

**Diagnosis and management of nasal obstruction in the athlete: A narrative review by subgroup 6 of the IOC Consensus Group on “Acute Respiratory Illness in the Athlete”**

Cameron McIntosh,<sup>a</sup> Hege Havstad Clemm,<sup>b,c</sup> Nicola Sewry,<sup>d</sup> Harald Hrubos-Strøm,<sup>e,f</sup> Martin Schwellnus,<sup>d, f</sup>

<sup>a</sup> Dr CND McIntosh INC, Edge Day Hospital, Port Elizabeth, South Africa; <sup>b</sup> Department of Pediatric and Adolescent Medicine, Haukeland University Hospital, Bergen, Norway; <sup>c</sup> Department of Clinical Science, University of Bergen, Bergen, Norway; <sup>d</sup> Sport, Exercise Medicine and Lifestyle Institute (SEMLI), Faculty of Health Sciences, University of Pretoria, South Africa; <sup>e</sup> Department of Otorhinolaryngology, Surgical Division, Akershus University Hospital, Lørenskog, Norway; <sup>f</sup> Department of Behavioural Sciences, Institute for Basic Medical Sciences, University of Oslo; <sup>g</sup> International Olympic Committee (IOC) Research Centre of South Africa, University of Pretoria, South Africa

\*Corresponding Author: Prof. Martin P. Schwellnus

Sport, Exercise Medicine and Lifestyle Institute (SEMLI), Faculty of Health Sciences, University of Pretoria, South Africa, SEMLI building Sports Campus, Burnett Street, Hatfield, Pretoria 0020, South Africa.

Telephone: +27-12-484 1749 Fax number: +27-86-480 6511

Email: mschwell@iafrica.com

## **ABSTRACT**

Protection of the health of the athlete is required for high level sporting performance. Acute respiratory illness is the leading cause of illness and can compromise training and competition in athletes. To date the focus on respiratory health in athletes has largely been on acute upper respiratory infections and asthma / exercise induced bronchoconstriction (EIB), while nasal conditions have received less attention. The nose has several important physiological functions for the athlete. Nasal conditions causing obstruction to airflow can compromise respiratory health in the athlete, negatively affect quality of life and sleep, cause mouth breathing and ultimately leading to inadequate recovery and reduced exercise performance. Nasal obstruction can be broadly classified as structural (static or dynamic) or mucosal. Mucosal inflammation in the nose (rhinitis) is the most frequent cause of nasal obstruction and is reported to be higher in athletes (21-74%) than in the general population (20-25%). This narrative review provides the sport and exercise medicine physician with a clinical approach to the diagnosis and management of common nasal conditions that can cause nasal obstruction, ultimately leading to improved athlete health and better sports performance.

**Key words:** Athletes, sports, exercise, nasal obstruction, rhinitis, sinusitis

## **Introduction**

Acute respiratory illness is one of the leading causes of competition- and training loss in elite athletes <sup>1-4</sup>. Sport performance at high level, including the Olympic and Paralympic Games, requires optimal respiratory health. Respiratory health, including effortless breathing without obstruction to airflow is also important for quality of life, good quality sleep, recovery from training and mood <sup>5,6</sup>. Effortless breathing requires a coherent respiratory organ system, starting with the nose and nasal passage. The nose and nasal passages have important physiological functions that are necessary for good health, but are often forgotten and neglected when it comes to discussing respiratory health, both in athletes and in general populations. In Sport and Exercise Medicine (SEM) the upper airways, particularly the nasal passages, should be considered in the prevention, diagnosis and treatment of respiratory illness in athletes. Structural (anatomical) causes of nasal obstruction in athletes can lead to a

reduction in airflow in the nose and nasal passages <sup>7</sup>. Mucosal swelling in the nose and nasal passages, resulting from rhinitis and rhinosinusitis are well known causes of nasal obstruction in athletes with a high reported prevalence, ranging from 27-74% <sup>8-10</sup>. Despite the high prevalence of nasal inflammation in athletes, previous reports lack precise differentiation of possible causes <sup>10,11</sup>.

Our main aim is to provide the SEM physician involved in athlete care with a clinical diagnostic and management approach to nasal obstruction in athletes. The specific focus of this narrative review is to briefly review nasal anatomy and nasal function at rest and during exercise, followed by a general and more detailed diagnostic and treatment approach to nasal obstruction in athletes. Ultimately, we believe this will contribute to the protection of the athletes' general and respiratory health, and aid in recovery and improved sports performance.

## **Anatomy and Physiology of the Nose and Paranasal Sinuses**

### *Anatomy of the nose and paranasal sinuses*

The bones of the skull (frontal, nasal, maxillae) contribute to the skeletal framework of the nose, but most of the external shape of the nose is comprised of soft tissue and cartilage <sup>12</sup>. The internal nose consists of two nasal cavities, separated by the nasal septum. The lateral nasal wall is made up of three paired turbinates (inferior, middle and superior). The lining of the nasal valve contains a rich blood supply under sympathetic nervous system control <sup>12</sup>. The area between the lateral nasal wall and the adjacent septum, up to the middle and inferior turbinate and upper edge of the vestibule, is the mobile part of the nose. This mobile part of the nose is made up of cartilage and facial muscles. The central quadrangular cartilage is also known as the septal cartilage. There are two paired upper lateral cartilages and two paired lower lateral cartilages. This mobile area has the ability to both open and close, which creates

a varying amount of resistance to airflow through the nose and nasal passages <sup>7</sup>. Lymphoid tissue (called the adenoids) may be present in the posterior part of the nose, the nasopharynx <sup>13</sup>. While the lymphoid tissue normally regresses in early childhood, enlarged adenoidal tissue can contribute to nasal obstruction in children and also in adults. In close relation to the nose, there are four, paired sinuses (frontal, ethmoid, maxillary and sphenoid). The sinuses are air-filled extensions of the nasal cavity. Ciliated pseudostratified columnar epithelium lines the majority of these nasal cavities.

### ***Normal functions of the nose and airflow dynamics at rest***

At rest and during sleep, humans are predominantly nose-breathers. The nose is not only an inlet of air to the lower airways, but has several highly developed physiological functions including heating, humidification, olfaction, phonation, chemo sensation, air-conditioning and filtration of air, and regulating airflow dynamics <sup>12</sup>. While all these functions are important, three functions deserve further discussion in the context of athletes. These are the defence against infections, allergen entrapment and the role of the nose in airflow dynamics.

The nose is an important defence system against respiratory tract infections. The nose filters the air for particles and pathogens. As air reaches the posterior nasopharynx it is exposed to adenoid tissue, which is a part of the immunological defence system against respiratory infections <sup>13</sup>. The paranasal sinuses may also play a part in protecting against infections. In addition to lightening the skull and contributing to voice resonance, the paranasal sinuses produce nitric oxide, which seems to be bacteriostatic and may serve as a further protective mechanism against infections <sup>14</sup>.

Allergen entrapment in the nose is an important protective function against lower airway inflammation and asthma and was first proposed in 1978 <sup>15</sup>. This protective function was confirmed by a recent review, although methodological challenges in the assessment of mouth breathing still exist <sup>16</sup>.

Regarding flow dynamics, inhaled air passing through the external nostrils is presented to a large mucosal surface. The narrow nasal valves and bony turbinates initially accelerate the inspired air, creating both laminar flow and turbulence. Most inhaled airflow travels between the inferior and middle turbinates. The inferior turbinates are the largest and these structures are responsible for the majority of the humidification, heating, filtering and resistance to air flow. The vascular tissue, lining the nasal valve, swells up and increases both airflow resistance and turbulence. In periods ranging from 1-7 hours, an alternating unilateral congestion of the nasal passage normally occurs, and this is called the nasal cycle <sup>17</sup>. This is of clinical importance because the nasal cycle per se may erroneously be identified as pathologic nasal obstruction. Other causes of transient swelling of the nasal turbinates are oestrogen containing oral contraceptives, pregnancy, sexual arousal and the supine position during sleep, which is associated with a shift in fluid distribution and a change of the nasal cycle <sup>18</sup>.

### ***Normal physiology of the nose during exercise***

Nasal function during exercise is particularly important for the athlete. As exercise intensity increases, minute ventilation rapidly increases and breathing switches from nasal to oral breathing to reduce resistance to the airflow. This switching occurs at a minute ventilation of approximately 35-45 ml/min <sup>19</sup>. Furthermore, at increasing exercise intensity, the sympathetic nervous system response results in vasoconstriction of the vessels in the nasal valve, which

increases the size of the nasal cavity and increases airflow in the nose and nasal passages <sup>5</sup>. There is unclear evidence to indicate that nasal airflow is reduced by exercise <sup>20,21</sup>.

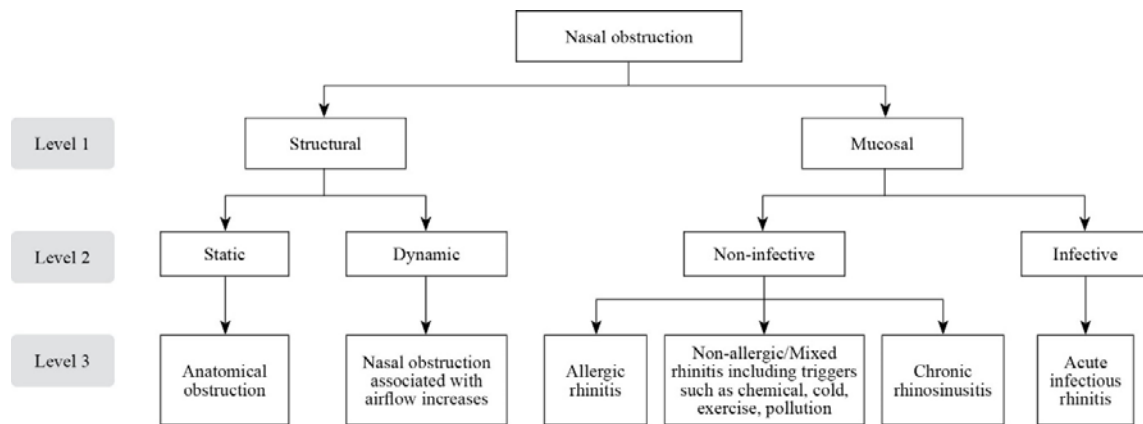
At maximal exercise intensity, the nasal airways normally contribute to only 10% of the overall minute ventilation, and maximal aerobic performance is not reduced when using a nose-clip <sup>22</sup>. The nasal airways do not seem to present a limiting factor in maximal exercise and attempts to increase maximal exercise capacity by reducing nasal resistance have not been successful <sup>23</sup>. On the other hand, there are studies showing that highly trained competitive athletes can adapt to exclusively nasal-breathing during running at maximal effort without loss in performance or peak aerobic capacity <sup>24,25</sup>. However, not all studies support these findings, as nasal breathing may lead to higher breathing-effort than oral breathing <sup>26</sup>.

Interestingly, all these studies reported a lower peak expiratory exchange ratio (RER) and lower maximal ventilation rate during nasal breathing. Even if the majority of athletes choose oral breathing during exercise, 8-17% may actually prefer nasal breathing <sup>27</sup>. Nasal breathing during exercise may have some benefits. Two recent meta-analysis reported an association between mouth breathing and asthma <sup>16</sup>, allergic rhinitis and poor sleep <sup>28</sup>. Moreover, there are studies reporting that bronchoconstriction during exercise is markedly reduced by nasal breathing compared to oral breathing <sup>15,29</sup>. Even if there seems to be an association between asthma, rhinitis, and sleep, the relationship is complex and beyond the scope of this review. However, it should be a reminder of how important it is to always consider interactions between the upper and lower airways when evaluating respiratory problems during exercise <sup>30</sup>. The long-term impacts of exercise on nasal function and nasal function on athletic performance are still unknown, and may depend on type of sport and duration.

In summary, nasal health may affect the athlete in different situations inside or outside the exercise environment. It is well documented that nasal obstruction influences sleep quality, mood and quality of life in athletes <sup>5,6</sup> and that this may have significant secondary negative effects on an athlete’s health and performance.

### Definition and Classification of Nasal Obstruction

Nasal obstruction can be defined as either “*narrowing of the nasal cavity*” (anatomical definition) <sup>31</sup>, or “*the subjective feeling of not being able to or inability to breathe optimally through the nose at rest and during exertion*” (functional definition) <sup>31</sup>. In general, nasal obstruction can originate from structural or mucosal causes (level 1), which can then be further classified into four main types of pathology (level 2) with a sub-classification of the non-infective group of pathologies (level 3) (Figure 1).

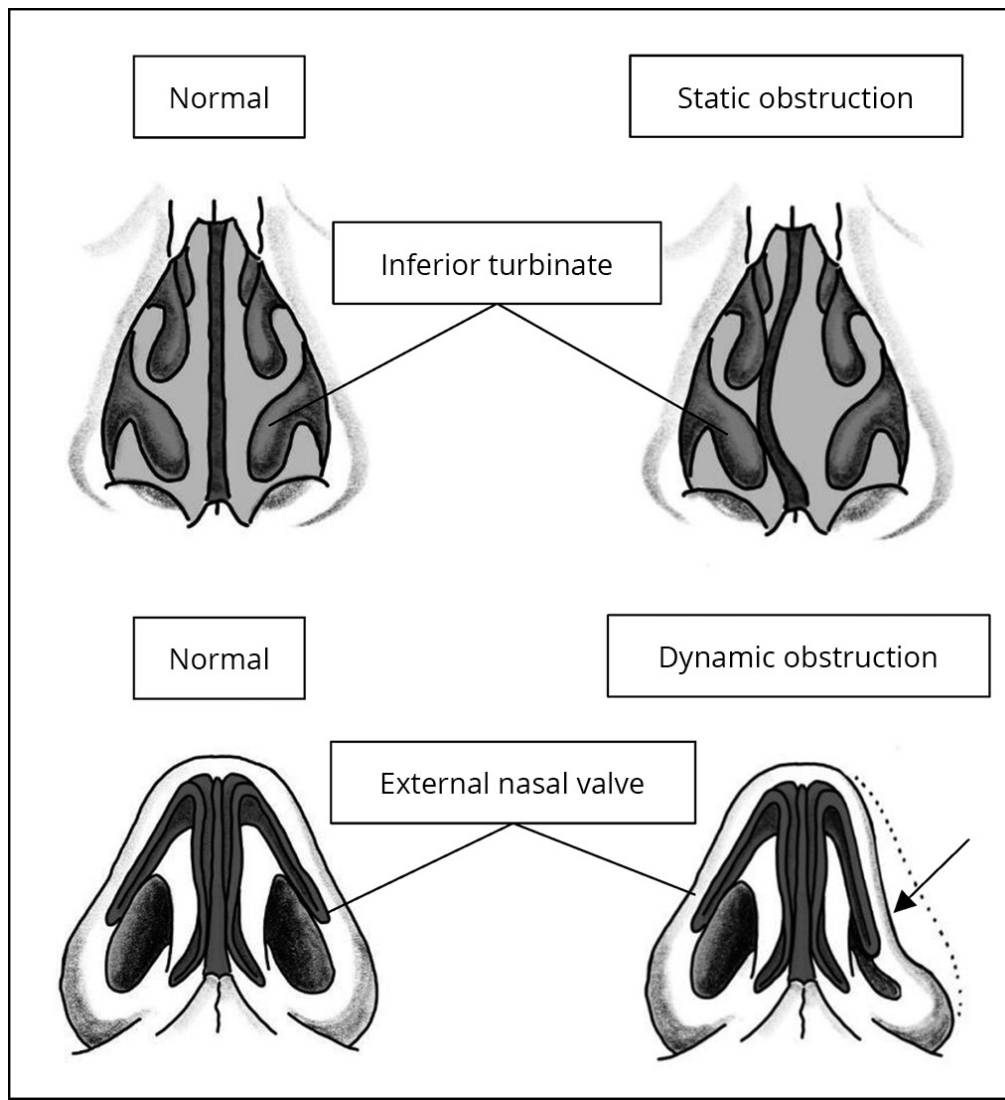


**Figure 1.**—Classification of nasal obstruction.

#### ***Structural (anatomical) causes of nasal obstruction***

Structural (anatomical causes) can be further classified into static (permanent) or dynamic (intermittent) causes. In static (permanent) obstruction, nasal airway narrowing remains constant and is not affected or aggravated by an increase in airflow. Static obstruction is caused by pathology in the structural layer of the nose. Dynamic (intermittent) nasal

obstruction refers to transient nasal airway narrowing when an increase in airflow through the nose causes collapse of the external valve (vestibule/lateral crura of lower lateral cartilages) or the internal nasal valve (area boarded by the upper lateral cartilage, septum and head of the inferior turbinate) in some individuals<sup>31</sup>. The collapse is a result of an increase in negative pressure created by the Venturi effect when there is a high rate of airflow (Figure 2).



**Figure 2.**—Anatomy of the nose and structural obstructions (static and dynamic). Illustrated by Hege Clemm.



### ***Mucosal causes of nasal obstruction***

Mucosal inflammation, with swelling and congestion, can cause nasal obstruction. The causes of inflammation of the nasal mucosa can be further classified as either infective or non-infective. In non-infective rhinitis, mucosal inflammation is caused by a trigger, irritant or exposure, which together with nasal hyperreactivity (NHR) leads to mucosal swelling and nasal obstruction. Causes of rhinitis are often mixed and can then be difficult to differentiate. NHR triggered by allergy is perhaps the easiest type of rhinitis to distinguish from the other causes. Non-allergic rhinitis can both be idiopathic or differentiated into different sub-classifications depending on the trigger. In athletes, irritants or exposures like chlorine in swimmers and exercise in endurance athletes act as triggers <sup>20,32</sup>. In chronic rhinosinusitis, the paranasal sinuses are also affected, often causing pressure and pain. However, for the SEM physician, it is often difficult to differentiate between non-allergic rhinitis and acute or chronic rhinosinusitis. Accordingly, our recommendations are based on a recent review on rhinology in SEM <sup>11</sup>, and central figures in two recently published European position papers on non-allergic rhinitis and rhinosinusitis and nasal polyps respectively <sup>33,34</sup>.

### **Prevalence of Nasal Obstruction in Athletes**

Nasal symptoms are significantly more frequent in athletes than non-athletes (70% vs. 52%) and upper respiratory tract infections are significantly particularly common in athletes suffering from nasal symptoms <sup>32</sup>. There is a paucity in the literature regarding prevalence on structural nasal obstruction in athletes. The estimated prevalence of rhinitis in the general population is 20-25% <sup>35,36</sup> and a systematic review from 2017 reported that the prevalence of rhinitis in the athlete ranges from 27-74% <sup>10</sup>. However, studies reporting prevalence of allergic rhinitis, non-allergic rhinitis and chronic rhinosinusitis in athletes are lacking and most studies only report allergic rhinitis. According to the 2020 European position paper on

rhinosinusitis and nasal polyps (EPOS 2020), self-reported rhinosinusitis has a higher prevalence than rhinosinusitis diagnosed by physicians<sup>33</sup>. One study on non-allergic rhinitis, reported the prevalence to be 45% in elite-swimmers, which was higher than in non-elite swimmers and a previously reported prevalence of allergic rhinitis in about 12% in elite and non-elite swimmers<sup>6</sup>. We could only identify one study reporting on chronic rhinosinusitis in athletes, where 3.2% of swimmers with nasal symptoms were diagnosed with chronic rhinosinusitis<sup>37</sup>. Because of exercise in chlorinated water, which can act as a trigger for rhinitis, the prevalence of rhinitis in swimmers is not representative of the overall athletic population<sup>38</sup>. In summary, there are very few studies reporting the prevalence of nasal obstruction in athletes caused by structural obstruction, non-allergic rhinitis and chronic rhinosinusitis.

#### **A general clinical approach to the diagnosis of nasal obstruction in athletes**

It is important for the SEM physician to have a general clinical approach to the screening, diagnosis, management and prevention of causes of nasal obstruction in athletes. In athletes with suspected nasal obstruction, the ability to distinguish between a structural or mucosal cause for obstruction is an essential first step, as the management differs. It is also important to have clear indications for referral to an otorhinolaryngologist for further management in certain cases of nasal obstruction.

A variety of validated questionnaires are used by otorhinolaryngologists to screen for nasal conditions. These include the Nasal Obstruction Symptom Evaluation (NOSE) scale<sup>39</sup>, the sino nasal outcome test (SNOT) 20<sup>40</sup>, and later the SNOT 22<sup>41</sup>. Currently, there is no athlete-specific screening questionnaire to aid SEM physicians in the screening for nasal conditions.

The only sports-related validated questionnaire is the AQUA <sup>42</sup>, which has a focus on allergic rhinitis.

In athletes that present with nasal symptoms, we propose that SEM physicians consider the following stepwise approach to the diagnosis of nasal obstruction that is based on the classification of nasal obstruction (Supplemental file 1).

*Step 1: Screen for possible nasal obstruction (NOSE score)*

*Step 2: Obtain a detailed history of nasal symptoms, followed by a general medical history*

*Step 3: Conduct a systemic examination of the nose to determine the causes of obstruction*

*Step 4: Decide on the use of special investigations to determine the nature, severity and cause of nasal obstruction*

A key element in the diagnosis is a good history and the type and severity of symptoms can often distinguish between causes of nasal obstruction (Table I).

We suggest that the SEM physician follow this general stepwise approach to the diagnosis of nasal conditions causing obstruction (Supplemental file 1). A more specific diagnostic and management approach to each of the groups of nasal conditions causing obstruction will now be reviewed.

**Table I: Nasal symptoms and athletes at risk for different causes of nasal obstruction**

Symptom/history	Structural		Mucosal*			
	Static	Dynamic	Non-infectious			Infectious
			CRS	Non-allergic/ Mixed	Allergic	
Symptoms of nasal obstruction (NOSE score)	+++	++	+++	+++	++	+++
Rhinorrhoea/Discharge	-	-	+++	+	+	+++
Post-nasal drip	-	-	+++	-	+++	+++
Sneezing	-	-	+	+	++	++
Nasal/palatal itch	-	-	-	+	+++	-
Facial pain/pressure/headache	-	-	+++	+	-	++
Loss of smell	-	-	+++	-	-	++
Sleep disturbance	+++	+	+++	++	++	+++
Unilateral obstruction	+++	+++	+	+	-	+
Exercise induced nasal obstruction	-	+++	?	+++	+	+
Trigger-induced nasal obstruction**	-	-	?	+++	+++	+++
<b>Athletes at risk</b>						
Contact sport	+++	+	-	-	-	+
Swimmers	-	-	+	+++	+++	++
Winter sport	-	-	?	+++	+	++
Endurance athletes	-	+	?	+++	+++	++

The number of + signs indicate the relative frequency / severity of symptoms for different causes of nasal obstruction and athletes at risk

NOSE: NOSE questionnaire

CRS: Chronic rhinosinusitis

\* Mucosal symptoms - adapted from Fokkens et al<sup>33</sup> and Hox et. al<sup>11</sup>.

\*\*Triggers can include cold, pollution, chlorine, chemical, allergens and others

## Structural Nasal Obstruction

### Structural static (permanent) nasal obstruction

A structural static (permanent) anatomical cause of nasal obstruction is continually present usually because of a bony anatomical abnormality that results in narrowing of the nasal airway. The degree of nasal obstruction does not increase if breathing rate during exercise intensity increases, because the anatomical bony structures are not influenced by changes in airflow and pressures. Examples of static obstruction can be a deviated quadrangular

cartilage, a deviated bony septum, a septal spur, a concha bullosa, an enlarged inferior turbinate, pyriform narrowing, choanal atresia or an ethmoid bulla <sup>31</sup>.

In athletes, the most frequent risk factor associated with a structural static (permanent) anatomical cause of nasal obstruction is a history of previous trauma to the nose and face. Although this is more common in athletes participating in contact sports, it may also be unrelated to any sporting activity. Other risk factors for static obstruction are congenital abnormalities of the nose (e.g. cleft lip, palate or both), and previous nasal surgery (failed septal surgery or rhinoplasty).

#### *Specific diagnostic considerations*

In general, athletes with a structural static (permanent) anatomical cause of nasal obstruction will have a high nasal score (NOSE) <sup>39</sup> 7. Other symptoms that are more specific will be a chronic history of symptoms, obligate mouth breathing, snoring, poor sleeping habits and a dry throat in the morning. On inspection, there may be facial or nasal asymmetry and anterior rhinoscopy may identify the cause of unilateral or bilateral structural abnormalities. The Cottle manoeuvre, which is a clinical office test to determine if there is dynamic nasal valve collapse (see Supplemental file 1 for a detailed description to perform the manoeuvre), will not improve nasal airflow after a topical decongestant. The SEM may suspect a structural static (permanent) anatomical cause of nasal obstruction, but the definitive diagnosis is usually made by an otorhinolaryngologist using nasal endoscopy and / or a computerised tomography (CT) scan of the nasal passages and paranasal sinuses. The severity of nasal obstruction is then assessed by the otorhinolaryngologist using rhinomanometry, acoustic rhinometry, the peak nasal inspiratory flow (PNIF) test and rigid nasal endoscopy.

### *Principles of management, prevention and referral*

Although some athletes with an anatomical cause of nasal obstruction may be able to perform on the sports field, chronic permanent nasal obstruction can lead to several health problems, including poor quality of life, poor sleep and inadequate recovery from training or competition. The SEM physician needs to be aware of these risks and consider early intervention for their patients.

The most important preventative strategy to reduce the risk of a structural static (permanent) anatomical cause of nasal obstruction is to reduce the risk of nasal trauma during sport, particularly contact sports. If acute nasal trauma does occur, these should be treated according to advance trauma life support (ATLS) principles<sup>43</sup>. If possible, immediate reduction of nasal fractures on the field by skilled medical staff is recommended, otherwise surgical reduction of fractures is preferable within 7 days. A septal hematoma needs urgent referral because it can lead to infection and subsequent destruction of the quadrangular cartilage, resulting in a saddle nose deformity and complete nasal obstruction<sup>44</sup>.

Early referral to a qualified otorhinolaryngologist for further surgical management is recommended<sup>45</sup>. However, it is recommended to wait for at least 3 months post injury to do a formal surgical repair if the injury has taken place >7 days previously. An athlete would then have to wait for at least 6 weeks before contact sports may resume. Timing surgery for the off-season would be advisable<sup>46</sup>.

### **Structural dynamic (intermittent) nasal obstruction**

A dynamic/intermittent nasal obstruction becomes evident when the breathing rate or intensity increases. As the flow of air through the nose increases, the Venturi effect causes higher negative pressures in the nasal cavities. If there is weakness of the soft tissues of the

nose (nasal muscles, ligaments and lateral cartilages) then these structures can collapse medially leading to an incomplete or complete obstruction of the nasal passage on that side. An inherent weakness of the upper lateral cartilages is associated with an internal nasal valve collapse, while a weakened lower lateral cartilage is associated with an external nasal valve collapse <sup>47</sup>.

Risk factors associated with a dynamic nasal obstruction are similar to those for a static nasal obstruction. However there are two key differences: injury to cranial nerve 7 (after previous surgery or trauma) resulting in weakness of the nasal muscles can lead to nasal valve insufficiency, and dynamic obstruction is only evident in sports that require an athlete to do forceful nasal breathing <sup>48</sup>.

#### *Specific diagnostic considerations*

A key feature in the history is that normal nasal breathing at rest is not associated with any symptoms of obstruction and that symptoms only become noticeable during forced nasal inspiration. Recent onset symptoms are most often associated with trauma or failed surgery, while longstanding symptoms are suggestive of a congenital cause. The diagnosis can be made by inspecting each nostril during forced inspiration through the nose. A differentiation between external (lower lateral cartilage) and internal (upper lateral cartilage) valve collapse can be made by inspecting from below (“worms eye view”) during anterior rhinoscopy. Special investigations to confirm the diagnosis and assess the severity of dynamic nasal obstruction are usually done by an otorhinolaryngologist. These tests, which include four-phase rhinomanometry, acoustic rhinometry and peak nasal inspiratory flow (PNIF) testing, are done before and after mechanical intervention manoeuvres. The results of these tests will be able to prove dynamic obstruction and give an indication of a possibly surgical outcome <sup>49</sup>.

### *Principles of management, prevention and referral*

For the management of a dynamic nasal obstruction, temporary relief can be achieved with various internal or external nasal dilating devices that are available on the market. These can be internally or externally placed by the athlete themselves when they feel it is necessary<sup>50 51</sup> and can be used during training, competition, recovery or even during sleep. However, surgery is often the only permanent solution. There are different surgical techniques to address internal or external valve collapse<sup>52</sup>, but a detailed discussion of these is beyond the scope of this review<sup>45</sup>. Nasal valve surgery is not a common Otorhinolaryngology procedure and care should be taken to refer to an appropriate surgeon experienced in this field.

The main indications for referral are reduced quality of life, reduced training/performance and if nasal dilators have failed. In these instances, it is advised to refer the athlete to an Otorhinolaryngologist or Facial Plastic Surgeon for possible nasal valve surgery<sup>53</sup>.

### **Mucosal Causes of Nasal Obstruction**

The general pathophysiological mechanism responsible for mucosal nasal obstruction is inflammation of the mucosa of the nose (rhinitis) and paranasal sinuses (rhinosinusitis). In general, chronic rhinitis / rhinosinusitis can occur as a result of an infection (acute rhinitis / rhinosinusitis) or a non-infectious cause. Athletes may be exposed to several triggers for non-infectious rhinitis because they are exposed to various sports-specific environments such as cold, pollution, irritants, chlorinated water and allergens. All these triggers are believed to stimulate the respiratory epithelium of the nose as well as the underlying nervous system. Activation of the nervous system can lead to direct induction of nasal symptoms and nasal hyperreactivity. Prolonged and / or repetitive exposure of different sport-specific triggers, might also lead to an activation of the immune system, inducing nasal inflammation<sup>11</sup>. Given



all the risk factors that athletes are exposed to, it can be a challenge to differentiate between the different types of non-infectious rhinitis. Non-allergic rhinitis can result from a variety of different triggers, while different triggers can also aggravate allergic rhinitis. Regardless of the interactions between these causes of mucosal obstruction, the extent of mucosal nasal obstruction in athletes is probably greater than what is generally appreciated and further research is needed. A diagnostic and management approach to each of the causes of mucosal nasal obstruction will now be reviewed, with the aim to provide the SEM physician with a clinical approach to these conditions.

### **Acute infectious rhinitis / rhinosinusitis**

Upper respiratory infections are common in elite athletes<sup>54</sup>. They account for 30-40% of visits to sports medicine clinics<sup>55</sup> and were one of the main reasons for athletes to consult a physician during the 2002 Winter Olympic Games<sup>56</sup>. Although acute rhinitis / rhinosinusitis is one of the most common clinical presentations of upper respiratory illness, these infections may be over diagnosed. Physicians often diagnose acute upper respiratory illness as viral or bacterial-related infections, but there are data showing that a pathogen or other factors indicative of infections cannot be confirmed in about 50% of suspected cases<sup>57</sup>. Therefore, non-infectious and non-allergic causes of acute rhinitis must be considered as a possible differential diagnosis.

In the majority of cases, viral rhinitis is self-limiting and with few long-term consequences. Acute bacterial rhinosinusitis can often be distinguished from other causes of acute rhinitis by additional symptoms of discoloured nasal discharge, facial pain or loss of smell<sup>33</sup>.

There are several risk factors associated with acute respiratory infections in athletes including poor personal hygiene habits, nutritional deficiencies, low energy availability, travel, and increased training and competition load <sup>58</sup>. In general, elite athletes suffer more frequently from infectious rhinitis than non-elite athletes <sup>59</sup>. Pre-existing nasal conditions also predispose to infectious disease <sup>5</sup> and in a recent review article it was suggested that exercise-induced decrease in immunoglobulin (Ig) A secretion is the most probable explanation for a higher prevalence of infectious rhinitis in athletes <sup>11</sup>. However, strong evidence to link acute physical stress to reduced immunodeficiency in athletes is lacking <sup>5,59</sup>. Recovery from training and sleep also affects post-exercise immune function <sup>60</sup>. In summary, the relation between exercise, rest, sleep, nutrition, and low energy availability as risk factors for infectious rhinitis require further investigation.

#### *Specific diagnostic considerations*

The SEM physician will suspect an acute infectious rhinitis by following the general approach to the diagnosis of nasal obstruction. According to the EPOS 2020 care pathway for acute rhinosinusitis (Figure 3), it is important to differentiate between single infections and recurrent infections <sup>33</sup>. Acute infections are distinguished from other rhinitis by short duration of symptoms, recurrent symptoms with symptom free in-between, and sometimes fever and malaise <sup>33</sup>. Other characteristics are the sudden onset of two or more symptoms; one of which should be either nasal obstruction or nasal discharge, and facial pain/pressure and/or reduction/loss of smell. A pattern of double sickening should also raise suspicion of an acute bacterial rhinosinusitis (Figure 3).



As illustrated by the recent COVID-19 pandemic, the principles of prevention of infectious rhinitis are personal hygiene (frequent hand washing), physical distancing and wearing of surgical masks. Additional general prevention measures include training and competition load management, optimal nutrition, adequate duration and quality of sleep <sup>58</sup>. Recent publications emphasized the importance of sleep and rest in athletes <sup>61</sup>, particularly the important role of sleep for the immune system <sup>60</sup>. However, there are no well-defined studies on the effects of these prevention measures on reducing the frequency of infectious rhinitis in athletes.

The indications for referral of an athlete with infectious rhinitis / rhinosinusitis to a tertiary care facility (otorhinolaryngologist) are acute “red flag” or alarm symptoms (Figure 3) and ongoing or recurrent acute infection (athletes that report  $\geq 3$  episodes of rhinosinusitis per year) because bacterial reservoirs may be surgically removed. In recurrent rhinosinusitis, the importance of the immune system in relation to recovery and the overtraining syndrome should be considered <sup>58</sup>.

### **Allergic rhinitis**

Allergic rhinitis is caused by sensitization leading to future allergic responses involving antigen presenting cells, T helper cells, B cells, mast cells and immunoglobulin E (IgE) <sup>62</sup>.

The allergic reaction is often divided in an early phase response (5-15 minutes) that is characterized by symptoms from histamine released from mast cells and a late phase response (lasting 2-4 hours), which is characterized by cytokine recruitment leading to chronic inflammation.

Athletes with a genetic predisposition to allergies and atopy are at risk, particularly if outdoor athletes are also exposed to seasonal allergens. In general, the prevalence of allergic rhinitis in

the athletic population is comparable to the prevalence in the general population <sup>10</sup> but there are sport-specific athletes that are at higher risk such as aquatic athletes <sup>6</sup>.

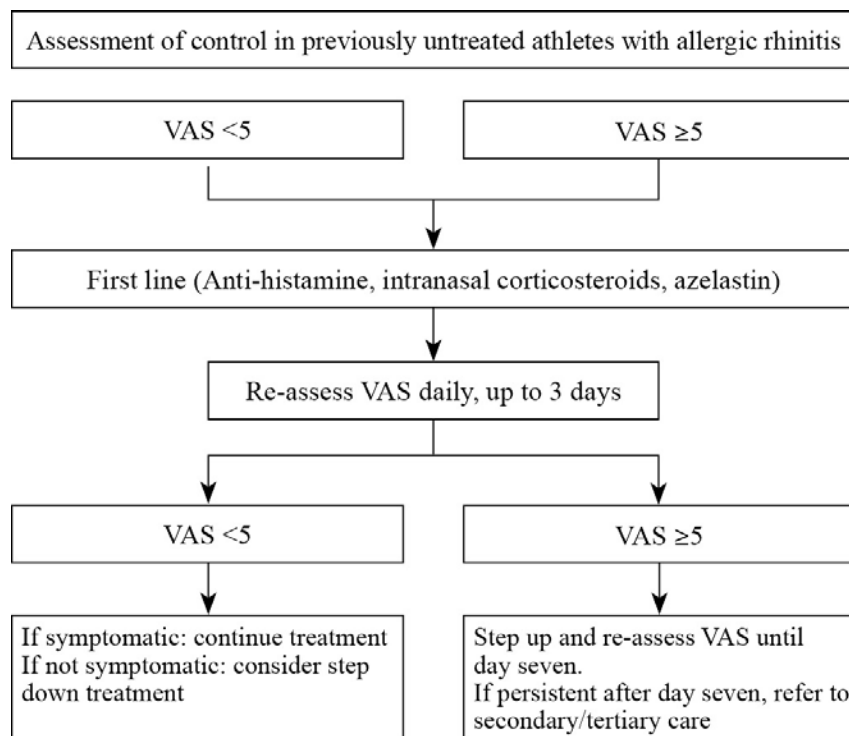
#### *Specific diagnostic considerations*

The SEM physician will suspect allergic rhinitis by following the general approach to the diagnosis of nasal obstruction. Itching of the nose and sneezing are two important symptoms that are caused by allergy as a result of histamine release. Depending on the allergen that induces the rhinitis, symptoms are mostly intermittent and are related to the allergen exposure. Other diagnostic features of allergies are genetic predisposition seasonal exacerbation, involvement of other sites such as the conjunctiva, skin and lower respiratory tract symptoms <sup>62</sup>, and symptoms of nasal obstruction, discharge, nasal/palatal itching. Allergic rhinitis can be confirmed with a positive blood sample for the specific IgE antibodies. If the test results are difficult to interpret, such as too many positive findings, an allergy specialist can order more specific blood tests or perform skin prick testing to confirm the diagnosis of specific allergies.

#### *Principles of management, prevention and referral*

A useful practical tool in the management of athletes with suspected allergic rhinitis is to ask athletes to complete a daily self-rating using a visual analogue scale (VAS) from 0-10, based on the following statement: *Overall how much are your allergic symptoms bothering you today, from “not at all bothersome” to “extremely bothersome”* <sup>63</sup> (Figure 4). The main treatment of allergic rhinitis is the identification of allergens by anamnesis and to avoid exposure to allergens <sup>62</sup>. Nasal rinse, rather than a topical nasal spray, is recommended as first line treatment in all athletes with allergic rhinitis during the allergic season. If pharmacotherapy is needed, antihistamines (oral or topical), decongestants for short-term

relief and nasal saline should be considered. For further treatment, the recent “*Allergic Rhinitis and Its Impact on Asthma (ARIA)*” guideline should be followed <sup>64</sup> using the VAS (Figure 4).



**Figure 4.**—Assessment of control in previously untreated athletes with allergic rhinitis (modified from Bousquet et al.).<sup>63</sup>

VAS: Visual Analogue Scale.

During follow-up, if the VAS remains  $\geq 5$  after 7 days of initial treatment, the SEM can consider referral of the athlete to an Otorhinolaryngologist or a specialist in allergy. When allergic rhinitis causes impaired sports performance and quality of life in general, and regular treatment does not solve it, immunotherapy (sublingual (SLIT) or subcutaneous (SCIT)) may be considered in a secondary health care setting <sup>64</sup>. Treatment with anticholinergics, leukotriene receptor antagonists, mast cell stabilisers, mucolytics, and corticosteroids (oral or topical) can also be considered in a secondary health care. There is no known prevention for sensitization of allergens, and avoidance of allergen exposure is not always possible. Other

practical clinical considerations are to start with allergy treatment before the onset of pollen season and use saline nasal rinsing to remove pollen after exercise.

### **Non-allergic rhinitis**

Non-allergic rhinitis is defined as symptomatic inflammation of the nasal mucosa in the presence of a minimum of two nasal symptoms such as nasal obstruction, rhinorrhoea, sneezing, and/or itchy nose, without clinical evidence of endonasal infection and without systemic signs of sensitization to inhalant allergens<sup>34</sup>. In non-allergic, (non-infectious) rhinitis and rhinosinusitis, inflammation and hyper reactivity to various stimuli or triggers is the major pathophysiological mechanism<sup>33</sup>. A vasomotor, parasympathetic dominance is the main cause of hyper reactivity and stimulation of the parasympathetic system can be triggered by eating, freezing and gazing at the sun<sup>65</sup>. Thus, non-allergic rhinitis can be sub-classified according to various triggers including the following: hormonal, occupational (inhaled irritant), drug-induced (non-steroidal anti-inflammatory drugs, NSAIDs, angiotensin converting enzyme (ACE) inhibitors, beta-blockers and oral contraceptives), gustatory, rhinitis medicamentosa (rebound congestion after >5 days of topical decongestant use), senile rhinitis and idiopathic rhinitis (IR)<sup>34</sup>.

More specifically in athletes, exercise itself can be a trigger as well as exposure to irritants that are characteristic of certain sport types (e.g. chlorine in swimmers, cold air in winter athletes, and changes in humidity, particularly dry air). The resultant rhinorrhoea, itch, sneezing, congestion and oedema causes nasal obstruction<sup>11</sup>.

There are certain athlete populations that may be at higher risk for non-allergic rhinitis. Data from several studies show that swimmers seem to be at a higher risk of especially non-allergic rhinitis, related to nasal hyper responsiveness due to chemical irritation<sup>6 32</sup>. Increased nasal

symptoms and nasal epithelial injury was reported in swimmers but not in indoor athletes<sup>38</sup>. Although nasal changes seem to normalize after a few weeks of training cessation the potential long-term consequences of repeated epithelial injury are not known. There is a risk that, before nasal mucosa is normalized, the defence barrier may be impaired leading to increased risk of upper respiratory tract infections.

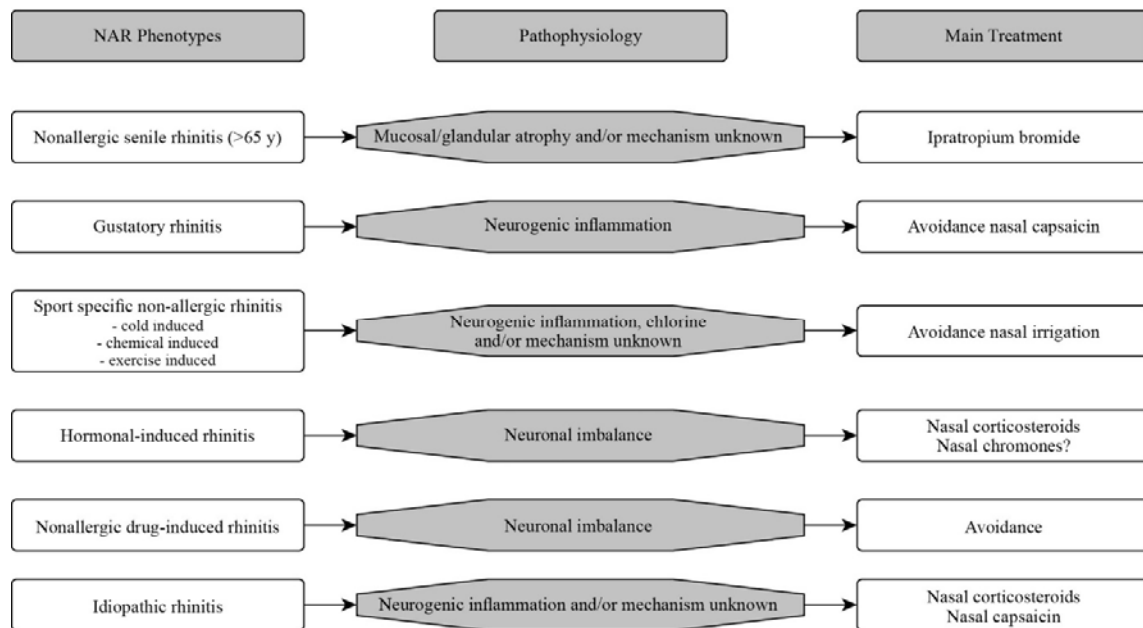
#### *Specific diagnostic considerations*

The diagnosis of non-allergic rhinitis is made by following the general approach of a history, clinical examination and special investigations, if needed. Non-allergic rhinitis should be suspected in athletes with recurrent, non-infectious episodes of nasal obstruction. The hallmark of non-allergic rhinitis is nasal hyper responsiveness and by taking a good history, the SEM physician could differentiate between the main sub-types of non-allergic rhinitis. However, to differentiate between non-allergic rhinitis and chronic rhinosinusitis (CRS) without a nasal endoscope or computer tomography of the paranasal sinuses may be challenging<sup>33</sup>. Facial pain/pressure and loss of smell are features of CRS rather than non-allergic rhinitis. Special investigations are helpful, and if there is no evidence of infection and no positive findings on allergy tests, non-allergic rhinitis is likely to be the cause.

#### *Principles of management, prevention and referral*

We recommend that the treatment of non-allergic rhinitis should be in line with the position paper of the European Academy of Allergy and Clinical Immunology (Figure 5)<sup>34</sup>.





**Figure 5.**—The phenotypes, pathophysiology and main treatment of athletes with non-allergic rhinitis (NAR) (modified from Hellings et al.).<sup>34</sup>

The first line of treatment (as in allergic rhinitis) is avoidance of the triggers that cause mucosal inflammation<sup>34</sup> and, as with allergic rhinitis, saline nasal rinsing is the first level of active treatment. If pharmacotherapy is needed, anticholinergics, leukotriene receptor antagonists, mast cell stabilisers, mucolytics and nasal corticosteroids may be considered, dependent on phenotype (Figure 5). The mainstay of prevention is avoidance of the triggers and referral to secondary care should be considered athletes do not responding to treatment.

### **Chronic rhinosinusitis (CRS)**

Chronic rhinosinusitis is a heterogeneous disease characterised by inflammation, mucociliary dysfunction and changes in the microbial environment in the nose and paranasal sinuses<sup>33</sup>.

Prior to 2020, chronic rhinosinusitis sub-classified as either “CRS with or without nasal polyps”. Recently EPOS 2020 classified chronic rhinosinusitis as either primary and secondary, with a further sub-classification into localized and diffuse disease.

The most important modifiable risk factors for primary CRS are tobacco smoke and occupational irritants. The role of allergy as a predisposing factor for CRS is controversial, while the association between asthma and CRS is indisputable<sup>33</sup>. A particular subtype of CRS, with relevance to sport medicine, is non-steroidal anti-inflammatory drug (NSAID) - exacerbated respiratory disease. The prevalence of aspirin insensitivity is as high as 2-3 percent in the general population.

The development of chronic rhinosinusitis is also related to other factors such as the immune system and the ciliary function in the nose<sup>64</sup>, sleep disorders and snoring that can progress to obstructive sleep apnea (OSA)<sup>60,66-69</sup>. The prevalence of OSA can be higher in certain athlete populations such as forward rugby players<sup>70</sup>. Finally, OSA is also associated with nocturnal gastroesophageal reflux, which is believed to be an important risk factor for secondary chronic rhinosinusitis<sup>71</sup>.

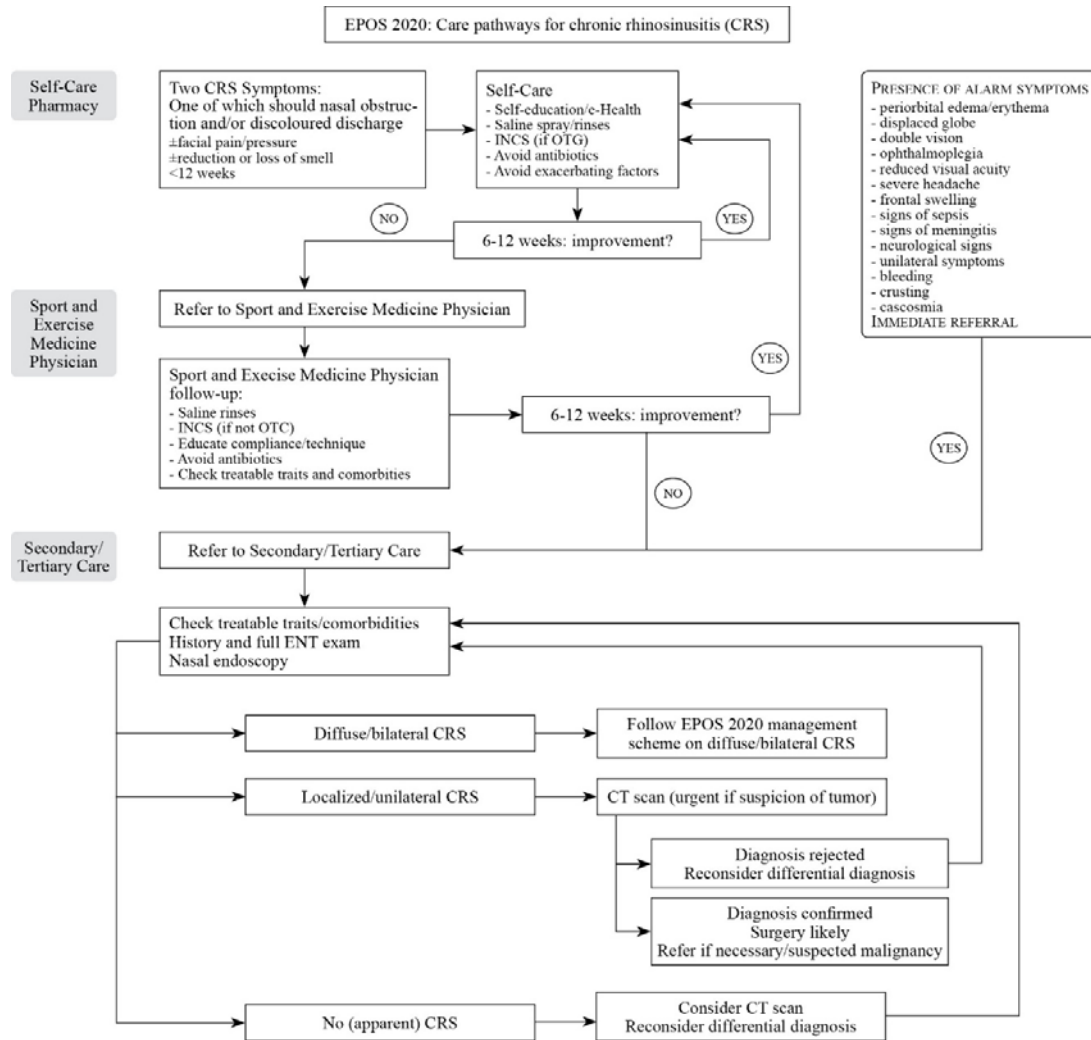
#### *Specific diagnostic considerations*

The diagnosis of chronic rhinosinusitis is made on history, clinical assessment and use of special investigation. On history, CRS is suspected if an athlete presents with a history of two or more symptoms, one of which should be either nasal obstruction or nasal discharge, facial pain/pressure and reduction or loss of smell, lasting for  $\geq 12$  weeks<sup>33</sup>. The inspection of breathing pattern (oral vs nasal) and inspection of the outer nose and nasal valve may provide useful information. However, the definitive diagnosis is made by CT scan and nasal endoscopy<sup>33</sup>.

*Principles of management, prevention and referral*

There is very limited literature on the management of chronic rhinosinusitis in athletes <sup>72</sup>.

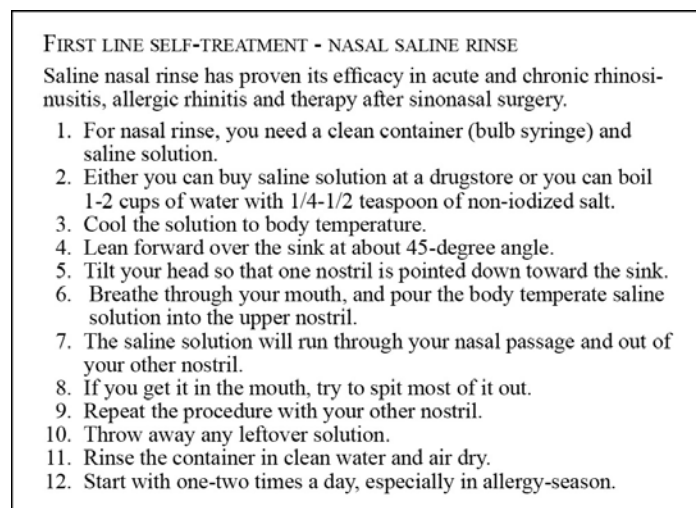
Therefore, we advise that SEM physicians follow the general recommendations of EPOS 2020 (Figure 6).



**Figure 6.**—The diagnosis, treatment and follow-up of athletes with chronic rhinosinusitis (modified from Walker et al.).<sup>5</sup>

EPOS: European Position Paper on Rhinosinusitis and Nasal Polyps; INCS: intra-nasal corticosteroids; OTC: over the counter; ENT: ear nose throat; CT: computer tomography.

As a first line management, the athlete can be advised to perform self-care for at least 6-12 weeks using a mobile application that has been developed to support self-care in all chronic disorders of the nose <sup>73</sup>. The first-line treatment is saline rinse (Figure 7).



**Figure 7.**—First line self-treatment: nasal saline rinse.

If additional pharmacotherapy is needed, nasal corticosteroids are the treatments of choice. Antibiotic therapy is not recommended in the treatment of CRS <sup>33</sup>. Obstructive sleep apnoea (OSA) is an important pathological factor in the development of chronic rhinosinusitis <sup>74</sup> and unilateral nasal congestion is associated also with the development of OSA <sup>75</sup>. However, the effect of nasal surgery in OSA is limited regarding objective measurement indices <sup>76</sup>. More research is needed regarding the role of the relation between chronic rhinosinusitis and OSA in athletes.

The principle of prevention of chronic rhinosinusitis is to remove modifiable risk factors. Tobacco smoke is seldom a problem for athletes and chemical irritants may be effectively removed with saline nasal irrigation <sup>33</sup>. The two main indications for referral are: 1) the presence of “red flag” or alarm symptoms, and 3) no improvement after 6-12 weeks of management by the SEM.

## Summary and Future Perspectives

Nasal obstruction can be divided into static, dynamic and mucosal causes. Mucosal causes of obstruction can again be divided into allergic and non-allergic rhinitis, acute and recurrent infectious rhinitis, and chronic rhinosinusitis. All different types of obstruction will affect athletes' performance, mainly due to poor level of sleep and restoration, and reduced level of quality of life. First line treatment is avoidance of triggers and self-care, however if a structural obstruction is suspected or if the obstruction is recurrent with no improvement after ordinary treatment, referral of the athlete to an otorhinolaryngologist is recommended. The diagnosis, management and prevention of causes of nasal obstruction are summarized in Table II.

Improved diagnosis and management of nasal obstruction in athletes is important to optimize athletes' health, recovery and performance, and may reduce the risk of acute respiratory illness such as infections.

The aim of this review was to increase awareness among SEM physicians of possible nasal obstruction in athletes presenting with nasal symptoms. Lack of recognition may lead to both under-treatment and erroneous treatment of nasal obstruction. This review focussed on risk factors, causes management and prevention of nasal obstruction. Future studies can focus on differentiating causes of nasal obstructions in athletes, the prevalence of non-allergic rhinitis and dynamic and static causes of nasal obstruction, the impact that nasal obstruction has on athletes' quality of life and performance, effects of different treatment modalities for nasal obstruction in athletes, and the validation and use of targeted athlete questionnaires to screen for nasal obstruction in athletes.

**Table II: A summary of the diagnosis, management and prevention of common causes of nasal obstruction in athletes**

		Structural		Mucosal			
		Static (Permanent)	Dynamic (Intermittent)	Non-Infectious rhinitis			Infectious rhinitis
				Chronic rhinosinusitis	Non-allergic/mixed rhinitis	Allergic rhinitis	
<b>Risk factors *</b>		<ul style="list-style-type: none"> <li>• Contact sports</li> </ul>	<ul style="list-style-type: none"> <li>• Contact sports</li> <li>• Endurance athletes</li> <li>• Previous trauma or facial surgery</li> </ul>	<ul style="list-style-type: none"> <li>• Swimmers</li> </ul>	<ul style="list-style-type: none"> <li>• Swimmers</li> <li>• athletes exposed to cold air and change in humidity</li> </ul>	<ul style="list-style-type: none"> <li>• Genetic predisposition to allergy and atopy</li> <li>• Swimmers and outdoor athletes exposed to pollen</li> </ul>	<ul style="list-style-type: none"> <li>• All athletes</li> <li>• Those with a previous history of rhinitis</li> </ul>
<b>Diagnosis</b>	<b>History General symptoms and signs *</b>	<ul style="list-style-type: none"> <li>• History of previous trauma.</li> <li>• Obligate mouth breathing.</li> <li>• Continual unilateral obstruction</li> </ul>	<ul style="list-style-type: none"> <li>• Unilateral symptoms appear on forced inspiration or during exercise</li> </ul>	<ul style="list-style-type: none"> <li>• Nasal obstruction &gt; 12 weeks</li> <li>• Rhinorrhoea/ discharge</li> <li>• Postnasal drip</li> <li>• Sneezing</li> <li>• Facial pain/pressure</li> <li>• Loss of smell</li> <li>• Sleep disturbance</li> </ul>	<ul style="list-style-type: none"> <li>• Nasal obstruction</li> <li>• Rhinorrhoea/ discharge</li> <li>• Sneezing</li> <li>• Itchy nose</li> <li>• Sleep disturbance</li> </ul>	<ul style="list-style-type: none"> <li>• Nasal obstruction</li> <li>• Rhinorrhoea/discharge</li> <li>• Postnasal drip</li> <li>• Sneezing</li> <li>• Nasal/palatal itch</li> <li>• Sleep disturbance</li> </ul>	<ul style="list-style-type: none"> <li>• Nasal obstruction</li> <li>• Rhinorrhoea/discharge</li> <li>• Postnasal drip</li> <li>• Sneezing</li> <li>• Facial pain/pressure</li> <li>• Loss of smell</li> <li>• Sleep disturbance</li> </ul>
	<b>Clinical examination findings Specific symptoms and signs</b>	<ul style="list-style-type: none"> <li>• Unable to visualise nasal cavity due to cause of obstruction</li> </ul>	<ul style="list-style-type: none"> <li>• Internal or external valve collapse during increased inhalation from exertion during training and competition</li> </ul>	<ul style="list-style-type: none"> <li>• Nasal obstruction and/or discoloured discharge, facial pain and loss of smell.</li> <li>• Minimum 12 weeks of duration.</li> </ul>	<ul style="list-style-type: none"> <li>• Symptoms triggered by various triggers like exercise, cold, pollution, chlorine, drugs</li> <li>• No systemic signs of allergen sensitization.</li> </ul>	<ul style="list-style-type: none"> <li>• Symptom variation follows the allergy exposure</li> <li>• +/- triggers like exercise</li> <li>• Involvement of other sites, like skin and eyes</li> </ul>	<ul style="list-style-type: none"> <li>• Acute</li> <li>• Double sickening **, fever &gt;38, pain, unilateral disease, severe pain, raised CRP</li> <li>• Be aware of alarm symptoms for severe infections</li> </ul>
	<b>Special investigations to confirm the diagnosis</b>	<ul style="list-style-type: none"> <li>• Nasal endoscopy</li> <li>• CT scan</li> </ul>	<ul style="list-style-type: none"> <li>• Nasal endoscopy</li> <li>• CT scan</li> </ul>	<ul style="list-style-type: none"> <li>• Nasal endoscopy</li> <li>• Negative allergy test</li> <li>• No pathogen detected</li> </ul>	<ul style="list-style-type: none"> <li>• Nasal endoscopy</li> <li>• Negative allergy test</li> <li>• No pathogen detected</li> </ul>	<ul style="list-style-type: none"> <li>• Nasal endoscopy</li> <li>• History confirmed by skin prick test and/or by specific IgE antibodies</li> </ul>	<ul style="list-style-type: none"> <li>• Nasal endoscopy</li> <li>• Pathogen detection, C-reactive protein</li> </ul>
<b>Principles of management</b>		<ul style="list-style-type: none"> <li>• Nasal rinse**</li> <li>• Pharmacotherapy</li> <li>• Surgery</li> </ul>	<ul style="list-style-type: none"> <li>• Nasal rinse**</li> <li>• Nasal strips</li> <li>• Surgery</li> </ul>	<ul style="list-style-type: none"> <li>• Removal of triggers</li> <li>• Environmental control</li> <li>• Nasal rinse** (See Figure 6)</li> </ul>	<ul style="list-style-type: none"> <li>• Removal of triggers,</li> <li>• Environmental control</li> <li>• Nasal rinse** (See Figure 5)</li> </ul>	<ul style="list-style-type: none"> <li>• Removal of triggers</li> <li>• environmental control</li> <li>• Nasal rinse**.</li> <li>• Pharmacotherapy (See Figure 4)</li> </ul>	<ul style="list-style-type: none"> <li>• Self-Care</li> <li>• Nasal rinse**</li> <li>• Pharmacotherapy</li> <li>• Only use antibiotic if <math>\geq 3</math> additional symptoms (See Figure 3)</li> </ul>

<b>Prevention</b>	<ul style="list-style-type: none"> <li>• Immediate intervention after trauma</li> <li>• Avoid secondary trauma</li> </ul>	<ul style="list-style-type: none"> <li>• Avoid induced obstruction</li> </ul>	<ul style="list-style-type: none"> <li>• Avoidance of triggers</li> <li>• Nasal rinse**</li> </ul>	<ul style="list-style-type: none"> <li>• Avoidance of triggers</li> <li>• Nasal rinse**</li> </ul>	<ul style="list-style-type: none"> <li>• Avoidance of triggers</li> <li>• Nasal rinse**</li> </ul>	<ul style="list-style-type: none"> <li>• Hand hygiene, distance</li> <li>• Avoidance of contagious environments</li> <li>• Increase rest and sleep</li> </ul>
<b>Indication for referral to secondary health care</b>	<ul style="list-style-type: none"> <li>• If nasal endoscopy, CT scan or objective testing is abnormal</li> </ul>	<ul style="list-style-type: none"> <li>• When nasal valve collapse causes impaired breathing</li> </ul>	<ul style="list-style-type: none"> <li>• No response to first line treatment</li> </ul>	<ul style="list-style-type: none"> <li>• No response to first line treatment</li> </ul>	<ul style="list-style-type: none"> <li>• No response to first line treatment</li> <li>• When immunotherapy is considered</li> </ul>	<ul style="list-style-type: none"> <li>• Alarm symptoms when <math>\geq 3</math> episodes per year</li> </ul>

\* See Supplemental file 1 for symptoms and risks. \*\*For nasal rinse, see Figure 7.

\*\* Double sickening – This indicates that a patient has a history of initial improvement followed by a worsening of symptoms, between days 5 and 10 of the illness and this is consistent with acute bacterial rhinosinusitis<sup>77</sup>.

**Conflict of Interest:**

The authors report no conflict of interest pertaining to this manuscript.

**Funding:**

IOC Research Centre (South Africa) (partial funding)

**Author contributorship:**

All authors read and approved the final version of the manuscript

**Author ORCID iD's:**

Cameron McIntosh: 0000-0001-9448-754X

Hege Clemm: 0000-0002-0096-4032

Harald Hrubos-Strøm: 0000-0003-0065-0145

Nicola Sewry: 0000-0003-1022-4780

Martin Schwellnus: 0000-0003-3647-0429

**References**

1. Hull JH, Jackson AR, Ranson C, Brown F, Wootten M, Loosemore M. The benefits of a systematic assessment of respiratory health in illness susceptible athletes. *Euro Respir J*. 2020.
2. Derman W, Runciman P, Jordaan E, et al. Incidence rate and burden of illness at the Pyeongchang 2018 Paralympic Winter Games. *Br J Sports Med*. 2019;53(17):1099-1104.



3. Soligard T, Steffen K, Palmer D, et al. Sports injury and illness incidence in the Rio de Janeiro 2016 Olympic Summer Games: A prospective study of 11274 athletes from 207 countries. *Br J Sports Med.* 2017;51(17):1265-1271.
4. Engebretsen L, Soligard T, Steffen K, et al. Sports injuries and illnesses during the London Summer Olympic Games 2012. *Br J Sports Med.* 2013;47(7):407-414.
5. Walker AC, Surda P, Rossiter M, Little SA. Nasal disease and quality of life in athletes. *J Laryngol Otol.* 2018;132(9):812-815.
6. Surda P, Putala M, Siarnik P, Walker A, Bernic A, Fokkens W. Rhinitis and its impact on quality of life in swimmers. *Allerg.* 2018;73(5):1022-1031.
7. Becker DG, Ransom E, Guy C, Bloom J. Surgical treatment of nasal obstruction in rhinoplasty. *Aesthet Surg J.* 2010;30(3):347-378; quiz 379-380.
8. Bougault V, Turmel J, Boulet LP. Effect of intense swimming training on rhinitis in high-level competitive swimmers. *Clin Experiment Allerg.* 2010;40(8):1238-1246.
9. Kurowski M, Jurczyk J, Kryzstofiak H, Kowalski ML. Exercise-induced respiratory symptoms and allergy in elite athletes: Allergy and Asthma in Polish Olympic Athletes (A(2)POLO) project within GA(2)LEN initiative. *Clin Respir J.* 2016;10(2):231-238.
10. Surda P, Walker A, Putala M, Siarnik P. Prevalence of rhinitis in athletes: systematic review. *Int J Otolaryngol.* 2017;2017:1-5.
11. Hox V, Beyaert S, Bullens D, et al. Tackling nasal symptoms in athletes: Moving towards personalized medicine. *Allerg.* 2021.
12. Jones N. The nose and paranasal sinuses physiology and anatomy. *Adv Drug Deliv Rev.* 2001;51(1-3):5-19.
13. Brandtzaeg P. Immunology of tonsils and adenoids: everything the ENT surgeon needs to know. *Int J Pediat Otorhinolaryngol.* 2003;67 Suppl 1:S69-76.

14. Selimoglu E. Nitric oxide in health and disease from the point of view of the otorhinolaryngologist. *Curr Pharmac Des.* 2005;11(23):3051-3060.
15. Shturman-Ellstein R, Zeballos RJ, Buckley JM, Souhrada JF. The beneficial effect of nasal breathing on exercise-induced bronchoconstriction. *Am Rev Respir Dis.* 1978;118(1):65-73.
16. Araujo BCL, de Magalhaes Simoes S, de Gois-Santos VT, Martins-Filho PRS. Association between mouth breathing and asthma: a systematic review and meta-analysis. *Curr Allerg Asth Rep.* 2020;20(7):24.
17. Kahana-Zweig R, Geva-Sagiv M, Weissbrod A, Secundo L, Soroker N, Sobel N. Measuring and characterizing the human nasal cycle. *PloS one.* 2016;11(10):e0162918.
18. Rohrmeier C, Schittek S, Ettl T, Herzog M, Kuehnel TS. The nasal cycle during wakefulness and sleep and its relation to body position. *Laryngoscop.* 2014;124(6):1492-1497.
19. Niinimaa V, Cole P, Mintz S, Shephard RJ. The switching point from nasal to oronasal breathing. *Respir Physiol.* 1980;42(1):61-71.
20. Surda P, Walker A, Limpens J, Fokkens W, Putala M. Nasal changes associated with exercise in athletes: systematic review. *J Laryngol Otol.* 2018;132(3):191-197.
21. Saketkhou K, Kaplan I, Sackner MA. Effect of exercise on nasal mucous velocity and nasal airflow resistance in normal subjects. *J Appl Physiol.* 1979;46(2):369-371.
22. Meir R, Zhao GG, Zhou S, Beavers R, Davie A. The acute effect of mouth only breathing on time to completion, heart rate, rate of perceived exertion, blood lactate, and ventilatory measures during a high-intensity shuttle run sequence. *J Streng Cond Res.* 2014;28(4):950-957.

23. Overend T, Barrios J, McCutcheon B, Sidon J. External nasal dilator strips do not affect treadmill performance in subjects wearing mouthguards. *J Athl Train*. 2000;35(1):60-64.
24. Hostetter K MS, Cox DG, Dallam G. Triathlete adapts to breathing restricted to the nasal passage without loss in VO<sub>2</sub>max or VVO<sub>2</sub>max. *J Sport Human Perf*. 2016;4:1-7.
25. Recinto C ET, Boffelli PT, Navalata JW. Effects of nasal or oral breathing on anaerobic power output and metabolic responses. *Int J Exerc Sci*. 2017;10(4):506-514.
26. LaComb CO TR, Lee SP, Young JC, Navalta JW. Oral versus Nasal Breathing during moderate to high intensity submaximal aerobic exercise. *Int J Kinesiol Sports Sci*. 2017;5(1):8-16.
27. Niinimaa V. Oronasal airway choice during running. *Respir Physiol*. 1983;53(1):129-133.
28. Liu J, Zhang X, Zhao Y, Wang Y. The association between allergic rhinitis and sleep: A systematic review and meta-analysis of observational studies. *PloS one*. 2020;15(2):e0228533.
29. Mangla PK, Menon MP. Effect of nasal and oral breathing on exercise-induced asthma. *Clin Allerg*. 1981;11(5):433-439.
30. Togias A. Rhinitis and asthma: evidence for respiratory system integration. *J Allerg Clin Immunol*. 2003;111(6):1171-1183; quiz 1184.
31. Apaydin F. Nasal valve surgery. *Facial Plast Surg*. 2011;27(2):179-191.
32. Walker A, Surda P, Rossiter M, Little S. Rhinitis in elite and non-elite field hockey players. *Int J Sports Med*. 2017;38(1):65-70.
33. Fokkens WJ, Lund VJ, Hopkins C, et al. European position paper on rhinosinusitis and nasal polyps 2020. *Rhinol J*. 2020;0(0):1-464.

34. Hellings PW, Klimek L, Cingi C, et al. Non-allergic rhinitis: Position paper of the European Academy of Allergy and Clinical Immunology. *Allergy*. 2017;72(11):1657-1665.
35. Bauchau V, Durham SR. Prevalence and rate of diagnosis of allergic rhinitis in Europe. *Euro Respir J*. 2004;24(5):758-764.
36. Jones NS, Smith PA, Carney AS, Davis A. The prevalence of allergic rhinitis and nasal symptoms in Nottingham. *Clin Otolaryngol Allied Sci*. 1998;23(6):547-554.
37. Gelardi M, Ventura MT, Fiorella R, et al. Allergic and non-allergic rhinitis in swimmers: clinical and cytological aspects. *Br J Sports Med*. 2012;46(1):54-58.
38. Steelant B, Hox V, Van Gerven L, et al. Nasal symptoms, epithelial injury and neurogenic inflammation in elite swimmers. *Rhinol J*. 2018;56(3):279-287.
39. Stewart MG, Witsell DL, Smith TL, Weaver EM, Yueh B, Hannley MT. Development and validation of the Nasal Obstruction Symptom Evaluation (NOSE) scale. *Otolaryngol Head Neck Surg*. 2004;130(2):157-163.
40. Piccirillo JF, Merritt MG, Jr., Richards ML. Psychometric and clinimetric validity of the 20-Item Sino-Nasal Outcome Test (SNOT-20). *Otolaryngol Head Neck Surg*. 2002;126(1):41-47.
41. Hopkins C, Gillett S, Slack R, Lund VJ, Browne JP. Psychometric validity of the 22-item Sinonasal Outcome Test. *Clin Otolaryngol*. 2009;34(5):447-454.
42. Bonini M, Braido F, Baiardini I, et al. AQUA: Allergy questionnaire for athletes. development and validation. *Med Sci Sports Exer*. 2009;41(5):1034-1041.
43. Subcommittee A, American College of Surgeons' Committee on T, International Awg. Advanced trauma life support (ATLS(R)): the ninth edition. *J Trauma Acute Care Surg*. 2013;74(5):1363-1366.

44. Lennon P, Jaber S, Fenton JE. Functional and psychological impact of nasal bone fractures sustained during sports activities: A survey of 87 patients. *Ear Nose Throat J.* 2016;95(8):324-332.
45. Kasperbauer JL, Kern EB. Nasal valve physiology. Implications in nasal surgery. *Otolaryngol Clinics N Am.* 1987;20(4):699-719.
46. van Egmond M, Rovers MM, Tillema AHJ, van Neerbeek N. Septoplasty for nasal obstruction due to a deviated nasal septum in adults: a systematic review. *Rhinol.* 2018;56(3):195-208.
47. Most SP. Analysis of outcomes after functional rhinoplasty using a disease-specific quality-of-life instrument. *Arch Facial Plast Surg.* 2006;8(5):306-309.
48. Spielmann PM, White PS, Hussain SS. Surgical techniques for the treatment of nasal valve collapse: a systematic review. *Laryngoscope.* 2009;119(7):1281-1290.
49. Lekakis G, Dekimpe E, Steelant B, Hellings PW. Managing nasal valve compromise patients with nasal dilators: objective vs. subjective parameters. *Rhinol.* 2016;54(4):348-354.
50. Dinardi RR, de Andrade CR, Ibiapina Cda C. External nasal dilators: definition, background, and current uses. *Int J Gen Med.* 2014;7:491-504.
51. Gelardi M, Porro G, Accettura D, Quaranta VN, Quaranta N, Ciprandi G. The role of an internal nasal dilator in athletes. *Acta Biomed.* 2019;90(2-S).
52. Fischer H, Gubisch W. Nasal valves--importance and surgical procedures. *Facial Plast Surg.* 2006;22(4):266-280.
53. Charest J, Grandner MA. Sleep and athletic performance: impacts on physical performance, mental performance, injury risk and recovery, and mental health. *Sleep Med Clin.* 2020;15(1):41-57.

54. Weidner TG, Anderson BN, Kaminsky LA, Dick EC, Schurr T. Effect of a rhinovirus-caused upper respiratory illness on pulmonary function test and exercise responses. *Med Sci Sports Exer.* 1997;29(5):604-609.
55. Gleeson M PD. Respiratory infections and mucosal immunity in athletes. *Am J Med sports.* 2001;3:159-165.
56. Reeser JC, Willick S, Elstad M. Medical services provided at the Olympic Village polyclinic during the 2002 Salt Lake City Winter Games. *WMJ.* 2003;102(4):20-25.
57. Cox AJ, Gleeson M, Pyne DB, Callister R, Hopkins WG, Fricker PA. Clinical and laboratory evaluation of upper respiratory symptoms in elite athletes. *Clin J Sport Med.* 2008;18(5):438-445.
58. Schweltnus M, Soligard T, Alonso JM, et al. How much is too much? (Part 2) International Olympic Committee consensus statement on load in sport and risk of illness. *Br J Sports Med.* 2016;50(17):1043-1052.
59. Nieman DC, Johanssen LM, Lee JW, Arabatzis K. Infectious episodes in runners before and after the Los Angeles Marathon. *J Sports Med Phys Fitn.* 1990;30(3):316-328.
60. Besedovsky L, Lange T, Haack M. The sleep-immune crosstalk in health and disease. *Physiol Rev.* 2019;99(3):1325-1380.
61. Jaworski CA, Rygiel V. Acute illness in the athlete. *Clin Sports Med.* 2019;38(4):577-595.
62. Wheatley LM, Togias A. Allergic Rhinitis. *New Engl J Med.* 2015;372(5):456-463.
63. Klimek L, Bergmann K-C, Biedermann T, et al. Visual analogue scales (VAS): Measuring instruments for the documentation of symptoms and therapy monitoring in cases of allergic rhinitis in everyday health care. *Allergo J Int.* 2017;26(1):16-24.

64. Bousquet J, Schünemann HJ, Togias A, et al. Next-generation Allergic Rhinitis and Its Impact on Asthma (ARIA) guidelines for allergic rhinitis based on Grading of Recommendations Assessment, Development and Evaluation (GRADE) and real-world evidence. *J Allerg Clin Immunol*. 2020;145(1):70-80.e73.
65. Baraniuk JN, Merck SJ. New concepts of neural regulation in human nasal mucosa. *Acta clinica Croatica*. 2009;48(1):65-73.
66. Värendh M, Janson C, Bengtsson C, et al. Nasal symptoms increase the risk of snoring and snoring increases the risk of nasal symptoms. A longitudinal population study. *Sleep Breath*. 2021.
67. Ji K, Risoli TJ, Kuchibhatla M, Chan L, Hachem RA, Jang DW. Symptom profile of chronic rhinosinusitis versus obstructive sleep apnea in a tertiary rhinology clinic. *The Annals Otol Rhinol Laryngol*. 2019;128(10):963-969.
68. Kao LT, Hung SH, Lin HC, Liu CK, Huang HM, Wu CS. Obstructive sleep apnea and the subsequent risk of chronic rhinosinusitis: a population-based study. *Scientif Rep*. 2016;6:20786.
69. Jiang RS, Liang KL, Hsin CH, Su MC. The impact of chronic rhinosinusitis on sleep-disordered breathing. *Rhinol*. 2016;54(1):75-79.
70. Caia J, Halson SL, Scott A, Kelly VG. Obstructive sleep apnea in professional rugby league athletes: An exploratory study. *J Sci Med Sport*. 2020;23(11):1011-1015.
71. Kasasbeh A, Kasasbeh E, Krishnaswamy G. Potential mechanisms connecting asthma, esophageal reflux, and obesity/sleep apnea complex--a hypothetical review. *Sleep MedRev*. 2007;11(1):47-58.
72. Steelant B, Hox V, Hellings PW, Bullens DM, Seys SF. Exercise and sinonasal disease. *Immunol Allerg Clinics N Am*. 2018;38(2):259-269.

73. Seys SF, De Bont S, Fokkens WJ, et al. Real-life assessment of chronic rhinosinusitis patients using mobile technology: The mySinusitisCoach project by EUFOREA. *Allerg.* 2020;75(11):2867-2878.
74. Bengtsson C, Jonsson L, Holmström M, et al. Incident Chronic Rhinosinusitis Is Associated With Impaired Sleep Quality: Results of the RHINE Study. *J Clin Sleep Med.* 2019;15(06):899-905.
75. Varendh M, Andersson M, Bjornsdottir E, et al. Nocturnal nasal obstruction is frequent and reduces sleep quality in patients with obstructive sleep apnea. *J Sleep Res.* 2017.
76. Ishii L, Roxbury C, Godoy A, Ishman S, Ishii M. Does nasal surgery improve osa in patients with nasal obstruction and osa? a meta-analysis. *Otolaryngol Head Neck Surg.* 2015;153(3):326-333.
77. Aring AM, Chan MM. Current concepts in adult acute rhinosinusitis. *Am Fam Physic.* 2016;94(2):97-105.