Review of Exercise-Induced Asthma

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ABSTRACT

STORMS, W. W. Review of Exercise-Induced Asthma. Med. Sci. Sports Exerc., Vol. 35, No. 9, pp. 1464–1470, 2003. Purpose: The purpose of this manuscript is to review the recent literature on exercise-induced asthma (EIA) and summarize the pathogenesis, diagnosis, and treatment of this condition. Method: A review of the English language medical literature was performed to obtain articles on EIA. Results: The pathophysiology of EIA is not fully understood, but there are two theories: 1) the hyperosmolar theory and 2) the airway rewarming theory. In addition, there have been data to show that airway inflammation is present in some elite athletes, especially in cold weather sports. The diagnosis of EIA is usually straightforward in most patients, but a number of patients may have atypical symptoms and may be more difficult to diagnose. They may well need exercise testing or eucapnic voluntary ventilation testing. Most people respond to treatment with an inhaled beta agonist and or cromolyn before exercise, but some patients will also need other medications, including daily medications such as inhaled steroids. When treatment does not control the problem, then further diagnostic evaluation should be done to rule out conditions other than EIA, such as vocal cord dysfunction or cardiac or pulmonary problems. Conclusions: EIA is a condition that may occur in schoolchildren in gym class and also in Olympic athletes. The diagnosis and treatment is usually fairly straightforward, but at times it may be challenging. However, all patients should be followed to make sure that the correct diagnosis is made and to make sure that treatment is effective. Key Words: EXERTIONAL DYSPNEA, EXERCISE-INDUCED BRONCHOSPASM, SPORTS-INDUCED ASTHMA, ASTHMA IN SPORTS, EXERCISE-INDUCED ASTHMA, ASTHMA

Exercise-induced asthma (EIA) is a condition that may affect persons at any level of exercise, from children to Olympic athletes (24). There are many antiasthmatic medications in the past few years that have led to better control of asthma and have allowed many patients with EIA to participate in athletic endeavors. There have been some new products for asthma in recent years that can be used in EIA, and these will be discussed. The goal of this review is to provide information to healthcare professionals, certified athletic trainers, coaches, and athletes so that EIA can be recognized and patients can be given proper therapy to allow them to compete at any level, whether they are recreational or elite athletes.

WHAT IS EIA?

EIA is a condition characterized by symptoms of coughing, wheezing, shortness of breath, and chest tightness during or after exercise, and associated with airway obstruction after exercise, as noted by a drop in FEV₁ or other spirometric parameters (24). The symptoms are not always straightforward; patients may present with cramps, stomach pain, headache, etc.; these are discussed in the section on diagnosis. EIA can be seen in subjects at any level of exercise, from children in gym class to elite Olympic athletes.

There are two descriptions commonly used, exercise-induced bronchospasm (EIB) and EIA or exercise induced asthma. EIB is the bronchial obstruction (“spasm”) found after exercise in persons with normal lung function at rest. EIA is sometimes used to describe the exacerbation of a patient’s asthma when they exercise. For this article, EIA will encompass both of these definitions. Airway hyperreactivity (AhR) is used to describe the hyperresponsive to stimuli that is seen in patients with asthma. This reactivity may be seen with cold air inhalation, pollen, mold, or animal dander exposure, upper respiratory infections, and laboratory testing such as methacholine inhalation. In patients with asthma, it is felt that airway inflammation is the primary underlying disease process, even when patients are asymptomatic. This inflammation is associated with airway hyperreactivity. The underlying inflammation in asthma determines the need for daily preventative anti-inflammatory controller medications, taken on a daily basis to forestall worsening asthma. If the inflammation goes unchecked,
there is a possibility that airway remodeling may occur in the lungs; this might lead to chronic irreversible airways obstruction.

Some patients with EIA only have symptoms with exercise and have no signs or symptoms of asthma at rest, and they have a normal physical examination and spirometry. It is only with exercise that these patients have respiratory symptoms and a drop in pulmonary function after exercise. They have EIA with no evidence of underlying chronic asthma.

Other patients may have chronic asthma, as evidenced by reduced lung function at rest and they will usually have exercise-related increase in symptoms and reduction in lung function. These individuals may also have symptoms of asthma when exposed to pollens, animal danders, or with an upper respiratory infection. In other words, there is chronic asthma present that can be triggered by other stimuli than exercise. In all likelihood, these individuals need daily medication for chronic asthma plus preexercise medication for the exercise-induced exacerbation.

**EIA in elite athletes.** Up to 50% of cold weather athletes will have respiratory symptoms after exercise and a drop in FEV$_1$ that is compatible with exercise-induced asthma (see prevalence section). Many of these athletes have neither prior history of asthma nor a family history of asthma (24,25,40). They can exercise in warm air without a drop in FEV$_1$. In these athletes, there may be some chronic airway injury because of the inhalation of large volumes of cold air during training, which leads to findings that mimic true asthma. Sue-Chu (39) has studied cross-country skiers with symptoms of exercise asthma and found evidence of airway inflammation but no beneficial effect of treatment with inhaled steroids. This study suggests that there may be a condition that occurs in cold weather athletes that is different than asthma and these subjects may not respond to the usual therapy. Support for this concept comes from Wilber (48), who studied elite speed skaters and found that the use of short-acting beta 2 agonists did not predictably prevent the fall in FEV$_1$ after exercise.

**WHAT IS THE MECHANISM OF EIA? HYPEROSMOLARITY THEORY AND AIRWAY REWARMING**

Chronic asthma and EIA may be caused by different mechanisms; each of these will be discussed. Chronic asthma is considered a disease of the airways of the lungs characterized by: 1) inflammation of the bronchial mucosa and submucosa; 2) hyperresponsiveness to various inhaled stimuliants (methacholine, allergens, cold air, etc.); and 3) airway narrowing (31). The mechanisms that underlie EIA are not totally understood; there are two current theories on the pathogenesis of EIA (2). The hyperosmolarity theory (3) proposes that water loss from the airway surface liquid during exercise leads to hypertonicity of the airway surface liquid and a hyperosmolar condition within the airway cell. This could lead to release of pro-inflammatory mediators that lead to bronchoconstriction; these mediators include histamine, prostaglandins, chemotactic factors, leukotrienes, etc., and the potential for chronic airway damage through inflammation and remodeling. When inflammation is present in the airways this may amplify the process because water loss may be facilitated by this inflammation. The airway rewarming theory (26) is the other theory for the mechanism of EIA. This states that the hyperventilation of exercise leads to cooling of the surface cells in the airway. After exercise, there is a rewarming process that causes a dilatation of the small bronchiolar vessels around the airways, and this leads to hyperemia of the airway lining, fluid exudation from the blood vessels into the submucosa of the airway wall and subsequent mediator release and bronchoconstriction. Neither of these theories considers inflammation as the basic underlying mechanism, as it is in chronic asthma, but there have been recent data to show that inflammation is present in cold weather athletes, as described below.

**WHAT IS THE MECHANISM OF EIA? INFLAMMATION IN EIA**

Asthma is well recognized as a disease of inflammation of the airways (31). The inflammatory reaction starts with the mast cell releasing preformed mediators such as histamine and proteases; newly synthesized mediators that are released shortly afterward include cysteinyl leukotrienes, bradykinin, prostaglandins, platelet activating factor, interleukins, and other mediators. At the same time, chemotactic factors are released, which attract various cells into the area of inflammation. These cells include eosinophils, monocytes, lymphocytes, and basophils. These cells subsequently release other mediators that amplify the inflammatory response, including interleukins and other mediators. The cysteinyl leukotrienes have been of considerable interest recently because of the new products that block the leukotriene receptors, zafirlukast (Accolate) and montelukast (Singulair). Leukotrienes are generated from arachidonic acid that is found in cell membranes. The inflammatory reaction causes new generation of leukotrienes from arachidonic acid in the cell membrane of the mast cell and other cells. Based on these findings, leukotriene receptor antagonists have been developed and have been shown to be effective in asthma and EIA. There are currently no specific drugs that target other mediators such as proteases, interleukins, prostaglandins, etc. Because inflammation has been understood as the pathophysiological basis for chronic asthma, evidence for inflammation has been studied in EIA and similar mediators have been found. In two studies, Sue-Chu et al. (20,39) showed elevations in some cell types but not others. Karjalainen et al. (20) studied 40 cross-country skiers and performed endobronchial biopsies at rest. The skiers had higher airway T-lymphocytes, macrophages, and eosinophil counts than the control group. The skiers also had increased subepithelial basement membrane tenascin expression, a marker of airway remodeling. These authors concluded that cross-country skiers not only had symptoms and signs of
exercise-induced asthma postexercise, but they also had evidence of chronic airway inflammation that was caused by their constant exercise in cold air. The authors also found that some of their cross-country skiers had evidence of airway inflammation even though they did not have any EIA symptoms or airway hyperreactivity. Other authors have found evidence of inflammation using these and other markers of inflammation, but not all investigators agree. Gauvreau et al. (13) studied subjects with mild asthma in whom they performed exercise challenges. They found no change in the sputum inflammatory cells or blood inflammatory cells after exercise. Nitric oxide (NO) has been used as a marker of asthmatic inflammation and is measured in the exhaled air. Some studies (19,22,38,42) have shown that exhaled NO changes after exercise in patients with EIA and that this change is directly correlated with the drop in FEV\textsubscript{1} after exercise. This rise in NO in EIA patients has been correlated with the increased vascular permeability of the bronchial circulation during the hyperventilation of exercise. Adenosine is another inflammatory mediator of asthma that has been studied in EIA. Vizi et al. (45) showed increased levels of plasma adenosine after exercise in patients with asthma. Other studies have shown other inflammatory markers to be present in patients with EIA at rest and also after exercise. This suggests that EIA is more than just a set of independent events after exercise but rather that frequent exercise may lead to chronic inflammatory changes in the airways. This is an important concept when treatment is considered; daily antiinflammatory asthma medications for the inflammation may be required in addition to preexercise medications. There are other data supporting inflammation in EIA, coming from studies showing the presence of the late asthmatic response in some patients with EIA (7). There have not been many studies that look for the late response after exercise because it requires doing pulmonary function tests up to 12 h after exercise, but Chhabra and Ojha (7) have shown a 50% incidence of a late asthmatic response in exercise-induced asthma.

**HOW PREVALENT IS EIA?**

In recent years, studies have shown a significant prevalence of EIA in both recreational and elite athletes (Table 1). In winter Olympic athletes, the prevalence of EIA has been shown to be 17% (46). This is higher than one might expect, as it might be assumed that asthmatics would not be able to reach such an elite level of performance. However, this has been corroborated by many other studies showing that competitive and recreational athletes seem to have a higher prevalence of EIA than previously thought; some of these studies use symptoms questionnaires and others use pulmonary function testing to make the diagnosis. The preferred diagnostic method is sport-specific challenge testing with spirometry before and after exercise, but this is more tedious and is not always done. Hallstrand et al. (14) found 9% of school children had EIA; Rupp et al. (37) found 12% of school children had it. These studies were done in nonathletes, and in many cases, the findings were a surprise to the children and their parents. Competitive athletes in cold weather sports have a high incidence of EIA: 35% in figure skaters (25) and 35% in ice hockey players (23). A study by Wilber et al. (49) was particularly important because the subjects were tested with spirometry before and after exercise in a sport-specific challenge that was performed in the environment of that specific sport. For all athletes, they found the incidence of EIA was 23%, but with some sports such as cross-country skiing, it was as high as 50%. In the Salt Lake City Olympics in 2002, 14.6% of the cross-country skiers used beta agonists for EIA, a number that is comparable to other studies (4). In warm weather sports, EIA appears to be less prevalent: 14% in cross-country runners (43) and 16% in the 1996 Atlanta summer Olympics (47). Because of these data showing that even elite athletes have EIA, it is important for physicians, certified athletic trainers, coaches, and athletes to be knowledgeable about the symptoms of EIA and to make sure that the subject gets a full evaluation by a physician who is knowledgeable about this condition. Symptoms are not always typical, and the athlete should have an evaluation if there is any suggestion of EIA.

**HOW IS EXERCISE-INDUCED ASTHMA DIAGNOSED?**

The usual presenting symptoms of exercise-induced asthma include cough, wheezing, shortness of breath, or chest tightness during or after exercise (Table 2). However, this would assume that the individual is aware of these abnormal symptoms and will seek medical attention. Many people do not recognize these as symptoms but just think that they are out of shape and never seek medical advice unless someone urges them to do so. The example noted in Table 3 highlights the importance of having all persons dealing with athletes to be alert for any respiratory symptoms with exercise and bring them to the attention of the athlete so that the athlete may seek proper medical attention. There are other symptoms besides coughing, wheezing, and shortness of breath that may occur in some patients because of EIA, such as the subtle symptoms of “feeling out of shape,” headaches, abdominal pains, muscle cramps, fatigue, or dizziness, and these should always be considered (Table 3). Although symptoms are usually what triggers the

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**TABLE 1. Incidence of EIA.**

<table>
<thead>
<tr>
<th>Sport</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cross-country skiers</td>
<td>50</td>
</tr>
<tr>
<td>Ice hockey</td>
<td>35</td>
</tr>
<tr>
<td>Speed skaters</td>
<td>43</td>
</tr>
<tr>
<td>Figure skaters</td>
<td>35</td>
</tr>
<tr>
<td>Summer and winter Olympic athletes</td>
<td>17</td>
</tr>
<tr>
<td>School children</td>
<td>12</td>
</tr>
</tbody>
</table>

**TABLE 2. Symptoms of EIA.**

<table>
<thead>
<tr>
<th>Typical</th>
<th>Atypical</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cough: during or after exercise</td>
<td>Stomach cramps</td>
</tr>
<tr>
<td>Wheezing</td>
<td>Headache</td>
</tr>
<tr>
<td>Shortness of breath during or after exercise</td>
<td>&quot;Being out of shape&quot;</td>
</tr>
</tbody>
</table>

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athlete to seek medical attention, it has been shown that symptoms do not necessarily correlate well with the presence of EIA. Both Thole et al. (43) and Rundell et al. (34) showed that self-reported symptoms are a poor predictor for EIA, giving both false-positive and false-negative results.

There is some information in the patient history that supports the diagnosis of EIA and would increase the likelihood of this diagnosis, such as increased symptoms with cold air exposure, and with certain sports such as running, ice skating, and cross-country skiing. Also a family history of asthma or a personal history of recurrent allergic rhinitis or sinusitis increase the likelihood of EIA.

After a thorough history is taken, a physical exam should be performed including an ear, nose, and throat exam looking for signs of nasal allergies, sinusitis, or otitis; a cardiac exam to look for murmurs or arrhythmias; and a chest exam to evaluate for wheezing, rales, or rhonchi. In most patients with EIA, the chest exam will be totally normal with no evidence of any wheezes or other abnormal lung sounds. Any positive findings in the physical exam should be fully evaluated and treated including such things as otitis media, sinusitis, etc. The next step in diagnosis is spirometry and this can be done in the office setting. In the patient with pure EIA, the FEV₁, from the spirometry would be expected to be normal, or at least 90% of normal. If the FEV₁ is less than 90%, this may indicate the presence of undiagnosed chronic mild persistent asthma, and daily antiasthmatic therapy should be initiated. It is important to realize that EIA may not be easy to diagnose from the history and the physical exam; this means that continued follow-up is needed to make sure that the treatment is working. If treatment is not effective, then further evaluation is important.

CHALLENGE TESTING

Challenge testing is important in various circumstances: 1) to confirm the diagnosis of EIA when it is in doubt, 2) to screen athletes in sports with a high incidence of EIA, 3) for screening epidemiology studies to evaluate the incidence of EIA, or 4) to provide proof of EIA in Olympic athletes to allow them to take antiasthmatic medications. There are a number of different challenge tests that can be performed to confirm the diagnosis of EIA. The type of test chosen may be determined by availability and preference of the treating physician. Simple step-testing has been suggested if one does not have a treadmill or cycle ergometer (11), but the step-test or outdoor running or treadmill tests may not be sensitive enough to make the diagnosis. The most specific test is to test the athlete in the field in their sport. Rundell et al. (36) has shown that this is more specific than exercise testing in the laboratory, but the temperature and humidity cannot be controlled with exercise in the field. Because not everyone can exercise their patients in the field, other types of laboratory challenges have been evaluated to attempt to predict EIA. The eucapnic voluntary hyperventilation (EVH) challenge with dry air has been used to assess EIA in athletes (3,10). Another challenge test that has been used in asthma for many years is the methacholine inhalation challenge. It has been used to classify the severity of a patient’s asthma, and it has a long history in epidemiologic studies. The methacholine challenge does not predict responsiveness to EVH challenge and therefore is not used to diagnose EIA. Although the EVH test has been used widely and has been recommended by the IOC Medical Commission for testing of Olympic athletes with asthma it requires some expensive equipment and is a relatively complex test. Because of this, other tests have been developed, such as the inhaled mannitol test. Holzer et al. (16) assessed the response to inhaled mannitol in 27 summer elite athletes with a diagnosis of EIA. Twenty-five of the 27 subjects had a positive EVH test, 26 of the 27 subjects had a positive mannitol inhalation test, and 24 subjects were positive to both tests. The authors suggest that the mannitol inhalation challenge may be the best test to evaluate EIA because it is at least as sensitive as the EVH test and it is easier and less expensive to perform. It is also easier and less expensive to perform than the sport-specific field challenge.

TREATMENT OF EXERCISE-INDUCED ASTHMA

Once the diagnosis, or presumed diagnosis, of EIA is made, then treatment is recommended. Treatment of EIA first involves advising the athlete regarding the types of exercise that are less likely to cause asthma (see Tables 4 and 5). For some athletes, this is not practical because they are competing in a specific sport, but for many recreational athletes, the type of exercise might be changeable, and they might be able to avoid certain weather conditions or high pollen days (21,15) that could make them worse. In addition, a special warm-up routine has been shown to reduce the severity of EIA: the athlete should warm up to 80–90% of their maximum workload before the formal exercise. This had been shown to partially reduce the severity of the exercise asthma but probably will not completely block EIA (28).

| TABLE 4. Factors most likely to cause EIA. |
| Continuous hard exercise, such as running |
| Exercise in cold environment |
| Exercise in polluted air, either indoor (pool, skating rink) or outdoor |
| Exercise during the pollen season for allergic athletes |
| Exercise during an upper or lower respiratory infection |

| TABLE 5. Factors least likely to cause EIA. |
| Intermittent exercise or team games |
| Swimming |
| Exercise in warm, humid air |
| Exercise outside of the pollen season in the allergic athlete |
| Exercise in nonpolluted air |
In addition to giving advice on the types of exercise to perform, the health care provider should recommend pharmacotherapy to prevent EIA (Fig. 1). The most effective treatment for EIA is a short-acting beta agonist (5) within 15 min before exercise (two puffs of albuterol [Proventil, Ventolin], metaproterenol, [Alupent], or pirbuterol [MaxAir] or one puff of formoterol [Foradil]) (12). If this does not give enough protection against exercise EIA, then add four to eight puffs of cromolyn (Intal) (29,41) with the beta agonist.

There is another class of inhalers that have limited usage but may be worth an empiric trial: the anticholinergics ipratropium (Atrovent) and tiotropium (Spiriva). Tiotropium is a long-acting once-daily inhaled anticholinergic that is about 80% as effective as ipratropium, but at a lower dose. There are less than 80% of predicted and symptoms greater than twice a week), daily controller medicines should be started and pretreatment before exercise should be continued. The daily therapy can be any of the controllers listed below, but inhaled steroids are the gold standard for chronic persistent asthma therapy.

If preexercise therapy has not prevented the EIA, then daily antiasthmatic therapy should be added, and the preexercise therapy should be continued. There are a number of daily medications that can be used alone or in combination: inhaled corticosteroids [budesonide (18,44) (Pulmicort), triamcinolone (Azmacort), fluticasone (Flovent), and flunisolide (Aerobid)]; leukotriene receptor antagonists (8) [montelukast (9) (Singular) and zafirlukast (33) (Accolate)]; long-acting beta agonists [salmeterol (Serevent) (6) and formoterol (Foradil) (12)]; and theophyllines (17) (Uniphyll, Theo-24).

There are potential side effects of the short- and long-acting beta agonist inhalers. The most common side effect is tremor or tachycardia; in athletes, this may be unacceptable, as it may lead to false starts or may impair fine motor skills. The other potential side effect of chronic use of beta agonists is tachyphylaxis or tolerance to the product. In this scenario, daily use of short- or long-acting beta agonists may render them less effective when really needed (31); the process by which this occurs is felt to be a down-regulation of the beta 2 receptor on the airway cells. Because this effect is seen with chronic daily dosing, it would only apply to those athletes who need it every day for training; in these subjects, it may be necessary to replace the beta agonist pretreatment with other medications, such as cromolyn.

Potential future therapies for asthma and EIA include Symbicort, Xolair, and PDE 4 inhibitors, which may be available in 2–3 yr. Symbicort is a combination of an inhaled steroid (budesonide) and long-acting beta agonist (formoterol) and is similar to Advair; it will be an alternative to Advair and will be used as chronic daily therapy. Xolair is the anti-IgE antibody that is given by injection and is another chronic preventative therapy. If any PDE 4 inhibitors make it to market, they will be similar to theophylline in their activity and will be used as chronic daily therapy. In the absence of data on EIA for any of these drugs, we will have to wait until these studies are done to identify their place in therapy of EIA.

If the patient does not respond to the usual treatment or if a challenge test is negative, then other causes of the symptomatology should be looked for, including vocal cord dysfunction (VCD), idiopathic arterial hypoxemia of exercise, cystic fibrosis, atrial septal defect, interstitial pneumonitis, and cardiac arrhythmias as other causes of dyspnea. In recent years, there has been much written about VCD as the cause of symptoms of EIA (27). A flow-volume curve with both expiration and inspiration at the time of symptoms after exercise may help in the diagnosis if it shows truncation of the inspiratory flow loop. This is not totally diagnostic, because this finding may be associated with any type of extrathoracic airway obstruction (tumor, enlarged thyroid, etc.). Confirmation of VCD requires direct visualization of the vocal cords with rhinolaryngoscopy, preferably after exercise when symptoms are present. The typical finding is the abnormal adduction of the vocal cords on inspiration. Rundell and Spiering (35) have recently reported that VCD may be identified in athletes if they have inspiratory stridor after exercise; this was identified by auscultation over the larynx and the lungs by an experienced investigator. If the evaluation for VCD is negative, then other conditions noted above need to be pursued. To evaluate for these, the patient may need a full metabolic exercise challenge with arterial blood gases, exhaled gases, cardiac monitoring, and pre- and postrhinolaryngoscopy. Other tests that may be helpful include ventilation-perfusion lung scan, echocardiogram, pulmonary venogram, etc.

HERBAL SUPPLEMENTS

Athletes will sometimes take herbal supplements, vitamins, or experiment with special diets to try to improve their performance, and they may even do this to improve their exercise asthma. The data supporting herbs, vitamins, or diets for exercise asthma are very limited. There are pre-
liminary data to suggest antioxidants may have mild beneficial effect (32), and there are data to suggest that a low salt diet may also benefit EIA (30). Until further confirmatory studies are performed with long-term use of these therapies, it is premature to recommend supplements or special diets for athletes with exercise-induced asthma.

Inhaled low molecular weight heparin has been shown to partially block EIA. These studies have been done with enoxaparin, a low molecular weight fraction of heparin (1). This is an interesting area of research that may not only help identify the mechanisms of exercise-induced asthma but may lead to an additional therapeutic option if this low molecular weight heparin can be produced in a metered dose inhaler.

DOPING

Doping is the use of any performance enhancing substance by any athlete. The governing bodies of the various organizations (International Olympic Committee, National Collegiate Athletic Association, International Skating Union, etc.) identify substances that are considered banned because of their performance enhancing effects. This information can be obtained from their respective web sites. This becomes an important issue for athletes with asthma, as they are frequently required to take beta agonist inhalers and steroid inhalers for control of their asthma; these medications fall into the “stimulant” and “steroi” categories, respectively, in the list of banned substances. However, certain inhaled beta agonists are allowed in competition as long as the athlete has well-documented asthma as identified by the IOC Medical Commission. The same is true for inhaled steroids; in both of these cases, a special form must be filled out and the governing body must be notified before participation in competition. For U.S. athletes, this information and this form can be obtained from the USOC web site. These same medications would not be allowed in athletes who do not have asthma. A list of banned substances will not be provided here because the list is subject to change, and the athlete and health care provider should check with the governing organization for the most up to date information. The new IOC Medical Commission requirements were implemented for the Olympic Games in Salt Lake City in 2002; a summary of the testing performed on athletes was reviewed by Anderson et al. (4), who concluded that proof of objective evidence of asthma in athletes is a feasible method of assuring that only those athletes with asthma are taking beta agonists and that nonasthmatics are not taking them. The issue of potential ergogenic effects of beta agonists has been discussed for years; a recent IOC medical commission document addressed this and stated “there is no scientific evidence to confirm that inhaled beta 2 agonists enhance performance in doses required to inhibit EIB.”

CONCLUSIONS

EIA is a condition that may be found in athletes at any level, from recreational to elite athletes. The diagnosis may be very obvious, but some subjects may present with unusual symptoms. It is important for the health care provider, certified athletic trainer, and coach to be aware of the possible symptoms that may suggest this condition. Treatment is effective in preventing the symptoms of EIA, but when treatment does not work, then other diagnoses should be sought after.

REFERENCES


