

Lower Respiratory Infections and Potential Complications in Athletes

Todd M. Lorenc, MD, and Michael T. Kernan, MD

Corresponding author

Michael T. Kernan, MD
St. Joseph's Hospital Health Center, 301 Prospect Avenue,
Syracuse, NY 13203, USA.
E-mail: kernam@pol.net

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A review and synthesis of recently published literature regarding lower respiratory infections in athletes was conducted. Diagnosis and treatment of common etiologies, specifically acute bronchitis, pneumonia, and influenza, are examined and discussed. In addition, potential complications, including spontaneous pneumothorax, bronchiectasis, hemoptysis, and acute respiratory failure that may result from inadequate diagnosis and treatment, are addressed. Criteria for allowing athletes to return to play were reviewed and current accepted guidelines for return to activity are discussed.

Introduction

Despite the fact that athletes are at an elevated level of health when compared with the general population, they are still prone to respiratory infections as a common cause of short-term fatigue and weakness resulting in inability to compete. If not addressed and treated properly, these infections can progress into life-threatening conditions. In addition, given the close person to person contact present in many sports, as well as shared use of training equipment and supplies by teammates, increased physical demand, and relative deprivation of sleep involved in training, athletes are subject to increased pathogen exposure and physical stress elevating the potential for subsequent illness. This article discusses the common lower respiratory illnesses occurring in athletes, as well as current practices in diagnosis and treatment. In addition, potential complications that may arise are addressed, along with recommendations concerning return to play criteria.

Lower Respiratory Infections

Acute bronchitis

Acute bronchitis is often defined as an inflammation of the trachea, bronchi, and bronchioles due to a respiratory tract infection or the presence of chemical irritants. It is the most common lower respiratory ailment in adults and the ninth most common reason adults seek care in an ambulatory setting [1]. The cause of acute bronchitis is often viral in nature and, when a definite cause is determined, the most common culprits are influenza A and B, parainfluenza, coronavirus (types 1–3), rhinovirus, and adenovirus [2,3]. Less common documented causes include *Mycoplasma pneumoniae*, *Chlamydia pneumoniae*, the Taiwan acute respiratory strain of *C. psittaci*, and *Bordetella pertussis* infection (Table 1) [4]. More frequently occurring in the winter months, upper respiratory symptoms such as coryza, nasal congestion, malaise, myalgias, and sore throat commonly precede its occurrence. Acute bronchitis usually presents with a cough lasting less than 3 weeks as its only symptom. This cough is initially dry and nonproductive but can become progressively productive of more viscous, mucoid sputum in subsequent days. In one out of four cases, the acute bronchitis cough lasts beyond a period of 3 weeks. When the length of duration of the athlete's cough extends beyond this length of time, it is often necessary for the physician to consider additional etiologies as to the cause of the cough. Additional considerations should include postnasal drip, asthma, and gastroesophageal reflux disease. Patients will sometimes complain of a burning substernal chest pain aggravated with coughing, deep inspiration, and movement. Physical examination usually yields few specific signs for diagnosis. Occasionally, scattered rhonchi will be appreciated on auscultation with or without crackles or rales at the lung bases. In some instances, wheezing may also be present, occurring more often after the patient coughs. Patients may exhibit a slight fever or may be afebrile. These signs and symptoms, along with a history of recent upper respiratory infection, are often sufficient for diagnosis with no additional workup or testing required [2,3]. Chest films often appear normal and

Table 1. Common pathogens detected in athletes with acute bronchitis or pneumonia

Acute bronchitis
Influenza A and B
Parainfluenza
Coronavirus types
Rhinovirus
<i>Mycoplasma pneumoniae</i>
<i>Chlamydia pneumoniae</i>
<i>Bordetella pertussis</i>
Pneumonia
<i>M. pneumoniae</i>
Respiratory viruses
<i>Streptococcus pneumoniae</i>
<i>Chlamydia pneumoniae</i>
<i>Legionella</i> spp
<i>Haemophilus influenzae</i>

(Data from Auble et al. [30].)

findings, when present, are often minimal and similar to those seen in asthmatics (atelectasis, hyperinflation, peribronchial thickening) [5].

Treatment of athletes with acute bronchitis usually entails symptomatic treatment and temporary suspension of training/playing activity to enhance convalescence. If the patient is a smoker, cessation should be a primary goal because symptoms are often prolonged and more severe in the smoking population. Given the common viral etiology of acute bronchitis, patients will often present with common cold symptoms and receive relief through treatment with nonsteroidal anti-inflammatory drugs and/or nasal decongestants. For a chronic persistent cough, relief can often be obtained with a cough suppressant [4]. Because acute bronchitis and asthma share similar pulmonary findings, (ie, bronchial inflammatory changes) bronchodilators, specifically β -2 agonists, have been studied. They relieve coughing within 1 week when compared with placebo in patients who are given inhaled albuterol (two puffs four times daily), oral albuterol (4 mg four times daily) or fenoterol (not available in the United States) [6]. In patients with continued cough despite bronchodilator therapy, inhaled corticosteroids may be of benefit. This is due to the fact that frequent and repetitive use of β -2 agonists has been associated with β -2 receptor subsensitivity and down-regulation. These processes are reversed with corticosteroids [5]. Antihistamines should be avoided to prevent inspissation of secretions. Vitamins C and E in addition to zinc and echinacea have been studied for their effectiveness in treating common cold symptoms, but no conclusive evidence has been elicited proving their benefit [7–10]. During febrile periods, it is often recommended

that athletes consume about 3 to 4 liters of fluids to maintain adequate hydration status and decrease viscosity of respiratory secretions [11]. In athletes with lower respiratory tract infections, the secretion of antidiuretic hormone is elevated. In the presence of recommended increased fluid intake during illness, this phenomenon can predispose the athlete to developing hyponatremia in certain instances and should be a source of vigilance for the treating physician. In some cases, less common microorganisms such as *Mycoplasma*, *Chlamydia*, and *Bordetella* species are determined as the cause of symptoms. Arguments have been made for treating athletes with bronchitis with antibiotics to which these organisms are susceptible, specifically tetracyclines, macrolides, and fluoroquinolones in the case of *Mycoplasma* and *Chlamydia* infection, and macrolides or sulfonamides in the case of *Bordetella* infection. In cases of *Bordetella* infection, 1 to 2 g of erythromycin four times a day for 14 days or trimethoprim-sulfamethoxazole double strength can be given twice a day for 14 days. However, despite this information, these antibiotics are not routinely prescribed for treatment of infection with *Mycoplasma* and *Chlamydia* organisms because there is insufficient quality evidence to support the notion that these interventions are beneficial. Also, in many cases, an etiologic agent is often not identified, therefore antibiotics are not routinely recommended in an effort to decrease the likelihood of perpetuating drug resistance in bacteria [2].

Pneumonia and influenza

Cough accompanied by purulent sputum production, crackles on auscultation, nausea, vomiting, and myalgias in addition to constitutional signs of fever higher than 38°C, tachycardia, or tachypnea with or without hypoxemia should raise the suspicion of acute bronchitis complicated by influenza or pneumonia. Patients presenting with these additional symptoms require a chest radiograph for accurate diagnosis of pneumonia. Radiologic findings will often consist of thickened bronchial shadows with areas of infiltration and atelectasis. Less common findings will include hilar adenopathy, nodular infiltrates, and pleural effusions. Depending on the etiological agent, empyema may also be detected. In cases of pneumonia, it is recommended that sputum Gram stain and culture be obtained to determine the specific cause. To determine if a patient requires inpatient or outpatient management of their pneumonia, physicians should use clinical judgment along with the pneumonia severity index (Table 2) to guide their decision making [12]. If influenza is suspected, diagnosis can be made most accurately through detection of virus or viral antigens in throat swabs, nasal washes, or sputum analysis [2].

Accurate determination of the pathology is important in deciding if the symptoms are bacterial or viral in nature and if antibiotics are appropriate treatment choice. In cases of suspected or diagnosed influenza A, amantadine or rimantadine can be prescribed for

Table 2. Pneumonia severity index

Characteristic	Points assigned	
Demographic factor		
Age		
Men	Age (yrs)	
Women	Age (yrs) - 10	
Co-existing illnesses		
Neoplastic disease	+30	
Liver disease	+20	
Congestive heart failure	+10	
Cerebrovascular disease	+10	
Renal disease	+10	
Physical examination findings		
Altered mental status	+20	
Respiratory rate ≥ 30 breaths/min	+20	
Systolic blood pressure < 90 mm Hg	+20	
Temperature $< 35^{\circ}\text{C}$ (95°F) or $\geq 40^{\circ}\text{C}$ (104°F)	+15	
Pulse ≥ 125 beats/min	+10	
Laboratory and radiographic findings (if study performed)		
Arterial blood pH < 7.35	+30	
Blood urea nitrogen level ≥ 30 mg/dL	+20	
Sodium level < 130 mmol/L	+20	
Glucose level ≥ 250 mg/dL	+10	
Hematocrit $< 30\%$	+10	
Partial pressure of arterial O_2 < 60 mm Hg or O_2 saturation $< 90\%$	+10	
Pleural effusion	+10	
Class	Points	Inpatient vs outpatient
I	< 51	Outpatient
II	51–70	Consider outpatient treatment for some
III	71–90	Consider outpatient treatment for some
IV	91–130	Hospitalize
V	> 130	Hospitalize

(Data from Fine et al. [31].)

treatment. In cases in which influenza B is more likely, neuraminidase inhibitors can be given due to their efficacy in treating both influenza A and B [2,4]. When atypical pneumonia is established in the face of mycoplasmal or chlamydial infection, treatment will often be empiric due to the inability to reliably distinguish between typical and atypical causes of pneumonia. Prescribed treatments are determined based on the patient's past 3-month history of antibiotic use (Table

3). The Infectious Disease Society of America recommends a macrolide or doxycycline for patients not previously treated. For those previously treated, recommended treatment includes a fluoroquinolone with enhanced activity against *Streptococcus pneumoniae* (eg, levofloxacin, moxifloxacin), an advanced generation macrolide plus high-dose amoxicillin (1 g orally three times daily), or high-dose amoxicillin/clavulanate (2 g orally two times daily) [13,14].

Table 3. Current treatment recommendations for community-acquired pneumonia in adults

Outpatients
No comorbidities or recent antibiotics use in the past 3 months:
Azithromycin, 500 mg PO on day 1; 250 mg PO qd on days 2–5
Clarithromycin XL, 2 500-mg tablets qd x 5 days or until afebrile for 48–72 h
Doxycycline, 100 mg PO bid x 7–10 days
With comorbidities (COPD, liver or renal disease, cancer, diabetes, heart failure) or antibiotic use in the past 3 months:
Gatifloxacin, 400 mg PO qd
Gemifloxacin, 320 mg PO qd
Levofloxacin, 750 mg PO qd
Moxifloxacin, 400 mg PO qd
Telithromycin, 800 mg PO qd if Gram-negative organisms are not of concern
Combination therapy with a beta-lactam effective against <i>Streptococcus pneumoniae</i> (high-dose amoxicillin, 1 g tid or amoxicillin-clavulanate, 2 g bid or cefpodoxime, 200 mg bid or cefprozil, 500 mg bid or cefuroxime, 500 mg bid) plus either a macrolide (azithromycin, 500 mg on day 1 followed by 250 mg/d on days 2–5 or clarithromycin, 250 mg bid or clarithromycin XL, 1000 mg qd) or doxycycline (100 mg bid)
Hospitalized patients not in the ICU
Combination therapy with ceftriaxone (2 g IV qd; 1 g IV qd in patients > 65 years of age) or cefotaxime (1 g IV every 8 h) plus azithromycin (500 mg IV qd)
Monotherapy with a respiratory fluoroquinolone given either IV or PO except as noted (gatifloxacin, 400 mg qd or levofloxacin, 750 mg qd or moxifloxacin, 400 mg qd or gemifloxacin, 320 mg qd [only available in oral formulation])
Hospitalized patients in the ICU
Intravenous combination therapy with a potent antipneumococcal beta-lactam (ceftriaxone, 2 g qd or cefotaxime, 1 g every 8 h) plus either an advanced macrolide (azithromycin, 500 mg qd) or a respiratory fluoroquinolone (gatifloxacin, 400 mg qd or levofloxacin, 750 mg qd or moxifloxacin, 400 mg qd)
bid—twice daily; COPD—chronic obstructive pulmonary disease; ICU—intensive care unit; IV—intravenous; PO—by mouth; qd—every day; tid—three times daily. (Data from File [32].)

Complications

Spontaneous pneumothorax

The presence of a continued cough in the diagnosis of bronchitis or pneumonia should elevate the physician's vigilance in suspecting and detecting the occurrence of spontaneous pneumothorax in athletes. The occurrence of spontaneous pneumothoraces is rare, with only 2000 new cases occurring in the United States each year, 10% of which occur during athletic activity. The increased intrathoracic and intrapleural pressure associated with coughing has been shown in some cases to increase the likelihood of occurrence [15].

Spontaneous pneumothorax is often seen in males in their late teens to late 30s who are of a generally taller and slender build. Athletes who smoke and those who abuse inhaled substances are also at an increased risk, with light smokers (defined as 1–12 cigarettes per day) having a relative risk of occurrence seven times higher than the general population [15–17]. Athletes with documented connective tissue disorders such as Marfan syndrome (most commonly seen in tall, slender basketball and volleyball players) are also at increased risk for occurrence [4].

There is no known cause of spontaneous pneumothorax but the most widely accepted hypothesis relates the cause to subpleural blebs in apical lung tissue resulting from airway inflammation. When subjected to injurious stimuli (eg, outside trauma or, in the case of chronic cough, repeated increased intrathoracic pressure with the Valsava-like mechanism of forcing air against a closed glottis present before coughing), these blebs can rupture and result in the formation of a pneumothorax [15].

The presentation of an athlete afflicted with a spontaneous pneumothorax can be varied. An athlete's most common complaint will be of dyspnea and pleuritic chest pain, which are present 80% to 90% of the time. Other athletes may only complain of chronic cough and, in 10% of cases, some may not even present with a chief complaint due to their increased state of conditioning or due to their familiarity with compensating for discomfort or injury [15,16]. In light of these various presentations, it is the physician's responsibility to collect adequate information via a detailed history and examination to make an accurate diagnosis.

Patient history may often be unremarkable due to the frequent occurrence of spontaneous pneumothorax at rest [16]. Depending on the size of the pneumothorax, the physical examination may be unremarkable. If large enough to yield physical findings, auscultation will reveal an area of decreased breath sounds over the area affected with decreased chest excursion. In addition, the affected area will reveal hyper-resonance to percussion accompanied by decreased fremitus. Athletes may also experience tachycardia or tachypnea along with hypoxemia due to continued perfusion of poorly ventilated lung tissue [4,17]. Should the pneumothorax be complicated by tension upon the mediastinum, more evident signs will be present including tracheal deviation to the affected side, decreased blood pressure, tachypnea, and cyanosis. These clinical signs make the diagnosis of tension pneumothorax easier; however, this situation is life threatening and requires immediate intervention with simple aspiration or tube thoracostomy to equalize intrathoracic pressure.

In spontaneous pneumothorax uncomplicated by tension, the physical findings during examination should be sufficient for the clinician to make the correct diagnosis. In situations in which the physical examination is less telling, the gold standard for diagnosis continues to be the posterior-anterior inspiratory plain chest film. Diagnostic films will show an absence of lung markings over the affected area with demonstration of a pleural line representing the border of the collapsed lung [4].

Once the correct diagnosis is made, treatment is often influenced by the relative size of the pneumothorax with 15% of the lung space of the affected side being the cutoff point in decision making. Pneumothoraces that occupy less than 15% of the pleural space of the affected side in clinically stable athletes are often treated observantly. A follow-up radiograph is often checked within 6 hours to determine that the pneumothorax is not progressing [4]. When stable, weekly chest films are obtained to monitor for reoccurrence until radiographic resolution is obtained [16]. Pneumothoraces occupying an area greater than 15% of the pleural space are often managed in a hospital setting and treated via simple aspiration or tube thoracostomy with or without instillation of a pleurodesis agent in the form of intrapleurally injected tetracycline, doxycycline derivate, or talc slurry. In addition, surgical removal of any remaining tissue blebs via thoracoscopy or open thoracotomy can be attempted to prevent reoccurrence [17].

Bronchiectasis

Bronchiectasis, the acquired abnormal dilation and destruction of bronchial walls occasionally occurring after lower respiratory infections, is another potential complication athletes may face. To accurately diagnose bronchiectasis after recent lower respiratory infection, patients will often need chest imaging. Plain chest films will reveal airway dilatation appearing as tram-tracking or end-on ring shadows in lung fields. Mucopurulent

plugs or obstruction due to other debris with postobstructive air-trapping is often noted, appearing as irregular peripheral opacities. If plain films are relatively normal in appearance, high-resolution CT scans of the chest will provide a more definitive diagnosis. In addition to these findings, scans will also show a lack of tapering of bronchi in the presence of bronchial dilatation, which is more specific for bronchiectasis. Airways of affected athletes will often be inflamed, edematous, and possibly cratered or ulcerated [18]. Irritation of the airways causes hypertrophy and tortuosity of the bronchial arteries that run along branches of the bronchial tree. The increased intrapleural pressures and forces associated with coughing increases the possibility of hemoptysis resulting from structural compromise of these arteries [19]. For patients with bronchiectasis uncomplicated by hemoptysis, treatment is often centered on the principle of adequate pulmonary hygiene. Although many forms of pulmonary hygiene are currently in use, the most common is general hydration with oral liquids and nebulization with hypertonic saline solutions or mucolytic agents [20]. Given the inflammatory mechanism behind bronchiectasis following bronchitis and pneumonia, bronchodilators and corticosteroids have also been suggested as possible remedies. Studies exploring the efficacy of bronchodilators to treat bronchiectasis are lacking. Steroids have been shown to cause some degree of immunosuppression when prescribed [18,21,22]. This relative compromise in immune function can lead to potential worsening of the condition the steroids were used to treat.

Hemoptysis

In the face of chronic cough due to a lower respiratory infection, bronchial arteries are repeatedly subjected to increased pressure and force associated with coughing. The resultant extent of hemoptysis is subject to the amount of coughing, the amount of arterial hypertrophy and the degree of expansion of vascular plexuses in the affected area of the bronchial tree. The blood produced will often be minimal and present as blood-tinged or streaked sputum. In the unfortunate circumstance of massive hemoptysis due to bronchial arterial rupture, the most important initial treatment is ensuring a protected airway by inserting a large-bore endotracheal tube. Having secured an adequate airway, management can then focus on treatment of the source of bleeding with initial intervention in the form of bronchoscopy with balloon tamponade of the compromised vasculature. If this intervention fails, resolution is then sought with arterial embolization of the affected vessels followed potentially by resection of affected lung tissue if unresolved [23].

Acute respiratory failure

At the end of the spectrum for possible complications of lower respiratory infection in athletes is acute respiratory failure. If left untreated, the persistent inflammation and

edema present with acute lung injury from lower respiratory infection can ultimately lead to increased hypoxia resulting from progressive pulmonary edema and diffuse alveolar damage. Patients affected to this degree require positive pressure ventilation and treatment of the initial underlying respiratory condition along with any subsequent complicating factors. To accurately determine the underlying respiratory condition, the previous course of the patient's symptoms should be reviewed for any circumstances raising suspicion of a particular etiology. Lacking a distinct etiology based on the patient's disease history and course, bronchoscopy with bronchoalveolar lavage with or without subsequent lung biopsy can be used to determine the cause of the patient's respiratory difficulty [24].

Return To Play Criteria

To date, there is an inadequate number of reliable studies to accurately guide physicians in confidently determining when is the appropriate time for an athlete to resume training and return to play following lower respiratory infections and their potential complications. Given this lack of guiding information, it is of prime importance for the physician caring for the athlete to understand not only the mechanism of the illness affecting the athlete but also the demands of the sport in which he or she may be participating in order to make safe recommendations as to what level of activity may be undertaken.

When treating lower respiratory tract infections, the physician should consider the issue of contagiousness given the athlete's close person to person contact when competing. Return to play considerations will often hinge on the etiologic agent causing the athlete's symptoms and the length of time the athlete is considered contagious. In cases in which athletes do receive antibiotic treatment, there is no reported documentation limiting return to play. An additional consideration when determining an athlete's ability to return to play is the "neck check" put forth by Eichner. The guideline states that if patient's respiratory symptoms are mainly above the neck (eg, coryza, sneezing, and headache), then the athlete may return to play. If, however, patient symptoms are confined below the neck (eg, fever, cough, myalgias), strenuous activity should be restricted until the symptoms resolve [25]. For athletes returning to play, caution should be taken to prevent them from returning too fast and incurring additional illness. Intense exercise can increase the severity of viral illness during recovery and manifest as muscle impairment of skeletal, respiratory, or cardiac muscle strength and function. This impairment can trigger such complications as activation of latent reactive airway disease, myocarditis and musculoskeletal injury resulting from impaired mechanics [26].

In the case of spontaneous pneumothorax, after diagnosis, the athlete should be restricted from strenuous training activities until complete resolution of the con-

dition is documented. After resolution, reports suggest it is safe return to training activities immediately with a gradual increase in activity slowly progressing to full competitive activity as tolerated by the athlete. Athletes treated with more invasive procedures such as thoracotomy can resume participation in all sports after a period of 2 to 4 weeks [15,27]. During recovery, athletes should be appropriately counseled regarding the risk of possible recurrence given that the incidence of such occurrence averages 30%. The most common recurrences occur in patients who are younger, female, tall in stature, and smokers. With recurrence, athletes should be advised to discontinue contact sports and pursue relatively sedentary leisure activities unless they are treated with a corrective procedure [16,27].

Prevention

To help athletes avoid lower respiratory infections and their potential complications, preventative efforts must be an underlying theme in all training and competitive activities. Avoiding sick contacts and limiting contact with those known to be ill is essential in minimizing the spread of disease. Simple, frequent hand washing should also be implemented to help reduce the spread of droplet-borne viral infections such as common colds and influenza. Immunization is a crucial aspect of prevention; most physicians recommending athletes be immunized against tetanus/diphtheria, measles/mumps/rubella, hepatitis B, and poliomyelitis. Other immunizations to be considered given the athlete's specific situation are hepatitis A (for travel to endemic areas), pneumococcal (for older, immunocompromised, or splenectomized athletes), meningococcal (for athletes who are splenectomized, traveling to endemic areas, or living in dorms), and varicella (for athletes not exposed to the disease by age 12). The influenza vaccine should also be strongly considered for athletes competing during the flu season [26].

Vitamins have been a focus of numerous studies for their efficacy in prevention of respiratory infections in athletes. Some studies have shown that vitamin C may be of benefit as prophylaxis against respiratory infections for those athletes exposed to extremely cold environments for short periods (eg, ultra-marathoners). For athletes exposed to less extreme conditions, however, this treatment is of minimal efficacy as prophylaxis [28].

Another focus of prevention has been the "inverted U hypothesis" when referring to exercise load. In essence, this theory states that athletes participating in moderate amounts of training and exercise will be less prone to getting respiratory infections than people with more sedentary pursuits due to the enhancing effect of moderate exercise on immune system function. Training at more exhausting and demanding levels can cause relative immunodepression, thereby causing the athlete to be more susceptible to infection. Despite studies examining the effect of exercise

on different components of the immune system, there is no definitive study proving this relationship [26,29].

Conclusions

Lower respiratory infections and the potential complications that emerge with improper or inadequate treatment are conditions even well-conditioned athletes are at risk for developing given the physical stresses of training and close contact with other athletes during training and competition. When these situations arise, it is necessary for the physician following these athletes to make the correct diagnosis and administer the proper treatment, stressing rest and recovery, to ensure adequate patient recovery and prevention of more serious sequelae.

A common concern among affected athletes is the increased apprehension of deconditioning or, if they are competing in a team setting, letting the team down. To stress the importance of a brief period of rest during illness, athletes should be informed deconditioning does not begin until after 4 to 5 days of inactivity. As symptoms gradually resolve and training intensity increases, physicians should continue to monitor athletes for any potential relapse with appropriate treatment prescribed as necessary.

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This reference has been highlighted for its discussion of treatment regimens currently accepted for the treatment of community acquired pneumonia in varied inpatient and outpatient populations.