CORRESPONDENCE

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In Reply—Noel points out that the catheters used in our study retrieved less than half of the venous air emboli and that a continuous column of frothy blood extending from the superior vena cava into the pulmonary vasculature was noted on necropsy. He indicates that, if the catheters were positioned in the pulmonary artery, a larger portion of the air emboli may have been extracted, and more animals would have survived. The rationale for positioning multiorifice catheters at the superior vena cava-right atrial (SVC-RA) junction was based on previous in vitro studies that concluded that an air lock occurs in the right atrium. Those evaluations documented that more than 80% of an air emboli can be removed by a multiorifice catheter located at the SVC-RA junction. The in vivo animal studies have confirmed that the majority of the air can be removed by a catheter located at the SVC-RA junction. However, the design of those studies may have contributed to those findings. Those studies injected air into the internal jugular or other large central vein. This may cause streaming of the air and a right heart air lock. Our study design attempted to mimic a venous air embolism occurring from a dural sinus during a decayed procedure.

Because of the results of the investigation, we postulate that there may be a fundamental difference in the blood fluid interface presented to the catheters when air is injected into a major vessel as opposed to a dural sinus. The air injected into the dural sinus may undergo considerable fractionation, as opposed to air injected into the central circulation. Is the frothy mixture less capable of creating a right heart air lock and thus less capable of aspiration? If so, it would explain the relatively low percentage of air aspirated compared to the other animal study designs. Based on the low volume of aspiration and the necropsy findings, should we reevaluate the location of the catheter for air aspiration? Would a high-volume aspiration catheter in the pulmonary vasculature be more effective than one at the SVC-RA border? Questions remain to be answered in future investigations.

Regarding the design of the catheter, Noel is correct that the electrocardiogram electrode design of the JX-318 catheter (Arrow International, Reading, PA) is probably better. The design of the JX-318 catheter for aspiration of air, however, appears to be inferior and has never been evaluated in any study of air aspiration.

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Difficulty Using a Laryngeal Mask Airway in a Patient with Lingual Tonsil Hyperplasia

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To the Editor—The successful use of a laryngeal mask airway (LMA) in three patients with undiagnosed lingual tonsil hyperplasia has been described by Biro and Shahinian. The LMA was used without apparent difficulty to treat a synchronously occurring unexpected airway complication. We cared for a patient who was found to have lingual tonsil hyperplasia at the time of induction but in whom the lungs could be ventilated with only marginal success via an LMA.
A 55-yr-old woman scheduled for ankle surgery had normal preoperative airway examination results. After induction with propofol and rocuronium, standard mask ventilation, eventually used both an oral airway and two anesthesiologists yielded no detectable gas exchange. Multiple attempts at laryngoscopy revealed only an abundance of soft, redundant, almost papillomaous appearing tissue at the base of the tongue. Transtracheal jet ventilation was attempted, but after several attempts, subcutaneous crepitus occurred without evidence of gas exchange. The catheter was removed, and a #4 LMA was inserted easily. Although gas exchange was occurring, the highest pulse oximeter reading attained was 92% with an FIO₂ of 1. The inadequate gas exchange was confirmed further by a simultaneous blood gas analysis (Pao₂, 62 mmHg). Using a fiberoptic bronchoscope through the LMA, only a very small portion of the vocal cords could be seen posteriorly. The anterior aspect of the cords was covered with folds of hyperelastic tissue from the lingual tonsil. The bronchoscope was guided through a tiny opening between the cords, and a normal appearing trachea was identified. We were unable, however, to pass a 6.0 mm ID endotracheal tube over the bronchoscope through the vocal cords. The Pao₂ decreased to 30% during this apneic intubation attempt, increasing to 85% over about 1 min after the bronchoscope was removed and ventilation resumed via the LMA. Because of the apparently edematous periglottic tissue and decreasing pulse oximeter readings, it was decided to proceed to a cricothyrotomy. Subsequent biopsy revealed a clear diagnosis of lingual tonsillar hyperplasia.

This case illustrates, as has been described previously, that the LMA can be a life-saving tool. It also serves as a reminder that this tool at times may be only limited efficacy as a superglotic ventilatory conduit in the presence of a significant periglottic obstruction.

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Postoperative Management Strategies May Obviate the Need for Most Preoperative Cardiac Testing

To the Editor.—As clinicians who run a preoperative evaluation center and provide intraoperative anesthesia and postoperative intensive care, we, like Mangano,1 have been frustrated with the problem of preoperative cardiac risk assessment. However, as we question whether “a cardiac risk assessment paradigm is possible,” our answer is slightly different. Whereas Mangano concludes with a call for development of screening algorithms and large-scale trials assessing testing technologies, we suggest that a more sensitive, specific, and cost-effective paradigm is probably not feasible and, equally important, may not be necessary. Rather, we believe that the focus should be shifted from preoperative testing to development of improved methodologies for postoperative ischemia detection and treatment.

For the past two decades, the conventional approach to cardiac risk management in anesthesia has been preoperative monitoring and interventions (to arrest or immediately correct evolving ischemia). A variety of screening tests have been proposed to identify high-risk patients who are either asymptomatic or have stable symptomatology; however, these tests have a low positive predictive value and a real incidence of false negatives. The positive predictive value of these tests is low; not because the tests are unable to detect significant coronary disease, but rather, because current management strategies have reduced the likelihood of patients with coronary disease experiencing major cardiac complications. Moreover, because plaque rupture can occur in physiologically insignificant lesions, there always will be a low but real incidence of false negatives.

The current economic environment is challenging all of us to examine our practice patterns and evaluate whether they are cost-effective. We believe that the way preoperative cardiac screening tests are used does not meet the challenge of cost-effectiveness. Available tests are expensive, and detection of unrecognized coronary atherosclerosis, by necessity, entails fairly widespread testing. Data on the societal costs of preoperative cardiac testing are difficult to obtain, but a recent survey2 indicates widespread preoperative testing in vascular surgery patients (60% of 400,000 cases/year). According to current estimates, 9 million patients are at risk for cardiac complications each year; whereas the actual number undergoing preoperative testing is unknown, the cost of cardiac screening is enormous. Cost-effective testing also requires that the results can be used to change outcome. Available data do not support that this is the case for preoperative cardiac screening tests. When angiography and angioplasty or bypass surgery are performed for the sole purpose of reducing

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