CONCLUSION. Clustered groups of self-organized terminal bronchiolar constriction, not large airways obstruction, contribute to large ventilation defects in acute asthma.

REVIEWER COMMENTS. The nature of functional changes of both small and large airways affecting ventilation during acute asthma attacks has been unclear. Previously, MRIs of asthmatic lungs during bronchoprovocation suggested large-airway obstruction as a major cause of large ventilation defects (eg, J Allergy Clin Immunol. 2003;111:1205–1211). In this study, Venegas et al demonstrated the role of clustered terminal bronchiolar constriction resulting in ventilation defects in acute asthma. On the basis of this model, inhaled bronchodilators could be ineffective because the inhaled form might reach only well-ventilated regions and could further impede lung expansion of problematic regions and exacerbate regional ventilation defects. The concept of catastrophic shifts might account for sudden, unexplained, and severe asthma attacks in some patients. Systemic bronchodilators may be needed in some asthmatics to bypass this problem. This line of investigation is further elucidated elsewhere (J Appl Physiol. 2005;99:2388–2397).

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The Bronchial Lavage of Pediatric Patients With Asthma Contains Infectious Chlamydia


PURPOSE OF THE STUDY. To examine the frequency of Chlamydia pneumoniae infections in pediatric patients with asthma.

STUDY POPULATION. Seventy pediatric patients undergoing flexible fiber-optic bronchoscopy as a part of their ongoing clinical care.

METHODS. Bronchoaveolar lavage (BAL) fluid and blood were examined for the presence of C pneumoniae by smear examination and culture. The BAL and blood samples were cultured on human or mouse macrophages to determine infectivity. Polymerase chain reaction (PCR) amplification of BAL samples was performed to confirm specificity of the culture technique. Blood was examined for total immunoglobulin E (IgE). Blood samples from 70 matched, nonrespiratory control patients were cultured for Chlamydia.

RESULTS. Forty-two patients undergoing bronchoscopy had asthma and 28 had various other respiratory diseases. Thirty-eight (54%) BAL samples were positive for Chlamydia by PCR and 22 (31%) samples were positive for Chlamydia by culture. Of the positive BAL samples, 28 (74%) of 38 PCR-positive and 14 (64%) of 22 culture-positive samples were from children with asthma. Culture-positive blood samples were found in 24 (34%) of 70 respiratory patients and 8 (11%) of 70 nonrespiratory controls. In the blood culture–positive respiratory group, 17 (71%) of 24 were from children with asthma. Elevated total serum IgE was associated with BAL culture–positive results, and this relationship was stronger than total IgE and asthma diagnosis.

CONCLUSIONS. Viable C pneumoniae organisms are frequently present in the lung lavage in a cohort of predominately asthmatic pediatric patients.

REVIEWER COMMENTS. Results from this study suggest that infectious C pneumoniae may be common in BAL fluid of children with asthma. Historically, C pneumoniae has been associated with exacerbation and increased incidence of respiratory conditions in adults, but studies to examine similar associations in children have not been performed. This is the first investigation to report viable and infectious C pneumoniae in the BAL fluid of children with asthma. These findings are intriguing and should encourage investigators to examine the clinical implications of Chlamydia infection among pediatric patients with asthma.

Repeat Exercise Normalizes the Gas-Exchange Impairment Induced by a Previous Exercise Bout in Asthmatic Subjects


PURPOSE OF THE STUDY. To determine the effects of a second exercise bout on the gas-exchange impairment caused by an initial exercise-induced bronchospasm (EIB) response in asthmatic subjects.

STUDY POPULATION. Twenty-one subjects with a known history of asthma participated after meeting at least 1 inclusion criteria: (1) ≥12% increase in the forced expiratory volume in 1 second (FEV1) after β-agonist inhalation, (2) ≥10% decrease in FEV1 after exercise test to exhaustion, or (3) a provocative concentration ≤4.0 mg/mL of methacholine causing a 20% decrease in FEV1.

METHODS. The subjects performed 2 submaximal workloads for 3 minutes. After 3 to 5 minutes of rest, constant work-rate exercise was performed until exhaustion at 90% of maximal O2 uptake (EX1). Arterial blood and expired gases were collected at 3 (early recovery) and 35 (late recovery) minutes after EX1. Subjects then per-
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