have previously noted1 that the Swedish study agrees quite well with the data from the Australian Twin Registry.

In conclusion, the findings of the Finnish group are not as incompatible with the results of previous genetic studies of asthma as was suggested in their discussion. It will be interesting to see further analysis of the (unpublished) self-report data on asthma they describe, where sex differences in concordance seem far less marked, possibly because of the higher number of affected subjects due to inclusion of less severe wheezers. A broadening of the case definition of asthma to include intermittent and childhood asthma, although possibly less reliable, is almost always needed in studies of the genetics and epidemiology of asthma in the general population.

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Auto-PEEP Is Favored by Weakness of the Posterior Wall of the Trachea

To the Editor:

The presence of intrinsic positive end-expiratory pressure (PEEP), or auto-PEEP, is now a widely recognized source of problems in the management of patients in the intensive care unit (ICU). It becomes greater when patients with COPD need mechanical ventilation despite the use of long expiratory time, intensive bronchodilator treatment, and the use of partial ventilatory support as early as possible. We report the case of a COPD patient who showed auto-PEEP in spite of the use of the aforementioned strategies and in whom an important role in the increased expiratory resistance could be attributed to dynamic compression of the trachea.

A 70-year-old man was admitted with a history of acute febrile dec ompensation of COPD. His past medical history included gastrectomy for peptic ulcer 20 years previously and COPD due to a long-term smoking habit; the COPD had been treated regularly with continuous home oxygen, corticosteroids, and bronchodilators. Pulmonary function tests showed the following values: FVC, 2.130 ml (54 percent of predicted); FEV1, 740 ml (26 percent); FEF25-75%, 300 ml (14 percent) of predicted. A bronchodilator test was positive. In the previous three months, he had suffered two attacks of severe bronchospasm necessitating hospitalization.

On the day of admission, he suddenly became dyspneic with cyanosis and diaphoresis. Arterial blood gas analysis showed mild hypoxemia and severe hypercapnia with respiratory acidosis (PaO2, 65 mm Hg; PaCO2, 65 mm Hg; and pH, 7.14 with 24 percent inspired O2). After 2 h, his clinical condition and blood gas values had deteriorated (PaO2, 63 mm Hg; PaCO2, 94 mm Hg; and pH, 7.01 with 30 percent inspired O2).

The patient was admitted to the ICU, and noninvasive mechanical ventilation with pressure support via a face mask was instituted with initially good results. After several hours the bronchospasm became aggravated, and endotracheal intubation was required. After vigorous intravenous bronchodilator treatment, wheezes persisted, and tests of lung mechanics revealed an inspiratory airway resistance of 27 cm H2O/L/s, static compliance of 70 ml/cm H2O, and auto-PEEP of 8 cm H2O. Fiberoptic bronchoscopy showed indentation of the posterior wall of the trachea into the lumen during expiration with a critical reduction in airway diameter. External PEEP was added in a stepwise manner under direct vision via the bronchoscope. The inward motion of the posterior wall of the trachea progressively decreased until the maximum reduction was reached at 8 cm H2O (Fig 1). Neither peak and plateau airway pressures nor auto-PEEP increased. The wheezes disappeared, and the capnogram profile showed a reduction in the slope of CO2.

Table 3—Heterogeneity Testing of Finnish and Swedish Data Using Threshold Models Fitted with MX*

<table>
<thead>
<tr>
<th>Twin Group</th>
<th>χ² Fit of ACE Model to Each Group (df)</th>
<th>Heritability from Separate Models</th>
<th>χ² Fit of ACE Model to Combined Data (df)</th>
<th>Heritability from Combined Groups</th>
</tr>
</thead>
<tbody>
<tr>
<td>Finnish males</td>
<td>0.26 (3)</td>
<td>0.00</td>
<td>4.65 (8)</td>
<td>0.34</td>
</tr>
<tr>
<td>Finnish females</td>
<td>2.10 (3)</td>
<td>0.47</td>
<td>13.56 (13)</td>
<td>0.55</td>
</tr>
<tr>
<td>Swedish twins†</td>
<td>3.62 (3)</td>
<td>0.63</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*ACE model comprises additive genetic, shared, and unshared environmental sources of variation.
†Using contingency tables reconstructed from Tables 3.1 and 3.2.1 in reference 15.
elimination from 5 to 2 mm Hg/s. The patient died three days later of septic shock caused by Pseudomonas pneumonia.

Auto-PEEP is attributed to high minute ventilation, short expiratory time, small diameter of endotracheal tubes, and, more often than not, elevated airway resistance. It is normally a function of small airway tone, mucus plugging, and dynamic collapse. While bronchomalacia is a common disorder in COPD patients, the weakness of the posterior wall of the trachea to such an elevated degree is exceptional. Moreover, to our knowledge it has not been described as a source of this amount of auto-PEEP. The response to external PEEP suggests an internal pneumatic stabilization component.2

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Foreign Bodies in the Tracheobronchial Tree

To the Editor:

In the August 1991 issue of Chest, Drs Marik and Ballhausen1 described a patient who aspirated a mercury thermometer into the right lower bronchus. The authors stated that they were unaware of any earlier similar reports. I wish to bring to the authors' attention a nearly identical case described in the article reporting on our experience with 66 foreign bodies in the tracheobronchial tree.' Figure 3 in that article is remarkably similar to the one shown in the communication by Drs Marik and Ballhausen. There is, however, one noteworthy difference in the management of our patients: We elected to extract the thermometer through a bronchotomy, rather than resecting the lobe, thus saving the normal lung parenchyma. Among our 66 patients, there were only six instances in which resection of lung tissue was necessary (four lobectomies, two segmentectomies); the resections were done only in cases of a long-standing obstruction (up to 12 years in case 2), with destruction of the distal parenchyma.

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Alternation between Atrial Flutter and Atrial Fibrillation

To the Editor:

I read with interest the article by Tunick et al., which appeared in the January 1992 issue of Chest. The frequency of coexistence of atrial flutter in patients with atrial fibrillation, especially in those with periods of sinus rhythm (20/24 [83 percent]), and its significant association with the use of a type 1A antiarrhythmic drug are not unexpected.

In 1956 I reported2 that atrial flutter was a frequent finding (51 percent) in 115 patients with atrial fibrillation during administration of quinidine, a type 1A antiarrhythmic agent. The more frequently the electrocardiograms were recorded during quinidin therapy, the more frequently the atrial flutter was observed. It is therefore not surprising that a much higher incidence was seen with Holter monitor recording, as reported by Tunick et al. After all, the two arrhythmias share a similar macro-reentrant mechanism, fibrillation being due to random reentry and flutter being due to nonrandom localized reentry.

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