Airway Malacia Disorders in Children

To the Editor:

We read with interest the article by Boogaard et al (November 2005) in CHEST on tracheomalacia (TM) and bronchomalacia (BM) in children. They reported the incidence of those conditions and patient characteristics, and they calculated the predictive value of a clinical diagnosis of airway malacia by pediatric pulmonologists. However, certain points need clarification.

The authors detected airway malacia in 160 children at a median age of 4 years. The age of the oldest malacic patient was 17 years, which seems very old. We recently reported3-43 patients with TM and/or BM, whose median age was 9 months (range, 1.5 months to 5 years). We did not identify any malacic patient older than 5 years of age, although we performed flexible bronchoscopy in patients up to 18 years of age. We wonder what kind of diseases or conditions contributed to the presence of malacia in patients from >5 years of age to adolescence in the series by Boogaard et al.1 The authors calculated the incidence of primary airway malacia based on the annual birth rate; however, this means of calculation taking into consideration only the annual birth rate does not seem reasonable.

In our series,2 we also evaluated the clinical and radiologic characteristics of our malacic patients, and we demonstrated that BM was associated with parenchymal findings more often than TM. In the presence of BM (with TM or alone), wheezing, recurrent or persistent pneumonia, and unilateral persistent atelectasis were common findings. Were there also any clinical and/or radiologic findings in the study of Boogaard et al?1

Boogaard et al1 reported that the diagnosis was confirmed by bronchoscopy in 74% of patients who were suspected of having airway malacia by pediatric pulmonologists. The authors had better detailed information and comments on which to base clinical signs and symptoms that would be predictive for the presence of malacic disorders.

The authors found that most of the patients with isolated airway malacia had no evidence of gastroesophageal reflux (GER) based only on the lipid-laden macrophage index,1 which was not consistent with that in other reports.2,3 They also explained this difference in their study with the exclusion of patients who had associated syndromes or medical conditions (who are at greater risk for aspiration). If GER was investigated not only by the lipid-laden macrophage index but also by other techniques (eg, barium esophagography, 24-h esophageal monitoring, or gastric scintigraphy), they could have found more patients with GER.

We detected GER, which was most commonly seen in those patients with tracheobronchomalacia, in 29% of the patients in our study.2

This may support the occurrence of the highest intrapleural negative pressure in these children than in those with TM or BM alone. Since GER can aggravate malacia symptoms, its treatment is important.2-3 It should also be emphasized that, since therapy with β2-adrenergic bronchodilator agents aggravates airway obstruction by reducing airway muscle tone, their use should be avoided in malacic disorders.4

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REFERENCE


There Is No Evidence To Support the Use of Aerosolized Magnesium for Acute Asthma

To the Editor:

I read with interest the systematic review about the use of aerosolized magnesium in acute asthma recently published in CHEST (July 2005) by Blitz et al.3 The authors concluded that nebulized MgSO4, particularly in addition to a β2-agonist, appears to produce benefits with respect to improved pulmonary function and may reduce hospital admissions. However, the data of this review are far from supporting these conclusions. The facts are as follows:

(1) In agreement with the review data, therapy with MgSO4 with or without a β2-agonist, was superior to therapy with β2-agonist alone (standardized mean difference [SMD], 0.30; 95% confidence interval [CI], 0.05 to 0.55; p = 0.02; five studies) with no between-study heterogeneity. However, a careful inspection of the data shows that pulmonary function values of the study of Bessmerteny et al2 were entered erroneously: in Table 2 and Figure 2 of their study (at 65 min), the mean values for FEV1 (percentage of predicted) are 68 ± 22.5% for the control group (not for the treatment group as appeared in Fig 1 of the review) and 63 ± 20.6% for the MgSO4 group (not for the control group). This mistake changes the results dramatically (Fig 1). Thus, the effect of aerosolized MgSO4 on posttreatment lung function is not superior to therapy with β2-agonists alone (SMD, 0.16; 95% CI, -0.08 to 0.41), with low-to-moderate heterogeneity (I2 = 40.8%). The effect was similar when therapy with β-agonists and MgSO4 was compared with a β-agonist alone (four studies): SMD, 0.21; 95% CI, -0.05 to 0.48; p = 0.11, with low-to-moderate heterogeneity again (I2 = 47.6%).

(2) The Cochrane version4 of this review showed the same data mistake. In an update of May 22, 2005, and as result of a letter sent by me to the authors, they recognized the mistake and corrected the data. Nevertheless, in an inexplicable form, they modified the standard deviation values of another one of the studies included in the review4 (to almost half of original value).

(3) Recently, we have identified a new randomized in-press study5 on the use of nebulized MgSO4 for acute asthma. When this study is included in the analysis, the results remain without modification. Lung function was similar when MgSO4 and β-agonists were compared with a β-agonist alone (SMD, 0.20; 95% CI, -0.05 to 0.45; p = 0.12; I2 = 31.8%; five studies).

(4) The authors of review4 failed to demonstrate a significant reduction of hospital admissions (with nebulized MgSO4 alone or in combination with β-agonists).

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