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The athlete “out of breath”

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Summary

Athletes often complain about breathing problems. This is a crucial issue due to potential implications not only on their general health, but also on their competing performance. Asthma and exercise-induced bronchoconstriction are prevalent conditions in elite athletes, which leads doctors to rely most of the times on asthma medication to treat athletes feeling “out of breath”. However, there are several other conditions that may mimic asthma and cause dyspnea in athletes. Effective treatment of dyspnea requires appropriate identification and treatment of all disorders. Proper knowledge and accurate diagnosis of such entities is mandatory, since asthma medication is not effective in those conditions. Herein we review the most common differential diagnosis of dyspnea in athletes, and describe the diagnostic strategies in order to increase awareness and to improve doctor’s confidence on dealing with these patients.

Introduction

Regular physical exercise and participation in sports are considered to be important components of a healthy life and are recommended for all individuals (1). However, in patients with respiratory symptoms, physical exertion is a potent stimulus that can produce episodes of airway distress. These individuals may show less tolerance to exercise due to the worsening of respiratory symptoms during exercise and this can preclude them from playing sports or attempting to keep fit. For instance, regarding asthma, evidence has shown that physical training improves cardiopulmonary fitness (2) and may even improve quality of life of both asthmatic children and their caregivers (3). Therefore, given the beneficial effects of exercise, every effort should be taken by doctors to recognize respiratory diseases and yield all actions so they are not a limitation for its practice. This issue is particularly relevant when concerning athletes. Respiratory

symptoms induced by exercise have potential implications not only on athlete’s general health, but also on their training capacity and competing performance.

Respiratory complaints pose several issues unique to athletes as they face special challenges managing their respiratory symptoms while practicing sport. Discrimination between physiological and pathological limitations to maximum exercise is difficult given the heavy training with extremely high level of physical fitness and maximum oxygen uptake (VO₂max) reached (4). This limits the ability of diagnosis. On the other hand, some athletes will not reveal their respiratory symptoms due to fear that the disclosure of their disorders will be detrimental. Therefore, athlete’s respiratory disorders often perplex, frustrate and distress both patients and their physicians. However, as stated by International Olympic Committee, “*all care should be taken to ensure that sports do not affect the health or welfare of the participants*”. So, the aim of this study is to review

several respiratory diagnostic hypotheses and how to differentiate and approach each, in order to provide the best treatment for athletes complaining of breathless. As a general purpose, we aim to demystify this subject and improve doctor’s confidence on dealing with these patients.

Asthma, the most common respiratory disorder among athletes

Well known by *Aretaeus* since the year 100 AD (5), asthma induced by sport practicing is not always easy to describe and recognize. For this reason, in 2008, a Joint Task Force was established by the European Respiratory Society (ERS) and the European Academy of Allergy and Clinical Immunology (EAACI) in cooperation with GA²LEN; accordingly, exercise-induced asthma (EIA) is defined as the presence of lower airway obstruction and symptoms of cough, wheezing or dyspnea induced by exercise in patients with underlying asthma (4); the same clinical presentation occurring after exercise in subjects without asthma is called exercise-induced bronchoconstriction (EIB), and does not imply underlying chronic asthma (4). For the purposes of this document, terminology EIA or EIB will be used interchangeably because, when occurring with exercise, presentation and treatment of both conditions are similar.

Substantial data show EIB occurs very commonly in athletes at all levels (6). Many studies performed in elite-level athletes have documented a prevalence of EIB varying between 30 and 70%, depending on the population studied and the methods implemented (7). In any case, asthma is definitely the most common chronic medical condition among Olympic athletes (8). Asthma is well-known to be more frequent in athletes than in general population, as well as more prevalent in elite athletes, particularly those who participate in endurance events, in swimming, and winter sports, than in recreational athletes (4,9). Several studies indicate subjects prone to EIB have increased levels of exhaled nitric oxide, leukotrienes, and airway epithelial shedding (10-12). The epithelium may play a key role in sensing the transfer of water and heat out of lower airways, but also in activation of sensory nerves (6,13). Also, atopy and type of sport are known to influence risk of asthma in athletes (9). Furthermore, in some specific sports, environmental training and competing conditions may also have a detrimental effect to airways (13).

Diagnosis is particularly relevant because of potential implications on performance both in training and competition, since airway narrowing during exercise compromises ventilatory capacity and efficiency (14). Additionally, asthma has been pointed out as a significant risk factor for unexplained death in young and healthy subjects (15), and a high proportion of asthma-related deaths have been reported in athletes associated with a sporting event (16). Besides, a subgroup of athletes who are asymptomatic present objective evidence of EIB (17), which raises the question of its potential underdiagnosis and the

resulting underperformance. On the other hand, untreated or undertreated asthma results in chronic sustained inflammation associated with persistent epithelial damage, which contributes to airway remodeling and fibrotic changes, fixed obstruction and progressive lung function decline over time (18).

It has become quite clear the importance of a correct diagnosis. *But how to recognize EIA?*

Clinical presentation of EIA includes wheezing, cough, shortness of breath and/or chest tightness, generally occurring within 5 to 30 minutes *after* intense exercise, sometimes, but unusually, during exercise (7,17). Gradual spontaneous improvement is common after ending exercise. Symptoms are often mild to moderate in severity and may cause impairment of athletic performance, but are often not severe enough to cause significant respiratory distress (6), which may mislead the doctor to pursue this diagnostic hypothesis.

Asthma-like symptoms in elite athletes are not necessarily associated with classic features of asthma (14). Athletes may not suffer from the obvious symptoms as regular asthmatic patients do, but rather cough (19) or some nonspecific complaints such as poor performance or “feeling out of shape”, abdominal pain, headaches, muscle cramps, fatigue, and dizziness (20). So, symptoms of EIB are variable and nonspecific, and the presence or absence of specific respiratory symptoms has very poor predictive value to objectively confirm EIB (6,19,21,22).

Physical examination can reveal expiratory dyspnea, expiratory wheezing or rhonchi and other signs of bronchial obstruction such as respiratory retractions (4,23) if the athlete is observed closely after training, but it is often normal during a scheduled appointment.

Not all breathing complaints mean asthma...

Although asthma is the most frequent respiratory chronic disorder in athletes, several other clinical entities can produce similar symptoms (24,25). Overlooking these conditions might therefore lead to wrong diagnosis and unsuccessful treatments. Actually, *it is quite interesting to note that most of the elite athletes who are referred for respiratory problems do not suffer from asthma or EIB* (26). Exercise-induced dyspnea, in particular, is associated with many disease processes and is a remarkably uncommon complaint among those who suffer from EIB (24). Wheezing or stridor can also be caused by other airway abnormalities and closely mimic EIB (14). On the other hand, in the particular case of athletes, their underlying high cardiorespiratory fitness make the diagnostic process even more complex, since also a variety of rare alternative diagnosis must be considered.

Exercise-induced stridor

Vocal cord dysfunction (laryngeal obstruction or inspiratory stridor) is one of the most frequent causes of exercise-induced

dyspnea in athletes. Symptoms such as shortness of breath, increased inspiratory effort, and stridor can be caused by exercise-induced laryngeal obstruction (EILO), and in many subjects only arise during exercise (24). There is no consensus definition, but the following has been proposed: an intermittent extrathoracic airway obstruction, mainly during inspiration, leading to dyspnea of varying intensity (27). Symptoms are thought to occur due to relatively small cross-sectional area of laryngeal orifice, which may be even further reduced by negative pressure created on inspiration during heavy exercise, and the paradoxical movement (adduction instead of normal abduction) of the vocal cords during inspiration (4,24). This condition is frequently associated with psychologically stressful events such as competitions. It has also been associated with gastroesophageal reflux (24). Elite athletes are more frequently affected than general population. Prevalence is reported between 5 and 35% of those athletes referred for routine evaluation for asthma and/or EIB (28,29). Prevalence of EILO appears to be gender related and is highest among young females (30). Differential diagnosis is important, as asthma treatment will have definitively no effect. However, it should be remarked that about half of athletes with asthma can present concomitant EIB (28). Laryngomalacia is a less common cause of exercise-induced stridor (24). This condition is characterized by collapse of the arytenoid area with normal vocal cord motion and primarily affects female competitive athletes who abruptly develop stridor at near peak exercise (31). Females may be predisposed to collapse because larynx is shorter and narrower than in males (24). Moreover, exertional inspiratory stridor may be caused by foreign body aspiration, poor-performance, psychogenic stridor, infectious croup, subglottic stenosis, and exercise-induced anaphylaxis, although these diagnoses are much more infrequent.

Upper respiratory tract infections

Athletes practicing regular strenuous exercise may be at increased risk of upper respiratory tract infections (URTIs) during periods of heavy exercise and for a couple of weeks following competition events (32,33). In contrast to moderate or intermittent physical activity, prolonged and intensive exertion causes changes in immunity that possibly reflects physiological stress and suppression (13). URTIs are so frequent in elite athletes that give rise to respiratory complaints over prolonged periods of time often related to competition seasons or heavy training blocks (34), and may resemble a chronic condition.

Poor physical fitness / Deconditioning

Poor physical fitness is a very frequent cause of exercise-induced breathless in children and adolescents testing because of respiratory complaints (35). It is not very common in elite athletes,

but may occur during the “off season” when they become deconditioned and an increase in respiratory drive with lesser amounts of exercise may be interpreted as pathologic (14). Deconditioned subjects have a lower lactate/ventilatory threshold, accumulating lactate and increasing minute ventilation with lesser amounts of exercise; excess lactate buildup results in exercise-associated increases in ventilation and ultimately hypoxemia (24).

Physiologic limitation

Normal physiologic exercise limitation was the most common reason for exercise-induced dyspnea after cardiopulmonary exercise testing in a study by Abuhasan et al (35). It occurred in 52% of referrals for EIB; of those, two thirds had normal or above normal cardiovascular conditioning. The dyspnea is likely related to the increase in ventilation which is necessary to meet increased metabolic demands of high intensity exercise. The increase in respiratory drive and work is a normal physiologic response to exercise but may be interpreted as pathologic by subjects who find that it limits their ability to perform to their expectations (14).

Rhinitis

Elite competitive athletes have a significant increased prevalence of allergic rhinitis and present a variety of both usual and rare symptoms and signs (14). Common symptoms of rhinitis include sneezing, anterior rhinorrhea, post nasal drip / chronic cough, and nasal obstruction (36). However, in athletes, the clinical presentation of rhinitis is frequently more subtle and might present as reduced exercise performance, fatigue, poor-quality sleep, and difficulty to recover after more demanding exercise sessions (36). Especially the lower performance and the cough induced by post-nasal dripping might mislead the doctors to pursue other respiratory disease such as asthma.

Exercise-induced hyperventilation and Dysfunctional breathing

Dysfunctional breathing is defined as chronic or recurrent changes in breathing pattern that cannot be attributed to a specific medical diagnosis; symptoms include exercise-induced breathlessness, as well as a variety of other asthma-like symptoms such as dyspnea with normal lung function, chest tightness, chest pain, deep sighing, frequent yawning and hyperventilation. Exercise-induced hyperventilation is a common physiologic response to exercise but also it may be interpreted as a primary problem since it can be associated with chest tightness and shortness of breath (24,37). The decreases in end-tidal CO₂ during exercise can be associated with chest discomfort perceived as dyspnea (37). An associated personality pattern

characterized by a high degree of competitiveness among the patients with perceived dyspnea from exercise-induced hypoxemia has been pointed out (37).

A higher incidence of moderate/severe rhinitis in patients with dysfunctional breathing has been reported (38), which is not surprising considering the consequences of oral breathing. The nasal congestion could be either the cause or the result of the abnormal breathing pattern with low PaCO₂ levels increasing nasal resistance.

Exercise-induced arterial hypoxemia

This condition occurs especially in well-trained athletes with high VO₂max and is thought to be primarily because of diffusion limitations and ventilation-perfusion mismatch (39). Hypoxemia develops at all exercise intensities with varying patterns and is more common in aerobically trained subjects (40). It is postulated that incomplete diffusion in the healthy lung may be because of a rapid red blood cell transit time through the pulmonary capillary (4). Ventilatory requirement rises with increased ionotropic and chronotropic capacities of the cardiovascular system induced by physical training; however, there is a limited capability of the airways and the lungs to produce higher flow rates or higher tidal volumes with little or no change in the pressure-generating capability of inspiratory muscles (41). Exercise-induced arterial hypoxemia is defined as reduced arterial oxygenation, which may result from a fall in PaO₂ (and thus also in SaO₂), from a rightward shift of the O₂ dissociation curve without a fall in PaO₂ or from a combination of these processes (42). It may occur in up to 50% of highly trained athletes (4,43-45). By virtue of their smaller lung volumes and airway diameters, women develop more mechanical ventilatory constraints during exercise, which may result in increased vulnerability to hypoxemia during exercise (40).

Other causes

Other chronic disorders are possible to less often cause exercise-induced symptoms in athletes. Heart diseases and other respiratory disorders should be also considered. In previously healthy persons, cardiac abnormalities are a rare cause of exercise-induced dyspnea (24). On the contrary, some pulmonary abnormalities can present with exercise-induced dyspnea. Chest wall or other musculoskeletal abnormalities can impair pulmonary mechanics and *pectus excavatum* has been associated with exercise intolerance and dyspnea (35); also mild scoliosis in adolescents has been linked with abnormal ventilator response to exercise (46). Pulmonary arteriovenous malformations and interstitial lung disease are very uncommon. Obesity, which may represent a differential diagnosis to EIA in the common asthmatic patient, is rare in most athletes.

In the particular case of swimmers and scuba-divers, attention must be paid to a rare condition called swimming-induced pulmonary edema (SIPE). SIPE occurs in well-trained water athletes after a heavy water training session (4). This condition was reported early in the 70s in previously healthy swimmers who developed typical symptoms of pulmonary edema together with a restrictive pattern in pulmonary function, which remained for up to one week after the swimming incident (4,47). Pulmonary edema is the accumulation of water in the lung extravascular spaces. This reflects a breakdown of the normal homeostatic mechanisms that maintain lung fluid balance, predominantly increased hydrostatic pressures and increased capillary membrane permeability (48). During exercise, elite athletes increase their cardiac output. A combination of additional pulmonary capillary bed recruitment and capillary distension mitigate against sharp rises in pulmonary artery pressures in healthy individuals. It is postulated that in susceptible individuals, compensatory mechanisms are overwhelmed. Increased hydrostatic capillary pressures result in mechanical failure, which produces exercise-induced pulmonary hemorrhage (48).

It is important to bear in mind that *more than one condition may coexist in a given subject*. In particular, EILO can be present concomitantly in about half of the athletes with EIB (28,29).

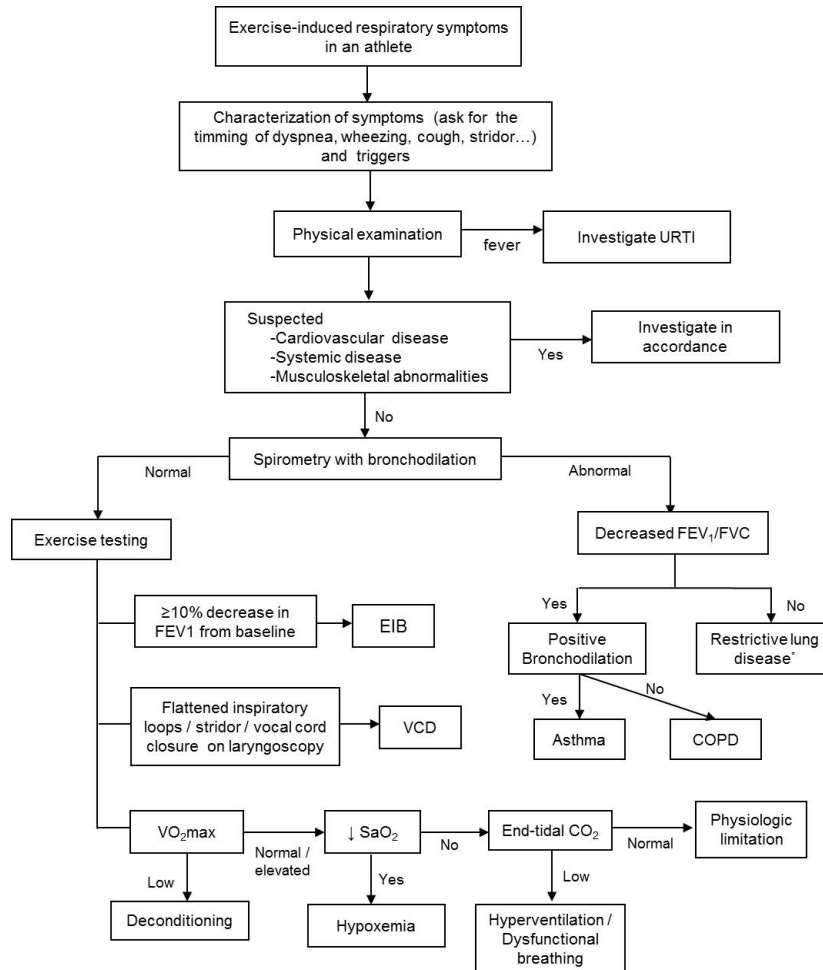
How to diagnose each respiratory disorder?

A systematic approach such as the one suggested in **figure 1** is often useful. In the specific case of competitive athletes, diagnosis of respiratory diseases poses several issues unique to this population. First, the majority of symptoms only occur when exercising at a high level of intensity and VO₂max, which are sometimes difficult to reproduce in the laboratory. To discriminate between physiological and pathological limitations to maximum exercise based on symptoms or resting exams is not easy most of the times (4). On the other hand, some athletes will not reveal their symptoms due to fear that their respiratory disorders disclosure will be detrimental (14), as others without asthma will try to secure asthma treatments in an attempt to gain a competitive advantage (7); although several studies proved that anti-asthmatic drugs do not enhance performance in healthy subjects (49), this is still a general misbelieve. Therefore, *objective evidence of respiratory diseases should always be part of these subjects' assessment*.

Asthma

Baseline spirometry is poorly predictive of asthma in competitive athletes (14). Often they record lung function values higher than the general population and so they may appear to be within the “normal” range, although, in reality, corresponds to a pulmonary deficit on the basis of what is expected for an athlete (9,50).

Figure 1 - Flow-chart for diagnosis of most common respiratory diseases in athletes. CO_2 : carbon dioxide; COPD: chronic obstructive pulmonary disease; EIB: exercise-induced bronchoconstriction; FEV_1 : forced expiratory volume in 1st second; FVC: forced vital capacity; SaO_2 : oxygen saturation; URTI: upper respiratory tract infections; VO_2max : maximum oxygen uptake; VCD: vocal cord dysfunction. *consider SIPE if occurring after a heavy water training, and in the presence of hemoptysis.



The evaluation of asthma requires therefore a combination of patient's history, clinical examination and judgment, as well as adequate tests, in a stepwise approach (4). However, as recommended by the Medical Commission of International Olympic Committee, in this special population is mandatory to obtain objective evidence to validate an asthma diagnosis by either a positive bronchodilator (figure 2) or bronchoprovocation test. Diagnostic tests and positivity criteria are presented in table 1. There are several bronchoprovocation tests that can be used and are approved for diagnosis, but the most recent guidelines recommend the use of a standardized exercise test (6). Exercise test has high specificity for asthma, but lower sensitivity. Standardization of the test is very important for the outcome: ex-

ercise load should be high, and it should also be standardized with regard to environmental temperature and humidity (6). The severity of EIB can be graded as mild, moderate, or severe if the percent fall in FEV1 from pre-exercise level is $\geq 10\%$ to $< 25\%$, $\geq 25\%$ to $< 50\%$, and $\geq 50\%$, respectively (6) (figure 3). A number of surrogates for exercise testing have been developed that may be easier to implement. These surrogates are validated for asthma diagnosis and include eucapnic voluntary hyperpnea, hyperosmolar aerosols, and dry powder mannitol (table 1); however, only exercise test allow for differential diagnosis using only one method. Testing directly for bronchial responsiveness using bronchial challenge with methacholine has a higher sensitivity, but a lower specificity, for asthma (23,51-

Figure 2 - A positive bronchodilation test (reversibility of 16% and 550 mL after 400 µg inhaled salbutamol).

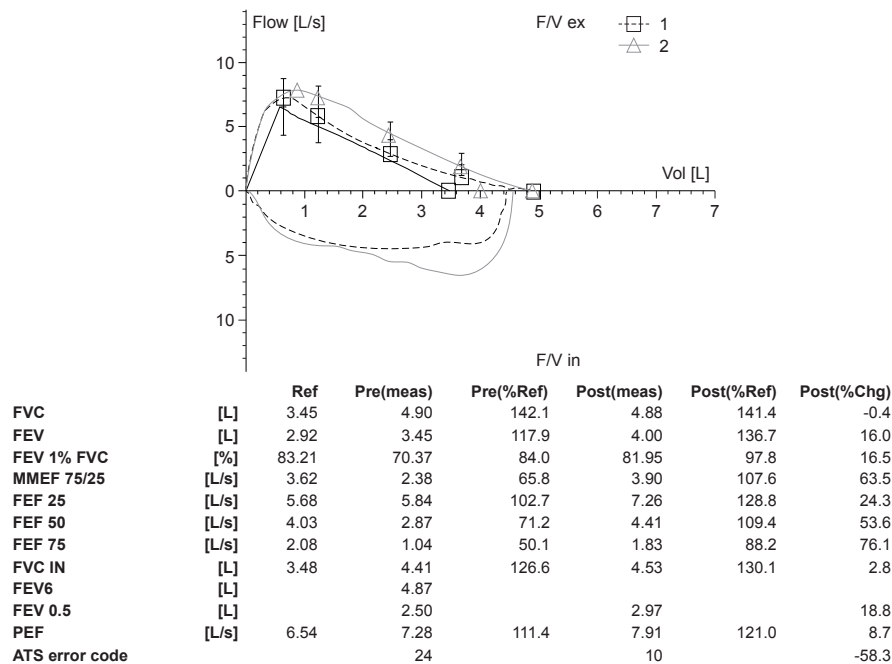
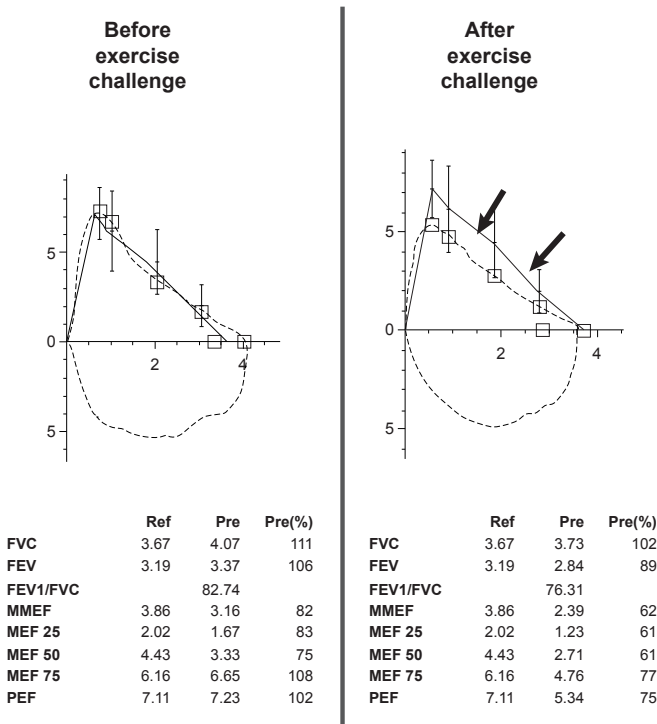


Table 1 - Methods for diagnosis and positivity criteria set by the International Olympic Committee to document exercise-induced bronchoconstriction in athletes.

Method	Protocol	Positivity criteria
Bronchodilation test	FEV ₁ before and 15 minutes after inhalation of a standard beta-2 agonist	Increase in FEV ₁ ≥ 200mL and ≥ 12% from baseline
Bronchial provocation challenges		
Methacholine test	Provocative dose (PD ₂₀) or concentration (PC ₂₀) of inhaled methacholine inducing a FEV ₁ decrease from baseline ≥ 20%	PC ₂₀ ≤ 4 mg/ml or PD ₂₀ ≤ 400 µg (cumulative dose), or ≤ 200 µg (noncumulative dose) in those not taking ICS PC ₂₀ ≤ 16 mg/ml or PD ₂₀ ≤ 1600 µg (cumulative dose) or ≤ 800 µg (noncumulative dose) in those taking ICS for at least 1 month
Eucapnic voluntary hyperpnoea	FEV ₁ before and within 30 min of 6 min dry (or dry and cool) air inhalation at 85% of predicted maximum voluntary ventilation	≥10% decrease in FEV ₁ from baseline
Hypertonic saline inhalation	FEV ₁ before and after inhaling 22.5 mL of 4.5 g% NaCl	≥15% decrease in FEV ₁ from baseline
Mannitol inhalation	Provocative dose of inhaled mannitol inducing a FEV ₁ decrease from baseline ≥15% (PD ₁₅ M)	PD ₁₅ M ≤ 635 mg of mannitol
Exercise challenge (field or laboratory)	FEV ₁ before and within 30 min of exercise challenge achieving heart rate > 85% for at least 4 min	≥10% decrease in FEV ₁ from baseline

FEV₁: Forced expiratory volume in one second; ICS: inhaled corticosteroids; NaCl: sodium chloride.

Figure 3 - A positive bronchoprovocation challenge with exercise. A reduction of 16% in FEV1, 5 minutes after exercise challenge test is observed, representing mild (fall in FEV1 from pre-exercise level $\geq 10\%$ and $< 25\%$) bronchial hyperresponsiveness to exercise.



53) and if negative represents a useful measurement to exclude that diagnosis (23).

Detailed information about past or current atopic disorders should be addressed and skin prick tests (SPT) or specific IgE (in case of the SPT could not be performed) should also be carried out in order to assess for atopy (14).

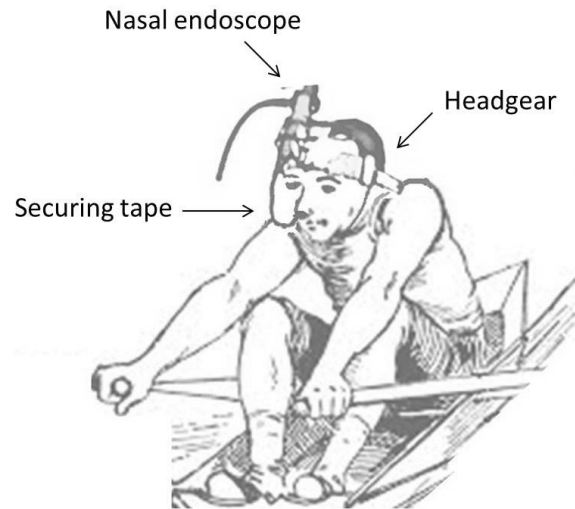
Exercise-induced stridor

Diagnosis may be suspected by history of inspiratory wheeze and throat tightness. Diagnosis of VCD is suggested by flow-volume loops which may reveal variable blunting of inspiratory loop (24). A ratio of less than one for maximal inspiratory flow at 50% of forced vital capacity / maximal expiratory flow at 50% of forced vital capacity, after a methacholine bronchial provocation, has been taken as suggestive of EILO (27).

Definitive diagnosis can be made by laryngoscopy during a maximum-intensity treadmill run or other exercise depending on the sport practiced (figure 4). That reveals audible inspiratory stridor occurring during maximum intensity and observable

evidence of the paradoxical motion of the vocal cords. Typical findings from laryngoscopy are inspiratory vocal cord closure with posterior “chinking” (a small opening at the posterior aspect of the cords) or, less commonly, complete closure (24). Laryngomalacia is differentiated from vocal cord dysfunction by fiberoptic rhinolaryngoscopy.

Figure 4 - Laryngoscopy during a maximum-intensity rowing session.



Upper respiratory tract infections

There are more uncertainties than evidence based facts on the nature of URTIs associated with exercise, particularly in high performance athletes (54). Physician confirmation of an infective cause, based on clinical signs and symptoms, has until recently been considered the ‘gold standard’, due to the costs associated with identification of the underlying causes of upper respiratory symptoms and the delay in obtaining results of investigative tests (54). However, recently, the ‘gold standard’ of physician verified diagnosis of URTIs has also come under scrutiny, and has been found less than ideal (55). The infectious etiology of upper respiratory symptoms has been questioned since few of them had no infectious agent identified, leading to hypothesize that symptoms might be due only to an increased inflammation state (54). A definite diagnosis of bacterial or viral infection must therefore be actively pursued with laboratory investigations.

Poor physical fitness / Deconditioning

During an exercise challenge, oxygen consumption (VO_2) peak is decreased, peak heart rate is normal/slightly decreased, the breathing reserve is $> 20\%$ of maximal voluntary ventilation, the

ventilatory equivalent for CO₂ at anaerobic threshold is normal, as well as SpO₂ (> 95%, with 4% drop during exercise) (56,57).

Physiologic limitation

Breath-by-breath analysis of gas exchange during exercise identifies exercise-induced dyspnea in very well-conditioned teenage athletes who simply reach a point of exercise limitation well beyond their anaerobic threshold and that interpret this perceived physiologic dyspnea as a pathologic condition (14,37). It is easily differentiated from poor physical fitness by maximal oxygen consumption (VO₂max).

Rhinitis

Diagnosis of allergic rhinitis in athletes is based in the concordance of a suggestive history of allergic symptoms and physical examination, supported by diagnostic tests (58,59). A thorough allergic history remains the best diagnostic tool available (59,60). In an athlete with persistent symptoms or when an allergic etiology for upper respiratory symptoms is suspected, SPT with standardized allergens or measurement of allergen-specific IgE in serum (in case SPT could not be performed) should be used (36).

Nasal challenge tests are not necessary to confirm diagnosis (36). Imaging of the nose and sino-nasal cavity is used to differentiate the source of sino-nasal symptoms, relation of sino-nasal problem with surrounding structures and the extent of the disease (59). To evaluate severity in an objective way, measurements of nasal obstruction and smell are used (58), but often unnecessary in daily clinical practice. Nasal patency can be monitored objectively using nasal peak inspiratory and expiratory flow, acoustic rhinometry that measures the volume of nasal cavity, and rhinomanometry that measures nasal airflow and pressure (59).

Exercise-induced hyperventilation and Dysfunctional breathing

A symptom-limited exercise test with a cycle ergometer has been proposed, starting with unloaded cycling and using a step-wise increase in work load of 16 W/min (56). The procedure is performed in room air according to current guidelines for exercise testing, with continuous monitoring of ECG, blood pressure and oxygen saturation. The test is continued until symptom-limitation (dyspnea), in the absence of chest pain or ECG abnormalities. Presence of an abnormal breathing pattern such as an increase in deep sigh rate in response to exercise or unsteadiness and irregularity of breathing with no evidence of bronchoconstriction on spirometry and good exercise tolerance is considered diagnostic for dysfunctional breathing (38). Abnormal breathing pattern should be assessed by the same physician in comparison with baseline breathing pattern (38).

Other proposed standardized methods for diagnosing dysfunctional breathing are Respiratory Induction Plethysmography and Manual Assessment of Respiratory Motion, a technique evaluating and quantifying breathing pattern. Both methods are able to differentiate between abdominal and thoracic breathing patterns (61).

Exercise-induced arterial hypoxemia

When EIAH is present, it usually peaks at or near maximal exercise intensity. Reduction in arterial oxygen saturation might be observed by using pulse oximetry. Noninvasive ear oximetry is commonly used in exercise studies in healthy subjects who would not be expected to desaturate < 10%. Thus the great majority of these changes lie on the relatively flat portion of the HbO₂ dissociation curve, and it is very difficult to accurately quantify changes in SaO₂, and especially in PaO₂, with this indirect measurement (42). Therefore, EIAH must be identified by direct measurements of arterial blood gases, and these measurements should be corrected to the *in vivo* arterial blood temperature (42). Arterial blood temperature is commonly measured directly or estimated from esophageal temperature. Temperature correction is very important, because temperature commonly increases ~1.5-2°C over the course of a standard progressive exercise test and even more in heavy constant-load endurance exercise (42). Failure to temperature-correct PaCO₂ would correspondingly overestimate ideal alveolar PO₂. Mild EIAH is defined as PaO₂ saturation of 93-95% (or 3-4% < rest), moderate EIAH as 88-93%, and severe EIAH as < 88% (42).

Other causes

Other hypothesis must follow diagnostic procedures course in accordance to what is suspected; if cardiac symptoms are present, a proper cardiac evaluation must be performed. In the presence of fatigue / weakness, consider the hypothesis of a myopathy. Physical examination may reveal other musculoskeletal abnormalities.

SIPE is easily identified by the occurrence of hemoptysis, cough and respiratory distress after heavy swimming exercises. A restrictive lung function can occur until one week afterwards (47), and infiltrates on chest radiographs may be observed.

Conclusions

It is crucial to recognize and accurately diagnose breathing complaints in athletes because it has potential implications not only on their general health, but also on their competing performance. Effective treatment of dyspnea requires appropriate identification of underlying disorder. Due to high prevalence of asthma in athletes, doctors often rely *a priori* on asthma medi-

cation to manage respiratory symptoms. Proper knowledge and accurate diagnosis of other entities is mandatory, since asthma medication is not effective in such cases. Self-reported symptoms are most of the time misleading doctors and are poor diagnostic predictors of disease. So, objective testing is frequently required and can prevent exposing patients to medications that are ineffective and have potential adverse side effects.

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