[Primary Care]

CEU

Exercise-Induced Bronchospasm

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Context: Exercise-induced bronchospasm (EIB) is a phenomenon of airway narrowing that occurs during or after exercise or physical exertion. This condition has been reported in a range of sporting activities but is most common in participants of cold-weather sports (eg, Nordic skiing) and indoor sports (eg, ice-skating and swimming). Traditionally, the terms *exercise induced-asthma* (*EIA*) and *EIB* have been used interchangeably; however, more recent evidence suggests that these entities are separate and should be described as such, given that their treatments differ.

Evidence Acquisition: Literature from 2000 to 2010 was obtained through searches of PubMed, Medline, and Google, with the keywords *exercise-induced asthma*, *exercise-induced bronchospasm*, *asthma and athlete*, and *asthma and sport* and with an emphasis on the current literature (last 3 to 4 years).

Results: Although the current literature suggests a differentiation between EIA and EIB, this differentiation is not always clear, and the terms are still often used interchangeably. This lack of distinction makes it difficult to draw conclusions on optimal diagnosis and treatment of EIB.

Conclusion: EIB is prevalent in elite-level athletes, with certain groups being at increased risk. Diagnostic testing should be used when possible, given that recent studies suggest poor correlation between symptoms and testing. The mainstay of treatment remains the use of short-acting β-adrenergic agonists.

Keywords: exercise-induced asthma; exercise-induced bronchospasm; diagnosis

Historically, the terms *exercise-induced asthma* (*EIA*) and *exercise-induced bronchospasm* (*EIB*) have been used interchangeably; however, these can be considered 2 separate entities that should be treated as such. EIA describes patients who have underlying asthma, and exercise is a trigger that exacerbates their asthma. EIB describes patients who do not have a history of asthma and who have bronchospasm associated with only exercise. One reason for this differentiation is the treatment of asthma/EIA versus EIB. Although current theories suggest that EIB is mediated by the release of inflammatory mediators, this process appears different from the chronic inflammation seen in EIA. In EIA, exercise can be one of many triggers, but treatment is aimed at reducing symptoms all the time. The mainstay of treatment is that of inhaled corticosteroids: Such athletes are commonly treated with a combination of a long-acting β-agonist and an inhaled corticosteroid. For EIB, the effectiveness of an inhaled corticosteroid and the role that this medication should play in treatment are debatable. Treatment of EIB centers on reducing symptoms while the athlete is exercising.

EPIDEMIOLOGY

Because of the previously stated concerns regarding the differentiation between EIB and EIA, it is difficult to ascertain how many athletes are affected by EIB. Current literature suggests that the prevalence of EIB is higher in elite athletes than in the general population and that it occurs equally between sexes and at any age.[4,19] Although the true incidence of EIB in the general population is not known, it has been reported in about 12% of the general population in Australia,[6] whereas 40% to 90% of asthmatics have EIA.[1] EIB can be detected in 41% of people with a history of allergic rhinitis.[7]

Rates are highest among athletes in cold weather (Nordic skiing) and indoor sports (ice skating and swimming), but EIB has been reported in many different arenas.[7] EIB also appears to be more prevalent in endurance athletes.[7,17,24] A study of the US Olympic winter athletes from the 1998 Winter Games revealed that 25% of the athletes were affected by EIB.[26] Cross-country skiing had the highest incidence (50%), and women were slightly more affected than men. This finding is consistent

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Table 1. Potential risk factors and triggers of exercise-induced bronchospasm.

Risk factors	Personal or family history of atopy Allergic rhinitis Cold weather sports Indoor sports Sports requiring high ventilation rates—including (but not limited to) Nordic skiing, soccer, distance running, hockey, swimming, football
Triggers	Cold, dry air Irritant exposure: allergens, high pollen counts, chlorine, pollution Intense exercise

Table 2. Differential diagnosis of exercise-induced bronchospasm.

Vocal cord dysfunction
Chronic lung disease, including asthma
General deconditioning
Exercised-induced arterial hypoxemia
Hyperventilation
Gastroesophageal reflux disease
Swimming-induced pulmonary edema
Other cardiovascular conditions

with other studies on Nordic skiing where reported rates of up to 55% are thought to be related to inhalation of cold, dry air.[7] EIB was found in up to 35% of ice-skating athletes and is thought to be due to the inhalation of cold, dry air and a high concentration of emission pollutants from the exhaust of ice-resurfacing machines.[7] Competitive swimmers have prevalence rates approaching 50%, with inhalation of chloramines from the pool water considered the inciting factor.[7,17] Rates in Olympic/elite summer athletes have been noted as high as 20%, with a rate of 17% reported in distance track and field athletes.[7]

RISK FACTORS AND TRIGGERS

Risk factors for EIB and potential inciting factors include a family or personal history of atopy to environmental factors (Table 1). Oral breathing does not warm the air, thus making it more likely to provoke airway cooling and EIB. Allergens, high pollen counts, pollution, and dry air can trigger symptoms. Intense exercise (eg, cross-country skiing, basketball, running) is more likely to cause bronchospasm because of the increased ventilation. Last, chemicals, insecticides, pesticides, and fertilizers can all trigger symptoms of EIB.[20,24]

MECHANISM

The pathophysiology of EIB is not entirely understood, and a complete review of the proposed mechanisms is beyond the scope of this article. Our current understanding of the pathophysiology of EIB is that hyperventilation during exercise causes a loss of heat and a drying of the airways, leading to dehydration of the airway cells and increased intracellular osmolarity. The osmotic gradient that is created stimulates the release of inflammatory mediators, including histamines, cytokines, and leukotrienes, among others.[3,7] These mediators, along with airway dehydration, cause an exaggerated response that results in EIB.[3,7]

Once the exercise is completed, airway cooling reverses as smaller bronchial vessels warm, creating a reactive hyperemia. This warming establishes another osmotic gradient that releases mediators, causing bronchospasm and airway edema, which can further contribute to EIB.[3,7]

Recent studies suggest that EIB is related to airway epithelial injury from breathing poorly conditioned air at high flow rates for long periods or a high volume of irritant gases or particles.[3] Breathing air saturated with chloramine pool water or even repeatedly breathing cold, dry air can injure the airway. The inflammatory mediators from the airway cells (eg, histamine, leukotrienes, prostaglandins) can lead to alteration of the smooth muscle contractile properties, causing hypersensitivity that can produce bronchoconstriction.[3]

DIFFERENTIAL DIAGNOSIS

Many symptoms of EIB are nonspecific and can been seen in a variety of disorders (Table 2). A complete history and physical examination will help to alert clinicians to potential other causes or associated conditions. Chronic lung disease, including asthma, other cardiovascular disorders, and generalized deconditioning, should be suspected on the basis of the history, physical examination, and results of pulmonary function testing. A complete discussion of all the conditions is not possible; however, a brief review will be provided.

Vocal Cord Dysfunction

Vocal cord dysfunction (VCD) can produce respiratory symptoms whenever the ventilatory rate rises. VCD typically causes inspiratory wheezing and/or stridor, as opposed to EIB and EIA, which primarily produce expiratory wheezing. The stridor in VCD occurs secondary to paradoxical closure of the vocal cords. Patients complain of difficulty "getting air in." VCD is frequently misdiagnosed as asthma or EIB and warrants special consideration when EIB patients do not respond to treatment.[7]

The diagnosis of VCD is often made clinically. If a flow-volume loop is performed while the patient is symptomatic, a flattening of the inspiratory loop occurs. The diagnosis can also be made during direct laryngoscopy, visualizing vocal cord adduction (anterior two-thirds) during inspiration with a posterior diamond-shaped opening remaining. Treatment of VCD is reassurance, education, and speech therapy.[6]

Gastroesophageal Reflux Disease

Gastroesophageal reflux disease may present with atypical symptoms, such as chronic cough and wheezing, and it has been associated with asthma, although the mechanism of this association is not entirely clear.[24] This diagnosis should be considered in athletes who have symptoms of gastroesophageal reflux disease or worsening symptoms of EIB associated with regurgitation, dyspepsia, large meals, or alcohol.

Swimming-Induced Pulmonary Edema

Swimming-induced pulmonary edema presents with shortness of breath and cough during or immediately after swimming with associated evidence of pulmonary edema. Spirometry reveals an acute restrictive pattern. These changes may remain for up to 1 week. This condition has been described in young healthy men but not elite-level swimmers, and risk factors for development and recurrence are unclear.[7]

Exercise-Induced Arterial Hypoxemia

Exercise-induced arterial hypoxemia has been reported in highly trained athletes. Although the exact mechanism is not clear, it is thought that rapid transit of red blood cells through pulmonary capillaries results in incomplete diffusion, resulting in a reduction in arterial oxygen saturation.[7] Suspicion for this condition should be entertained in highly trained athletes who present with decreased exercise capacity at high workloads. Pulse oximetry monitoring can help to differentiate this condition from other causes.

DIAGNOSIS

Clinical Signs and Symptoms

EIB was a disease classically diagnosed and treated on the basis of self-reported symptoms. Recent studies have shown a lack of diagnosis specificity and sensitivity based on symptoms.[16-19] These studies have demonstrated that athletes with presumed symptoms of EIB did not have EIB with testing. Testing also found a number of athletes that fit criteria for EIB but did not have symptoms, emphasizing a need for objective testing in athletes. Therefore, although a history and a physical examination are important, they should not be relied on to diagnose or exclude the diagnosis of EIB.

The presentation of EIB can vary; the most common symptoms include wheezing, shortness of breath, chest tightness, chest pain (children), cough (after exercise), early fatigue, and poor performance. Symptoms are similar to those that occur in an acute asthma attack, but they are induced by 10 to 15 minutes of intense exercise and are much shorter in duration. Symptoms often dissipate when the activity stops. A late-phase response can occur 4 to 8 hours after exercise.[20]

Physical examination immediately after exercising may reveal wheezing, which usually dissipates with rest. Athletes with wheezing at rest should be suspected of having asthma rather than EIB. Dermatologic examination may demonstrate atopic disease such as eczema. Nasal examination may show enlarged, boggy turbinates. The throat may have posterior pharynx cobblestoning and enlarged tonsils, which are both signs of allergic rhinitis.[20]

Table 3. Bronchial provocation tests approved by the World Anti-doping Agency for the diagnosis of exercise-induced bronchospasm.[27]

Bronchial Provocation Test	Decrease in FEV_1 for Positive Test Result[a]
Eucapnic voluntary hyperpnea	>10%
Methacholine aerosol challenge	>20%
Mannitol inhalation	>15%
Hypertonic saline aerosol challenge	>15%
Exercise challenge (field or laboratory)	>10%
Histamine challenge	>20%[b]

[a]FEV_1, forced expiratory volume in the first second.
[b]At a histamine concentration of 8 mg/mL or less during a graded test of 2 minutes.

Diagnostic Testing

A variety of testing procedures can be used to diagnosis EIB. Table 3 lists the World Anti-doping Agency's (WADA's) approved tests and diagnostic criteria.[27]

During pulmonary function testing, related medications should be stopped to prevent testing confusion and false-negative results (Table 4). Other testing in suspected cases of EIB should either focus on associated conditions (eg, allergies) or be undertaken if alternative or comorbid diagnosis is suspected by history and clinical examination. From a practical standpoint, some authors have advocated a clinical trial of a short-acting inhaled β-adrenergic agonist 15 to 20 minutes before exercise.[22] This approach uses resolution of symptoms as a successful test result. Although practical and inexpensive

Table 4. Stopping medications for pulmonary function tests.

β-Agonists (long- and short-acting)	24 hours
Inhaled steroids	1 week
Leukotriene receptor antagonists	24 hours
Cromolyn sodium/ nedocromil sodium	24 hours
Theophylline	24 hours
Antihistamines	Can be taken before pulmonary function testing but should be stopped at least 72 hours before skin testing for allergies[1,9]

for athletes who do not require testing for WADA or other governing bodies, this testing runs a high risk of both false-positive and false-negative results; ideally, formalized testing should be used. However, in circumstances where financial and testing resources are limited, this approach is reasonable and has low risk of adverse events.

Although a reversal of the decreases in forced expiratory volume in the first second (FEV_1), as found during testing with pharmacological treatment (mainly, a short-acting inhaled β-adrenergic agonist), may be helpful in monitoring treatment response, it is not required for diagnostic purposes in EIB.

Exercise Challenge Tests

Exercise challenge tests can be performed in laboratory or field settings. Spirometry should initially be done at rest, then after exercising to establish baselines and comparison tests. The suggested exercise challenge should be performed for 8 minutes. Testing conditions should allow the athlete to reach >90% of peak heart rate at 2 minutes and maintain this level for another 6 minutes. These parameters allow most athletes to achieve an adequate associated ventilation rate, >85% maximum voluntary ventilation. Maximum voluntary ventilation is the maximum volume of gas that a person can inhale and exhale by voluntary effort per minute by breathing as quickly and deeply as possible. For highly fit or elite athletes, peak heart rates closer to 95% may be required.[19]

The response to the exercise challenge is considered positive when there is a decrease in FEV_1 of 10% or more compared to baseline. The postexercise FEV_1 should be done immediately after exercise. Some experts recommend postexercise spirometry at 5, 10, 15, and 30 minutes.[19] The International Olympic Committee (IOC) recommends that the FEV_1 be measured at least 3 minutes after the challenge, and it accepts a 10% decline in FEV_1 as an indication for a β-2 agonist.[13] The results should be sustained over 5 minutes to be consistent with EIB.[19]

Field Versus Laboratory Testing

Sport-specific field testing is ideal when resources allow. Studies have had some athletes with negative testing in the laboratory setting but positive results with field testing.[19] Variations in temperature, humidity, and other environmental factors can make testing difficult, but these variables may also account for the differences found between the field and the laboratory. Sport-specific tests for activities such as Nordic skiing, ice hockey, speed skating, and figure skating have been shown to accurately reproduce the symptoms of EIB.[19,24] If sport-specific field testing is not feasible, then free running at maximal effort can be used to induce symptoms of EIB: It is cost-effective and requires few resources, but caution should be used because it may not trigger EIB in all athletes.[19] Difficulty in standardization of the testing environment and cardiovascular monitoring are the main drawbacks to field testing. Potential false-negative results can occur if testing parameters (peak heart rate and ventilation rates) are not achieved.

Laboratory testing allows for easier control of the testing environment and parameters. Ideally, dry air (<5 mg $H_2O \cdot L^{-1}$) should be used. False-negative results may occur if ambient humid air is used because airway drying is thought to be a trigger in EIB.[19] Although it is advantageous to have a controlled environment, relative humidity, pollutants, and other irritants may decrease the likelihood of provoking symptoms of EIB.

Bronchial Provocation Tests

During bronchial provocation tests, the patient inhales a substance designed to induce bronchoconstriction. These may be used as the primary diagnostic test or when exercise testing is equivocal or negative and a diagnosis of EIB is still suspected. Current evidence does not suggest superiority of any one of these tests, but the IOC prefers the eucapnic voluntary hyperpnea test for the diagnosis of EIB.[13]

Eucapnic voluntary hyperpnea. Eucapnic voluntary hyperpnea testing is performed by inhalation of dry air containing 5% carbon dioxide; it has a high sensitivity for EIB. Maximum voluntary ventilation should be between 60% and 85%, with elite-level athletes at 85% minimum. Increased ventilation is thought to cause drying of the airway-surface liquid and increased osmolarity with subsequent release of inflammatory mediators.[19] Spirometry is used at baseline and within 3 minutes after the challenge of the dry-air inhalation for 6 minutes. A decrease in FEV_1 of more than 10% is considered a positive test.[2,10,19]

Hypertonic saline challenge (hyperosmolar aerosols). The hypertonic saline challenge is performed by inhalation of a

4.5% hypertonic saline initially for 30 seconds. It is thought to act by increasing airway surface liquid osmolarity, which triggers sensitized cells to release inflammatory mediators.[19] If the percentage decline from baseline FEV_1 is less than 10%, the exposure time is subsequently doubled (60 seconds, 2 minutes, 4 minutes, 8 minutes).[17] Spirometry is performed within 1 minute of exposure. A 15% decline is considered a positive test for EIA.

Inhaled powdered mannitol challenge. The inhaled powdered mannitol challenge test begins with 5 mg and is doubled (10 mg, 20 mg, etc) up to 160 mg; the protocol recommends cumulative exposure of 635 mg.[19] The mannitol is thought to cause smooth muscle contraction by stimulating the release of inflammatory mediators.[19] In addition, the testing is thought to better represent the neural and cellular contribution to airway hyperresponsiveness because mannitol does not directly act on the airway smooth muscle.[19] FEV_1 is measured 1 minute after each dose, with a 15% decline in FEV_1 considered a positive test result. Although used in some parts of the world, this test does not have Food and Drug Administration approval in the United States.

Methacholine challenge. Despite different techniques and dosing methods, the methacholine challenge generally involves inhalation of nebulized methacholine. Methacholine stimulates acetylcholine receptors, causing smooth muscle contraction and bronchoconstriction.[1,8] Even normal airways have some hyperresponsiveness to this challenge, so close monitoring of the dosing regime is of utmost importance. The test has traditionally been used for the diagnosis of asthma and has been reported to have low sensitivity in the diagnosis of EIB.[8] The IOC considers a positive test result a fall in FEV_1 of greater than 20% at a dose less than or equal to 400 mcg (cumulative dose) or 200 mcg (noncumulative) or a concentration less than or equal to 4 mg/mL (tidal breathing technique).[13] There is some evidence for the use of other provocative agents—carbachol, histamine, and adenosine monophosphate—but the IOC does not currently accept tests with these agents.[13]

PREVENTION AND TREATMENT

Pharmacologic: β-Adrenergic Agonists

Several classes of medications have been used in the treatment of EIB, but the most commonly recommended are the short-acting inhaled β-adrenergic agonists. Because the majority of the studies on the pharmacological treatment of EIB do not clearly differentiate EIB from EIA, optimal treatment is difficult to assess. Most recommended medications are well tolerated and are allowed in competition, although some require a declaration of use or a therapeutic use exemption (TUE). Clinicians and athletes should always check with their sports' governing bodies before initiating any pharmacological treatment.

Short-acting β-adrenergic agonists are the treatment of choice for EIB.[22,25] These should be taken 15 to 20 minutes before exercise, ideally using a spacer with the inhaler to maximize the concentration of the medication. Short-acting β-adrenergic agonists are generally well tolerated, but common side effects can include tachycardia, palpitations, and anxiety.

Long-acting β-adrenergic agonists have been effectively used to treat EIB.[24,25] However, because of recent data linking these medications to adverse outcomes, they should not be used as monotherapy for the treatment of EIB.[13,24] These agents are generally used in conjunction with an inhaled steroid for athletes with underlying asthma, and they may be helpful with EIA in this setting.

As of January 1, 2010, all β-adrenergic agonists are prohibited by WADA, except salbutamol (albuterol) and salmeterol. These 2 medications require a declaration of use, whereas all other β-adrenergic agonists need a TUE. Note, however, that urinary concentrations of salbutamol greater than 1000 ng/mL are not presumed to be therapeutic.[28] Additionally, athletes and clinicians should be aware that tolerance may develop to both short- and long-acting forms of this medication, which can become less effective if used on a regular basis.[24]

Other medication classes are commonly used in the treatment of EIA, but their place in the treatment of EIB is unclear. Inhaled steroids are the mainstay of treatment in persistent asthma and EIA but have less of an indication in EIB.[20] Inhaled steroids require a declaration of use, but systemic use is prohibited and requires a TUE. Leukotriene antagonists can be used as second-line medication or occasionally used as monotherapy. They are as effective as long-acting β-adrenergic agonists but have not been compared to short-acting β-adrenergic agonists.[21] They may help treat EIA, and they may be effective in EIB that does not respond to a β-agonist (short- or long-acting).[21,24] Cromolyn sodium can be used as an adjunctive treatment for asthma and EIA.[11,19,22,24] Antihistamines and intranasal steroids are indicated for seasonal allergies and allergic rhinitis that may exacerbate EIA. These medications can be used alone or in conjunction with one another.[24] Leukotriene antagonists, antihistamines, and cromolyn sodium are not prohibited substances.[5]

Nonpharmacologic Treatment

Although the mainstay of treatment of EIB in athletes is pharmacologic, other measures have been found to be effective. Improved conditioning is generally thought to decrease the symptoms and severity of EIB and increase the threshold at which symptoms develop.[6,22] Whether findings represent improved conditioning alone and not a decrease in EIB severity is unknown, but a military study found that recruits with EIB were able to make gains in physical fitness similar to those of recruits without EIB.[23] Given the high prevalence of EIB in elite endurance athletes, it does not appear to impede improved conditioning. Warm-up routines consisting of submaximal work loads and sprints reduce the symptoms or EIB for subsequent exercise.[6] Short bursts of vigorous exercise (wind sprints) may extinguish EIB and induce short-term resistance to EIB, particularly if EIB is due to an endogenous release of prostaglandins.[10,22]

Warming and humidifying of the air may help decrease the symptoms of EIB. Masks and nasal breathing have been found to be helpful but are not always obtainable. Avoiding exposure to cold, pollutants, and irritants can be helpful in some circumstances, but environmental conditions are often dictated by the sport. Diet may also influence the symptoms of EIB. Low-salt diets and those high in antioxidants and fish oils may decrease the frequency and severity of symptoms.[15] No studies compare these nonpharmacologic treatments with medication.

SPECIAL CONSIDERATIONS

Screening

Given the relatively high prevalence in elite-level athletes, some organizations have been screening athletes for EIB.[12] No current data exist to guide this practice, and many questions exist about its efficacy and cost-effectiveness. Additionally, studies do not address whether the treatment of EIB improves athletic performance. Before institution of widespread screening, further study is needed to assess the potential benefits and harm.

Governing Bodies

National Collegiate Athletic Association. The National Collegiate Athletic Association requires a doctor's note or prescription for the use of a medication to treat EIB or asthma. The only restriction is that albuterol must be in the inhaled form.[14]

WADA and the IOC. A high percentage of Olympic athletes were using β-2 agonists for EIB and EIA, with increasing numbers from the Atlanta Games in 1996 to Sydney in 2000.[8] These numbers clearly exceeded the prevalence of EIB and EIA in the general population, so the IOC Medical Commission established new criteria in 2002 (updated in 2008) for the use of β-2 agonists during competition for the diagnosis of EIB or asthma.[4,5,8,13]

The IOC follows the recommendations of the WADA.[29] Note that WADA and the IOC do not differentiate between EIA and EIB for testing and medication use. To secure an exemption for a banned medication, an athlete must have a TUE validated by an appropriate physician. The TUE is reviewed by a Therapeutic Use Exemption Committee online at WADA (http://www.wada-ama.org) and the IOC (http://www.olympic.org) (Table 5), which ensures an equal process for the athletes across countries and sports.

A TUE can be canceled if the athlete does not comply with the rules of WADA or the IOC. The application must include a comprehensive medical history, with relevant examination, laboratory, and imaging results. The application must also include a statement by a qualified physician attesting to the necessity of the prohibited substance, and it must explain why an alternative permitted medicine cannot be used in the treatment of the condition. The dose, frequency, route, and duration of administration of the medicine must be specified.

As previously stated under WADA regulations, all β-2 agonists (including their D- and L-isomers) are prohibited, except salbutamol (albuterol) and salmeterol, which now require only a declaration of use, as filed by the athlete with WADA or declared on a Doping Control Form at the time of testing.[29]

Table 5. Criteria for acceptance of a therapeutic use exemption.

Therapeutic use exemption must be submitted 21 days or more in advance of the event.
The athlete would experience significant impairment to health if the prohibited substance were withheld.
The use of the prohibited substance would produce no additional enhancement of performance other than that which might be anticipated if the athlete was in a state of normal health.[a]
There is no reasonable alternative to the prohibited substance.

[a]Use of a prohibited substance to treat "low-normal" levels of any endogenous hormone is not considered an acceptable therapeutic regimen.

CONCLUSION

The literature uses the terminology for EIB and EIA interchangeably, thereby making it difficult to assess the ideal diagnostic testing and treatment strategies for EIB. Controversy still exists as to whether these entities are different or part of the same spectrum of asthma. It appears to be prevalent in elite-level athletes—particularly, those who participate in cold environments; however, the degree to which EIB limits an athlete's ability to perform is unknown. Diagnostic testing is important because history and physical examination alone do not reliably predict the presence or absence of EIB. The treatment of asthma and EIA should focus on controlling symptoms with inhaled steroids, whereas EIB appears to be best controlled by a short-acting β-adrenergic agonist. Other pharmacologic and nonpharmacologic treatments may be effective in the treatment of EIB, but their role is less well defined. Further studies are required to determine if EIB is indeed a separate disease and to define its pathophysiology, optimal diagnostic testing, effects on performance, and treatment.

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Clinical Recommendations

SORT: Strength of Recommendation Taxonomy
A: consistent, good-quality patient-oriented evidence
B: inconsistent or limited-quality patient-oriented evidence
C: consensus, disease-oriented evidence, usual practice, expert opinion, or case series

If an athlete's clinical history is suspicious for exercise-induced bronchospasm (EIB), a trial of a short-acting β-adrenergic agonist, such as albuterol, is acceptable.[1,6,7,9,11,20,22,24] Note that salbutamol (albuterol) is the only short-acting β-adrenergic agonist allowed by the World Anti-doping Agency and requires a declaration of use. All other forms require a therapeutic use exemption.

Clinical Recommendation	SORT Evidence Rating
History and physical examination are not adequate to diagnose EIB.[16-19]	B
Short-acting β-adrenergic agonists are the treatment of choice in EIB.[22,24]	C
Field-based testing is optimal for the diagnosis of EIB.[19]	B
The eucapnic voluntary hyperpnea test is the laboratory diagnostic bronchoprovocation test of choice.[13]	C

For more information about the SORT evidence rating system, see www.aafp.org/afpsort.xml and Ebell MH, Siwek J, Weiss BD, et al. Strength of Recommendation Taxonomy (SORT): a patient-centered approach to grading evidence in the medical literature. *Am Fam Physician.* 2004;69:549-557.

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