importance of coordinated public health messaging, reliable distribution channels, and clear clinical pathways that help families receive either maternal vaccination during pregnancy or infant monoclonal antibody protection after birth.[4] In summary, effective prevention requires both rigorous in-hospital infection control to prevent nosocomial spread and robust immunization implementation strategies to translate maternal vaccine and long-acting monoclonal antibody innovations into consistent, equitable reductions in severe RSV disease [4].

Differential Diagnosis

The clinical syndrome produced by RSV—particularly when it progresses to bronchiolitis—

overlaps substantially with other pediatric respiratory conditions that present with cough, tachypnea, wheezing, increased work of breathing, and hypoxemia. Accordingly, the differential diagnosis should be approached as a structured consideration of alternative viral etiologies, airway reactivity disorders, parenchymal lung disease, upper-airway obstruction syndromes, aspiration events, and cardiogenic contributors in children with underlying heart disease. In many infants, especially during peak respiratory-virus seasons, RSV cannot be reliably distinguished from other viral bronchiolitides on clinical grounds alone, because rhinovirus, human metapneumovirus, influenza, and parainfluenza may produce an indistinguishable constellation of rhinorrhea, cough, wheeze, and diffuse crackles. The practical implication is that clinicians should interpret the "RSV-like" phenotype primarily as a severity and disposition problem rather than a pathogen-labeling exercise unless testing is needed for cohorting or outbreak control. Asthma or reactive airway disease should be considered when wheezing is prominent, especially in older infants and toddlers with prior episodes, personal or family atopy, or clear bronchodilator responsiveness. In contrast, bronchiolitis is more likely when the patient is younger, has a first wheezing episode, and demonstrates diffuse findings with feeding compromise. Pneumonia—viral or bacterial—enters the differential when there is focal auscultatory asymmetry, persistent high fever, or disproportionate systemic toxicity; however, radiographic findings may overlap with bronchiolitis, so the diagnosis should be anchored in clinical coherence rather than imaging alone. Croup (laryngotracheobronchitis) should be suspected when inspiratory stridor, a barking cough, and hoarseness dominate, indicating upper-airway involvement rather than small-airway obstruction. Foreign body aspiration requires heightened suspicion when onset is sudden, symptoms are unilateral or episodic, or there is a choking history, as delayed recognition can mimic recurrent "viral wheeze." Finally, congestive heart failure must be considered in infants with congenital heart disease who present with tachypnea, poor feeding, hepatomegaly, diaphoresis, or failure to thrive, because pulmonary edema and bronchiolitis can coexist or be confused clinically, and management priorities differ substantially [19].

Prognosis

The overall prognosis of RSV infection in children is favorable, with most infants and children improving spontaneously over time without requiring hospitalization. Even among those admitted, recovery is typically complete and uncomplicated, reflecting the self-limited nature of RSV in immunocompetent hosts and the effectiveness of modern supportive care. For hospitalized children, the usual length of stay is approximately 3 to 7 days, although the clinical course is more variable in high-risk groups. Infants born extremely premature, those with congenital heart disease, or children with chronic lung disease often experience prolonged hospitalization and higher rates of intensive care utilization, largely due to limited cardiopulmonary reserve and greater vulnerability to hypoxemia and fatigue.[9] Mortality in high-income settings remains low; in the United States, overall mortality is less than 1%, with fewer than 400 deaths annually, but the risk is concentrated in children with significant comorbid conditions, especially congenital heart disease, extreme prematurity, and chronic lung disease.[2] From a transmission standpoint, RSV contagiousness has prognostic relevance for household spread and reinfection risk rather than for individual clinical severity. Most RSV-infected individuals shed virus for roughly 3 to 8 days, but shedding may persist for up to 4 weeks in some infants and immunocompromised patients, which can prolong exposure risk in families, daycare settings, and inpatient units.[11] Beyond the acute episode, long-term outcomes have drawn considerable attention because substantial evidence links severe early-life RSV lower respiratory tract infection with recurrent wheezing and childhood asthma. Observational studies have reported a 2- to 12-fold higher asthma risk among children with prior RSV lower respiratory infection, and children without RSV infection in the first year of life have been reported to have a 26% lower risk of asthma by age 5.[22][23] While the extent to which RSV is causal versus a marker of underlying susceptibility remains debated, the association is clinically meaningful for counseling families, planning follow-up for children with severe bronchiolitis, and reinforcing prevention strategies in infancy [20][21][22][23].

Complications

Complications of RSV infection arise from a combination of airway obstruction, increased work of breathing, impaired feeding, and—in severe disease—systemic stress responses that can affect organs beyond the lungs. In typical pediatric cases, the most frequent complications are dehydration due to poor feeding and increased metabolic demand, acute otitis media related to viral upper-airway inflammation and Eustachian tube dysfunction, and progression from upper respiratory symptoms to lower respiratory tract involvement such as bronchiolitis or viral pneumonia. In young infants, especially those under two to three months of age or born prematurely, apnea represents a particularly concerning complication, sometimes occurring early and requiring inpatient monitoring even when wheezing is not prominent. Severe bronchiolitis can culminate in hypoxemic respiratory failure, necessitating escalation to supplemental oxygen, high-flow therapy, noninvasive ventilation, or mechanical ventilation, with risk amplified by baseline cardiopulmonary disease. Secondary bacterial infection is comparatively uncommon in otherwise healthy infants but may occur, particularly in those requiring intensive care or with prolonged hospitalization, and should be considered when fever is persistent, focal lung findings appear, or clinical toxicity is disproportionate to viral bronchiolitis. Long-term respiratory sequelae are among the most clinically relevant complications, especially after severe early-life infection. Recurrent wheezing episodes and an increased risk of asthma development later in childhood have been repeatedly associated with RSV lower respiratory tract disease, shaping follow-up priorities and family counseling.[9] In addition, extrapulmonary complications—though less frequent—have been increasingly recognized across age groups. Cardiac involvement may include myocarditis and arrhythmias, while neurologic events can include seizures and encephalopathy, underscoring that RSV can be a systemic illness in its most severe forms.[24] Recent data also suggest RSV may directly infect nerve cells, triggering inflammation and raising concern for potential longer-term neurocognitive effects, although the magnitude and persistence of such outcomes remain areas of active investigation.[24] In critically ill infants, systemic abnormalities such as troponin-elevated myocardial injury, arrhythmias, hyponatremia, and hepatitis have been documented, reflecting the physiologic stress of severe infection and the complex interplay of hypoxemia, inflammation, and organ vulnerability.[25] Recognizing these complications early supports timely escalation of care, targeted monitoring, and reduction of downstream morbidity [22][23][24][25].

Patient Education

Effective deterrence of RSV in children depends on integrating practical infection-control behaviors with evidence-based immunoprophylaxis and risk-reduction strategies that limit exposure and severity. At the household and community level, hand hygiene remains the single most impactful preventive measure because RSV is readily transmitted through direct contact with respiratory secretions and contaminated surfaces. Families should be counseled to wash hands thoroughly before and after contact with infants, to disinfect frequently touched objects such as doorknobs and toys, and to avoid close interaction—particularly kissing or face-to-face contact—with individuals who have active respiratory symptoms.[26] Reducing tobacco smoke exposure is another key preventive intervention. Exposure to household smoke increases airway inflammation and vulnerability, and it is associated with more severe RSV disease and a higher likelihood of respiratory complications; therefore, counseling should explicitly address smoke-free environments around infants and pregnant caregivers.[27] Breastfeeding also functions as a protective health behavior: encouraging exclusive breastfeeding for at least six months can enhance infant immune defenses, reduce overall respiratory infection morbidity, and may mitigate RSV severity through passive immune factors and improved baseline health resilience.[28] Patient education should be structured around recognition, response, and prevention. Caregivers benefit from clear guidance on early symptoms—rhinorrhea, cough, feeding difficulty—and on red flags that warrant urgent evaluation, such as persistent tachypnea, retractions, grunting, cyanosis, apnea, lethargy, or reduced urine output. Practical instructions on supportive care at home should include maintaining hydration with small frequent feeds, managing fever appropriately, and using saline drops with gentle nasal suctioning to improve feeding. Education should also address how RSV spreads within households, emphasizing that infants can become ill from siblings and adult caregivers with mild “cold” symptoms. Finally, clinicians should counsel families regarding preventive biomedical options, including maternal vaccination during pregnancy and long-acting monoclonal antibodies for eligible infants, while clarifying that these tools complement—rather than replace—basic infection-control practices. By combining behavioral precautions, environmental risk reduction, breastfeeding support, and immunoprophylaxis awareness, healthcare providers can empower caregivers to reduce RSV incidence, protect high-risk infants, and respond early when clinical deterioration begins.[26][27][28]

Enhancing Healthcare Team Outcomes

RSV remains the leading cause of acute respiratory infection requiring hospitalization in young children worldwide, and optimizing outcomes depends on coordinated interprofessional care across outpatient, emergency, inpatient, and public health settings. Because most infections begin with nonspecific upper respiratory symptoms yet can progress to bronchiolitis—particularly in infants under two years and those with prematurity or chronic comorbidities—teams must prioritize early severity recognition, frequent reassessment, and consistent supportive management. Severe disease can manifest with apnea, hypoxemia, and respiratory failure, making timely escalation of oxygen and ventilatory support a core determinant of safety.[9] Evidence-based practice also requires shared restraint: avoiding routine bronchodilators, corticosteroids, and antibiotics in otherwise healthy infants reduces low-value interventions, limits medication harms, and supports guideline adherence. Prevention strategies—maternal vaccination, long-acting monoclonal antibodies, hand hygiene, breastfeeding, and tobacco smoke avoidance—can reduce morbidity and hospitalization, but suboptimal uptake highlights the importance of team-driven education and implementation. In high-functioning systems, clinicians align roles around a common care pathway. Physicians and advanced practitioners assess risk, define severity, determine hospitalization needs, and initiate supportive orders, while nurses provide continuous monitoring of respiratory status, feeding tolerance, hydration markers, and evolving distress, often identifying deterioration earlier than intermittent provider assessments. Respiratory therapists contribute specialized expertise in oxygen titration, high-flow delivery, escalation to noninvasive support, airway humidification, and safe suctioning practices, all of which directly affect work of breathing and gas exchange. Pharmacists support safe procurement and administration workflows for monoclonal antibodies, reconcile dosing in weight-based pediatric populations, and reinforce medication counseling when comorbidities or concurrent therapies complicate care. Infection-control teams and unit leadership are essential for preventing nosocomial transmission through screening, isolation, cohorting, and staff education, particularly during seasonal surges. Laboratory and radiology services contribute by supporting selective testing and imaging when results will alter cohorting or clarify atypical presentations, reducing unnecessary exposure and costs. When these disciplines communicate effectively—using shared protocols, consistent caregiver messaging, and clear escalation triggers—teams improve patient-centered outcomes, reduce complications, and enhance operational readiness during peak RSV seasons [9][28].

Conclusion:

RSV remains a dominant cause of pediatric hospitalization worldwide, with severe outcomes concentrated among infants and high-risk groups. Although most cases are self-limiting, the potential for rapid deterioration underscores the importance of early recognition and timely escalation of care. Supportive management—hydration, oxygen therapy, and respiratory support—remains the cornerstone of treatment, while evidence strongly discourages routine use of bronchodilators, corticosteroids, and antibiotics in typical bronchiolitis. Recent advances in prevention represent a paradigm shift. Maternal vaccination during late pregnancy and long-acting monoclonal antibodies for infants now provide season-long protection, reducing RSV-related hospitalizations and mortality. These innovations, coupled with rigorous infection-control practices, offer unprecedented opportunities to mitigate RSV's global impact. However, implementation challenges persist, particularly in low-resource settings where access to immunoprophylaxis and advanced respiratory care is limited. Future priorities include improving coverage of maternal and infant immunization programs, strengthening public health education, and ensuring equitable access to preventive and supportive interventions. By combining biomedical advances with coordinated healthcare strategies, RSV-related morbidity and mortality can be significantly reduced, marking a transformative era in pediatric respiratory care.

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