

Return to Play in Asthma and Pulmonary Conditions

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57.1 Introduction

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Football (soccer) is a sport of tremendous physical demand. The dynamic nature of the sport places great stress on an individual athlete. Those sports-specific demands not only involve the musculoskeletal system but also place a lot of stress on the cardiopulmonary system. In this chapter, we will address the effect of asthma and other pulmonary conditions in the football athlete.

57.1.1 Introduction: Physiologic Demands

The physiologic and strength demands are high, with METs (metabolic equivalents) being in the 5–12 range [1]. The oscillation of low-intensity

and high-intensity varies throughout a football match. The body must be able to meet the metabolic needs during those variances.

There are many pathologic conditions that may affect an athlete's ability to inspire, exchange gases, and expire. One of those conditions is asthma.

57.1.2 Introduction: Potential Conditions

Asthma in sports tends to break down into two main categories: exercise-induced asthma (EIA) and exercise-induced bronchospasm (EIB). EIA is exercise causing bronchial obstruction in patients with clinical asthma, while EIB is an airway obstruction in association with exercise without a clinical diagnosis of asthma [2]. Other topics of discussion will include pneumothorax and pulmonary infections. Finally, this chapter will try to address the current dogma and recommendations on return to play in each of those conditions.

57.2 Asthma

Asthma is a chronic pulmonary condition that can negatively affect athletes to varying degrees with regard to airway inflammation, hyperresponsiveness, and reversible obstruction. Asthma affects approximately 25 million people in the United States, 7 million of which are children [3]. The prevalence of asthma in athletes overall is actually higher than the general population ranging from 23% to 55%, with higher rates noted in endurance athletes [4].

57.2.1 Asthma: Pathophysiology

In asthmatics there is a chronic underlying inflammatory state in the lungs with increased levels of cytokines and inflammatory molecules within the epithelium of the bronchial tree. When exposed to certain triggers, such as pollen, mold, allergens, cold air, or exercise, these inflamma-

tory markers induce a cascade of molecular reactions that result in increased mucus production, swelling of the bronchial epithelium, and bronchial muscle tightening.

57.2.2 Asthma: Symptoms and Presentation

The previously discussed changes in asthma lead to airway narrowing and obstruction. Athletes will present with symptoms including cough, wheezing, shortness of breath, or chest tightness—in severe cases, these symptoms can lead to a life-threatening state of hypoxia due to obstruction of the airways. The diagnosis of asthma can typically be made by taking a thorough history with athletes that have had episodes of reversible symptoms. These episodes are typically associated with certain triggers that may also be identified with skilled history taking. Lung function testing with spirometry can be used to diagnose the condition with objective evidence.

57.2.3 Asthma: Diagnosis

Spirometry is a functional test used to diagnose asthma. This test can objectively measure the forced expiratory volume in 1 second (FEV₁) as the athlete forcibly exhales, and if that value is <80% predicted and the FEV₁/forced vital capacity (FVC) ratio is <70%, then asthma is the diagnosis as long as there is reversibility by a FEV₁ increase of >12% from baseline or \geq 10% of the predicted FEV₁ after the use of a short-acting beta-agonist inhaler such as albuterol [5]. Based on history and spirometry results, the diagnosis of asthma can be classified into four separate categories that will guide treatment depending on the severity of symptoms as shown in Table 57.1. The diagnosis of asthma can also be made after a 4-6-week trial of controller medication with reassessment to determine if the symptoms have improved or resolved [3].

Intermittent Mild persistent Moderate persistent Severe persistent Symptoms ≤2 days/week >2 days/week Once daily Throughout the day Nighttime >Once/week Often 7 days/week ≤2 events/month 3–4 events/month awakenings Use of SABA ≤2 days/week >2 days/week and not Daily Several uses daily > once per day Treatment Low-dose ICS with Medium-dose ICS High-dose ICS with SABA as needed for SABA as needed with LABA LABA and possible symptoms oral corticosteroid

Table 57.1 Asthma classification and treatment

ICS inhaled corticosteroid, SABA short-acting beta-agonist, LABA long-acting beta-agonist

Fact Box 1 Spirometry Diagnostic Values

 $FEV_1 < 80\%$

FEV₁/FVC < 70%

Reversibility of values with inhaled SABA:

 $FEV_1 > 12\%$

Predicted FEV₁ $\geq 10\%$

57.2.4 Asthma: Treatment

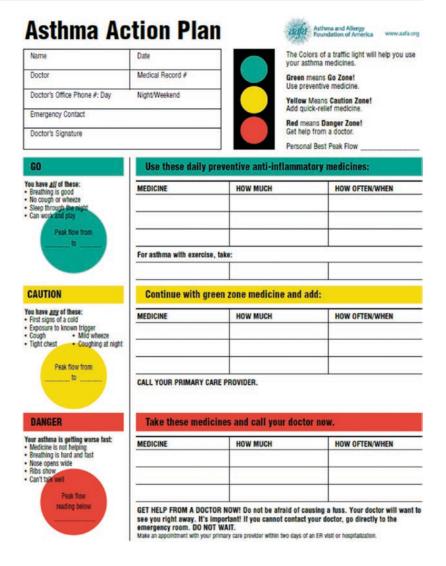
When it comes to treatment of asthma in footballers, it is important to verify which medications may be prohibited based on the World Anti-Doping Agency. This agency bans all inhaled beta-agonists, except salmeterol, salbutamol (1600 µg maximum over 24 h), and formoterol (54 µg maximum over 24 h) [6]. However one recent study did show that inhaled salbutamol up to 1600 µg did not improve lung exercise performance [7]. The Medical Commission of the International Olympic Committee imposed its ban on inhaled beta-agonists in 1993 due to the growing concern of increased use among elite athletes in order to help improve oxygen uptake and thus lead to an unfair advantage in endurance, speed, and strength. Other organizations have imposed their bans as well, and it is important to be aware of sport governing bodies when it comes to treating asthma and bronchospasm.

Along with the pharmacological treatment of asthma, education and prevention of symptoms is also a key component to maintaining adequate control of the athlete's symptoms. The athlete should attempt to identify triggers for their symptoms,

which may vary from seasonal allergies, like pollen to dust mites or even weather changes. For example, cold weather changes can be correlated to the development of asthma symptoms. An individual must demonstrate vigilance to keep their symptoms under control by altering environmental exposures and utilizing controller medication. These environmental changes may include changing practice locations based on the weather, using a humidifier or anti-allergen air filters at home, or even avoiding the use of NSAIDs, as they may predispose athletes to asthma flares [8]. Also, as part of the education process, athletes should be reminded of their asthma action plan that provides them with a step-by-step process of what to do when their symptoms start to become uncontrolled (Fig. 57.1).

The athlete and medical provider should be aware of when the last asthma exacerbation took place, if the athlete was ever hospitalized, and if they ever had to have a breathing tube placed (intubation) due to severe symptoms. Peak flows may also be utilized to monitor asthma symptom severity; however, this may be unfeasible or cumbersome when it comes to its proper and judicious use among the athletic population. Other modifying factors include increasing endurance training as studies have shown that endurance athletes tend to have higher lung volumes by way of strengthening respiratory muscles, reducing resistance through bronchial canals, and increasing lung elasticity and alveolar expansion in order to adapt to the demands brought forth by endurance training [9]. This training may theoretically help reduce incidences of asthma exacerbation and symptoms that can prevent footballers from participating at their highest ability.

Fig. 57.1 Asthma action plan from AAFA



57.2.5 Asthma: Return to Play

It is generally acceptable to allow an athlete to return to sport once his or her lung function has returned to baseline and is able to participate in all sport-related activities without adverse pulmonary symptoms. Medications have demonstrated quicker improvement in symptoms and may allow for a sooner return to play timeline.

57.3 Exercise-Induced Bronchoconstriction

Exercise-induced bronchoconstriction (EIB) is a pulmonary condition characterized by transient reversible airway narrowing that increases respiratory resistance resulting in coughing, shortness of breath, wheezing, or chest tightness shortly after vigorous exercise. EIB is present in 7–20% of the general population. However, its prevalence in athletes has been found to be much higher with one study finding up to 50% depending on the sport and environmental conditions [10].

57.3.1 Exercise-Induced Bronchoconstriction: Symptoms and Presentation

EIB is a condition outside that of asthma, as these patients have no symptoms while at rest. Symptoms will typically present after

5–10 minutes of vigorous exercise and may last up to 20–30 minutes after exercise has concluded [8]. The key factor is that symptoms are reversible and do not occur at rest. Symptoms may also be influenced by other factors including weather, allergens, and intermittent bursts of exercise intensity during match play [11].

57.3.2 Exercise-Induced Bronchoconstriction: Pathophysiology

The etiology of EIB is centered on the idea of having to warm and condition air that is normally done so by the upper airway prior to the cool air reaching the lower airway and bronchial tree. While at rest, the upper airway's ability to warm the air is adequate secondary to the respiratory rate being slow enough to complete the task. However, when athletes exert themselves and the respiratory rate increases, this allows for the cool environmental air to reach the lower airways, thus giving the distal bronchial tree the task of warming and conditioning the air. It is this alteration in physiologic role that may lead to changes in an individual's symptoms.

There are two main theories as to the pathophysiology of EIB, thermal expenditure and osmotic. The thermal expenditure theory states that the airway cooling from the increased respiratory rate will cause vasoconstriction surrounding the lower airway bronchial tree and that upon the rewarming of the airway, the surrounding vasculature will dilate to engorge and rewarm the bronchial epithelium and leads to vascular leakage and airway edema [10]. The osmotic theory focuses on the water loss mechanisms that the bronchial epithelium and submucosa utilize in an attempt to warm the cool air that reaches the lower airways, which then causes changes in the epithelial pH and creates a hyperosmolar state that then leads to a cascade of molecular changes that release immune-modulators and mediators instigating bronchial constriction [10]. Though no one theory is definitive, it is likely a combination of the two that leads to the manifestation of EIB.

57.3.3 Exercise-Induced Bronchoconstriction: Diagnosis

The diagnosis of EIB is typically made through history and physical exam; however, it must be noted that this can lead to either overdiagnosis or underdiagnosis of the condition given the vast variance of symptom severity and presentation as well as a refractory period that some individuals may have. The refractory period is the time following spontaneous resolution of EIB symptoms where athletes will not experience any further symptoms for the next 1-2 hours, however, may have return of symptoms following this grace period [6]. This period may mask the diagnosis as athletes may not report the issue to their medical staff but then have recurrence of symptoms afterward. To properly diagnose the condition, pulmonary function tests (PFTs) should be conducted while at rest to rule in or out any underlying chronic asthma. Following this, other pathologies should be ruled out including vocal cord dysfunction, gastroesophageal reflux, and cardiac abnormalities.

The diagnosis of EIB can be made with the use of an exercise challenge test, eucapnic voluntary hyperventilation test (EVH), hyperosmolar saline challenge test, mannitol challenge test, or a direct challenge test with the use of methacholine to induce bronchoconstriction. The most commonly used tests are the exercise challenge test and the EVH, which focus on the FEV1 and its decline from baseline following provocation with either exercise or voluntary hyperventilation measured at various time limits following introduction of the stimulus. A decline in FEV1 of >10% in EVH or peak expiratory flow rate (PEFR) of 15% or greater indicates positive results with these tests [8, 11].

57.3.4 Exercise-Induced Bronchoconstriction: Treatment

When it comes to the treatment of EIB, it is important to look at all modifiable factors as well as pharmacologic options to maximize a football player's pulmonary capabilities. The use of gaiters, scarves, or masks can theoretically limit the amount of cool air that reaches the lower respiratory tree, thus limiting the effects of bronchoconstriction. Short warm-ups 15–20 min prior to activity at 80–90% of maximum exertion, calisthenics, and proper cooldown post-workouts have also been shown to limit the EIB response with athletes [4].

Many of the same medications that treat asthma can be utilized. Two to four puffs of a short-acting beta-agonist (SABA) (Fig. 57.2), 20 min prior to exercise or a match, can help control the onset of symptoms, if exercise is expected to last no more than 6 h. If there is a tournament or the player is expected to exert themselves for a longer period of time, then a long-acting beta-agonist (LABA) can be considered as their effects can last up to 12 h after administration. Leukotriene antagonists and cromolyn can also be utilized for those that cannot tolerate the beta-agonist inhalers; however, these may not be as effective. Inhaled corticosteroids do not play a role in the management and treatment of EIB. The use of these medications has

shown improvement of symptoms and have allowed for quicker resolution as well.

57.3.5 Exercise-Induced Bronchoconstriction: Return to Play

A football player experiencing symptoms of EIB should be removed from play and evaluated properly on the sideline. If a baseline PEFR is known, then a repeat at the time of symptom onset should be conducted to assist in the evaluation of symptom severity [9]. If the PEFR is below 15% from baseline, then two puffs of a SABA may be utilized. If symptoms are still present after 5 min, then two more puffs may be administered [9]. Serial PEFR readings can be conducted until the value returns to normal limits or back to baseline. If values do not return to normal or baseline, then the athlete should be evaluated more properly off the sideline in case further treatment modalities are indicated, including emergency options as the condition could progress a life-threatening state such as status



Fig. 57.2 Athlete demonstrating proper inhaler technique with spacer

asthmaticus [9]. Once the athlete's respiratory function has improved and he or she is no longer symptomatic, then he or she may return to the playing field.

57.4 Pneumothorax

A pneumothorax (PTX) is a rare but potentially life-threatening pulmonary condition that requires prompt recognition and expedited treatment and care. The condition is defined as a collection of air within the pleural space between the chest wall and the lung. Due to the intrinsic intrathoracic negative pressure created by inspiration, the accumulation of this air can eventually reach enough pressure to induce collapse of the lung and potentially lead to cardiopulmonary compromise. Only 2% of all adult pneumothoraces are associated with sports and can be spontaneous or tension-related [12].

Spontaneous PTX is found more often in tall, thin, and young individuals. It will occur, as its name suggests, spontaneously from primary or secondary etiology, secondary being in the setting of underlying pulmonary disease including pneumonia, asthma, cystic fibrosis, or interstitial lung disease. Tension PTX is much less common and typically occurs due to blunt or penetrating trauma from a fracture rib that disrupts the pleura. Particular to football, blunt trauma causing tension PTX is typically the result of thoracic collisions at high speed; athletes are more prone to this type of injury during keeper attacks or going for headers.

57.4.1 Pneumothorax: Symptoms and Presentation

Patients can present with difficulty in breathing, pleuritic chest pain with inspiration, rapid respiratory rates, increased heart rate, or even a presence of anxiety. These symptoms will most often develop in a progressive nature, which highlights the importance of serial exams. Associated

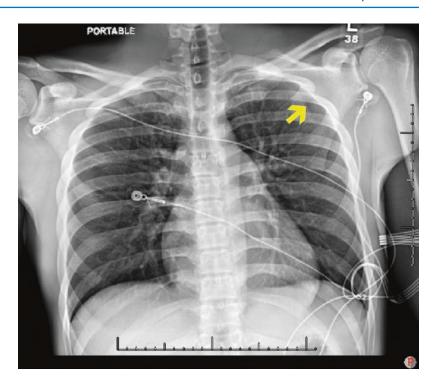
pathology must be excluded including internal organ injury following blunt trauma as well, including pulmonary contusion, hemothorax, pneumomediastinum, splenic rupture, kidney laceration, rib fracture, or other internal derangement. Examination with a focus on primary assessment that includes airway, breathing, circulation, and frequent vital sign checks is crucial to obtaining rapid treatment in order to avoid progressive lung collapse or potential cardiovascular compromise.

57.4.2 Pneumothorax: Diagnosis

The physical exam plays an essential role in determining the potential concern for a pneumothorax. The pulmonary exam may include diminished breath sounds, rales, hyperresonance on percussion, hypoxia, hypotension, or even tracheal deviation away from the affected side. It is also imperative to assess for thoracic cage injury including rib fractures and potential cardiac involvement with a full cardiovascular exam.

Imaging studies are also useful in making the diagnosis. Chest X-ray (CXR) (Fig. 57.3) and ultrasound are good initial studies; however, helical CT imaging could also be utilized once the patient is stabilized in order to assess for any other associated pathology such as pulmonary contusion, laceration, and rib fractures. Once the diagnosis is even suspected, the transport of the athlete to a nearby emergency department for higher-level care, evaluation, and treatment is necessary if there is concern for respiratory compromise and overall decompensation. Football players will typically have high health reserve that may allow them to compensate for a period of time before becoming limited by PTX symptoms; therefore, a high index of suspicion is required with serial physical exams in order to make the diagnosis. Following blunt trauma that is limiting the player, he or she should be evaluated thoroughly as a missed diagnosis of PTX could result in serious complications.

Fig. 57.3 Left-sided pneumothorax after blunt trauma with *yellow arrow* pointing to visceral pleural line



57.4.3 Pneumothorax: Treatment

Treatment for an uncomplicated pneumothorax may be observational depending on size. An example of this would be a non-tension pneumothorax <10%. However, if a tension pneumothorax is suspected, supplemental oxygen and needle decompression with either a 14- or 16-G needle can be performed. The needle is inserted into the second intercostal space in the midclavicular line to relieve the pressure, which may be followed by a rush of air [4, 13]. Following this procedure, the placement of a chest tube to allow for lung re-expansion is needed. Repeat CXR to visualize re-expansion of the lung and ensure proper chest tube placement is recommended (Fig. 57.4). This should be followed up with serial CXRs, a minimum of 2 days later, to ensure stability. If the PTX remains resolved, then the chest tube can be removed after 2-3 days of monitoring in an acute care setting.

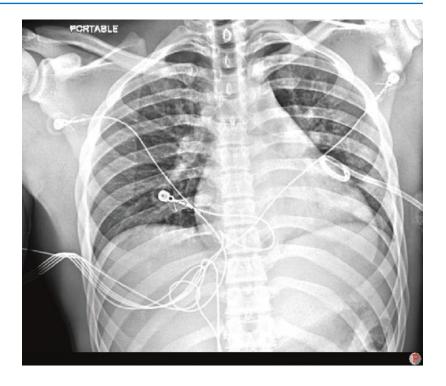
With regard to a spontaneous PTX, the American College of Chest Physicians published that a small PTX (<3 cm apex-to-cupola distance) in a hemodynamically stable patient with-

out significant symptoms may be managed by observation alone with close follow-up after the exclusion of progression with 3–6 h of observation and repeat CXR in an emergency setting; larger PTX (>3 cm) should re-expanded with decompression [13].

57.4.4 Pneumothorax: Return to Play

There are no consensus guidelines when it comes to return to play (RTP) from a resolved pneumothorax. Case reports and opinion typically cite return to play ranging anywhere from 2 to 10 weeks, with an average of 3–4 weeks prior to RTP [13]. Air travel should be avoided in the first 1–3 weeks following a pneumothorax as the change in air pressure may result in hypoxemia and gas expansion in a closed parenchymal space. Recommends may differ if an athlete has associated pathology like multiple rib fracture or flail chest. During this time, repeat CXR should be obtained to ensure continued resolution of the PTX, and treatment with a pulmonary toilet including beta-agonists, mucolytics, and cough

Fig. 57.4 Left-sided pneumothorax with re-expanded lung after chest tube placement



suppressants may be utilized for symptom control. A chest wall protector may also be implemented for extra protection.

Close follow-up should also be implemented to ensure that the development of acute respiratory distress syndrome (ARDS) does not occur. All in all, a slow return to physical activity with using pain as a guide is typically the mainstay of return to play from this pulmonary condition.

57.5 Respiratory Infections

Respiratory infections and their associated symptoms are a very common complaint among the general population as well as the athletic population. Symptoms can present in a variety of ways and in different orders. The most common symptoms are nasal congestion, sore throat, cough, postnasal drip, headache, fatigue, nausea, and fever. Although the symptoms may be similar, it is important to keep a broad differential when determining the etiology, as many ailments could present with those symptoms.

57.5.1 Respiratory Infections: Types of Infection

Types of infection include, among others, viral upper respiratory infection (URI), bacterial or viral pharyngitis, mononucleosis, viral or bacterial sinusitis, peritonsillar abscess, lower respiratory infection (LRI), bronchitis, and/or pneumonia. The athlete diagnosis is made mostly through history and physical exam; however, at times, it may be necessary to rule out other potential causes if red flags exist, and, thus, lab work including a CBC, CMP, EBV titers, or even a CXR may be needed to make the correct diagnosis.

57.5.2 Respiratory Infections: Treatment

Once the etiology of the symptoms is determined, the proper course of treatment can then be initiated. Viral URIs can be treated symptomatically with decongestants, antihistamines, or other over-the-counter medications as indicated based on symptoms. Vitamin C and zinc may also play a role in symptom duration as well. Most viral-induced URIs typically will resolve after 4–5 days. If the etiology for the athlete's symptoms is determined to be bacterial in origin, then the proper antibiotic is indicated with special attention paid to the most likely bacterial culprit, age, medical allergies, or other specific issues that may alter the antibiotic selection (Table 57.2). It is also important to monitor the athlete's hydration status and ensure that they are obtaining the proper nutrient and caloric intake during their illness.

57.5.3 Respiratory Infections: Return to Play

Return to sports decisions should be made in conjunction with close follow-up, as reevaluation and examination may be necessary. One special consideration to be mindful of when it comes to return to play is the presence of a fever greater than 101 °F. If an athlete engages in sports with a fever greater than 101 °F, then he or she does have an increased risk of developing myocarditis, a condition that affects cardiac function and output, and should be held from practice until the fever resolves without the use of antipyretic for 24 h. Also, it is important to ensure that the

symptoms are not due to mononucleosis and EBV, as this ailment could lead to splenic enlargement and prohibit a footballer from playing for at least 3 weeks from symptom onset.

There are no clear "return to play" guidelines when it comes to URI or LRI; however, the "above the neck" rule is typically implemented for most athletes, including those in football. This rule refers to that if the symptoms of sore throat, nasal congestion, and others are mainly "above the neck" without other systemic involvement, then the athlete may continue with training uninterrupted. If the symptoms are present "below the neck" or include systemic symptoms such as fever or myalgia, then it may be necessary for the athlete to rest until they improve. Return to play guidelines with pneumonia are limited as well, but typically the athlete should rest for 10-14 days as needed. However, this timeline is variable and depends on the individual athlete and his or her ability to safely play [6].

Fact Box 2 Return to play considerations with URI

Resolution of fever (oral temp. <101 °F)
Euvolemic hydration status
Respiratory capacity to athletically perform
No diagnosis of mononucleosis

Table 57.2 Common respiratory infections and treatment

	Common pathogens	Treatment
Bacterial pneumonia	S. pneumoniae, M. pneumoniae, H. influenzae	Azithromycin, clarithromycin, doxycycline
Bacterial sinusitis	S. pneumoniae, H. influenzae, M. catarrhalis	Augmentin, doxycycline
Bacterial pharyngitis	Group A streptococcus	Penicillin G IM, amoxicillin
Peritonsillar abscess	Group A streptococcus, S. aureus, MRSA	Augmentin, clindamycin, drainage
Influenza	Influenza A or B	Tamiflu if within 24–48 h of symptoms onset
Viral etiologies	RSV, rhinovirus, adenovirus, coronavirus, parainfluenza	Antihistamine, decongestant, nasal spray, expectorants, zinc, vitamin C

Conclusions

Take-Home Message: Summary

Pulmonary conditions in football athletes present a diverse spectrum of challenges to a sports medicine team. To adequately diagnose and appropriately manage, a strong understanding of each pathologic entity is necessary. A thorough clinical history accompanied by a systematic physical exam will frequently establish the diagnosis. With respect to non-emergent scenarios, peak flow measurements, environment exposure management, pulmonary conditioning, pharmacological management, and action plans are useful modalities. In emergent scenarios, diligent attention should be placed on assessment of airway, breathing, and circulation as well as prompt diagnosis. It is stressed that return to play criteria should be determined on case-by-case bases and that recommendations may vary depending on an athlete's individual circumstances.

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