National Athletic Trainers' Association Position Statement: Management of Asthma in Athletes

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Objective: To present guidelines for the recognition, prophylaxis, and management of asthma that lead to improvement in the quality of care certified athletic trainers and other heath care providers can offer to athletes with asthma, especially exercise-induced asthma.

Background: Many athletes have difficulty breathing during or after athletic events and practices. Although a wide variety of conditions can predispose an athlete to breathing difficulties, the most common cause is undiagnosed or uncontrolled asthma. At least 15% to 25% of athletes may have signs and symptoms suggestive of asthma, including exercise-induced asthma. Athletic trainers are in a unique position to recognize breathing difficulties caused by undiagnosed or uncontrolled asthma, particularly when asthma follows exercise. Once the diagnosis of asthma is made, the athletic trainer should play a pivotal role in supervising therapies to prevent and control asthma symptoms. It is also important for the athletic trainer to recognize when asthma is not the underlying cause for respiratory difficulties, so that the athlete can be evaluated and treated properly.

Recommendations: The recommendations contained in this position statement describe a structured approach for the diagnosis and management of asthma in an exercising population. Athletic trainers should be educated to recognize asthma symptoms in order to identify patients who might benefit from better management and should understand the management of asthma, especially exercise-induced asthma, to participate as active members of the asthma care team.

Key Words: airway hyperresponsiveness, airway obstruction, exercise-induced asthma, exercise-induced bronchospasm, pulmonary function tests, certified athletic trainer

INTRODUCTION

sthma is defined as a chronic inflammatory disorder of the airways characterized by variable airway obstruction and bronchial hyperresponsiveness.¹ Airway obstruction can lead to recurrent episodes of wheezing, breathlessness, chest tightness, and coughing, particularly at night or in the early morning.1 Asthma can be triggered by many stimuli, including allergens (eg, pollen, dust mites, animal dander), pollutants (eg, carbon dioxide, smoke, ozone), respiratory infections, aspirin, nonsteroidal anti-inflammatory drugs (NSAIDs), inhaled irritants (eg, cigarette smoke, household cleaning fumes, chlorine in a swimming pool), particulate exposure (eg, ambient air pollutants, ice rink pollution), and exposure to cold and exercise.¹⁻⁵ Airflow limitation is often reversible, but as asthma symptoms continue, patients may develop "airway remodeling" that leads to chronic irreversible airway obstruction.^{6,7} Severe attacks of asthma can also cause irreversible airflow obstruction that can lead to death.^{4,8}

The National Heart, Lung, and Blood Institute of the National Institutes of Health launched the National Asthma Education and Prevention Program (NAEPP) in March 1989 to address the increasing prevalence of asthma in the United States and its economic costs to the society; the program was updated in 1997 (as NAEPPII).¹ An updated expert panel report from the NAEPP is expected to be released in 2006. The Global Initiative for Asthma (GINA) was also developed to provide worldwide guidelines for asthma awareness and management.² These guidelines are extremely comprehensive and have been regularly updated to reflect advances in the diagnosis and management of asthma. Nevertheless, the guidelines do not describe the role of certified athletic trainers or other allied health care professionals in recognizing and managing asthma in an athletic population.

PURPOSE

The purpose of this position statement is to provide athletic trainers and other allied health care professionals who care for athletes with information to:

1. Identify the characteristics and diagnostic criteria of asth-

ma, especially exercise-induced asthma (EIA) or exercise-induced bronchospasm (EIB).

- 2. Provide guidelines for referral so that patients with asthma and those in whom asthma is suspected can receive a thorough evaluation.
- 3. Describe management plans to prevent attacks and to control asthma exacerbations when they occur.
- 4. Educate certified athletic trainers and athletes about pharmacologic and nonpharmacologic therapies and techniques to help control asthma.

RECOMMENDATIONS

Based on current research and literature, the National Athletic Trainers' Association provides the following guidelines for the identification, examination, management, and prophylaxis of asthma, including EIA, and the education of athletes, parents, coaches, and health care personnel about asthma. Not all individuals who suffer from asthma present in the same manner, nor do they all respond to the same management or treatment plan. Therefore, these recommendations are intended to provide the certified athletic trainer and other health care professionals with an overall guide for a better understanding of the asthmatic condition.

Asthma Identification and Diagnosis

- 1. All athletes must receive preparticipation screening evaluations sufficient to identify the possible presence of asthma.^{9–12} In most situations, this evaluation includes only obtaining a thorough history from the athlete. However, in special circumstances, additional screening evaluations (eg, spirometry testing or the challenge testing described below) should also be performed because the history alone is not reliable.¹⁰
- 2. Athletic trainers should be aware of the major signs and symptoms suggesting asthma, as well as the following associated conditions^{5,13,14}:
 - a. Chest tightness (or chest pain in children)
 - b. Coughing (especially at night)
 - c. Prolonged shortness of breath (dyspnea)
 - d. Difficulty sleeping
 - e. Wheezing (especially after exercise)
 - f. Inability to catch one's breath
 - g. Physical activities affected by breathing difficulty
 - h. Use of accessory muscles to breathe
 - i. Breathing difficulty upon awakening in the morning
 - j. Breathing difficulty when exposed to certain allergens or irritants
 - k. Exercise-induced symptoms, such as coughing or wheezing
 - 1. An athlete who is well conditioned but does not seem to be able to perform at a level comparable with other athletes who do not have asthma
 - m. Family history of asthma
 - n. Personal history of atopy, including atopic dermatitis/ eczema or hay fever (allergic rhinitis)

Note: Although there is a correlation between the presence of symptoms and EIA, the diagnosis should not be based on history alone.⁵ Rather, these symptoms should serve to suggest that an athlete may have asthma.

3. The following types of screening questions can be asked to seek evidence of asthma¹³:

- a. Does the patient have breathing attacks consisting of coughing, wheezing, chest tightness, or shortness of breath (dyspnea)?
- b. Does the patient have coughing, wheezing, chest tightness, or shortness of breath (dyspnea) at night?
- c. Does the patient have coughing, wheezing, or chest tightness after exercise?
- d. Does the patient have coughing, wheezing, chest tightness, or shortness of breath (dyspnea) after exposure to allergens or pollutants?
- e. Which pharmacologic treatments for asthma or allergic rhinitis, if any, were given in the past, and were they successful?
- 4. Patients with atypical symptoms, symptoms despite proper therapy, or other complications that can exacerbate asthma (such as sinusitis, nasal polyps, severe rhinitis, gastroesophageal reflux disease, or vocal cord dysfunction) should be referred to a physician with expertise in sports medicine (eg, allergist; ear, nose, and throat physician; cardiologist; or pulmonologist with training in providing care for athletes).¹⁵ Testing might include a stress electrocardiogram, upper airway laryngoscopy or rhinoscopy, echocardiogram, or upper endoscopy.

Pulmonary Function Testing

- 5. Athletes with a history of asthma or of taking a medication used to treat asthma and those suspected of having asthma should consult a physician for proper medical evaluation and to obtain a classification of asthma severity (Table 1). This evaluation should include pulmonary function testing.^{16–18}
- 6. An exercise challenge test is recommended for athletes who have symptoms suggestive of EIA to confirm the diagnosis.¹⁹
- 7. If the diagnosis of asthma remains unclear after the above tests have been performed, then additional testing should be performed to assist in making a diagnosis.^{15,20,21} Physicians should be encouraged, when possible, to test the athlete using a sport-specific and environment-specific exercise-challenge protocol, in which the athlete participates in his or her venue to replicate the activity or activities and the environment that may serve to trigger airway hyperresponsiveness.^{20,21} In some cases, athletes should also be tested for metabolic gas exchange during strenuous exercise to determine fitness (eg, to assess anaerobic threshold and $\dot{V}o_2max$), especially to rule out the diagnosis of asthma or to rule in another diagnosis (eg, pulmonary fibrosis) for a patient with an unclear diagnosis.¹⁶

Asthma Management

8. Athletic trainers should incorporate into the existing emergency action plan an asthma action plan for managing and urgently referring all patients who may experience significant or life-threatening attacks of breathing difficulties (Figure 1).^{1.2} Immediate access to emergency facilities during practices and game situations should be available. For example, athletic trainers should be familiar with appropriate community resources and must have a fully functional telephone (mobile or cellular) available, preprogrammed with emergency medical care access numbers. A telephone might be the single most important de-

Table 1. National Asthma Education and Prevention Program II: Classification of Asthma Severity ¹
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	Clinical Features Before Treatment†		
	Symptoms‡	Nighttime Symptoms	Lung Function
Step 4 Severe persistent	Continual symptomsLimited physical activityFrequent exacerbations	Frequent	 FEV₁ or PEF ≤60% predicted PEF variability >30%
Step 3 Moderate persistent	 Daily symptoms Daily use of inhaled short-acting beta₂-agonist Exacerbations affect activity Exacerbations ≥2 times/wk; may last days 	>1 time/wk	 FEV₁ or PEF >60%-<80% predicted PEF variability ≥30%
Step 2 Mild persistent	 Symptoms >2 times/wk but <1 time/d Exacerbations may affect activity 	>2 times/mo	 FEV₁ or PEF ≥80% predicted PEF variability 20–30%
Step 1 Mild intermittent	 Symptoms ≤2 times/wk Asymptomatic and normal PEF between exacerbations Exacerbations brief (from a few hours to a few days); intensity may vary 	≤2 times/mo	 FEV₁ or PEF ≥80% predicted PEF variability <20%

*FEV₁ indicates forced expiratory volume in 1 s; PEF, peak expiratory flow.

†The presence of one of the features of severity is sufficient to place a patient in that category. An individual should be assigned to the most severe grade in which any feature occurs. The characteristics noted in this figure are general and may overlap because asthma is highly variable. Furthermore, an individual's classification may change over time.

*Patients at any level of severity can have mild, moderate, or severe exacerbations. Some patients with intermittent asthma experience severe and life-threatening exacerbations separated by long periods of normal lung function and no symptoms.

vice to have on the practice field for a patient who is experiencing an asthma exacerbation. In addition, athletic trainers should have pulmonary function measuring devices (such as peak expiratory flow meters [PFMs] or portable spirometers) at all athletic venues for athletes for whom such devices have been prescribed and should be familiar with how to use these devices.²²

- 9. Patients who are experiencing any degree of respiratory distress (including a significant increase in wheezing or chest tightness, a respiratory rate greater than 25 breaths per minute, inability to speak in full sentences, uncontrolled cough, significantly prolonged expiration phase of breathing, nasal flaring, or paradoxic abdominal movement) should be referred rapidly to an emergency department or to their personal physicians for further evaluation and treatment. Referral to an emergency room or equivalent facility should be sought urgently if the patient is exhibiting signs of impending respiratory failure (eg, weak respiratory efforts, weak breath sounds, unconsciousness, or hypoxic seizures).
- 10. All patients with asthma should have a rescue inhaler available during games and practices, and the certified athletic trainer should have an extra rescue inhaler for each athlete for administration during emergencies. In case of emergencies, a nebulizer should also be available. With a metered dose inhaler (MDI), athletes should be encouraged to use a spacer to help ensure the best delivery of inhaled therapy to the lungs.²³
- 11. Athletic trainers and coaches should consider providing alternative practice sites for athletes with asthma triggered by airborne allergens when practical. Indoor practice facilities that offer good ventilation and air conditioning should be considered for at least part of the practice if this can be accomplished, although in most cases it will not be practical. For example, indoor and outdoor allergens or

irritants, tobacco smoke, and air pollutants might trigger asthma, and attempts should be made to limit exposure to these triggers when possible. Another option is to schedule practices when pollen counts are lowest (eg, in the evening during the ragweed pollen season). Pollen count information can be accessed from the National Allergy Bureau at http://www.aaaai.org/nab.

12. Patients with asthma should have follow-up examinations at regular intervals, as determined by the patient's primary care physician or specialist, to monitor and alter therapy. In general, these evaluations should be scheduled at least every 6 to 12 months, but they may be more frequent if symptoms are not well controlled.

Asthma Pharmacologic Treatment

- 13. Athletic trainers should understand the various types of pharmacologic strategies used for short- and long-term asthma control and should be able to differentiate controller from rescue or reliever medications (Figure 2).^{24–30}
- 14. Patients with EIA may benefit from the use of short- and long-acting β_2 -agonists. Rapid-acting agents can be used for prophylaxis during practice and game participation. When the goal is to prevent EIA, a short-acting β_2 -agonist, such as albuterol, should be inhaled 10 to 15 minutes before exercise. The excessive need (3–4 times per day) for short-acting β_2 -agonist therapy during practice or an athletic event should cause concern, and a physician should evaluate the patient before return to participation. Long-acting β_2 -agonists should, in general, only be used for asthma prophylaxis and control and are usually combined with an inhaled corticosteroid. Athletic trainers should understand the use, misuse, and abuse of short-acting β_2 -agonists.
- 15. Patients with asthma may also benefit from the use of

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Homeroom Teach	er:		Room:		
Parent/Guardian	Name:		Ph:(h):		ID Photo
	Address:		Ph: (w):		
Parent/Guardian	Name:		Ph: (h):		
	Address:		Ph: (w):		
Emergency Phone	Contact #1	Name	Polotion		Phone
Emergency Phone	Contact #2		Relationship		Phone
canorgoney r none	connot no	Name	Relationship	,	Phone
Physician Treating	g Student for Asthma:			Ph:	
Other Physician:				Ph:	
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Figure 1. Sample asthma action plan. Extracted from *Managing Asthma: A Guide for Schools*. National Heart, Lung, and Blood Institute. Available at: http://www.nhlbi.nih.gov/health/prof/lung/asthma/asth_sch.htm. Accessed June 7, 2005.

leukotriene modifiers, inhaled or parenteral corticosteroids, and cromones (such as cromolyn sodium).

- 16. Pharmacotherapy should be customized for each asthma patient, and a specialist (an allergist or pulmonologist with expertise in sports medicine) should be consulted to maximize therapy when symptoms break through despite apparently optimal therapy.
- 17. Patients with past allergic reactions or intolerance to aspirin or NSAIDs should be identified and provided with alternative medicines, such as acetaminophen, as needed.

Asthma Nonpharmacologic Treatment

- 18. Health care providers should identify and consider nonpharmacologic strategies to control asthma, including nose breathing, limiting exposure to allergens or pollutants, and air filtration systems.^{31–33} However, these therapies should be expected to provide only limited protection from asthma in most circumstances.
- 19. Proper warm-up before exercise may lead to a refractory period of as long as 2 hours, which may result in de-

DAILY ASTHMA MANAGEMENT	PLAN	
 Identify the things which start 	an asthma episode (Check each th	nat applies to the student.)
Exercise	□ Strong odors or fumes	• Other
Respiratory infections	Chalk dust / dust	
Change in temperature	Carpets in the room	
Animals	Pollens	
G Food	Molds	
Comments		
 Control of School Environmen 	t	
	ures, pre-medications, and/or dietary restr	ictions that the student needs to prevent an asthm
Peak Flow Monitoring		
Monitoring Times:		
 Daily Medication Plan 		
Name	Amount	When to Use
1		
4		
COMMENTS / SPECIAL INSTRU	UCTIONS	
11 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1	*****	
FOR INHALED MEDICATIONS		
		roper way to use his/her medications. It is my
professional opinion that him/herself.	should b	e allowed to carry and use that medication by
	at should not carry h	sis/her inhaled medication by him/herself.
		maner minutes intereation by minutesent
Phys	ician Signature	Date
Parer	nt/Guardian Signature	Date

Figure 1. Continued.

creased reliance on medications by some patients with asthma. $^{\rm 34}$

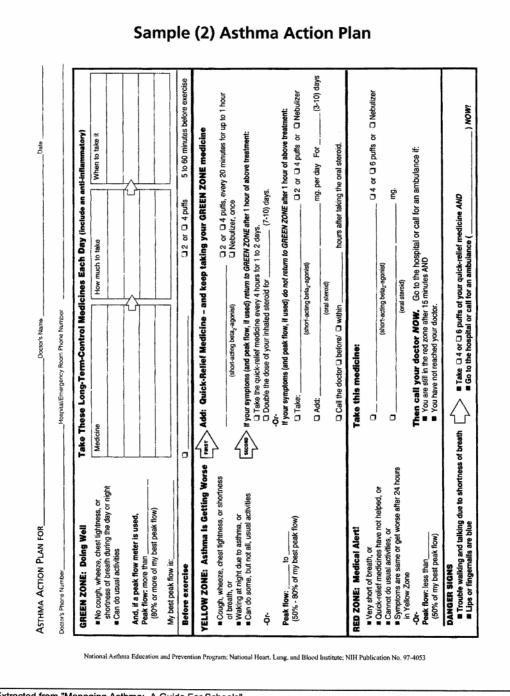
20. Patients who have been diagnosed previously as having asthma or suspected of having asthma should follow the recommendations of NAEPPII and GINA for evaluation and everyday management and control.^{1,2}

Asthma Education

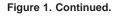
21. Athletes should be properly educated about asthma, especially EIA, by health care professionals who are knowl-

edgeable about asthma.^{35–52} Athletes should be educated about the following:

- a. Recognizing the signs and symptoms of uncontrolled asthma.
- b. Using spirometry recording devices to monitor lung function away from the clinic or athletic training room.
- c. Methods of limiting exposure to primary and secondary smoke and to other recognized or suspicious asthma triggers (eg, pollens, animal allergens, fungi, house dust, and other asthma sensitizers and triggers). Patients with asthma who smoke should be provided with



Extracted from "Managing Asthma: A Guide For Schools"



information about smoking cessation and encouraged to participate in classes to change socialization patterns.

- d. The need for increased asthma rescue medication (eg, short-acting β_2 -agonists) as a signal for asthma flareup. Increased use of short-acting β_2 -agonists signals a need for enhanced treatment with asthma controller therapy.
- e. The proper techniques for using MDIs, dry powder inhalers, nebulizers, and spacers to control asthma symptoms and to treat exacerbations. Health care professionals should periodically check the patient's

medication administration techniques and should examine medication compliance.

- f. Asthma and EIA among competitive athletes. These conditions are common, and athletic performance need not be hindered if the patient takes an active role in controlling the disease and follows good practice and control measures.
- 22. The athletic trainer should also be familiar with vocal cord dysfunction and other upper airway diseases, which can sometimes be confused with asthma.^{15,53,54} Vocal cord dysfunction may be associated with dyspnea, chest tight-

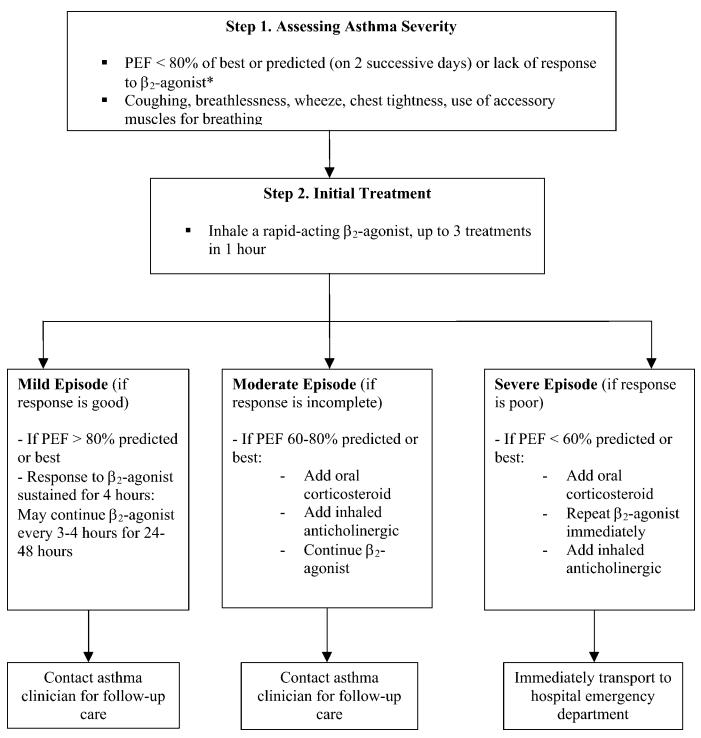


Figure 2. Asthma pharmacologic management.² PEF indicates peak expiratory flow.

ness, coughing, wheezing, and inspiratory stridor. In many cases, the condition is triggered with exercise. Visual inspection of the vocal cords by a physician experienced in examining the upper airway during exercise to differentiate vocal cord dysfunction from asthma is recommended.

- 23. Patients with asthma should be encouraged to engage in exercise as a means to strengthen muscles, improve respiratory health, enhance endurance, and otherwise improve overall well-being.⁵⁵
- 24. The athletic trainer should differentiate among restricted, banned, and permitted asthma medications. Athletic train-

ers should be familiar with the guidelines of the International Olympic Committee Medical Commission, the United States Anti-Doping Agency, the World Anti-Doping Agency, and the doping committees of the various federations.

25. The athletic trainer should be aware of the various Web sites that provide general information and frequently asked questions on asthma and EIA. These sites include the American Academy of Allergy, Asthma and Immunology (www.aaaai.org); the American Thoracic Society (www. thoracic.org); the Asthma and Allergy Foundation of Amer-

ica (www.aafa.org); the American College of Allergy, Asthma, & Immunology (www.acaai.org); and USA Swimming (http://www.usaswimming.org/USASWeb/_Rainbow/ Documents/6c812467-b717-4c16-a32c-a1d9bcc9f444/ Asthma-%20Comprehensive%20Guide%2004%20Nov% 2029.pdf).

BACKGROUND AND LITERATURE REVIEW

Definition and Pathophysiology of Asthma

Asthma is a common condition that has been recognized for more than 2000 years.⁵⁶ Asthma is usually defined operationally as a chronic inflammatory disorder of the airways.^{1,2,4,56} In many patients, this chronic inflammation causes an increase in airway hyperresponsiveness, leading to recurrent episodes of wheezing, breathlessness, chest tightness (or chest pain in children), and coughing, particularly at night or in the early morning and after exercise, especially in cold, dry environments. These episodes are associated with widespread but variable airflow obstruction that is often reversible, either spontaneously or with treatment.^{1,2,4} This definition implies that asthma has multiple causes, and indeed, it is a complex disorder.

The chronic inflammatory process causes excess mucus production and bronchial smooth muscle constriction,^{57–61} which result from a release of inflammatory mediators that include histamine, tryptase, prostaglandin, and leukotrienes from mast cells.⁶²⁻⁶⁶ Airways may also accumulate thick, viscous secretions produced by goblet cells and submucosal glands; moreover, there is leakage of plasma proteins and accumulated cellular debris.^{57–61,67} Although airway narrowing affects the tracheobronchial tree, small bronchi (2-5 mm in diameter) are most affected.⁶⁸⁻⁷⁰ Maximal expiratory flow rate is reduced and residual lung volumes are increased as air is trapped behind the blocked airways.⁷¹ As a result, during an asthma attack, the respiratory rate increases to compensate for the increased obstruction of the airways and the inability of the usually elastic lung to recoil (dynamic hyperinflation). The patient must work harder to breathe as the thorax becomes overinflated. With progression of the attack, the diaphragm and intercostal muscles must compensate and contribute more energy during respiration.⁷² In a severe attack, muscle efficiency is eventually lost, and the increased breathing rate leads to respiratory muscle fatigue and physical distress that may result in death. Indeed, as many as 4200 to 5000 people die from asthma each year in the United States.⁷³

Environmental Factors

Environmental factors, such as allergens, air pollution, occupational sensitizers, and tobacco smoke, may cause or exacerbate asthma.^{74–79} These factors are important triggers that should be considered when evaluating a patient with asthma.^{79–83}

Recently, concern about indoor air pollution has been heightened.^{79,80,82–84} Indoor ambient air can contain allergens and pollution that can cause or exacerbate asthma and other respiratory ailments when susceptible individuals are exposed to these environments.⁸⁵ Many factors should be considered indoors, such as adequacy of ventilation, humidity level, presence of allergens, presence of wall-to-wall carpeting and up-

holstered furniture, and types of building materials.^{85–87} Indoor animal allergens (such as from cats, dogs, and other pets) are an important trigger of symptoms in many people.⁸⁸ Cockroach allergens are commonly seen indoors (such as in swimming pool locker rooms), and the cockroach allergenic materials can remain for a long period of time, even after extermination.^{89–92} Indoor mold and fungal spores, house dust mites, and particulate matter, such as aerosols or smoke from cigarettes, wood, or fossil fuels, also can trigger asthma symptoms.^{93–107} Tobacco smoke is a risk factor for the development of asthma, and smoking tobacco appears to increase asthma severity.^{108–110}

Other indoor air irritants, such as chlorine, can exacerbate asthma and cause eye and lung irritation.^{111,112} School-age children who frequently visit chlorinated pools may have an increased risk of developing asthma, especially if they have other risk factors for asthma.¹¹³ Even individuals who do not usually enter the water but who are exposed to indoor chlorinated pools (eg, lifeguards, coaches) may have respiratory irritation on exposure to chlorine.^{114,115}

Many actions can be taken to limit indoor allergen exposure, including prohibiting smoking indoors, using air cleaners equipped with a high-efficiency particulate air cleaner (HEPA) filter, washing walls, vacuuming carpets, and cleaning mattress covers weekly.^{116–119} Additional measures include removing carpets and installing linoleum or wood flooring, washing pets (dogs and cats) and their bedding twice a week, keeping pets out of the bedroom or living room at all times to reduce exposure, covering mattresses and pillows, and controlling humidity to help manage dust mites and mold.^{81,82,120} Some of these measures, such as the removal of a family pet from the home, can be very difficult, so it is necessary to discuss the effect of these exposures with the asthma patient. Although air filters might help, the house should be cleaned thoroughly before their use and regularly thereafter.³³

When inhaled, outdoor air pollution, caused by sulfur dioxide, carbon monoxide, nitrogen dioxide, ozone, and particulate matter, can cause pulmonary function decrements, increased reliance on medications, bronchial hyperresponsiveness, and increased asthma symptoms.^{121–125} Many pollens (trees, grasses, and weeds) are inhaled into the bronchi and cause allergeninduced asthma.^{126,127} Tree pollens predominate in the spring, grass pollens in the late spring or early summer (and fall), and weed pollens in the late summer and fall in the United States, depending on geographic location, but may be present at other times of the year in locations outside of the United States.^{128,129} Information about the pollen seasons in the United States can be accessed from the National Allergy Bureau Web site.

Although it is virtually impossible to avoid all outdoor pollution and allergens, some practical precautions can be implemented: move indoors and close all windows, close car windows when traveling, limit exposure when pollen is at its highest levels, monitor local weather stations for allergy reports, and practice indoors if possible when pollen counts are high.

Diagnosis and Classification of Asthma

Asthma can be difficult to diagnose and classify (see Table 1). Some individuals, especially elite athletes, do not display consistent signs or symptoms of asthma.^{9,10,130} Asthma symptoms may be present only during certain times (seasons) of

the year or only after exercise and may be highly variable, depending upon the athlete, the environment, and the activity. 13

The first step in determining whether asthma is present is to obtain a detailed medical history. Questions regarding past experiences, symptoms, smoking history, and family history can help to rule out other respiratory disorders such as chronic bronchitis, emphysema, bronchiectasis, allergic rhinitis, upper respiratory infection, congestive heart failure, disorders of the upper airway (eg, vocal cord dysfunction), and nonrespiratory conditions such as anxiety. Most importantly, the athletic trainer should ask general questions as listed in Recommendation 3 to assist in making a proper diagnosis.¹³ If a patient appears to have one or more symptoms suggestive of asthma, then lung function testing should be performed (see Table 1).⁹ However, it is important to recognize that the history and baseline physical examination will fail to identify many patients with EIA.^{9,10,12}

Lung function tests are essential to assess asthma severity and airflow limitation and to determine whether the obstruction is fully reversible with treatment.^{16-18,131-133} The most common measures of airway function are the forced expiratory volume in 1 second (FEV_1), the forced vital capacity (FVC), and the peak expiratory flow rate (PEFR). These tests can be performed while the patient is at rest or after a challenge. The FEV₁ measures the volume of air in liters forcefully exhaled out of the airway in 1 second after a full inspiration. The FVC measures the total volume of air in liters forcefully exhaled out of the airway when the breath continues (usually for a period of 6 or more seconds). The FVC procedure is effort dependent and requires the patient to fully understand that he or she needs to inhale a deep breath and then "blast" the air out of the lungs into the measurement device. The FVC testing also requires considerable expertise by the technician and the ability to communicate with the patient. The PEFR measures the maximal flow rate of air (in L/s or L/min) out of the airways and is easier to perform than an FEV₁ or FVC maneuver, although PEFR testing is also effort dependent. A flow volume curve (flow loop) provides a graphic depiction of the breathing effort in which flow rate is plotted against volume (in L/s) of air exhaled and inhaled (in L), as shown in Figure 3. A volume-time curve (called a spirogram) is another way to plot the breath, in which the volume of air exhaled (in L) is plotted against time (in seconds). The FEV₁/FVC ratio is also examined, along with a variety of measures of flow, such as the flow between 25% and 75% of the FVC (called the FEF_{25-75}).

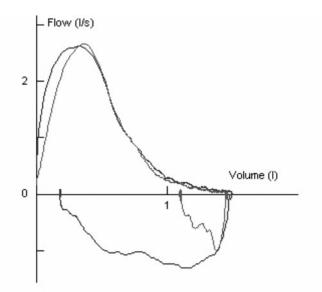
Asthma is an example of an obstructive lung disease in which the airways obstruct the outflow of air. In contrast, pulmonary fibrosis is an example of a restrictive lung disease in which the functional size of the lungs decreases. In obstructive lung diseases, the FEV₁ decreases, whereas the FVC remains relatively normal, so the FEV₁/FVC ratio decreases (until late in the disease or with severe exacerbations, when the FVC may also decrease).¹³¹ In restrictive lung disease, both the FEV_1 and the FVC decrease proportionally (so that the FEV_1 / FVC ratio is normal). Nomograms exist to provide guidance as to normal ranges for FEV1 and FVC based on age, size and race.134,135 It is important to recognize racial differences in the normal values for these tests. Generally, levels should be at least 80% of the predicted values to be considered "normal." The FEV₁/FVC ratio should also be above 80%. An increase of 12% or more in FEV₁ after an inhaled bronchodilator (eg, a β_2 -agonist such as albuterol) suggests reversible airway disease and may be used as a diagnostic criterion of asthma.¹³¹ A decrease in FEV_1 after a challenge (such as after inhalation of methacholine or running on a treadmill) suggests that the airways are reactive to the stimulus.¹⁹ It is important to determine a patient's personal best FEV_1 , FVC, and PEFR, which are identified by plotting these values over time. These values can also demonstrate variability between morning and evening and over time, which may reflect airway hyperreactivity.

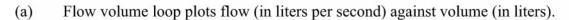
Spirometers are used in the clinic or training room to determine these pulmonary function values.¹³⁶ Patients may also be given a PFM to measure PEFR away from the clinic or athletic training room (Tables 2 and 3). The PFM is a small, handheld device that measures maximal flow rate during forced exhalation.¹³⁷⁻¹⁴⁰ Maximal flow rate usually occurs within the first 120 to 150 milliseconds of a forced exhalation.¹⁴⁰ When used properly, a PFM can be somewhat helpful in following the course of asthma and might even be useful in suggesting the presence of asthma. The PFM can also be used to identify asthma triggers and to monitor medication changes, and it may help to reduce asthma morbidity.^{141–147} The device allows the asthmatic patient to measure lung variability over time to assist in determining when to seek medical attention. The PFM should be used at least daily (in the morning after awakening) and preferably also in the late afternoon or evening.^{1,2,148} At least 2 or 3 trials should be performed at each specified time and the highest value recorded. The device should be used before taking any medications and at least 4, but preferably 8, hours after inhalation of a bronchodilator, if possible. The device should be used for at least a 2- to 3-week monitoring period. Some devices record the PEFR electronically, which can assist the patient in keeping the data secure and available. Over time, the patient's personal best value will be determined. Subsequently, if the PEFR value is less than 80% of the personal best or if daily variability is greater than 20% of the morning value, then the patient should be reevaluated in an attempt to find better control measures. If the PEFR value is less than 50% of the personal best, the individual should seek immediate attention (see Table 2).¹ Spirometry, including the use of PFMs, may be especially useful in patients who do not perceive the severity of their symptoms.149,150

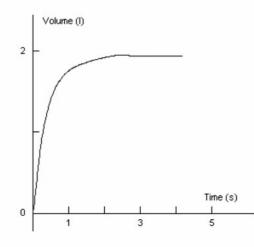
The PFM should be used regularly, even if asthma symptoms appear to be well controlled.^{132,151–153} Patients who have good PFM technique (see Table 3) adhere to their treatment plan and control their symptoms better than those who have poor technique. The PFM should be available in all athletic training facilities and on the field in medical kits.

Unfortunately, some reports suggest that PFM recordings are not always reliable indicators of airflow obstruction.^{154–159} In certain cases, elite or well-trained athletes may possess large lung capacities, which may exceed the measuring capacity of the PFM. In addition, it appears that PFM values are not consistent from one to another PFM from the same manufacturer, across different PFM devices, when men and women use PFMs, with different techniques while using a single PFM, and at high altitudes.^{159–164} The most accurate spirometry testing is performed with office-based spirometry testing requipment.¹⁴⁰ However, as noted above, spirometry testing requires training^{22,165} and may be impractical for everyday asthma management, especially in primary care settings in which most patients are not being seen for asthma.¹⁴⁰

If baseline lung function tests are within normal values and







(b) Volume time curve (spirogram) plots volume (in liters) against time (in seconds). Figure 3. Flow volume loop and volume time curve.

Table 2. Peak Flow Zones for Asthma Management*	Table 3. General Instructions for Using a Mechanical Peak Flow Meter*		
 Green zone PEF values are between 80% and 100% of personal best No asthma management changes are necessary at this time 	 The movable indicator is placed at the beginning of the numbered scale. The patient stands or sits tall and straight. 		
 Yellow zone PEF values are between 50% and 80% of personal best Caution is warranted; use of medications is required 	 The patient then inhales maximally. The patient places his or her lips tightly around the mouthpiece. The patient is told to "blast" the air out of his or her lungs and into the device. The patient then forcefully exhales as fast and hard as 		
 Red zone PEF values are less than 50% of personal best Danger: emergency action is needed, including medications or hospital visit 	 6. The value is recorded. 7. Steps 1–6 are repeated 2 more times. 8. The highest number is marked as the value for that time period. 		
*Adapted with permission from Li JTC. $^{\rm 140}$ PEF indicates peak expiratory flow.	*For example, a Mini-Wright peak flow meter (Clement Clarke Interna- tional Ltd, Essex, UK). Adapted with permission from Li JTC. ¹⁴⁰		

Medication	Dosage Form	Adult Dose	Child Dose
Inhaled corticosteroids			
Systemic corticosteroids			
Methylprednisolone Prednisolone	2, 4, 8, 16, 32 mg tablets 5 mg tablets 5 mg/5 cc	 7.5–60 mg daily in a single dose in AM or qod as needed for con- trol 	 0.25–2 mg/kg daily in single dose in AM or qod as needed for con- trol
Prednisone	15 mg/5 cc 1, 2.5, 5, 10, 20, 50 mg tablets: 5 mg/cc, 5 mg/5cc	 Short-course "burst" to achieve control; 40–60 mg/d as single or 2 divided doses for 3–10 d 	 Short-course "burst": 1–2 mg/kg/ d, maximum 60 mg/day for 3– 10 days
Long-acting inhaled beta ₂ -a	agonists (Should not be used for sympto	om relief or for exacerbations. Use with i	nhaled corticosteroids.)
Salmeterol	MDI 21 mcg/puff DPI 50 mcg/blister	2 puffs q12h 1 blister q12h	1–2 puffs q12h 1 blister q12h
Formoterol	DPI 12 mcg/single-use capsule	1 capsule q12 h	1 capsule q12h
Combined medication			
Fluticasone/salmeterol	DPI 100, 250, or 500 mcg/50 mcg	1 inhalation bid; dose depends on severity of asthma	1 inhalation bid; dose depends on severity of asthma
Cromolyn and nedocromil			
Cromolyn	MDI 1 mg/puff	2–4 puffs tid–gid	1–2 puffs tid–gid
Nedocromil	Nebulizer 20 mg/ampule MDI 1.75 mg/puff	1 ampule tid–qid 2–4 puffs bid–qid	1 ampule tid–qid 1–2 puffs bid-qid
Leukotriene modifiers			
Montelukast	4 or 5 mg chewable tablet 10 mg tablet	10 mg qhs	4 mg qhs (2–5 yrs) 5 mg qhs (6–14 yrs) 10 mg qhs (>14 yrs)
Zafirlukast Zileuton	10 or 20 mg tablet 300 or 600 mg tablet	40 mg daily (20 mg tablet bid) 2,400 mg daily (give tablets qid)	20 mg daily (7–11 yrs) (10 mg tablet bid)
Methylxanthines (Serum m	nonitoring is important [serum concentra	tion of 5–15 mcg/mL at steady state])	
Theophylline	Liquids, sustained-release tablets, and capsules	Starting dose 10 mg/kg/d up to 300 mg max; usual max 800 mg/d	 Starting dose 10 mg/kg/d; usual max: <1 year of age: 0.2 (age in wk) +5 = mg/kg/d
			• \geq 1 year of age: 16 mg/kg/d

*qod indicates every other day; bid, twice a day; tid, 3 times a day; qid, 4 times a day; qhs, at bedtime; MDI, metered dose inhaler; DPI, dry powder inhaler.

the reversibility test with a β_2 -agonist is equivocal, then a challenge test (eg, with methacholine) may be performed to test for bronchial hyperresponsiveness.¹⁹ During these tests, progressively increasing concentrations of the aerosolized drug are administered during a multistage procedure.¹⁹ After each stage, spirometry testing is performed to determine whether a 20% reduction in FEV₁ from baseline is obtained. If the reduction is less than 20% after all stages have been performed, then the test is considered negative, and the patient is determined not to have bronchial hyperresponsiveness. It is important to note, however, that these tests alone are not diagnostic of asthma. A positive test must be interpreted in the context of other information to make the definitive diagnosis of asthma.

Exercise challenge and other surrogate challenges (such as eucapnic hyperventilation) are described in the EIA section of this statement.^{19,21}

Pharmacotherapy for Asthma

It is important to ascertain the correct diagnosis before medications are prescribed and for the health care professional to know the types of medications that are prescribed.^{1,2,24–30,166,167} Only a small percentage of asthmatic patients take their medications precisely as prescribed by their physician; the most common cause of treatment failure is failure to use the prescribed treatment.¹⁶⁷ Regardless, asthma can be managed through various medications to prevent or control symptoms; for an updated medication list, refer to the NAEPPII and GINA guidelines.^{1,2} The medications used to treat asthma are classified as either controller or rescue (reliever) medications.

Controller Medications. Controller medications are daily, long-term interventions used prophylactically to manage the symptoms of mild, moderate, and severe asthma and generally should not be used to manage acute asthmatic symptoms.^{1,2,25–28} Examples include inhaled corticosteroids, systemic corticosteroids, cromones (sodium cromolyn and nedocromil sodium), long-acting inhaled β_2 -agonists, theophylline, and leukotriene modifiers (Table 4 provides sample agents).

Inhaled corticosteroids are effective controller medications for treating persistent mild, moderate, and severe asthma.^{1,2} They act by decreasing airway inflammation, mucus production, and bronchial hyperresponsiveness.^{168–173} Proper use of inhaled corticosteroids can lead to a decrease in the number and severity of asthma exacerbations, improve lung function, lessen bronchial hyperresponsiveness, and reduce the need for symptom relief with short-acting β_2 -agonists.^{174–180} Inhaled corticosteroids should not be used to treat acute asthmatic attacks.²⁵ Their adverse effects include hoarseness, coughing, and occasionally thrush or oral candidiasis.^{177,181} However, rinsing the mouth after inhalation and using a spacer generally help to prevent oral candidiasis.

Systemic corticosteroids are administered orally or parenterally for individuals who have severe persistent asthma that remains poorly responsive to inhaler therapy.¹⁸² Systemic therapy has the same mechanisms of action as inhaled corticosteroids. However, long-term use of systemic agents can cause more significant systemic adverse events than inhaled corticosteroids, including osteoporosis, glucose intolerance, glaucoma, weight gain, skin thinning, bruising, fluid and electrolyte abnormalities, growth suppression, and muscle weakness.^{1,2,183} To minimize these adverse events, oral corticosteroids are often taken daily in the morning or every other day, and patients should be monitored closely by a physician.¹⁸³ Oral corticosteroids are used much less frequently today than in the past since the advent of high-potency inhaled corticosteroids such as fluticasone and budesonide.^{1,2}

The cromones, cromolyn sodium and nedocromil sodium, are inhaled asthma medications used to control mild persistent asthma and are considered less effective anti-inflammatory agents than inhaled corticosteroids.^{25,26,184} Although the exact mechanisms of action are poorly understood, they are thought to inhibit IgE-mediated mediator release from mast cells and, thus, to inhibit acute airflow limitations induced by exercise, cold air, and allergens. The cromones are generally used to treat mild persistent asthma and to prevent EIA.^{1,2} Cromones should be used as second-line drug therapy alternatives for treating mild persistent asthma, perhaps combined with an inhaled β_2 -agonist; however, several doses each day are usually needed to control asthma.^{1,2} Only minimal adverse events are seen with these agents, including occasional coughing and an unpleasant taste, particularly with nedocromil sodium.

Long-acting inhaled β_2 -agonists (eg, salmeterol and formoterol) have the same mechanisms of action as short-acting β_2 -agonists but a 12-hour duration of action, compared with 4 to 6 hours for the short-acting agents.^{185–190} A single dose of formoterol or salmeterol before exercise can protect the athlete from asthma symptoms associated with exercise for up to 12 hours.^{191–193} Formoterol has a shorter onset of action than salmeterol, approximately 5 minutes as compared with 15 to 30 minutes.^{192,194} All β_2 -agonists, including formoterol and salmeterol, are restricted asthma medications according to the International Olympic Committee, World Anti-Doping Agency, and United States Anti-Doping Agency; elite athletes taking these medications, their physicians, and their athletic trainers should review the guidelines posted by these agencies.

Patients using long-acting β_2 -agonists regularly may display a decrease in the duration of action.^{195–197} In one study, formoterol had a shortened duration of action by day 14 of regular daily use.¹⁹⁸ Thus, patients should not expect these drugs to remain effective over the 12-hour dosing interval after regular, daily, extended use. Studies also show that the use of long-acting β_2 -agonists does not affect persistent airway inflammation.¹⁹⁹ These agents should only be used in combination with inhaled corticosteroids, which may be more beneficial than ingesting each drug separately.^{200–202} Combination therapy (an inhaled, long-acting β_2 -agonist and an inhaled corticosteroid) has been shown to decrease the need for shortacting β_2 -agonists, decrease nocturnal asthma, improve lung function, decrease asthma exacerbations, and prevent EIA.^{200,202–205}

Leukotriene modifiers, taken orally, block leukotriene syn-

thesis or block leukotriene receptors.²⁰⁶⁻²⁰⁸ Leukotriene modifiers can be used to control allergen-, aspirin-, and exerciseinduced bronchoconstriction; improve lung function; and decrease asthma exacerbations.²⁰⁹⁻²¹⁵ Used primarily as secondline therapy, leukotriene modifiers can reduce the dose of an inhaled corticosteroid required to treat mild persistent asthma and may improve asthma control.^{214–217} Adverse events are usually minimal, with reports of headaches and gastrointestinal discomfort. However, zileuton (Zyflo, Abbott Laboratories, Abbott Park, IL) may be associated with liver toxicity; therefore, liver function should be monitored regularly when using this medication.²¹⁸ Unlike β_2 -agonists, the duration of action of the leukotriene modifiers does not diminish over time.²¹⁵ In the past, theophylline has been used alone as a controller agent, but now it is usually used in combination with another agent, such as an inhaled corticosteroid.219

Finally, some patients who have allergic asthma may benefit over the long term from the administration of various forms of allergy immunotherapy.^{220–222} The decision to initiate such therapy must be made by the patient and physician after a careful evaluation. Even in the most successful cases, additional medical therapy is often required in conjunction with immunotherapy.

Rescue (Reliever) Medications. Rescue medications act rapidly to treat acute bronchoconstriction and associated symptoms of coughing, wheezing, shortness of breath (dyspnea), and chest tightness.^{1,2,25–28} Several classes of drugs act in this manner, including rapid-acting inhaled β_2 -agonists, inhaled anticholinergics, and short-acting theophylline.

Rapid-acting inhaled β_2 -agonists are the most commonly used reliever therapy for chronic asthma. These β_2 -agonists act quickly to cause bronchodilation by relaxing airway smooth muscle, decreasing vascular permeability, and modifying mediator release from mast cells.¹⁸⁵ Rapid-acting inhaled β_2 -agonists are also the most frequently used agents to prevent EIA and to treat its symptoms.²²³ These medications can be used as "rescue therapy" at times of an acute attack. However, because of their relatively short duration of action (2–4 hours), repeat treatments may be necessary for EIA. Some authors^{224,225} suggest that repeated use of short-acting β_2 -agonists can lead to tolerance and less effectiveness over time. Furthermore, the chronic use of long-acting inhaled β_2 agonists can decrease the effectiveness of the short-acting inhaled β_2 -agonists.^{189,225}

Inhaled anticholinergic agents (eg, ipratropium) may be used as bronchodilators.²²⁶ These agents block acetylcholine release from cholinergic innervation in airway smooth muscle but have no anti-inflammatory action.

Short-acting theophylline is a bronchodilator that has been used for many years to relieve asthma exacerbations.^{29,227} Theophylline's onset of action is delayed when compared with that of β_2 -agonists, and so it is not currently used as first-line rescue therapy.^{1,2} Theophylline is a controversial drug because its benefits might be outweighed by potential adverse events such as seizures.²²⁸ Adverse events can be serious and severe if dosing is not closely monitored. Short-acting theophylline should not be administered to patients who are already receiving chronic therapy with sustained-release theophylline therapy.

Short-acting oral β_2 -agonists, although rarely used, function primarily by relaxing airway smooth muscle within a few minutes after administration and for a period of up to 4 hours; however, they have no anti-inflammatory actions.^{24,29} Adverse events include tachycardia, hypertension, and decreased appetite. If used chronically, increasing doses of short-acting oral β_2 -agonists might indicate loss of control of asthma.^{1,2} All athletes using short-acting oral β_2 -agonists should be advised that many sporting organizations restrict or prohibit the use of these agents.

Finally, systemic glucocorticosteriods are administered orally or parenterally for individuals who have acute asthma exacerbations. The mechanisms of action are similar to those for corticosteroids used to treat chronic severe asthma.^{1,2}

Asthma Medication Delivery Techniques

Many of the newer asthma medications are delivered to the lungs by inhalation devices.²³ The most common types of inhalers are MDIs and dry powder inhaler devices. The MDIs release a specific amount of a drug from a pressurized canister to propel medication into the lungs when the patient takes a breath. When using MDIs, patients must exhale first, then place the inhaler at or slightly in front of the lips, and slowly inhale at the same time that they are activating the inhaler to release the drug. Patients hold their breath for a few seconds (approximately 10) before exhaling. Patients who have difficulty coordinating MDI activation with breathing generally benefit from the use of a spacer.^{229–231} A spacer is attached to the MDI device to reduce side effects of inhaled corticosteroids in the mouth and for patients who have difficulty coordinating the activation of an MDI and breathing. Dry powder inhalers are often easier to use than MDIs, and they do not permit use of a spacer.

Aspirin, Nonsteroidal Anti-Inflammatory Drugs, and Asthma

Aspirin-sensitive athletes may manifest nasal congestion; itchy, watery, or swollen eyes; coughing; difficulty breathing; wheezing; urticaria; and possible shock when they ingest aspirin or other NSAIDs.^{232–236} This is not a true allergy because it is not caused by IgE, but it is treated in the same manner as an allergic reaction. Athletic trainers should also be aware of triad syndrome: athletes with asthma, nasal polyps, and aspirin sensitivity may have a severe asthma attack when they take an NSAID.²³⁷

Although only a small percentage of the population has aspirin-sensitive asthma,^{238–240} this condition is particularly concerning in an athletic population because many athletes who have asthma use anti-inflammatory drugs to treat injuries. Therefore, athletic trainers must understand that some patients who have asthma could suffer fatal consequences if they take aspirin or NSAIDs.

Aspirin-sensitive athletes should also avoid COX-2 inhibitors, but acetaminophen in moderate doses can usually be taken without difficulty.²⁴¹ Salsalate, choline magnesium trisalicylate, and dextropropoxyphene may be used as substitute medication in patients with aspirin sensitivity if tolerated.^{240,242} Athletic trainers should be familiar with the many prescription and over-the-counter products that contain aspirin and other NSAIDs, including ibuprofen (eg, Motrin, McNeil-PPC, Fort Washington, PA; Advil, Wyeth, Madison, NJ) and naproxen (eg, Aleve, Bayer, Morristown, NJ). Health care professionals should supply as much information to the patient as possible, including a list of products to avoid (Table 5).

Table 5. Sample Nonsteroidal Anti-Inflamn

Generic	Brand	
Name	Name(s)	Manufacturer
Diclofenac, miso- prostol	Arthrotec	GD Searle LLC, Chicago, IL
Celecoxib	Celebrex	GD Searle LLC
Diclofenac	Voltaren	Novartis Pharmaceuticals Corp, East Hanover, NJ
Diclofenac	Cataflam	Novartis Pharmaceuticals Corp
Diflunisal	Dolobid	Merck & Co, Inc, West Point, PA
Etodolac	Lodine	Wyeth-Ayerst International Inc, Madison, NJ
Flurbiprofen	Ansaid	Pharmacia & Upjohn Co, Kalama- zoo, MI
Ibuprofen	Motrin	McNeil-PPC, Fort Washington, PA
	Advil	Wyeth, Madison, NJ
Indomethacin	Indocin	Merck & Co, Inc
Ketoprofen	Orudis	Rhône-Poulenc Rorer New Zealan Ltd, Auckland, New Zealand
Ketorolac	Toradol	Roche Pharmaceuticals, Nutley, N
Nabumetone	Relafen	GlaxoSmithKline, Research Trian- gle Park, NC
Naproxen	Naprosyn	Roche Pharmaceuticals
	Aleve	Bayer, Morristown, NJ
Oxaprozin	Daypro	GD Searle LLC
Piroxicam	Feldene	Pfizer Inc, New York, NY
Rofecoxib	Vioxx	Merck & Co, Inc
Salsalate	Disalcid	3M Pharmaceuticals, Northridge, CA
Sulindac	Clinoril	Merck & Co, Inc

Nonpharmacologic Treatment for Asthma

Athletes with asthma need to keep their asthma under optimal control to prevent exercise-induced breathing symptoms.^{31,32,243,244} Masks and nose breathing help to warm and moisturize inhaled air before it reaches the smaller airways. This may decrease the inflammatory reaction in the airways and thus decrease the frequency and intensity of EIA. These maneuvers are effective for some but not all athletes.²²⁴ Nose breathing is not effective at high ventilation rates. Limiting environmental exposures (eg, to cold air and pollen) may decrease symptoms in susceptible athletes; however, this may not be practical in some sports.

Theoretically, exercise training might decrease symptoms by conditioning the body to exercise, but research has not supported this theory.^{245,246} Nevertheless, an asthmatic individual should participate in exercise programs tailored to his or her capacity to perform.⁵⁵

A refractory period can occur after exercise, when the airway response to exercise is inhibited for up to 2 to 3 hours. Some athletes have taken advantage of this phenomenon to help control EIA.^{31,32,34,247–251} However, there are no specific guidelines to follow, and each athlete must experiment to determine the best individual protocol.

Because hyperosmolarity plays a role in mediating EIA, limiting sodium in the diet has received some attention. Restricting dietary salt may cause a relative decrease in airway obstruction.^{252,253} Both sodium and chloride appear to play roles, but this remains an area of active investigation, and no specific guidelines are available.²⁵⁴ A diet supplemented with n-3 polyunsaturated fatty acid in fish oil has shown favorable results in elite athletes with EIA.²⁵⁵

Exercise-Induced Asthma or Bronchospasm

Most asthmatic individuals have a flare of their asthma after exercise.^{256,257} Some individuals only have asthma signs and symptoms associated with exercise.¹³ By definition, a temporary narrowing of the airways (bronchospasm) induced by strenuous exercise in which the patient has no symptoms is known as EIB.¹³ When symptoms are present, EIB is described as EIA.¹³ This section reviews the incidence of EIA and EIB in the athletic population and considers special diagnostic or therapeutic measures that should be taken in an athletic population.

Exercise-induced asthma is commonly seen in athletes in all levels of athletic competition.^{5,9,10,31,243,244,258–269} In most patients who have chronic asthma (at least 80%), exercise is a trigger for bronchoconstriction.^{13,243} Exercise-induced asthma can also occur in patients who do not otherwise have asthma, such as in about 40% of patients who suffer from allergic rhinitis in season.^{243,270,271} The incidence of EIA in the general population has been estimated to be between 12% and 15%.^{13,271} Rates as high as 23% have been reported in schoolage children, and the incidence in athletes may also be this high.^{258,262,263,265} Exercise-induced asthma may be more common in urban environments than in rural areas.²⁶¹ Other factors, such as high ozone levels, might also account for increased EIA rates.²⁷²

Exercise-induced asthma can be a significant disability for the athlete, especially in endurance sports.^{262,263,273} For example, EIA is relatively common in cross-country skiers, and some studies suggest that the cold air athletes breathe while cross-country skiing may provoke inflammation.^{274,275} Similarly, athletes who participate in swimming and long-distance running have a high incidence of asthma.²⁶² Among Olympic athletes, asthma appears to be more common in those who participate in winter sports than in those who participate in summer sports.^{262,263} At least 1 in 5 United States athletes who participated in the 1998 Winter Olympics had the condition, compared with 1 in 6 at the 1996 Summer Olympic Games.^{262,263} Wilber et al²⁶⁵ found a 23% overall incidence of EIA among athletes in the 7 winter sports tested. In addition, more females than males participating in the Winter Games reported an asthma condition or medication use.^{262,263,265} Of the winter sports athletes tested, females had an incidence of 26%, compared with 18% in males.²⁶⁵

Although EIA impairs performance, it can be overcome. Amy Van Dyken, an athlete who suffered from relatively severe asthma, won 4 gold medals in swimming in the 1996 Olympic Games.²⁷³ Other well-known elite athletes have also been able to excel when their asthma was under good control.²⁷³

Pathophysiology of Exercise-Induced Asthma

Two major theories exist to explain EIA: the cooling/warming hypothesis and the drying hypothesis.^{31,256,257,260,276–287} As ventilation increases, airways progressively cool, which results in bronchoconstriction. This theory is supported by the higher incidence of EIA in athletes participating in cold environments.²⁶³ In addition to cooling, the increased ventilation can lead to airway dehydration as inhaled air is humidified. The main effect of inhaling cold air is actually attributable to the fact that cold air carries less moisture. As with chronic asthma, inflammatory cells and mediators may increase in the lung in response to exercise in patients with EIA.^{248,279,288–290} Environmental allergens may enhance the likelihood of bronchoconstriction, and irritants such as sulfur dioxide, nitrogen dioxide, ozone, and chlorine have been implicated as causing patients to have exercise-induced symptoms.^{31,243,291}

Exercise-Induced Asthma Diagnosis

Two requirements are needed to diagnose EIA: symptoms and obstructed airways, both associated with exercise.^{13,269}

First, the patient has any of a constellation of symptoms associated with exercise, including shortness of breath (dyspnea), coughing, chest tightness (or chest pain in children), wheezing, and decreased exercise tolerance.^{13,262,263} Symptoms generally occur 5 to 8 minutes after sufficiently intense exercise starts. The EIA may be associated with specific sports as well as specific environments.^{262–267} Where allergens are present, outdoor activities and cold air exposure may be more likely to foster the appearance of EIA, which would not occur in other environments.

Second, the patient should have objective evidence of airway obstruction associated with exercise.²²⁴ Generally, a drop from baseline of at least 10% to 15% in FEV₁ after a challenge test supports the diagnosis of EIA.^{19,292} Pulmonary function should be monitored 5, 10, 15, and 30 minutes after the challenge.¹⁹ The exercise needs to last for 6 to 8 minutes at an intensity level high enough to raise the athlete's heart rate to at least 80% of maximum¹⁹ and ventilation to approximately 40% to 60% of maximum.¹⁹ Exercise challenges can be performed in a laboratory (using a treadmill, a cycle ergometer. a rowing machine, or a free running asthma screening test [FRAST]).²⁹³ Alternatively, an exercise challenge test in the laboratory can attempt to mimic the conditions and intensity of the sport.^{20,21} Indeed, 78% of cross-country skiers reported a false-negative test during standard laboratory exercise challenges, suggesting that the standard tests are not as sensitive as sport-specific exercise challenge tests for endurance athletes.^{20,21} Cold, dry air and near-maximal exercise intensity (greater than 90% peak heart rate) are required to provoke a positive result, especially in the cold-weather athlete. Time of day can be important: in a group of asthmatics, a greater drop in pulmonary function (FEV₁) to exercise challenge was measured in the evening than in the morning.²⁹⁴

Additional challenge tests include eucapnic voluntary hyperventilation or inhalation of hypertonic saline.^{21,269,295} The former test requires the athlete to hyperventilate dry air containing 5% carbon dioxide, 21% oxygen, and the balance of nitrogen at 30 times FEV_1 for 6 minutes.²¹

It is important to evaluate athletes with atypical EIA symptoms because upper airway conditions such as vocal cord dysfunction or abnormal movement of the arytenoids region may be the cause.^{15,53} The signs and symptoms of vocal cord dysfunction can be similar to asthma and can be confused with EIA.^{54,296,297} This larvngeal disorder involves the unintentional paradoxic adduction of the vocal cords with breathing and can be triggered by exercise.^{296,297} The patient is often female and may also have gastroesophageal reflux disease or a psychiatric illness. Vocal cord dysfunction often occurs with asthma, making control of EIA difficult. Diagnosis of vocal cord dysfunction involves the direct visualization of the paradoxic vocal cord motion, but the condition is often suspected when voice changes and inspiratory stridor occur during an attack, as well as when the inspiratory (bottom) portion of the flow volume loop is truncated.

One goal of management is to enable patients to participate in any activity they choose without experiencing asthma symptoms. EIB should not limit either participation or success in vigorous activities.

Recommended treatments include:

- Beta₂-agonists will prevent EIB in more than 80 percent of patients.
 - Short-acting inhaled beta₂-agonists used shortly before exercise (or as close to exercise as possible) may be helpful for 2 to 3 hours.
 - Salmeterol has been shown to prevent EIB for 10 to 12 hours (Kemp et al 1994³⁰¹).
- Cromolyn and nedocromil, taken shortly before exercise, are also acceptable for preventing EIB.
- A lengthy warmup period before exercise may benefit patients who can tolerate continuous exercise with minimal symptoms. The warmup may preclude a need for repeated medications.
- $\circ\,$ Long-term–control therapy, if appropriate.
 - There is evidence that appropriate long-term control of asthma with anti-inflammatory medication will reduce airway responsiveness, and this is associated with a reduction in the frequency and severity of EIB (Vathenen et al, 1991³⁰²).

Teachers and coaches need to be notified that a child has EIB, should be able to participate in activities, and may need inhaled medication before activity. Individuals involved in competitive athletics need to be aware that their medication use should be disclosed and should adhere to standards set by the U.S. Olympic Committee (Nastasi et al, 1995³⁰³). The U.S. Olympic Committee's Drug Control Hotline is 1-800-233-0393.

EIB indicates exercise-induced bronchospasm.

Exercise-Induced Asthma Treatment

Most of the drugs described for the treatment of chronic asthma are used to prevent EIA attacks.^{1,2,269,298–300} Table 6 contains recommendations from the NAEPPII for the treatment of EIA.¹ The key feature is that a β_2 -agonist can be used both to prevent attacks and to treat them when they occur. Once an asthmatic individual meets the requirements for stage 1 through 4 asthma, the NAEPPII treatment guidelines should be followed.

Asthma Education

Throughout this position statement, information has been presented to inform and educate the athletic trainer and allied health personnel about asthma and asthma management. Of particular importance is a properly prepared asthma management plan. Educating athletes about asthma and having a written management plan will help control their disease.^{1,2,304} Several groups^{35–44,48–51} have shown that an effectively written management plan can reduce medication errors, asthma exacerbations, and hospital visits. Without a written asthma action plan, many patients have a difficult time controlling their asthma symptoms.^{45–47,305} It is also imperative that an accessible line of communication between the patient and health care professional be identified.

An effective management plan should include a written document that addresses the following: (1) goals of the patient, (2) proper use and frequency of PEFR monitoring, (3) guidelines for altering medications based upon readings from PFMs or asthma symptoms, (4) contact numbers for all health care professionals, including emergency numbers, and (5) environmental factors to avoid or monitor. The health care professionals developing the asthma management plan should dis-

Table 7. United States Anti-Doping Agency Regulated Asthma Medications

Drug class: β_2 -agonists:
Advair* (GlaxoSmithKline, Research Triangle Park, NC)
albuterol*
bambuterol bitolterol
Brethaire* (Riker Laboratories, Inc, Northridge, CA)
Combivent* (Boehringer Ingelheim Pharmaceuticals Inc, Ridgefield
(CT)
fenoterol
Foradil* (Novartis Pharmaceuticals Corp, East Hanover, NJ)
formoterol*
metaproterenol
orciprenaline
pirbuterol
Proventil* (Schering-Plough Corp, Kenilworth, NJ)
reproterol
salbutamol*
salmeterol*
Serevent* (GlaxoSmithKline)
terbutaline*
Ventolin* (GlaxoSmithKline)
Xopenex* (Sepracor, Marlborough, MA)

Available at: http://www.usantidoping.org. Accessed June 6, 2005. *Allowed by inhaler or nebulizer only to prevent or treat asthma or exercise-induced asthma. Abbreviated Therapeutic Use Exemption (TUE) must be on file with United States Anti-Doping Agency or international federations, as appropriate. A salbutamol (albuterol) level greater than 1000 ng/mL is prohibited even with abbreviated TUE.

cuss all goals or expectations with the athlete. This education empowers the athlete and promotes better compliance.

Athletic trainers working with elite or Olympic athletes must be familiar with International Olympic Committee, World Anti-Doping Agency, and United States Anti-Doping Agency medication guidelines (Table 7). Certain asthma medications may be banned, restricted, or permitted, depending on the organization and the medication. A banned medication is one the athlete cannot take. In some cases, a prohibited substance is prohibited at all times or only prohibited in competition, meaning the athlete must allow sufficient time for the substance to clear the system before competition. Restricted medications must have prior physician approval and completion of forms (such as the Therapeutic Use Exemption, or TUE) before the athlete can compete. For example, the United States Anti-Doping Agency lists salbutamol/albuterol, salmeterol, terbutaline, and formoterol as restricted β_2 -agonists that require a TUE before competition.

Additional information about the diagnosis and management of asthma can be obtained at the National Asthma Education and Prevention Program Web site (http://www.nhlbi.nih.gov/ health/prof/lung/index.htm or http://www.nhlbi.nih.gov/about/ naepp/) and the GINA Web site (www.ginasthma.com).

CONCLUSIONS

Asthma can affect individuals regardless of age, sex, or socioeconomic status. This National Athletic Trainers' Association asthma position statement is designed to present guidelines for the recognition, prophylaxis, and management of asthma. The information should lead to improvements in the quality of care certified athletic trainers and other heath care providers can offer to patients with asthma, particularly athletes and especially those with EIA. In the end, these guidelines should reduce the incidence of asthma complications and improve the quality of life for patients with asthma, especially those in whom exercise is an important trigger.

DISCLAIMER

The NATA publishes its position statements as a service to promote the awareness of certain issues to its members. The information contained in the position statement is neither exhaustive not exclusive to all circumstances or individuals. Variables such as institutional human resource guidelines, state or federal statutes, rules, or regulations, as well as regional environmental conditions, may impact the relevance and implementation of these recommendations. The NATA advises its members and others to carefully and independently consider each of the recommendations (including the applicability of same to any particular circumstance or individual). The position statement should not be relied upon as an independent basis for care but rather as a resource available to NATA members or others. Moreover, no opinion is expressed herein regarding the quality of care that adheres to or differs from NATA's position statements. The NATA reserves the right to rescind or modify its position statements at any time.

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