Controlling Postoperative ARDS

To the Editor:

I believe that there may be an undue incidence of post-operative ARDS in patients previously treated with bleomycin, as indicated in the article by Gilson and Sahn (Chest 1985; 88:304-06), even without preoperative pulmonary toxicity.1 I do not, however, feel that oxygen can be held responsible when other important stresses are present in these patients. Even the evidence presented by those authors highlights alternate factors.

Specifically, Goldiner et al1 compared five such patients who died from post-operative ARDS to the next 12 patients, who benefited from meticulous invasive hemodynamic monitoring. Notably, as a result of this monitoring, the positive fluid balance in survivors was significantly less than that in non-survivors by a factor of 2.5. Damaged lungs are leakier than healthy lungs,2 and it is possible that bleomycin does cause sub-clinical lung injury. Increases in filtration pressure, even within the “normal” range, readily increase lung water volume in such lungs. Since an additional 1 to 2 L of infused volume may significantly increase pulmonary vascular filtration pressure, it is likely that intraoperative hydrostatic pulmonary edemagenesis contributed to ARDS. Neither Gilson and Sahn nor Goldiner2 includes intraoperative hemodynamics or transfusion history in their reports, both of which may be important in defining the cause of ARDS.

While oxygen may have played a causative role in the reported cases, I think one would be myopic to avoid considering other possible causes of ARDS in these patients. Certainly, it seems prudent to use just enough oxygen to maintain hemoglobin saturation, but arbitrary limits to FIO2 cannot be established on the basis of current evidence.

Alan C. Jasper, M.D., F.C.C.P.,
Director, Respiratory Care Unit,
Cedars-Sinai Medical Center,
Los Angeles

REFERENCES

To the Editor:

It was not our intent to imply that oxygen inspired in low fractions is the only factor responsible for ARDS in patients previously administered bleomycin, but it is clearly the most critical.1 Certainly, elevations in hydrostatic pressure in abnormal pulmonary capillaries can lead to leak, which would not occur in a normal capillary bed; therefore, judicious fluid administration is necessary in these patients. However, clinical data from the literature2 supports the occurrence of injury resulting from clinically non-toxic concentrations of oxygen potentiated by bleomycin in lung tissue with a latent period of 48 to 120 hours before the development of the adult respiratory distress syndrome (ARDS). Experimental data also substantiate the clinical observations.4 In the present case,1 the patient was euvolemic in the immediate postoperative period and over the next 2½ days prior to the development of ARDS. Clinically, he was asymptomatic, with room air arterial oxygen tension level of 75mm Hg. Sixty hours following surgery, he developed the insidious onset of dyspnea and high fever. Pulmonary capillary wedge pressure at the time of decapsulation was 3mm Hg. Similarly, in Goldiner’s paper,2 five patients developed respiratory distress three to five days following surgery and, at autopsy, had evidence of interstitial pneumonia, fibrosis, and destructive changes in alveolar walls and capillaries; hemodynamics were not commented upon. The latent period from the time of surgery to ARDS in these six cases would support an insult from oxygen radicals, possibly exacerbated by positive fluid balance in Goldiner’s patients, rather than volume overload as the primary factor.

Furthermore, we did not suggest arbitrary limits of FIO2 but simply recommended the use of ambient air or the lowest FIO2 to maintain adequate tissue oxygenation. To consider overzealous fluid administration as the critical factor in this syndrome, and not as a contributing factor following reactivation of bleomycin-oxygen toxicity, would be hyperopic.

Steven A. Sahn, M.D., F.C.C.P.;
Allen J. Gilson, M.D.,
Division of Pulmonary and Critical Care Medicine,
Medical University of South Carolina,
Charleston

Reprint requests: Dr. Sahn, Pulmonary and Critical Care Medicine,
Medical University of South Carolina, Charleston 29425

REFERENCES
5 Tryka AF, Skorinka WA, Godleski JJ, Brain JD. Potentiation of bleomycin-induced lung injury by exposure to 70% oxygen. Am Rev Respir Dis 1982; 126:1074-79