Communications for this section will be published as space and priorities permit. The comments should not exceed 350 words in length, with a maximum of five references; one figure or table can be printed. Exceptions may occur under particular circumstances. Contributions may include comments on articles published in this periodical, or they may be reports of unique educational character. Specific permission to publish should be cited in a covering letter or appended as a postscript.

Postpneumonectomy Pulmonary Edema
What's the Cause?

To the Editor:

We appreciate the succinct summary of the pathophysiologic factors contributing to the development of postpneumonectomy pulmonary edema (PPE) in the editorial by Mathru and Blakeman that accompanied our recent article concerning this complication of pneumonectomy. However, we wish to respond to several of their comments, which are important in understanding PPE and applying the results of our study in clinical practice. The editorial writers correctly summarized the results of our study as finding no significant difference between the amount of fluid administered to the patients who developed PPE and those who did not. However, we did not state that any patient received an "excess" of fluid. We actually made the contrary point: patients who developed PPE did not receive an excess of fluid when compared with patients who developed PPE. We also believe the mean ± SD of the fluid received by all patients (about 1,500 to 3,500 ml) is well within reason for the length of operation and degree of blood loss associated with a pneumonectomy; indeed, it would probably be considered too low for other general thoracic operations. The clinical correlate of this observation is that a patient undergoing pneumonectomy may still develop PPE in spite of efforts to keep the lung "dry." The medicolegal application is that if PPE occurs, the patient's physicians cannot automatically be assumed to have "flooded" the remaining lung. The additional observation we made was that intensive treatment with positive pressure ventilation, fluid restriction, and diuretics did not decrease the pulmonary edema and resulted in eventual discharge of the patient from the hospital—the patient died anyway, usually with worsening pulmonary compliance and gas exchange. This lack of response to treatment for a "drowned" lung provides further evidence that fluid balance alone is not responsible for PPE.

We agree with Drs. Mathru and Blakeman that fluid administration regimens tend to be rather nebulous, and that fluid therapy guided by invasive hemodynamic monitoring would be much more satisfactory. They cite a study by Wittnich et al indicating that the pulmonary artery wedge pressure may be falsely low after pneumonectomy. We were aware of this study at the time we cared for the two patients who inspired our interest in this topic. We both obtained pulmonary artery occlusion pressure tracings on multiple occasions from these patients and observed no decline in the radial artery pressure during pulmonary artery catheter balloon inflation. We, therefore, cannot confirm the findings of Wittnich et al, but we agree their results should be considered in any interpretation of hemodynamic data from pneumonectomy patients.

We continue to believe pneumonectomy patients should receive the "just right" amount of fluid: the volume of fluid necessary to maintain adequate tissue perfusion. What constitutes "just right" varies among patients, but generally can be guided by observation of the vital signs, urine output, record of fluid intake/output, and daily weight. In appropriate patients, invasive hemodynamic monitoring may help guide fluid administration decisions; however, the physician should consider the additional effort required to position a pulmonary artery catheter in the nonoperative pulmonary artery and the potential risk for disruption of the catheter during the operative procedure. Further, the editorial writers cited the work of Little et al indicating dogs who underwent pneumonectomy were more prone to pulmonary edema with fluid administration. Later work done by the same group, however, showed pneumonectomized dogs did not develop excess extravascular lung water at normal left atrial pressures if the hematocrit was maintained constant. It required raising the left atrial pressure before the dogs in this study developed a significant increase in extravascular lung water.

We trust readers of Chest will agree our data support our conclusion that PPE is not merely a fluid overload problem. We agree with Drs. Mathru and Blakeman that much further basic laboratory and clinical inquiry is needed to better define the fundamental pathophysiology of this complication of pneumonectomy. We strongly agree with their recommendation to educate physicians about the seriousness of PPE so patients exhibiting any sign of dyspnea and early pulmonary infiltrate after pneumonectomy will receive appropriate supportive treatment as early as possible. While we gain a more sophisticated understanding of PPE, we continue to advise administering a sufficient quantity of fluid to maintain vital organ perfusion and appropriate monitoring of fluid therapy. Most importantly, we recognize that if PPE should develop, it is not necessarily a function of excess fluid and measures to treat it as such will likely meet with limited success. While some degree of fluid restriction may limit the amount of pulmonary edema formation in PPE, definitive treatment will require eliminating the cause of the abnormal capillary permeability described by Mathru et al.

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Neurologic Disease Requiring Long-term Ventilation

To the Editor:

I read with interest the recent article by Hammond and Potgieter concerning the use of selective decontamination of the digestive tract (SDD) in patients with neurologic disease requiring prolonged mechanical ventilation. Their conclusion that SDD was of no benefit in reducing secondary infections in these patients was interesting, but to some extent misleading. In an apparent effort to suggest that their group of patients may represent a unique subset who may not benefit from SDD, they compare their findings with four previous studies all of which showed a significant reduction in the incidence of secondary infection in a larger group of patients with various disease states. While this comparison may have some merit, the authors fail to mention that an earlier study that they coauthored (in which the present group of patients with neurologic disease was included) failed to show any benefit of SDD in reducing the incidence of secondary infections.

While patients with neurologic disease may not benefit from SDD, the comparison of the present study with other studies that involved different patient populations, SDD regimens, infection control policies, and inclusion criteria should be scrutinized carefully. Would it not have been better for the authors to compare the subset of patients with neurologic disease with the remainder of their larger population, rather than retrospectively with other studies over which they had no control?

The issues addressed by Drs. Hammond and Potgieter are indeed important, as the proper role of SDD remains controversial. The usefulness of SDD, however, in patients with neurologic disease probably remains unanswered. A larger, more indepth study is probably required to determine the true effectiveness of SDD in patients with neurologic disease and to determine if these patients do indeed represent a population that is refractory to the benefits of SDD.

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