

International Olympic Committee (IOC) consensus statement on acute respiratory illness in athletes part 2: non-infective acute respiratory illness

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ABSTRACT

Acute respiratory illness (ARill) is common and threatens the health of athletes. ARill in athletes forms a significant component of the work of Sport and Exercise Medicine (SEM) clinicians. The aim of this consensus is to provide the SEM clinician with an overview and practical clinical approach to non-infective ARill in athletes. The International Olympic Committee (IOC) Medical and Scientific Committee appointed an international consensus group to review ARill in athletes. Key areas of ARill in athletes were originally identified and six subgroups of the IOC Consensus group established to review the following aspects: (1) epidemiology/risk factors for ARill, (2) infective ARill, (3) non-infective ARill, (4) acute asthma/exercise-induced bronchoconstriction and related conditions, (5) effects of ARill on exercise/sports performance, medical complications/return-to-sport (RTS) and (6) acute nasal/laryngeal obstruction presenting as ARill. Following several reviews conducted by subgroups, the sections of the consensus documents were allocated to 'core' members for drafting and internal review. An advanced draft of the consensus document was discussed during a meeting of the main consensus core group, and final edits were completed prior to submission of the manuscript. This document (part 2) of this consensus focuses on respiratory conditions causing non-infective ARill in athletes. These include non-inflammatory obstructive nasal, laryngeal, tracheal or bronchial conditions or non-infective inflammatory conditions of the respiratory epithelium that affect the upper and/or lower airways, frequently as a continuum. The following aspects of more common as well as lesser-known non-infective ARill in athletes are reviewed: epidemiology, risk factors, pathology/pathophysiology, clinical presentation and diagnosis, management, prevention, medical considerations and risks of illness during exercise, effects of illness on exercise/sports performance and RTS guidelines.

INTRODUCTION

Protection of the health of athletes and illness prevention have been formally identified as high priorities by the International Olympic Committee (IOC) since 2009.¹ Acute illness that threatens

Key points

- ⇒ Acute illness accounts for up to ~50% of all medical consultations at major sporting events with ~50% of cases involving the respiratory system.
- ⇒ Acute respiratory illness (ARill) includes acute respiratory infections and non-infective ARill.
- ⇒ Causes of non-infective ARill in athletes can involve predominantly the upper (eg, structural nasal obstruction, acute allergic and non-allergic rhinitis/rhinosinusitis and exercise-induced laryngeal obstruction) or the lower airways (lower airway dysfunction and rarer conditions such as excessive dynamic airway collapse) and tracheobronchomalacia).
- ⇒ In this consensus, we provide the Sport and Exercise Medicine clinician with an overview and practical clinical approach to the diagnosis, management, return-to-sport decision-making and prevention of these non-infective causes of ARill in athletes.

athlete health is a significant component of Sport and Exercise Medicine (SEM) clinicians' work at international single sport²⁻⁴ and multisport events, including the Olympic^{5,6} and Paralympic Games.⁷ The IOC Medical and Scientific Committee identified 'acute respiratory illness (ARill) in the athlete' as a high priority area to protect the health of athletes and is the focus of this IOC Consensus paper.

Acute illness can account for ~50% of all medical consultations at major sporting events,^{2-4,7} and in ~50% of cases involve the respiratory system.^{2-4,7} Although the majority of ARill in athletes are suspected acute respiratory infections (ARinf), mostly by clinical diagnosis,^{2-4,6,8} ARill can have a non-infective cause.⁴

The aim of this consensus is to provide the SEM clinician with an overview and practical clinical approach to acute non-infective respiratory illness in athletes. This consensus document complements part 1, which focuses on ARinf.⁹ The specific focus



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of part 2 is a review of epidemiology, risk factors, pathology/pathophysiology, clinical presentation and diagnosis, management, prevention, medical considerations, risks of illness during exercise, effects of illness on exercise/sports performance and return-to-sport (RTS) guidelines. This consensus group recognises that there are international guidelines for several of the respiratory conditions. In this consensus statement, we aim to be consistent with these guidelines, but we specifically highlight unique aspects of non-infective ARill in athletes.

METHODS AND CONSENSUS GROUP WORKFLOW

The IOC Medical and Scientific Committee appointed a chair (MS) for this consensus group in July 2019. This consensus statement process took a unique approach, following key steps as detailed in part 1.⁹ In brief, subgroups of the IOC Consensus group were originally established to review the literature in the following six areas: (1) epidemiology/risk factors for ARill, (2) infective ARill, (3) non-infective ARill, (4) acute asthma/exercise-induced bronchoconstriction (EIB) and related conditions, (5) effects of ARill on exercise/sports performance, medical complications/RTS and (6) acute nasal/vocal cord dysfunction presenting as ARill. A chair, who was a 'core' member of the consensus group, was identified for each subgroup. The six subgroups were created, and subgroup chairs nominating and adding content-specific expert 'corresponding' members to their group. Following the work of the subgroups, the sections of the consensus documents were allocated to 'core' members for drafting and internal review. An advanced draft of the consensus document was discussed during a meeting of the main consensus core group in Lausanne, Switzerland on 11–12 October 2021. Final edits were completed in the weeks after the meeting, prior to submission of the manuscript.

GENERAL APPROACH TO NON-INFECTIVE ACUTE RESPIRATORY ILLNESS IN ATHLETES

ARill in the athlete can present with a variety of symptoms or signs, with considerable overlap in the predisposing factors, pathophysiology and clinical presentation. ARill in athletes can be broadly classified by: (1) an anatomical classification predominantly affecting the upper or lower airways and (2) a pathological classification of infective and non-infective ARill. However, both these classifications have limitations. For example, non-infective conditions presenting predominantly in the upper airways in athletes, such as allergic rhinitis, are frequently associated with lower airway dysfunction (LAD) (eg, asthma).¹⁰ Similarly, non-infective conditions such as allergies can predispose individuals to ARinf. It is important that the SEM clinician is aware of these limitations in the classifications. If concurrent or overlapping ARill in athletes are not recognised, this shortcoming may compromise recognition, diagnosis and management. With these qualifications, and for the purposes of this consensus statement, the following terminology, and classifications for ARill in athletes were agreed on by the consensus group.

Anatomical classification of ARill in athletes

The consensus group recognises that in many conditions causing ARill, including allergy, asthma, infection and other inflammatory conditions,^{11 12} there is an anatomical and pathological continuum given the interaction between upper and lower airways. However, the usage of the terms 'upper' or 'lower' airways disease is still common in the literature when referring to both infective and non-infective causes of ARill. In this context, 'upper' refers to symptoms, signs and pathology

arising from the larynx and above, while 'lower' refers to symptoms, signs and pathology arising from below the level of the larynx. In this consensus statement, the group has referred to 'predominantly' upper or lower airway conditions, based on a predominant cluster of upper or lower airway symptoms, signs or pathological involvement.

Pathological classification of ARill in athletes

ARill in an athlete can present with general non-specific symptoms. In many studies among athletes, the pathology of ARill could not be attributed specifically to an infection or a non-infective cause. In these studies, the ARill in athletes was classified as 'undiagnosed'. ARinf in athletes is defined and discussed in part 1 of this consensus statement.⁹ Non-infective ARill in athletes is the focus of this consensus paper and was defined as an illness not caused by an infection. There are several conditions that cause non-infective ARill and a broad pathological subclassification of either 'inflammatory' or 'non-inflammatory' causes can be applied.

In summary, the consensus group classified non-infective ARill based on the anatomical and pathological classification of ARill (table 1) and these conditions will now be reviewed.

GENERAL APPROACH TO ACUTE NON-INFECTIVE RHINITIS/RHINOSINUSITIS IN ATHLETES

Rhinosinusitis is defined as inflammation of the nasal (rhinitis) and paranasal sinus (sinusitis) mucosa and characterised by: (1) two or more symptoms, one of which should be either nasal blockage/obstruction/congestion/discharge, facial pain/pressure, reduction/loss of smell and (2) endoscopic nasal examination of nasal polyps/mucopurulent discharge/mucosal obstruction and/or CT changes.^{11 13} Acute rhinosinusitis can be either infective or non-infective. Acute infective rhinosinusitis in the athlete is reviewed in part 1 of the IOC Consensus on 'ARill in the athlete'.⁹ Non-infective rhinosinusitis can be subclassified as either allergic or non-allergic. In non-allergic rhinitis (NAR), mucosal inflammation is caused by a trigger, irritant or exposure, which together with nasal hyperreactivity leads to mucosal swelling and nasal obstruction. Mucosal inflammation can lead to nasal obstruction. A detailed review of nasal obstruction in the athlete was specifically commissioned for this IOC Consensus statement and has been published.¹³ A clinical diagnostic approach to an athlete presenting with symptoms or signs of nasal obstruction is summarised in figure 1. In this diagnostic approach, nasal obstruction can originate from either structural (anatomical) causes or mucosal causes (usually swelling). Causes of mucosal swelling can be subclassified into infective (acute infective rhinitis/rhinosinusitis) or non-infective (non-infective acute rhinitis/rhinosinusitis).¹³ The structural causes of nasal obstruction and the non-infective causes of acute rhinitis/rhinosinusitis in athletes will be reviewed first.

NASAL OBSTRUCTION (STRUCTURAL/ANATOMICAL)

Introduction and definition

Athletes with ARill can present with symptoms and signs of nasal obstruction. Nasal obstruction can be defined as either permanent anatomical 'narrowing of the nasal cavity' or 'the subjective feeling' of reduced nasal airflow. Structural causes of nasal obstruction are subclassified as static or dynamic. In general, static obstruction is a permanent anatomical cause of obstruction whereas dynamic obstruction is intermittent.

Table 1 Classification of non-infective acute respiratory illness (ARill) in athletes

Anatomical classification	Pathological classification	Conditions in athletes
Predominantly upper airways	▶ Inflammatory non-infective (ARill)	▶ Non-infective rhinitis/rhinosinusitis * – Allergic rhinitis/rhinosinusitis – Non-allergic rhinitis/rhinosinusitis (chemically/mechanically mediated, for example, pollution, gases, air temperature) ▶ Non-infective pharyngitis/laryngitis (chemically/mechanically mediated for example, gastro-oesophageal reflux disease (GORD), pollution, gases, air temperature)
	▶ Non-inflammatory non infective (ARill): upper airway obstruction (anatomical/mechanical)	▶ Structural (anatomical) nasal obstruction† – Static – Dynamic (non-inflammatory) ▶ Oropharyngeal obstruction ▶ Laryngeal obstruction – Exercise induced laryngeal obstruction (EILO) – Static (anatomical) laryngeal obstruction
Predominantly lower airways	▶ Inflammatory non-infective (ARill)	▶ Lower airway dysfunction (allergy mediated, chemically/mechanically mediated, for example, pollution, gases, air temperature, etc) – Asthma – Exercise-induced bronchoconstriction (EIB) – Airway hyper-responsiveness (AHR)
	▶ Non-inflammatory non-infective (ARill): lower airway obstruction (anatomical/mechanical)	▶ Expiratory/Excessive Central Airway Collapse (ECAC) – Excessive Dynamic Airway Collapse (EDAC) – Tracheobronchomalacia (TBM)

*Can cause nasal obstruction due to mucosal swelling (inflammatory).
†Can cause nasal obstruction due to anatomic/structural causes and can be static (fixed) or dynamic (non-inflammatory, for example, nasal valve collapse).

Epidemiology

The incidence of nasal trauma leading to static structural nasal obstruction in athletes has not been thoroughly investigated. Nasal injuries potentially resulting in static obstruction are more common in competitive sports, adolescent and young adult athletes and men. The majority of nasal injuries affect the nasal bone and septum.^{14–16} The prevalence and incidence of

structural static and dynamic nasal obstruction in athletes are not well documented and this requires investigation.¹⁶

Risk factors

A history of previous trauma to the nose is the most frequent risk factor associated with a structural static (permanent) anatomical

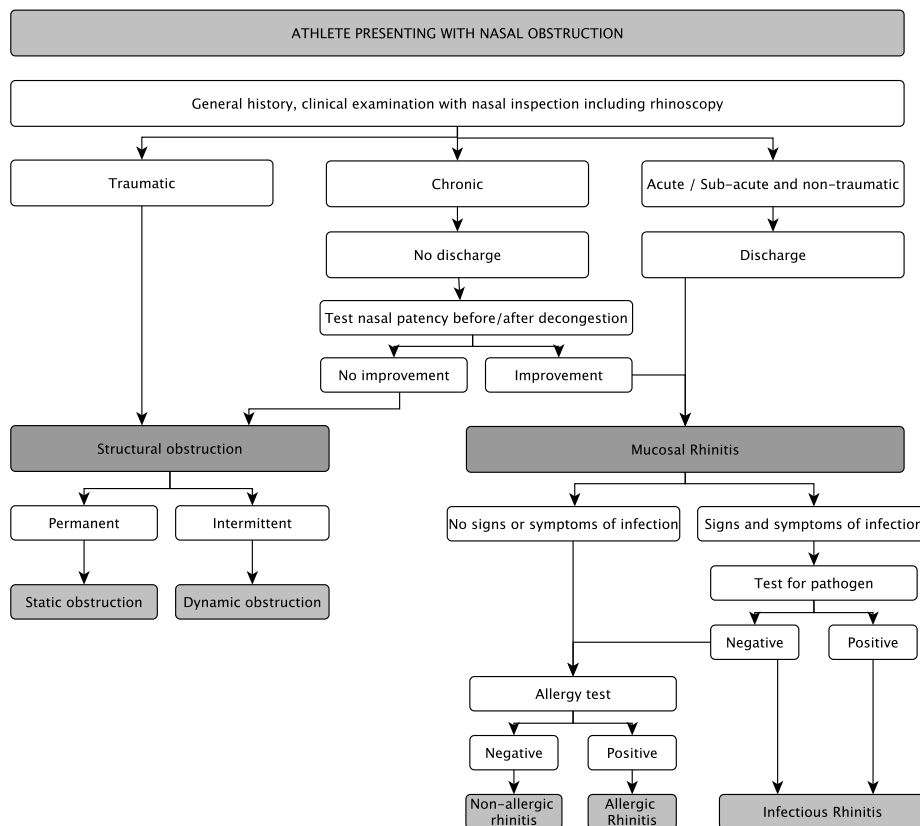


Figure 1 A clinical diagnostic approach to the athlete presenting with symptoms and signs of nasal obstruction.

cause of nasal obstruction. Risk factors associated with dynamic nasal obstruction in athletes are not well documented but include cranial nerve injury (after previous surgery or trauma), which can lead to nasal valve insufficiency during forceful nasal breathing.¹⁷

Pathology and pathophysiology

The pathophysiology of static and dynamic nasal obstruction in athletes varies. Static obstruction is a permanent anatomical abnormality narrowing the nasal airway not influenced by airflow and pressure changes. Dynamic obstruction is influenced by airflow and pressure changes due to a weakness in the soft tissue of the nose. Collapse occurs when the breathing rate or intensity increases, given the Venturi effect where increased flow results in higher negative pressures in the nasal cavity.¹⁸

Clinical presentation and diagnosis

A comprehensive clinical approach to the diagnosis of nasal obstruction in athletes is described in the commissioned published review for this IOC Consensus statement.¹³ A key element in the diagnosis is a good history and the most common acute symptoms are blocked nose, nasal congestion or nasal discharge. More chronic symptoms are obligate mouth breathing, snoring, poor sleeping habits and a dry throat in the morning. There are a variety of validated questionnaires adopted by otorhinolaryngologists to screen for nasal conditions, but no athlete-specific screening tool exists. In athletes who present with nasal symptoms, the following stepwise approach to the diagnosis of static and dynamic nasal obstruction is proposed¹³: (1) screen for possible nasal obstruction (Nasal Obstruction Symptom Evaluation (NOSE) survey score), (2) obtain a detailed history of nasal symptoms, followed by a general medical history, (3) conduct a systematic examination of the nose to determine the causes of obstruction and (4) decide on the use of special investigations (such as cone beam sinus CT scans) to determine the nature, severity and cause of nasal obstruction (online supplemental video link 1).

Management

In athletes with structural dynamic causes nasal obstruction, temporary relief with various internal or external nasal dilating devices during training, competition, recovery or during sleep can be considered.^{19–21} Early referral to an experienced otorhinolaryngologist for further surgical management is recommended.^{22–23} In general, surgery is the only permanent solution for structural static causes of nasal obstruction.

Prevention

Reducing the risk of nasal trauma during sport is the most important primary prevention strategy. If acute nasal trauma does occur, secondary prevention strategies to reduce the risk of complications are important. Nasal trauma should first be treated according to advanced trauma life support principles.²⁴ Immediate reduction of a displaced nasal fracture is recommended and surgical reduction within 7 days. A septal haematoma requires urgent intervention given the risk of infection, or destruction of the septum with saddle nose deformity, resulting in complete nasal obstruction.²⁵

Medical considerations and risks

In athletes, permanent structural nasal obstruction can lead to several health problems. These include poor quality of life, poor sleep and inadequate recovery from training or competition.

Mild symptoms of structural obstruction can worsen when athletes are exposed to trigger from various sports-specific environments that cause additional mucosal swelling (rhinitis) such as cold, pollution, irritants, chlorinated water and allergens.²⁶

Effect on exercise and sports performance

There is little evidence that alterations in nasal airflow negatively impact exercise or sports performance, or that nasal airflow is reduced by exercise.^{27–28} Two main reasons for this are: (1) as exercise intensity increases, ventilation increases concomitantly and breathing switches from nasal to oral, usually at a minute ventilation of approximately 35–45 L/min, resulting in a reduced resistance to the airflow²⁹ and (2) the sympathetic nervous system response during exercise results in vasoconstriction of the vessels in the nasal valve region, which increases airflow through the nose and nasal passages.³⁰

RTS considerations

In general, athletes with mild to moderate nasal obstruction due to structural abnormality can continue training even though they may experience mild symptoms during exercise. However, sleep disturbance and inadequate recovery may cause reductions in sport or exercise performance. RTS after acute nasal trauma is dependent on the extent of the injury, which should be discussed with an otolaryngologist. If there is a delay of more than 7 days after the acute injury, the recommendation is to delay surgical repair for at least 3 months. In general, contact sports can resume after ~6 weeks after surgery and the timing of surgery to coincide with the off-season period is advisable.

ALLERGIC RHINITIS/RHINOSINUSITIS

Definition

Allergic rhinitis is a symptomatic IgE-mediated inflammatory mucosal nasal condition, resulting from allergen introduction in a sensitised individual.^{31–32} Where allergens are also the cause of inflammation in the mucosa of the paranasal sinuses, the condition is referred to as allergic rhinosinusitis.¹¹ In this consensus document, allergic rhinitis/rhinosinusitis will be discussed as a single clinical syndrome (and abbreviated as AR), unless otherwise stated.

Epidemiology

A recent systematic review reported that 20%–60% of athletes can be classed as having allergic rhinitis³³ based on a diagnosis of nasal symptoms and a positive skin prick test to at least one allergen. This prevalence is similar to the general population.³³ Except for a few studies in swimmers, the prevalence of allergic rhinosinusitis in athletes is not reported.¹³

Risk factors

Athletes with a predisposition to allergy and atopy are at increased risk for AR. A few studies indicate a linear association between the volume of training per week in elite endurance sport athletes and allergic symptoms, Th1 lymphocytes and raised serum IgE levels.^{34–35} In some sports with environment-specific exposure, for example, swimmers' training in a chlorinated pool, there may be a higher risk of developing AR than other sports, but this remains to be determined.^{36–37}

Pathology and pathophysiology

Increased ventilation during exercise results in a greater volume and a deeper penetration of aeroallergens into the respiratory tract, potentially compromising the immunoregulatory function

of the epithelial barrier.^{13,38} Atopy, which can be a strong predictor of allergic symptoms in the general population, is observed in many endurance athletes but does not appear to be directly linked to allergic symptoms.^{34,35} Exercise-induced changes in immune function observed during and after intense exercise may predispose, at least in endurance athletes, to atopy and allergic symptoms.^{34,39} For example, atopic athletes, compared with non-atopic athletes, have a lower Th1/Th2 lymphocyte-type balance, which can increase susceptibility to upper ARinf.^{34,39} Environmental conditions (ie, chlorinated water, polluted air and cold air) can also increase allergenic sensitisation, via reaction with airway surface lining fluid components, airway oxidative stress and associated inflammatory cascade.^{40–42}

Clinical presentation and diagnosis

The classical symptoms of AR are nasal obstruction due to mucosal swelling, itching, sneezing, rhinorrhoea and postnasal drip.³² Athletes with allergies may also complain of associated ocular itching, erythema and/or tearing, oral cavity or pharyngeal pruritus, sore throat, aural itching or congestion, wheezing or cough and/or asthma.³² A clinical diagnosis of AR is made when athletes present with a history consistent with an allergic cause and at least one of the classical symptoms.^{32,43} The diagnosis can be confirmed by determining serum IgE as a general indicator of allergies, and by performing skin prick testing or in vitro testing methods^{32,43} for specific allergens. In cases of suspected rhinosinusitis, a definitive diagnostic is made by a CT scan and/or nasal endoscopy.¹³ In resource scarce settings, where skin prick tests or laboratory testing are not available, validated questionnaires such as the allergy questionnaire for athletes can be used to predict allergic disease.^{44,45} However, due to the potentially multiple causes of rhinitis in athletes, it is important to: (1) consider other causes such as those of infectious origin and (2) provide evidence of allergies. If a diagnosis of AR is made, pulmonary function testing (spirometry) should also be considered to diagnose concomitant LAD. Rhinitis is a risk factor for, and associated with, LAD, specifically asthma.^{39,43}

Management

Management decisions are based on making a correct diagnosis and identifying the causative allergen/s. In the first instance, the causative allergen/s should be avoided if possible, and nasal rinsing advised, even if evidence is limited.⁴⁶ A 10 point visual analogue scale (VAS) from 0 to 10 can be used to assess and track symptomatic relief.^{13,47} If allergen avoidance and nasal rinsing is not fully effective to bring sufficient symptom relief, pharmacotherapy should be considered. When AR is acute and/or symptoms are mild (VAS <5), either antihistamines (oral or topical) or intranasal corticosteroids (with or without antihistamines) can be used.⁴⁸ When symptoms are more severe (VAS ≥5) or experienced for more than a week, intranasal corticosteroids should be considered with or without antihistamines.^{13,47} In addition, if AR impairs sports performance, negatively affects quality of life in general, or where regular treatment is insufficient to control symptoms, other treatment options may be considered.⁴⁷ These include anticholinergics, leukotriene receptor antagonists, mast cell stabilisers or mucolytics. For bouts of acute exacerbation of rhinosinusitis, decongestants may be considered. The SEM clinician should refer to World Anti-Doping Agency (WADA) current standards when prescribing medications such as oral pseudoephedrine.⁴⁹ Finally, patient preferences, treatment availability and costs will determine the choice of treatment among first-line medication.⁴⁸ Athletes should be followed up and if the

VAS remains ≥5, despite correct use of intranasal corticosteroids, good compliance and adherence, the SEM clinician should consider referral of the athlete to an otorhinolaryngologist or an allergy specialist.¹³

Prevention

The only known prevention for sensitisation of allergens is avoidance, at home and/or during sport. Assessing allergen levels in ambient air and pollutants' concentration through official websites may be useful when advising athletes where or when to practice their sport. Alpine regions above 1600 m generally show markedly lower exposure to pollens, moulds and mites, which may reduce risk of AR in athletes and improve lung function in asthmatic subjects.⁵⁰

Medical considerations and risks

The main risk during exercise for untreated athletes suffering from allergic rhinitis is LAD, which includes EIB and asthma.^{51,52} Of interest, there are recent data showing an association between allergies in general and gradual onset musculoskeletal injuries in long-distance runners and cyclists.^{53,54} However, a causal relationship between allergies and gradual onset injuries remains to be established, and the potential mechanisms for this relationship are not clear. For example, it is not known if this may be because of allergies or medications used to treat allergies such as antihistamines or corticosteroids.^{55,56}

Effect on exercise and sports performance

Allergic rhinitis causing nasal obstruction and postnasal drip can disturb sleep and impair quality of life, thereby reducing exercise or sports performance.^{26,57–62} A decrease in endurance sports performance has been reported in healthy endurance athletes treated with histamine receptor blockers (selective H1 and H2-receptor blocker).^{56,63} However, to the best of our knowledge, there is no comparative study on the effects on performance with untreated symptomatic allergies versus those treated with oral antihistamine (H2-receptor blocker) treatment. While intranasal corticosteroids can be effective in improving quality of life and performance in elite athletes,⁵⁸ a recent study showed that 80% of athletes (endurance sports and football) with rhinitis reported impaired performance, including those under treatment.⁵⁹ In this study, 13% could not exercise at all due to allergic symptoms and among athletes using treatments, ~80% still had impaired training capabilities.⁵⁹ Therefore, the relationship between AR, allergies in general and exercise /sport performance in athletes requires further investigation.

RTS considerations

In most cases of AR in athletes, training can continue, but the SEM clinician may suggest, in individual athletes, decreases in training volume and intensity based on AR symptom severity. An important clinical consideration in RTS is that in case of a new diagnosis of AR, the lower airways should also be investigated to exclude LAD before an athlete continues with normal training.

NON-ALLERGIC RHINITIS (NAR)/RHINOSINUSITIS

Introduction and definition

NAR/rhinosinusitis is defined as acute or chronic rhinitis/rhinosinusitis in the absence of clinical symptoms and signs of infection, without evidence of allergies (normal systemic allergen-specific IgE and negative skin prick tests for allergens).⁶⁴ This definition includes a variety of clinical syndromes (phenotypes) that vary according to severity, aetiology and type of inflammation.⁶⁴ For

athletes, exercise itself can be a trigger as well as exposure to environmental irritants in certain sports (eg, chlorine in swimmers, cold air in winter athletes and changes in environmental conditions such as humidity, dry air and ambient air pollution that athletes may be exposed to).^{26 65 66}

Epidemiology

The incidence/prevalence of NAR in athletes is poorly documented and good evidence that unfavourable environmental conditions cause ARill in athletes is limited. The exceptions are cold air-induced rhinitis in winter athletes⁶⁷ and chemically-induced rhinitis in swimmers, linked at least partly to the chlorine disinfection.^{37 68}

Risk factors

The type of sport and exposure to certain environmental conditions are risk factors for NAR. Predominantly upper airway respiratory symptoms, especially rhinorrhoea, are common in water sport and winter sport athletes.^{37 69} There is evidence that time spent in a chlorinated swimming pool and the level of chloramines in the air are two risk factors associated with swimming-induced rhinitis.^{37 70 71} Some early studies reported a dose-dependent effect of pollution during exercising on the frequency and severity of upper respiratory symptoms in healthy or asthmatic athletes, especially pharyngeal irritation and nasal congestion.⁷²

Pathology and pathophysiology

A detailed review of each of the clinical syndromes of NAR is beyond the scope of the consensus document, but the differing pathophysiological mechanisms that influence management principles are summarised in figure 2. There are two important clinical considerations for athletes: first, athletes may present with NAR syndromes not related to sport. Second, there may be several pathophysiological mechanisms responsible for a clinical syndrome in an athlete including: various types of inflammation (eg, non-allergic rhinitis with eosinophilia syndrome, with eosinophils and mast cells, neutrophilic rhinitis, neurogenic inflammation) as well as neuronal imbalance or atrophic rhinitis (figure 2).^{26 32 36 73} The physiological response to air pollutants and chlorine likely includes stimulation of cell and neurogenic airway inflammation as well as epithelial cell damage through the oxidative stress provoked by the reaction of pollutants with the airway surface lining content.^{65 74 75}

Clinical presentation and diagnosis

It is important to make a specific diagnosis of the clinical syndrome of NAR in the athlete because this will influence decision-making on management. The diagnosis of NAR is made by clinical examination and appropriate tests, and based on: (1) typical recurrent nasal respiratory symptoms when exposed to specific triggers and (2) the exclusion of other or coexisting causes of allergic and infective rhinitis or respiratory disease.¹³

It is important to note that, as in the general population, mixed rhinitis may occur in some athletes, that is, rhinitis symptoms that have both allergic and non-allergic components.³² A diagnosis of a mixed rhinitis should be considered in cases where an athlete has an ineffective or partial response to medication.

Management

There is limited research on the management of athletes with NAR caused by environmental factors. Therefore, management of NAR in athletes should align with the position paper of the European Academy of Allergy and Clinical Immunology.⁶⁴ The principles of management are both non-pharmacological and pharmacological. The mainstay of non-pharmacological treatment is avoidance of environmental and other conditions that cause NAR. Nose rinsing with saline before or after swimming may help some swimmers exposed to chlorine-related products in pools. If environmental avoidance fails to suppress symptoms, pharmacotherapy may be considered. The clinical phenotype of NAR could influence decision-making on pharmacological management using topical intranasal corticosteroid or antihistamines (figure 2).⁷⁶ In athletes with underlying inflammation, topical intranasal corticosteroid is the treatment of choice.^{76 77} In cases where there is no suspected inflammatory component, ipratropium is the preferred treatment, for example, watery rhinorrhoea⁷⁷ and cold-induced rhinitis.⁶⁹ Intranasal capsaicin can be useful if ipratropium is insufficient.⁷⁷ In cases of suspected mixed rhinitis (allergic and non-allergic), a combination of anti-inflammatory and ipratropium is an option.

Prevention

The key to prevention of NAR is avoiding environmental stimuli by reducing exposure.⁷⁸ During swimming, a nose clip may be effective in relieving nasal symptoms related to the chlorinated environment or decrease their severity.³⁶ Facemasks may be advised to humidify and heat the inhaled air (cold air)⁷⁹ or partly filter at least particles, but not gases (air pollution).⁸⁰

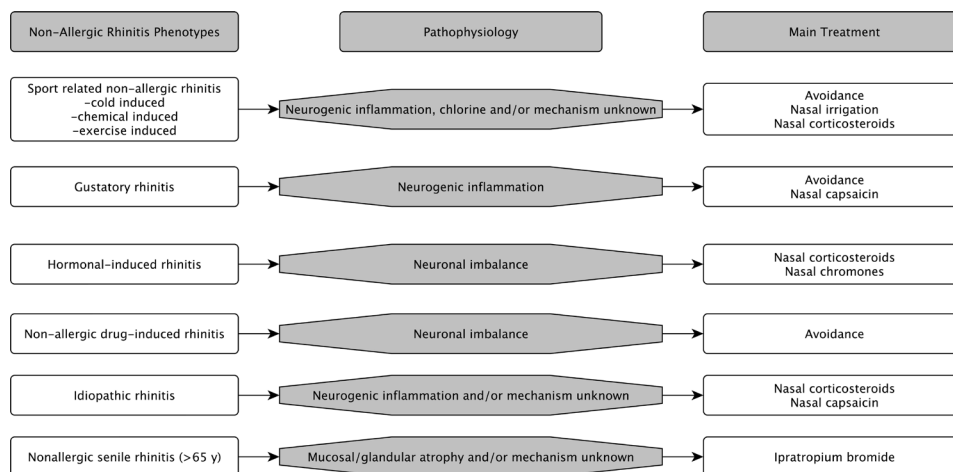


Figure 2 Phenotypes, pathophysiology and treatment of non-allergic rhinitis (adapted from Hellings *et al*⁶⁴).

Medical considerations and risks

There are no studies on acute or long-term risks associated with environmentally induced NAR in athletes. It is not known if athletes with acute NAR are at higher risk of developing acute medical complications during exercise. Athletes with chronic NAR may be at higher risk of allergic disease or asthma, but this requires further investigation. Any long-term risk is likely related to the environmental trigger, the pathophysiology and the frequency of exposure.

Effect on exercise/sports performance

There are no data on the effects of NAR related to pollution, cold air or chlorine on exercise/sports performance and this requires further investigation. In general, detrimental effects on exercise/sports performance are more likely if there is exposure to pollution or if LAD coexists with NAR.

RTS considerations

In most instances, there are no contraindications to training with environmentally induced NAR. However, as with AR, the lower airways should also be investigated to exclude LAD before an athlete with NAR continues with normal training.

EXERCISE-INDUCED LARYNGEAL OBSTRUCTION

Introduction and definition

Exercise-induced laryngeal obstruction (EILO) is defined as a paradoxical inspiratory narrowing of the laryngeal structures during exercise in an otherwise normal larynx at rest. EILO can mimic LAD (asthma and/or EIB), may impair exercise performance⁸¹ and is, therefore, an important cause of non-infective ARill in athletes. A detailed review of EILO in the athlete was specifically commissioned for this IOC Consensus statement and has been published.⁸²

Epidemiology

There are only a few studies reporting the prevalence of EILO in athletes. In most studies, the diagnosis was not confirmed using the gold-standard diagnostic test of continuous laryngoscopy during exercise (CLE) testing.⁸³ In the general paediatric population, the prevalence of EILO is approximately 5%–10%.^{84 85} In the most extensive study in high-level (Olympic) athletes, the point prevalence of EILO, based only on clinical assessments of stridor without CLE, was 5%.⁸⁶ This contrasts with data from one study where EILO was confirmed by CLE testing in 24 out of 89 (27%) elite cross-country skiers, with only one athlete reporting stridor.⁸⁷

Risk factors

The precise causes and risk factors associated with EILO are still unknown. Several factors have been suggested as both causes or contributory factors, including: asthma, environmental conditions (exposure to humid or cold air), anatomic factors related to the upper airway in women, ‘high achiever’ personality and a genetic factor.⁸⁸ EILO seems to be more prevalent in athletes,^{89 90} and this has been attributed to interactions between the high airflows required in some sports and anatomical and/or psychological characteristics.^{91–93} There is evidence that LAD (asthma, EIB) and EILO can coexist in some individuals^{90 94} and that symptoms of EILO may worsen when exercising in cold and humid air⁸³ or other adverse environmental conditions.⁸⁶ Although gastro-oesophageal reflux disease might increase the risk of EILO, treating EILO patients with proton pump inhibitors has not proven to be effective in reducing symptoms.⁹⁵

An anatomical contributor to EILO may explain the observed female preponderance^{84 88 90} in pubertal years and beyond, as women typically have smaller upper airways than men.⁸⁵ A genetic predisposition to EILO has been suggested,^{88 96} but to date, there is no definite proof of a hereditary component.

Pathology and pathophysiology

The larynx is a highly complex ‘valve’ that protects the lower airway from aspiration, permits vocalisation and abducts fully (opens) during exercise, to optimise airflow. Typically, a patient with EILO has a normal laryngeal structure and no symptoms at rest. In individuals with EILO, the laryngeal structures adduct (close) during inspiration and reach maximum obstruction at the peak of intense exercise. Symptoms typically resolve rapidly (<5 min) on exercise cessation. When viewed from a physical airflow perspective, the Venturi effect arising from a narrowed structure results in higher negative pressure and induces obstruction in the larynx during exercise.

The anatomical area of obstruction in EILO can be either supraglottic or glottic (vocal cords) or a combination.⁹⁷ Supraglottic obstruction followed by glottic obstruction is common, while pure glottic obstruction is rare (ie, seen in <5% of cases). A scoring system is widely used to grade the severity of EILO and ranges from no obstruction (complete patency=0), mild, moderate or severe obstruction (almost complete closure=3). The grading is based on direct observation of the obstruction at moderate to maximum intensity of exercise.^{98–100}

Clinical presentation and diagnosis

The diagnosis of EILO is based on a high index of suspicion when an athlete presents with symptoms during exercise that follows a specific time course. The key elements of a typical presentation are wheeze, whistle/stridor when breathing in that: (1) are usually most evident during the inspiratory phase of the respiratory cycle, (2) are most prominent at peak exercise, when ventilation is at its greatest and (3) typically resolve rapidly (within 1–5 min) after cessation of exercise unless there is coexistent (ongoing) panic or hyperventilation.¹⁰¹ This pattern is in contrast to EIB, where symptoms are related to expiratory limitation and are worst 3–15 min postexercise. These differentiating features can distinguish suspected EILO from EIB in athletes (figure 3). It is important that the SEM clinician makes the correct diagnosis to avoid treatment failure.^{90 94}

Athletes may find it difficult to attribute their exertional respiratory symptoms to a specific phase of the respiratory cycle.

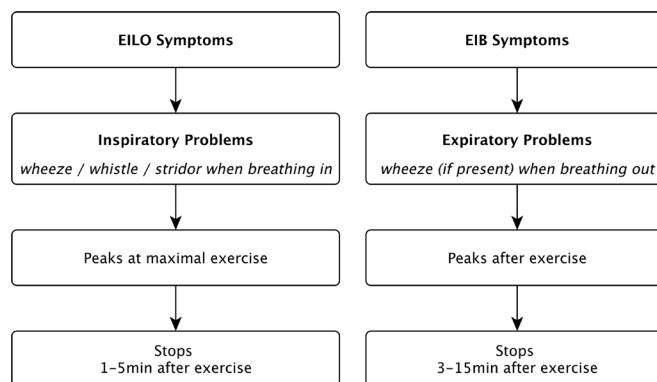


Figure 3 Key differences in the clinical presentation of exercise induced laryngeal obstruction (EILO) and exercise induced bronchoconstriction (EIB).

They may present with other exercise-related symptoms such as breathing that simply feels ‘harder’, throat tightness, choking sensations, upper chest tightness and chest pain, noisy breathing, stridor, hoarseness, voice changes, cough and panic reactions.⁹⁷ Typical symptoms of EILO, however, may also occur in athletes with diseases such as asthma, structural airway abnormalities, cardiac diseases, vascular anomalies, tumours/cancer, primary hyperventilation/breathing pattern disorders, poor breathing techniques, low physical fitness and psychological causes. Given the difficulties inherent in discerning the features of exercise-related phenomena in a clinical consultation at rest, it may be helpful to: (1) seek a collateral witness report describing the symptoms, (2) obtain a video recording of the athlete during a symptomatic period or (3) directly assess an athlete when they are symptomatic (eg, during an exercise test).

If the diagnosis of EILO is suspected, referral to a centre where CLE can be performed is recommended. The CLE test involves placement of a flexible laryngoscope to allow real-time visualisation of the larynx throughout a complete exercise session. Typically, moderate obstruction defined by a CLE-score ≥ 2 at the glottic or supraglottic level is considered pathological and is diagnostic for EILO^{98–100} (online supplemental video link 2).

Management

Treatment for EILO is non-surgical in the first instance and includes behavioural therapies (breathing advice, speech therapy, laryngeal control therapy),^{102–103} biofeedback¹⁰⁴ and inspiratory muscle training.^{105–106} The aim of these strategies is to provide athletes with tools to better control their larynx during exercise and enable them to continue exercising without experiencing EILO. The CLE test can be used as a biofeedback tool to guide athletes in using breathing control techniques during exercise. There is little scientific evidence on the efficacy of these treatment strategies, and most are based on anecdotal experiential evidence or single-centre intervention studies. Surgery is an option, and the CLE test is also used to select those in need of surgery.^{107–109} Surgery should be limited to athletes with moderate to severe obstruction caused by the supraglottic structures, with little or no effect from conservative treatments.

Prevention

To date, there are no evidence-based preventative strategies for EILO. However, anecdotal experiential evidence indicates that if athletes employ effective breathing techniques and have knowledge of laryngeal function, they may have a lower risk of some types of EILO. This area requires further study.

Medical considerations and risks

An important medical consideration is that EILO can be incorrectly diagnosed as LAD (asthma/EIB). This may lead to over-prescription of asthma medication with subsequent side effects and unnecessary doping implications.^{49–110} A more widespread understanding of the key clinical features that distinguish EILO from asthma/EIB will improve assessment of athletes with exercise-related breathing problems. There is a clinical impression from the author group (not based on scientific evidence) that athletes and young individuals who have a prolonged (years) history of EILO appear more difficult to treat than those who seek advice early (months).

Effect on exercise and sports performance

EILO may lead to airflow limitation and excessive breathlessness, which are all contributory factors to impairment of exercise

and sports performance.⁸¹ Performance decrements can lead to loss of motivation and willingness to participate in competitive sports. Several studies from the general population indicate that EILO left untreated or undertreated can negatively affect a healthy lifestyle through the avoidance of exercise.^{99–100}

RTS considerations

The general recommendation for RTS in an athlete with ongoing severe EILO causing distress±complications (eg, syncope/dizziness) is to only exercise below or up to the threshold of intensity where symptoms of EILO develop. Once an athlete with EILO is treated and has a working strategy to control the larynx during exercise, RTS is recommended. In highly symptomatic athletes, a more cautious RTS protocol is recommended. Unsupervised or aquatic-based sports, with a risk of injury, are to be avoided until symptoms are better controlled.

LOWER AIRWAY DYSFUNCTION

Introduction, definition and classification

A variety of terms are used to describe clinical entities causing asthma-related issues in athletic individuals. Terms used include exercise-induced asthma, EIB and airway hyper-responsiveness (AHR). These terms are often used interchangeably in real-life practice to describe a similar clinical entity. In this consensus document, the term ‘lower airway dysfunction’ (LAD) is used as an umbrella term to encompass the clinical entities causing asthma-related issues in athletes. Where indicated, definitive reference is made to a specific condition, for example, studies specifically evaluating asthma, EIB or AHR.

Epidemiology of LAD in athletes

The prevalence of LAD is greater in athletes than in the general population.¹¹¹ A recent systematic review and meta-analysis, specifically commissioned for this IOC consensus statement, re-evaluated the prevalence of LAD in athletes. In this review, studies employing different diagnostic approaches revealed an overall prevalence of LAD in ~20% of athletes.¹¹² The highest prevalence rates were reported in elite-level endurance athletes and in those participating in aquatic and winter-based sports. A higher prevalence of LAD was also evident in studies using objective testing methodology (figure 4).

Key deficits in the available epidemiological data in this area include a lack of robust data from developing nations, detail on LAD in para-athletes, and insight regarding the incidence and impact of acute asthma-related events during training or competition in the athletic population.

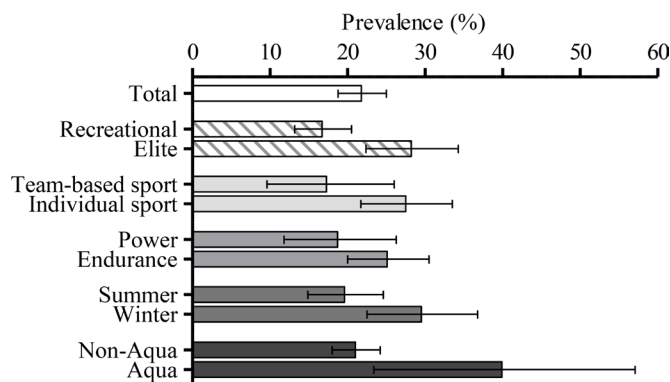


Figure 4 Prevalence of lower airway dysfunction according to athletic standard and sporting discipline (adjusted from Price *et al*).

Risk factors

Several risk factors are associated with the development of LAD, reflecting both host and environmental influences. For example, as in the general population, an atopic disposition in athletes is associated with a 25-fold to 75-fold increased likelihood of EIB.⁵¹ In the context of sport, the training environment in which exercise is undertaken is further important risk factor.^{113–114} For example, in the aquatic environment, exposure to high levels of trichloramines (ie, a chlorine by-product) is associated with AHR.^{115–117} When endurance exercise is performed in cold air, as witnessed in elite cross-country skiers, there is a high prevalence of respiratory symptoms and AHR.^{118–121} Other environmental risk factors include exposure to pollution, that is, emissions from fossil-fuelled ice resurfacing machines in sports arenas and air pollutants such as ozone and particulate matter.¹²²

Pathology and pathophysiology

Over the past 40 years, there has been considerable progress in understanding the pathophysiological pathways, leading to the development of EIB in athletes.^{123–124} In this context, two questions are particularly pertinent (1) how EIB develops acutely and (2) why there is a heightened prevalence of LAD in endurance athletes. The development of airway dysfunction may be viewed as being akin to an ‘occupational lung disease’ in athletes.¹²⁵

The key pathophysiological mechanism driving acute EIB is the release of local mediators in response to evaporative water loss from the airway surface. Specifically, hyperpnoea during exercise places stress on the airway surface to replenish evaporative water loss. In susceptible individuals, failure to replenish evaporative water loss at the airway surface leads to cellular changes and activation of sensory pathways that release local mediators, which promote bronchoconstriction.^{126–127} This process is amplified by the following factors: dry and cold air exposure, air pollution,¹²²

allergic asthma and the extent of epithelial damage and inflammatory status of the airway at baseline.¹²⁷ A recent acute viral infection may also act to sensitise airway responses, with some reports indicating development of a postviral AHR syndrome. In general, this condition resolves spontaneously within 12 weeks.¹²⁸

The reason elite-level endurance athletes appear to have a higher prevalence of lower dysfunction remains to be fully established. Repeated and prolonged episodes of hyperpnoea, specifically in endurance athletes, particularly when they exercise in potentially noxious environments, may drive airway structural, cellular and inflammatory changes, in a pattern similar to that witnessed pathologically following injury.^{38–123} Epithelial damage may facilitate sensitisation to circulating allergic and inflammatory mediators, cause sensory irritation and a local hyper-inflammatory response, thereby precipitating heightened AHR.¹²³ Increased parasympathetic nervous system may also be relevant in the development of AHR in endurance athletes.^{129–131} Certainly, there are key differences in the phenotypic and inflammatory pattern of LAD in some groups of elite athletes, and this may be relevant for treatment and also for strategies to protect airway health.¹³²

Clinical presentation and diagnosis

There are two main clinical scenarios, where an SEM clinician will need to follow an approach to make a diagnosis of LAD. In the first instance, there may be a need to screen asymptomatic athletes as part of a periodic health assessment, and, in the second instance, a diagnosis is required for athletes that may present with symptoms suggestive of LAD. An approach to these two scenarios is presented in figure 5.

Symptoms suggestive of LAD, in athletes, typically include breathlessness, chest tightness, wheeze and cough during or

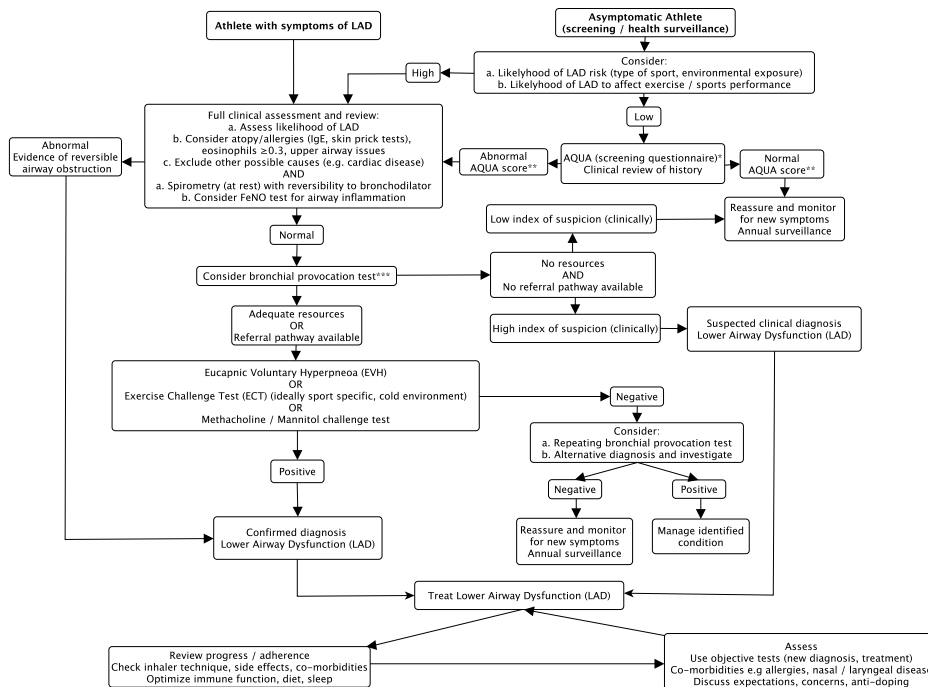


Figure 5 A clinical approach to the detection and evaluation of LAD in athletes. *: AQUA: Allergy Questionnaire for Athletes Bonini *et al.*⁴⁴ **: AQUA screening uses five as the cut-off score for normal. ***: Objective testing is recommended to ensure diagnosis is robust, to be guided by local availability of tests; sport-specific exercise testing and/or another indirect bronchoprovocation testing protocols are recommended. EIB, exercise-induced bronchoconstriction; LAD, lower airway dysfunction.

after exercise.^{132–133} Other less distinct symptoms are ‘difficulty obtaining a satisfying breath’ or ‘catching their breath’, particularly at the onset of vigorous exercise and exercising in cold environments.

In athletes, there is a poor relationship between clinical features suggestive of LAD and proof of these diagnoses when objective testing is undertaken.^{134–136} This is because conditions such as EILO or dysfunctional breathing may mimic or coexist with asthma^{10 84 90} or respiratory symptoms may arise because of sensory irritation (eg, as is seen in cough hypersensitivity).¹³⁷ A careful history of the clinical features can help discern EILO from EIB (figure 3).¹³⁸

The diagnosis of LAD in an athlete must be robust and supported by objective tests; a position supported by The Global Initiative for Asthma (GINA), prior IOC-Medical Commission statements¹³⁹ and the WADA.¹⁴⁰ Moreover, WADA recommend that a childhood diagnosis of asthma should be reconfirmed in adulthood.⁴⁹

The key first-line objective test in the assessment of an athlete with suspected LAD is spirometry at rest. If expiratory airflow limitation/obstruction is evident at rest (based on appropriate predicted normative values), then bronchodilator reversibility testing should be undertaken (eg, following administration of a short acting beta-2 agonist (SABA)). This process may provide immediate evidence of reversible airflow obstruction, supporting a diagnosis of LAD. In most cases; however, athletic individuals typically have normal resting spirometry values and, thus some form of additional testing will be required.

A broad range of additional tests have been used in this context, including: (1) sport-specific/field-based exercise challenge tests (ECTs) with spirometry performed before and after exercise and (2) other bronchoprovocation-based tests that can be performed and interpreted in line with international guidelines.¹⁴¹ The optimal test to detect LAD in athletes remains debated and will most often be dictated by factors such as local availability and cost. In general, it is recommended that test replicates the process that precipitates symptoms during exercise, that is, one that promotes relevant physiological changes at the airway surface. Accordingly, an approach that uses either exercise or an alternative ‘indirect’ form of bronchoprovocation testing (eg, eucapnic voluntary hyperpnoea (EVH) or mannitol provocation testing) is recommended in preference to direct bronchoprovocation tests (eg, using methacholine or histamine for bronchoprovocation).

A recent systematic review, specifically commissioned for this IOC consensus statement, revealed the following: (1) a symptom-based approach/clinical diagnosis by a physician was sensitive but very non-specific for diagnosis of LAD in athletes and (2) EVH, followed by a sport-specific ECT, had the highest specificity when compared with other forms of provocation tests to confirm the diagnosis of LAD in athletes (Reier-Nilsen *et al.*, 2022, unpublished). Therefore, in a resource-scarce setting where EVH testing is not an option, the alternative test is a sports-specific ECT, that is, conducted with appropriate consideration of the environmental conditions and type of activity to obtain precise results.^{119 142 143} The sensitivity/specificity of ECT is enhanced if testing is: (1) performed in cold air,^{51 144} (2) at a very high exercise intensity (>85% of age predicted maximum heart rate) for 6–8 min¹⁴² and (3) in an environment and time of year when the athlete is symptomatic,⁵¹ for example, during a grass pollen season. Finally, repeat testing can increase sensitivity, specifically in athletes where there are initial negative or inconclusive test findings, but there is an ongoing high suspicion

of LAD (ie, without features of a differential diagnosis such as EILO).¹⁴⁵

A further key component in the modern-day assessment of an athlete with suspected LAD and is the detection and an assessment of airway inflammation.¹⁴⁶ This can be undertaken with measurement of fractional exhaled nitric oxide (FeNO), and interpretation of results can be guided by international guidelines (see GINA). In addition, a comprehensive assessment of LAD in athletes includes a full blood count to detect eosinophilia and tests for atopy (eg, serum IgE and skin prick testing).

Management

Both pharmacological and non-pharmacological strategies play a role in a successful holistic approach to the treatment of LAD in athletes. The overall goal of treatment is to ensure good symptom control and minimise risk of acute exacerbations. In athletes, a key aim is to ensure that participation in sport is not hampered by respiratory symptoms. Different phenotypes of asthma in athletes have been described, and identification of biomarkers of inflammation (ie, termed type 2) is now considered relevant to guide management.¹⁴⁷ The evidence-based management of EIB was reviewed in 2013.¹⁴⁸

There are several non-pharmacological strategies to help manage LAD in athletes. The first strategy is to reduce exposure to cold and dry air, allergens, pollution or other airway irritants. Other strategies include wearing a heat and moisture exchange face mask,¹⁴⁹ alterations to dietary intake with vitamin D,¹⁵⁰ omega-3 polyunsaturated fatty acids,^{151–154} low-salt diet^{155 156} and use of prebiotics.¹⁵⁷ An attenuation of EIB has also been reported after short high-intensity intervals and variable intensity pre-exercise warm-ups.¹⁵⁸

The pharmacological approach to management of asthma is constantly evolving. In this consensus, we recommend SEM clinicians refer to www.ginasthma.org for latest updates and management advice. At the current time, there is no contemporary and robust evidence, to indicate that management of asthma in athletes should deviate from these international asthma guidelines.

The classical pharmacological approach of recommending a SABA alone, before sport for an individual with EIB, has been challenged by changes to international guidelines on the management of asthma. Specifically, as outlined in the GINA guidelines (www.ginasthma.org), it is now recommended that all patients with asthma, even with mild intermittent asthma, should use an inhaled corticosteroid daily to prevent exacerbations and control symptoms. This recommendation represents an important change to the general treatment of asthma, and SEM clinicians should consider this updated guidance when caring for asthmatic athletes.

Some clinicians may choose to consider the use anticholinergic bronchodilators or leukotriene antagonists (eg, montelukast), based on their expertise when managing a specific athletic population, and the environmental conditions in which sport is being undertaken. Approach to therapy should be modified by baseline lung function (ie, if an athlete has spirometry at rest that indicates airflow obstruction) and the presence of biomarkers of type 2 (eg, raised FeNO±blood eosinophilia) inflammation; that is, highlighting a potentially steroid responsive process.

Treatment should be regularly reviewed with evaluation of adherence and inhaler technique. When taken correctly and with use of a spacer or volumetric device, it is not necessary to use nebulised preparations of inhaler therapy; this type of treatment should be restricted to management of severe or life-threatening asthma, when administered in an emergency scenario. It is

also important that athletes with asthma are aware of how to manage an acute exacerbation, and for competitive athletes, there is a discussion regarding antidoping regulations.¹⁵⁹ In this respect, various web-based resources including www.wada-ama.org, www.globaldro.com, international sports federations and national antidoping agencies are important sources of information to provide clarity regarding anti-doping regulations on asthma medication.

Prevention

A key strategy to prevent LAD in athletes is to ensure that the air an athlete breathes is as clean as possible.¹⁶⁰ Environmental exposure factors such as cold air, pollution (carbon monoxide, nitrogen dioxide, ozone and fine particulate matter) and allergens may contribute to development of LAD. It is recommended that sporting organisations and facilities should endeavour to improve air quality in the sporting arena, and where possible limit athletes' exposure to polluted areas. Airway health should be considered when sports authorities develop extreme weather protocols (eg, if air temperature is very cold). It is also important to consider strategies to reduce allergen exposure in atopic individuals.¹⁶⁰ This may include advice on how to modify training plans (ie, time of day and duration of session), according to the local environmental conditions. In scenarios where it is not possible to modify exposure to a noxious environment (eg, polluted or in very cold air), it may be appropriate to consider use of a face mask or filter,⁸⁰ both at rest and during exercise, although it is acknowledged that this is impractical in many sporting scenarios. More work is needed to assess climatic conditions and the concentration of pollutants through various official monitoring sites or individual sensor systems, with the aim of helping inform clinicians involved with the care of athletes.

Medical considerations and risks

In the general population, asthma is associated with recognised morbidity (eg, exacerbations) and mortality.¹⁴⁶ However, data on medical complications in athletes with LAD are currently limited. Most athletes with LAD will only develop or report symptoms in the context of sporting participation, that is, they do not generally report other classical complications of asthma, such as frequent exacerbations or night waking with symptoms. In one study, the presence of asthma/EIB in athletes was associated with a twofold increased likelihood of more frequent ARinf in the preceding 18 months.¹⁰ However, other factors, respiratory issues, such as the presence of laryngeal dysfunction or breathing pattern irregularities, were also relevant predictors of risk of ARinf. With respect to asthma-related mortality, very few studies have evaluated asthma as a cause of sudden death in athletes or in the context of sport. A recent study found that sudden death due to asthma in athletes is uncommon when compared with cardiac causes, accounting for 0.8%–4.9% of all causes.¹⁶¹ A further potential medical consideration in athletes with asthma relates to potential complications arising from treatment with β_2 -adrenoceptor agonists (eg, tachyarrhythmia).^{162 163}

Effect on exercise and sports performance

LAD may impact breathing and gas exchange efficiency and limit performance secondary to the development of dyspnoea. Data from athletes competing at international level events suggest that asthmatic athletes are still capable of excelling in their chosen sport, and in some studies appear to outperform their competitors without asthma.¹⁶⁴ A systematic review of 10 studies evaluating the impact of LAD on athletic performance measures could

not draw any firm conclusions because of several methodological limitations within the included studies.¹⁶⁵ Further work is needed to study the impact of LAD in sport-specific settings and with environmental conditions likely to be provocative to the lower airways.¹⁶⁵ A diagnosis of LAD, when managed appropriately, should not be perceived as a negative consequence for athletic performance nor an athlete's aspirations. Finally, although often raised in discussion with athletes, the best available evidence to date indicates that inhaled asthma therapy, when taken at routinely prescribed doses, does not enhance endurance-based athletic performance.^{166 167}

RTS considerations

There are currently no dedicated studies informing athletic RTS following an acute exacerbation of asthma. In the general population, it is recommended that vigorous exercise is avoided completely in the period immediately following an acute exacerbation of asthma.¹¹⁰ It would appear logical to extend this recommendation to athletes, that is, an athlete requiring maximal treatment for an acute exacerbation of asthma (eg, with oral glucocorticoid therapy) should, therefore, not partake in vigorous exercise. This recommendation is made to mitigate the risk of an acute serious adverse event; for example, worsening respiratory tract infection, development of hypoxaemia \pm arrhythmias. Some sporting activities likely present a greater risk than others (eg, high-intensity exercise in a cold environment) and, thus, decision-making regarding RTS should be guided by the identification of features prompting a need for a cautious approach to RTS. These include clinical features such as recent hospitalisation, ongoing respiratory symptoms during low-intensity physical activity/activities of daily living and ongoing significant need for bronchodilator medication (ie, in excess of typical utilisation). The SEM clinician should also consider using both clinical and physiological assessment tools, to help assess and accurately appraise an athlete's recovery. It is recommended that specialist input and advice is sought for more complex scenarios.

OTHER CONDITIONS CAUSING NON-INFECTIVE ACUTE RESPIRATORY ILLNESS

Differential diagnoses of acute breathing problems in athletes

The SEM clinician should also consider the broader range of differential diagnoses when an athlete presents with acute breathing problems. These conditions include the following: cardiac-related diseases, acute pulmonary emboli, acute pneumothorax (acute dyspnoea with chest pain, often related to strenuous activity), altitude-related pulmonary oedema (acute dyspnoea and other features of altitude related illness)¹⁶⁸ and exercise-induced pulmonary oedema (dyspnoea and haemoptysis, in relation to endurance sports generally free water swimming, often with wetsuits in cold conditions).^{169 170} These conditions should be assessed and managed appropriately. More recently, focus has been placed on the potential for respiratory symptoms that arise from excessive dynamic airway collapse (EDAC) or dysfunctional breathing in athletic individuals.

EDAC and tracheobronchomalacia

EDAC and tracheobronchomalacia are conditions characterised by excessive inward movement of the trachea and/or main bronchi during the expiratory phase of the respiratory cycle.¹⁷¹ The terms are often used interchangeably, both defined as >50% collapse of the airway lumen.¹⁷² Clinically, these conditions present with unexplained breathlessness, a barking or

Table 2 Summary of unanswered questions and potential research priorities in non-infective acute respiratory illness in athletes

Unmet need/unanswered questions	Future research priorities
<p>Nasal conditions in athletes (eg, nasal obstruction, rhinitis/rhinosinusitis)</p> <ul style="list-style-type: none"> ▶ Epidemiological data on structural nasal obstruction ▶ Data on impact of nasal obstruction on training capability, resuscitation ▶ Epidemiological data from developing countries and para-athlete populations. ▶ Data on the impact of various severities of rhinitis/rhinosinusitis and anti-allergic treatment on training capability and return to play. ▶ Impact of pollution on the nose and possible treatments. 	<ul style="list-style-type: none"> ▶ Effect on health and performance following correction of structural nasal obstruction. ▶ RCT on nasal obstruction treatment. ▶ More inclusive epidemiological studies to help characterise global burden of disease. ▶ Characterisation of the physiopathology of nasal symptoms during exercise in various type of sports. ▶ The effects of immunotherapy on allergic rhinitis in athletes, comparatively to acute treatments. ▶ Various treatments or prevention measures efficacy to manage NAR according to the environment.
<p>Exercise-induced laryngeal obstruction (EILO)</p> <ul style="list-style-type: none"> ▶ Epidemiological data from developing countries and para-athlete populations. ▶ Data on the impact of EILO on training capability. ▶ Impact of cold, chlorine, pollution and thermal stress ▶ Evidence based treatment guidelines ▶ Pathophysiological studies on EILO—why is EILO more prevalent in athletes? 	<ul style="list-style-type: none"> ▶ RCT on EILO treatment. ▶ Impact from environmental factors on EILO. ▶ More available diagnostic options for EILO. ▶ More inclusive epidemiological studies on EILO to help characterise global burden of disease. ▶ Prevention and risk considerations in different types of sports.
<p>Lower airway dysfunction (LAD)</p> <ul style="list-style-type: none"> ▶ Epidemiological data from developing countries and para-athlete populations. ▶ Data on the impact of asthma exacerbations on training capability and return to play. ▶ Impact of pollution and thermal stress on airway health in athletes. ▶ Data on the effect of nebulised isotonic saline post strenuous exercise on pulmonary function, inflammation and on respiratory symptoms. 	<ul style="list-style-type: none"> ▶ More inclusive epidemiological studies to help characterise global burden of disease. ▶ Establish prospective longitudinal monitoring systems to evaluate and ensure athlete respiratory health is protected from early in an athlete's career. ▶ Prospective evaluation of lower airway function, inflammatory biomarkers during exacerbations/evaluating the interplay with acute respiratory infections (ARInf). ▶ Mechanistic studies to evaluate 'airway injury' and interaction with environmental conditions. ▶ Placebo-controlled studies on nebulised isotonic saline.

RCT, randomised controlled trial.

honking-type cough and difficulty clearing respiratory secretions with or without recurrent lower respiratory tract infections.

Dysfunctional breathing/breathing pattern disorders

Dysfunctional breathing can be defined as an altered biomechanical pattern of breathing, leading to intrusive symptoms that can impact athletic performance and quality of life.^{173 174} Several terms have been used to describe this issue, including exercise-associated hyperventilation or a breathing pattern disorder. This condition can occur in the presence or absence of other comorbidities including EIB, EILO and/or anxiety and can overlap or amplify symptoms making them refractory to treatment.

Dysfunctional breathing is characterised by the following features: frequent sighing, apical/upper chest movement during respiration, asynchrony between upper and lower chest, excessive mouth breathing and limited lateral movement of abdominal rib cage. A suspected diagnosis of dysfunctional breathing can be supported by affirmative responses to a questionnaire (eg, Nijmegen questionnaire) or breathing pattern assessment tools that are used in the general population.¹⁷⁵ Ideally, a breathing pattern assessment should be conducted in a sport-specific context by an individual with expertise in the area. Assessment and subsequent treatments are usually undertaken by respiratory therapists. The focus is optimised breathing patterns but also to identify and address relevant psychophysiological and musculo-skeletal factors, including core stability.

FUTURE DIRECTIONS

In this IOC consensus statement, we provide an evidence-based overview of common and lesser-known causes of non-infective ARill in athletes. However, in this process, several unanswered questions were identified by the group translating into future research priorities (table 2). The group encourages researchers and institutions to consider these as research priorities, so that knowledge can be expanded and contributed to the protection of respiratory health in athletes.

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