isolate were identical to those of the organisms recovered from all four patients. The organism was resistant to all antibiotics except colistin.

In our practice a sterile needle is connected to the distal end of the tubing from the analyzer and the needle is inserted through the corrugated rubber tubing of a Mörch swivel to aspirate a sample of gas for analysis. Although the flow of gas in the oxygen analyzer is in a one-way direction away from the patient, condensate may have contaminated the needle or the fingers of personnel using the analyzer. The tubing, once established as an environmental reservoir of *Pseudomonas maltophilia*, might quickly have become the source of additional airway contamination via tubing → needle → tracheal tube or tubing → hand → tracheal tube routes. It is important to note that dry areas of the oxygen analyzer have been sterile in the past. Although generally considered nonpathogenic, nonfermentative gram-negative bacilli such as *Pseudomonas maltophilia* have been increasingly associated with a wide variety of infections, particularly in critically ill patients.[6,7]

In this present instance, *Pseudomonas maltophilia* was responsible for airway colonization in four seriously ill patients within a brief period. Each patient had a tracheal tube in place and had been treated with broad-spectrum antibiotics. The only environmental source of this organism found in the R-SICU was a length of oxygen analyzer tubing containing condensate.

Continued awareness of all respiratory equipment shared by patients as potential foci of hospital infections cannot be overemphasized. Items of respiratory equipment frequently considered free from contamination because of presumed dry surfaces, such as peak-negative-pressure meters, oxygen analyzers, and spirometers, remain potential sources of airway contamination. Such equipment must be sterilized between uses. Alternatively, sterile connectors should be inserted between instrument and patient when this equipment cannot be adequately decontaminated. In addition, there will be less likelihood of oxygen analyzer contamination if the inspired oxygen concentration is measured near the gas source rather than near the patient.

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The Use of PEEP to Identify the Source of Cardiopulmonary Air Embolism

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Air embolism is an acknowledged hazard when patients are placed in the sitting position for surgical procedures.[1,2] When the surgical field is higher than the right atrium, air may enter the cardiovascular system. The following report describes the identification of the site of air entry by application of positive end-expiratory pressure (PEEP) during an intraoperative episode of massive air embolization.

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Report of a Case

A 75-year-old man experienced rapid deterioration of mental status 12 hours after blunt trauma to the head. Respiratory arrest occurred during diagnostic radiography; with the endotracheal tube in place, and comatose, the patient was brought immediately to the operating room for emergency suboccipital craniotomy with exploration of the posterior fossa. Anesthetic induction and maintenance were accomplished with intermittent iv administration of thiopental and nitrous oxide in oxygen (50 per cent); pancuronium

bromide was given for muscle relaxation. Cerebral ventriculostomy, iv administration of mannitol and dexamethasone, and mechanically-controlled hyperventilation (Pa_{CO_2} 23 to 26 torr) were used to control intracranial pressure. A central venous catheter was passed by the subclavian route; the catheter tip was estimated by stylet measurement to be in the superior vena cava just above the right atrium. Other monitors included a radial arterial cannula with pressure transducer, precordial Doppler ultrasonic flow transducer, and an electrocardiograph.

The operation was begun with the patient in the sitting position, occiput approximately 35 cm above the sternal angle. Blood pressure was 140–160 torr systolic, 80–100 torr diastolic, with pulse rate 77/min, controlled by demand pacemaker. Ninety minutes into the operation, with the posterior fossa exposed for evacuation of a cerebellar hematoma, an abrupt coarseness of the Doppler tones was noticed. Aspiration through the central venous catheter produced an intermittent stream of gas bubbles, totaling about 15 ml. Nitrous oxide was discontinued. Additional increments of air of approximately 200 ml total volume were withdrawn from the central venous catheter by syringe during the subsequent 30 minutes. A slow decline of arterial pressure to 95/55 torr responded to phenylephrine, 125 μg, iv. The electrocardiogram remained unchanged. Although repeated ausculation of the heart revealed resonance and splashing sounds indicative of large amounts of intracardiac gas, intensive scrutiny of the surgical field, the intravenous catheter entry site, and the catheter connections failed to uncover the source of embolization. A Boehringer PEEP valve (15 cm H_2O) was added to the exhalation limb of the anesthesia circuit in an attempt to stop what appeared to be continuing passive entry of air into the thorax. Seconds later, the surgeon remarked that he could see a cluster of gas bubbles at the corner of the operative field, and he traced them to a laceration of a vein deep in the divided neck muscles. Within ten minutes of closure of this venous defect, vital signs had stabilized and the Doppler and auscultatory heart tones had returned to normal. PEEP was discontinued and no further therapeutic intervention was necessary.

Postoperatively, chest x-ray and the patient's level of consciousness were indistinguishable from those seen preoperatively, with no gross evidence of pulmonary or systemic air embolization. Substantial improvement in the patient's mental status occurred over the next four days.

Discussion

The use of ultrasonic precordial flow transducers to detect small quantities of intracardiac air has demonstrated that the frequency of cardiopulmonary air embolism in patients undergoing operations in the sitting position may approach 50 per cent.[3–6] Clinically useful modalities available for treatment of cardiovascular air embolization are, however, limited to aspiration of intracardiac air from right atrial or Swan-Ganz catheters, discontinuation of nitrous oxide in the inspired gas mixture, external cardiac compression and the use of positive inotropic agents, and elevation of venous pressure at the operative site by compression of jugular veins, intermittent positive-pressure ventilation, or other means. The effectiveness with which these techniques minimize morbidity and mortality depends upon their prompt initiation and the simultaneous identification and closure of the site of air entry. Although generally presumed to be within the operative field, and therefore, visible to the surgeon, the site of air entry could not be found in half of the cases reported in one large series,[4] a situation that produced the persistent massive embolization described in a report of a single case.[7]

In our case, the air entry point was not readily apparent, and delay in its location resulted in continued embolus formation. Bilateral compression of the internal jugular vein was not attempted. Instead, we imposed upon our patient sustained positive intrathoracic pressure with a PEEP valve in order to prevent further aspiration of air, and found that PEEP also permitted identification of the site of air entry. Although a reduction in venous return and the possibility of transient hypotension were anticipated, blood pressure remained stable during the 10 minutes of PEEP application. PEEP at levels as low as 10 cm H_2O has reportedly been associated with barotrauma and systemic air embolization in mechanically ventilated rodents,[8] but a similar hazard has not, to our knowledge, been demonstrated in man.

In summary, we conclude that temporary application of PEEP can be of value during intraoperative cardiopulmonary air embolization, not only as a means to minimize further embolus formation, but also as a technique to facilitate identification of the site of air entry.

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