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DOES NOT AFFECT THE HEMODYNAMIC
RESPONSE TO VENOUS AIR EMBOLISM IN
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Body Position Does Not Affect the Hemodynamic Response to Venous Air Embolism in Dogs

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Current therapy for massive venous air embolism (VAE) includes the use of the left lateral recumbent (LLR) position. This recommendation is based on animal studies, conducted 50 yr ago, which looked primarily at survival. Little is known, however, about the concomitant hemodynamic response after VAE in various body positions. The purpose of this study was to investigate the hemodynamic and cardiovascular changes in various body positions after VAE. Twenty-two mechanically ventilated supine mongrel dogs received a venous air infusion of 2.5 mL/kg at a rate of 5 mL/s. One minute after the infusion, 100% oxygen ventilation was commenced and the body position of the dogs was changed to either the LLR ($n = 6$), the LLR with the head 10° down (LLR-10°; $n = 6$) or the right lateral recumbent (RLR; $n = 5$) position. Five dogs were maintained in the supine position (SUP; $n = 5$). One dog died in every group except in the SUP group, where all the dogs recovered. There were no significant differences among the various body positions in terms of heart rate, mean arterial pressure, pulmonary artery pressure, central venous pressure, left ventricular end-diastolic pressure, or cardiac output. The acute hemodynamic changes occurring during the first 5–15 min after VAE recovered to 80% of control within 60 min. Our data suggest that body repositioning does not influence the cardiovascular response to VAE. Specifically, our data do not support the recommendation of repositioning into the LLR position for the treatment of VAE.

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Venous air embolism (VAE) may develop as a complication of various diagnostic or therapeutic maneuvers or may result from accidental trauma or decompression sickness (1–8). Depending on the amount and rate of air entry, the physiologic responses to VAE can range from mild, transient pulmonary hypertension to cardiovascular collapse or arterial air embolism (9). Current therapy for VAE includes 100% oxygen ventilation and cessation of nitrous oxide (where applicable) to reduce bubble size as well as withdrawal of air from central veins, the right atrium, and pulmonary arteries (4,5,10–13). In cases of massive VAE that are not responsive to these measures alone, additional maneuvers, such as the left lateral recumbent (LLR) position, either horizontal or head down, external cardiac massage, and recompression therapy, with or without hyperbaric oxygen, may be necessary (4,5,10–12,14–16). The recommendation of the LLR position, known as "Durant's maneuver," is based primarily on animal studies performed by Durant et al. (14) beginning in 1947. Using survival as the primary measure of outcome, they found the LLR position to be superior to other body positions. As recently as 1992, the LLR position has been described as a cornerstone in the management of VAE (5), even though the effectiveness of this maneuver has been questioned (12,17). Apart from survival outcome studies, there has been little investigation of the concomitant hemodynamic responses to body positioning after VAE. Therefore, the purpose of this study was to investigate the influence of body positioning on the hemodynamic and cardiovascular changes after VAE.

Methods

All procedures were approved by the University of Texas Animal Welfare Committee and were consistent with the National Institutes of Health *Guide for the Care and Use of Laboratory Animals.* Twenty-two mongrel dogs (19.2 ± 3.5 kg) were anesthetized with intravenous administration of 25 mg/kg thiopental, intubated, and mechanically ventilated with room air using

a volume-cycled respirator (Harvard Apparatus, Boston, MA). Anesthesia was maintained with halothane at an inspired concentration of 1%–2%. We placed fluid-filled catheters into the left femoral artery and vein for mean arterial pressure (MAP) monitoring and fluid administration, respectively, and into the left ventricle via the right carotid artery for measurement of left ventricular end-diastolic pressure (LVEDP). Via the right jugular vein we placed a 7 Fr Swan-Ganz thermodilution catheter into the pulmonary artery for pressure (PAP) and cardiac output (CO) determinations as well as a superior vena caval catheter for the air infusions. The pressure monitoring catheters were connected to pressure transducers (Isotec™; Healthdyne Cardiovascular Inc., Irvine, CA) and data were recorded on an eight-channel chart recorder (Grass Instrument Co., Quincy, MA). CO was determined in triplicate by injection of 5 mL ice-cold Ringer's solution. Subcutaneous needle electrodes were used to record lead II of the electrocardiogram (ECG). End-tidal carbon dioxide concentration (ET_{CO_2}) was measured with a capnograph (Datex; Puritan-Bennett Corp., Wilmington, MA).

After a stable baseline (BL) period of at least 30 min in the supine (SUP) position, each dog received 2.5 mL of air per kg body weight at a rate of 5 mL/s through the air infusion catheter. One minute after the air infusion, 100% oxygen ventilation was commenced and the body position of the dogs was changed to either the LLR ($n = 6$), the LLR with the head 10° down (LLR-10°; $n = 6$), or the right lateral recumbent (RLR; $n = 5$) position. Five dogs remained in the SUP position ($n = 5$) throughout the entire experiment. The four groups were randomly selected. We collected data for 3 h after the air infusion. During the first hour, vascular pressures, ECG, and ET_{CO_2} were recorded every 5 min, and CO and arterial blood gases were measured every 15 min. Thereafter, we measured all variables at 120 and 180 min after the air infusion. At the end of the experiment the dogs were euthanized with an intravenous thiopental overdose and saturated potassium chloride. We then examined the hearts for the presence of intracardiac anomalies, especially patent foramen ovale. Lung edema was measured using a gravimetric analysis of extravascular fluid (EVF) with correction for residual blood (18). Lung EVF is expressed as the unitless blood-free (wet weight-dry weight)/dry weight ratio.

To determine the effect of position changes in the absence of VAE, we measured hemodynamic variables in 10 of the dogs in the SUP position and 5 min after turning them successively to each of the following positions: the RLR, the LLR, the LLR-10°, and back to the SUP position. We did not observe significant hemodynamic changes due to body positioning.
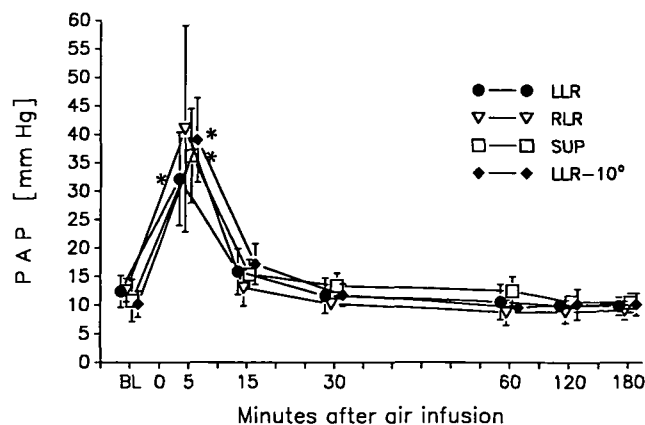

Figure 1. Response of pulmonary artery pressure (PAP; mean ± sd) to venous air infusion. LLR = left lateral recumbent; RLR = right lateral recumbent; SUP = supine position; LLR-10° = left lateral recumbent with the head 10° down; BL = baseline; *$P < 0.05$ vs BL.

Hemodynamic data are presented as mean ± sd. Recovery time after the air infusion was defined as the time needed for each hemodynamic variable to return to within 80% of BL.

Data analysis was carried out on a Macintosh Centris 650 computer using the StatView® 4.01 software package (Abacus Concepts Inc., Berkeley, CA). Data from the different groups were compared by repeated measures analysis of variance and F test. Post hoc comparison was made using a Student's t statistic with a Bonferroni correction for multiple comparisons. A value of $P < 0.05$ was considered significant.

Results

Nineteen of the 22 dogs recovered from the air infusion. In the SUP group all five dogs survived, whereas in each of the other three groups one dog died. At autopsy, we did not observe intracardiac anomalies in any of the 22 dogs. Lung EVF was not increased significantly in any of the animals. Mean EVF was 3.75 ± 0.50, which is a normal value for dog lungs (19). After the air infusion, the ECGs showed several changes in the ST segments (i.e., ST segment depression, T inversion) as well as arrhythmias, such as ventricular and supraventricular extrasystoles and periods of bradycardia or tachycardia. In some cases the ECG changes had resolved as early as 10 min after the air infusion, whereas in some others they were still present at the conclusion of the experiment. There were no differences in the ECG findings between the various body positions.

Figures 1–3 show the typical responses to VAE for the different variables: VAE was followed by a significant increase in PAP (Figure 1) and a concomitant decrease in MAP (Figure 2). Both PAP and MAP had returned to baseline levels within 60 min after the air
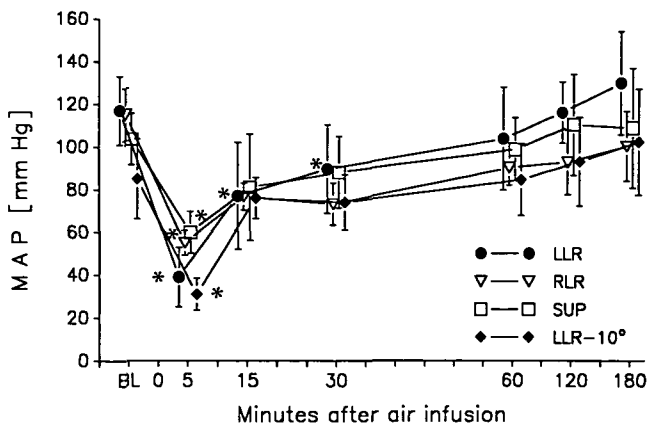
Figure 2. Response of mean arterial pressure (MAP; mean ± SD) to venous air infusion. LLR = left lateral recumbent; RLR = right lateral recumbent; SUP = supine position; LLR-10° = left lateral recumbent with the head 10° down; BL = baseline; *$P < 0.05$ vs BL.

Figure 3. Response of end-tidal carbon dioxide concentration (ET-CO2; mean ± SD) to venous air infusion. LLR = left lateral recumbent; RLR = right lateral recumbent; SUP = supine position; LLR-10° = left lateral recumbent with the head 10° down; BL = baseline; *$P < 0.05$ vs BL.
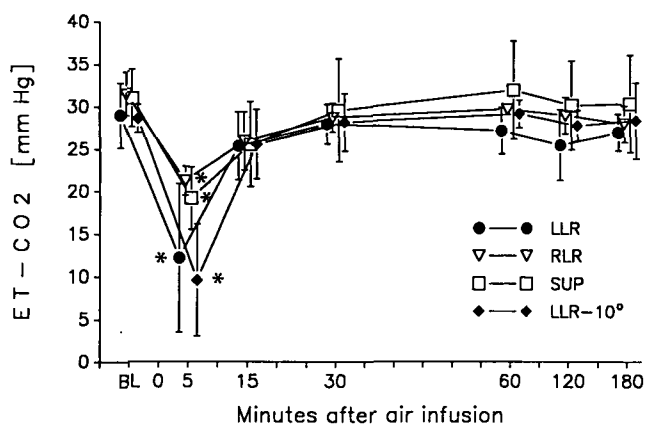
infusion. In all groups ET_{CO_2} decreased significantly during the first 5 min and returned to baseline levels within 30 min (Figure 3). Minute ventilation was maintained constant throughout the experiments. Slight decreases in LVEDP in the RLR and LLR groups compared with the SUP and LLR-10° groups did not reach statistical significance (Table 1). Compared with BL levels, we did not observe significant changes in CO (Table 1). For all of the variables there were no significant differences between the various body positions (Figures 1–3, Table 1). In Figure 4 the recovery times for all variables are shown: almost all acute changes noted during the first 5 to 15 min after the VAE were recovered to 80% of BL level within 60 min. There were no significant differences for the recovery times in the four body positions.

Discussion

The results of this study suggest that body repositioning does not influence the cardiovascular responses to VAE. We did not observe significant differences between the various body positions in either the magnitude of change or the recovery times of the hemodynamic variables. Specifically, these findings do not indicate a benefit of LLR positioning as part of the management of massive VAE.

The suggestion of therapeutic value of the LLR was originally based on the studies by Durant et al. in the 1940s and 1950s (14,20). They found that dogs receiving venous infusions of 25 to 150 mL of air had higher survival rates when lying in the LLR position at the time of VAE compared with dogs lying in the RLR or in the SUP position. Furthermore, they observed greater survival in dogs that were turned from the SUP position into the LLR position after the air infusion. They demonstrated that VAE forms an air trap in the right ventricular outflow tract that acts as an effective circulatory block. They postulated that in the LLR position the right ventricular outflow tract lies inferior to the body of the right ventricle, thus displacing the air trap by the principle of air buoyancy. In additional studies they found that the survival rates in dogs receiving venous air infusions of 5.0 or 7.5 mL/kg in the SUP position were 95% and 30%, respectively (21). When dogs were receiving 7.5 mL/kg of air while lying in the LLR position at the time of infusion or were repositioned into the LLR within 1 min after the air infusion, the survival rates increased statistically significantly to 62.5% and 60.1%, respectively.

Although the pathophysiology of VAE has been well documented (9,12,14,17,20–23), there has been little investigation into the hemodynamic benefits of body positioning for the treatment of VAE. This is important, because the efficacy of this therapy has been questioned (12,17). Adornato et al. (17) investigated the pathophysiology of slow venous air infusion (0.01–2.00 mL·kg⁻¹·min⁻¹) compared with bolus (25–200 mL) injections of air in dogs weighing between 10 and 21 kg. They concluded that there were no differences in physiologic modalities, regardless of whether the dog was in the SUP, prone, erect, left lateral, or right lateral position at slow infusion rates. However, they reported that dogs better tolerated the same infusion rates when lying in a head-down position. In conclusion, they postulated different mechanisms causing circulatory collapse: 1) in slow air infusion, shock was due to a decrease in peripheral resistance induced by a sympatholytic reflex, whereas 2) a bolus of air resulted in failure of cardiac output due to an air lock in the right ventricle. Alvaran et al.

Table 1. Response of Cardiac Output, LVEDP, Pulmonary Vascular Resistance, Po$_2$, and Pco$_2$ to Venous Air Embolism

	BL	5'	15'	30'	60'	120'	180'
LLR							
CO (L/m^2)	2.64 ± 0.48		2.63 ± 0.50	2.77 ± 0.47	2.71 ± 0.69	2.29 ± 0.51	2.41 ± 0.52
LVEDP (mm Hg)	2.3 ± 2.0	-4.1 ± 4.0	-2.0 ± 2.2	-2.6 ± 1.7	-1.0 ± 1.6*	-1.0 ± 2.4	3.0 ± 3.2
PVR (dynes·s·cm^{-5})	308 ± 101		556 ± 89	422 ± 119	308 ± 137	297 ± 74	190 ± 63
Po$_2$ (mm Hg)	151 ± 33		318 ± 143	388 ± 59	393 ± 58	379 ± 71	345 ± 117
Pco$_2$ (mm Hg)	37.0 ± 5.3		44.6 ± 5.7	40.1 ± 6.6	35.8 ± 5.0	34.0 ± 3.8	33.4 ± 4.9
RLR							
CO (L/m^2)	3.03 ± 0.97		3.23 ± 0.91	3.08 ± 0.66	2.83 ± 0.82	2.56 ± 0.74	2.56 ± 0.93
LVEDP (mm Hg)	5.6 ± 3.7	0.1 ± 5.5	-1.0 ± 4.3	-0.8 ± 4.8	1.5 ± 6.8	1.1 ± 3.5	0.4 ± 4.2
PVR (dynes·s·cm^{-5})	215 ± 162		384 ± 180	248 ± 129	315 ± 245	346 ± 273	283 ± 251
Po$_2$ (mm Hg)	139 ± 14		314 ± 80	304 ± 76	312 ± 83	338 ± 92	330 ± 88
Pco$_2$ (mm Hg)	36.4 ± 2.4		43.4 ± 2.8	40.9 ± 2.1*	37.3 ± 2.5	35.8 ± 2.6	38.5 ± 3.9
SUP							
CO (L/m^2)	2.88 ± 0.81		2.78 ± 0.80	3.21 ± 0.71	3.23 ± 0.33	2.79 ± 0.16	2.42 ± 0.25
LVEDP (mm Hg)	5.0 ± 2.4	6.4 ± 2.5	6.2 ± 2.6	6.5 ± 2.9	7.0 ± 2.3	5.3 ± 2.1	6.0 ± 2.5
PVR (dynes·s·cm^{-5})	248 ± 121		368 ± 141	307 ± 77	238 ± 89	198 ± 74*	180 ± 81
Po$_2$ (mm Hg)	127 ± 21		372 ± 47	384 ± 44	387 ± 51	372 ± 80	372 ± 84
Pco$_2$ (mm Hg)	35.5 ± 3.7		45.8 ± 7.8	42.9 ± 4.3	38.3 ± 3.7	36.1 ± 4.4	36.6 ± 4.1
LLR-10°							
CO (L/m^2)	2.05 ± 0.34		2.01 ± 0.33	2.22 ± 0.47	2.44 ± 0.45	2.35 ± 0.73	1.99 ± 0.42
LVEDP (mm Hg)	2.6 ± 1.1	2.1 ± 3.7	2.7 ± 3.0	0.7 ± 3.4	0.3 ± 3.2	1.4 ± 3.7	3.4 ± 2.1
PVR (dynes·s·cm^{-5})	292 ± 92		573 ± 149*	424 ± 180	323 ± 143	316 ± 114	292 ± 116
Po$_2$ (mm Hg)	150 ± 32		346 ± 89	343 ± 83	338 ± 81	296 ± 119	333 ± 84
Pco$_2$ (mm Hg)	38.8 ± 4.3		47.7 ± 6.8	43.6 ± 7.0	39.9 ± 5.4	39.4 ± 7.2	39.4 ± 6.9

Values are mean ± sd.
BL = baseline; ' = minutes after the air infusion; CO = cardiac output; LVEDP = left ventricular end-diastolic pressure; PVR = pulmonary vascular resistance; Po$_2$ = arterial oxygen partial pressure; Pco$_2$ = arterial carbon dioxide partial pressure; LLR = left lateral recumbent; RLR = right lateral recumbent; SUP = supine position; LLR-10° = left lateral recumbent with the head 10° down.
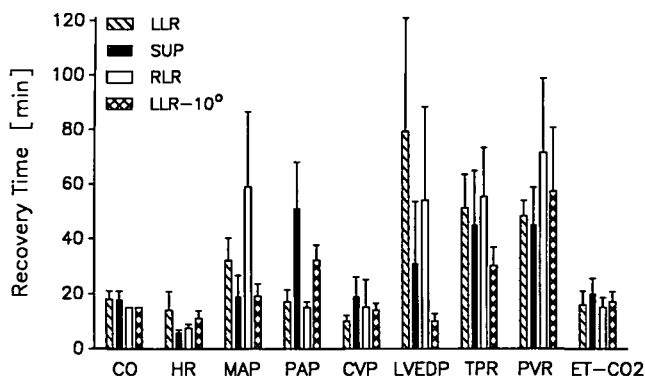* $P < 0.05$ vs BL.

Figure 4. Recovery times (mean ± SEM) for all hemodynamic variables. Recovery time was defined as the time needed for each hemodynamic variable to return to within 80% of baseline. LLR = left lateral recumbent; RLR = right lateral recumbent; SUP = supine position; LLR-10° = left lateral recumbent with the head 10° down; CO = cardiac output; HR = heart rate; MAP = mean arterial pressure; PAP = pulmonary artery pressure; CVP = central venous pressure; LVEDP = left ventricular end-diastolic pressure; TPR = total peripheral resistance; PVR = pulmonary vascular resistance; ET-CO2 = end-tidal carbon dioxide concentration.

(12) compared the effects of external cardiac massage, intracardiac aspiration, or LLR position on survival rates and resuscitation times in dogs receiving 15 mL/kg air. There were no differences in the survival rates between the groups, although the time required for successful resuscitation was significantly shorter when air was aspirated. They concluded that positioning the body in the LLR position alone may not be sufficient therapy for massive VAE.

In our study, we did not find the LLR position to be superior to the other body positions in terms of survival rates, hemodynamic response, or recovery times. However, because death due to massive venous air embolism is often due to cardiovascular collapse, mortality cannot be separated from the hemodynamic changes after VAE. In other words, if there were differences in the survival rates between different body positions, one would also expect differences in the hemodynamic responses. We therefore chose an air dose which was not associated with as high a mortality as previously evaluated (14,22). Since the average lethal dose of air in dogs has been found to be between 3.0 and 7.5 mL/kg (21,22,24–26), we selected a dose of 2.5 mL/kg. A rapid rate of infusion (5 mL/s) was selected to mimic the clinical situation of massive VAE. After the infusion of this dose we observed profound hypotension associated with significant increases in PAP, which were the same for the various body positions. Within 1 h these acute hemodynamic changes had returned to BL levels. Since we did not observe any hemodynamic benefit of body repositioning after VAE, our results

suggest that the recommended LLR position for the treatment of VAE should be questioned.

The purpose of body positioning is to manipulate the air by the principle of buoyancy in a nondependent region prior to entering the pulmonary vasculature. On the other hand, body positioning may be of therapeutic value when the air has already reached the pulmonary circulation. Chang et al. (27) investigated the effects of gravity and buoyancy on the distribution of blood and air bubbles within the pulmonary vasculature. In prone as well as in SUP dogs they demonstrated a significant shift of blood perfusion toward the dependent areas of the lung after VAE, suggesting an important role of buoyancy. Transesophageal echocardiography allows the visualization of the bubbles themselves (28) and thus may guide titration of body position to minimize pulmonary embolization. We are currently investigating this modality.

Besides body positioning there are many other factors that can affect the hemodynamic response to VAE, including rate of air entry, CO, or gas composition (29). Accordingly, Durant et al. (14) described one dog that survived a total of 1000 mL of air injected in amounts of 100 mL every 5 to 10 min. At slow infusion rates between 0.4 and 0.6 mL·kg^{-1}·min^{-1}, Adornato et al. (17) described an increase in CO which compensated for the decrease in blood pressure. This compensation began to fail at higher rates (1.2–1.8 mL·kg^{-1}·min^{-1}); above approximately 1.8 mL·kg^{-1}·min^{-1}, profound shock occurred and most of the dogs did not recover. Interestingly, in the present study one RLR dog (19.7 kg) with a high BL CO of 4.3 L/min demonstrated minimal effects after receiving the air infusion. There was only a moderate increase in PAP from 12 to 21 mm Hg associated with a decrease in MAP from 105 to 80 mm Hg, indicating a fast clearance of the air emboli. This observation is in agreement with the findings of Martin et al. (30), who demonstrated an inverse relationship between CO and clearance time of venous air emboli from the superior vena cava. Neither their study nor the present experiments addressed the question of whether manipulations of CO would have any value in the management of VAE, however. In the present study we did not detect significant changes in CO. We suspect that there were alterations in the CO, but they had resolved by 15 min after the air infusion. Measurements of CO taken immediately after the VAE would be subject to considerable error due to the probable incomplete mixing of injectate and blood.

In conclusion, the results of this study demonstrate that repositioning into the LLR position failed to demonstrate any positive hemodynamic effect on VAE in dogs. Because changing body positions during surgical procedures may pose some risk to patients, the potential benefits should first be weighed.

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