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Effect of Body Repositioning after Venous Air Embolism

An Echocardiographic Study

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Background: **Current therapy for massive venous air embolism (VAE) may include the use of the left lateral recumbent (LLR) position, although its effectiveness has been questioned. This study used transesophageal echocardiography to evaluate the effect of body repositioning on intracardiac air and acute cardiac dimension changes.**

Methods: **Eighteen anesthetized dogs in the supine position received a venous air injection of 2.5 ml/kg at a rate of 5 ml/s. After 1 min the dogs were repositioned into either the LLR, LLR 10° head down (LLR-10°), right lateral recumbence, or remained in the supine position.**

Results: **Repositioning after VAE resulted in relocation of intracardiac air to nondependent areas of the right heart. Peak right ventricular (RV) diameter increase and mean arterial pressure decrease were greater in the repositioned animals compared with those in the supine position ($P < 0.05$). Right ventricular diameter and mean arterial pressure showed an inverse correlation ($r = 0.81$). Peak left atrial diameter decrease was greater in the LLR and LLR-10° positions compared with the supine position ($P < 0.05$). Repositioning did not influence peak pulmonary artery pressure increase, and no correlation was found between RV diameter and pulmonary artery pressure. All animals showed electrocardiogram and echocardiographic changes reconcilable with myocardial ischemia.**

Conclusions: **In dogs, body repositioning after VAE provided no benefit in hemodynamic performance or cardiac dimension changes, although relocation of intracardiac air was demonstrated. Right ventricular air did not appear to result in significant RV outflow obstruction, as pulmonary artery pressure increased uniformly in all groups and was not influenced by the relocation of intracardiac air. The combination of increased RV afterload and arterial hypotension, possibly with subsequent RV ischemia rather than RV outflow obstruction by an airlock appeared to be the primary mechanism for cardiac dysfunction after VAE. (Key words: Air embolism. Body repositioning. Myocardial ischemia. Transesophageal echocardiography.)**

VENOUS air embolism (VAE) remains a serious complication of various diagnostic or therapeutic interventions[1–4] or may develop from accidental trauma,[5] decompression sickness,[6] pulmonary barotrauma,[7] or during organ transplantation.[8] Symptoms can vary from asymptomatic transient pulmonary hypertension to complete cardiovascular collapse. If acute cardiac decompensation occurs, right ventricular (RV) failure due to outflow obstruction by the air embolus is considered to be the main mechanism.[9,10] Durant *et al.*[10] postulated that the obstructing air embolus could be displaced and RV failure prevented by repositioning the patient to the left lateral recumbent (LLR) position. The LLR position, either horizontal or head down, is still recommended for VAE treatment[11–13], although its effectiveness has been questioned.[14,15] In a recent study[16] investigating the effect of body repositioning on hemodynamic response after VAE, we showed no difference among various body positions. Because transesophageal echocardiography (TEE) is not only well established for the early detection of VAE[17,18] but allows localization of intracardiac air and determination of cardiac diameter changes, this technique was chosen to determine why repositioning failed to improve cardiac function. This study used TEE to investigate the influence of body repositioning after VAE on relocation and persistence time of intracar-

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diac air as well as the correlation between cardiac dimension changes and concomitant hemodynamic changes.

Methods

All procedures were approved by the University of Texas Animal Welfare Committee and were consistent with the National Institutes of Health *Guidelines for the Care and Use of Laboratory Animals*. Eighteen mongrel dogs (22.8 ± 1.1 kg) of either sex were anesthetized by intravenous administration of 25 mg/kg body weight thiopental sodium, tracheally intubated, and their lungs mechanically ventilated with room air using a volume-cycled respirator (Harvard Apparatus, Boston, MA) at a rate and volume to maintain baseline end-expiratory carbon dioxide pressure at 35–45 mmHg. Anesthesia was maintained with halothane at an inspiratory concentration of 1–2%.

General Preparation

Fluid-filled catheters were placed into the right femoral artery and vein for mean arterial pressure (MAP) monitoring and fluid administration, respectively. We placed a superior vena caval catheter into the right jugular vein for the air injection and a 5-Fr balloon-tipped thermodilution catheter into the pulmonary artery for pressure (PAP) and cardiac output measurements. The pressure monitoring catheters were connected to calibrated pressure transducers (Isotec, Healthdyne Cardiovascular, Irvine, CA), and data were recorded on a six-channel chart recorder (Gould Instruments, Cleveland, OH). Cardiac output was determined in triplicate by injecting 5 ml ice-cold Ringer's solution. Subcutaneous needle electrodes were used to record lead II of the electrocardiogram. A biplane TEE probe (Aloka UST-5233, 5 MHz; Corometrics Medical Systems, Wallingford, CT), connected to an echocardiograph (Aloka 870) was placed in the esophagus. Measurements of RV and left atrial (LA) diameters were obtained from a transverse plane through the LA, aortic root, and RV. All echocardiographic images were videotaped for later analysis. Diameter measurements of three consecutive cardiac cycles at each of the time points noted subsequently were averaged. Right ventricular diameters were determined at end diastole and LA diameters at end systole. No measurements were taken during extrasystoles.

Procedures

After a stabilization period of at least 30 min in the supine position, baseline readings of all parameters were recorded. Thereafter each dog received 2.5 ml air per kilogram body weight at a rate of 5 ml/s through the air-injection catheter followed by a 10-ml saline flush. One minute after the air injection, 100% oxygen ventilation was commenced and the body position of the dogs was changed to either the left lateral recumbent (LLR; n = 4), the left lateral recumbent with the head 10° down (LLR-10°; n = 4), or the right lateral recumbent (RLR; n = 6) position. The remaining dogs stayed in the supine (n = 4) position during the entire experiment. In all animals, RV and LA diameters were measured each minute for the first 15 min and then at 15-min intervals for the rest of the first hour. In ten of these animals, the echo image was videotaped continuously until all visible air was cleared to evaluate persistence time and intracardiac location of the air embolus. In the first hour, vascular pressures were recorded continuously for the first 15 min to obtain the maximal deviation from baseline and thereafter in 15-min intervals. Cardiac output and arterial blood gases were measured every 15 min during the first hour. Thereafter, all variables were measured 120 and 180 min after the air injection. At the conclusion of each experiment, the dogs were killed with an intravenous thiopental overdose and saturated potassium chloride. After death, the hearts were examined for the presence of intracardiac anomalies, including patent foramen ovale.

To determine the effect of position changes in the absence of VAE, hemodynamic variables and RV and LA diameters were measured in eight of the dogs before the experiment. Measurements were taken in the supine position and 5 min after turning the animals successively to each of the following positions: the RLR, the LLR, the LLR-10°, and back to the supine position. Significant hemodynamic or diameter changes due to body positioning alone were not observed after 5 min of repositioning.

Hemodynamic data from seven of the 18 dogs were reported in a recent publication.[16] That report concerned hemodynamic recovery, not acute hemodynamic or cardiac dimension changes after VAE. The seven animals were assigned to the groups as follows: LLR (n = 1), LLR-10° (n = 2), RLR (n = 4).

Statistics

All data are presented as means ± SE. Data analysis was performed on a Macintosh Centris 650 computer

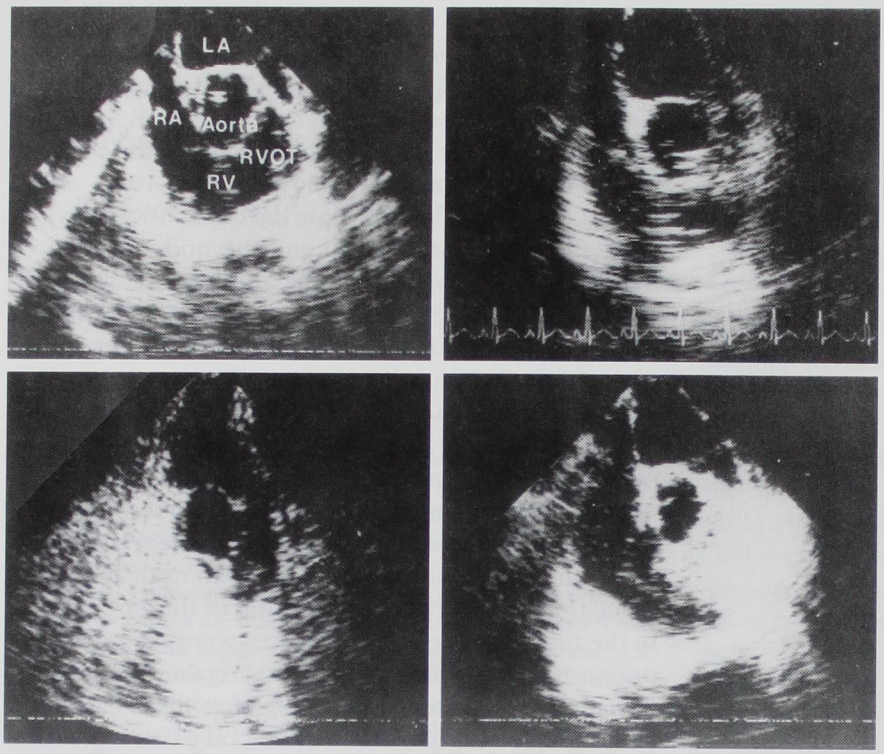

Fig. 1. The effect of repositioning on intracardiac air. (*Top left*) Baseline; left atrium (LA), right atrium (RA), right ventricle (RV), right ventricular outflow tract (RVOT). (*Top right*) One minute after air injection: the animal is positioned supine (SUP), with the air bubble located in the ventral RV, with most air already cleared. (*Bottom right*) One minute after air injection, the dog is repositioned to the right lateral recumbent (RLR) position, with air located in the RVOT. (*Bottom left*) One minute after air injection, the dog is repositioned to the left lateral recumbent (LLR) position, with air located in the RA and RV inflow tract.

using the StatView 4.01 software package (Abacus Concepts, Berkeley, CA). Data from the supine group were compared with those from the LLR, LLR-10°, and RLR groups using analysis of variance, and *post hoc* comparisons were made using a Student's *t* test with Bonferroni correction for multiple comparisons. For nonparametric data, a Mann-Whitney U test was performed. Regression analysis was done using the Sigma Plot 1.02 software package (Jandel Corporation, San Rafael, CA). A value of $P < 0.05$ was considered significant.

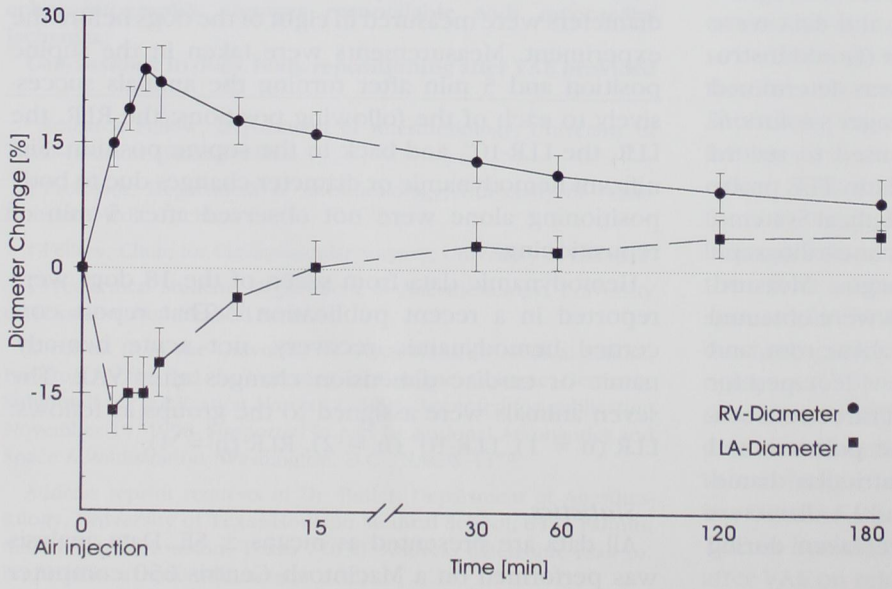

Fig. 2. The time course of right ventricular (RV) and left atrial (LA) diameter change (all groups, n = 18). After the air injection, an instantaneous decrease in LA diameter and increase in RV diameter was observed. Left atrial diameter returned to baseline values within 15 min, whereas RV diameter increase persisted for 3 h. Values are expressed as means ± SE.

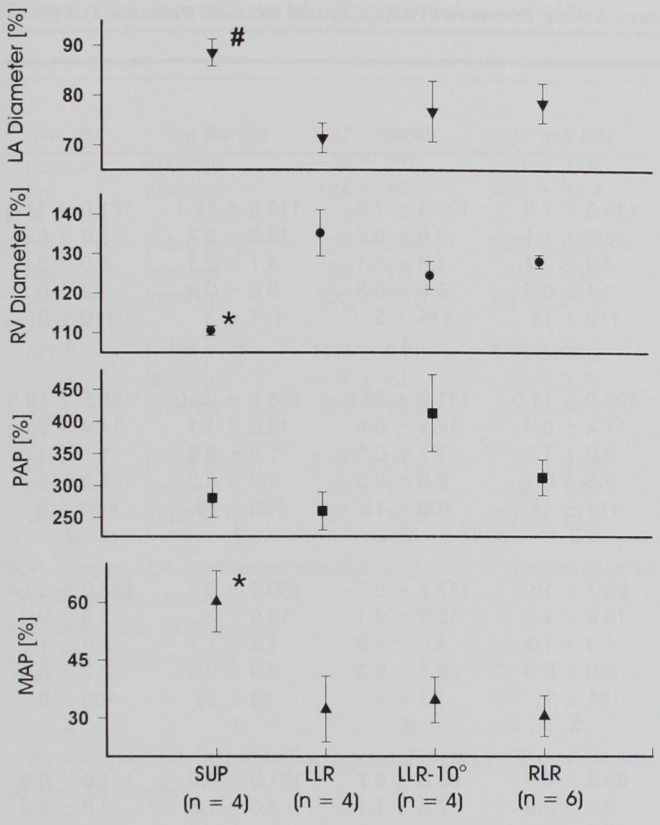

Fig. 3. Maximal change in percentage of baseline values for mean arterial pressure (MAP), mean pulmonary artery pressure (PAP), right ventricular (RV) diameter, and left atrial (LA) diameter (0–5 min). Values are presented as means ± SE. *$P < 0.05$ supine *versus* left lateral recumbent (LLR), LLR with head positioned down 10° (LLR-10°), and right lateral recumbent (RLR) position. #$P < 0.05$ supine *versus* LLR and LLR-10°.

Results

Outcome

Thirteen of the 18 dogs recovered from the VAE. Five deaths were due to asystolic cardiac arrest (confirmed by TEE) that followed a period of gradually deteriorating bradycardia and occurred in all cases within 10 min (6.6 ± 4 min) after the air injection. There were two deaths each in the LLR and RLR groups and one death in the LLR-10° group. All four dogs in the supine group survived.

Transesophageal Echocardiographic Findings

Air appeared in the RV approximately 10–20 s after initiation of the air injection and was visualized in the right atrium, tricuspid area, and RV outflow tract. The major portion of air collected along the anterior wall of the RV. With repositioning to the RLR, the bubbles relocated to the RV outflow tract, which was the nondependent region of the RV in this position. In the animals repositioned to the LLR, the major portion of air was visualized in the right atrium and tricuspid area, which were superior to the other structures of the right heart in this position (fig. 1). The same pattern of air distribution was observed in the animals in the LLR-10° position, although the RV apex was superior in this position. It was impossible to visualize the RV apex because of the accessory lobe of the right lung in dogs.

Most of the air cleared after 5–10 min in the surviving animals, but small amounts of air were visualized in the RV for periods lasting as long as 30 min after the air injection. No difference in RV air persistence time was found among the four groups. However, in the region of the right atrium that was visualized, air persisted for a significantly shorter time in the supine position (n = 3; 1, 3, and 3 min) compared with the LLR, LLR-10°, and RLR positions combined (n = 7; 10.6 ± 2.2 min; $P < 0.05$).

Right ventricular hypokinesis of varying severity was observed in all animals briefly after the air injection. It was most severe in the nonsurviving animals. In the surviving animals, RV function recovered gradually after approximately 10–15 min.

Immediately after the air injection we observed a simultaneous decrease in LA and an increase in RV diameter. Whereas the LA diameter recovered to baseline values within 15 min, the RV diameter increase persisted more than 3 h (fig. 2). The greatest deviation from baseline was observed during the first 5 min. During this time, the maximal increase in RV diameter was significantly greater in the LLR, LLR-10°, and RLR groups compared with the supine group ($P < 0.05$; fig. 3). In the same groups, the decrease in LA diameter was also greater but not statistically significant (fig. 3). However, when the analysis was limited to the comparison among the supine group and the clinically relevant positions (LLR and LLR-10°) the difference was significant ($P < 0.05$; fig. 3).

Hemodynamic Findings

Venous air injections were associated with an immediate increase in PAP and concomitant MAP decrease. Consistent with the TEE findings, the greatest deviation from baseline was observed within the first 5 min. The maximal decrease in MAP during this time was significantly greater in the LLR, LLR-10°, and RLR groups compared with the supine group ($P < 0.05$; fig. 3). There

Table 1. Response of Mean Arterial Pressure (MAP), Mean Pulmonary Artery Pressure (PAP), Central Venous Pressure (CVP), Cardiac Output (CO), and Heart Rate (HR) to VAE

	BL	0–5 min (minimum or maximum)	15 min	30 min	60 min	120 min	180 min
SUP							
MAP (mmHg)	126.3 ± 8.0	77.8 ± 15.8*	111.5 ± 14.2	119.3 ± 7.8	120.3 ± 7.2	116.8 ± 12.1	121.0 ± 11.8
PAP (mmHg)	13.1 ± 1.5	35.5 ± 1.8*	19.2 ± 1.5	16.2 ± 1.1	12.9 ± 0.7	12.3 ± 0.7	13.0 ± 0.6
CVP (mmHg)	3.2 ± 1.3	7.3 ± 3.6*	5.3 ± 2.9	4.9 ± 2.7	4.8 ± 3.1	4.7 ± 3.1	5.0 ± 3.0
CO (L/min)	3.4 ± 0.6	—	3.0 ± 0.5	3.4 ± 0.5	3.6 ± 0.3	3.6 ± 0.4	3.2 ± 0.3
HR (beats/min)	125 ± 11	107 ± 14	113 ± 9	112 ± 11	115 ± 5	111 ± 3	110 ± 3
n	4	4	4	4	4	4	4
LLR							
MAP (mmHg)	118.3 ± 9.5	37.5 ± 9.8*†	90.5 ± 10.5	106.0 ± 11.0	111.0 ± 33.0	138.0 ± 33.0	136.5 ± 19.5
PAP (mmHg)	14.8 ± 1.0	38.2 ± 4.3*	20.1 ± 3.0	17.4 ± 0.4	12.2 ± 0.4	13.5 ± 1.1	14.4 ± 0.6
CVP (mmHg)	1.8 ± 0.4	11.5 ± 3.8*	1.8 ± 0.3	2.0 ± 1.0	1.7 ± 0.5	1.8 ± 0.8	1.1 ± 1.0
CO (L/min)	2.5 ± 0.3	—	2.5 ± 0.1	2.8 ± 0.1	2.8 ± 0.3	3.0 ± 0.5	3.2 ± 0.7
HR (beats/min)	135 ± 9	96 ± 19	102 ± 17	114 ± 14	106 ± 12	109 ± 15	110 ± 8
n	4	4	2	2	2	2	2
LLR-10°							
MAP (mmHg)	109.5 ± 3.6	38.0 ± 7.0*†	95.7 ± 9.2	95.7 ± 16.2	117.3 ± 8.7	130.3 ± 5.5	122.0 ± 10.4
PAP (mmHg)	11.2 ± 2.0	43.5 ± 4.8*	19.9 ± 4	16.9 ± 4.6	12.9 ± 2.1	13.9 ± 2	13.6 ± 0.9
CVP (mmHg)	1.7 ± 1.1	14.8 ± 2.8*	2.6 ± 1.4	1.5 ± 1.3	4.5 ± 4.0	1.3 ± 1.7	1.2 ± 1.6
CO (L/min)	2.3 ± 0.3	—	2.8 ± 0.6	3.0 ± 0.3	2.8 ± 0.3	2.9 ± 0.5	2.7 ± 0.4
HR (beats/min)	120 ± 7.0	82 ± 10	88 ± 3	101 ± 9	97 ± 9	99 ± 12	103 ± 9
n	4	4	3	3	3	3	3
RLR							
MAP (mmHg)	115.7 ± 5.2	35.3 ± 6.4*†	85.5 ± 8.7	83.8 ± 8.8	95.3 ± 6.7	101.5 ± 7.9	111.5 ± 8.9
PAP (mmHg)	13.2 ± 1.0	41.1 ± 4.0*	13.3 ± 1.8	9.1 ± 2.0	7.6 ± 1.1	7.0 ± 1.6	8.8 ± 0.9
CVP (mmHg)	3.0 ± 0.5	15.4 ± 3.7*	1.7 ± 0.8	2.5 ± 1.2	1.1 ± 0.3	1.0 ± 0.6	1.4 ± 0.7
CO (L/min)	2.9 ± 0.4	—	3.1 ± 0.6	3.0 ± 0.3	2.9 ± 0.4	2.6 ± 0.4	2.7 ± 0.4
HR (beats/min)	133 ± 17	105.2 ± 14.2	116 ± 18	105 ± 12	100 ± 10	103 ± 8	112 ± 10
n	6	6	4	4	4	4	4

SUP = supine; LLR = left lateral recumbence; LLR-10° = LLR with head 10° down; RLR = right lateral recumbence; VAE = venous air embolism.

* $P < 0.05$ *versus* baseline.

† $P < 0.05$, SUP *versus* LLR, LLR-10°, and RLR.

was no significant difference for the maximal PAP increase in the first 5 min among the four groups (fig. 3). Central venous pressure (CVP) increased immediately after the air infusion in all four groups, although the differences among the groups were not significant (table 1). No cardiac output measurements were conducted immediately after the air infusion under the assumption that RV air would interfere with the mixing of blood and injectate, causing considerable error. At 15 min, when the first cardiac output measurements were taken, cardiac output had returned to at least 80% of baseline in all animals and we found no significant difference among the groups (table 1). However, because all deaths occurred within 10 min, we could not obtain cardiac output measurements in the nonsurviving animals.

In the surviving animals MAP, PAP, and CVP returned to at least 80% of baseline within 60 min, with no significant difference related to position.

Electrocardiographic, Blood Gas Analysis, and Autopsy Results

Electrocardiographic changes occurred within the first 5 min after the air injection and included S-T segment depression or elevation and T-wave inversion. Most of the animals showed arrhythmias such as ventricular or supraventricular extrasystole and bradycardia. There were no significant differences among the groups.

Blood gas analysis showed a decrease in *p*H and a significant increase in carbon dioxide pressure after VAE, with no significant differences among groups (table 2).

Table 2. Response of Arterial *p*H, p_{CO_2}, and p_{O_2} to VAE

		BL*	15 min†	30 min†	60 min†	120 min†	180 min†
SUP							
*p*H	(−log [H⁺])	7.37 ± 0.01	7.26 ± 0.02‡	7.29 ± 0.02‡	7.33 ± 0.02	7.35 ± 0.03	7.37 ± 0.02
p_{CO_2}	(mmHg)	39 ± 2.0	51 ± 3.5‡	46 ± 2.8‡	44 ± 2.6	44 ± 2.8	41 ± 1.3
p_{O_2}	(mmHg)	124 ± 16.5	450 ± 89.4	453 ± 87.0	367 ± 126.5	551 ± 86.5	554 ± 59.1
LLR							
*p*H	(−log [H⁺])	7.36 ± 0.04	7.22 ± 0.05	7.30 ± 0.04	7.34 ± 0.008	7.33 ± 0.01	7.32 ± 0.02
p_{CO_2}	(mmHg)	38 ± 1.3	49 ± 6.9‡	42 ± 1.8	39 ± 0.4	42 ± 0.9	39 ± 1.4
p_{O_2}	(mmHg)	123 ± 22.0	547 ± 32.0	575 ± 39.0	557 ± 44.5	577 ± 15.5	562 ± 12.5
LLR-10°							
*p*H	(−log [H⁺])	7.39 ± 0.02	7.26 ± 0.02	7.30 ± 0.04	7.34 ± 0.03	7.34 ± 0.03	7.35 ± 0.05
p_{CO_2}	(mmHg)	37 ± 2.9	51 ± 3.7‡	43 ± 4.8	41 ± 4.0	42 ± 5.3	43 ± 6.5
p_{O_2}	(mmHg)	133 ± 18.5	400 ± 54.9	424 ± 32.7	480 ± 52.1	445 ± 46.0	447 ± 47.9
RLR							
*p*H	(−log [H⁺])	7.37 ± 0.03	7.30 ± 0.02	7.35 ± 0.02	7.37 ± 0.02	7.40 ± 0.03	7.38 ± 0.02
p_{CO_2}	(mmHg)	37 ± 1.7	42 ± 1.0‡	41 ± 1.1‡	35 ± 1.1	35 ± 1.7	38 ± 2.5
p_{O_2}	(mmHg)	151 ± 16.3	403 ± 77.6	407 ± 81.5	401 ± 76.7	435 ± 69.3	423 ± 70.9

SUP = supine; LLR = left lateral recumbence; LLR-10° = LLR with head 10° down; RLR = right lateral recumbence.

* Ventilation with room air.

† Ventilation with 100% oxygen.

‡ $P < 0.05$ *versus* baseline.

At autopsy, no intracardiac anomalies were found in any of the 18 dogs.

Discussion

The TEE results showed that repositioning caused intracardiac air to relocate to nondependent parts of the right heart, but this relocation did not result in better hemodynamic performance. Furthermore, there were greater changes in cardiac dimensions after repositioning. To our knowledge, this is the first report of echo imaging of both air localization and cardiac dimensions during acute VAE.

The recommendation for repositioning after VAE arises from the work by Durant and colleagues more than 40 yr ago.[10,19,20] Using biplane angiocardiography, these investigators described an obstructing air lock in the RV outflow tract in dogs that were either left in the supine position or turned to the RLR position.[19] They concluded that hemodynamic compromise was primarily due to this air lock and suggested that LLR repositioning after VAE, notably Durant's maneuver, would displace the bubble away from the RV outflow tract and result in RV output recovery. In our studies, we did not find any evidence of improved cardiac function with repositioning. Furthermore, the RV air did not appear to result in significant RV outflow tract obstruction because PAP uniformly increased immediately after VAE independent of body position. This finding corresponds with the results of other investigators[19,21] and was consistent even with the application of considerably higher VAE doses of 3.0–7.5 ml/kg. In fact, Oppenheimer *et al.*[19] noted the contradiction between the observed PAP increase independent of body position and the expected decrease in PAP implied by the concept of a hemodynamically relevant air lock.

If the air does not obstruct the RV outflow tract, then repositioning would not be expected to be beneficial. This is consistent with our data, which suggest that repositioning not only failed to improve cardiac function but may in fact be detrimental. Our data suggest that right atrial air persistence time might be increased in dogs repositioned to the LLR, possibly because of venous inflow obstruction by air relocated to the right atrium and RV inflow tract. The shorter persistence time of right atrial air in the supine group was probably due to a more physiologic venous return flow in this position. Aside from the worse hemodynamic and dimension changes seen in the repositioned dogs, all of the acute deaths occurred in these dogs as well.

In our experiments, the possible occurrence of myocardial ischemia in the early phase of VAE was indicated by typical electrocardiographic changes and acute RV hypokinesis and dilatation. Myocardial ischemia has

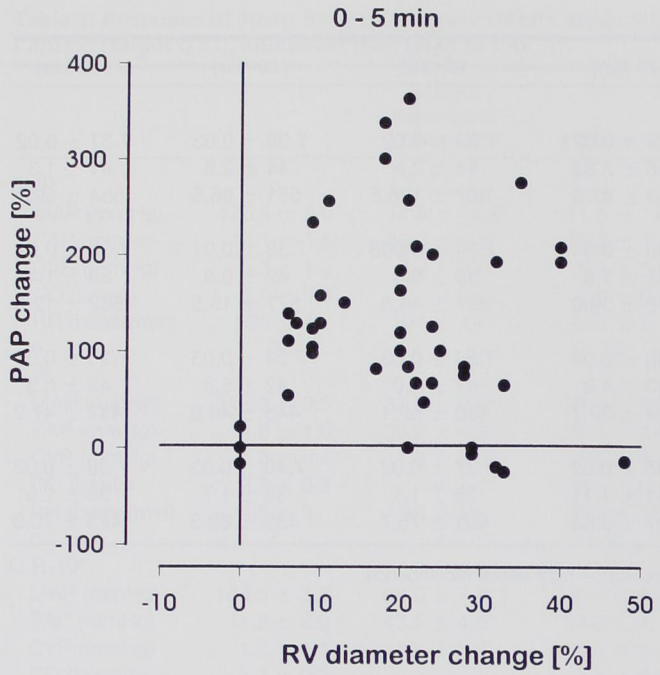

Fig. 4. Mean pulmonary artery pressure (PAP) and right ventricular (RV) diameter change (0–5 min, all groups). Regression statistics: $y = 56.32 + 2.54\,x$; $r^2 = 0.12$.

been acknowledged as contributing to cardiac decompensation after VAE by various authors,[4,14,21] including Durant *et al.*[10,19] Holt *et al.*[21] found improved cardiac function after VAE by increasing coronary perfusion pressure with intermittent cross clamping of the ascending aorta. Myocardial ischemia in VAE is thought to occur due to severe arterial hypotension and increased CVP, impairing coronary blood flow due to the diminished pressure gradient between the coronary arteries and coronary sinus. Simultaneously, RV oxygen demand and thus its risk for ischemia are markedly increased by acute pulmonary hypertension. Although the increased afterload is generally regarded as the cause of the acute RV dilatation after VAE,[10,14] we found no relation between PAP and RV dimension change during the acute phase (fig. 4). However, we did note an inverse relation between RV diameter and MAP (fig. 5), which implicates RV ischemia as a contributing factor to acute RV dysfunction after VAE. The detrimental effect of repositioning appears to be mediated by arterial hypotension because MAP was significantly lower and RV diameter increase significantly greater in the groups that were repositioned, whereas PAP and CVP were not different among the groups. Arterial hypotension after VAE was due primarily to decreased left ventricular filling, indicated by an acute decrease in LA diameter observed in all animals (fig. 2). Echocardiographic assessment of left ventricular function was not possible, because of the accessory lobe of the right lung in dogs that blocks acoustic transmission from the esophagus to the left ventricle. However, if left ventricular hypokinesis were responsible for arterial hypotension and RV dilatation, then we would expect to observe an increase in LA diameter, due to increased left atrial pressure.

Acute arterial hypotension after VAE may be partially the result of autonomic cardiovascular reflexes. Activation of vagal afferents in the atria or ventricles by volume or pressure loading can result in hypotension and decreased sympathetic nerve activity.[22,23] Aibiki *et al.*[24] studied the role of vagal afferents in hypotension induced by VAE. They found that MAP decreased less and survival improved in vagotomized animals.[24] Decrease in MAP after VAE may also be due to decreased venous return.[14] These findings support the conclusion that arterial hypotension and subsequent myocardial ischemia may be significant contributors to cardiac failure after VAE.

Previous studies have suggested that VAE may stimulate the release of various metabolic products of arachi-

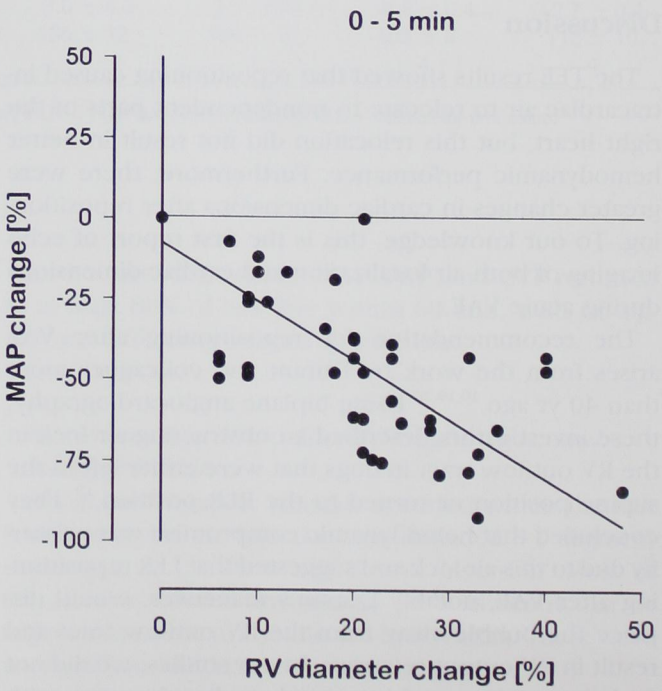

Fig. 5. Mean arterial pressure (MAP) and right ventricular (RV) diameter change (0–5 min, all groups). Regression statistics: $y = -7.51 - 1.82x$; $r^2 = 0.66$.

donic acid, including thromboxane B_2 and leukotrienes.[25,26] Such mediators have vasoactive features that may contribute to the pulmonary hypertension seen in this study. Fukushima *et al.*[25] found that thromboxane synthase inhibition did not result in reduction of VAE-induced pulmonary hypertension, suggesting a principal role of the leukotrienes. However, pharmacologic blockade of mediator-induced pulmonary vasoconstriction appears to be an important approach for future VAE therapy.

Our investigation focused on the acute phase after VAE, because the most pronounced changes in vascular pressures and cardiac dimensions were observed within the first 5 min. The significance of the acute phase is of particular interest because all observed deaths occurred within the first 10 min.

Our data suggest that repositioning provided no benefit in hemodynamic performance or cardiac dimension changes after VAE. This occurred despite TEE demonstration that the main portion of air relocated to the nondependent part of the right heart. However, we found no indication that RV air resulted in significant RV outflow obstruction in any body position. We conclude that the combination of acute increase in RV afterload and arterial hypotension with subsequent RV ischemia are the major contributing factors to cardiac dysfunction after VAE. Future directions for VAE treatment thus should consider measures to limit pulmonary vasoconstriction and arterial hypotension rather than body repositioning. Further investigation of the role of myocardial ischemia in acute VAE appears to be warranted.

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