Exercise and asthma: a review

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Introduction

The benefits of exercise cannot be overestimated as physical activity has been shown to be protective for a variety of chronic illnesses such as hypertension, ischaemic heart diseases, type 2 diabetes, osteoporosis, colonic cancer, anxiety, and depression. Low levels of activity are associated with increased mortality; 12% of deaths in the USA can be attributed to low levels of physical activity.[1] In spite of this, many asthma patients are still unwilling to undertake any exercise due to the fear of precipitating an attack and are, therefore, unfit. Studies have shown that asthma sufferers are able to exercise and improve their fitness and the limitation in exercise capacity tends to relate more to their lack of fitness rather than airflow obstruction.[2] Many asthmatics have won gold medals in the Olympic games.

The relationship between asthma and exercise has long been known; it was Aretaeus (120–200AD), over 1800 years ago, who noted that physical exertion provoked airway obstruction.[3] Over 300 years ago, Sir John Floyer, who was himself asthmatic, described the adverse effect of physical exercise on his asthma, noting that different exercises had a greater or lesser adverse effect,[4] and Herxheimer suggested that hyperventilation stimulated airway narrowing.[5]

While exercise-induced asthma (EIA) is a common and age-long phenomenon, many health practitioners do not have an adequate understanding of its characteristics, diagnosis, management, and prevention. The public health consequences of unrecognised or inadequately diagnosed EIA are significant: asthma was found to be the single greatest risk factor for unexplained death in a 30-year review of Israel military recruits.[6] In addition, the prevalence of EIA is increasing among athletes.[7] This article seeks to present a broad overview of EIA, use of objective tests for correct and precise diagnosis, and comprehensive management.

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Definition

EIA is a condition in which vigorous physical activity triggers acute or transient airway narrowing in people with heightened airway reactivity, occurring mostly after the exercise[8] and rarely during the exercise,[9] resulting in a decrease in post-exercise pulmonary function.

The term exercise-induced bronchoconstriction (EIB) is often used interchangeably with EIA but the former may be more appropriate when exercise is the only provoking factor in some individuals, especially elite athletes when there are no other symptoms or signs of asthma.[10]

Prevalence of EIA

The reported prevalence of EIA varies widely depending on the criteria for diagnosis, the population studied, environmental factors, and the differences in the intensity of the exercise. The prevalence of EIA in asthmatics ranges from 40 to 90%.[11–13] One reason for this is the lack of uniformity in methods used to detect the response; however, it is believed that almost all asthma patients will have EIA if the exercise is performed under standardised conditions.

In addition, 35–40% of patients who have allergic rhinitis experience EIA.[14,15] EIA can also be seen in 3–10% of the normal population.[16] This translates to an overall prevalence of 12–15%.

The prevalence of EIA among athletes is higher when compared with the general population. In summer sports events, the prevalence ranges between 3.7 and 22.8%[17] but this can rise to 54.8% in cross-country skiers.[18]

Characteristics of EIA

A typical response to exertion or exercise in asthmatics is initially bronchodilation, possibly due to the release of endogenous adrenaline and decreased vagal tone stimulation. This is apparently physiological as there is an increase in lung volume. However, towards the end of the exercise period, the lower airway begins to bronchoconstrict and the lung function deteriorates; the forced expiratory volume in one second (FEV_1) and peak expiratory flow (PEF) rate decrease significantly, with maximal bronchoconstriction generally occuring between 3 and 15 minutes post-exercise.[19] The lung function usually returns spontaneously to baseline over 30–60 minutes depending on the magnitude of bronchial narrowing. The magnitude of the response depends on a number of factors.

The type of exercise

It has been observed that certain activities, e.g. running, produce greater airflow limitation than other activities, e.g. swimming. This is caused by the differences in absolute ventilation level and by differences in characteristics of inhaled air content. The idea that there are inherently unique aspects of a specific task is outdated.[20] Table 1 shows examples of high-ventilation and low-ventilation sport although EIA can occur in any setting. It is especially prevalent in endurance events such as cross-country skiing, swimming, and long-distance running, in which ventilation is increased for long periods of time during training and competition, allowing for relatively more evaporative water loss and subsequent airway narrowing. There is also an increased prevalence of EIB in winter sports athletes, Although swimming is a high-ventilation sport the characteristics of the inhaled air make it less likely to cause EIA.

High-ventilation sports	Low-ventilation sports
Track	Golf
Cross-country	Baseball
Soccer	Bowling
Ice-hockey	Weight-lifting
Swimming	Volleyball
Cross-country skiing	Football

Table 1 Examples of high- and low-ventilation sports

Ambient air condition (cold air, low humidity, pollutants)

Athletes who participate in environments in which there may be environmental pollutants are at increased risk for the development of EIB. Chlorine compounds in swimming pools and chemicals related to ice-resurfacing machinery in ice rinks may put certain populations of athletes at additional risk.

Other factors contributing to the severity of the response are:

- exposure to allergies in sensitised individuals;
- overall control of asthma;
- poor physical conditioning;
- respiratory infections;
- underlying bronchial hyper-reactivity.

Although most patients recover from EIA within 1 hour, late asthmatic responses that occur 3 to 9 hours after exercise have been reported. Some investigators have doubted the existence of this late response or its clinical relevance because bronchoconstriction during late response is usually less severe. Patients at higher risk for late responses appear to be children and those with severe early response.

With a repeat exercise challenge under identical conditions, less than half of the original degree of obstruction will occur; this is defined as the refractory period. Some 40%–50% of asthma patients will be refractory to exercise when a second exercise is undertaken within 2 hours.[14]

The pathogenesis

The pathogenesis of EIA has been the subject of great debate among scientists over the years, although the mechanism is becoming fairly well understood, some aspects still require clarification.

Inspired air has a temperature of 68–73.4°F (20–23°C) and a water content of 6–12 mg/l. At 30–60% relative humidity with normal inspiration, inhaled air is conditioned by the nasal passages to body temperature (37°C) and a water content of 44 mg/l before its arrival in the lower airways. However, during exercise with resulting high ventilation of a large volume of air, the nasal cavity is bypassed and the conditioning and humidification process shift to the lower airways.[13]

The exact stimulus for bronchocontriction leads to two hypotheses: the thermal hypothesis (cooling of the airways) and the osmotic hypothesis (loss of water vapour). However, the two hypotheses may account for the resultant bronchoconstriction.

The thermal hypothesis suggests that the increased ventilation causes airway cooling by transferring of thermal energy to the inspired air; this is followed by a rapid rewarming of the airways. The rewarming of the respiratory mucosa induces a reactive hyperaemia of bronchial microvasculature and oedema of the airways. The larger the quantity of thermal energy that needs to be transferred, the more rapidly the airways rewarm and the more bronchi are narrowed.[7,19,21] Support for this theory came from observation that inhalation of cold, dry air during exercise caused a greater reduction of FEV_1 than did inhalation of ambient or hot dry air.[22,23] However, this theory did not fully explain the documentation of quite severe EIA that occurs under conditions of inspiring hot, dry air.

Osmotic theory was developed later and it suggested that it was the increased rate of respiratory water loss caused by hyperpnoea of exertion that induces hyperosmolality of the epithelium, and that this provides a favourable environment for the release of mediators that cause airway narrowing.[24–26] The evidence that hypertonic aerosol challenge is capable of inducing airway obstruction in asthmatic subjects supports the theory that hyperosmolality induced by hyperpnoea initiates EIA.[27,28] In addition, the osmotic effect of mucosal dehydration causes an increase in blood flow during exercise.

The relative role of heat and water loss in pathogenesis is still being debated; however, there has been increasing documentation of the release of mediators from inflammatory cells in EIA. These mediators cause the resultant bronchoconstriction.

Mast cell degranulation has been shown in bronchial biopsies from humans after exercise.[29] A significant correlation has been reported between the severity of EIB and the degree of peripheral and blood eosinophilia,[30] and eosinophilia in induced sputum of asthma patients.[31] Hallstrand and his co-researchers demonstrated that EIB is associated with injury to the airway epithelium, over expression of cysteinyl leukotrienes, airway eosinophilia,

and under-production of prostaglandin E_2.[32]

There is suggestion that nitric oxide (NO) plays a significant role in the development of airway obstruction that follows hyperventilation.[33]

Although inflammation is a critical component of asthma and EIB, the role or significance of inflammation in the pathogenesis of EIB in subjects without asthma is unclear, further studies are needed to clarify this.

Clinical features

Most subjects with EIA have classic symptoms of airway obstruction following exercise: dyspnoea, cough, chest tightness that starts 10–15 minutes into exercise and peaks 8–15 minutes after the exertion is completed. However, history alone is not a reliable indicator of EIA; 50% of children with asthma who gave a negative history of EIA have a positive response to exercise challenge. Erhabor and colleagues[34] showed that 66.15% of asthma patients with no history of EIA were found to demonstrate the classic pattern of EIA. The reason for this asynchrony between history and exercise challenge is not known; however, poor perception among asthma sufferers is suggested as one of the causes. Other factors in patients' history that may suggest the prevalence of EIA are: symptoms that vary by season or outdoor temperature; decreased, or altered exercise regimen; complaints of decreased or limited endurance; minimal problems with swimming or being in a warm humid environment. A thorough family history and occupational history should also be obtained.

A complete physical examination should be carried out on patients who have exercise-related respiratory complaints. Other medical problems that can mimic EIA need to be considered in the initial evaluation of exertional dyspnoea. The nose, throat, sinuses, pharynx, heart, and lungs should be examined to exclude other differential diagnoses as described below. However, examination findings are often normal in patients with EIA.

Diagnosis

For confirmation of EIA, a challenge test is often warranted because a symptoms-related diagnosis has a low sensitivity and specificity.[34] The challenge test may be direct or indirect.

The exercise challenge test

The exercise can be performed in the laboratory or in the field. Generally subjects are advised to stop medications 8–48 hours before the test according to the type of drugs, and also avoid vigorous exercise at least 4 hours before the challenge. Care must be taken to rule out any contraindications to exercise testing as shown in Table 2.

Field exercise challenge test

Athletes undergo the test while performing their usual exercise or sport with different protocols, depending on the type of exercise. Exercise in the field is an effective[35] and more sensitive way to identify EIA in cold weather compared with exercise under laboratory conditions of temperature and humidity.[36] Whatever the type of exercise, the FEV_1 is measured pre- and post-exercise at 5 minute intervals up to 30 minutes. A positive response to exercise is a fall in FEV_1 of ≥10% in accordance with European Respiratory Society (ERS)[37] and American Thoracic Society (ATS)[38] guidelines. A cut-off point of 10% seems to be based on a co-efficient of variation of 6% for repeated manoeuvres of FEV_1.[39]

For those who are asthmatic, a continuous and rigorous free-running exercise can be done for 6–8 minutes, according to established protocols.[38] The disadvantages are that it may not be standardised for cardiovascular work load and for environmental conditions.

Laboratory exercise challenge

The exercise must be hard and brief and the patients must inspire air with the appropriate characteristics of a temperature of 20–25°C and a humidity of <50%. The target ventilation and/or heart rate must be reached quickly within the first 2 to 3 minutes of the exercise.

A treadmill or bicycle ergometer are the most commonly used exercise stimulators in physiological laboratories. The choice depends on the availability, experience, and expertise of the investigators, although a motor-driven treadmill may be preferred because it induces a faster increase in ventilation. During exercise it is necessary to monitor some variables such as electrocardiogram (ECG), pulse oximetry, and blood pressure. Standard protocols are available in ATS guidelines.[38]

The laboratory test has the advantage that the measurement can be made under controlled conditions; however, few laboratories are equipped with appropriate cycle ergometers to test trained athletes and few have treadmills that can be safely use at high speed; other disadvantages are that it is not sport specific and equipment is expensive.

However, the sensitivity of these laboratory-based tests to identify airway hyper-responsiveness to exercise, even when the inspired air is cool and dry, is only approximately 65% in treated adult asthma patients,[40] 50% in asthmatic children who inhaled air in temperate climate conditions[41] and even less than 25% to identify

Absolute	Relative
Severe air flow limitation	Moderate air flow limitation
FEV_1 <50% of predicted < 1l	FEV_1 < 60% of predicted <1.5l
Heart attack or stroke in the last 3 months	Inability to perform acceptable spirometry
Uncontrolled hypertension	Pregnancy
Aortic aneurysm	
Unstable cardiac ischaemia	Nursing mothers
Malignant arrhythmias	Current use of cholinesterase for myasthenia.

Table 2 Contraindications to exercise testing

EIB in athletes that exercise in the field.[36] For these reasons other bronchial provocation tests have been used as surrogate tests for EIA or bronchocontriction.

Eucapnic voluntary hyperpnoea (EVH)/hyperventilation test

The eucapnic voluntary hyperpnoea (EVH) is the test that is currently recommended by the International Olympic Committee Medical Commission (IOC-MC) for diagnosis of EIB in elite athletes, and is the best known surrogate test to identify EIB. The test is well standardised.[42–44] It is performed by making the subject inhale a dry mixture containing 5% carbon dioxide, 21% oxygen (the other component is nitrogen) for 6 minutes.

The mixture is safe, stimulates ventilation, and maintains a normal end-tidal partial pressure. The target ventilation for 6 minutes is 30 x FEV_1, and this is close to 85% of the predicted maximum voluntary ventilation.[45] The FEV_1 is measured at least up to 15 minutes after the end of the test. The test is considered positive when the FEV_1 fall is 10%. This was based on the findings of Hurwitz et al[46] who found that a 10% fall in FEV_1 has a specificity of 90% for identifying those with asthma and also maintains the value of exercise. The advantages are that it is relatively inexpensive, easily standardised among laboratories, achieves high ventilation required to induce bronchoconstriction in elite athletes, and if necessary, time, ventilation, and inspired air condition can be adjusted to simulate the conditions under which the athlete is competing.

Osmotic challenge test

Many studies have been carried out comparing the effects of exercise and eucapnic hyperpnoea with non-isotonic aerosols, which clearly show that asthmatics are sensitive to these stimuli.[47–49] This is based on one of the mechanisms for EIA – that the airway narrowing is due to increased osmolarity of the airway surface in response to the loss of water by evaporation in conditioning the inspired air.[24,26,27] It consists of administrating progressive doses of hypertonic saline (4.5%) obtained by increasing the duration of administration. The wet aerosol is usually generated by a large volume (200 ml) of ultrasonic nebuliser. Measurements of FEV_1 are made before the challenge and 60 seconds after each exposure (at 1, 2, 4, and 8 minutes). A fall of FEV_1 ≥15% is diagnostic of EIB although a lower value of 10% has been suggested. The test is safe and inexpensive but it is performed in a laboratory.

A new osmotic challenge involving the inhalation of progressively increasing doses of dry powder mannitol has also been developed.[50] It consists of administration of increasing doses of encapsulated dry power mannitol via a spinhaler. It may fulfil the criteria for evaluating EIB at the point of care

Measurement of airway hyper-responsiveness by methacholine

This is also used to identify EIB. The dose or concentration (PD_{20} or PC_{20}) of methacholine that will cause a fall of 20% in FEV_1 from baseline level is used as the cut-off point. For those who have not used inhaled steroid, PC_{20} ≤4 mg/ml or PD_{20} ≤0.4 mg (2 µmol) is usually diagnostic (see Table 3).

Management

The main goal of management is to allow patients to participate fully in athletic activities without difficulty. The management is optimised by combining pharmacological therapy with non-pharmacological therapy.

The main goal of pharmacological therapy is preventing the onset of symptoms and treating breakthrough episodes that may occur following exercise.

The β2 selective agonists, administered by inhalation, are the class of drugs that have been shown to be most effective in protecting against EIA, and therefore remain the drug of choice.[51] The short-acting β2 agonists provide symptomatic relief and a rapid onset of bronchodilation. Treatment with two puffs of a short-acting agonist (salbutamol) shortly before (15 minutes) exercise will provide peak bronchodilation in 15 to 60 minutes and protection for at least 3 hours in most patients.[52] However, the overuse of β2 agonist has been shown to result in tachyphylaxis or tolerance and to worsen the symptoms of EIB and asthma.[51]

Long-acting β2 agonists, such as salmeterol and formoterol, work in a pharmacologically similar manner to short-acting bronchodilators and the bronchoprotection can last up to 12 hours.[53] Ferrari and colleagues demonstrated that inhalation of formoterol is effective in protecting the asthma patient as early as 15 minutes after dosing, but salmeterol was not rapidly effective.[54,55] Palmquist and colleagues[56] discovered that formoterol was effective in 12 minutes, while salmeterol was effective in 31 minutes. These studies suggest that there are differences in the pharmokinetics between the two drugs and that the administration of salmeterol should occur significantly in advance of exercise (at least 30 minutes) for it to be optimally effective. Long-acting bronchodilators are not rescue medications and should not be used more than twice a day. Tachyphylaxis also occurs after repeated doses of a long-acting β2 agonist.

Mast cell stabilisers (disodium cromoglycate and nedcromil sodium)

These drugs have been shown to be effective in preventing EIA.[57,58] They prevent mast cell degranulation, but although these agents are effective they are often used as a second-line treatment due to their cost, lower efficacy, and poorer duration of action compared with β2 agonists. They are usually given 15–20 minutes before exercise and are highly effective as a combination therapy in patients who do not respond to single-medication therapy.

Leukotriene modifiers

This group of drugs has been studied in depth and have been shown to be effective in controlling asthma

Test	Characteristics
1. Clinical history of respiratory symptoms during exercise and clinical evaluation	• It is not sensitive and not specific • Clinical examination may exclude other causes of respiratory related symptoms. • It is required by IOC-MC for diagnosis of EIB in athletes
2. Lung function (spirometry) or maximum expiratory flow volume loops. An increase of FEV_1 ≥12% after inhaled bronchodilator therapy is considered positive	• May identify people with abnormal baseline function necessitating treatment even when standardised exercise test has not been performed. However, many athletes may have normal baseline function
3. Standardised exercise test in the laboratory: ≥10% fall in FEV_1 is diagnostic[37,38]	• Equipment is not widely available • Equipment is relatively expensive • Has high specificity but only moderate sensitivity for EIB • Has standardised exercise load and stable environmental conditions regarding temperature and humidity but may not achieve desired ventilation • Patients have to perform unfamiliar exercise
4. Field exercise test: A reduction of FEV_1 ≥ 10% is diagnostic	• Types of exercise may be varied in accordance with sport practised. However, free running may be best suited • May be inconvenient for logistical reasons • It is not standardised for cardiovascular workload and for environmental conditions of humidity and temperature
5. Eucapnic voluntary hyperventilation test: A reduction in FEV_1 ≥ 10% is diagnostic	• Best known surrogate test to identify EIA • Currently recommended by IOC-MC • Has good specificity for active asthma • Well standardised • It is possible to achieve and maintain high minute ventilation higher than exercise test
6 Osmotic challenge test: (a) hypertonic 4.5% saline (b) mannitol dry powder inhalation ≥ 15% reduction in FEV_1 is considered diagnostic	• Laboratory based and relatively inexpensive compared with exercise test and more portable progressive protocol increases relative safety • Can simulate some reported-on exercise and demonstrates whether they are associated with bronchoconstriction • Inhalation of dry powder mannitol may be useful in office-based setting
7. Bronchial hyper-responsive to methacholine provocative concentration or dose PC_{20} or PD_{20} causing a 20% reduction in FEV_1, PC_{20} ≤ 4 mg/ml or PD_{20} ≤ 2µmol for those not taking inhaled steroid .	• Not widely available and laboratory based • Non-specific test of bronchial hyper-responsiveness • May not necessarily diagnose • Has low sensitivity to detect EIB in elite athletes

Table 3 The diagnostic evaluation in EIA

in patients with EIA.[59–61] Leukotriene modifiers include montelukast, zarfirlukast, and zileuton. They have become attractive as a result of their efficacy, once-a-day dosing, and oral formulation; and compare well with long-acting β2 bronchodilator. In addition, tolerance to medication and rebound worsening after discontinuation of treatment were not seen.

Inhaled corticosteroids

These are recommended as first-line therapy in terms of controlling medication in athletes who have asthma and experience EIA. They improve asthma symptoms by reducing airway inflammation and bronchial hyperreactivity. In a 6-week study of 40 adults with asthma, the post-exercise fall in FEV_1 was 22% after 6 weeks of placebo and 7% after inhalation of 0.8 mg of budesonide, an almost 70% reduction in airway response.[62] Good control of underlying inflammation in asthmatics reduced the need for rescue medication. They do not have immediate bronchodilator effects and are not effective if used alone prior to exercise. The efficacy of inhaled corticosteroid has not been studied in non-asthmatic athletes who have EIB.

Other drugs

Other drugs that have been tried in EIAasthma include oral theophylline. However, its long onset of action limits its use in the athletic setting. Calcium blockers have been demonstrated to protect against EIA.[63,64] Anticholinergic and antihistamines have not been found useful against EIA; other drugs include heparin and frusemide.

Overall, the treatment should follow common guidelines after appropriate diagnosis, and taking into consideration (when prescribing for athletes) the rules set up by doping authorities: the World-AntiDoping Agency (www.wada-ama.org) for international sports and the IOC-MC for the Olympic Games. The regulations for the use of asthma drugs among athletes have been repeatedly changed so physicians treating asthmatic athletes should be aware of present regulations. Applications for the use of drugs are generally made before the event to the respective authorities.

The non-pharmacological therapy

The success of pharmacological therapy also depends on non-pharmacological therapy, which includes activity modification, improvement in ambient air conditions, and patients' education. Management should begin with an effort to increase physical conditioning as improving fitness lowers minute ventilation for a given workload and thereby decreases the likelihood of EIA.[65] A warm-up can also be used effectively in decreasing the airway response to subsequent exercise, as this is the only natural precipitant of asthma that induces tachyphylaxis.[66] Cooling down or slowly lowering the exercise rate instead of stopping abruptly also has a beneficial effect by making airway rewarming and the resultant vascular dilation and oedema more gradual and less intense.[67]

Some other strategies to control the characteristics of inhaled air include breathing through the nose rather than through the mouth; this will filter, warm, and humidify the inspired air. Wearing a face mask or scarf, especially in a dry, cold environment, can also ameliorate EIA.

Education can boost the effectiveness of other therapeutic intervention. Patients must be aware of the conditions that exacerbate the disease and know how to avoid them, any early signs of impending attacks, and the appropriate use of medication.

Medication	Route of administration	Effectiveness in EIA
Short-acting β2 agonists		
Salbutamol	Aerosol	Excellent
Terbutaline	Aerosol	Excellent
Long-acting β2 agonists		
Salmeterol	Aerosol	Excellent
Formoterol	Aerosol	Excellent
Mast cell stabilisers		
Sodium cromoglycate	Aerosol	Good
Nedocromil sodium	Aerosol	Good
Antileukotrienes		
Montelukast	Oral	Excellent
Zafirlukast	Oral	Excellent
Zileuton	Oral	Excellent
Corticosteriods		
Beclomethasone	Aerosol	Excellent (prophylaxis)
Budesonide	Aerosol	Excellent (prophylaxis)
Fluticasone	Aerosol	Excellent (prophylaxis)
Flunisolide	Aerosol	Excellent
Methylxanthines		
Theophylline	Oral	Good

Table 4 Some of the drugs used for treating EIA

The differential diagnosis

EIA is easily recognisable and can be demonstrated by the exercise test; however, some conditions can mimic it and also cause exercise-induced respiratory symptoms that do not respond to standard therapy.

The closest differential diagnosis is exercise-induced laryngeal dysfunction, which is an abnormal laryngeal response to exercise that encompasses three different

but closely related entities:

- exercise-induced paradoxical vocal cord dysfunction;
- exercise-induced laryngeal prolapse;
- exercise-induced laryngomalacia.

All these conditions present as exercise-induced inspiratory stridor and are more common among female athletes during adolescence. They present immediately after maximal exercise with typical pattern of variable extra-thoracic obstruction and flattening of the maximal inspiratory curve in the flow volume loop during spirometry. Laryngoscopy is abnormal during acute episodes.

Another differential diagnosis is hyperventilation during exercise, although the pathophysiological mechanism in EIA is thought to be due to increased ventilation during exercise and voluntary hyperventilation with dry air is a surrogate laboratory test for EIA. Hyperventilation during exercise can produce respiratory symptoms not directly linked to bronchial obstruction but hypocapnia.[69,70] Other conditions that can produce respiratory symptoms during exercise include swimming-induced pulmonary oedema and exercise–induced arterial hypoxaemia.

Gastro-esophageal reflex disease (GERD) is another possible cause of exercise-induced respiratory symptoms and chronic cough and dyspnoea are common extra oesophageal symptoms of GERD. Other chronic disorders such as heart disease may have an effect upon physical performance and thus be a possible differential diagnosis. Poor physical fitness may be another differential. All these conditions should be considered and ruled out with a thorough physical examination and appropriate investigation in order that athletes do not receive unnecessary medication for asthma, which would not improve their exercise-induced respiratory symptoms.

Conclusion

Exercise tailored to the patient's asthma severity has physical, social, and emotional benefits. Early recognition and correct diagnosis are the first steps towards appropriate management. Important therapeutic strategies include individual environmental control measure exercise advice, and prophylactic pharmacological treatment.Moreover, better education of physicians, athletes, and coaches should make them aware of possible exercise-induced respiratory symptoms that are caused by other diseases.

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