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Use of a Pulmonary-artery Catheter for Detection and Treatment of Venous Air Embolism:

A Prospective Study in Man

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The use of pulmonary arterial pressure (PAP) monitoring was compared with precordial Doppler ultrasound monitoring and continuous infrared end-tidal CO_2 fraction (FET_{CO_2}) analysis for detection and treatment of venous air embolism in 52 consecutive patients undergoing neurosurgical procedures in the seated position. Doppler air sounds were identified 44 times during 20 operations. During 12 operations there were 17 episodes of Doppler air sounds associated with increased PAP (mean increase = 13 ± 2.8 torr SE, $P < .001$), lasting for an average of 7 ± 2 min (SE). Only small volumes (2–20 ml) of air were recovered from the pulmonary artery and right atrium via the pulmonary-artery catheter during periods of increased PAP. During 12 episodes when PAP was increased, mean FET_{CO_2} decreased, from 3.8 ± .2 to 2.7 ± .3 per cent (SE), $P < .05$. Monitoring of PAP afforded prompt diagnosis of clinically significant air embolism and an estimate of the severity of the condition, usually before systemic circulatory changes occurred. Monitoring of PAP also indicated when it seemed prudent to continue the surgical procedure after air embolism and when surgical intervention had corrected the cause of air entrainment. Although successful pulmonary arterial catheterization was not possible in five patients, no significant complication resulted from the procedure in this series. The authors believe that monitoring of PAP for detection and treatment of air embolism is justified during seated neurosurgical procedures. (Key words: Anesthesia, neurosurgical. Embolism, air. Equipment: catheters, pulmonary artery; Doppler ultrasound. Monitoring: carbon dioxide; pulmonary arterial pressure.)

VENOUS AIR EMBOLISM is a potential hazard whenever the operative site is above the level of the heart, although it is most frequently associated with neurosurgical procedures performed with the patient in the seated position. Recent work by English *et al.*[1] has indicated that changes in canine pulmonary arterial (PA) pressure accurately reflect the volume of air embolized experimentally, and Munson *et al.* have described two cases[2,3] in which a pulmonary-artery catheter was used for early detection of intraoperative air embolism. Since pulmonary arterial catheterization is not without hazard, we undertook this prospective evaluation of routine pulmonary arterial pressure monitoring in order to determine its usefulness for diagnosis and treatment of venous air embolism in the clinical setting.

Materials and Methods

Fifty-two consecutive patients scheduled for elective cervical laminectomy or craniotomy in the seated position underwent attempted preoperative PA catheterization via either the right internal jugular or basilic veins, by use of a modified Seldinger guidewire technique and a triple-lumen nonthermistor balloon-directed catheter. Catheter position was verified by pulse-pressure configuration and wedge positioning with balloon inflation on a Hewlett-Packard Model 78304 A oscilloscope. All but five attempts were successful. The only complication other than ventricular extrasystoles associated with right ventricular catheterization was one carotid-artery puncture with a 16-gauge introducing catheter. There was no instance of neurologic deficit or pneumothorax.

General anesthesia was induced with thiopental, nitrous oxide, 70 per cent, oxygen, 30 per cent, and pancuronium, and was maintained with a variety of anesthetics. After the patient was placed in a seated position with the knees at heart level, a precordial Doppler probe‡ was affixed and its position over the right atrium was verified from the change in signal produced by a 2-ml bolus injection of 0.9 per cent NaCl solution through the central venous port of the pulmonary-artery catheter. End-tidal CO_2 fraction (FET_{CO_2}, Beckman LB-2) and PA, central venous, and radial arterial pressures were recorded continuously throughout the surgical procedure using Bentley Model 800 transducers and a Brush Model 440 recorder. Instances of Doppler air sounds were noted manually on the strip-chart record.

All instances of air embolism suggested by either an increase in PA pressure or a decrease in FET_{CO_2} were treated by promptly packing the incision with saline-soaked sponges. Nitrous oxide was discontinued, and a vigorous attempt was made to aspirate blood and air from the central venous and PA ports of the pulmonary-artery catheter.

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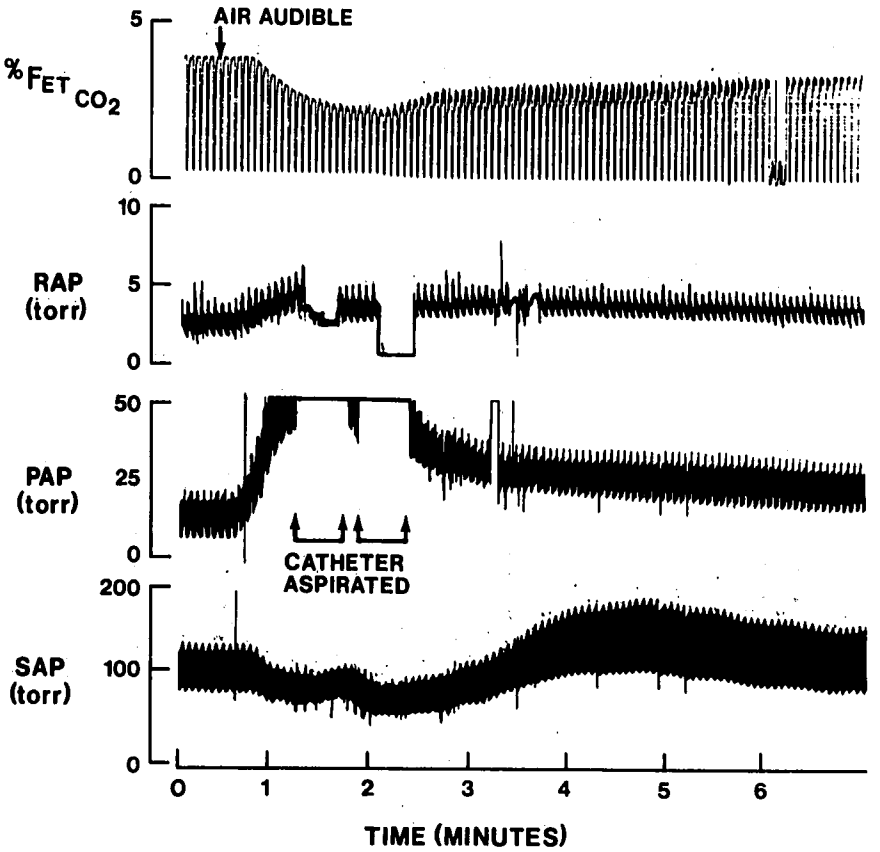

FIG. 1. Record of end-tidal CO_2 fraction (% $F_{ET_{CO_2}}$) and right atrial (RAP), pulmonary arterial (PAP), and systemic arterial (SAP) pressures during air embolism in one patient. Systemic hypotension and ventricular irritability occurred shortly after Doppler air sounds were audible, and were associated with marked pulmonary hypertension. We withdrew 80 ml of blood from the PA and RA, but recovered only 10 ml of air. Prompt packing of the incision stopped air entrainment, and the source was identified and corrected after the circulatory values had stabilized.

All changes in recorded variables during air embolism were compared with pre-embolism values using the Student *t* test for paired data. Comparisons between groups of patients were performed using the non-paired *t* test. $P < .05$ was regarded as significant.

Results

Forty-four separate episodes of Doppler air sounds were identified during 20 of the 52 operations performed. During 27 of these episodes no change in $F_{ET_{CO_2}}$ or PA or systemic arterial pressure was observed, and no therapeutic measure was taken.

Seventeen episodes of air embolism occurring in 12 patients were accompanied by a significant mean increase in PA pressure of 13 ± 2.3 torr (SE) and lasted an average of 7 ± 2 min (SE). Small volumes of air (2–5 ml) were aspirated from both the central venous and PA ports of the pulmonary-artery catheter during most periods of air embolism associated with increased PA pressure. During the two most severe instances of air embolism (associated with highest PA pressures and systemic hypotension), 10 and 20 ml of air, respectively, were recovered. One of these episodes was heralded by PA hypertension, and Doppler air sounds were only minimally audible after systemic hypotension had occurred. Doppler air sounds often persisted after the PA pressures had returned to normal values. Central venous pressure increased slightly during marked PA hypertension (fig. 1), but was of no diagnostic value during air embolism. There was no appreciable difference between the amounts of air recovered from the CVP port and the PA port of the catheter.

During the 12 instances of air embolism in the nine patients who sustained the largest increases in PA pressure, $F_{ET_{CO_2}}$ decreased significantly from 3.8 ±.2 to 2.7 ± .3 per cent (SE). One additional patient had a small decrease in $F_{ET_{CO_2}}$ during an episode of Doppler air sounds without a corresponding change in PA pressure. All five of the patients whose pulmonary arteries could not be cannulated were monitored by continuous $F_{ET_{CO_2}}$ measurements. One instance of Doppler air sounds occurred, but no change in $F_{ET_{CO_2}}$ was observed in this patient.

After clinically significant episodes of air embolism had been diagnosed and treated by immediately packing the incision, the return of PA pressure toward baseline values served as a guide to the timing of definitive therapy (surgical obliteration) of the air leak (fig. 1). Before additional surgical manipulation was undertaken, we reintroduced N_2O, 70 per cent, into the anesthetic gases. On two occasions an increase in

PA pressure and decrease in $F_{ET_{CO_2}}$ was observed, and further operation was deferred until a N_2O challenge did not cause changes in pulmonary arterial pressure.

Comparing the patients who underwent cervical laminectomies (31) with those who had craniotomies (21), there was no difference in the incidences of air embolism as detected by Doppler sounds, PA pressure, or $F_{ET_{CO_2}}$ monitoring. The mean peak changes in PA pressure and $F_{ET_{CO_2}}$ during air embolism, however, were significantly greater during craniotomies than during laminectomies (16 ± 2 versus 7 ± 2 torr [SE], and 1.7 ± 0.2 versus 0.4 ± 0.2 per cent [SE]).

Air embolism tended to occur early during craniotomies, with four of the five episodes of increased PA pressure developing as neck muscles were dissected away from the skull. In contrast, during cervical laminectomies, nine of 12 episodes of increased PA pressure occurred with excision of lamina or bony fragments, whereas only three episodes were observed while the incision was being opened or closed.

At the termination of anesthesia all pulmonary-artery catheters were either removed or withdrawn so that the tip was located in the right atrium. Continuous measurement of $F_{ET_{CO_2}}$ and constant controlled mechanical ventilation during removal of the pulmonary-artery catheter demonstrated no instance of a sudden decrease in $F_{ET_{CO_2}}$ or change in arterial blood-gas values suggestive of pulmonary thrombotic embolism.

All patients were nursed in the head-up position postoperatively. None sustained significant hematoma formation at the site of internal jugular-vein catheterization, and none developed signs or symptoms of interstitial pulmonary edema.

Discussion

Venous air embolism probably occurs most frequently in the clinical setting as a steady stream of small air bubbles entrained over a period of time.[2] While it has been shown that a large bolus of air can cause an air lock in the right heart, necropsies of dogs dying from slow infusion of air demonstrated bubbles dispersed throughout the cardiopulmonary circulation.[4] Small air bubbles lodge in peripheral branches of the pulmonary circulation and cause mechanical obstruction as well as reflex pulmonary vasoconstriction, resulting in increased PA pressure and wasted ventilation.[2] When air entrainment is slow, cardiovascular collapse probably develops either from acute right heart failure or from occlusion of the pulmonary outflow tract when the volume of air exceeds the capacity of the pulmonary arterial tree.

The most sensitive method for detecting embolized air is a correctly positioned precordial Doppler device. It can document bubbles smaller than 1 ml passing through the right atrium, although we and others[5] have observed that it may not detect all instances of clinically significant air entrainment. The Doppler device cannot quantitate the severity of circulatory impairment, however, and frequently alerts the anesthesiologist and surgeon to episodes of air embolism that are clinically insignificant.

Monitoring of end-tidal CO_2 fraction is less sensitive than the precordial Doppler device, and is a noninvasive technique that can alert the anesthesiologist to entrainment of volumes of air sufficient to affect the pulmonary circulation before deterioration of systemic circulation occurs. Unlike the Doppler device, $F_{ET_{CO_2}}$ monitoring reflects the relative severity of air embolism[1] and indicates when the condition is abating. At present, however, cost factors limit use of end-tidal CO_2 monitoring to busy neuroanesthesia services.

We observed that clinical pulmonary arterial pressure monitoring appears to be intermediate in sensitivity between the precordial Doppler device and end-tidal CO_2 monitoring, a finding previously demonstrated in supine dogs given graded air emboli.[1] Our experience indicates that pulmonary arterial pressure monitoring promptly reflects clinically significant air embolism in 39 per cent of occurrences of positive Doppler air sounds. The amount of increase in PA pressure reflects the severity of the condition, and successful surgical treatment is documented by a rapid return to baseline pulmonary arterial pressure. The widespread availability of arterial pressure monitoring capability and the ease of insertion of a pulmonary-artery catheter suggest that it is a feasible method for detection of air embolism.

It is possible that our use of nitrous oxide anesthesia exaggerated the changes observed with PA pressure monitoring. Rapid diffusion of nitrous oxide into embolized air bubbles probably enlarged the effective size of the embolus and caused a greater change in pulmonary hemodynamics than would have occurred had nitrous oxide not been used in the anesthetic mixture.[6] Conversely, this may have resulted in more prompt diagnosis and surgical intervention than if PA pressure had increased very gradually. The addition of nitrous oxide to the anesthetic mixture after treatment of venous air embolism proved to be a valuable diagnostic maneuver. The presence of residual air in the pulmonary vasculature became evident when PA pressure promptly increased, suggesting to us that further delay was prudent before surgical manipulation was reinitiated.

We were not able to document the usefulness of the pulmonary-artery catheter for recovery of em-

bolized air. In contrast to the massive volumes (as much as 500 ml) of air recovered from right atrial catheters by Albin *et al.*,[7] our evaluation showed only small amounts (2–20 ml) of air that could be removed from either the PA or central venous ports of the pulmonary-artery catheter during embolism associated with increased PA pressure. Our findings agreed with the observations of Munson *et al.*,[2,3] who likewise could withdraw only a few ml of air from pulmonary-artery catheters during air embolism. Rather than reflecting a therapeutic failure, we feel that these findings support the contention that PA pressure monitoring facilitates diagnosis and prompt surgical treatment of air embolism before the pulmonary artery and heart chambers fill with bubbles and impair blood flow.

In summary, we have found pulmonary arterial catheterization and pressure monitoring to be a safe, reliable, and versatile technique. It rapidly detected clinically significant episodes of air embolism in 23 per cent of our patients, quantitated the severity of the condition, and identified successful therapeutic intervention on the part of the surgeon. It also afforded a route for removing air from the pulmonary artery and right heart. We believe its use for monitoring air embolism during neurosurgical procedures with the patients seated is justified.

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