Late complications of collapse therapy generally present as pyogenic, caseous, or myotic 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 thoracentesis. Although prothrombin values were within the therapeutic range, transient overanticoagulation most probably enhanced pleural bleeding. We believe that pleuropleural 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 potential 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 defect (FVC, 82 percent; FEV1, 56 percent; FEV1/FVC, 55 percent). Values obtained at blood gas analysis were as follows: PO2, 65 mm Hg; PCO2, 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 Steinigrub 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 Shippy 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-
moglobin saturation) and its relationship to oxygen demand. For example, a high cardiac output, 8 L/min, will have less DO₂ than a low cardiac output, 4 L/min, when the hemoglobin concentrations are 8 and 15 g/dl and the SaO₂ values are 90 percent and 100 percent, respectively. But how much DO₂ is necessary? Does it depend on VO₂? Does it depend on need? Is gastric tonometry an answer? Perhaps by explicitly recording oxygen delivery instead of cardiac output, there would be a more proper emphasis on this more important variable.

Previous studies have shown that physicians are poor predictors of central hemodynamics. However, does therapy based on central hemodynamics rather than on clinical status improve outcome? If based on central hemodynamics, what therapy should be used? Studies have shown that therapy aimed to achieve supranormal values improves outcome. Rather than doing studies to show that pulmonary artery catheterization affects therapy, randomized studies, such as Guyatt et al. are doing, comparing pulmonary artery catheterization with clinical judgment, different therapies, or different therapeutic goals should be conducted to see whether they affect outcome.

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To the Editor:

We have reviewed the comments by Dr Engoren and share his thoughts regarding the need for prospective studies to compare different therapeutic goals. As to his remarks concerning suboptimal performance classification and associated lowest mortality observed in groups 1 and 2, the review panel of critical care physicians concluded that these cases did not follow any logical management pattern that fit with the initial pathophysiologic measurements obtained. This may imply that pulmonary artery catheterization (PAC) information was ignored or distrusted, both of which are not necessarily unusual in patients with complicated conditions. Furthermore, this analysis was never intended to be an outcome study in a population whose mortality rates are high; we attempted only to determine how PAC data are interpreted and how information is used. The selected case-mix variables were chosen to eliminate other factors that may have affected the performance grade.

Undoubtedly, PAC has clearly provided insight into the metabolic and hemodynamic disturbances of septic shock. Measurements of cardiac output, blood oxygen consumption, and lactate do play a role in managing sepsis syndrome. Furthermore, measurement of PAWP may help to differentiate between hydrostatic and pulmonary edema of the lung and thus facilitate fluid management. It remains less clear, however, whether PAC should be performed in every patient with sepsis syndrome. The impact of invasive monitoring and its measurements of physiologic variables in sepsis syndrome are not well defined and perhaps only favorably affects survival if infection is eradicated. The prognostic significance of hemodynamic and metabolic disturbances in sepsis remains controversial. Which parameter should be monitored and treated in order to improve outcome? The prognostic value of changes in oxygen uptake and the response to oxygen delivery remains unclear in different clinical situations. Although several studies suggest that oxygen uptake is higher in survivors than in nonsurvivors, other studies do not support this view.

We agree with Dr Engoren that catheterization is unlikely to influence prognosis until more effective therapy is available. A strict treatment protocol based on standardization of physician skills and hemodynamically derived data accumulation is critical in any study to assist in defining efficacy of technique. The question of how capable certain physicians are in planning therapy without catheterization remains to be answered. Are there some who can do it as well without invasive monitoring? Certainly, there are subgroups of patients in whom PAC reveals otherwise unobtainable information. Further work is needed in large samples of patients with emphasis on outcome of clinical importance. An additional study to distinguish those patients whose hemodynamic status can be reliably predicted noninvasively from those whose hemodynamic status is unpredictable is warranted, the latter patients being more likely to benefit from invasive hemodynamic monitoring.

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