Exercise-induced Asthma: Past, Present, Future

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Exercise-induced asthma (EIA) involves series of stages of bronchoconstriction, bronchodilation, and a refractory period (1). Although there were recent advances in the treatment of EIA by breathing warm humid air during exercise instead of cold dry air, other areas such as the mechanism or the refractory period of EIA had conflicting results (4, 6 & 10). In fact, even what constitutes asthmatic and normal subjects were not definitive which warrants further investigation. Thus, the trends in recent studies have focused more on the accurate diagnosis and the underlying mechanism behind EIA and the refractory period.

Exercise-induced asthma is defined as a narrowing of the airways with symptoms of wheezing, chest tightness, and coughing that is triggered by exercise (6). It can be diagnosed by completing variety of tests including methacholine, histamine challenge, and eucapnic voluntary hyperpnea (EVH) test (11). However, each test varies in their predictive value of EIA, cost and ease of administration (11). To clarify the terms, FEV1 is forced expiratory volume in 1 second, peak expiratory flow (PEF) is how fast a person can exhale air and forced vital capacity (FVC) is the maximum volume of air that a person can exhale after maximum inhalation (5). Compared to non-asthmatics, subjects with EIA had significant reduction in PEF, FEV1/FVC values and developed post-exercise bronchoconstriction (9). Nevertheless, a contrary finding from Lefcoe, Carter, and Ahmad showed that group differences between asthmatics and normal subjects were not clear-cut because some normal subjects exhibited significant post-exercise bronchoconstriction (9). Yet, another study by Kenneth and Scanlon clarified contradictory results of EIA (1). In previous studies of EIA, investigators have found an increase in airway resistance after brief exercise (5 to 10min) in either constant or incremental load (1). However, Kenneth and Scanlon found that for subjects with EIA, bronchoconstriction can occur during exercise of lower intensity but can also cause bronchodilation if exercise intensity is returned to a higher level (1).

Although the exact mechanism behind EIA is still unclear, many studies suspect thermal and osmotic effects of water lost from the airways from inspired air (6). However, Belcher, Rees, Clark and Lee found that respiratory water loss did not have an effect on EIA, but rather pointed to a transient hyperosmolarity of the airway
that led to bronchoconstriction (2). Still other research theorized that the release of stored mediators such as leukotrienes, histamines, and prostaglandins or enzyme precursors cause EIA (5 & 6). According to this study, the mediators would cause a chain of events that would narrow the airways and lead to bronchoconstriction, coughing, and breathlessness (6).

Another unknown post-exercise response is the refractory period. It is defined as the time after an asthma attack when an individual is at decreased risk of developing a second attack (7). According to McFadden, the refractory period after exercise lasts up to four hours (8). Although the reason for the refractory period is also unknown, some suggest that the depletion of the mast cell mediators is the primary mechanism (6). This was also supported by a study done by Edmunds, Tooley, and Godfrey (3). Still, alternative theory proposed that it was the catecholamines that protected the airways during the initial challenge (10). Yet, a third theory argued that it is a reduction in airway smooth muscle responsiveness (10). However, not everyone with EIA has a refractory period. Numerous studies have shown that only half of asthmatics who recovered from an initial EIA were less responsive after second exercise stimulus (2, 6 & 10). Also, among those who exhibited refractory period, even a preliminary warm-up exercise reduced the symptoms of EIA significantly (4). However, this refractory period disappears if asthmatics are treated with anti-inflammatory medication for several days (6).

As research on the process and the refractory period of EIA develops, there will be more theories and explanations to understand this complex phenomenon. Nevertheless, many studies suggest that these mechanisms cannot be explained by one or two factors (10). As a result, any research findings that speculate any one cause should be met with rigorous scientific analysis.

Works Cited:
Respiratory Critical Care Medicine. 149 (1994): 352-357


