

Dyspnoea in the competitive paediatric athlete

S Kansra

H Vyas

Abstract

Adolescent athletes are participating in sport in highly competitive environments. Dyspnoea associated with exercise is a common reason they are referred to paediatricians and respiratory specialists. The presentation with dyspnoea in adolescent athletes needs teasing out on history, examination and investigations from numerous conditions including exercise-induced bronchoconstriction, exercise-induced asthma (where exercise is a trigger for exacerbation of pre-existing asthma), vocal cord dysfunction, laryngeal dysfunction and physiological limitation to exercise.

Exercise-induced bronchoconstriction is diagnosed by evaluating the magnitude of fall in FeV1 post exercise. This bronchoconstriction can be completely ameliorated by short acting beta agonists given prior to exertion. Their use is permitted by the WADA but there are concerns over tachyphylaxis with long-term use. Warm up before strenuous exercise can also help in limiting the degree of bronchoconstriction. Athletes with pre-existing atopic asthma benefit from inhaled corticosteroids and leukotriene receptor antagonists.

Exercise associated upper airways obstruction maybe due to abnormal adduction of vocal cords or prolapse of the arytaenoid (subglottic) region. There may be psychological overlay. Diagnosis is by flexible nasoendoscopy and assessing the flow volume loops. Management is by reassurance and speech therapy.

Exercise-induced hyperventilation and physiological limitations to exercise may also lead to perception of dyspnoea. Absence of cardiopulmonary abnormalities during exercise points towards these diagnosis.

keywords asthma; athlete; dyspnoea; vocal cord dysfunction

Introduction

Young adolescents participating in sports at the highest levels are highly motivated and endure strenuous training regimens in sometimes unfriendly environments. These activities place extraordinary physical and psychological stresses on these young athletes. While they are susceptible to various respiratory ailments similar to the general population, that may have an adverse impact on their performance; they may additionally encounter certain unique respiratory problems related to the high intensity exercise and demands of competitive sport. The

commonest reason these young athletes present to a respiratory physician is a feeling of dyspnoea on exercise or worsening of performance. This review aims to look at the various aetiologies of dyspnoea in this population and also the diagnostic and treatment strategies in these conditions.

A competitive athlete for the purpose of this review would be considered as someone who is participating in sports at the county or national level. Dyspnoea is a clinical term signifying a subjective sensation of breathlessness or shortness of breath experienced by individuals. The sensation of dyspnoea derives from interactions between multiple physiological, psychological, social, and environmental factors that may then induce secondary physiological and behavioural responses.

The presentation with dyspnoea in adolescent athletes needs teasing out on history, physical examination and investigations from numerous conditions such as

- Exercise-induced bronchoconstriction
- Exercise-induced asthma
- Vocal cord dysfunction
- Laryngeal dysfunction
- Physiological limitation to exercise (Table 1)

Exercise-induced bronchoconstriction (EIB)

Elite or highly trained athletes participating in sports such as long distance running, cross-country skiing, swimming and ice hockey are exposed to adverse environmental factors such as inhalant allergens, presence of chlorine in pool water and cold air. Sustained increase in ventilation as a result of frequent high intensity training are responsible for an increased risk of exercise-induced bronchoconstriction (EIB) in competitive endurance athletes. This risk is exacerbated in atopic athletes.

An observation of a greater than 10 % drop in FEV1 from pre-exercise levels after 20–30 minutes of exercise is taken to be suggestive of EIB. The term exercise-induced asthma (EIA) is imperfect as it implies that exercise causes asthma rather than being a trigger that exacerbates it. EIB especially in athletes can be the only manifestation in the absence of other symptoms and signs of asthma. EIB is a more accurate descriptive term and for the purposes of this review, EIB will be used preferably.

Prevalence

Many studies report increased prevalence of asthma and EIB in elite athletes. Data from last 3 Olympic Games indicated that a high proportion of endurance athletes were using b2 agonists. Athletes have a significantly greater prevalence of doctor diagnosed asthma, use of asthma medication and allergic rhinitis. It also seems that the higher the level of performance of the athlete, the greater is the prevalence of asthma. Athletes who were competing at the Commonwealth Games level had a higher prevalence of asthma than ordinary public but lower prevalence as compared to Olympic athletes. Paradoxically asthmatic Olympic athletes have outperformed their peers at both summer and winter Olympic Games from 2002 to 2010.

Pathogenesis

Air gets heated and humidified during inspiration and rapid breathing while exercising, causes an increased loss of heat and water. Low inspired air temperatures (more convective loss) and breathing in dry air (more evaporative loss) produces more

S Kansra MRCPCH is Speciality Registrar (ST8), in Nottingham Children's Hospital, Nottingham University Hospitals NHS Trust, Nottingham, UK.

H Vyas DM(Notts) FRCP FRCPC is Professor in PICU and Respiratory Medicine at Nottingham University Hospitals, Queen's Medical Centre Campus, Nottingham, UK.

Major presentation in competitive paediatric athletes

Exercise-induced bronchoconstriction	Dyspnoea shortly after cessation of exercise Gradual improvement on its own or with SABA
Exercise-induced upper airways obstruction	Dyspnoea during exercise (maximal exertion) Symptoms resolve once exercise is stopped Inspiratory stridor may be heard
Exercise-induced hyperventilation	Hyperventilation during exercise Decreased end tidal CO ₂ during exercise Related to anxiety
Physiological limitation to exercise	Usually well-trained athletes Older and female athletes Inefficient alveolar-arterial exchange and expiratory flow limitations

Table 1

cooling of mucosa and rapid breathing accentuates these losses. The pathway by which thermal events lead to bronchial narrowing remains uncertain although according to different theories changes in osmolality of periciliary layer and cooling-rewarming gradients post exercise are argued to be the cause of the subsequent obstruction.

According to the first theory, airway-cooling causes parasympathetic nerve stimulation. This in turn leads to a reflex bronchoconstriction and a reflex venoconstriction of bronchial venules to conserve heat. At cessation of exercise the increased minute ventilation and cooling ceases and there is rebound vasodilation. So both smooth muscle constriction and mucosal oedema due to reflex vasodilatation lead to decrease in size of bronchial lumen and increased airway resistance.

Alternatively others believe that hyperventilation causes airway drying which increases the osmolality of the extracellular fluid of bronchial mucosal membranes. This causes an efflux of intracellular water to the extracellular space causing an intracellular ion concentration. This leads to release of mediators like eicosanoids and histamine that cause bronchoconstriction.

Airway inflammation in elite athletes has been observed in swimmers, ice hockey players and cross country skiers. These groups of athletes have been shown to have higher inflammatory cell counts in induced sputum as compared to controls. This inflammation seems to remain active or aggravate during follow up. Increased ventilation and exposure to cold and a mixture of irritant agents induce symptoms and airway inflammation in elite athletes.

Symptoms of EIB

The clinical presentation of EIB is quite characteristic. The airways dilate during exercise. When the workload is over; airway obstruction begins almost immediately and then progresses to reach its peak in about 5–10 minutes. Spontaneous resolution occurs over 30 minutes.

The severity of exercise-induced asthma varies with both the type of exercise and the nature of the environment. More strenuous exercise leads to greater ventilatory demand resulting in more severe bronchoconstriction. Running limits airflow more than jogging, which in turn limits it more than walking. Temperature and humidity of the inspired air further affect the severity of obstruction. Obstruction is maximized in cold dry air and is least in warm humid air. Patient's sensitivity to exercise and intensity of bronchoconstriction induced by activity are related to underlying reactivity. Though exercise does not cause prolonged or intense airway obstruction that is dangerous or requires hospitalization it may be detrimental to the performance of the adolescent athlete. Exercise does not induce long-term deterioration of lung function.

Diagnosis

In EIB, the symptoms of airway obstruction develop **after** cessation of exercise. Symptoms **during** exercise are as a result of changes in lung function arising from an existing airflow limitation or represent excessive dyspnoea associated with inefficient delivery of oxygen to the working muscles.

Clinical diagnosis of EIB may be relatively inaccurate. The diagnosis of EIB is confirmed by changes in pulmonary functions after bronchial provocation. The most commonly used provocation test is exercise. A fall in FeV1 of 10% or more after an exercise challenge is usually considered suggestive. Exercise protocols based on achievement of specific physiological parameters such as heart rate are often used. Treadmills or bicycle ergometers can be used to achieve exercise endpoints. The exercise load needs to be high as assessed by the heart rate, the test should be standardized in respect to environment temperature and humidity. The exercise test has high specificity but lower sensitivity especially in the adolescents on inhaled steroids.

This approach to diagnosis of EIB may need to be modified in athletes. Subjects may be required to be tested in their usual environment (sport specific) at times.

Surrogate provocation tests have been used and are accepted by sports bodies in diagnosis of exercise-induced asthma. There may be advantages to use surrogate tests especially when evaluating children and elderly or obese adults.

Since EIB is thermally mediated, hyperventilation with dry cold air (eucapnic hyperventilation) has also been employed as a stimulus instead of exercise. This is a physically demanding test and requires the athlete to perform hyperpnoea by inhaling air containing 5% carbon dioxide at ventilation equivalent to 30 times the baseline FeV1. Other indirect stimuli often used are hyperosmolar aerosols Mannitol and hypertonic saline. Additionally, methacholine and histamine are also accepted by the IOC and WADA for identifying bronchial hyper responsiveness.

International Olympic Committee (IOC) accepted following provocation tests as a basis to apply for a therapeutic use exemption (TUE) for asthma medication at the Beijing Olympic games.

- Eucapnic voluntary hyperpnoea test
- Exercise challenge in the laboratory or an exercise test in the field
- Hyperosmolar aerosols i.e. 4.5 g% saline or dry powdered mannitol
- Methacholine test

Download English Version:

<https://daneshyari.com/en/article/4172357>

Download Persian Version:

<https://daneshyari.com/article/4172357>

[Daneshyari.com](https://daneshyari.com)