Airway Hyperresponsiveness in Asthma: Its Measurement and Clinical Significance

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Airway hyperresponsiveness is a characteristic feature of asthma, and its measurement using direct inhalation challenges, particularly with inhaled methacholine, or indirect challenges with inhaled mannitol or exercise is important in establishing a correct diagnosis. This is particularly true in excluding asthma as a diagnosis in patients with symptoms that suggest asthma, but are caused by another condition. This is because airway hyperresponsiveness measurements are very sensitive in determining a diagnosis of asthma (therefore, they have a high negative predictive value) but are not very specific because patients with other diseases, such as allergic rhinitis or COPD, can have airway hyperresponsiveness without asthma.

This supplement to CHEST highlighted the many insights that have been made into the role of measuring both airway hyperresponsiveness and airway inflammation. These methods have proven very helpful clinically, as well as in mechanistic studies of asthma. The importance of airway inflammation in increasing airway responsiveness after exposure to environmental or occupational stimuli, such as allergens, or small-molecular-weight chemicals, such as toluene diisocyanate, is now established; however, the role of airway inflammation and its associated structural changes (airway remodeling) in initiating airway hyperresponsiveness remains unclear.

Finally, the value of measuring both airway hyperresponsiveness and airway inflammation in making treatment decisions, particularly about the doses of inhaled corticosteroids (ICS) used in patients, has been evaluated. Adjusting the doses of ICS to optimize the measurements of methacholine airway responsiveness leads to improved asthma control, whereas adjusting ICS doses to minimize sputum eosinophils reduces asthma exacerbations. These studies have not been conducted yet for the indirect airway challenges, such as mannitol or exercise.

The symposium described in this supplement has identified the many advances that have been made since the original symposium in 1979 in understanding the value of measuring airway hyperresponsiveness and airway inflammation for establishing a diagnosis of asthma and optimizing treatment, as well as the association between these two markers of asthma. However, the concluding statement made by Dr Roland Ingram at that meeting that “neither I, nor I suspect anyone else here, truly understands the genesis or modulating mechanisms” of airway hyperresponsiveness remains almost as true today. Although this could be regarded as a failure of the past research efforts into understanding the mechanisms of airway hyperresponsiveness, it is almost certain that, with the extent of the research endeavors into clarifying these issues, this problem will be solved and likely soon.

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REFERENCES


