The concept that chronic inflammation of foregut structures can be associated with airway inflammation and damage struck a cord with us since we have noted a striking excess of cases of treated hypothyroidism among a population of patients with idiopathic chronic cough\(^2\) and in nonsmoking patients with fixed airflow obstruction.\(^3\) Hypothyroidism is usually due to autoimmune destruction of the thyroid gland and is associated with intrathyroid lymphocytic infiltration. We have shown that idiopathic chronic cough is associated with a BAL lymphocytosis\(^2\) and have suggested that this is due to homing of activated lymphocytes from the primary site of autoimmune inflammation to embryologically related structures such as the airways.\(^2\) The mechanism of airway inflammation and damage in autoimmune thyroid disease, and perhaps in chronic hepatitis C infection, may be analogous to that thought to be responsible for airway complications of inflammatory bowel disease. The concepts that inflammatory bowel disease and autoimmune thyroid disease are associated with airway disease, and that the pathogenesis is similar and not related to thyroid hormone status, are supported by a recent study\(^4\) showing a twofold to threefold excess of cough, sputum production, and breathlessness, and a remarkably similar profile of respiratory symptoms, among a cohort of patients with inflammatory bowel disease and among another cohort with treated autoimmune thyroid disease.

One important difference between the primary sites of inflammation in chronic hepatitis C infection and autoimmune thyroid disease is that the former condition is treatable. The findings of Kanazawa et al\(^1\) with interferon therapy raise the interesting possibility that treatment may modify the airway consequences of chronic inflammation of the foregut. The search is on for other treatable causes of chronic foregut inflammation that might be relevant to airway diseases. COPD is associated with peptic ulcer disease,\(^5\) so one possibility worth investigating is that chronic gastric inflammation secondary to Helicobacter pylori infection is a potentially modifiable factor underlying the amplified immune response to cigarette smoking and other pollutants that characterize COPD.

**To the Editor:**

We thank Dr. Birring for an interest regarding our study (February 2003).\(^1\) Hepatitis C virus (HCV) infection is a major cause of chronic liver disease. However, it has previously been reported that chronic HCV infection is associated with several other syndromes, including cardiomyopathy\(^2\) and proliferative glomerulonephritis.\(^3\) Therefore, we speculated that there is also an interaction between chronic HCV infection and some pulmonary disorders. Birring et al suggest that airway disease may be related to the underlying chronic inflammatory disorder.\(^4\) We were very interested in their concepts. In our previous study,\(^5\) we discussed the role of T lymphocytes in airway inflammation in asthmatic patients with chronic HCV infection. They also emphasized that idiopathic chronic cough with hypothyroidism and inflammatory bowel disease is due to the homing of activated lymphocytes from the primary site of inflammation to embryologically related structures such as airways. However, we did not have any data that chronic inflammation of the foregut structure can be associated with airway inflammation. In future studies, we will examine the relationships between chronic inflammation of the foregut structure and airway inflammation according to their concepts.

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A Reappraisal of Nasal Saline Solution Use in Chronic Sinusitis

**To the Editor:**

We read with interest the article by Tsoa et al\(^1\) in the March 2003 issue of CHEST. Although we agree with the idea that the chronic sinusitis that ensues concomitantly in children with mild asthma and allergic rhinitis should definitely be treated, we have some objections to the aggressive treatment methods they have recommended.

First of all, saline solution nasal washing certainly facilitates nasal drainage and clears the airway from any postnasal dis...
charge; however, it can be effective when applied appropriately. As described in the aforementioned studies, saline solution is applied (five drops in each nostril) at least four times a day (before going to bed, in the morning, and twice before feeding) until the symptomatology subsides. In the study by Tsao et al., nasal saline solution was applied once a week, whereas the technique was not designated. At this point, one can reasonably speculate that inappropriate saline solution administration could have a role in the worse treatment outcome when compared to groups of children treated with antibiotics.

There is still much debate on the use of antibiotics in chronic sinusitis treatment; as opposed to Tsao et al., there are studies that have shown the ineffectiveness of either topical or systemic use of antibiotics in chronic sinusitis. Besides, antibiotics are widely known allergens and can readily cause allergic reactions in this atopic group of asthmatic patients. Asthma exacerbations have been reported during various antibiotic treatments including amoxicillin. Moreover, it has also been mentioned that the use of antibiotics can even bring about asthma in early childhood. Thus, we strongly underscore the necessity of a delicate decision before utilizing antibiotics in asthmatic children.

Overall, the treatment strategy for asthmatic children with chronic sinusitis has not been established. Antibiotics—with their high costs, possible side effects, disputed treatment outcomes, and likely contribution to asthma pathogenesis—comprise one group of treatment alternatives. However, nasal saline solution—with the evidence of its beneficial effects in the treatment of upper respiratory tract infections and acute sinusitis—is a cheap and convenient way of treating these patients. We do imply that placebo-controlled cohort trials are obviously awaited for rendering its success in the treatment of concurrent asthma and chronic sinusitis in children.

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To the Editor:

We appreciate the recommendation regarding the method of nasal washing for patients with chronic sinusitis. From the results of our study and Oliveria et al., nasal saline solution irrigation can be safely used for symptom relief without any influence on bronchial hyperreactivity (BHR) in asthmatic children with concomitant chronic sinusitis; however, we cannot neglect the possibility of a different method of nasal saline solution irrigation influencing the result of our study.

Sinusitis is not uncommon in asthmatic patients. As mentioned by Karadag et al., there is still much debate on antibiotic treatment for chronic sinusitis. However, some studies have shown that children with asthma had remarkable improvement of lower airway symptoms and pulmonary function after diagnosis and concomitant treatment of sinusitis. The impressive result of our study also reveals a trend. For mild asthmatic children with concomitant chronic sinusitis, if aggressive antibiotic treatment is provided there is significant improvement on symptoms and BHR. From this point of view, for those asthmatic children who show an unpredictable response to appropriate treatment, earlier intervention and treatment for chronic sinusitis will not only lower the cost for unnecessary medications for asthma control but will also resolve the concomitant sinusitis problem.

A marked increase in allergic diseases has been linked to the hygiene environment in early infancy. Infants exposed to more children at home or day-care experienced less frequent wheeze from year 8 through year 13 and were less likely to have elevated serum IgE levels. Improved hygiene in industrialized societies and the use of vaccine and antibiotics have been reported to reduce the incidence of infections that would normally stimulate the immune system in some way that mitigates against asthma. Whether exposure to endotoxin (lipopolysaccharide), a potent inducer of interleukin-12 and interferon-γ, is protective or harmful is likely to depend on a complex mixture of timing of exposure during the life cycle, environmental cofactors, and genetics. In both animal models and studies in humans, exposure to endotoxin early in life, during the development of the immune system, seems to be most important in providing protection against the development of allergic disease. For older children, the use of antibiotics should not bring about increased allergic diseases.

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References


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**Tracheobronchomalacia and Noninvasive Ventilation?**

*To the Editor:*

I read with great interest the report by Baydur and Kanel (April 2003)1 on seven autopsies that had been performed on patients with Duchenne muscular dystrophy (DMD) who had been long-term users of tracheostomy intermittent positive-pressure ventilation (IPPV). Five of the seven patients had tracheobronchomalacia with areas of dilatation, stenosis, erosions, and bleeding. All patients had scoliosis. One patient had undergone repeated bronchoscopies for secretion management. Two patients had tracheal perforations. One of these two patients had a fatal hemorrhage from a tracheovascular fistula, and one died from ventilator-associated pneumonia. The authors cautioned that patients using noninvasive IPPV might develop the same pressure-related airway changes. However, we think this is unlikely for the following reasons:

1. The greatest damage they reported was in the trachea at the tube opening and then just downstream. The damage was most likely due to the direct contact between the tube and the trachea, and the turbulence of the air entering the wider trachea. Such damage would be much less likely with the more linear flow patterns of breathing or receiving noninvasive IPPV via the upper airway. A similar effect is seen in the destruction of airway cilia by the high flows through the small holes in suction catheters, by comparison with effective suction via the upper airway using mechanical in-exsufflation (MI-E).2
2. In 257 noninvasive IPPV users over a 2,350-patient-year period, no deaths occurred due to airway hemorrhage or any other clinically apparent tracheobronchomalacia.3 Indeed, the same author has now treated > 700 noninvasive IPPV users for > 5,000 patient-years, including 10 patients for 45 to 53 years of continuous noninvasive IPPV at pressures up to 45 cm H2O (for one obese patient with no measurable vital capacity since 1956) with no airways ventilation-associated morbidity.4
3. The facts that five of seven DMD patients experienced morbidity or died from causes associated with tracheostomy, and that in at least two centers tracheostomy has been avoided for > 200 DMD ventilator users with virtually no respiratory morbidity,2,4 argues strongly for avoiding tracheostomy in favor of noninvasive methods rather than cautioning clinicians about the latter.
4. The reason that patient 2 in the study by Baydur and Kanel5 underwent repeated bronchoscopies for the management of secretions was because MI-E was not used via the tube (or via the upper airway) had a tube not been used in the first place. The 34 DMD patients with access to MI-E and noninvasive IPPV for 170 patient-years in one center experienced no pulmonary mortality.2 This also argues for noninvasive IPPV rather than tracheostomy IPPV. Since we have not had to resort to tracheotomy for any DMD patient in > 20 years, we feel strongly that no DMD patient needs one to avoid respiratory mortality.

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