

to allergens, irritants, viral infections, and exercise). It also results in edema, increased mucus production in the lungs, influx of inflammatory cells into the airway tissue, and epithelial cell denudation. Chronic inflammation can lead to **airway remodeling**, which results from a proliferation of extracellular matrix proteins and vascular hyperplasia and may lead to irreversible structural changes and progressive loss of pulmonary function.

EPIDEMIOLOGY

Asthma is the most common chronic disease of childhood in industrialized countries, affecting over 5 million children younger than 18 years of age in the United States (7.5% of children). In adults, females are more likely than males to have asthma, but in children the reverse is true. In 2016 in the United States, pediatric hospitalizations for asthma exceeded 80,000, and there were nearly 550,000 emergency department visits for pediatric asthma. Mortality from asthma occurs; in 2018, 192 deaths were attributed to asthma. For children, the death rate is 2.6 per million.

CLINICAL MANIFESTATIONS

Children with asthma have symptoms of coughing, wheezing, shortness of breath, exercise intolerance, or chest tightness. The history should elicit the frequency, severity, and precipitating factors as well as a family history of asthma and allergy. Common exacerbating factors include respiratory viral infections, exposure to allergens and irritants (e.g., smoke, air pollution, strong odors, fumes), exercise, emotions, and change in weather/humidity. Nocturnal symptoms are common. Rhinosinusitis, gastroesophageal reflux, and aspirin can aggravate asthma. Treatment of these conditions may lessen the frequency and severity of asthma.

During acute episodes, tachypnea, tachycardia, cough, wheezing, and a prolonged expiratory phase may be present. Physical findings may be subtle. Wheezing may not be prominent if there is poor aeration from airway obstruction. As the attack progresses, cyanosis, diminished air movement, retractions, agitation, inability to speak, tripod sitting position, diaphoresis, and pulsus paradoxus (decrease in blood pressure of >15 mm Hg with inspiration) may be observed. Physical examination may show evidence of other atopic diseases such as eczema or allergic rhinitis.

LABORATORY AND IMAGING STUDIES

While no single test or study can confirm the diagnosis of asthma, many elements contribute to establishing the diagnosis.

Objective measurements of pulmonary function aid in the diagnosis and direct the treatment of asthma. **Spirometry** is used to monitor response to treatment, assess degree of reversibility with therapeutic intervention, and measure the severity of an asthma exacerbation. Children older than 5 years of age can usually perform spirometry maneuvers. **Peak expiratory flow** offers a simple, inexpensive way to measure asthma control at home; variability in predicted peak flow reference values make spirometry preferred over peak flow measures in the diagnosis of asthma. For younger children who cannot perform spirometry maneuvers or peak flow, a therapeutic trial of controller medications aids in the diagnosis of asthma.

CHAPTER 78

Asthma

ETIOLOGY

Inflammatory cells (mast cells, eosinophils, T lymphocytes, neutrophils), chemical mediators (histamine, leukotrienes, platelet-activating factor, bradykinin), and chemotactic factors (cytokines, eotaxin) mediate the underlying inflammation found in asthmatic airways. Inflammation contributes to **airway hyperresponsiveness** (airways constricting in response

TABLE 78.1 Differential Diagnosis of Cough and Wheeze in Infants and Children

UPPER AIRWAY DISEASES	OBSTRUCTION INVOLVING LARGE AIRWAYS	OBSTRUCTION INVOLVING SMALL AIRWAYS	OTHER
Allergic rhinitis Sinusitis	Tracheal or bronchial foreign body Paradoxical vocal fold motion Vascular rings or laryngeal webs Enlarged lymph nodes or tumor	Viral bronchiolitis or obliterative bronchiolitis Cystic fibrosis Bronchopulmonary dysplasia (chronic lung disease of prematurity)	Recurrent cough not caused by asthma (infection, habit cough, postnasal drip) Aspiration from swallowing mechanism dysfunction or gastroesophageal reflux disease Heart failure, pulmonary edema

From Guilbert TW, Lemanske RF, Jackson DJ. Diagnosis of asthma in infants and children. In: Adkinson Jr NF, Bochner BS, Burks AW, et al., eds. Middleton's Allergy: Principles and Practice. 8th ed. Philadelphia: Saunders; 2014:870.

TABLE 78.2 Mnemonic of Causes of Cough in the First Months of Life

C —Cystic fibrosis
R —Respiratory tract infections
A —Aspiration (swallowing dysfunction, gastroesophageal reflux, tracheoesophageal fistula, foreign body)
D —Dyskinetic cilia
L —Lung and airway malformations (laryngeal webs, laryngotracheomalacia, tracheal stenosis, vascular rings and slings)
E —Edema (heart failure, congenital heart disease)

From Schidlow DV. Cough. In: Schidlow DV, Smith DS, eds. A Practical Guide to Pediatric Disease. Philadelphia: Hanley & Belfus; 1994.

Measurement of a biomarker, **exhaled nitric oxide (FeNO)**, reflects eosinophilic airway inflammation and provides information on those patients who may better respond to corticosteroid treatment, aids in monitoring response to therapy and dose optimization, and detects nonadherence to corticosteroid therapy.

Allergy skin testing should be included in the evaluation of all children with persistent asthma but not during an exacerbation of symptoms. Positive skin test results, identifying sensitization to **aeroallergens** (e.g., pollens, mold, dust mite, pet dander), correlate strongly with bronchial allergen provocative challenges. In vitro serum tests, such as enzyme-linked immunosorbent assay (ELISA), are generally less sensitive in defining clinically pertinent allergens, are more expensive, and require several days for results, compared to several minutes for skin testing (see Table 77.4).

A **chest x-ray** should be performed with the first episode of asthma or with recurrent episodes of undiagnosed cough or wheeze to exclude anatomic abnormalities. Repeat chest x-rays are not needed with new episodes unless there is fever (suggesting pneumonia) or localized findings on physical examination.

DIFFERENTIAL DIAGNOSIS

Many childhood conditions can cause wheezing and coughing like asthma (Table 78.1), but not all cough and wheeze is asthma. Misdiagnosis delays correcting the underlying cause and exposes children to inappropriate therapy (Table 78.2).

Allergic bronchopulmonary aspergillosis is a hypersensitivity reaction to antigens of the mold *Aspergillus fumigatus*. It occurs primarily in patients with steroid-dependent asthma and in patients with cystic fibrosis.

TREATMENT

Optimal medical treatment of asthma includes several key components: environmental control, pharmacologic therapy, and patient education, including attainment of self-management skills. Because many children with asthma have coexisting allergies, steps to minimize allergen exposure should be taken (Table 78.3). For all children with asthma, exposures to tobacco and wood smoke and to persons with viral infections should be minimized. Influenza immunization is indicated. Asthma medications can be divided into long-term control medications and quick-relief medications.

Long-Term Control Medications

Inhaled Corticosteroids

Inhaled corticosteroids (ICS) are the most effective antiinflammatory medications for the treatment of chronic, persistent asthma and are the preferred therapy when initiating long-term control therapy. Early intervention with ICS reduces morbidity but does not alter the natural history of asthma. Regular use reduces airway hyperreactivity, the need for rescue bronchodilator therapy, risk of hospitalization, and risk of death from asthma. ICS are available as inhalation aerosols, dry powder inhalers, and a nebulizer solution.

The potential risks of ICS are favorably balanced with their benefits. A reduction in growth velocity may occur with poorly controlled asthma or ICS use. Low-to-medium dose ICS may decrease growth velocity, although these effects are small (approximately 1 cm in the first year of treatment), generally not progressive, and may be reversible. Height measurements should be monitored. ICS do not have clinically significant adverse effects on hypothalamic-pituitary-adrenal axis function, glucose metabolism, subcapsular cataracts, or glaucoma when used at low-to-medium doses in children. Rinsing the mouth after inhalation and using large volume spacers help lessen the local adverse effects of dysphonia and candidiasis and decrease systemic absorption from the gastrointestinal tract. ICS should be titrated to the lowest dose needed to maintain control of a child's asthma. For children with severe asthma, high-dose ICS may be needed to minimize oral

TABLE 78.3 Controlling Factors Contributing to Asthma Severity

MAJOR INDOOR TRIGGERS FOR ASTHMA	SUGGESTIONS FOR REDUCING EXPOSURE
Viral upper respiratory tract (e.g., RSV, rhinovirus, influenza virus)	Limit exposure to viral infections
	Annual influenza immunization
Tobacco smoke, wood smoke	No smoking around the child or in child's home
	Help parents and caregivers quit smoking
	Eliminate use of wood stoves and fireplaces
Dust mites	ESSENTIAL ACTIONS
	Encase pillow, mattress, and box spring in allergen-impermeable encasements
	Wash bedding in hot water weekly
	DESIRABLE ACTIONS
	Avoid sleeping or lying on upholstered furniture
	Minimize number of stuffed toys in child's bedroom
	Reduce indoor humidity to <50%
	If possible, remove carpets from bedroom and play areas; if not possible, vacuum weekly
Animal dander	Remove the pet from the home or keep outdoors (if removal is not acceptable)
	Keep pet out of bedroom/no sleeping with pet
	Use a filter on air ducts in child's room
	Wash pet weekly (the evidence to support this has not been firmly established)
Cockroach allergens	Do not leave food or garbage exposed
	Pesticides
	Sealing cracks and holes
Indoor mold	Avoid vaporizers
	Reduce indoor humidity to <50%
	Use of mold inhibiting cleaner
	Use of dehumidifier
	Fix leaky faucets, pipes

RSV, Respiratory syncytial virus.

From American Academy of Allergy Asthma & Immunology. Pediatric Asthma: Promoting Best Practice. Milwaukee, WI: American Academy of Allergy Asthma & Immunology; 1999:50.

corticosteroid use, but other add-on therapy should be considered (see the following sections).

Leukotriene Modifiers

Leukotrienes, synthesized via the arachidonic acid metabolism cascade, are potent mediators of inflammation and smooth

muscle bronchoconstriction. Leukotriene modifiers are oral, daily-use medications that inhibit these biologic effects in the airway. Two classes of leukotriene modifiers include cysteinyl leukotriene receptor antagonists (zafirlukast and montelukast) and leukotriene synthesis inhibitors (zileuton). The leukotriene receptor antagonists have much wider appeal than zileuton. Zafirlukast is approved for children older than 5 years of age and is given twice daily. Montelukast is dosed once daily at night as 4-mg granules or chewable tablets (for children 6 months to 5 years of age), 5-mg chewable tablets (6–14 years of age), and 10-mg tablets (≥ 15 years of age). Pediatric studies show the usefulness of leukotriene modifiers in mild asthma and the attenuation of exercise-induced bronchoconstriction. These agents may be helpful as steroid-sparing agents in patients with asthma that is more difficult to control.

Long-Acting β_2 Agonists

Long-acting β_2 agonists (LABAs) relax airway smooth muscle for 12–24 hours but do not have any significant antiinflammatory effects. LABAs are not intended for use as monotherapy, but adding a LABA to ICS therapy is more beneficial than doubling the dose of inhaled corticosteroids. Multiple formulations are available. Formoterol is approved for use in children older than 5 years of age for maintenance asthma therapy and for prevention of exercise-induced asthma. It has a rapid onset of action similar to albuterol (15 min) and is dosed twice daily. Salmeterol is approved for children 4 years of age or older, has an onset of 30 min, and is given twice daily. Vilanterol, an ultralong-acting β_2 agonist, is approved in patients aged 18 years and older and has the advantage of once-daily dosing. Combination ICS/LABA products are available. Because combination inhalers administer two medications simultaneously, compliance is generally improved.

Theophylline

Theophylline was more widely used previously, but because current management is aimed at inflammatory control, its use has declined. It is mildly to moderately effective as a bronchodilator and is considered an alternative, add-on treatment to low- and medium-dose inhaled corticosteroids. Its therapeutic window is narrow necessitating monitoring of blood levels.

Biologics

Biologic agents are genetically engineered proteins that target specific antibodies, inflammatory molecules, and receptors; several biologics are U.S. Food and Drug Administration (FDA) approved for the treatment of asthma in children (Table 78.4). Omalizumab, the first biologic approved for asthma, is a humanized anti-IgE monoclonal antibody that prevents binding of free IgE to high-affinity receptors on basophils and mast cells. It is delivered by subcutaneous injection every 2–4 weeks, depending on body weight and pretreatment serum IgE level.

Mepolizumab is an add-on maintenance therapy for severe asthma with an eosinophilic phenotype. It decreases the production and survival of eosinophils, a major inflammatory cell involved in asthma pathogenesis.

Dupilumab binds to the IL-4 receptor, which blocks intracellular signaling of IL-4 and IL-13. This medication can be administered at home.

Benralizumab binds the IL-5 receptor inhibiting IL-5 activity and induces eosinophil apoptosis. Reslizumab also binds

TABLE 78.4 List of U.S. Food and Drug Administration–Approved Biologic Medications for the Treatment of Asthma in Children and Their Characteristics

NAME	INDICATION	MECHANISM OF ACTION	DOSE	MOST COMMON SIDE EFFECTS	ADMINISTRATION CONSIDERATIONS	OTHER
Benralizumab (Fasenra)	Add-on controller therapy for severe eosinophilic asthma >12yr of age	- IL-5 receptor (at the α-subunit) blocker - Induces apoptosis of eosinophils through ADCC*	30 mg SC every 4wk x 3 - Then every 8 wk	- Injection site reactions - Headache - Pharyngitis	- Treat preexisting helminth infections 30-min observation from first dose	Improved efficacy if AEC† >150 cells/μL
Dupilumab (Dupixent)	Add-on controller therapy for moderate to severe persistent asthma eosinophilic phenotype or oral corticosteroid dependent >12yr of age	IL-4 receptor blocker (at α subunit) preventing IL-4 and IL-13 signaling	400 mg SC once then 200 mg SC every 2 wk OR 600 mg SC once then 300 mg every 2 wk - With concomitant moderate to severe atopic dermatitis, 600 mg once then 300 mg every 2 wk	- Injection site reactions - Eosinophilia - Oropharyngeal pain	- Avoid live vaccines - Treat preexisting helminth infections - Option for self-administration	Improved efficacy if AEC >150 cells/μL
Mepolizumab (Nucala)	Add-on controller therapy for severe persistent asthma eosinophilic phenotype >6yr of age	IL5 antagonist preventing binding of IL-5 receptor on eosinophils	- Patients >12yr of age: 100 mg SC every 4wk - Patients 6–11yr of age SC 40 mg every 4wk	- Injection site reactions - Herpes zoster - Headache - Back pain - Fatigue	- Zoster vaccination - Treat preexisting helminth infections 30-min observation from first dose	- AEC >150 in last 6 wk OR - AEC 300 in last 12 months
Omalizumab (Xolair)	Moderate to severe persistent asthma aged >6yr of age with sensitivity to perennial aeroallergens	Binds free IgE preventing IgE binding to high affinity IgE receptor on mast cells and basophils minimizing release of chemical mediators involved in allergic inflammatory cascade	- Dependent on age, weight, and total IgE level - Administered SC every 2–4 wk	- Anaphylaxis - Injection site reactions - Arthralgia	- Patients should have autoinjectable epinephrine at time of injection 90-min wait after first 3 injections, then 30-min wait with subsequent doses - Equipment available to treat anaphylaxis	- Total IgE >30 ku/L AND - Positive skin prick test or serum-specific IgE to perennial aeroallergen

*Antibody-dependent cell-mediated cytotoxicity.

AEC, Absolute blood eosinophil count; IL, interleukin; SC, subcutaneous.

to IL-5, preventing its binding to the IL-5 receptor but is only approved for patients over 18 years of age.

Quick-Relief Medications

Short-Acting β_2 Agonists

Short-acting β_2 agonists (SABAs) such as albuterol and levalbuterol are effective bronchodilators that relax bronchial smooth muscle within 5–10 minutes of administration. These effects last 4–6 hours. Generally, a SABA is prescribed for acute symptoms and as prophylaxis before allergen exposure and exercise. The inhaled route is preferred because adverse effects—tremor, prolonged tachycardia, and irritability—are less. Overuse of SABAs implies inadequate control, and a change in medications may be warranted. The definition of *bronchodilator overuse* depends on the severity of the child's asthma; use of more than one metered-dose inhaler canister per month or daily use suggests poor control.

Anticholinergic Agents

Ipratropium bromide is a short-acting anticholinergic bronchodilator that relieves bronchoconstriction, decreases mucus hypersecretion, and counteracts cough-receptor irritability by binding acetylcholine at the muscarinic receptors found in bronchial smooth muscle. It seems to have an additive effect with SABAs when used for acute asthma exacerbations.

A long-acting anticholinergic agent (tiotropium) is approved for use in moderate to severe persistent asthma in children 6 years or older. It inhibits muscarinic receptors on bronchial smooth muscle to induce bronchodilation.

Oral Corticosteroids

Short bursts of oral corticosteroids (1–10 days) are administered to children with acute exacerbations. The usual dose is 1–2 mg/kg/day of prednisone for 5 days or 0.6 mg/kg/day of dexamethasone for 1–2 days. Oral corticosteroids are available in liquid or tablet formulations. Prolonged use of oral corticosteroids can result in systemic adverse effects such as hypothalamic-pituitary-adrenal suppression, cushingoid features, weight gain, hypertension, diabetes, cataracts, glaucoma, impaired immunity, osteoporosis, and growth suppression. Children with severe asthma may require oral corticosteroids over extended periods. The dose should be tapered as soon as possible to the minimum effective dose, preferably administered on alternate days (for prednisone).

Administration of one or two doses of dexamethasone for the treatment of *acute asthma* exacerbations is used, usually in an emergency room setting for intermittent, mild persistent, or moderate persistent asthma. This long-acting oral corticosteroid has properties that may increase compliance with therapy, decrease missed school days, and decrease readmission rates.

Approach to Therapy

Therapy is based on the concept that chronic airway inflammation is a fundamental feature of asthma, which can vary in intensity over time, requiring treatment to be adjusted accordingly. Classification of asthma severity is emphasized for initiation of therapy in patients not currently receiving controller

medications. Assessing control is emphasized for monitoring and adjusting therapy. A stepwise approach is used for management of infants and young children 0–4 years of age, children 5–11 years of age (Fig. 78.1), and youths 12 years or older, and adults (Fig. 78.2). Medication type, dose, and dosing intervals are determined by the level of asthma severity or asthma control. Therapy is then increased (**stepped up**) as necessary and decreased (**stepped down**) when possible. A short-acting bronchodilator should be available for all children with asthma. A child with intermittent asthma has asthma symptoms less than two times per week. To determine whether a child is having more persistent asthma, using the **Rules of Two** is helpful: daytime symptoms occurring two or more times per week or nocturnal awakenings two or more times per month implies a need for daily antiinflammatory medication.

Inhaled corticosteroids are the preferred initial long-term control therapy for children of all ages (Fig. 78.3). Daily long-term control therapy is recommended for infants and young children 0–4 years of age who had four or more episodes of wheezing in the previous year that lasted more than 1 day, affected sleep, and who have a positive asthma predictive index. For children over 5 years of age with moderate persistent asthma, combining long-acting bronchodilators with low-to-medium doses of inhaled corticosteroids improves lung function and reduces rescue medication use. For children with severe persistent asthma, a high-dose inhaled corticosteroid combined with a long-acting bronchodilator is the preferred therapy. The guidelines also recommend that treatment be reevaluated within 4–6 weeks of initiating therapy. Once asthma is under control, control should be assessed on an ongoing basis every 1–6 months. Asthma should be well controlled for at least 3 months before stepping down therapy.

COMPLICATIONS

Most asthma exacerbations can be successfully managed at home. **Status asthmaticus** is an acute exacerbation of asthma that does not respond adequately to therapeutic measures and may require hospitalization. Exacerbations may progress over several days or occur suddenly and can range in severity from mild to life threatening. Significant respiratory distress, dyspnea, wheezing, cough, and a decrease in **spirometry** or **peak expiratory flow rate (PEFR)** characterize deterioration in asthma control. During a severe episode, pulse oximetry is helpful in monitoring oxygenation. In status asthmaticus, arterial blood gases may be necessary for measurement of ventilation. As airway obstruction worsens and chest compliance decreases, carbon dioxide retention can occur. In the face of tachypnea, a *normal* P_{CO_2} (35–45 mm Hg) indicates impending respiratory arrest.

First-line management of asthma exacerbations includes supplemental oxygen if needed and repetitive or continuous administration of SABAs. Early administration of oral or intravenous corticosteroids (Fig. 78.4) is important in treating the underlying inflammation. Administration of anticholinergic agents (ipratropium) with bronchodilators decreases rates of hospitalization and duration of time in the emergency department. Intravenous magnesium sulfate may be administered in the emergency department if there is clinical deterioration despite treatment with β_2 agonists, ipratropium, and systemic corticosteroids. The typical dose of magnesium

		Step up if needed (first check inhaler technique, adherence, environmental control, and comorbid conditions)						→						
		Assess control												
		Step down if possible (and asthma is well controlled at least 3 months)							←					
		Step 1		Step 2		Step 3		Step 4		Step 5		Step 6		
		Notes												
Children 0–4 years of age		Intermittent asthma		Persistent asthma: Daily medication Consult with asthma specialist if Step 3 care or higher is required. Consider consultation at Step 2.							• The stepwise approach is meant to assist, not replace, the clinical decision making required to meet individual patient needs. • If an alternative treatment is used and response is inadequate, discontinue it and use the preferred treatment before stepping up. • If clear benefit is not observed within 4–6 weeks, and patient's/family's medication technique and adherence are satisfactory, consider adjusting therapy or an alternative diagnosis. • Studies on children 0–4 years of age are limited. Step 2 preferred therapy is based on Evidence A. All other recommendations are based on expert opinion and extrapolation from studies in older children. • Clinicians who administer immunotherapy should be prepared and equipped to identify and treat anaphylaxis that may occur. Key: Alphabetical listing is used when more than one treatment option is listed within either preferred or alternative therapy. ICS, Inhaled corticosteroid; LABA, inhaled long-acting beta ₂ -agonist; LTRA, leukotriene receptor antagonist, oral corticosteroids, oral systemic corticosteroids; SABA, inhaled short-acting beta ₂ -agonist			
	Preferred	SABA PRN	Low-dose ICS	Medium-dose ICS	Medium-dose ICS + LABA or Montelukast	High-dose ICS + LABA or Montelukast	High-dose ICS + LABA or Montelukast + Oral corticosteroids ICS							
	Alternative		Cromolyn or Montelukast											
		Each Step: Patient Education and Environmental Control												
	Quick-Relief Medication	• SABA as needed for symptoms. Intensity of treatment depends on severity of symptoms. • With viral respiratory symptoms: SABA q 4–6 hours up to 24 hours (longer with physician consult). Consider short course of oral systemic corticosteroids if exacerbation is severe or patient has history of previous severe exacerbations. Caution: Frequent use of SABA may indicate the need to step up treatment. See text for recommendations on initiating daily long-term-control therapy.												
Children 5–11 Years of Age		Intermittent Asthma		Persistent Asthma: Daily Medication Consult with asthma specialist if Step 4 care or higher is required. Consider consultation at Step 3.					• The stepwise approach is meant to assist, not replace, the clinical decision making required to meet individual patient needs. • If an alternative treatment is used and response is inadequate, discontinue it and use the preferred treatment before stepping up. • Theophylline is a less desirable alternative due to the need to monitor serum concentration levels. • Steps 1 and 2 medications are based on Evidence A. Step 3 ICS and ICS plus adjunctive therapy are based on Evidence B for efficacy of each treatment and extrapolation from comparator trials in older children and adults—comparator trials are not available for this age group; Steps 4–6 are based on expert opinion and extrapolation from studies in older children and adults. • Immunotherapy for Steps 2–4 is based on Evidence B for house-dust mites, animal danders, and pollens; evidence is weak or lacking for molds and cockroaches. Evidence is strongest for immunotherapy with single allergens. The role of allergy in asthma is greater in children than adults. • Clinicians who administer immunotherapy should be prepared and equipped to identify and treat anaphylaxis that may occur. Key: Alphabetical listing is used when more than one treatment option is listed within either preferred or alternative therapy. ICS, Inhaled corticosteroid; LABA, inhaled long-acting beta ₂ -agonist; LTRA, leukotriene receptor antagonist; SABA, inhaled short-acting beta ₂ -agonist					
	Preferred	SABA PRN	Low-dose ICS	Low-dose ICS + LABA, LTRA, or Theophylline	Medium-dose ICS + LABA	High-dose ICS + LABA	High-dose ICS + LABA + Oral corticosteroids							
	Alternative		Cromolyn LTRA Nedocromil, or Theophylline	Medium-dose ICS	Medium-dose ICS + LTRA or Theophylline	High-dose ICS + LTRA or Theophylline	High-dose ICS + LTRA or Theophylline + Oral corticosteroids							
		Each Step: Patient Education and Environmental Control, and Management of Comorbidities Steps 2–4: Consider subcutaneous allergen immunotherapy for patients who have persistent, allergic asthma.												
	Quick-Relief Medication	• SABA as needed for symptoms. Intensity of treatment depends on severity of symptoms: up to 3 treatments at 20-minute intervals as needed. Short course of oral systemic corticosteroids may be needed. Caution: Increasing use of SABA or use >2 days a week for symptom relief (not prevention of EIB) generally indicates inadequate control and the need to step up treatment.												

FIGURE 78.1 Stepwise long-term approach for managing asthma in children, 0–4 years of age and 5–11 years of age. (From National Heart Lung Blood Institute, National Asthma Education and Prevention Program. Expert panel report 3: guidelines for the diagnosis and management of asthma. Summary report 2007, NIH Publication No. 08-5846. Bethesda, MD: U.S. Department of Health and Human Services; 2007:42. <https://www.nhlbi.nih.gov/sites/default/files/media/docs/asthsumm.pdf>.)

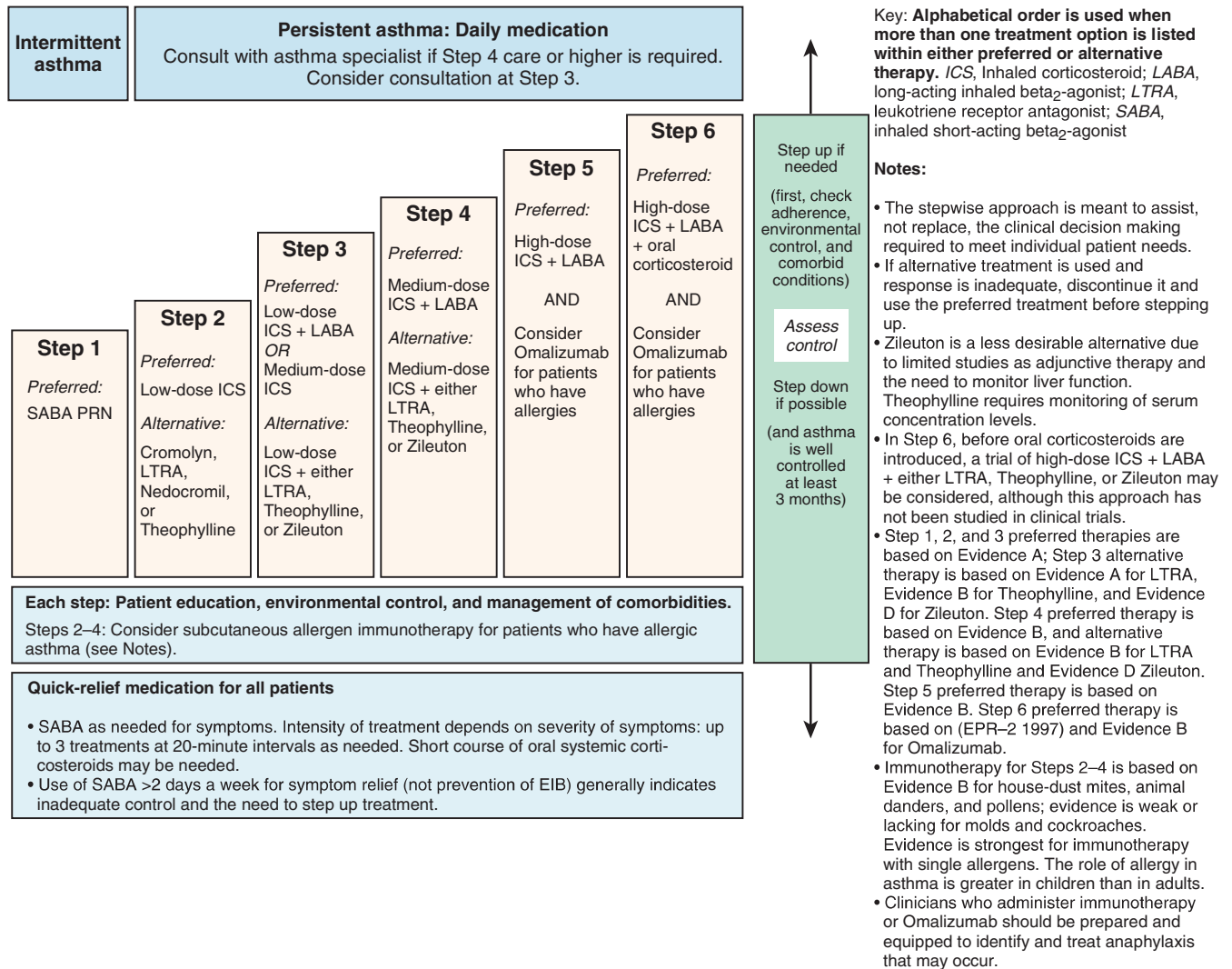


FIGURE 78.2 Stepwise approach for managing asthma in youths 12 years of age or older and adults. (From National Heart Lung Blood Institute, National Asthma Education and Prevention Program. Expert panel report 3: guidelines for the diagnosis and management of asthma. Summary report 2007, NIH Publication No. 08-5846. Bethesda, MD: U.S. Department of Health and Human Services; 2007:45. <https://www.nhlbi.nih.gov/sites/default/files/media/docs/asthsumm.pdf>.)

sulfate is 25–75 mg/kg (maximum 2.0 g) intravenously administered over 20 min. Intramuscular epinephrine or subcutaneous terbutaline is rarely used except when severe asthma is associated with anaphylaxis or is unresponsive to continuous administration of SABAs.

PROGNOSIS

For some children, symptoms of wheezing with respiratory infections subside in the preschool years, whereas others have more persistent asthma symptoms. Prognostic indicators for children younger than 3 years of age who are at risk for persistent asthma are known as the Modified Asthma Predictive Index for children (Table 78.5). Atopy is the strongest predictor for wheezing continuing into persistent asthma (Table 78.6).

PREVENTION

Education plays an important role in helping patients and their families adhere to the prescribed therapy and needs to begin at the time of diagnosis. Successful education involves teaching basic asthma facts, explaining the role of medications, teaching environmental control measures, and improving patient skills in the use of spacer devices for metered-dose inhalers and peak flow monitoring. Families should have an asthma management plan (Fig. 78.5) for daily care and for exacerbations.

Peak flow monitoring is a self-assessment tool that may be helpful for children with asthma over 5 years of age who are poor perceivers of airway obstruction, have moderate to severe asthma, or have a history of severe exacerbations. Peak flow monitoring also can be useful in children who are still learning to recognize asthma symptoms. It must be emphasized that

Drug	Low Daily Dose			Medium Daily Dose			High Daily Dose		
	Child 0–4 Years of Age	Child 5–11 Years of Age	12 Years of Age and Adults	Child 0–4 Years of Age	Child 5–11 Years of Age	12 Years of Age and Adults	Child 0–4 Years of Age	Child 5–11 Years of Age	12 Years of Age and Adults
Beclomethasone HFA 40 or 80 µg/puff	NA	80–160 µg	80–240 µg	NA	>160–320 µg	>240–480 µg	NA	>320 µg	>480 µg
Budesonide DPI 90, 180, or 200 µg/inhalation	NA	180–400 µg	180–600 µg	NA	>400–800 µg	>600–1,200 µg	NA	>800 µg	>1,200 µg
Budesonide Inhaled Inhalation suspension for nebulization	0.25–0.5 mg	0.5 mg	NA	>0.5–1.0 mg	1.0 mg	NA	>1.0 mg	2.0 mg	NA
Flunisolide 250 µg/puff	NA	500–750 µg	500–1,000 µg	NA	1,000–1,250 µg	>1,000–2,000 µg	NA	>1,250 µg	>2,000 µg
Flunisolide HFA 80 µg/puff	NA	160 µg	320 µg	NA	320 µg	>320–640 µg	NA	640 µg	>640 µg
Fluticasone HFA/MDI : 44, 110, or 220 µg/puff	176 µg	88–176 µg	88–264 µg	>176–352 µg	>176–352 µg	>264–440 µg	>352 µg	>352 µg	>440 µg
DPI : 50, 100, or 250 µg/inhalation	NA	100–200 µg	100–300 µg	NA	>200–400 µg	>300–500 µg	NA	>400 µg	>500 µg
Mometasone DPI 200 µg/inhalation	NA	NA	200 µg	NA	NA	400 µg	NA	NA	>400 µg
Triamcinolone acetonide 75 µg/puff	NA	300–600 µg	300–750 µg	NA	>600–900 µg	>750–1,500 µg	NA	>900 µg	>1,500 µg

Key: *DPI*, Dry power inhaler; *HFA*, hydrofluoroalkane; *MDI*, metered-dose inhaler; *NA*, not available (either not approved, no data available, or safety and efficacy not established for this age group)

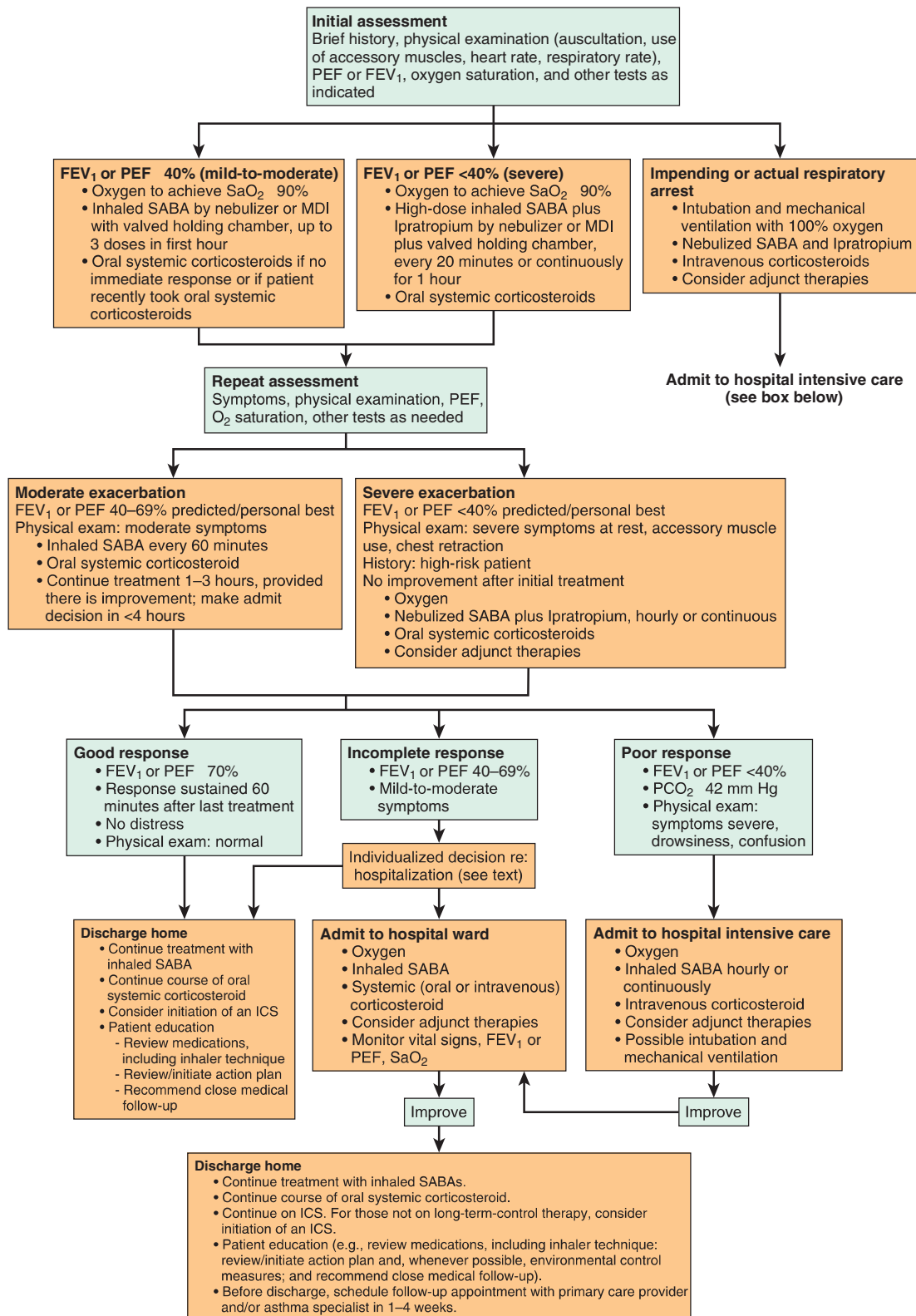
Therapeutic Issues:

- The most important determinant of appropriate dosing is the clinician's judgment of the patient's response to therapy. The clinician must monitor the patient's response on several clinical parameters and adjust the dose accordingly. Once control of asthma is achieved, the dose should be carefully titrated to the minimum dose required to maintain control.
- Preparations are not interchangeable on a µg or per puff basis. This figure presents estimated comparable daily doses. See EPR-3 Full Report 2007 for full discussion.
- Some doses may be outside package labeling, especially in the high-dose range. Budesonide nebulizer suspension is the only inhaler corticosteroid (ICS) with FDA-approved labeling for children <4 years of age.
- For children <4 years of age: The safety and efficacy of ICSs in children <1 year has not been established. Children <4 years of age generally require delivery of ICS (budesonide and fluticasone HFA) through a face mask that should fit snugly over nose and mouth and avoid nebulizing in the eyes. Wash face after each treatment to prevent local corticosteroid side effects. For budesonide, the dose may be administered 1–3 times daily. Budesonide suspension is compatible with Albuterol, Ipratropium, and Levalbuterol nebulizer solutions in the same nebulizer. Use only jet nebulizers, as ultrasonic nebulizers are ineffective for suspensions. For Fluticasone HFA, the dose should be divided 2 times daily; the low dose for children <4 years of age is higher than for children 5–11 years of age due to lower dose delivered with face mask and data on efficacy in young children.

Potential Adverse Effects of Inhaled Corticosteroids:

- Cough, dysphonia, oral thrush (candidiasis).
- Spacer or valved holding chamber with non-breath-actuated MDIs and mouthwashing and spitting after inhalation decrease local side effects.
- A number of the ICSs, including Fluticasone, Budesonide, and Mometasone, are metabolized in the gastrointestinal tract and liver by CYP 3A4 isoenzymes. Potent inhibitors of CYP 3A4, such as Ritonavir and Ketoconazole, have the potential for increasing systemic concentrations of these ICSs by increasing oral availability and decreasing systemic clearance. Some cases of clinically significant Cushing syndrome and secondary adrenal insufficiency have been reported.
- In high doses, systemic effects may occur, although studies are not conclusive, and clinical significance of these effects has not been established (e.g., adrenal suppression, osteoporosis, skin thinning, and easy bruising). In low-to-medium doses, suppression of growth velocity has been observed in children, but this effect may be transient, and the clinical significance has not been established.

FIGURE 78.3 Estimated comparative daily dosages for inhaled corticosteroids. (From National Heart Lung Blood Institute, National Asthma Education and Prevention Program. Expert panel report 3: guidelines for the diagnosis and management of asthma. Summary report 2007, NIH Publication No. 08-5846. Bethesda, MD: U.S. Department of Health and Human Services; 2007:49. <https://www.nhlbi.nih.gov/sites/default/files/media/docs/asthsumm.pdf>.)



Key: FEV₁, Forced expiratory volume in 1 second; ICS, inhaled corticosteroid; MDI, metered-dose inhaler; PCO₂, partial pressure carbon dioxide; PEF, peak expiratory flow; SABA, short-acting beta₂-agonist; SaO₂, oxygen saturation

FIGURE 78.4 Management of asthma exacerbations: emergency department and hospital-based care. (From National Heart Lung Blood Institute, National Asthma Education and Prevention Program. Expert panel report 3: guidelines for the diagnosis and management of asthma. Summary report 2007, NIH Publication No. 08-5846. Bethesda, MD: U.S. Department of Health and Human Services; 2007:55. <https://www.nhlbi.nih.gov/sites/default/files/media/docs/asthsumm.pdf>.)

TABLE 78.5 Modified Asthma Predictive Index for Children	
At least four wheezing episodes <i>plus</i> :	
One major criteria	Or two minor criteria
Parental asthma	Allergic rhinitis
Eczema	Wheezing apart from colds
Inhalant allergen sensitization	Eosinophils $\geq 4\%$
	Food allergen sensitization

From Liu AH, Martinez FD. Natural history of allergic diseases and asthma. In: Leung DYM, Szefer SJ, Bonilla FA, et al., eds. *Pediatric Allergy: Principles and Practice*. 3rd ed. Philadelphia: Elsevier; 2016 (fig. 2.4).

peak flow monitoring is not for the diagnosis of asthma but for monitoring.

A child's personal best PEFr is the highest value achieved over a 2-week period when asthma is controlled. Percentages based on the best PEFr reading may then be incorporated into a written asthma management plan as a means of providing an objective indication of airway obstruction to guide home-based asthma care. Limitations exist, however, to PEFr: they are effort and technique dependent, peak flow meters may lose accuracy with time, and appropriate range meters (low vs high) must be provided.

Utilizing PEFr and written asthma management plans simultaneously may impact asthma monitoring and management. Children with asthma should be seen not only when they are ill but also when they are healthy. Regular office visits allow the health care team to review adherence to medication and control measures and to determine whether doses of medications need adjustment.

PEARLS FOR PRACTITIONERS

See *Allergy: Pearls for Practitioners* at the end of this section.

TABLE 78.6 Risk Factors for Persistent Asthma	
Allergy	Atopic dermatitis
	Allergic rhinitis
	Elevated total serum IgE levels (first year of life)
	Peripheral blood eosinophilia $>4\%$ (2–3yr of age)
	Inhalant and food allergen sensitization
Gender	Males
	• Transient wheezing
	• Persistent allergy-associated asthma
	Females
	• Asthma associated with obesity and early-onset puberty
	• “Triad” asthma (adulthood)
Parental asthma	
Lower respiratory tract infections	Rhinovirus, respiratory syncytial virus
	Severe bronchiolitis (i.e., requiring hospitalization)
	Pneumonia
Environmental tobacco smoke exposure (including prenatal)	

Triad asthma—nasal polyps, aspirin sensitivity, asthma.

From Liu AH, Martinez FD. Natural history of allergic diseases and asthma. In: Leung DYM, Szefer SJ, Bonilla FA, et al., eds. *Pediatric Allergy: Principles and Practice*. 3rd ed. Philadelphia: Elsevier; 2016:10.

Asthma Action Plan

For: _____
 Doctor's Phone Number: _____
 Hospital/Emergency Department Phone Number: _____

Doctor: _____
 Date: _____

GREEN ZONE

Doing Well

- No cough, wheeze, chest tightness, or shortness of breath during the day or night
- Can do usual activities

And, if a peak flow meter is used,
Peak flow: more than _____
 (80 percent or more of my best peak flow)

My best peak flow is: _____

Take these long-term-control medicines each day (include an anti-inflammatory).
 Medicine _____ How much to take _____
 When to take it _____

Identify and avoid and control the things that make your asthma worse, like (list here):

Before exercise, if prescribed, take: ☐ 2 or ☐ 4 puffs _____ 5 to 60 minutes before exercise

YELLOW ZONE

Asthma Is Getting Worse

- Cough, wheeze, chest tightness, or shortness of breath, or
- Waking at night due to asthma, or
- Can do some, but not all, usual activities

-Or-
Peak flow: _____ to _____
 (50 to 79 percent of my best peak flow)

First

Add: quick-relief medicine-and keep taking your GREEN ZONE medicine.

_____ ☐ 2 or ☐ 4 puffs, every 20 minutes for up to 1 hour
 (short-acting beta₂-agonist) ☐ Nebulizer, once

If applicable, remove yourself from the thing that made your asthma worse.
 If your symptoms (and peak flow, if used) return to GREEN ZONE after 1 hour of above treatment:
☐ Continue monitoring to be sure you stay in the green zone.

Second

-Or-
 If your symptoms (and peak flow, if used) do not return to GREEN ZONE after 1 hour of above treatment:
☐ Take: _____ ☐ 2 or ☐ 4 puffs or ☐ Nebulizer
 (short-acting beta₂-agonist) mg per day. For _____ (3-10) days
☐ Add: _____ (oral corticosteroid)
☐ Call the doctor _____ within _____ hours after taking the oral corticosteroid.
 (phone)

RED ZONE

Medical Alert!

- Very short of breath, or
- Quick-relief medicines have not helped, or
- Cannot do usual activities, or
- Symptoms are same or get worse after 24 hours in Yellow Zone.

-Or-
Peak flow: less than _____
 (50 percent of my best peak flow)

Take this medicine:

☐ _____ ☐ 4 or ☐ 6 puffs or ☐ Nebulizer
 (short-acting beta₂-agonist) mg
☐ _____ (oral corticosteroid)

Then call your doctor NOW. Go to the hospital or call an ambulance if

- You are still in the red zone after 15 minutes AND
- You have not reached your doctor.

DANGER SIGNS

- Trouble walking and talking due to shortness of breath
- Lips or fingernails are blue

Now!
 Take ☐ 4 or ☐ 6 puffs of your quick-relief medicine AND
 Go to the hospital or call for an ambulance _____ (phone)

FIGURE 78.5 Asthma self-management guideline. (From National Heart Lung Blood Institute, National Asthma Education and Prevention Program. Expert panel report 3: guidelines for the diagnosis and management of asthma. Summary report 2007, NIH Publication No. 05-5251. Bethesda, MD: U.S. Department of Health and Human Services; 2007:119. <https://www.nhlbi.nih.gov/sites/default/files/media/docs/asthsumm.pdf>.)

CHAPTER 79

Allergic Rhinitis

ETIOLOGY

Rhinitis describes diseases that involve inflammation of the nasal epithelium and is characterized by sneezing, pruritus, rhinorrhea, and congestion. There are many different causes of rhinitis in children, but approximately half of all cases are caused by allergies.

Allergic rhinitis, commonly known as **hay fever**, is caused by an IgE-mediated allergic response. During the early allergic phase, mast cells degranulate and release preformed chemical mediators, such as histamine and tryptase, and newly generated mediators such as leukotrienes, prostaglandins, and platelet-activating factor. After a quiescent phase in which other cells are recruited, a late phase occurs approximately 4–8 hours later. Eosinophils, basophils, CD4 T cells, monocytes, and neutrophils release their chemical mediators, which leads to the development of chronic nasal inflammation.

Allergic rhinitis can be seasonal, perennial, or episodic depending on the specific causative allergen and exposure. Some children experience perennial symptoms with seasonal exacerbations. **Seasonal allergic rhinitis** is caused by airborne pollens, which have seasonal patterns. Typically, trees pollinate in the spring, grasses in late spring to summer, and weeds in the summer and fall. The pollen, microscopic in size, can travel airborne hundreds of miles and be inhaled easily into the respiratory tract. **Perennial allergic rhinitis** is primarily caused by indoor allergens, such as house dust mites, animal dander, mold, and cockroaches. **Episodic rhinitis** occurs with intermittent exposure to allergens, such as visiting a friend's home where a pet lives.

EPIDEMIOLOGY

Chronic rhinitis is one of the most common disorders encountered in infants and children. Overall allergic rhinitis is observed in 10–25% of the general population, with children and adolescents more commonly affected than adults. The prevalence of physician-diagnosed allergic rhinitis has been estimated at 40%.

CLINICAL MANIFESTATIONS

The hallmark symptoms of allergic rhinitis are clear thin rhinorrhea, nasal congestion, sneezing paroxysms, and pruritus of the eyes, nose, ears, and palate. Postnasal drip may result in frequent attempts to clear the throat, nocturnal cough, and hoarseness. It is important to correlate the onset, duration, and severity of symptoms with seasonal or perennial exposures, changes in the home or school environment, and exposure to nonspecific irritants such as tobacco smoke.

The physical examination includes a thorough nasal examination and an evaluation of the eyes, ears, throat, chest, and skin. Physical findings may be subtle. Classic physical findings include pale or bluish, boggy and edematous nasal turbinates with clear, watery secretions. Frequent nasal itching and rubbing of the nose superiorly with the palm of the hand, the **allergic salute**, can lead to a transverse nasal crease found across the nose. Children may produce clucking sounds by rubbing

the soft palate with their tongue. Oropharyngeal examination may reveal lymphoid hyperplasia of the posterior oropharynx, visible mucus, or both. Orthodontic abnormalities may be seen in children with chronic mouth breathing. **Allergic shiners**, dark, edematous infraorbital areas caused by venous congestion, along with eyelid edema or conjunctival injection, are often present in children. Retracted tympanic membranes from eustachian tube dysfunction or serous otitis media also may be seen. Evidence of other atopic diseases, such as asthma or eczema, may be present, which helps lead the clinician to the correct diagnosis.

LABORATORY AND IMAGING STUDIES

Allergy testing can be performed by in vivo skin tests or by in vitro serum tests (enzyme-linked immunosorbent assay [ELISA]) to pertinent allergens found in the patient's environment (see Table 77.4). Skin tests (prick/puncture) provide immediate and accurate results. Positive tests correlate strongly with nasal and bronchial allergen provocative challenges. In vitro serum tests are useful for patients with diffuse dermatitis, dermatographism, poor tolerance to skin testing, or for those taking medications that interfere with skin testing. Disadvantages of serum tests include increased cost, inability to obtain immediate results, and reduced sensitivity compared with skin tests. Testing should include relevant allergens in the home and regional flora. Broad screening testing without regard to symptoms is not recommended. Measurement of total serum IgE or blood eosinophils generally is not helpful. The presence of eosinophils on the nasal smear suggests a diagnosis of allergy, but eosinophils also can be found in patients with **nonallergic rhinitis with eosinophilia**. Nasal smear eosinophilia is often predictive of a good clinical response to nasal corticosteroid sprays but is not commonly performed in clinical practice.

DIFFERENTIAL DIAGNOSIS

Rhinitis can be divided into allergic and nonallergic rhinitis (Table 79.1). Nonallergic rhinitis describes a group of nasal diseases in which there is no evidence of allergic etiology. It can be divided further into nonanatomic and anatomic etiologies. The most common form of nonallergic rhinitis in children is infectious rhinitis, which may be acute or chronic. Acute infectious rhinitis (**the common cold**) is caused by viruses, including rhinoviruses and coronaviruses, and typically resolves within 7–10 days (see Chapter 102). An average child has three to six common colds per year, with the most affected being younger children and children attending day care. Infection is suggested by the presence of sore throat, fever, and poor appetite, especially with a history of exposure to others with colds. Chronic infectious **rhinosinusitis**, or sinusitis, should be suspected if there is mucopurulent nasal discharge with symptoms that persist beyond 10 days (see Chapter 104). Classic signs of acute sinusitis in older children include facial tenderness, tooth pain, headache, and fever. Classic signs are usually not present in young children who may present with postnasal drainage with cough, throat clearing, halitosis, and rhinorrhea. The character of the nasal secretions with infectious rhinitis varies from purulent to minimal or absent. Coexistence of middle ear disease, such as otitis media or eustachian tube dysfunction, may be additional clues of infection.

TABLE 79.1 Classification of the Etiology of Rhinitis in Children

ALLERGIC	NONALLERGIC	
	Nonanatomic	Anatomic
Seasonal	Nonallergic, noninfectious (vasomotor) rhinitis	Adenoidal hypertrophy
Perennial	Infectious rhinosinusitis	CSF rhinorrhea
Episodic	Nonallergic rhinitis with eosinophilia	Choanal atresia
	Rhinitis medicamentosa	Congenital anomalies
		Foreign body
		Nasal polyps
		Septal deviation
		Tumors
		Turbinate hypertrophy

CSF, Cerebrospinal fluid.

Nonallergic, noninfectious rhinitis (formerly known as *vasomotor rhinitis*) can manifest as rhinorrhea and sneezing in children with profuse clear nasal discharge. Exposure to irritants, such as cigarette smoke and dust, and strong fumes and odors, such as perfumes and chlorine in swimming pools, can trigger these nasal symptoms. **Nonallergic rhinitis with eosinophilia syndrome** is associated with clear nasal discharge and eosinophils on nasal smear and is seen infrequently in children. Cold air (**skier's nose**), hot/spicy food ingestion (**gustatory rhinitis**), and exposure to bright light (**reflex rhinitis**) are examples of nonallergic, noninfectious rhinitis. Treatment with topical ipratropium before exposure may be helpful.

Rhinitis medicamentosa is due primarily to overuse of topical nasal decongestants, such as oxymetazoline, or phenylephrine, resulting in rebound nasal congestion. This condition is not common in younger children. Adolescents or young adults may become dependent on these over-the-counter medications. Treatment requires discontinuation of the offending decongestant spray, topical corticosteroids, and frequently a short course of oral corticosteroids.

The most common anatomic problem seen in young children is obstruction secondary to **adenoidal hypertrophy**, which can be suspected from symptoms such as mouth breathing, snoring, hyponasal speech, sleep disordered breathing, and persistent rhinitis with or without chronic otitis media (see [Chapter 135](#)). Infection of the nasopharynx may be secondary to infected hypertrophied adenoid tissue.

Nasal polyps typically appear as bilateral, gray, glistening tissue originating from the sinus ostia and may be associated with anosmia, nasal obstruction, and clear or purulent nasal discharge. Nasal polyps are rare in children younger than 10 years of age, but if present they warrant evaluation for an underlying disease process such as cystic fibrosis or primary ciliary dyskinesia. **Aspirin exacerbated respiratory disease** consists of asthma, aspirin sensitivity, and nasal polyps with chronic or recurrent sinusitis.

Foreign bodies are seen more commonly in young children who place food, small toys, stones, or other objects in their nose. The index of suspicion should be raised by a history of unilateral, purulent nasal discharge or foul odor. The foreign body can often be seen on anterior nasal examination.

TREATMENT

Management of allergic rhinitis is based on disease severity, impact of the disease on the patient, and the ability of the patient to comply with recommendations. Treatment modalities include allergen avoidance, pharmacologic therapy, and immunotherapy. Environmental control and steps to minimize allergen exposure, similar to preventive steps for asthma, should be implemented whenever possible (see [Table 78.3](#)).

Pharmacotherapy

Intranasal corticosteroids are the most effective pharmacologic therapy for treatment of allergic and nonallergic rhinitis. These include beclomethasone, budesonide, ciclesonide, flunisolide, fluticasone, mometasone, and triamcinolone. These topical agents work to reduce inflammation, edema, and mucus production. They are effective for symptoms of nasal congestion, rhinorrhea, pruritus, and sneezing but less helpful for ocular symptoms. Daily administration is necessary for maximum effectiveness. Nasal corticosteroid sprays have been used safely in long-term therapy. Deleterious effects on adrenal function or nasal membranes have not been reported when these agents are used appropriately. The most common adverse effects include local irritation, burning, and sneezing, which occur in 10% of patients. Nasal bleeding from improper technique (spraying the nasal septum) can occur. Rare cases of nasal septal perforation have been reported.

Antihistamines are the medications used most frequently to treat allergic rhinitis. They are useful in treating rhinorrhea, sneezing, nasal itching, and ocular itching but are less helpful in treating nasal congestion. **First-generation antihistamines**, such as diphenhydramine and hydroxyzine, easily cross the blood-brain barrier with sedation as the most common reported adverse effect. Use of first-generation antihistamines in children has an adverse effect on cognitive and academic function. In very young children, a paradoxical stimulatory central nervous system effect resulting in irritability and restlessness has been noted. Other adverse effects of first-generation antihistamines include anticholinergic effects, such as blurred vision, urinary retention, dry mouth, tachycardia, and constipation. **Second-generation antihistamines**,

such as cetirizine, desloratadine, fexofenadine, levocetirizine, and loratadine, are less likely to cross the blood-brain barrier resulting in less sedation. Cetirizine, fexofenadine, levocetirizine, and loratadine are available without prescription. Azelastine and olopatadine, topical nasal antihistamine sprays, are approved for children older than 5 years and 6 years, respectively.

Decongestants, taken orally or intranasally, may be used to relieve nasal congestion. Oral medications, such as pseudoephedrine and phenylephrine, are available either alone or in combination with antihistamines. Adverse effects of oral decongestants include insomnia, nervousness, irritability, tachycardia, tremors, and palpitations; oral decongestants are not recommended for children less than 4 years old. For older children participating in sports, oral decongestant use may be restricted. Topical nasal decongestant sprays are effective for immediate relief of nasal obstruction but should not be used more than 3 consecutive days to prevent rhinitis medicamentosa.

Topical ipratropium bromide, an anticholinergic nasal spray, is used primarily for nonallergic rhinitis and rhinitis associated with viral upper respiratory infection. Leukotriene modifiers have been studied in the treatment of allergic rhinitis. Montelukast is approved for use in seasonal allergic rhinitis.

Allergen Immunotherapy

If environmental control measures and medication intervention are only partially effective or produce unacceptable adverse effects, immunotherapy may be recommended. The mechanism of action for allergen immunotherapy is complex but includes increased production of an IgG-blocking antibody, decreased production of specific IgE, and alteration of cytokine expression produced in response to an allergen challenge. Immunotherapy is effective for desensitization to pollens, dust mites, and cat and dog proteins. Use in young children may be limited by the need for frequent injections. *Subcutaneous immunotherapy* (injections or “allergy shots”) must be administered in a physician’s office with 30 min of observation after the allergen injection. Anaphylaxis may occur, and the physician must be experienced in the treatment of these severe adverse allergic reactions. *Sublingual immunotherapy* (tablets placed under the tongue) is only indicated for ragweed pollen, some grass pollens, and dust mite allergy. It can be used in children 10 years or older. The first dose must be given in a physician’s office, but subsequent doses can be administered at home.

COMPLICATIONS

Approximately 60% of children with allergic rhinitis have symptoms of bronchial hyperresponsiveness (see [Chapter 78](#)). Chronic allergic inflammation leads to chronic cough from postnasal drip, eustachian tube dysfunction and otitis media, sinusitis, and tonsillar and adenoid hypertrophy, which may lead to obstructive sleep apnea. Children with allergic rhinitis may experience sleep disturbances, limitations of activity, irritability, and mood and cognitive disorders that adversely affect their performance at school and their sense of well-being.

PROGNOSIS AND PREVENTION

Seasonal allergic rhinitis is a common and prominent condition that may not improve as children grow older. Patients become more adept at self-management. Perennial allergic rhinitis improves with allergen control of indoor allergens.

Removal or avoidance of the offending allergen is advised. The only effective measure for minimizing pet allergen exposure is removal of the pet from the home. Avoidance of pollen and outdoor molds can be accomplished by staying indoors in a controlled environment. Air conditioning and keeping windows and doors closed lower exposure to pollen. High-efficiency particle air filters reduce exposure to allergens (e.g., pet dander). Sealing the mattress and pillow in allergen-proof encasings is the most effective strategy for reduction of dust mite allergen. Bedding should be washed in hot water (>130 °F) every week.

PEARLS FOR PRACTITIONERS

See *Allergy: Pearls for Practitioners* at the end of this section.