

Air Embolism: Further Basic Facts Relevant to the Placement of Central Venous Catheters and Doppler Monitors

To the Editor:—In an editorial about a year ago, Michenfelder¹ addressed the very important question of the placement of central venous catheters before neurosurgery in patients at risk for venous air embolization. The advent of the precordial Doppler technique* for detecting bubbles in the venous return to the heart and its recommended use during surgical anesthesia² has emphasized the common occurrence of air embolism, even though it rarely may prove clinically significant.

For major aspirations of air into the venous system, suctioning with a central venous catheter can be life-saving. Much more common, however, is the nagging question regarding the hazard from an odd bolus of air or the slow but persistent entry of air, which could admit bubbles to the systemic arterial circulation. This raises two obvious questions: 1) What factors determine whether venous air can spill over into the systemic arterial system? and 2) how much systemic arterial air can be tolerated, if any?

To take the second question first, fatal volumes for a bolus of air typically quoted for dogs³ are 0.05 ml in the coronary circulation or 0.5 ml in the pulmonary veins. Three times the critical bolus volume can be tolerated if the air is infused slowly as microbubbles,⁴ but it is still several orders of magnitude smaller than those that the venous system has been known to tolerate.

The first question, therefore, concerns the factors that might compromise the otherwise superb capability of the lungs to trap venous bubbles, which has been demonstrated with air ranging in form from bolus injections to microbubbles of diameters down to 14 μm .⁵ There is the isolated case of a patient with a patent foramen ovale or, more common, one with an unsealed interatrial tissue flap (probe patent) that could be opened with a reverse pressure gradient facilitated by the pulmonary hypertension accompanying venous air embolism. Otherwise, the vital air trap is the pulmonary vasculature in which bubbles elicit a reflex vasoconstriction.⁵ The factors that we have found to compromise this otherwise superb capability of the lung to trap bubbles are pulmonary oxygen toxicity⁶ and the volume of venous air infused. There is a remarkably well-defined threshold of 0.35 ml \cdot kg⁻¹ for spill-over in dogs when air is infused continuously over 10–20 min.

Another feature of spill-over, following all but massive overload, is the characteristic delay in the appearance of systemic arterial bubbles.⁵ This usually is 10–30 min in dogs but can be as long as 90 min. This delay is tantalizingly close to the time needed for a central venous catheter to be inserted following the event and still leaves open the question of whether it then would be effective. Another potential clinical aspect of the characteristic delay is that the absence of signs of systemic arterial embolism immediately following surgical procedures conducive to venous embolization should not be construed as indicating that the risk of systemic arterial bubbles has passed. It is hoped that these additional facts about air embolism will help anesthesiologists in their management of this potentially dangerous occurrence and put into better perspective the benefits of precordial Doppler monitoring as an early warning.

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