Relationship of fat embolism to haemodynamic and echocardiographic changes during cemented arthroplasty

Patricia Murphy MD FRCPC,
Gerald Edelist MD FRCPC,
Robert J. Byrick MD FRCPC,
J. Colin Kay,
J. Brendan Mullen MD FRCPC

Purpose: Pressurisation of the medullary cavity during cemented arthroplasty causes "intravasation" of marrow fat. The purpose of this study was to examine the relationship between the amount of pulmonary intravascular fat and the haemodynamic and echocardiographic changes.

Methods: Anaesthetised mongrel dogs (n=16) underwent bilateral cemented arthroplasty (BCA) to create a large embolic load. Haemodynamic measurements included blood pressure (BP), pulmonary artery pressure (PAP), right atrial pressure and cardiac output as well as transoesophageal echocardiographic (TEE) assessment of right ventricular (RV) and left ventricular (LV) areas. Using quantitative morphometry on postmortem lung specimens, the proportion of lung tissue occluded by fat was measured.

Results: Mean BP decreased within one minute of BCA, coinciding with the appearance of echogenic material in the RV. The RV area increased by 56% ($P < 0.05$) and LV area decreased by 34% ($P < 0.05$) while PAP increased from 15 ± 3 mmHg to 39 ± 10 mmHg within one minute ($P < 0.001$). The PAP remained elevated throughout the study (30 min). Stroke volume decreased in 14/15 dogs, yet cardiac output was maintained by increased heart rate. There was a curvilinear relationship ($r=0.87$) between the maximum increase in PAP and the proportion of lung occupied by fat.

Conclusion: In this model, stroke volume decreased within one minute of BCA when fat embolism accompanied prosthesis insertion. The TEE detected an increased RV area and reduced LV area associated with decreased stroke volume. The maintenance of cardiac output after intraoperative fat embolism depends primarily on the ability to increase heart rate.

Objectif : La pressurisation de la cavité médullaire pendant l'arthroplastie cimentée provoque l'«intravasation» de la graisse médullaire dans la circulation. Cette étude visait à examiner la relation entre la quantité de graisse intravasculaire interceptée au poumon et ses conséquences hémodynamiques et échocardiographiques.

Méthodes : Des chiens de race commune anesthésiés ont subi (n=16) une arthroplastie cimentée bilatérale (ACB) dans le but provoquer une forte décharge embolique. Les mesures hémodynamiques comprenaient la tension artérielle (TA), la pression artérielle pulmonaire (PAP), la mesure par l'échographie transoesophagienne (ÉTO) des surfaces du ventricule droit (VD) et du ventricule gauche (VG). La morphométrie quantitative réalisée sur des spécimens de poumons isolés a permis de mesurer la proportion du tissu pulmonaire obstrué par la graisse.

Résultats : La TA moyenne a diminué en moins d'une minute de l'ACB, ce qui coïncidait avec l'apparition de matériel échogène dans le VD. La surface du VD a augmenté de 56% ($P < 0,05$) et celle du VD a diminué de 34% ($P < 0,05$) alors que la PAP augmentait de 15 ± 3 mmHg à 39 ± 10 mmHg en moins d'une minute ($P < 0,001$). La PAP demeurait élevée pendant toute l'étude (30 min). Le volume d'éjection diminuait chez 14 des 15 chiens mais l'augmentation de la fréquence assurait le maintien du débit cardiaque. La relation entre l'augmentation maximale de la PAP et la proportion du poumon occupé par la graisse était curvilinéaire ($r = 0,87$).

Conclusion : Sur ce modèle, le volume d'éjection a diminué en moins d'une minute après l'ACB. L'embolie graisseuse suivait l'insertion de la prothèse et s'accompagnait d'une baisse du volume d'éjection. Après l'embolie graisseuse peropératoire, le maintien du débit cardiaque dépendait principalement de l'augmentation de la fréquence cardiaque.

From the Departments of Anaesthesia, St. Michael's Hospital and Sunnybrook Health Science Centre; Department of Pathology and Laboratory Medicine, Mt. Sinai Hospital; University of Toronto. This study was supported by the Ontario Thoracic Society.

Address correspondence to: Dr. Robert J. Byrick, Department of Anaesthesia, St. Michael's Hospital, 30 Bond Street, Toronto, Ontario M5B 1W8; Phone: 416-864-5892; Fax: 416-864-6014; E-mail: robert.byrick@utoronto.ca

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DURING cemented arthroplasty (CA) procedures with long-stemmed prostheses, hypotension, hypoxaemia, acute pulmonary hypertension, right ventricular failure and occasionally cardiac arrest may occur.[1-3] These complications are referred to as the "bone cement implantation syndrome" and are thought to be due to pulmonary embolisation of intramedullary contents at the time of cement and prosthesis insertion. Transoesophageal echocardiography (TEE) has demonstrated that these changes are associated with visualisation of echogenic material in the right ventricle (RV).[3-7]

Although nearly all patients demonstrate echogenic material during CA, most patients tolerate the embolic load and compensate for the increased RV afterload. However, descriptions of profound hypotension leading to cardiac arrest and sudden death continue to be reported.[8,9]

Clinical reports of this rare event have not clearly delineated the relationship between RV opacification on TEE and acute pulmonary hypertension with decreased cardiac output. No studies have explored the relationship between the number of intravascular fat emboli and the pulmonary hypertension. We report the cardiopulmonary response to massive fat embolism during CA in an animal model and relate these changes to the TEE opacification and the amount of pulmonary intravascular fat at postmortem.

Methods

This study was approved by the local animal care committee. All dogs were treated in accordance with the regulations of the Canadian Council on Animal Care.

Anaesthesia and cannulation

Sixteen mongrel dogs weighing 30 ± 4.6 kg were anaesthetised using 30 $mg \cdot kg^{-1}$ sodium pentobarbital *iv*. After tracheal intubation, anaesthesia was maintained with isoflurane 1–2%, in a mixture of oxygen:nitrous oxide (50:50) by positive pressure ventilation via a non-rebreathing circuit. Ventilation was adjusted to maintain $PaCO_2$ between 35 and 40 mmHg. We infused normal saline at 10 $ml \cdot kg \cdot hr^{-1}$ *iv* throughout the experiment. Pancuronium (0.15 $mg \cdot kg^{-1}$ *iv*) was given prior to surgery and intermittent doses of 250–500 μg fentanyl were given as required to maintain a baseline heart rate < 100 bpm. No fentanyl was given after the baseline measurements.

An arterial cannula (14 gauge) was inserted into a femoral artery for blood sampling. A 7F catheter was advanced into the pulmonary artery via an external jugular vein to measure PAP, pulmonary artery occluded pressure (PAOP) and right atrial pressure (RAP). A MIKRO-TIP® catheter pressure transducer (Millar Instruments, Houston, Texas) was inserted into a carotid artery to monitor aortic blood pressure (BP).

Echocardiography

Transoesophageal echocardiography was performed using a 5.0 MHZ Biplane probe (model 21363A Hewlett-Packard Co., Andover, Mass.). Under general anaesthesia, the TEE probe was inserted into the oesophagus and advanced a distance of approximately 50 cm from the incisors. At this position, all animals were examined using colour flow Doppler analysis to exclude the presence of a patent foramen ovale (PFO). The absence of a PFO was confirmed at postmortem examination. The TEE probe was then advanced into the stomach to a level 60 cm from the incisors to produce a view of the right and left ventricles at the level of the mitral valve. The mitral valve level view was chosen as it was the only short axis view that could be consistently reproduced to allow estimation of both the cardiac effects of the embolic load as well as of the function and size of the ventricles. This was technically difficult and required extreme anterior and rightward flexion of the scope. The animal occasionally had to be rotated to a position between supine and left lateral to obtain the optimal view. It appeared to be impossible to achieve an adequate short axis view at the papillary muscle level consistently due to the proximity of the left lung. The same view was maintained for each animal throughout the study. Right and left ventricular areas were calculated from cavitary measurements (planimetry with the trackball) at the level of the mitral valve at end-diastole (Sonos 1000 On-Line Analysis, Hewlett-Packard Co., Andover, Massachusetts).

The TEE images were assessed independently by two echocardiographers (PM and GE) who were unaware of the surgical procedure, haemodynamic responses of the dog and post mortem pathology results. Both echocardiographers independently examined the tapes and analysed the same representative frame at each measurement period. When there was a discrepancy of >10% in ventricular dimensions, the tapes were reviewed and a consensus reached. The state of RV and left ventricular (LV) dilatation was determined using the measured RV and LV areas as described. The presence or absence of interventricular septal flattening was noted and LV fractional area change (FAC) was calculated at baseline and the one minute interval. Fractional area change was calculated as end-diastolic area minus end-systolic area divided by end-diastolic area multiplied by 100 to convert to percentage. End-diastolic area was taken as the largest area at end-diastole and end-systolic area as the smallest area

at end-systole. The endocardial border was traced manually.

Surgical technique and protocol

In 16 dogs a bilateral cemented arthroplasty (BCA) was performed by exposing both distal femoral condyles using a transverse skin incision and division of the patellar tendons. The distal femoral canal was entered by drilling through the patellofemoral groove and reaming to a depth of approximately 10 cm using successively larger reamers up to 9 mm diameter. The intramedullary canal was filled with normal saline. At this stage, low viscosity bone cement (Surgical Simplex P, Howmedica International Ltd., London, England) was injected simultaneously under manual pressure into both medullary canals. A solid, contoured metal rod was then hammered into each medullary cavity to simulate prosthesis insertion.

Data collection

Ventricular function was monitored continuously throughout the surgical procedure and all TEE data were recorded on videotape for later analysis. Heart rate (HR), BP, RAP and PAP were recorded continuously on a Gould ES 1000 recorder (Gould Inc., Cleveland, Ohio) and at specific time periods simultaneously collected by analog to digital conversion using an AT-Codas system interface card (Dataq instruments, Akron, OH) at a rate of 250 Hz per channel on an IBM compatible 80386 computer.

Haemodynamic measurements were recorded on the computer before and after reaming and at 1, 3, 5, 15 and 30 min following impaction of the femoral prostheses. Thermodilution cardiac output $\dot{Q}$ was measured in triplicate at each period, with the exception of the one minute measurement. The one minute $\dot{Q}$ measurement was only a single reading as haemodynamic stability was not maintained at this time. The uncertainty related to thermal dilution cardiac output under these circumstances made concurrent evaluation of cardiac function by TEE important. The means of three determinations of $\dot{Q}$ at the other measurement periods were used to calculate stroke volume and pulmonary vascular resistance (PVR) using standard equations.

We used the measurements following reaming (i.e., immediately before cement and prosthesis impaction) as baseline values because they were made within three minutes of BCA, thus minimising haemodynamic changes resulting from surgical bleeding during reaming. All subsequent measurements were compared with these post-reaming baseline values. No fentanyl was given after the base line measurements and haemodynamic changes were not treated with inotropic agents or bolus fluid administration.

Morphometric analysis

At the end of the 30 min monitoring period the dogs were killed by an overdose of pentobarbital and injection of KCl. Our morphometric analysis of the pulmonary fat embolic load has been described in detail.[10] In brief, a numerical coding system was used so the pathologist who performed the morphometric analysis was unaware of the physiological or TEE changes found in the experiment. Six lung samples from each animal post-fixed in Flemming's solution were evaluated using a TV-based image analysis system (BQ Meg IV, R and M Biometrics, Nashville TN). The area and diameter were calculated for each embolus. The ratio of area occupied by fat emboli to total area of lung examined is equivalent to the volume proportion of lung tissue occupied by fat (Delesse's Principle).[11] The number of fat emboli and volume proportion of lung tissue occupied by fat was calculated for each dog.

Statistical analysis

Data are reported as the mean value ± standard deviation. Data were analysed using the SAS® (Statistical Analysis System) statistical package. We used the SAS general linear model repeated measures analysis of variance procedure to analyse data from sequential measurements. When a significant F-ratio was present ($P < 0.05$) multiple comparisons between baseline and other measurements were made using Dunnett's and Tukey's tests. Correlation coefficients were determined using linear functions for all correlations except the correlation between the amount of pulmonary intravascular fat and the PAP where a polynomial function was used.

Results

One dog was eliminated from the study because of a patent foramen ovale detected by TEE immediately after cement and prosthesis insertion.

Haemodynamics

The haemodynamic results for the remaining 15 dogs that underwent BCA are shown in the Table. There were no differences between the pre-ream and post-ream (baseline) measurements. Compared with the post-ream baseline values, mean PAP and PVR increased after BCA and remained above baseline values throughout the study period. Mean BP decreased within one minute of BCA. Mean PAOP increased but returned to baseline values within five minutes. There were no decreases in mean cardiac output at the measurement periods.

However, SV decreased and HR increased immediately after BCA but both returned to baseline by 15 min.

Transoesophageal echocardiography

All dogs had normal ventricular function prior to BCA. After BCA there was complete opacification of the RV for 30 sec with embolic material varying in size from 1–10 mm, marked RV dilation, interventricular septal flattening and reduced LV area (Figure 1). Septal flattening was noted in all animals and left ventricular FAC decreased significantly from baseline 43 ± 6.9% to 35 ± 9.4% ($P < 0.05$) at the one minute interval.

Figure 2 shows the mean right and left ventricular areas at each measurement period with the simultaneous PAP measurement. Within one minute of BCA, the RV area increased in all 15 dogs by 56% ($P < 0.05$). Concomitantly the LV area decreased by 34% ($P < 0.05$) (Figure 2) and PAP increased by 155%. The RV area returned to the baseline value by the 30 min measurement. The LV area remained below baseline values throughout the study period.

When all 15 dogs were analysed as a group there was a relationship between the change in RV area and the change in mean PAP ($r = 0.32$, $P < 0.05$), and the change in PVR and the change in SV ($r = 0.33$, $P < 0.05$). In response to the increase in PAP the change in RV area and SV was variable among dogs. In 14/15 dogs SV decreased and RV area increased at the one minute measurement. However, the slope of this relationship varied widely (Figure 3). Figure 4 shows the mean and SD of PAP and RV area at each time period.

Postmortem pulmonary intravascular fat

In one of the 15 animals technical problems prevented complete lung inflation for fixation hence only 14 animals are included in the analysis of volume of lung tissue occupied by fat. The mean proportion of lung

TABLE Haemodynamic data (mean ± standard deviation) at each measurement time period (n = 15).

	Pre-ream	*Post-ream (Baseline)*	*Time after BCA*				
			1 min	*3 min*	*5 min*	*15 min*	*30 min*
BP (mmHg)	98 ± 22	110 ± 24	79 ± 25*	98 ± 24	107 ± 30	102 ± 19	99 ± 22
PAP (mmHg)	14 ± 3	15 ± 3	39 ± 10*	35 ± 10*	32 ± 9*	28 ± 7*	27 ± 7*
RAP (mmHg)	8 ± 2	8 ± 2	9 ± 4	9 ± 3	9 ± 3	8 ± 3	8 ± 3
PAOP (mmHg)	8 ± 3	9 ± 3	13 ± 8*	12 ± 6*	11 ± 6	10 ± 5	9 ± 4
Q ($l \cdot min^{-1}$)	3.6 ± 1.2	3.3 ± 1.1	2.9 ± 1.4	3.0 ± 1.3	3.2 ± 1.3	3.7 ± 1.4	4.0 ± 1.3*
PVR ($dyne \cdot sec \cdot cm^{-5}$)	145 ± 53	154 ± 44	1071 ± 1326*	913 ± 1029*	680 ± 543*	447 ± 223*	384 ± 177*
HR (bpm)	94 ± 21	94 ± 17	140 ± 35*	122 ± 24*	116 ± 22*	104 ± 27	114 ± 28
SV ($ml \cdot min^{-1}$)	39 ± 14	36 ± 11	21 ± 9*	24 ± 9*	28 ± 13*	35 ± 10	35 ± 9

*significant difference from post reaming (baseline) measurement.

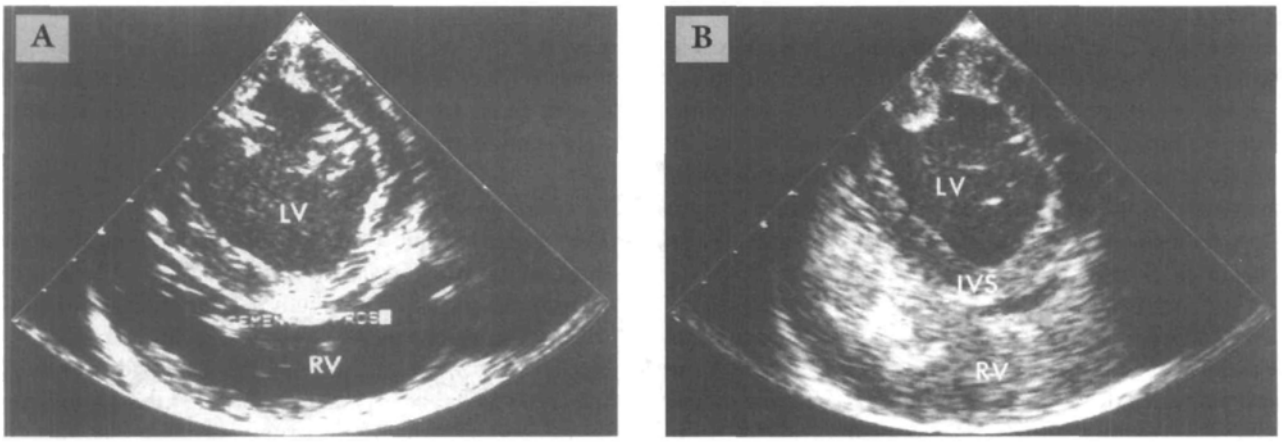

FIGURE 1 Echocardiographic images in one dog before bilateral cemented arthroplasty (A) and (B) three minutes after cement and prosthesis insertion

An opaque embolus is visualised in the right ventricle (RV) after BCA (B). The RV is dilated and large, the left ventricle (LV) area is reduced

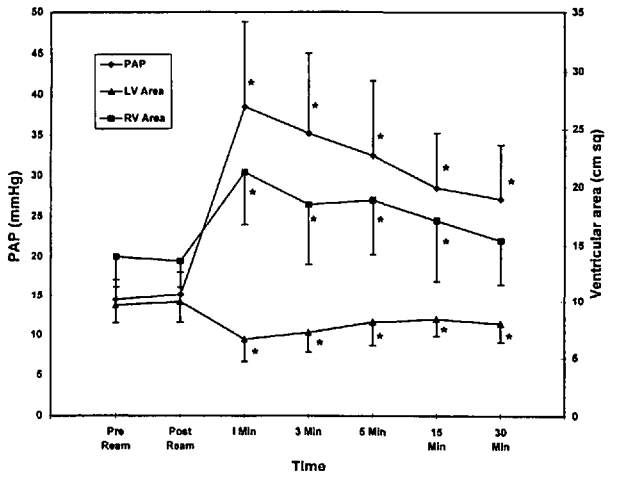

FIGURE 2 Mean (± SD) of pulmonary artery pressure (PAP), right ventricular (RV) area and left ventricular (LV) area at each measurement period.
*denotes a significant change from post-reaming baseline measurement.

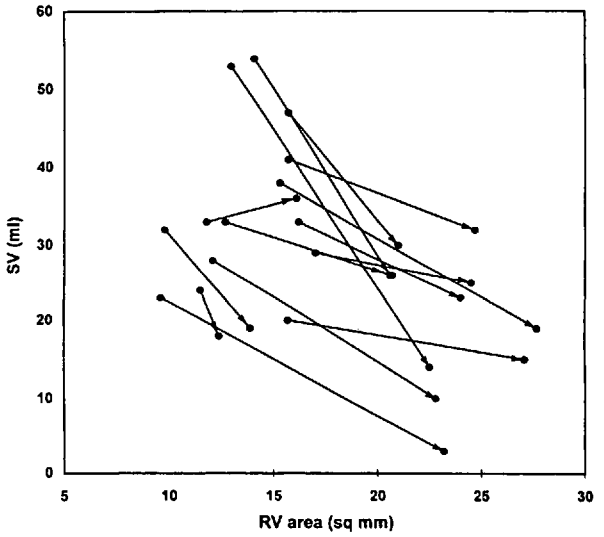

FIGURE 3 The right ventricular area (RV) and stroke volume (SV) at baseline and one minute for each dog. The arrow indicates the one minute measurement.

tissue occupied by fat ranged from 1.6 to 4.1% (Figure 5). There was a relationship between the post mortem proportion of lung tissue occupied by fat and the change in PAP from baseline to maximum value within three min of BCA ($P < 0.05$). The data are best described using curvilinear regression and a cubic polynomial was used (r= 0.87) (Figure 5). No relationship between the change in RV area and the mean proportion of lung tissue occupied by fat was found.

Discussion

This study examined the acute haemodynamic changes during cemented arthroplasty complicated by massive fat embolism. We simulated the clinical syndrome using an animal model and quantified the embolic fat load at postmortem examination. Thus, the echocardiographic and haemodynamic measurements could be related to the amount of lung vasculature occluded by fat.

The severity of embolism we observed corresponds to a 7–9 in Ereth's grading system (range 0–9).[4] This system graded venous embolism in patients based on TEE analysis. The features used included the amount of right atrium filled by echogenic particles, the duration of opacification and the diameter of particles seen. In his study, Ereth showed that the level of opacification that corresponds to grade 7–9 is associated with clinically important haemodynamic instability.[4]

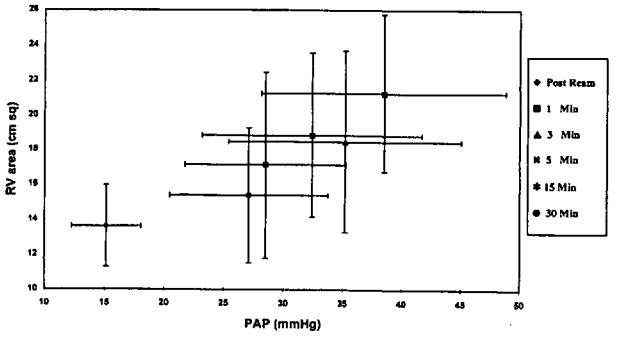

FIGURE 4 Mean (± SD) of pulmonary artery pressure (PAP) and right ventricular (RV) area at each measurement period.

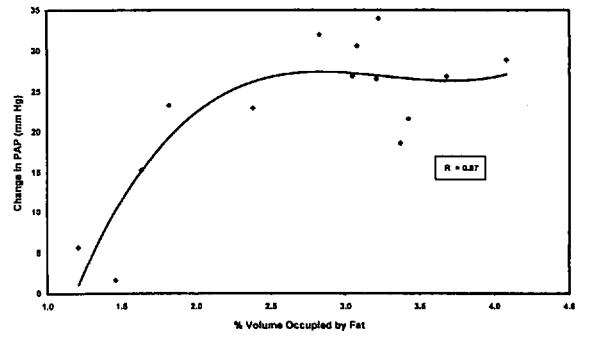

FIGURE 5 The post-mortem volume of lung tissue occupied by fat (%) and the maximum change in pulmonary artery pressure (PAP) within three minutes of BCA, with a cubic polynomial fitted to the data.

We used an animal model because, in clinical studies, one cannot quantify the amount of pulmonary vascular occlusion. Even with TEE, the quantity of embolised fat cannot be determined. Any material with sound wave transmission properties different from blood produces echoes and the echocardiographer cannot distinguish between blood clot, fat, air and marrow particles. Therefore, this is the first report of RV area and PAP changes correlated with the quantity of pulmonary intravascular fat after acute fat embolism.

We observed that the volume of lung tissue occupied by fat ranged from 1.6 to 4.1%. The extent of embolic occlusion is only one of the determinants of the severity of pulmonary hypertension after pulmonary embolism. The compliance of the normal pulmonary vasculature, the cardiovascular status before embolism and the activity of humoral or reflex mechanisms also play major roles in determining PAP. Because of these factors, the relationship between the volume of lung tissue occupied by fat and the change in PAP would not be expected to be a linear relationship and we found that a cubic polynomial produced the best empirical fit to our data (Figure 5). The degree of pulmonary hypertension in our dogs suggests that > 50% of the pulmonary vasculature is functionally occluded. Barie *et al.*[12] found an increase of PAP of only 3 mmHg with occlusion of the left pulmonary artery in sheep. This supports the findings of Ereth[4] who suggested that the degree of embolisation did not correlate with changes in haemodynamic variables. Kay *et al.*[13] using starch emboli in dogs also found there was not a good relationship between the quantity of embolus given and the increase in PAP. For any given quantity of starch there was a wide range of PAP values. Ebert *et al.*[14] showed that RV pressure increased in a stepwise fashion and parallelled the degree of pulmonary vascular occlusion as long as cardiac output was maintained. When cardiac output decreases PAP does not increase with progressive pulmonary vascular occlusion. These factors interact to determine the curvilinear relationship between PAP and the volume proportion of lung vasculature occluded (Figure 5).

Previous work with this model has confirmed that haemodynamic instability characterised by pulmonary hypertension and systemic hypotension was related to the number of fat emboli in the lung and not to the use of bone cement.[15] Orsini *et al.* showed that intramedullary pressure determined the extent of fat embolism, whether created by the use of bone cement or an inert material such as bone wax.[15] Ereth's data confirmed these findings in patients where non-cemented prostheses were associated with fewer echogenic emboli than cemented prostheses.[4] The role of bone cement appears to be primarily in sealing the medullary cavity resulting in increased pressure forcing fat into the venous system. The doses of methylmethacrylate monomer required to alter cardiopulmonary function are much larger than those resulting from cemented arthroplasty.[16]

Although statistically significant, the relationship between the PAP and either RV area or SV changes were not predictive (Figures 3 and 4). It is not possible to predict, from PAP changes, the decrease in SV or increase in RV area in individual animals. Several factors alone or together may contribute to the lack of a predictable relationship. A likely explanation for the lack of relationship between the increase in PAP and RV area is the quantitative difference between the true RV volume and the measured RV area. We chose to use the area of the RV at the level of the mitral valve as our measure of RV size. This is clearly not a measure of RV volume since the shape of the RV varies with the level at which images are taken and the RV cannot be assumed to be elliptical.[17] In humans, areas are measured most often at the level of the papillary muscle. We found, however, that in the dog, it was impossible to obtain consistently good views at the papillary muscle level due to interposition of the lung. We were able to reproduce consistent ventricular images at the mitral valve level only. Therefore, we did not wish to assume that our measurements reflect volume changes and reported area changes only.

Another explanation would be a varying level of ventricular reserve among animals. Data from individual dogs showed that some were able to maintain cardiac output in the presence of an increased PVR. These dogs had a large increase in PAP with smaller changes in SV and RV area. Other dogs could not tolerate the increased PVR. They developed varying levels of RV failure manifest by decreases in SV accompanied by dilation of the RV (Figure 3). This variability in response partially accounts for the large standard deviations found in the combined data (Figure 4) and the lack of a strong statistical correlation between the changes in PAP and either SV or RV area when all dogs were compared.

The ability to tolerate the embolic insult is a function of the ability of the heart to maintain an adequate RV output in the presence of an increased PVR. In this study FAC decreased within one minute of BCA. Left ventricular area decreased (Figure 2) and PAOP increased (Table) suggesting a decrease in LV compliance caused by RV dilation. Given a decrease in preload as suggested by the decrease in LV diastolic area and the decreased FAC, it is likely that the cause of the reduced SV is decreased LV volume within one minute of BCA. Previous work[18] has shown a transient

decrease in cardiac output within seconds of BCA. The modest decrease in FAC could have been due to a decrease in contractility but is more likely to be due to the large changes in loading conditions, since this measurement is known to be sensitive to changes in both preload and afterload.[19]

Our data suggest that the key factor in maintaining cardiac output after embolisation is the ability to increase HR in the presence of decreased SV. It is known that dogs can compensate for increases in pulmonary vascular resistance better than can many humans.[14] If similar embolic loads with increased PAP and decreased SV were encountered in elderly humans having cemented arthroplasty, haemodynamic changes should be clinically significant. Urban's data[20] suggest a similar pattern does occur in patients. In their study of 18 patients undergoing total hip arthroplasty, four experienced pulmonary hypertension accompanied by a decrease in RV ejection fraction and hypotension at the time of pulmonary embolisation. These patients did not tolerate this haemodynamic instability and required inotropic support. Urban's patients may have had a limited ability to increase HR because of the presence of a sympathetic blockade secondary to epidural analgesia to a sensory level of T4. In another report, a patient with a fixed heart rate (pacemaker dependent) suffered acute cardiovascular collapse within seconds of cement and prosthesis insertion.[21] The inability to increase HR in response to the decrease in SV may have been an important predisposing factor.

Guest *et al.*[22] using the same animal model showed that haemodynamic stability and improved outcome after BCA with fentanyl anaesthesia was associated with an increased HR. This potential to increase heart rate was not present in the dogs that received isoflurane anaesthesia. They had a tachycardia before BCA with no change after fat embolisation. Guest's study demonstrated that BCA was associated with a variable decrease in SV which required an increase in HR to maintain $\dot{Q}$. In our study low concentrations of isoflurane were used with intravenous fentanyl titrated to keep baseline HR around 100 beats·min^{-1} prior to BCA. Most dogs could increase HR which corresponded in time to the decreased SV (Table). As SV returned to base line, HR decreased (Table).

This study has extended our understanding of the acute haemodynamic changes that result from fat emboli during cemented arthroplasty. Firstly, a curvilinear relationship was found between the increased PAP and the proportion of lung volume occluded by fat. Secondly, we conclude that the decrease in $\dot{Q}$ within one min of CA demonstrated in a previous study with an open pericardium, also occurs with this closed chest model. The reduced $\dot{Q}$ is due to a markedly decreased SV which is accompanied by increased RV area and reduced LV area. The recovery and maintenance of $\dot{Q}$ for the 3–30 min after CA was dependent on an increased HR. Similar TEE findings have been reported in humans, therefore, patients with fixed heart rates may be at risk of intraoperative cardiac arrest during cemented arthroplasty.

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