Of Venous Air Embolism, Aminophylline, and Volatile Agents

To the Editor—In a recent Letter to the Editor, Hills and Butler\(^1\) raised the issue of "spill-over" of venous air embolism into the arterial circulation via the pulmonary vascular bed (as opposed to transit via a probe patent foramen ovale\(^2\)). Citing their own work,\(^3,4\) they state that the lungs are normally a highly efficient trap for air that enters the pulmonary circulation but that this function is impaired in the presence of oxygen toxicity and can be overwhelmed by large volumes of venous air. However, their studies may contain additional implications for anesthesiologists which are not mentioned in their letter. They demonstrated\(^5\) that aminophylline, a presumed pulmonary vasodilator, dramatically reduced the threshold for spill-over in pentobarbital-anesthetized dogs. Air was consistently (four of four) detected in the femoral artery of aminophylline pre-treated animals after the injection of less than 0.01 to 0.05 ml/kg of air into the right ventricle, but never (eight of eight) observed after comparable injections in non pre-treated dogs. Larger volumes of air were administered in a single non-pre-treated animal and air was not detected in the systemic circulation until injectate volume exceeded 2 ml/kg. This begs the question: Will the use of anesthetic agents such as enflurane, halothane, and isoflurane which have a pulmonary vasodilating effect,\(^6\) enhance the transit of venous air to the systemic circulation and increase the risk of coronary and cerebral insult after episodes of venous air embolization? And if the volatile agents do augment spill-over, does an agent that inhibits hypoxic pulmonary vasoconstriction (e.g., isoflurane\(^6\)) augment this effect to a greater extent than agents that do not (e.g., enflurane, halothane\(^6\))?

I raise these questions in this forum in the hope that they will come to the attention of an investigator with the opportunity to pursue them. In the interim, I feel that these questions are sufficiently speculative (because of the limited extent of the pulmonary vasodilating effect of volatile agents\(^6\)) that they do not justify any change in the anesthetic practices of those who would otherwise feel that volatile agents were the most suitable in surgical circumstances where venous air embolism might potentially occur. The observations of Butler and Hills\(^3\) do, however, suggest that the risk of systematic air embolization may be greater in patients receiving aminophylline. In those few situations where aminophylline administration and venous air embolism risk coincide, positions which minimize that risk (lateral, prone) and a more aggressive response to minor embolic events may be appropriate.

John C. Drummond, M.D.
Assistant Clinical Professor of Anesthesiology
Department of Anesthesiology
University of California, San Diego
La Jolla, California 92093

REFERENCES

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Tachycardia and Mitral Valve Disease

To the Editor—A recent clinical report by Kopman\(^1\) describes the use of intravenous verapamil for control of acutely accelerated ventricular responses to atrial fibrillation in two patients with mitral valve disease. One patient with moderately severe mitral stenosis became acutely dyspneic prior to the induction of anesthesia. At that time the patient was in atrial fibrillation with a ventricular rate of 190 beats/min and the pulmonary capillary wedge pressure (PCWP) was 55 mmHg. Five milligrams of intravenous verapamil produced the expected clinical and hemodynamic improvement concurrent with a decrease in the ventricular response to 75 beats/min. Tachycardia is detrimental to patients with mitral stenosis because the atrioventricular pressure gradient is proportional to the