

Atrial Pressures in the Seated Position

Implication for Paradoxical Air Embolism

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In order to investigate the circumstances in which air bubbles in the venous circulation could pass to the arterial circulation via a patent foramen ovale, simultaneous right atrial (RAP) and pulmonary capillary wedge pressures (PCWP) were determined in two patient populations undergoing elective neurosurgical procedures in the seated position. In Group 1 ($n = 24$), quadruple-lumen pulmonary arterial catheters were inserted prior to the induction of anesthesia, and RAP and PCWP were determined while the patients were: 1) awake and supine, 2) awake and seated, 3) anesthetized and supine, 4) anesthetized and seated, and 5) 60 minutes after skin incision. In Group 2 ($n = 31$), RAP and PCWP were measured prior to, and then during intraoperative episodes of clinical air embolism. In Group 1, placement in the seated position while awake resulted in a significant decrease in PCWP ($8.5 \text{ mmHg} \pm 0.9 \text{ SE}$ supine vs. $6.6 \text{ mmHg} \pm 0.9 \text{ SE}$ seated, $P < 0.05$), but no change in RAP ($6.3 \text{ mmHg} \pm 0.6 \text{ SE}$ supine vs. $6.5 \text{ mmHg} \pm 0.9 \text{ SE}$ seated). Similar changes were found after induction of anesthesia (PCWP supine = $7.8 \text{ mmHg} \pm 0.8 \text{ SE}$ vs. PCWP seated = $5.6 \text{ mmHg} \pm 0.8 \text{ SE}$, $P < 0.05$, and RAP supine = $6.6 \text{ mmHg} \pm 0.8 \text{ SE}$ vs. RAP seated = $4.4 \text{ mmHg} \pm 0.7 \text{ SE}$). Sixty minutes after skin incision, 13 of the 24 patients had PCWP lower than RAP, and mean PCWP was less than RAP ($5.6 \text{ mmHg} \pm 0.8 \text{ SE}$ vs. $6.2 \text{ mmHg} \pm 0.8 \text{ SE}$). In Group 2, mean pulmonary artery pressure increased markedly with venous air embolism ($10.9 \text{ mmHg} \pm 0.7 \text{ SE}$ to $18.0 \text{ mmHg} \pm 1.04 \text{ SE}$, $P < 0.01$). Smaller increases were seen in both RAP ($3.7 \text{ mmHg} \pm 0.7 \text{ SE}$ vs. $4.7 \text{ mmHg} \pm 0.6 \text{ SE}$, $P < 0.01$) and PCWP ($4.6 \text{ mmHg} \pm 0.6 \text{ SE}$ vs. $5.5 \text{ mmHg} \pm 0.7 \text{ SE}$, $P < 0.01$), but there was no evidence of RAP acutely exceeding PCWP. The authors conclude that use of the seated position inherently predisposes some neurosurgical patients to the risk of paradoxical air embolism, since the normal interatrial pressure gradient frequently becomes reversed in this position. (Key words: Anesthesia: neurosurgical. Embolism: air, paradoxical. Equipment: catheters, pulmonary artery. Monitoring: right atrial pressure; pulmonary capillary wedge pressure.)

PARADOXICAL AIR EMBOLISM is a well-recognized hazard of neurosurgical procedures performed in the seated position,¹ although the factors contributing to its occurrence have not been elucidated completely. While it is thought that air bubbles can pass from the right to

the left atrium via a probe patent foramen ovale (present in 20–30% of the adult population), this normally should not occur because left atrial pressures are usually higher than those in the right atrium.² Possible changes in the interatrial pressure gradient which might occur clinically have not been reported previously. The use of the pulmonary artery catheter for diagnosis and treatment of venous air embolism has provided a unique opportunity to study right atrial pressure directly and left atrial pressure indirectly at two critical junctures: 1) when patients are placed in the seated position; and 2) during episodes of clinical air embolism.

Materials and Methods

PART I

Twenty-four informed, consenting adult patients underwent pulmonary artery catheterization prior to elective neurosurgical procedures performed in the seated position. The protocol for the study was reviewed and approved by the institution's Human Investigation Committee. Catheter position was verified using pulse-pressure configuration and wedge positioning with balloon inflation. Simultaneous right atrial pressure (RAP) and pulmonary capillary wedge pressure (PCWP) measurements were made at the following times: 1) while awake and supine; 2) while awake and seated (back propped up 45° to horizontal); 3) while supine and anesthetized (60% N_2O in O_2 with either intravenous or volatile agent supplementation); 4) while seated and anesthetized; and 5) 60 min after skin incision. Pressures were transduced using Bentley® Model 800 transducers positioned in the midaxillary line when patients were supine and at the level of the fifth intercostal space anteriorly when the patients were seated (fig. 1). Pressures obtained in the awake-supine state were compared with values observed in the awake-seated state, and pressures determined in the anesthetized-supine state were compared with those observed when the patients were anesthetized and seated.

Intravenous fluid replacement consisted of 1,000 ml of 5% dextrose in lactated Ringer's solution infused prior to induction of anesthesia and a mean volume of $1,640 \text{ ml} \pm 278 \text{ SD}$ infused by one hour after incision. No patient's blood loss exceeded 200 ml up to this point,

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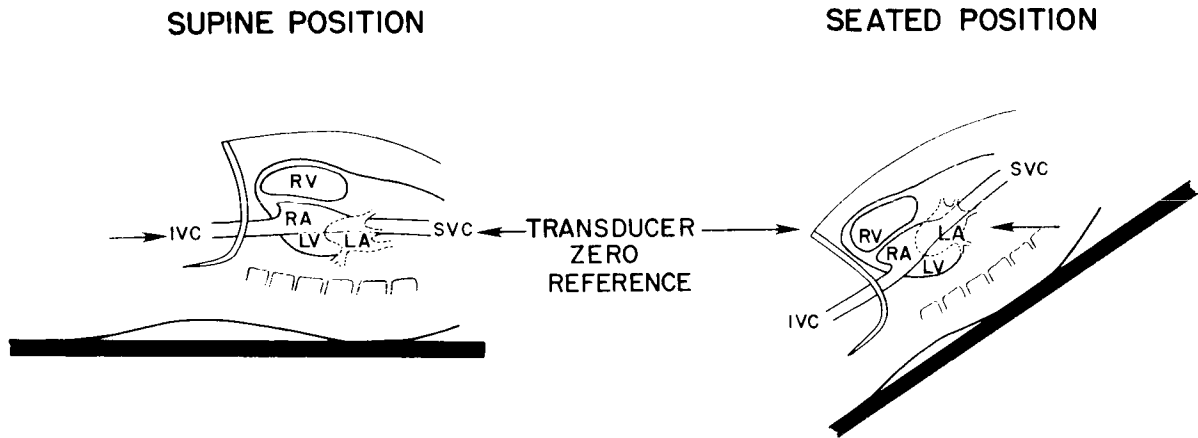


FIG. 1. Schematic representation of the anatomic relationship between the right atrium (RA) and the left atrium (LA) as visualized from left lateral view angiocardiograms. Pressure transducers were referenced to the midaxillary line while patients were supine. In the seated position, pressure transducers were referenced to the fifth intercostal space anteriorly.

and no episode of air embolism was detected during the study period.

PART 2

One hundred consecutive patients undergoing neurosurgical operations in the seated position (including the 24 in Part 1) were prospectively monitored for venous air embolism with precordial Doppler ultrasound signal and continuous recordings (Brush Model 440 recorder) of end-tidal CO₂ fraction (Beckman LB-2) and pulmonary arterial and right atrial pressures. The incidence of air embolism in these patients was 36%.

During 31 episodes of venous air embolism in which pulmonary artery pressure increased, simultaneous measurements of right atrial and pulmonary capillary wedge pressure were obtained and were compared with

the same variables determined in each patient during the 30-min period before air embolism was detected.

Statistical comparison for both parts of this study were performed using Student's *t* test for paired data. *P* values <0.05 were regarded as significant.

Results

Figure 2 summarizes our findings with regard to RAP and PCWP values measured during postural changes and surgery. In the awake, supine condition, mean PCWP was higher than mean RAP. When patients were placed in the seated position while awake, mean PCWP was reduced significantly. After induction of anesthesia, in the supine state, PCWP was greater than RAP, but in the seated position, there was significant reduction in PCWP although mean RAP was still lower than mean PCWP. One hour after skin incision, PCWP remained

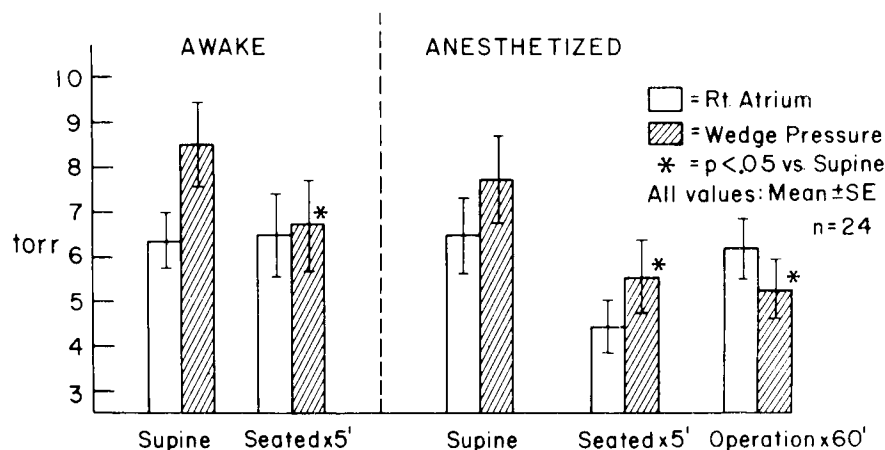


FIG. 2. Changes in right atrial and pulmonary capillary wedge pressures associated with placement in the seated position both before and after induction of anesthesia for neurosurgical operations. Sixty minutes after incision, the normal positive pressure gradient from the left to the right atrium had reversed, predisposing patients to systemic air embolism if venous air embolism occurred.

low and the mean right atrial to left atrial pressure gradient had become positive, thus predisposing patients to paradoxical air embolism. In all, 13 of the 24 patients exhibited an RAP which was greater than their PCWP after one hour of operation. In contrast to the changes in PCWP, mean pulmonary artery pressure (PAP) was 13.3 mmHg \pm 1.1 SE when patients were supine awake, and 13.8 mmHg \pm 0.8 SE when they were supine anesthetized. No change in mean PAP occurred when patients were placed in the seated position.

In 31 patients who actually suffered air embolism, mean PAP increased markedly, whereas RAP and PCWP were found to increase in concert, with the interatrial pressure difference remaining essentially constant (fig. 3). Mean RAP was lower than mean PCWP in this group of patients, presumably because some of the episodes of air embolism occurred relatively early in the operation. However, one patient in this group whose RAP was 5 mmHg higher than PCWP at the time of venous air embolism, subsequently developed signs and symptoms of systemic arterial air embolism, with a dense hemiparesis and multiple foci of cortical infarction seen with postoperative CT scans.

Discussion

Embolized air bubbles may pass from the venous to the arterial circulation either through an intracardiac defect or through the pulmonary circulation. Although several animal studies³⁻⁵ have tended to discount the latter etiologic factor, at least one clinical case report suggests it is possible.⁶ However, the most likely cause of paradoxical air embolism is thought to occur with the passage of air bubbles through a probe patent foramen ovale.¹ Although considered to be a normal anatomic variant, a probe patent foramen ovale may be as large as a centimeter in diameter. It will remain functionally closed as long as left atrial pressure exceeds right atrial pressure so that the flap-like septum secundum is applied over the ostium primum defect.

The incidence of a probe-patent foramen ovale in the general population is reported to be 20-30%.² Since venous air embolism occurs in 30-40% of patients during seated neurosurgical procedures,⁷ approximately 6-12% of these patients are at risk for developing paradoxical air embolism when right atrial pressure exceeds left atrial pressure. One reason why more episodes of paradoxical air embolism are not noticed may be that in the majority of patients with a positive RAP-LAP gradient the pressure difference is not great enough to force air bubbles through a probe-patent foramen ovale. Alternatively, since the superior vena cava is oriented so that blood flow is directed toward the tricuspid valve,

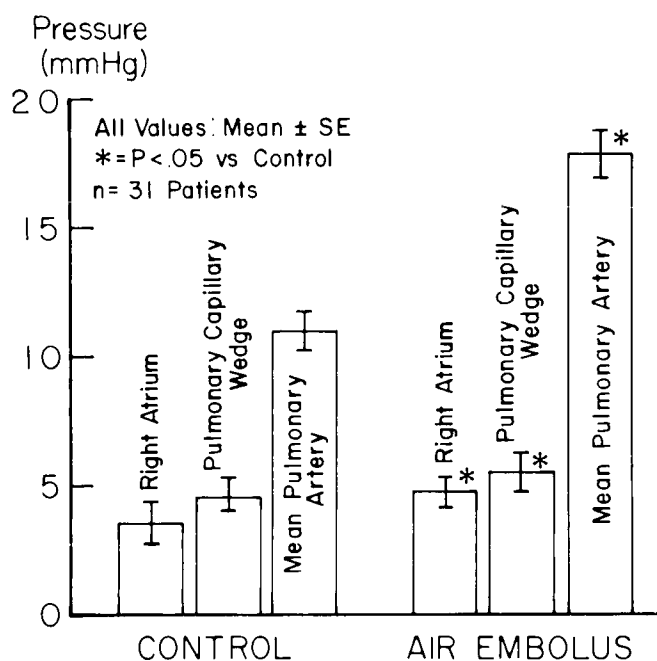


FIG. 3. Clinical episodes of venous air embolism caused marked increases in mean pulmonary artery pressure. Right atrial and pulmonary capillary wedge pressures increased concurrently but to a lesser extent than pulmonary artery pressure.

it may be that embolized air bubbles tend to be carried preferentially into the right ventricle and pulmonary circulation rather than passing through a probe-patent foramen ovale.

The critical pressure necessary for air bubbles to be forced through a probe-patent foramen ovale is not known. Experimentally induced canine atrial septal defects are associated with biphasic flow, although the preponderant direction of flow is linearly related to the interatrial pressure gradient.⁸ Only a 4-mmHg gradient can produce a 50% right-to-left intracardiac shunt after cardiac surgery,⁹ and the patient in the present study who developed signs and symptoms of paradoxical air embolism had a 5-mmHg RAP to PCWP pressure difference.

A possible source of error in this study is that a pulmonary artery catheter tip might become lodged in a region within the lung where alveolar pressure exceeds pulmonary arterial and venous pressure, and where PCWP will reflect airway pressure rather than left atrial pressure (LAP).¹⁰ Benunof *et al.*¹¹ found only 1.8% of patients with a high (6 cm above carina) cephalad placement which might "possibly be in Zone 1 when sitting." Thus, while this finding may be important in a single individual, we doubt that this occurred in many of the patients in the present study.

Does change in body position from the supine to the 45° upright position alter the apparent pressures measured with a pulmonary artery catheter? RAP and PCWP measured in the supine position with the transducers at the midaxillary line probably indicated a falsely low left atrial pressure and high right atrial pressure, since the right atrium is anatomically anterior to the left atrium (fig. 1). When the patient is placed upright at a 45° angle with the transducers affixed anteriorly at the fifth intercostal space (apparently over the right atrium as indicated by Doppler ultrasound signal), then the transduced right atrial pressure should become more negative relative to the left atrial pressure as reflected by PCWP. It seems very unlikely then, that the changes in the interatrial pressure gradient that we observed are the result of artifact due to alterations in the position of the pressure transducers relative to the heart chambers. Furthermore, the actual decrease in LAP was probably greater than that which we were able to measure when the patients were placed in the seated position since LAP is usually 1–2 mmHg lower than PCWP.¹²

What is the reason, then, for the acute reduction in PCWP when patients were placed in the seated position? The most likely explanation is that this reflects a decrease in pulmonary blood volume, since it is known that blood flow per unit of lung volume decreases in the seated position as compared with the supine position.¹³ The fact that mean pulmonary arterial pressure remained constant with postural change reflects the considerable compliance of the pulmonary circulation. If this is correct, then there must have been a period of time between when the seated position was achieved and when a new hemodynamic equilibrium was established during which the stroke volume of the left ventricle must have exceeded that of the right ventricle. Perhaps this is why the right-to-left atrial pressure gradient usually was not positive after only 5 min in the seated position, but was often positive after one hour of surgery.

It is conceivable that larger volumes of intravenous fluid replacement, perhaps including colloid solutions, might have prevented the reduction in PCWP. Although the infusion of 1,640 ml ± 278 SD of lactated Ringer's solution appeared to be adequate for the small amount of blood loss (<200 ml) plus maintenance fluid requirements, more vigorous replacement might have tended to expand circulatory volume and particularly the volume of the pulmonary circulation, thus increasing left ventricular preload. Investigations designed to confirm or refute these speculations are currently in progress.

During episodes of venous air embolism, it is possible

that PCWP may not accurately reflect true LAP. Air embolized into the pulmonary vascular bed may not be distributed uniformly, and, if the catheter tip is in a branch of the pulmonary vasculature that receives a portion of the air embolus, the increase in PCWP may reflect the increase in PAP. If no air is embolized into the catheterized pulmonary artery, PCWP would tend to remain low despite increases in PAP.¹⁴ Thus, Mehta and Sokoll¹⁵ recently found that directly measured LAP either remained the same or decreased during experimental venous air embolism in dogs, whereas RAP increased and exceeded LAP when large volumes of air were infused. Thus, our PCWP data during air embolism probably overestimate true LAP, and paradoxical air embolism may well occur when RAP is acutely elevated during venous air embolism.

The present study suggests, however, that PCWP data are useful clinically, particularly when patients are first placed in the seated position. At this time, 54% of our patients developed acute reductions in PCWP which rendered them at risk for developing paradoxical air embolism. When both RAP and PCWP have been measured and it is known that RAP is lower than PCWP, then systemic air embolism can be considered to be a relatively unlikely hazard unless a large air embolus capable of acutely increasing RAP occurs. Conversely, without pulmonary artery catheter monitoring, we believe that even small episodes of suspected venous air embolism must be regarded as potential causes of paradoxical air embolism and must be treated aggressively.

In light of the findings in this study, we insert pulmonary artery catheters whenever possible in patients scheduled for seated neurosurgical procedures. If the RAP exceeds PCWP in the seated position, we believe that the risk of paradoxical air embolism exists and, if possible, the patient is repositioned such that the incision site is at heart level and the likelihood of air embolism is minimized.

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