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Detection of Venous Air Embolism by Airway Pressure Monitoring

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Venous air embolism is a well-recognized complication of surgery conducted in the sitting position. A variety of useful and sensitive monitoring techniques allow detection of the air or its physiologic effects.

Pulmonary air emboli and thromboemboli cause vasoconstriction and bronchoconstriction with alterations in total airway resistance and dynamic lung compliance in animals and humans.[1,2] In experimental gas embolism in dogs, lung mechanics are reliably altered with volumes of air of 0.2 $cc \cdot kg^{-1}$ or greater[3] or when gas is administered at a rate of 0.1 to 0.15 $cc \cdot kg^{-1} \cdot min^{-1}$.[4,5] These airway pressure changes correspond to an increase in airway resistance and a decrease in lung compliance. Similar pulmonary changes occur with embolism of autologous thrombi,[1,6] fat,[7] barium sulfate,[8-9] or glass beads,[6] where constriction of airways less than 3 mm in diameter, notably alveolar ducts and terminal bronchioles, occurs within 30–45 s after embolism. Bronchoconstriction may be the result of release of histamine, serotonin,[6] or prostaglandin[10] mediators and can be blocked by pretreatment with indomethacin[10] or heparin. Bronchoconstriction occurs in human air embolism and thromboembolism and may be a clinical indicator of embolic events.[2]

Because airway pressure should detect these alterations in pulmonary mechanics,[10,12] monitoring ventilatory pressures may be a valuable adjunct for detection of venous air embolism. The following case demonstrates alterations in airway pressure during neurosurgery with documented air embolism.

REPORT OF A CASE

A 41-yr-old, 79-kg man was scheduled for a sitting-position right temporal and suboccipital craniotomy for removal of a recurrent cerebello-pontine angle meningioma. The patient had undergone a subtotal resection of the meningioma in 1981, after which he developed a right sixth nerve palsy and a seizure motor disorder, which had been increasing in frequency.

A computerized tomographic scan demonstrated a 4.5 cm-diameter hyperdense and enhancing tumor mass in the right parasellar and cerebello-pontine angle regions that compressed and displaced the fourth ventricle posteriorly. Carotid angiography showed narrowing and displacement of the presellar segment of the right internal carotid artery by the tumor; it had both infratentorial and supratentorial extensions.

The patient was brought to the operating room 90 min after premedication with oral diazepam, 10 mg, and intramuscular scopolamine, 0.4 mg. Two 14-g iv catheters and a left radial arterial cannula were inserted. A 90-cm polymer resin, 18-g central venous pressure (CVP) cannula was easily advanced through a right antecubital vein and its tip placed near the sinoatrial node by electrocardiographic monitoring.

The patient voluntarily hyperventilated with 100% oxygen before anesthesia was induced with thiopental (300 mg) and sufentanil (150 μg iv). Following pancuronium, 15 mg iv, the trachea was easily intubated and the lungs mechanically ventilated. Anesthesia was maintained with constant iv infusion drips of thiopental at 1.5 $mg \cdot kg^{-1} \cdot hr^{-1}$ and sufentanil at 0.5 $\mu g \cdot kg^{-1} \cdot hr^{-1}$. Additional sufentanil and pancuronium were administered as required during the 13.5-h case. The sitting position was accomplished uneventfully, and monitors included V_5 electrocardiogram (ECG), esophageal stethoscope, radial artery blood pressure, end-tidal CO_2 (ET CO_2), precordial Doppler ultrasound, CVP, and brainstem auditory and median nerve somatosensory evoked potentials. In addition to the above, airway pressure was continuously monitored by a technique similar to that described by Baker *et al.*[13] A Bentley Trantec® 1000 transducer and Pharmaseal® disposable dome (BD 240) placed externally on the chest at the level of the carina were connected by rubber tubing to a sidearm port of a 90°-angle connector placed at the Y-piece of the anesthesia circuit (fig. 1). This pressure was displayed on an oscilloscope and recorded on a strip chart with ECG, blood pressure, ET CO_2, and CVP.

Six and one-half h after incision, air embolism was noted by the Doppler ultrasound. Three serial episodes of air emboli occurred within 45 min (A, B, and C in fig. 2). Arterial blood pressure remained stable at 124/60 mmHg, and heart rate at 60 beats/min. With mechanical ventilation unchanged, the mean CVP increased stepwise from 4 to a high value of 10 mmHg, and the ET CO_2 decreased from 42 to a low value of 20 mmHg. Simultaneously, peak airway pressure abruptly increased from a stable baseline of 19 to a maximum of 25 mmHg. The chart recording of CVP, ET CO_2, and airway pressure is shown in fig. 2. Air embolism was confirmed by aspiration of 27 ml of air through the CVP (denoted by an *arrow* in fig. 2). During these emboli, the surgeon identified and cauterized the offending veins, and these changes resolved over the subsequent 20 to 30 min. No further episodes of air embolism were detected.

Resection of the tumor was completed without other incident except for three episodes of bradycardia associated with surgical manipulation of the brainstem. The patient was awake and responding to commands, and his trachea was extubated in the operating room. He had, however, a left-sided hemiparesis, right V, VI and VII nerve palsies, a decreased gag reflex, and dysarthria secondary to edematous changes. These deficits resolved rapidly over the next month with physical and speech therapy, at which time he was discharged.

DISCUSSION

The ability to detect venous air embolism early in neurosurgical operations in the sitting position has become

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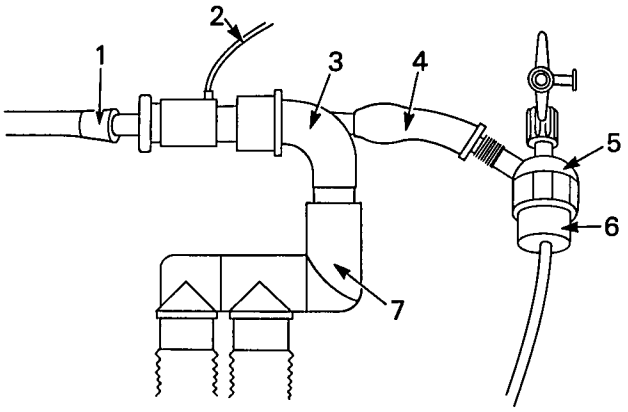

FIG. 1. A diagram of the apparatus used to monitor airway pressure. (*1*) Endotracheal tube; (*2*) sampling port for ET CO_2; (*3*) 90°-angle connector with sidearm; (*4*) rubber tubing; (*5*) Pharmaseal® Disposable Dome (BD 240); (*6*) Bentley Trantec® 1000 transducer; and (*7*) Y-piece connector of anesthesia circuit.

an important aspect of anesthetic intraoperative care. Several monitoring techniques have become available, and a combination of several methods may provide optimal detection.[14,15] The most commonly used methods include precordial Doppler ultrasound, central venous or pulmonary artery catheterization, and ET CO_2 monitoring.

Each of these techniques has problems that may limit its usefulness. Airway pressure monitoring is a simple technique. Any dynamic pressure transducing system such as those for monitoring invasive cardiovascular pressures can be used for this noninvasive technique. The system used in this case (fig. 1) is identical to the transducing apparatus used for measurement of the CVP and radial arterial pressure. These components are readily available and reusable if caution is taken to cleanse and sterilize each component as appropriate for ventilatory equipment. The waveform of the pressure measured at the endotracheal tube is displayed on the monitoring oscilloscope. Visual observation of the pressure on a breath-to-breath basis can be made from the oscilloscope display when a low-pressure scale is used (*e.g.*, 0–30 mmHg). In addition, a permanent record of the waveform and trend can be obtained.

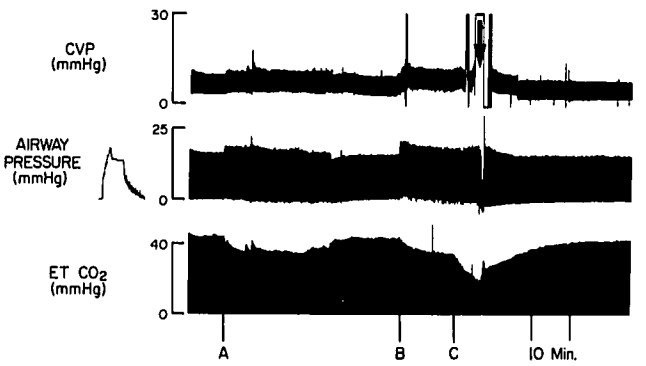

FIG. 2. Strip chart recording during air embolus episodes. CVP, airway pressure, and ET CO_2 are shown. Three air embolism incidents occurred with Doppler indications at A, B, and C. The *arrow* indicates the time at which air was removed through the CVP. The airway pressure profile is shown at the left.

The monitoring of airway pressure has previously been advocated for assessment of pulmonary mechanics of patients requiring ventilatory support.‡ Routine use has been suggested as a method to warn of airway and ventilatory equipment malfunction.[16] When actual tidal volume and flow characteristics are known, pulmonary compliance and airway resistance can be calculated[12,17] and alterations determined on a breath-to-breath basis. Thus, when the ventilator settings have not been changed, alterations in airway pressure will reflect changes in airway resistance, compliance, or both. Evidence in dogs demonstrates that airway pressure, lung compliance, and airway resistance are altered by venous air embolism at volumes of 0.2 $cc \cdot kg^{-1}$.[3] This volume is similar to the 0.5 $cc \cdot kg^{-1}$ reported for alterations in ET CO_2.[15] These separate studies suggest that these two modalities should have similar clinical sensitivity for air embolus detection. In the only dog study in which ET CO_2, airway pressure, and CVP were compared, calculated dynamic lung compliance was altered with volumes of air similar to that which altered the CVP.[16] Both methods were found to be less sensitive than ET CO_2. Airway resistance, however, was not determined; the type of ventilation and method of determination of compliance may have differed from the other studies; and these dogs were studied in the supine, rather than sitting, position. These differences may account for the apparent lack of consistency in air embolus detection sensitivity.

Airway pressure monitoring for venous air embolism lacks specificity in that thromboemboli or bronchoconstriction secondary to hyperactive airways or light anesthesia can produce increases in airway pressure. A patient receiving a prostaglandin inhibitor or heparin might not show the appropriate rise in airway pressure if the release of chemical mediators is blocked.

In this patient, airway pressure changes occurred simultaneously with changes in ET CO_2 and CVP during a documented venous air embolism. This technique appears to be a valuable adjunct to the other available monitoring techniques. Its simple, noninvasive nature makes it widely applicable, and the equipment is familiar and readily available to most anesthesiologists. Patient body habitus, availability of venous access, and electrical interference should not limit its application. Further, the decrease in lung compliance and increase in airway resistance

‡ Bone RC: Monitoring patients in acute respiratory failure. Respiratory Care 27:700–701, 1982.

will not occur with decreases in cardiac output from other forms of cardiac depression where CVP and ET CO_2 may be altered. Thus, alterations in airway pressure may aid in the differentiation of the etiology of incidents where the ET CO_2 and CVP change without other evidence of air embolism. In addition, like CVP and ET CO_2, recovery after the episode allows observation of resolution of the physiologic effect of the embolus.

In summary, this case demonstrates the alteration of airway pressure with venous air embolism. This monitoring technique is a simple, inexpensive, and noninvasive method that offers promise as an adjunct to other techniques for the detection of venous air embolism in sitting neurosurgical operations.

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Unexpected Hyperthermia Manifesting during Outpatient Anesthesia

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Malignant hyperthermia (MH) is a rare and usually unexpected complication of anesthesia. In most cases, the association of tachycardia with cyanosis, metabolic acidosis, hyperthermia, hyperkalemia, or muscle rigidity alerts the anesthesiologist to the rare but potentially lethal complication MH.[1] We describe a case of unexpected hyperthermia occurring during anesthesia in an outpatient. The development of hyperthermia was not associated with

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