A Mechanical Model of Auto-PEEP

To the Editor:

I read with interest in the September 1989 issue of Chest the editorial by Tobin and Lodato¹ about auto-PEEP. Since auto-PEEP is a common complication in mechanically ventilated patients, it is important to understand the pathophysiologic mechanisms involved in the development of this condition.

I have assembled a simple mechanical model that simulates quite well the development of auto-PEEP, produced either by dynamic hyperinflation or by flow limitation due to airway collapse.² The model consists of a test lung connected to a ventilator via an airway. The airway, made of conventional ventilator tubing, has an interposed mushroom expiratory valve connected to a water manometer. The application of pressure on this valve creates the effect of airway collapse. Pressure transducers are located proximal and distal to this valve.

When no pressure is applied to the expiratory valve and ventilator settings are chosen to critically decrease the expiratory time, a pressure gradient is generated between the proximal and distal pressure transducers at end expiration (dynamic hyperinflation). The higher distal pressure is detected only in the proximal transducer when an end-expiratory hold maneuver³ is performed and pressure equilibration occurs in the system. When expiratory time is prolonged and pressure is applied to the expiratory valve, a pressure gradient is also created at end expiration between the proximal and distal ends of the system (airway collapse). The higher distal airway pressure cannot be sensed by the proximal expiratory hold maneuver is performed. Therefore, this model predicts that when airway collapse exists, the end-expiratory hold maneuver may not give a good estimate of auto-PEEP. The application of external PEEP to the system when “airway collapse” is produced reduces the proximal-distal pressure gradient by diminishing airway collapse. This is done without increasing the test lung volume, as long as the external PEEP does not reach the auto-PEEP value.

The results obtained with this model support the judicious application of external PEEP in patients with airflow limitation and may be of help in understanding the pathophysiology of air-trapping mechanisms and auto-PEEP.

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Influence of Platelet-derived Microparticles on Coagulation in a Lung Cancer Patient Receiving Chemotherapy

To the Editor:

Coagulation abnormalities have been reported in patients with a variety of cancers.¹ In particular, Ruiz et al² investigated the influence of chemotherapy on coagulation and fibrinolysis in lung cancer patients, and found a decrease in fibrinolytic activity that was related to an increase in thromboembolic complications after chemotherapy. Sims et al³ have reported that platelet-derived microparticles (MPs) may play a role in the normal hemostatic response to vascular injury, since prothrombinase activity is exhibited on these particles.

We recently treated a 68-year-old man with small cell carcinoma of the lung using modified CODE therapy (cisplatin, 25 mg/m²; vincristine, 1 mg/m²; adriamycin, 30 mg/m²; etoposide, 100 mg/m²; and recombinant human granulocyte-colony stimulating factor, 75 µg). We measured MPs and several coagulation parameters (thrombin-antithrombin III complexes [TAT]; fibrinopeptide A [FPA], and E-type fibrin-fibrinogen degradation products [FDPE]) on 16 occasions in this patient. Microparticles were detected with the use of flow cytometry.⁴⁵

The mean (±SD) levels of MPs, FDPE, TAT, and FPA were 28.4 ± 11.3 percent (normal, 6.8 to 16.4 percent), 131 ± 29 ng/ml (normal, 0 to 70 ng/ml), 8.9 ± 6.2 µg/l (normal, 1.5 to 2.5 µg/l), and 10.5 ± 8.7 ng/ml (normal, 0 to 2 ng/ml), respectively. Thus, the levels of both MPs and the coagulation parameters were markedly raised in this patient. Furthermore, we found a significant difference in the TAT levels between the 6 occasions when the MP level was 30 percent or more (14.0 ± 5.7 µg/ml) and the 10 occasions when the MP level was less than 30 percent (7.3 ± 4.1 µg/ml) (p<0.001), although there was no significant correlation between the actual TAT and MP levels. The same result was obtained for FPA (p<0.01), but not for FDPE.

The functional significance of platelet-derived MPs is of considerable interest, because they are rich in membrane receptors for

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coagulation factor Va and provide a catalytic surface for the prothrombinase reaction. We observed that TAT and FPA were both increased in a lung cancer patient with a high level of MPs. Thus, platelet-derived MP may influence coagulation in lung cancer patients receiving chemotherapy.

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Closure of a Tracheoesophageal Fistula by Bronchoscopic Application of Tissue Glue

To the Editor:

We read with interest in the August 1991 issue of Chest the article by Antonelli et al regarding the closure of a tracheoesophageal fistula by bronchoscopic application of fibrin glue.

We would like to report briefly another case of tracheoesophageal fistula closed by means of a bronchoscopic procedure.

A 17-year-old boy was admitted to the ICU in October 1990 after a car accident. Flail chest, multiple bone fractures, and a brain concussion were noted. He was intubated, and mechanical ventilation was started. Twelve days later the patient underwent a tracheostomy for long-term respiratory assistance. Thirty-two days after the tracheostomy, the patient had recovered completely, and successful weaning from the ventilator permitted oral feeding instead of enteral feeding by nasogastric tube.

Immediately after the first fluid ingestion the patient experienced a severe cough, which recurred after each fluid intake. A Gastrografin swallow study showed a communication between the superior third of the esophagus and the trachea. A bronchofiberscopic attempt was made to close the fistula with tissue glue (N-buty1-2-cyanoacrylate [Histoacryl, Braun Melsungen, Germany]). The technique described by Roksvagg et al for closing a bronchial fistula was used. Twenty-four hours before the procedure the patient gargled with distilled water with 5 percent Betadine every 15 min. Atropine was given by intravenous instillation until the mouth was completely dry, beginning 1 h before the procedure and continuing 2 h afterward. Cardiac monitoring was performed throughout the same period. The bronchofiberscopic procedure was performed under local anesthesia. The tissue glue was applied to the fistula under direct vision; 2 ml of Histoacryl was used.

After the procedure the patient received nothing by mouth for 2 days. On the third day a second bronchofiberscopic procedure was done, which demonstrated complete visual obliteration of the fistula by the glue. A Gastrografin swallow showed no more communication between the esophagus and the trachea. On the same day fluid intake was permitted, and the patient did not cough. Six months of follow-up showed no recurrence of the fistula.

Histoacryl was chosen because of its fast solidification (10 to 30 s) and its associated inflammatory reaction, which enhances fibrosis with formation of a foreign-body resorptive granuloma. Fistula closure is rapid with Histoacryl, and as a safety measure, 3 days was allowed for complete fistula closure before the patient resumed oral feeding. To obtain good results, the tracheal and the esophageal mucosa must be clean, noninfected, and dry; for this reason, we used Betadine for decontamination and atropine for mucosal dryness.

This is believed to be the first report of use of Histoacryl for closing a tracheoesophageal fistula, sparing the patient a major surgical procedure and providing a very good result in a few days' time. In our opinion, bronchofiberscopic closure of tracheoesophageal fistula using tissue glue has to be tried before any surgical repair since it is less aggressive and less costly.

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Serologic Response to Itraconazole in Allergic Bronchopulmonary Aspergillosis

To the Editor:

In the excellent report by Denning et al., which appeared in the September 1991 issue of Chest, oral itraconazole (200 mg daily for a mean of 3.9 months), used as an adjunctive therapy in six patients with allergic bronchopulmonary aspergillosis (ABPA), was effective in improving the clinical (corticosteroid requirements), serologic (total serum immunoglobulin [Ig] E level), and pulmonary functional status. However, they found no significant effect on Aspergillus-specific IgG (IgG-Af), which could be due to the fact, mentioned by the authors, that the immunodiffusion technique they used is qualitative rather than quantitative. Moreover, they state that there are no reports in the literature documenting any consistent relationship between fluctuation in an individual patient's IgG-Af level and disease activity.

We have one experience in which IgG-Af was the only serologic parameter showing improvement with oral itraconazole. The patient was a 36-year-old woman who met the diagnostic criteria for ABPA proposed by Rickett et al. She was in the corticosteroid-dependent stage of ABPA and had had a mean of two exacerbations of asthma per year in spite of daily treatment with a mean of 20 mg of prednisone. After informed consent, a therapeutic trial of oral itraconazole, 200 mg/d for 4 months, was instituted. Along with a corticosteroid-sparing effect and an increase in spirometric values