Cough Pathophysiology – Lower Airway Perspective

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The pathophysiology of cough variant asthma (CVA) is poorly understood[1]. In particular, the mechanisms for the difference in symptomatology between typical asthma (in which wheeze predominates) compared with CVA (in which cough predominates) have not been determined. Higher wheezing thresholds, differences in cough sensitivity and/or differences in small airway function may play a role. This session will describe recent insights regarding a potential pathophysiologic role for altered pulmonary mechanics and small airway function gleaned from high-dose methacholine (MCT)[2] and mannitol challenge testing[3, 4]

MCT reliably mimics the main mechanical aberrations and sensory consequences (intensity of and quality of dyspnea) experienced during spontaneous bronchoconstriction in asthma[5, 6]. We have demonstrated that dynamic hyperinflation (DH), as measured by the reduction in inspiratory capacity (IC) (% predicted), is the strongest independently significant factor contributing to dyspnea during MCT in classic asthma [5-7]. Recently, we have shown that individuals with chronic cough who cough during MCT but have normal airway sensitivity to methacholine (PC20>16 mg/mL) develop small airway obstruction, DH, gas trapping and increases in positive end-expiratory pressure measured by esophageal balloon catheters before a cough, which partially resolve following a deep inspiration and cough [8, 9]. Preservation or loss of the bronchoprotective effect of a DI may be a fundamental pathophysiologic difference between asthma, CVA, methacholine-induced cough with normal sensitivity and eosinophilic bronchitis.

References


