

**Asthma**

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These guidelines should not be construed as including all proper methods of care or excluding other acceptable methods of care reasonably directed to obtaining the same results. The ultimate judgment regarding any specific clinical procedure or treatment must be made by the physician in light of the circumstances presented by the patient.

**Patient population:** Pediatric and adult.

**Problems:** Asthma is under-recognized and under-treated. Patients are often not taught to recognize and treat exacerbations. Management relies too much on “rescue” therapy with insufficient emphasis on chronic suppressive therapy. Inhaled corticosteroids are underutilized.

**Objectives:** Improve the patient's quality of life by achieving and maintaining control of symptoms; attaining normal lung function; minimizing need for as-needed  $\beta_2$ -agonists; avoiding adverse effects from asthma medications; preventing exacerbations; attaining normal activity levels, including exercise; and preventing emergency visits and hospitalizations.

**Key Points**

- **A high index of suspicion** for asthma is essential. A history of both symptoms and symptom triggers should be obtained. [*evidence: C\**]
- **Objective evaluation of airflow obstruction** is key to the diagnosis, classification, and management of the disease. Goals of treatment should include not only symptomatic relief, but normalization of lung function [*C\**].
- **Therapy should focus on long-term suppressive therapy.** Anti-inflammatory agents (in particular inhaled corticosteroids) are the cornerstone of therapy for moderately and severely affected patients. Inhaled  $\beta_2$ -agonists should represent “rescue” agents in most instances [*B\**].
- **Patient education** should emphasize how to identify and avoid environmental triggers of asthma and smoking cessation. Patients with moderate or severe asthma should be able to measure their peak expiratory flow rate (PEFR) at home and modify their therapy or seek help based on their performance relative to their personal best peak flow value. Self-management is fundamental to successful therapy [*A\**], so a structured asthma education program should be considered.

\* **Levels of evidence for the most significant recommendations**

A = randomized controlled trials; B = controlled trials, no randomization; C = decision analysis; D = opinion of expert panel

**Clinical Background**

**Clinical Problem  
and Management Issues**

- Asthma is under-diagnosed. Poor perception of asthma severity delays treatment.
- Asthma morbidity and mortality are increasing. African-Americans have asthma-related mortality rates that are higher than Caucasians’ rates.
- Asthma care, both by patients and by physicians, still relies on symptoms and perception of symptoms. There is a relative lack of the use of objective data to guide care.
- Because airway inflammation is now proposed as a principal factor in airway hyperresponsiveness, therapeutic agents to prevent or reverse this abnormality are considered first-line therapy.

**Rationale for Recommendations**

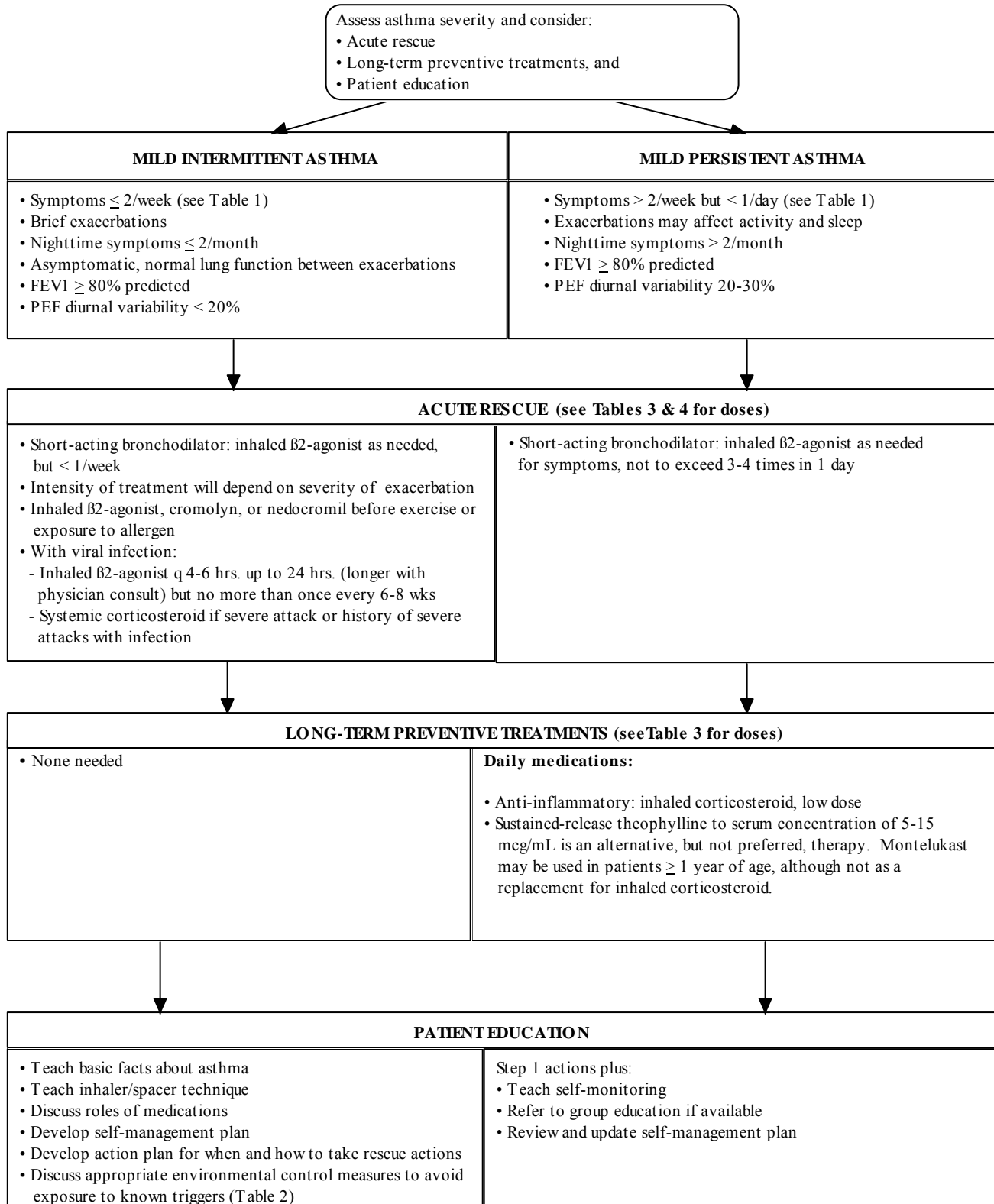
**Diagnosis of Asthma**

To establish a diagnosis of asthma, the clinician must determine that:  
(1) episodic symptoms of airway obstruction are present; (2) airflow obstruction is present and is at least partially reversible; and (3) alternative diagnoses are excluded. Initial assessment may be normal (e.g., no airflow obstruction) especially with episodic disease. When the diagnosis of asthma is not easily made, consider longitudinal assessment of diurnal airflow variation, bronchoprovocation testing (methacholine challenge test), or referral to a specialist.

(continued on page 5)

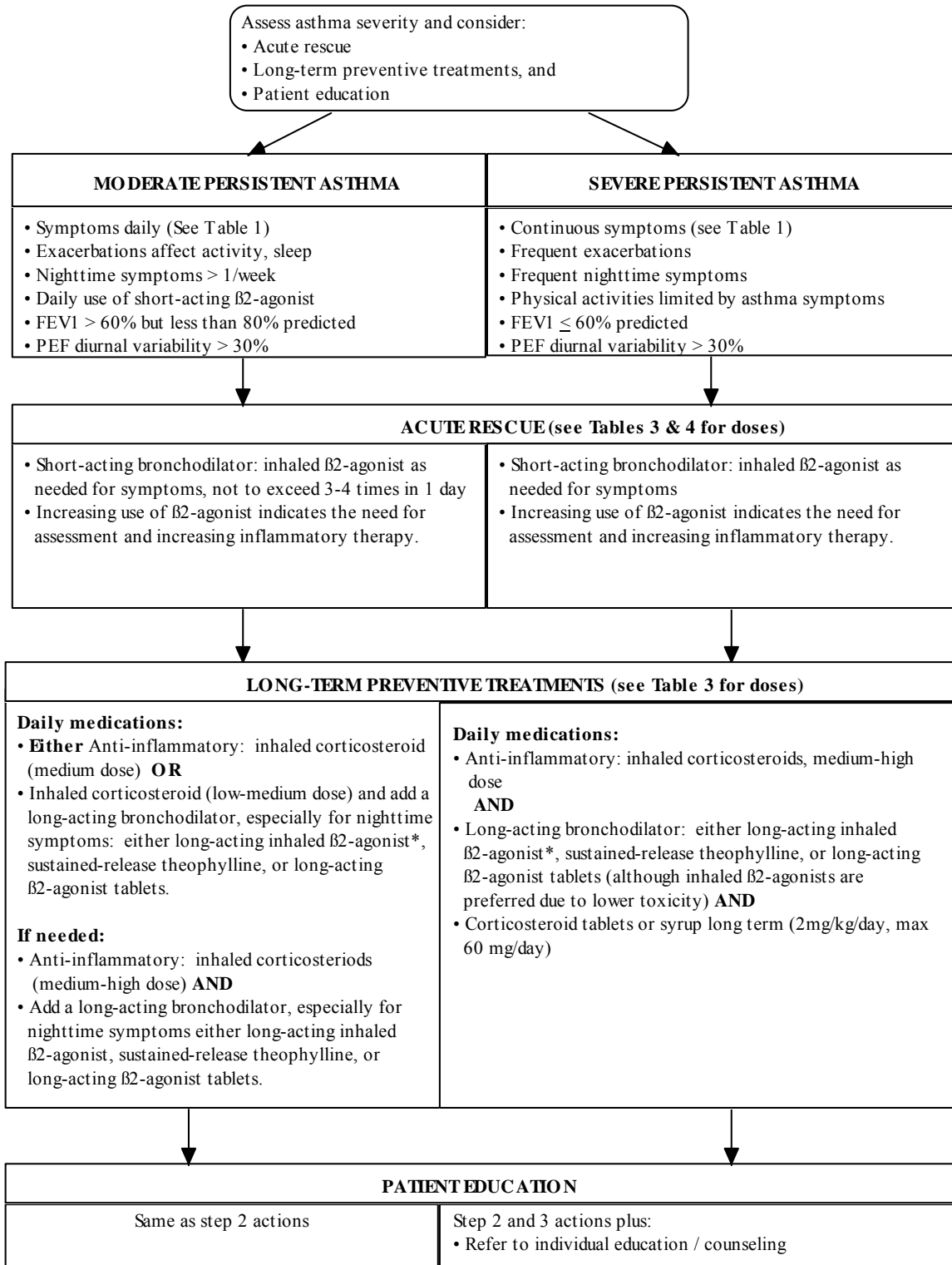
## Figure 1. Management of Asthma

(Adapted from the National Heart, Lung, and Blood Institute: Guidelines for the Diagnosis and Management of Asthma, July 1997)



## Figure 1. Management of Asthma, continued

(Adapted from the National Heart, Lung, and Blood Institute: Guidelines for the Diagnosis and Management of Asthma, July 1997)



\* As of 11/18/05, the US Food and Drug Administration (FDA) required manufacturers of products containing long-acting beta agonists (AdvairDiskus, Foradil Aerolizer, and Serevent Diskus) to add warnings to their product labels and to distribute a Medication Guide for patients regarding the risk that long-acting beta agonists may increase the chance of severe asthma episodes and asthma-related deaths.

**Table 1. Symptoms Suggestive of Asthma**

<ul style="list-style-type: none"> <li>• Cough</li> <li>• An attack, or recurrent attacks, of wheezing</li> <li>• Shortness of breath or chest tightness</li> <li>• Nocturnal cough</li> <li>• Exercise-induced cough or wheezing</li> <li>• Onset of symptoms after exposure to airborne allergens or other stimuli</li> <li>• History of persistent respiratory tract infections</li> <li>• Conditions associated with asthma (e.g., nasal polyyps, rhinitis, atopic dermatitis, etc.)</li> </ul>
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**Table 2. Common Asthma Triggers**

<ul style="list-style-type: none"> <li>• Smoking (or passive smoking)</li> <li>• Indoor allergens:             <ul style="list-style-type: none"> <li>House dust mites</li> <li>Animal dander</li> <li>Cockroaches</li> <li>Fungi</li> </ul> </li> <li>• Outdoor allergens:             <ul style="list-style-type: none"> <li>Pollens</li> <li>Fungi</li> </ul> </li> </ul>	<ul style="list-style-type: none"> <li>• Pollutants:             <ul style="list-style-type: none"> <li>Air pollutants</li> <li>Occupational exposures</li> </ul> </li> <li>• Medications:             <ul style="list-style-type: none"> <li>β-blockers</li> <li>Aspirin</li> <li>NSAIDs</li> </ul> </li> <li>• Exercise</li> <li>• Cold air</li> </ul>	<ul style="list-style-type: none"> <li>• Respiratory tract infections:             <ul style="list-style-type: none"> <li>Viral URI illnesses</li> <li>Sinusitis</li> <li>Bronchitis</li> </ul> </li> <li>• Gastroesophageal reflux</li> <li>• Sulfites</li> </ul>
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**Table 3. Pharmacologic Therapy for Chronic Asthma**

Generic Name	Brand Name	Usual Adult Dose	Usual Pediatric Dose	# Days canister will last (lowest-highest)	Cost for 30 day supply* (lowest-highest)		
<b>INHALERS</b>							
<i>Corticosteroids**</i>							
Beclomethasone (MDI) 40 or 80 mcg/puff	Qvar (HFA)	Low Dose	80-240	Low Dose	80-160	6-50 days (40 mcg) 12-50 days (80 mcg)	\$51-256 \$65-194
		Medium	240-480	Medium	160-320		
		High	>480	High	>320		
Budesonide (DPI)*** 200 mcg/inhalation	Pulmicort Turbuhaler	Low Dose	200-600	Low Dose	200-400	25-100 days	\$47-155
		Medium	600-1200	Medium	400-800		
		High	>1200	High	>800		
		Divided bid		Divided bid			
Budesonide (Nebulizer solution)*** 0.25 or 0.5 mg	Pulmicort Respules			Low Dose	0.5		\$279-302
				Medium	0.5		
				High	1.0		
				Divided bid			
Triamcinolone (MDI) 100 mcg/puff	Azmecort	Low Dose	400-1000	Low Dose	400-800	15-80 days	\$32-174
		Medium	1000-2000	Medium	800-1200		
		High	>2000	High	>1200		
		Divided bid-qid		Divided bid-qid			
Flunisolide (MDI) 250 mcg/puff	Aerobid	Low Dose	500-1000	Low Dose	500-750	12.5-25 days	\$87-182
		Medium	1000-2000	Medium	1000-1250		
		High	>2000	High	>1250		
		Divided bid		Divided bid			
Fluticasone (MDI)*** 44, 110 or 220 mcg/puff	Flovent	Low Dose	88-264	Low Dose	88-176	12-30 days (44 mcg)	\$44-129
		Medium	264-660	Medium	176-440		
		High	>660	High	>440	20-60 days (110 mcg) 60 days (220 mcg)	\$64-133
		Divided bid		Divided bid			
Salmeterol/fluticasone (DPI)	Advair Diskus	Low Dose	100/50 1 puff bid	Low Dose	1 puff bid	30 days	\$120
		Medium	250/50 1 puff bid	Medium	1 puff bid	30 days	\$152
		High	500/50 1 puff bid	High	1 puff bid	30 days	\$210

Table continues on next page

MDI= Metered Dose Inhaler; DPI = Dry Powder Inhaler

\* AWP = Average Wholesale Price. For brand drugs, Average Wholesale Price minus 10%. AWP from Amerisource Bergen Wholesale Catalog 8/1/044/13/05. For generic drugs, Maximum Allowable Cost plus \$3 from BCBS of Michigan MAC List, 5/25/05.

\*\* High doses of inhaled steroids may have significant side effects; see text for discussion.

\*\*\* Once-per-day dosing is an option only for patients whose asthma is controlled by oral corticosteroids may be considered

**Table 3. Pharmacologic Therapy for Chronic Asthma, continued**

Generic Name	Brand Name	Usual Adult Dose	Usual Pediatric Dose	# Days canister will last (lowest-highest)	Cost for 30 day supply (lowest-highest)
<b>Mast Cell Stabilizers</b>					
Cromolyn sodium (MDI)	Intal	2-4 puffs tid-qid (800 mcg/puff)	1-2 puffs tid-qid (800 mcg/puff)	7-56 days	\$39-230
Nedocromil sodium (MDI)	Tilade	2-4 puffs bid-qid (1750 mcg/puff)	1-2 puffs bid-qid (1750 mcg/puff)	7-56 days	\$37-301
<b>β<sub>2</sub>-adrenergic agonists</b>					
Albuterol (MDI)	Proventil	2 puffs tid-qid (90 mcg/puff)	2 puffs tid-qid (90 mcg/puff)	25-30 days	\$11 (generic) \$39 (brand)
	Proventil HFA	2 puffs tid-qid (90 mcg/puff)	2 puffs tid-qid (90 mcg/puff)	25-30 days	\$39
	Ventolin HFA	2 puffs tid-qid (90 mcg/puff)	2 puffs tid-qid (90 mcg/puff)	25-30 days	\$34
Pirbuterol (MDI)	Maxair Autohaler	2 puffs tid-qid (200 mcg/puff)	2 puffs tid-qid (200 mcg/puff)	37-50 days	\$37-50
<b>Long-Acting β-agonist*</b> (maintenance only, not acute use)					
Salmeterol (DPI)	Serevent Diskus	1 blister bid (50 mcg/dose)	1 blister bid (50 mcg/dose)	30 days	\$94
Fomoterol (DPI)	Foradil Aerolizer	1 capsule bid (12 mcg/dose)	1 capsule bid (12 mcg/dose)		\$91
<b>TABLETS</b>					
<b>Methyxanthine</b>					
Theophylline	Several long-acting forms (e.g., Theo-24)	300 mg bid	Starting dose 10mg/kg/day; usual max: ≥1 year of age: 16 mg/kg/day < 1 yr: 0.2 (age in weeks) + 5 = mg/kg/day	NA	\$24-48
<b>Leukotriene Modifiers</b>					
Montelukast	Singulair	10 mg hs	5 mg hs age 6-14 4 mg hs age 2-5 4 mg hs (oral granules) age 12-23 mo	NA	\$89
Zafirlukast	Accolate	20 mg bid	10 mg bid	NA	\$101
<b>Oral Steroids**</b>					
Prednisone: 1, 2.5, 5, 10, 20, 25 mg tabs; 1 mg/ml liquid		7.5-60 mg/day in a single or divided dose as needed for control	0.25-2mg/kg/day in single or divided dose as needed for control	NA	\$4-12 (generic)
Prednisolone: 5 mg tabs; 1mg/ml, 3 mg/ml liquid		short-course burst with taper, for example:	short course burst with taper: 1-2mg/kg/day, max 60 mg/day, for 3-10 days		generic liquid \$11/2 oz OraPred liquid \$31/2 oz
Methylprednisolone: 2, 4, 8, 16, 32 mg tablets		<u>Day</u> <u>Dose (mg)</u> 1 60 2 50 3 40 4 30 5 20 6 10 7 off			\$20(generic) \$52 (brand)

\* As of 11/18/05, the US Food and Drug Administration (FDA) required manufacturers of products containing long-acting beta agonists (Advair Diskus, Foradil Aerolizer, and Serevent Diskus) to add warnings to their product labels and to distribute a Medication Guide for patients regarding the risk that long-acting beta agonists may increase the chance of severe asthma episodes and asthma-related deaths.

\*\* If patients are started on inhaled corticosteroids, it may be necessary to taper the systemic corticosteroid dose depending on dose and duration of therapy. Ora Pred (3 mg/ml) brand of liquid prednisolone preferred for children due to taste

**Table 4. Dosages of Drugs in Acute Exacerbations of Asthma in Children**

DRUG & DOSAGE FORM		DOSE	COMMENT
Albuterol <i>MDI</i>	90mcg/puff	4-8 puffs every 20 min for 3 doses, then every 1-4 hrs as needed	If not improved, switch to nebulizer.
<i>Nebulizer solution</i>	0.5% (5mg/ml)	0.15mg/kg (minimum dose 2.5 mg) every 20 min for 3 doses, then 0.15-0.3 mg/kg up to 10 mg every 1-4 hrs as needed, or 0.5mg/kg/hr by continuous nebulization.	If improved, decrease to 1-2 hours. If not improved, use by continuous inhalation.  For optimal delivery, dilute aerosols to minimum of 4ml at gas flow of 6-8 L/min.
Levalbuterol Nebulizer solution	0.63 mg/3 ml 1.25 mg/3 ml	0.63-1.25 mg every 20 min for 3 doses, then increase interval to 1-4 hours as needed	No clinically significant advantage; albuterol should be used first
Epinephrine HCl <i>Systemic</i>	1:1000 (1 mg/ml)	SQ: 0.01 mg/kg up to 0.3-0.5 mg every 20 minutes for 3 doses	Inhaled $\beta_2$ - agonist preferred. No proven advantage of systemic therapy over aerosol.
Ipratropium Bromide <i>Nebulizer Solution</i>	0.25mg/ml	0.25 mg every 20 min. for 3 doses, then every 2-4 hrs.	Consider when response to initial albuterol nebulizer treatment is poor. Should not be used as first-line therapy; should be added to $\beta_2$ -agonist therapy.  May mix in same nebulizer with albuterol.
Corticosteroids		<b>Outpatient:</b> Short course "Burst" therapy: Use 1-2 mg/kg/day, maximum 60 mg/day for 3-10 days.  <b>Emergency Department or hospitalized patients:</b> 1 mg/kg every 6 hrs for 48 hrs then 1-2 mg/kg/day (max 60 mg/day) in 2 divided doses until PEF 70% of predicted or personal best.	Length depends on response. May need only a few days.  Use Ora Pred brand (3 mg/ml) of oral prednisolone for children due to taste  If patients are started on inhaled corticosteroids, it may be necessary to taper the systemic corticosteroid dose.  No advantage has been found for IV administration over oral therapy provided GI transit time or absorption is not impaired.
Prednisone	1, 2.5, 5, 10, 20, 25 mg tabs; 1mg/ml		
Prednisolone	1 mg/ml, 3 mg/ml		
Methylprednisolone	2, 4, 8, 16, 32 mg tablets 40mg, 125mg, 500mg, 1 gm, 2 gm per vial, powder for injection		

MDI = Metered Dose Inhaler

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## Rationale for Recommendations (continued)

**Symptoms.** Common symptoms of asthma are cough, wheezing, shortness of breath, and chest tightness (Table 1). These symptoms are not in themselves diagnostic. Recurrent symptoms, especially if provoked by exogenous factors, are very suggestive of asthma.

Isolated cough without clear etiology (e.g., acute viral infection) should be considered asthma until proven otherwise, especially in young patients and non-smokers.

While many children develop asthma in the first year of life, only 30% of infant wheezers persist with wheezing into later childhood. Early age of onset of wheezing is not clearly related to persistent asthma, but most asthmatic children do manifest symptoms at an early age. The pathophysiologic mechanisms of “wheezy bronchitis” in infancy may be different from those that produce asthma. Unfortunately, there is no way to distinguish the two entities on clinical grounds. For the purpose of establishing guidelines for therapy, the infant who has 3 or more episodes of wheezing, regardless of the trigger, should be considered to have asthma. History of maternal asthma and /or the presence of atopic dermatitis are strong predictors of asthma in the symptomatic young child.

**Signs.** Conditions associated with asthma (e.g., rhinitis, atopic dermatitis, etc.) may suggest the diagnosis. Nasal polyps are more predictive of cystic fibrosis than asthma in children, but among adults can suggest asthma. The patient experiencing an acute attack of asthma may exhibit wheezing, intercostal retraction during inspiration, chest hyperinflation, and prolonged expiratory phase. Because asthma is characteristically episodic, the physical examination may be completely normal even when the patient is symptomatic. Asthma patients frequently have poor recognition of symptoms and poor perception of the severity of the disease. Thus, objective measures of airflow obstruction and its variability are critical in establishing a diagnosis.

**Objective measures of airflow obstruction.** Spirometry measurements (FEV<sub>1</sub>, FVC, FEV<sub>1</sub>/FVC ratio before and after the patient inhales a short-acting bronchodilator) can document airway obstruction, demonstrate reversibility, and stage the severity of disease. Partially reversible airflow obstruction is defined as a 12% increase in FEV<sub>1</sub> after bronchodilator.

Asthma patients’ subjective symptom reports have been shown to correlate poorly with measured severity of obstruction. For this reason, an NIH expert panel recommends spirometry for diagnosing and grading the severity of asthma [D\*]. Caffeine-containing beverages may affect spirometry results and should be avoided for 4 hours before testing.

Measurements by peak flow meter in physicians’ offices should not be used to diagnose air flow obstruction because there is wide variability even in the best published reference values.

**Exclusion of alternative diagnoses.** Wheezing does not necessarily mean asthma. In young children, wheezing may be associated with the following: congenital and acquired extramural lesions, including bronchogenic cysts, tumors, (very rarely), vascular rings, vascular slings, and lymphadenopathy; intramural lesions, including tracheomalacia and bronchomalacia, tracheostenosis and bronchostenosis, and bronchiolitis; and intraluminal lesions, including tracheal web, cystic fibrosis, and foreign body aspiration. Intraluminal lesions, such as foreign body aspiration and tracheal webs should be considered. Cystic fibrosis may present as chronic cough or intractable wheezing. In adults, wheezing may be associated with upper respiratory infection, heart failure, acute bronchiolitis, foreign body aspiration, upper airway obstruction (functional or organic), and other conditions. In instances when the diagnosis is in doubt or specialized testing is required, referral to a specialist in asthma care may be appropriate (see general guidelines for referral under step 6).

## Asthma Management

For effective therapy, a six-part asthma management program is recommended.

### Step 1. Educate patients to develop a partnership in asthma management

Research shows that asthma education can be cost-effective and can reduce morbidity. From the time of diagnosis, the clinician should begin to build a partnership with the patient and family. Teaching asthma self-management and encouraging active participation should be integrated at every step of medical care. It is essential that the clinician demonstrate, review, evaluate, and correct inhaler/spacer technique. Current recommendations are for patients to have both a written plan for daily self-management and a written action plan for management of acute exacerbations (either symptomatic or revealed by deteriorating peak flows). The patient will have a great deal to learn to master self-management, and the services of a dedicated asthma educator may be valuable. Severe asthma patients (especially children) benefit from comprehensive education programs [A\*].

### Step 2. Assess asthma severity with objective measures of lung function

Several studies have shown a poor correlation between patient-reported symptoms of asthma and actual airflow obstruction. Consequently, to help guide management, several consensus committees have provided an asthma severity stratification based on symptoms and airflow obstruction (Figure 1). Objective evaluation of airflow obstruction should be utilized to monitor patient progress.

**Peak Expiratory Flow Rate (PEFR) monitoring.** Peak flow meters are designed as monitoring tools, not diagnostic tools. PEFR monitoring is a simple quantitative method for

detecting asymptotic deteriorations, monitoring severity, assessing response to therapy, establishing diurnal variation, diagnosing exercise-induced asthma, and identifying triggers. PEFR monitoring also provides objective information to assist in planning, starting, or stopping treatment and can be used by most adults as well as children as young as 5 years of age. Peak flow effort, and hence results, are less consistent in children under age 8. Spirometry can be used for monitoring response to therapy, as well as diagnosing asthma. It should be considered where doubt about peak flow reliability exists.

Clinicians should consider home peak flow monitoring in patients over 5 years of age who have: (1) moderate or severe asthma, (2) variable disease, or (3) a poor perception of the severity of their asthma. Long-term daily peak flow measurement in managing mild or intermittent asthma does not appear to affect outcomes significantly. For patients and families who have been well trained in the method, routine structured symptom assessment has been demonstrated to be as effective as peak flow measurement in monitoring severity.

Patients must be educated in using their peak flow meters. PEFR measurement is effort dependent, so patients need to be coached initially to give their best effort. Proper technique is simple, and nose clips are not required. The instrument pointer should be reset to baseline. The patient should stand up, take a deep breath, place the meter in the mouth with lips closed tightly around the mouthpiece, and blow out as hard and fast as possible. It is not necessary to blow out all the way as peak flow is obtained in the first portion of exhalation. Repeat the process two more times and record the best effort.

Patients should use the same peak flow meter over time, as different brands of meters can give different values. It is important for every patient to establish a personal best PEFR value and use that value as the standard against which future measurements will be evaluated. A personal best value is obtained by measuring twice daily peak flow over a 2-3 week period. If the patient takes a bronchodilator, the PEFR should be measured before and after using the bronchodilator. The personal best value is usually the highest PEFR measurement achieved in the afternoon/evening measurement after a period of maximum therapy. In children, the personal best value should be re-evaluated yearly to account for growth. Nomograms of normal values are available (appendix), but the most clinically useful standard for ongoing monitoring is the patient's personal best peak flow value.

### **Step 3. Avoid or control asthma triggers**

Asthma triggers can either induce airway inflammation or precipitate acute bronchospasm (Table 2). Efforts to identify and control patient-specific triggers should be made. Reducing exposure to allergens is also important in controlling asthma. Desensitization injections (allergy shots) are not indicated for most patients, but they may have a place in the management of a subset of selected patients with extrinsic (allergic) asthma.

Some patients with asthma continue to smoke. Smoking cessation for these patients is a critical first step in reducing inflammation. Cessation of smoking should be strongly advised for parents of children with asthma.

A high percentage of children with asthma are atopic, and aeroallergen exposure and sensitivity contribute significantly to the development and persistence of the asthma. The best therapy for allergen-induced asthma is avoidance, but success with allergen avoidance is predicated on accurate identification of the offending allergens. Skin testing correlates with bronchial allergen challenge and can, in many instances, identify controllable environmental allergens.

**Indoor allergens.** Because a large portion of any 24-hour period is spent in the bedroom, the most important continuous source of indoor allergen exposure comes from this room. The workplace is another important source of indoor allergen exposure.

House dust mites are microscopic animals that live in mattresses, bedding, furniture, and carpets. They thrive in high humidity. Upholstered furniture and wall-to-wall carpeting may increase dust-mite exposure; for many years, down pillows and comforters were also thought to harbor dust mites, but recent evidence demonstrate that this is not the case [B\*]. Most of the house dust mite allergen is found in the mite's fecal particles. Effective mite control measures include washing (every 1-2 weeks) bedding materials in hot water to denature mite allergens. Encasing the mattress, pillows and box spring reduces mite allergen levels. Additional control measures include steam cleaning, having non-carpeted floors, and reducing humidity to less than <50% [A\*]. Treating carpets and furniture with acaricides (to kill dust mites) or tannic acid (to denature dust mite allergen) have not been well studied clinically. Mite-allergic individuals should avoid the environment being cleaned or vacuumed as this causes allergens to become airborne. Individualized house dust mite control measures among atopic asthmatic children have reduced asthma associated morbidity in several randomized controlled trials, but allergen-impermeable encasements as a single intervention for avoidance of exposure to dust-mites was clinically ineffective in a study of adult asthmatics.

All warm-blooded pets--including cats, dogs, rodents, and birds--produce allergens that can trigger asthma. Removal of animals from the patient's environment is extremely important although perceived benefit may not be immediate because animal allergens may linger for months after animal removal. There are mixed data as to the efficacy of regular washing of the allergenic pet. This should never be attempted by the allergic patient him/herself.

In the workplace, there are numerous agents that have been identified as occupational allergens and irritants that can precipitate asthma. Once the worker is sensitized to a particular agent, the level of agent necessary to induce symptoms may be very low. PEFR monitoring on and off the job and Material Safety Data Sheets can help identify occupational triggers. Attempts to reduce occupational

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trigger exposure have been successful in a number of industrial settings.

**Outdoor allergens.** Outdoor allergens (pollens and molds) are common triggers and impossible to avoid completely. Exposure may be reduced by closing windows and doors and by using air-conditioning and filtering devices, especially during peak pollen and mold seasons. Seasonal symptoms may indicate pollen sensitivity (in Michigan: trees in March, April and May; grasses in late May and June; ragweed and sage in late August and September).

**Food triggers.** Foods are extremely rare asthma triggers; however, anaphylaxis due to food allergy is a potentially life-threatening condition. Strict avoidance of the food trigger and education in the proper self-administration of epinephrine for inadvertent food exposures is mandatory. The only food additive that has been substantiated as an asthma trigger is sulfites, which are common food and drug preservatives found in processed potatoes, shrimp, dried fruits, beer and wine. The ability of any other food additive to trigger asthma has not been well documented using controlled challenges.

**Indoor air pollution.** Tobacco smoke, smoke from wood stoves or heating, aerosols, household sprays, volatile organic compounds, strong odors and scents, and air pollutants can trigger asthma in a nonspecific manner. In a randomized study of schools using unflued gas heating in winter, either switching to flued gas or electric heaters to reduce NO<sub>2</sub> reduced student asthma symptoms and daytime asthma attacks. Smoking has been shown to impair the short term response to both systemic and inhaled corticosteroids.

**Medications.** In certain patients aspirin and nonsteroidal anti-inflammatory drugs (NSAIDs) can cause severe exacerbations, but may be useful for those without contraindications. (COX2 inhibitors do not trigger asthma in NSAID induced asthmatic patients.) Many clinicians still think of  $\beta$ -blockers as contraindicated in asthma patients, and these drugs do indeed produce exacerbations in some such patients. However,  $\beta$ -blockers provide substantial survival benefit among patients with coronary artery disease (CAD), especially those who have sustained a myocardial infarction. Current guidelines from NHLBI and ACC/AHA emphasize that this lifesaving therapy be withheld only for true absolute contraindications. Asthma should therefore not be regarded as grounds for withholding a trial of  $\beta$ -blocker therapy in patients with CAD.  $\beta$ -blocker therapy should be initiated under supervision for asthma patients, preferably with a short-acting agent for the first dose. Patients with CAD and asthma who respond with an exacerbation should have that adverse effect documented as a reason for withholding  $\beta$ -blocker therapy; others should continue on  $\beta$ -blockers.

**Exercise** Exercise-induced asthma is an expression of airway hyper-responsiveness and may indicate that asthma is not adequately controlled. Anti-inflammatory therapy often results in the disappearance of exercise-induced symptoms. The inhalation of a rapid-acting  $\beta$ 2-agonist 10

to 20 minutes before exercise is often the most effective means of preventing exercise-induced asthma exacerbations. Cromolyn inhalers and oral montelukast are also of demonstrated efficacy for exercise-induced asthma. Training and sufficient warm up also reduce the incidence and severity of exercise-induced asthma.

**Concurrent medical conditions** Concurrent medical conditions that can exacerbate asthma include infections (e.g., viral upper respiratory infections, bronchitis, sinusitis), allergic rhinitis, and gastroesophageal reflux disease.

#### **Step 4: Establish medication plans for chronic management (Table 3)**

Asthma medications are categorized into two general classes: anti-inflammatory and bronchodilator. Patients with persistent asthma require both; medication plans must accommodate the fact that asthma is both a chronic and a dynamic condition. A step-wise approach to pharmacologic therapy accomplishes this. Whatever medication is used, a poor or short-lasting response to treatment mandates immediate medical care.

**Anti-inflammatory medications.** Corticosteroids are the most potent and consistently effective anti-inflammatory treatment for asthma.

Inhaled corticosteroids are used in long-term control of asthma. The National Asthma Education Program consensus statement endorses the use of inhaled corticosteroids for all moderate asthmatics (i.e., daily bronchodilator use or abnormal pulmonary function tests). Since inflammation plays a central role in asthma, many clinicians find that the added control achieved with chronic inhaled corticosteroids allows inhaled  $\beta$ 2-agonists to be used on a PRN basis. Increasing reliance on inhaled  $\beta$ 2-agonists is a marker of worsening disease or an impending severe attack and should trigger an overall reassessment and possible alteration in therapy, usually involving increased efforts to control airway inflammation.

There is a dose-dependent reduction of short-term growth with the use of conventional doses of beclomethasone dipropionate. Long-term linear growth does not appear to be affected by moderate doses (400-800 mcg/day) of inhaled corticosteroids, except in prepubertal males. Little information exists on the dose-response relationship of triamcinolone acetonide and flunisolide on growth velocity. Little information is available on the long-term growth effects of inhaled corticosteroids at higher dosage regimens (>800 mcg/d) or in babies and young children at any dosage regimen. Inhaled corticosteroids in doses as high as 800 mcg/d exert much less short-term growth suppression than low-dose oral corticosteroids. Serial growth monitoring is advised in these situations. Studies of markers of bone deposition and resorption, and of bone mineral density, suggest that osteopenia is a concern with inhaled corticosteroids in the adult population and increases with dose and duration of use. Patients who need to take high doses of inhaled corticosteroids long-term may need

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prophylactic measures to prevent osteoporosis. There also appears to be some increase in risk of cataracts with long-term use of inhaled steroids.

High doses of inhaled corticosteroids may cause systemic side effects (though to a much lesser extent than oral steroids will). Risk of side effects with high-dose inhaled steroids can be minimized by having the patient rinse his/her mouth immediately after inhalation and before swallowing, and by always using a spacer device.

Dry powder inhalers are now available for select steroids (Pulmicort Turbohaler and Advair: Flovent/Serevent combination) in addition to traditional metered dose inhalers. Advantages of these devices include easier administration of doses and lack of chlorofluorocarbons. No coordination is necessary between device activation and inhalation; however, a minimal inspiratory flow rate is needed to activate the device, which may make usage difficult during exacerbations. The relative ease of use with these devices may make them more suitable for children  $\geq 5$  years of age and the elderly, who may experience difficulty with the MDI. The combination fluticasone/salmeterol product (Advair) allows simplification of the medication regimen which may be especially useful for patients with compliance problems, such as adolescents.

Systemic corticosteroids are often required to mitigate exacerbations in patients with moderate to severe asthma and are used in long-term therapy to gain control of the disease. In asthma, their mechanism is not entirely established but includes: interference with arachidonic acid metabolism and the synthesis of leukotrienes; prevention of the directed migration and activation of inflammatory cells; and increased responsiveness of  $\beta$ -receptors of the airway smooth muscle. No consensus exists on the specific type, dose, or duration of corticosteroid to be used in the treatment of asthma. Systemic steroid pulses with rapid taper are generally used for acute exacerbations. If chronic long-term therapy is required, alternate day administration of oral corticosteroids is preferable to daily treatment. In most cases, it is strongly recommended that inhaled corticosteroids be added to the regimen when oral corticosteroids are started to reduce or eliminate chronic long term oral therapy. Use of systemic corticosteroids within 1 hour of presentation of adults and children with acute asthma to the emergency department has been shown to significantly reduce hospital admission. Benefits were greatest in patients with more severe asthma and those not currently receiving steroids.

Chronic systemic corticosteroid therapy may be associated with obesity, moon facies, supraclavicular and nuchal fat pads, striae, easy bruisability, weakness, hypertension, osteopenia, and glucose intolerance. Children may also exhibit growth failure. Long-term ( $>2$  weeks) corticosteroid therapy may cause suppression of the hypothalamic-pituitary-adrenal axis. Full recovery of the axis can take up to 12 months depending on the dose, frequency, and duration of antecedent therapy. Symptoms and signs of secondary adrenal insufficiency include weakness, weight loss, and gastrointestinal discomfort.

Adrenal insufficiency can evolve into acute adrenal crisis precipitated by severe infection, trauma, or surgery. Clinical presentation includes fever, dehydration, hypotension, nausea, vomiting, and hypoglycemia. Systemic corticosteroid therapy can cause osteopenia.

Leukotriene receptor antagonists. The currently available data suggest that monotherapy with leukotriene modifier agents is more likely to be successful for prophylaxis in mild persistent asthma and exercise-induced asthma. Leukotriene modifiers have not been studied as monotherapy for severe persistent asthma. Leukotriene modifiers have been used as adjuvants to inhaled corticosteroids in severe persistent asthma, though not studied extensively for this purpose.

There are two leukotriene modifier agents approved by the FDA currently available for the treatment of chronic asthma. Zafirlukast (Accolate) and montelukast (Singulair) are leukotriene receptor antagonists and zileuton (Zyflo) is a 5-lipoxygenase inhibitor. Both agents have been shown clinically to inhibit exercise induced bronchoconstriction as well as reduce daytime symptom scores, nighttime asthma, rescue  $\beta$ -agonist use and improve PEFr and FEV1 in chronic asthma. In the only controlled study comparing these agents to inhaled corticosteroids, beclomethasone 200 mg bid outperformed montelukast 10 mg daily.

Montelukast can be administered without regard to food, while zafirlukast must be dosed one hour before or 2 hours after eating. Both zafirlukast and montelukast are category B in pregnancy while zileuton is category C. Zafirlukast is approved for ages  $>5$  years and montelukast is approved for ages  $> 1$  year using the oral granules. Zafirlukast can potentiate warfarin and theophylline as well as interact with several other medications. Drug interactions with montelukast are rare. Churg-Strauss vasculitis has rarely been reported in association with montelukast or zafirlukast in patients tapering chronic systemic corticosteroids. The FDA mandates monitoring hepatic enzymes with zileuton.

Cromolyn sodium and nedocromil are non-steroidal drugs with anti-inflammatory properties. Their mechanism of action in vivo remains unknown though it has been attributed to stabilization of mast cells and prevention of mediator release. Though these drugs are traditionally thought to have a special role in the treatment of exercise- and allergen-induced asthma, there is in fact no way to predict reliably which patients will respond to them. Cromolyn has no special advantage over  $\beta_2$ -agonists prophylactically or inhaled corticosteroids for maintenance therapy when these other medications are effective and well tolerated. Recent randomized controlled trials in children and observational data in adults do not demonstrate cromolyn or nedocromil to be superior to placebo for non-exercise-induced asthma. Some experts believe there is a subset (perhaps 5-10%) of adult asthmatics who derive additional benefit to that obtained from maximal tolerated doses of inhaled corticosteroids,  $\beta_2$ -agonists, and methylxanthines. While there may be situations where cromolyn sodium or nedocromil may be useful, they should in general be considered second or third line agents.

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**Bronchodilator Medications.** Four types of bronchodilators have been developed.

Inhaled, short-acting  $\beta$ 2-agonists have been and remain a therapy of choice for relief of acute symptoms and prevention of exercise-induced bronchospasm.

Intermittent, short-acting, inhaled  $\beta$ 2-agonists alone are frequently effective in controlling the symptoms of mild asthma. Both chronic and PRN therapy with inhaled  $\beta$ 2-agonists are acceptable as long as asthma symptoms are controlled. Chronic  $\beta$ -agonist use should be accompanied with inhaled corticosteroid therapy. Currently, available data demonstrate that the regular use of long-acting  $\beta$ 2-agonists in combination with inhaled corticosteroids may synergistically improve lung function and should be tried before increasing the dose of inhaled steroids. Inhaled  $\beta$ 2-agonists are as (or more) effective than parenteral  $\beta$ 2-agonists, with longer duration of action, and less likelihood of toxicity.

For patients experiencing difficulty with traditional MDI technique, other  $\beta$ 2-agonist options include use of the Autoinhaler (breath-activated MDI) and Diskus (breath-activated DPI). Similar to steroid DPI's, these delivery systems offer an advantage to patients who cannot coordinate activation and inhalation. Activation is, however, dependent upon a slow, deep inhalation by the patient

Several epidemiologic studies have found an association between excess use of  $\beta$ 2-agonist (short and long-acting) inhalers and asthma mortality. A causal relationship has not been demonstrated, and it is possible that  $\beta$ 2-agonists represent a mere marker for the severity of disease, being more frequently prescribed for patients with life-threatening asthma. If  $\beta$ 2-agonists do have a causative role, it may be an indirect one, such as delaying presentation until airway obstruction is more severe. While important to recognize, such an association would not necessarily mandate a change in current prescribing practices. A number of prospective studies have failed to demonstrate any alteration in airway hyperresponsiveness with chronic  $\beta$ 2-agonist use. In addition, tolerance to the effects of  $\beta$ 2-agonists with chronic use has been hard to demonstrate, suggesting that significant down regulation of  $\beta$ -receptors likely does not occur.

Inhaled, long-acting  $\beta$ 2-agonists. Salmeterol and formoterol, the only long-acting  $\beta$ -agonist DPIs demonstrate a 12-hour duration of action. Salmeterol may be used twice daily for maintenance or before bedtime for nocturnal asthma symptoms. Safety and efficacy for salmeterol has been established in children as young as 4 years of age and for formoterol as young as 5 years. Salmeterol and formoterol are not indicated for acute bronchospasm. The patient should always have a short-acting bronchodilator available for treating acute symptoms. Both agents should be administered in combination with an anti-inflammatory agent when used for maintenance therapy.

A recent secondary data analysis associated use of long-acting beta agonists (LABA) with an increase in asthma deaths, though the numbers in the study were small and the interpretation is still a matter of controversy.

LABAs should be used prudently: 1) as additional symptom control for patients on appropriate asthma control medications (e.g., inhaled corticosteroids), never for primary treatment of asthma; 2) on a scheduled basis not for management of worsening asthma; 3) not for exacerbations. Patients should be cautioned not to discontinue their established LABA without consulting their health care provider.

Methylxanthines have only mild to moderate bronchodilator activity but have additional effects that may be beneficial in the management of asthma. Methylxanthines are quite useful for managing patients with variable or nocturnal symptoms not readily controlled with inhaled medication (anti-inflammatory and  $\beta$ 2-agonist). Theophylline bronchodilator response is roughly linear with serum concentration. Traditionally, levels of 10-20 mcg/ml have been suggested as an optimal compromise between safety and efficacy. Recent data suggest that side-effects are minimized and safety more easily maintained with levels between 5-15 mcg/ml. Current practice tends to use theophylline as an adjunct to inhaled therapy, not as the mainstay. Sustained release theophylline is a convenient way to maintain steady-state levels in the therapeutic range. Sustained release products should be chosen on the basis of cost. Scientific evidence does not support or reject use of theophylline in severe acute asthma.

Anticholinergics are useful in COPD and in status asthmaticus. The benefits of daily use for asthma in children and adults have not been established, even though they are commonly used for refractory patients.

Omalizumab is a recombinant anti-IgE antibody that can be administered subcutaneously daily. It improves control for severe allergic asthma patients who are using maximal doses of conventional therapy [A\*].

Antireflux therapy can reduce airway hyperresponsiveness and improve pulmonary function for GERD patients with asthma [A\*].

Bronchodilator use. When using bronchodilators, consideration should be given to pediatrics management and to home nebulizers.

Pediatric considerations. There are few recommendations to guide the clinician and fewer criteria on which to base fundamental treatment decisions. The FDA has not approved the use of many effective anti-asthma drugs in the very young. "Lack of approval" does not constitute "disapproval."

Metered dose inhalers (MDIs) can be used in infants and smaller children with appropriate spacer devices and masks. Infants and smaller children need MDI doses equivalent to adult doses because they inhale aerosols during tidal

breathing without a breath hold, thus decreasing retention time and effective drug delivery to the lungs. Pediatric patients may be uncooperative with inhaled medication delivery. Compressed air-driven, wet nebulizers are commonly available for home administration of  $\beta_2$ -agonists, cromolyn sodium, ipratropium bromide, and the corticosteroid budesonide. Complete nebulization of medication is time consuming, and the infant or child must keep the mask in place or breath exclusively through the mouthpiece the entire time; "blow-by" administration is ineffective. If proper administration is not tolerated by the child, a metered-dose inhaler with spacer and mask should be used instead.

Home nebulizers have been shown to be no more effective than MDIs in the management of asthma, and may be less effective than MDIs with spacers (particularly the valved-holding-chamber type). They may be helpful in patients who cannot use a metered dose inhaler and spacer properly. It is important, however, that patients recognize that such therapy is not a substitute for initiating other measures, such as initiation or increase in inhaled or oral corticosteroids. Patients need instruction in the mechanical use of the nebulizer; this should be done by a clinician or pulmonary technician. Patients must also be instructed in frequency and dose of such medications.

Patients with nebulizers should always have peak flow meters and should measure peak flow before and after treatment. If peak flows after treatment do not respond significantly (i.e., return to the patient's "all clear zone", 80-100% of personal best PEFr), or if treatment needs to be repeated more frequently, patients should be told to contact their provider and/or start oral corticosteroids based on a previously arranged protocol.

It is critical that patients be given clear guidelines for nebulizer use so as not to delay initiating more definitive therapy with corticosteroids or antibiotics. Patients who regularly need to use a  $\beta_2$ -agonist nebulizer are unstable and need more aggressive baseline therapy.

### **Step 5. Establish plans for managing exacerbations**

All patients should have a written action plan based on signs and symptoms (Asthma Treatment Plan). Instructions for Yellow Zone management are most important. Early treatment is the best strategy. The principal goal of treatment is rapid reversal of airflow obstruction. This is best accomplished by repetitive or continuous administration of an inhaled  $\beta_2$ -agonist. Early administration of systemic corticosteroids should be done in patients with severe attacks or in patients who fail to respond promptly and completely to an inhaled  $\beta_2$ -agonist. Correction of hypoxemia is done by administering supplemental oxygen. Achieving this goal requires close monitoring of the patients' conditions by serial measurement of lung function.

To help asthma patients use home PEFr monitoring, the system of PEFr zones is helpful.

**Green Zone (80-100% of personal best)** = "all clear" no change in therapy; or if asymptomatic for a prolonged period, consider a reduction in medication with continued monitoring.

**Yellow Zone (50-80% of personal best)** = "caution" indicating suboptimal control or early exacerbation.

**Red Zone (<50% of personal best)** = "alert" indicating need for initiation of more intense treatment, often involving a course of corticosteroids.

Persistently symptomatic asthma is most effectively controlled with daily anti-inflammatory therapy. A step-wise approach to pharmacologic therapy is recommended to gain and maintain control of asthma. Initiate therapy at a higher level at the onset to establish prompt control and then step down. The amount and frequency of medication is dictated by asthma severity and directed toward suppression of airway inflammation. Continual monitoring is essential to insure that asthma control is achieved. Step-down therapy is essential to identify the minimum medication necessary to maintain control. All patients with persistent mild, moderate, and severe asthma should receive some form of daily anti-inflammatory medication to diminish chronic airway inflammation and airway hyper-responsiveness (Figure 1). Strong evidence from clinical trials support this recommendation.

### **Step 6. Provide regular follow-up care and consider consultation or referral**

Some situations may warrant a referral or consultation with a specialist in asthma care. These include cases with:

- Diagnosis in doubt. Signs and symptoms are atypical, or there are problems in differential diagnosis.
- Additional diagnostic testing indicated (e.g., skin testing, provocative challenge, rhinoscopy, bronchoscopy, complete pulmonary function studies).
- Inadequate response to asthma therapy. For example, patients considered to have mild or moderate asthma who are hospitalized for exacerbations, or who make more than two ER visits a year.
- Severe refractory asthma, particularly if the clinician cares for very few such patients.
- Other chronic pulmonary disease complicating management.
- Immunotherapy or other complications of therapy.

Special cases meriting consultation with health care team members include:

- Occupational-related asthma. In this situation either the primary care physician or the consultant may benefit from consultation with a physician expert in occupational medicine.
- Patients who chronically do not adhere to their treatment regimen may benefit from an intensive asthma health behavior/health education intervention.

- Significant psychiatric or family problems interfering with treatment.

Additional consideration should be given to consultation or referral in the following situations:

- When the cost of care for a patient becomes excessive.
- When patient disability is substantial despite adequate therapy.

### Management considerations in pregnancy

Asthma in pregnancy is generally managed as for nonpregnant patients. Good control is important for both maternal well-being and optimal fetal growth. It is safer for women to be treated than to suffer poorly-controlled asthma in pregnancy. Systematic reviews of evidence show that beta-agonists, cromolyn, and inhaled corticosteroids are safe in pregnancy. Inhaled steroids reduce the risk of pregnancy-associated exacerbations. The bulk of the data on inhaled steroids is with budesonide; other agents are not well studied. Systemic steroid use in the first trimester is associated with cleft lip and palate, and later in pregnancy with pregnancy-induced hypertension and possibly preeclampsia. Theophylline in high doses is associated with adverse pregnancy outcomes; while usual doses are safe, unacceptable side effects are frequent. Few data are available about antileukotrienes in pregnancy; animal data suggest leukotriene receptor antagonists (but not lipoxygenase inhibitors) may be safe.

### What the Patient Should Know

The greater the understanding that patients and their families have of asthma, the more successful their management will be. The key educational points for patients and their families include the following:

- **Understand mechanisms.** Understanding two major ingredients of asthma: bronchospasm and inflammation and which medications are used for each.
- **Triggers.** Being familiar with what triggers their asthma flare (e.g., viral URI, environmental allergens, exercise, cold, stress). Learning how to avoid triggers or self-medicate to prevent predictable exacerbations.
- **Signs.** Knowing their own warning signs (e.g., increased shortness of breath, chest tightness, or cough).
- **Peak flow meter use.** For moderate or severe asthma: using a peak flow meter and knowing how to interpret the results.
- **Metered dose inhaler use.** Knowing how to correctly use a metered dose inhaler and spacer.
- **Treat flares.** Learning how to self-medicate to initiate treatment of flares.
- **Acute exacerbation.** Knowing what constitutes an acute exacerbation and what to do in such circumstances.

### Strategy for Literature Search

Relevant data were identified using a Medline search that included the following terms: asthma, peak flow meter, spirometry, diagnosis, treatment, randomized controlled trials, practice guidelines. Also reviewed were literature referenced in the National Asthma Education Program's Executive summary: Guidelines for the diagnosis and management of asthma, 1994, and the international consensus report on diagnosis and treatment of asthma: A call to action for US practitioners, Clinical Therapeutics 1994;16(4):694-706.

### Related National Guidelines

This guideline is consistent with the National Heart, Lung, and Blood Institute's expert panel report: Guidelines for the diagnosis and management of asthma. National Institutes of Health. Bethesda, Maryland 20892. Publication No. 97-4051. 1997. It also uses material from the 2004 update for asthma in pregnancy, NIH Publication No. 05-3279.

### Disclosures

The University of Michigan Health System endorses the Guidelines of the Association of American Medical Colleges and the Standards of the Accreditation Council for Continuing Medical Education that the individuals who present educational activities disclose significant relationships with commercial companies whose products or services are discussed. Disclosure of a relationship is not intended to suggest bias in the information presented, but is made to provide readers with information that might be of potential importance to their evaluation of the information.

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## APPENDIX. Predicted Average Peak Expiratory Flow

**Note:** These charts are for informational purposes only. Spirometry should be used for diagnosis and staging. "Personal best" measures should be used for the asthma treatment plan.

### A. Predicted Average Peak Expiratory Flow for Normal Children and Adolescents (liters/minute)

Height (inches)	Males & Females	Height (inches)	Males & Females
43	147	56	320
44	160	57	334
45	173	58	347
46	187	59	360
47	200	60	373
48	214	61	387
49	227	62	400
50	240	63	413
51	254	64	427
52	267	65	440
53	280	66	454
54	293	67	467
55	307		

Adapted from: Polger G, Promedhat V: *Pulmonary function testing in children: Techniques and standards*, Philadelphia. W.B. Saunders, 1971.

### B. Predicted Average Peak Expiratory Flow for Normal Males (liters / minute)

AGE	HEIGHT				
	60"	65"	70"	75"	80"
20	554	602	649	693	740
25	543	590	636	679	725
30	532	577	622	664	710
35	521	565	609	651	695
40	509	552	596	636	680
45	498	540	583	622	665
50	486	527	569	607	649
55	475	515	556	593	634
60	463	502	542	578	618
65	452	490	529	564	603
70	440	447	515	550	587

Adapted from: Leiner GC, et al.: Expiratory peak flow rate. Standard values for normal subjects. Use as a clinical test of ventilatory function. *Am Resp Dis* 88:644, 1963.

### C. Predicted Average Peak Expiratory Flow for Normal Females (liters / minute)

AGE	HEIGHT				
	55"	60"	65"	70"	75"
20	390	423	460	496	529
25	385	418	454	490	523
30	380	413	448	483	516
35	375	408	442	476	509
40	370	402	436	470	502
45	365	397	430	464	495
50	360	391	424	457	488
55	355	386	418	451	482
60	350	380	412	445	475
65	345	375	406	439	468
70	340	369	400	432	461

Adapted from: Leiner GC, et al.: Expiratory peak flow rate. Standard values for normal subjects. Use as a clinical test of ventilatory function. *Am Resp Dis* 88:644, 1963.

# Asthma Treatment Plan

Name \_\_\_\_\_

Date \_\_\_\_\_

## Maintenance Program (Resume when symptoms return to normal.)

**Peak flow GREEN ZONE** = \_\_\_\_\_ (80% or higher of personal best) No breathing problems, no cough or wheezing, able to do normal activities.

**Personal Best:** \_\_\_\_\_

Medication	Morning	Lunch	Dinner	Bedtime
_____ (Anti-inflammatory)	_____	_____	_____	_____
_____ to be used 2 puffs 3-4 times daily only as needed and in an acute attack (Rescue/Short-acting bronchodilator )	_____	_____	_____	_____
_____ (Long-acting bronchodilator)	Do NOT use for an asthma attack!			
_____ (Other)	_____	_____	_____	_____

## Breathing Problems

**Peak flow YELLOW ZONE** = \_\_\_\_\_ (80% to 50% of personal best) Coughing, wheezing, chest tightness, breathing problems at night

Medication	Morning	Lunch	Dinner	Bedtime
_____ (Anti-inflammatory)	_____	_____	_____	_____
_____ to be used 2 puffs 3-4 times daily only as needed and in an acute attack (Rescue/Short-acting bronchodilator )	_____	_____	_____	_____
_____ (Long-acting bronchodilator)	Do NOT use for an asthma attack!			
_____ (Other)	_____	_____	_____	_____

## Severe Breathing Problems

**Peak flow RED ZONE** = \_\_\_\_\_ (less than 50% of personal best) Medicine is not helping, breathing is hard and fast, difficult to walk or talk

**If you develop severe breathing problems:**

- CALL 911 or HAVE SOMEONE DRIVE YOU TO THE EMERGENCY ROOM.
- Take \_\_\_ mg of prednisone by mouth.
- Continue to use your inhaled bronchodilator – two puffs every 20 minutes as needed up to three times in 60 minutes.

## Additional Information: