Exercise-Induced Asthma

The terms “exercise-induced asthma” (EIA) and “exercise-induced bronchospasm” (EIB) are used synonymously to describe acute lung airway narrowing occurring during and/or after physical activity. EIA is considered a more inclusive term. The bronchospastic response is thought to result from a summation of specific events, including smooth muscle contraction of the airway, bronchial mucosal edema, and mucus plug formation. The pathogenesis of these events is associated with the generation of inflammatory mediators including leukotrienes, prostaglandins, and other immune system factors from airway mast cells, epithelial cells, and macrophages interacting with in-situ hormonal components of the lungs.

The underlying factors governing the initiation of EIB are not clearly understood. Changes in airway temperature (cooling and rewarming), alterations in mucosal osmolarity (airway drying), and congestion of the bronchial arteries, resulting in bronchial mucosal vascular engorgement, have all been suggested as causes. At present, it seems that the bronchial blood flow/ bronchial heat exchange relationship influences the development of airway narrowing following exercise-related overbreathing. EIB typically occurs after ventilation with large quantities of air, especially cold, dry air that contains environmental pollutants and/or allergens. The frequency and severity of the reaction reflects the underlying allergic predisposition of the individual, the degree of overbreathing, coldness and/or dryness of inspired air, the burden of environmental agents inhaled, and the intensity of exercise. As a result, seasonal fluctuations in the bronchospastic response have been identified. After the occurrence of an EIB episode, approximately 50 percent of patients experience a relative refractory period lasting for up to two hours wherein another exercise challenge will fail to produce EIB or will produce a lesser reaction. Late asthmatic responses occurring six to eight hours after the initial bronchospasm also occur in about 50 percent of the EIB population, but are typically mild.

The method used to detect the EIB response critically affects the estimates of prevalence. Although the convenient peak flow meter is adequate for use with highly reactive and symptomatic individuals, it is relatively insensitive in mildly affected persons or elite athletes in whom small reductions in bronchial airflow may lead to a significant decrease in performance. In addition, peak flow measurements are critically effort-dependent, so this diagnostic technique may not be absolutely reliable. Spirometric measurements and maximal mid-expiratory flow rates are acceptably accurate and reproducible, as effort variation is detectable from the configuration of the tracings.

The intensity of the exercise challenge used to induce the EIB response is another important variable. Standard clinical protocols to provoke EIB apply exercise bouts of five to eight minutes at a level just below the lactate threshold (LT), 70-85 percent of maximal heart rate reserve. This work rate has been selected because the subject may not complete the exercise challenge at a higher intensity, and a more severe test promotes catecholamine release producing bronchodilation. A major problem with this standard protocol is identifying the LT for an individual, given the wide range of fitness levels in the general population. With elite athletes, sub-LT exercise is typically not sufficient to produce EIB. Additionally, laboratory-based exercise challenges are rarely performed in the environmental circumstances (e.g. cold, dry air) that produced the symptoms suggesting EIB.

As a result, the prevalence of EIB is reported to be ten to 50 percent, depending upon the study population, exercise protocol, detection measurement and environmental conditions. For the general population, an incidence of ten to 15 percent is a reasonable figure. Most moderately to severely allergic subjects will demonstrate some level of EIB. Recent studies show the frequency to be 20 to 75 percent (depending upon the sport) among elite cold weather athletes. The majority (73 percent) of the athletes who met diagnostic criteria during “field evaluations” did not meet the criteria when retested using the standard laboratory protocol.

Medications to modify or prevent the EIB response include bronchodilators, anti-inflammatory compounds such as inhaled cromolyn, nedocromil, and corticosteroids, and a variety of medications including antihistamines, calcium channel blockers, and inhaled heparin. More recently, immune system modifiers are available, including leukotriene or neurokinin receptor inhibitors and lipoxygenase inhibitors.

An important question to study in view of the wide use of pharmacological prophylaxis by elite winter sports athletes is whether or not bronchospasm is a natural phenomenon that serves as a physiological mechanism to protect the lower airways from the noxious insult of exposure to large volumes of cold dry air. Similar airway-narrowing is a common response to inhalation of such irritant gases as sulfur dioxide (SO2) and nitrogen dioxide (NO2), both of which can occur in high concentrations in ice rinks.

For most asthmatic patients, and the majority of people who do not suffer clinical asthma but exhibit EIB, individualized prophylactic treatment is safe and effective, allowing full participation in sports activities. In addition, proper timing of preparticipation warm-up exercise enables some athletes to utilize the refractory period to attenuate the bronchospastic response during exercise and achieve optimal performance.

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