Late complications of collapse therapy generally present as pyogenic, caseous, or mycotic empyema at the site of the previous pneumothorax. Hemorrhagic effusion associated with pleural malignant change has been reported. To our knowledge, extrapleural hematoma without malignancy has not yet been described.

Our patient's medical history excluded external violence or traumatic thoracotensis. Although prothrombin values were within the therapeutic range, transient overanticoagulation most probably enhanced pleural bleeding. We believe that pleuropulmonary decortication was the appropriate treatment, suppressing the pocket that might have become infected by repeated centesis or tube thoracostomy and allowing full pulmonary expansion.

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Increase of Intraocular Pressure during Nasal CPAP

To the Editor:

Nasal continuous positive airway pressure (CPAP) was first described by Sullivan et al and has proved to be of great value for the treatment of patients with obstructive sleep apnea (OSA). The therapeutic benefit of nasal CPAP is due to its good results and security, with very few complications described. To our knowledge, secondary increase of intraocular pressure during treatment with nasal CPAP has not been previously reported in the literature. We present the case of a patient with OSA and glaucoma in whom the intraocular pressure increased during nasal CPAP treatment.

A 62-year-old obese man was studied because of daytime sleepiness, loud snoring, and nocturnal arousals with apnea episodes. Glaucoma in the right eye had been diagnosed, and he had received the pertinent treatment. Respiratory functional testing revealed a moderate obstructive ventilatory deficit (FVC, 82 percent; FEV, 56 percent; FEV/FVC, 55 percent). Values obtained at blood gas analysis were as follows: PaO, 65 mm Hg; PaCO, 46 mm Hg; pH, 7.36. All-night polysomnography demonstrated severe OSA, with an apnea-hypopnea index of 40. Treatment with nasal CPAP was initiated at a pressure of 3 cm H2O and was then increased on the following nights until a pressure of 10 cm H2O was reached. The patient reported pain in the glaucomatous eye. The intraocular pressure in both eyes was measured at baseline and after 15 min of nasal CPAP at 10 cm H2O. An increase of 7 mm Hg in intraocular pressure in the glaucomatous eye was documented; no changes appeared in the healthy eye.

Nasal CPAP produces an increase in pressure in the oropharynx, keeping the airway open during sleep. The pressure of nasal CPAP can be transmitted through the esophagus and has been useful in reduction of gastroesophageal reflux. The relation between intrathoracic and intracranial pressure has also been studied in OSA. Transmission of intrathoracic pressure to the intracranial space occurs through the venous system or directly to the cerebrospinal fluid space via the thoracic vertebral foramina. Impairment of cerebral blood outflow across valveless veins and an increase in cerebrospinal fluid pressure can increase cerebral venous and intracranial pressures. The increase in intraocular pressure might be due to a similar pathogenetic mechanism associated with failure of the diseased eye to regulate the pressure. To our knowledge, no other published studies corroborate our findings, but it seems that nasal CPAP treatment increases intraocular pressure in patients with glaucoma, which suggests that glaucoma should be considered a relative contraindication for nasal CPAP. Further evidence is needed to support this possibility.

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Therapeutic Pulmonary Artery Catheterization

To the Editor:

I read with interest in the June 1991 issue of Chest the article by Steinbrug et al on therapeutic pulmonary artery catheterization. It is curious that groups 1 and 2, for whom the reviewers were "uncomfortable with care," had the lowest mortality (although not statistically significant) compared with the "comfortable with care" and "optimal management" groups. Why is this so? Does it imply that therapy makes no difference in chance of survival in these critically ill patients? Rather, I think it implies that (1) we need a better idea of what it is we measure and should measure; and (2) we need controlled studies to compare different therapies to determine optimal management.

First we may ask what does pulmonary artery wedge pressure (PAWP) measure? It is commonly used as a surrogate for fluid status. Yet Shipp et al showed that there is a poor correlation between PAWP and measured blood volume. Schuster and Haller showed that there is a poor correlation between PAWP and extravascular lung water. There was a better correlation when capillary permeability was included.

More important than cardiac output is oxygen delivery (the product of cardiac output, hemoglobin concentration, and oxyhe-