Exercise–Induced Asthma

A seemingly well-conditioned soccer player removes himself from a match because he is having severe difficulty breathing. He is coughing, wheezing, and complaining of tightness in his chest. As the athletic trainer or therapist performs the sideline evaluation it becomes evident that no trauma has occurred. Is it time for the athletic therapist to panic? Spontaneous pneumothorax? Anaphylactic reaction? Because of a thorough preparticipation exam, we know this athlete is suffering from an exercise-induced asthma (EIA) attack. He managed the attack with an albuterol inhaler that was brought to the field by the athletic trainer or therapist. After two puffs from his inhaler and 5 min of slow, rhythmic breathing, the athlete returned to the soccer match without further incident. Scenarios like this occur daily in gymnasiums and playing fields around the world. The purpose of this article is to provide you with the background you’ll need to successfully prevent, recognize, and manage EIA.

EIA is a chronic inflammatory disorder that is more prevalent and serious than was once believed (Tan & Spector, 1998). EIA affects 15–20% of the general population, 10% of all athletes, 11% of Olympic-level athletes, and 90% of all people with asthma (Voy, 1986). The prevalence of EIA among competitive athletes has been increasing in the past decade (Schoene et al., 1997). EIA affects individuals at all levels of activity, from recreational athletes to elite, highly competitive athletes. A correct diagnosis by a physician is critical to proper treatment and usually requires pulmonary function testing. Fortunately, with the efficient pharmacologic and nonpharmacologic treatments available, EIA can be adequately controlled, and participation in exercise and sport can occur safely.

Pathology

EIA is a clinical syndrome defined as an intermittent narrowing of the airways following 6–8 min of vigorous exercise (Weiler, 1996). The characteristic signs and symptoms of EIA include wheezing, chest tightness and discomfort, shortness of breath, sensitivity to cold air, feeling “out of shape,” and coughing. Some patients with EIA have no specific symptoms of typical asthma, but many experience stomach cramps, chest pain or discomfort, nausea, fatigue, and headaches. Some exhibit signs of asthma only when exercising, and most patients who have chronic asthma have increased symptoms with physical exertion (Storms & Joyner, 1997).
EIA is an inflammatory process that causes a narrowing of the bronchial tree, thus obstructing the flow of air in and out of the lungs. Bronchial-wall edema, mucous production, muscle contraction, and muscle hypertrophy contribute to the airflow obstruction. The obstruction can be caused by inflammatory agents in the airways, particularly the release of inflammatory mediators from mast cells, macrophages, and epithelial cells (Rupp, 1996). Airway hyperactivity is a magnified bronchoconstriction response to stimuli such as allergens and environmental irritants, respiratory infections, exercise, or cold, dry air (Rupp; Storms & Joyner, 1997). Other contributing factors include the type, intensity, and duration of exercise; fatigue; emotional stress; sinusitis; bronchitis; food sensitivity; overtraining; and hyperventilation (Katz, 1986; see sidebar).

There are different theories on the mechanisms of EIA. One is the water loss theory, which is based on the idea that an EIA response is caused by decreased hydration of the airways during exercise. This dehydration is thought to occur as water from the airways is lost into exhaled air during exercise. This water loss can trigger bronchospasm. Because of the large volume of air that is inhaled and exhaled, the loss of water causes changes in osmolarity, pH, and temperature in the airway, triggering an EIA response (Storms & Joyner, 1997; Weiler, 1996).

Another theory is the thermal expenditure theory, which suggests that an EIA response is a direct result of heat loss from the airways during and after exercise. During exercise, respiratory heat loss occurs as heat is discharged with exhaled air. This transfer of thermal energy is followed by rewarming of the airways after termination of exercise. Rewarming of the airways is associated with dilation and hyperemia of the bronchiolar vessels, which then can lead to an EIA response (Storms & Joyner, 1997).

Another suggested cause of EIA involves the release of inflammatory mediators from mast cells and basophils in response to vigorous physical exertion. This also can lead to bronchospasm (Weiler, 1996). Cold air can also directly lead to bronchoconstriction (Storms & Joyner, 1997).

Bronchodilation normally occurs in response to exercise. The widening of the glottis and bronchodilation help the body meet increased oxygen demands during exercise. In EIA, however, the glottis widens but the lower airways constrict. Respiratory demand increases with exercise, but forced expiratory volume and peak expiratory flow rate decrease significantly during an EIA attack. Maximal bronchoconstriction generally occurs 3–15 min after exercise. Although most patients recover from EIA within an hour, late asthmatic responses have been reported to occur 3–9 hr after exercise. This appears to be more prevalent in children and patients who suffer from severe bronchospasm (Rupp, 1996).

**Diagnosis**

EIA is often not diagnosed by physicians because the symptoms are not always characteristic. Many athletes think their respiratory symptoms mean that they are “out of shape.” For physicians to diagnose EIA, a thorough history must be obtained, and pulmonary function tests at rest and during exercise are necessary (Weiler, 1996). An exercise-induced decrease in