small decrease in systemic vascular resistance. In selected patients with myocardial dysfunction, this might well be expected to result in greater increases in right atrial than left atrial mean pressure.

Much of the interest in the use of PEEP during procedures performed on patients in the sitting position is derived from the initial hope that the incidence of air entrainment would be reduced. There have been no investigations of PEEP that have used the incidence of confirmed VAE as an endpoint. However, there is substantial evidence, some of which is cited by Zaslow et al., that hemodynamically acceptable levels of PEEP will not produce positive venous pressure at a cranial operative site. Accordingly, the prevention of venous air entrainment in patients in the sitting position is not a rationale for the use of PEEP, which should be viewed as firmly established.

The hemodynamic effects of PEEP, when applied in patients in the sitting position, may also be a matter of clinical significance. The effects of 10 cm of PEEP on mean arterial pressure (MAP) and cardiac output (CO) were negligible in the population studied by Zaslow et al. However, others have reported substantially greater alterations. For example, Perkins et al. observed reductions in MAP and CO of 14% and 15%, respectively. The differences may be related to the age of the patient population (not specified in either report) or to anesthetic technique. It follows that in some patients, hemodynamic changes may be a significant limitation to the use of PEEP.

My review of the available literature leads me to doubt that PEEP should be employed routinely during procedures performed in patients in the sitting position. It should probably be withheld for situations where there are specific pulmonary indications for its use. However, when the preoperative evaluation suggests the probable need for intraoperative PEEP, anesthesiologists at our institution are likely to approach their surgical colleagues about the feasibility of alternatives to the sitting position.

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In Reply.—We appreciate Dr. Drummond’s thoughtful comments concerning the safety and efficacy of PEEP during surgery in patients in the sitting position. We believe our article adequately addressed the issue of safety. In our study, 10 cm H$_2$O of PEEP increased both right atrial pressure (RAP) and left atrial pressure (LAP), but did not result in a positive RAP-LAP gradient in any of the 20 patients. Similarly, 10 cm H$_2$O of PEEP did not significantly affect the magnitude by which LAP exceeded RAP. These results are consistent with our prior study in dogs, which demonstrated no effect of PEEP on the LAP-RAP gradient either before or during venous air embolism. Our results differ from those of Perkins and Bedford, and we considered possible reasons for this discrepancy. The Perkins-Pearson et al. study referred to by Dr. Drummond did not investigate the effects of PEEP.

We agree that maneuvers that dynamically alter airway pressure can reverse the normally positive LAP-RAP gradient. This does not imply that 10 cm H$_2$O of PEEP will have a similar effect. Multiple studies have used contrast echocardiography to demonstrate paradoxical movement of microbubbles during or following the release of the strain phase of a Valsalva maneuver. The magnitude and timing of the airway pressure changes that occur are dramatically different from the changes that occur with 10 cm H$_2$O of PEEP. The study by Dr. Cucchiara et al. demonstrated paradoxical movement of microbubbles when 20 cm H$_2$O positive airway pressure was held for 5 s and released. As we discussed in our article, 20 cm H$_2$O of PEEP may be sufficient to reverse the direction of the LAP-RAP gradient in some patients.

We examined the effects of ventilation on RAP, LAP, and the LAP-RAP gradient during and 10 cm H$_2$O PEEP in the 20 patients in our study. Phasic positive pressure ventilation increased LAP at least as much as RAP, and the effect on the instantaneous LAP-RAP gradient did not change with the application of PEEP. We did not examine the transient effects of removal of PEEP on the LAP-RAP gradient in our clinical study. However, in our dog study, we continuously monitored LAP and RAP and observed no transient changes in the LAP-RAP gradient with either the application or removal of PEEP. We therefore believe that 10 cm H$_2$O of PEEP will not predispose to paradoxical air embolism during venous air embolism.

Dr. Drummond questions the effects of PEEP on systemic hemodynamics and systemic and pulmonary vascular resistances. The small effects of low levels of PEEP on systemic hemodynamics are primarily due to decreased venous return and can be reversed if necessary by fluid administration. High levels of PEEP can increase pulmonary vascular resistance, but this is a relatively minor phenomenon at low levels of PEEP. In our study, PEEP increased PVR an average of only 58 dynes · sec · cm$^{-5}$ (0.72 mmHg · 1$^{-1}$ · min$^{-1}$), a value unlikely to pro-

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duce right ventricular dysfunction. Patients with pre-existing myocardial dysfunction generally have a large LAP-RAP gradient, so that PEEP would be unlikely to provoke paradoxical movement of air.

Our study addressed only the safety and not the efficacy of PEEP for patients in the supine and seated positions. PEEP should be effective in decreasing venous air embolism during head and neck or pelvic procedures when the operative site is above the right atrium. It is not yet established whether PEEP is effective in decreasing venous air embolism during neurosurgical procedures in patients in the seated position. We do not advocate the routine use of PEEP during procedures with risk for venous air embolism. On the basis of our clinical and experimental studies, we believe that the application of up to 10 cm H2O of PEEP when venous air embolism does occur is not hazardous. Although data exist that suggest that such application of PEEP may decrease air embolism and aid in detecting its source, additional studies are required to determine the role of PEEP in such situations.

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Carboxyhemoglobin and P50 in Pregnancy

To the Editor—We have several comments regarding the report describing elevated carboxyhemoglobin levels as a mechanism for a decrease in P50 in preeclamptic women.1 First, the authors did not sufficiently specify the two groups of women compared in their study. For example, they did not mention whether these women were from the same geographic area. A national survey in North America found that 1–2% carboxyhemoglobin levels are not unusual in urban non-smokers as a result of environmental exposure.2 In addition, one should consider that even passive smoking can lead to elevated carboxyhemoglobin levels in nonsmokers. Second, and of greater importance, is that a decrease in P50 of 5.7 mmHg in preeclamptic women compared with normal pregnant women can hardly be explained by an increase in carboxyhemoglobin level of 2.1%. The average influence of carboxyhemoglobin on P50 can be estimated using the formula dP50/dCOHb = –0.27.3 This would account only for a 0.57 mmHg decrease in P50. In other words, the cause of 90% of the difference in P50 remains unexplained. Finally, when discussing shifts of the oxyhemoglobin dissociation curve during standardized conditions (pH = 7.4; pCO2 = 40 mmHg), the concentration of intraerythrocytic 2,3-DPG should always be stated.


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