HYPOTENSION DURING CEMENTED ARTHROPLASTY

RELATIONSHIP TO CARDIAC OUTPUT AND FAT EMBOLISM


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An episode of hypotension is common during cemented joint replacement, and has been associated with circulatory collapse and sudden death. We studied the mechanism of hypotension in two groups of six dogs after simulated bilateral cemented arthroplasty. In one group, with no lavage, the insertion of cement and prosthesis was followed by severe hypotension, elevated pulmonary artery pressure, decreased systemic vascular resistance and a 21% reduction in cardiac output. In the other group, pulsatile intramedullary lavage was performed before the simulated arthroplasties. Hypotension was less, and although systemic vascular resistance decreased, the cardiac output did not change.

The severity of the hypotension, the decrease in cardiac output and an increase in prostaglandin metabolites were related to the magnitude of pulmonary fat embolism. Pulsatile lavage prevents much of this fat embolism, and hence the decrease in cardiac output. The relatively mild hypotension after lavage was secondary to transient vasodilatation, which may accentuate the hypotension caused by the decreased cardiac output due to a large embolic fat load.

We make recommendations for the prevention and management of hypotension during cemented arthroplasty.

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Acute hypotension during cemented joint replacement is common (Charnley 1970; Jones 1975; Modig et al 1975), and has been associated with elevated pulmonary artery pressure (Samii et al 1980; Byrick, Kay and Mullen 1987) and transient hypoxaemia (Kallos 1975; Alexander and Barron 1979; Mebius and Hedenstierna 1982). These physiological changes have been demonstrated at the time of cement and prosthesis insertion both in patients (Herndon, Bechtol and Crickenberger 1974; Modig et al 1975; Samii et al 1980) and in experimental animals (Byrick et al 1987).

Although the pathophysiology of this hypotension remains poorly understood, particulate fat and marrow pulmonary embolism has been implicated (Sevitt 1972; Breed 1974; Herndon et al 1974; Modig et al 1975; Sherman et al 1983; Byrick et al 1987). Other possible mechanisms include PMMA-induced myocardial depression (Mir, Lawrence and Autian 1973), vasodilatation secondary to PMMA (Peebles et al 1972; Kim and Ritter 1972; McMaster, Bradley and Waugh 1974), reflex autonomic effects (Rudiger and Ritter 1983), thromboplastin generation (Modig et al 1975; Giercksky et al 1979; Dahl et al 1992) and prostaglandin-induced vasodilatation (Byrick et al 1991).

Simulated bilateral cemented arthroplasty (SBCA) performed in anaesthetised mongrel dogs has been shown to produce hypotension associated with pulmonary fat embolism (Byrick et al 1987). In this animal model, pulsatile lavage of the femoral medullary cavity before SBCA reduces pulmonary fat and marrow embolism, systemic hypotension and also pulmonary hypertension (Sherman et al 1983; Byrick et al 1989). There is a transient reduction in cardiac output in association with acute pulmonary fat embolism after SBCA (Byrick et al 1992) but it is not known whether prevention of fat embolism by medullary lavage stops this decrease. If a reflex autonomic phenomenon or vasodilatation from PMMA was responsible for the transient decrease in cardiac output, then lavage would not be expected to show the same haemodynamic response.

Our study was designed to investigate the mechanism of hypotension during SBCA and the relationship between haemodynamic changes and lavage. We hypothesised that a reduction in fat and marrow 'embolic load' by pulsatile lavage before cemented arthroplasty
would prevent both the acute decrease in cardiac output and the release of prostaglandin metabolites.

MATERIALS AND METHODS

Twelve mongrel dogs weighing 30.3 ± 7.3 kg were anaesthetised using intravenous pentobarbitone 30 mg/kg, followed by the continuous infusion of 5 mg/kg/hr of pentobarbitone as required to maintain anaesthesia and a stable haemodynamic state. The trachea was intubated and the lungs were mechanically ventilated by a rebreathing circuit and 100% oxygen. Ventilation was adjusted to maintain PaCO$_2$ at 35 to 40 mmHg. Animals were paralysed using pancuronium bromide (0.2 mg/kg) and Ringer's lactate solution was infused at a rate of 10 ml/kg/hr to maintain intravascular volume during the procedure.

The experimental model is shown in Figure 1. A size 7 French gauge triple-lumen, balloon-tipped catheter was positioned to measure pulmonary artery pressure (PAP) and right atrial pressure (RAP). The left carotid artery was cannulated with a pressure-tipped arterial catheter (Millar Instruments Inc, Houston, Texas) which was advanced into the ascending thoracic aorta to measure systemic arterial blood pressure (BP). A size 7 French gauge catheter was inserted into the right femoral artery for arterial blood sampling.

A left, fifth-interspace thoracotomy was performed. From within the pericardium, a T201 ultrasonic flow probe (Transonic Systems Inc, Ithaca, New York) was placed around the ascending thoracic aorta to measure continuously the pulsatile aortic flow and record the cardiac output (CO). A size 7 French gauge catheter was sutured directly into the left atrial appendage to monitor left atrial pressure (LAP).

All pressures and the heart rate (HR) were recorded continuously by a Gould ES 1000 recorder (Gould Inc, Cleveland, Ohio). The stroke volume (SV) was calculated using the following relationship:

\[ \text{Stroke volume} (l) = \frac{\text{Cardiac output (l/min)}}{\text{Heart rate (l/min)}} \]

Systemic vascular resistance (SVR) and pulmonary vascular resistance (PVR) were calculated using the following equations:

Systemic vascular resistance
\[ = \frac{\text{BP}-\text{RAP}}{\text{CO}} \times 80 \text{ (dyne/sec/cm}^{-5}) \]

Pulmonary vascular resistance
\[ = \frac{\text{PAP}-\text{LAP}}{\text{CO}} \times 80 \text{ (dyne/sec/cm}^{-5}) \]

Six dogs were assigned to each of a no lavage group and a pulsatile lavage group. In the no lavage group, both distal femora were exposed via bilateral knee arthrotomies, dividing the patellar tendons. Prereaming measurements were made and the distal femoral medullary canals were then entered by a drill, and reamed for 10 cm depth, using successively larger reamers up to 9 mm. After reaming, all the physiological measurements were repeated. Simulated bilateral cemented arthroplasties were then performed, using PMMA bone cement (Surgical Simplex; Howmedica International Ltd, London, England) mixed according to standard directions. One packet was mixed with a 20 ml ampoule and half of the 60 g of PMMA was inserted into each medullary canal by manual pressure. Solid, contoured metal rods were then hammered into both medullary cavities to simulate prosthesis insertion. To create an 'embolic load' which would produce haemodynamically significant pulmonary hypertension (Byrick et al 1987), the insertion was performed with no venting of the medullary canal and no tourniquet.

In the lavage group, the surgical procedure was identical except that the medullary cavities were thoroughly irrigated by high-pressure, high-volume pulsatile lavage from a Simpulse suction-irrigator (CR Bard, Cranston, Rhode Island) before the insertion of cement and prosthesis. The irrigator was detached from the suction tube of the device to permit lavage of the dogs'
Table I. Haemodynamic parameters and plasma arachidonic acid metabolite concentrations (mean ± SD) for preream, postream (no lavage) and postlavage (lavage) groups. The postream and postlavage values were used as baseline measurements for subsequent comparisons in the no lavage and lavage groups respectively.

<table>
<thead>
<tr>
<th></th>
<th>Preream Control</th>
<th>Preream Lavage</th>
<th>Postream Control (baseline)</th>
<th>Postream Lavage (baseline)</th>
</tr>
</thead>
<tbody>
<tr>
<td>BP (mmHg)</td>
<td>146.9 ± 10.0</td>
<td>140.3 ± 20.6</td>
<td>141.9 ± 6.3</td>
<td>131.5 ± 25.0</td>
</tr>
<tr>
<td>PAP (mmHg)</td>
<td>16.3 ± 3.4</td>
<td>15.6 ± 3.4</td>
<td>16.2 ± 3.9</td>
<td>14.6 ± 2.5</td>
</tr>
<tr>
<td>LAP (mmHg)</td>
<td>8.8 ± 1.7</td>
<td>10.2 ± 3.1</td>
<td>8.4 ± 2.4</td>
<td>8.6 ± 2.8</td>
</tr>
<tr>
<td>RAP (mmHg)</td>
<td>4.9 ± 1.9</td>
<td>6.7 ± 1.9</td>
<td>4.9 ± 2.0</td>
<td>5.5 ± 2.1</td>
</tr>
<tr>
<td>CO (l/min)</td>
<td>2.70 ± 0.74</td>
<td>2.57 ± 0.79</td>
<td>2.52 ± 0.69</td>
<td>2.23 ± 0.93</td>
</tr>
<tr>
<td>HR (/min)</td>
<td>149 ± 10</td>
<td>163 ± 11</td>
<td>145 ± 13</td>
<td>156 ± 14</td>
</tr>
<tr>
<td>SV (ml)</td>
<td>18.26 ± 5.38</td>
<td>15.74 ± 4.68</td>
<td>17.65 ± 5.39</td>
<td>14.14 ± 5.49</td>
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<tr>
<td>PVR (dyne/sec/cm²)</td>
<td>248 ± 135</td>
<td>195 ± 125</td>
<td>280 ± 171</td>
<td>273 ± 204</td>
</tr>
<tr>
<td>SVR (dyne/sec/cm²)</td>
<td>4485 ± 1289</td>
<td>4448 ± 1415</td>
<td>4634 ± 1340</td>
<td>5067 ± 1926</td>
</tr>
<tr>
<td>TxB2 (ng/ml)</td>
<td>0.61 ± 0.15</td>
<td>0.66 ± 0.15</td>
<td>0.59 ± 0.12</td>
<td>0.81 ± 0.13</td>
</tr>
<tr>
<td>6-keto PGF1α (ng/ml)</td>
<td>0.46 ± 0.07</td>
<td>0.38 ± 0.08</td>
<td>0.46 ± 0.05</td>
<td>0.32 ± 0.05</td>
</tr>
</tbody>
</table>

marrow cavities. A minimum of 1.5 litres of saline was used to irrigate each cavity, followed by thorough suction to maximise the removal of marrow.

Blood samples were taken for arterial blood-gas analysis and the measurement of plasma concentrations of the stable arachidonic acid metabolites thromboxane B₂ (TxB₂) and 6-keto prostaglandin F₁α (6-keto PGF₁α) before reaming, immediately after reaming, after lavage in the lavage group only, and at 30 seconds and 1, 3, 5 and 15 minutes after insertion of cement and prosthesis.

For the estimation of TxB₂ and 6-keto PGF₁α, 9 ml blood samples were collected in plastic syringes containing 1 ml of an indomethacin/anticoagulant (ethylenediaminetetra-acetic) solution adjusted to a pH of 7.4. (The indomethacin was added to inhibit any further production of arachidonic acid metabolites in vitro after sampling.) Each sample was then centrifuged, the plasma separated and immediately frozen at −20°C for storage.

The concentrations of plasma TxB₂ and 6-keto PGF₁α were determined by radioimmunoassay (RIA), utilising ¹²⁵I-labelled tracer kits (Dupont Canada, Mississauga, Canada). Standards, controls and samples were analysed in duplicate as previously described (Byrick et al 1991, 1992). Radioactivity measurement and data reduction were performed using an LKB Model 1277 gamma counter (LKB, Wallac Oy, Turku, Finland).

After completing the study, each dog was killed by injecting pentobarbitone (200 mg) and potassium chloride through the left atrial cannula. At post-mortem, the lungs and heart were removed en bloc and fixed in inflation with 10% buffered formalin at a pressure of 25 cm of fixative. After fixation for 72 hours, the lungs were sectioned in the mid-sagittal plane and three stratified random blocks of known size were taken from each lung. These samples were postfixed in Fleming's solution for 72 hours, washed in running water for 24 hours, and then processed for histological examination.

Sections were evaluated using a TV-based image-analysis system (BQ Meg IV, Nashville, Tennessee). This system has a Leitz Dialux 22 microscope attached to a Sony CCD video camera connected to an IBM AT compatible microcomputer. For each animal, ten stratified random fields (each 1 mm²) from the six lung samples were examined for a total area of 60 mm² per animal. The measurements were made at a final optical magnification of × 245. The grey scale image was converted to a binary black and white image by manual selection of the threshold value which best delineated the emboli from the background. The area and diameter of each embolus and the ratio of area occupied by fat emboli to total area of lung examined were calculated (Siegart et al 1971). This is equivalent to the volume proportion of lung tissue occupied by fat (Delesse's principle).

Data are reported as mean ± 1SD and were analysed using the SAS (Statistical Analysis Software) 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 within groups between baseline (postreaming in the no lavage group; postlavage in the lavage group) and other measurements were made by unadjusted pairwise comparisons using the LSMEANS comparison procedure. Adjustments were made on the probability of rejection using Bonferroni's correction. If there was significant group and time interaction, multiple comparisons with baseline values within groups were determined by Dunnett's test.
RESULTS

Figures 2, 3 and 4 show the changes from baseline for the haemodynamic results in the two groups. The maximum decrease in BP was observed from 30 seconds to 3 minutes after cement and prosthesis insertion. At the point of maximum decrease in BP, all haemodynamic variables were recorded.

Groups before SBCA. There were no significant differences between the mean weight of dogs in the no lavage (28.3 ± 4.7 kg) and lavage (32.4 ± 9.1 kg) groups or the baseline haemodynamic measurements in each group (Table I). In the lavage group, there were no significant differences between the poststreaming and postlavage measurements. There were no significant differences in baseline arterial blood gas measurements (PaO₂ and PaCO₂) between the groups.

Haemodynamic and gas exchange results

No lavage. In the no lavage group, there was a significant decrease in mean BP (55.6 ± 9.7 mmHg) within 3 minutes of SBCA when compared with baseline values (Fig. 2). At the time of the maximum decrease in mean BP, there was a significant increase in PAP (22.3 ± 10 mmHg) to values approximately double the baseline measurements. PAP (Fig. 2) remained significantly elevated above baseline values at both 5 minutes (19.9 ± 10.8 mmHg) and 15 minutes (15.1 ± 6.4 mmHg). There were no significant changes in RAP or LAP (Fig. 2).

At the time of maximum decrease in mean BP, there was a significant decrease in CO (Fig. 3; 0.57 ± 0.37 l/min; p < 0.05). This was a 21% reduction in CO from baseline values. It had returned to baseline values at 5 and at 15 minutes. HR (Fig. 3) did not change significantly during the study period, but there was a significant decrease in SV within 3 minutes of SBCA (Fig. 3; 4.1 ± 2.8 ml; p < 0.05), also equivalent to a 21% decrease from baseline values.

The calculated PVR increased significantly from baseline values (993 ± 396 dyne/sec/cm⁵) within 3 minutes of SBCA (Fig. 4). The increase in PVR remained significant at 5 and 15 minutes (p < 0.05). SVR decreased significantly compared with baseline values (Fig. 4; 1064 ± 1017 dyne/sec/cm⁵; p < 0.001) within 3 minutes, but had returned to baseline values at 5 and at 15 minutes.

There was a significant decrease in PaO₂ compared with baseline values at 5 minutes (60 ± 54 mmHg) and at 15 minutes (45 ± 52 mmHg) (p < 0.02). No change from baseline PaCO₂ was noted at any time period.

Lavage group. In the lavage group, there was a statistically significant decrease in mean BP within 3 minutes of SBCA (Fig. 2; 19.1 ± 14.8 mmHg; p < 0.05). At 5 and 15 minutes however, there was no significant change compared with baseline values. A significant (35% increase above baseline) rise in PAP also occurred (Fig. 2), which persisted throughout the study period (p < 0.05). No changes in LAP or RAP were noted.
HYPOTENSION DURING CEMENTED ARTHROPLASTY

Fig. 3
Changes from baseline values for cardiac output, heart rate and stroke volume in both groups (* = statistically significant change from baseline; † = significant difference in response between groups).

Fig. 4
Changes from baseline values for pulmonary vascular resistance and systemic vascular resistance for both groups (* = statistically significant change from baseline; † = significant difference in response between groups).
Changes from baseline values for thromboxane B₂ and 6-keto prostaglandin F₁α for both groups (* = statistically significant change from baseline; † = significant difference in response between groups).

There were no significant changes in CO in the lavage group at the three time intervals when compared with baseline values (Fig. 3). In addition, there were no demonstrable changes in either heart rate or stroke volume.

There was a significant increase in PVR compared with baseline values within 3 minutes, and at 5 and 15 minutes following SBCA (Fig. 4, p < 0.05). SVR decreased significantly compared with baseline values (931 ± 923 dyne/sec/cm²; p < 0.005) within 3 minutes of SBCA. There were no significant changes from baseline SVR at 5 and 15 minutes.

PaO₂ decreased significantly from baseline values at 5 minutes (45 ± 60 mmHg) after SBCA in the lavage group (p < 0.02). No change in PaCO₂ was found.

Comparison between groups. The decrease in BP measured within 3 minutes of SBCA was significantly greater in the no lavage group than in the lavage group (Fig. 2; p < 0.0005). There was also a significantly greater increase in PAP in the no lavage group than at all three time periods after SBCA (Fig. 2; p < 0.0005).

The 21% decrease in cardiac output in the no lavage group was significantly greater than that in the lavage group. This difference was apparent within 3 minutes (p < 0.0005) as well as at 5 minutes (p < 0.005) after SBCA (Fig. 3). The decrease in stroke volume in the no lavage group was significantly greater than that in the lavage group (Fig. 3; p < 0.0005 within 3 minutes; p < 0.001 at 5 minutes). Similarly, the change from baseline PVR at all three time intervals was greater in the no lavage group (Fig. 4; p < 0.01). There were similar decreases in SVR within 3 minutes in both groups.

Prostaglandin assay. In the no lavage group, plasma 6-keto PGF₁α concentration increased significantly above baseline values within 3 minutes (mean increase 0.77 ± 0.39 ng/ml) and at 5 minutes (0.28 ± 0.1 ng/ml), returning to baseline values at 15 minutes (Fig. 5). Plasma T₂B₂ concentrations were also significantly increased in the no lavage group at all three measurement intervals (0.6 ± 0.52 ng/ml, 0.43 ± 0.35 ng/ml and 0.32 ± 0.24 ng/ml, within 3 and at 5 and 15 minutes respectively).

In the lavage group, the plasma concentration of 6-keto PGF₁α within 3 minutes of SBCA was significantly increased (mean 0.17 ± 0.19 ng/ml), but had decreased towards baseline values at 5 and 15 minutes (Fig. 5). The plasma T₂B₂ concentrations were not significantly increased above baseline values.

The changes in plasma 6-keto PGF₁α concentrations in the no lavage group were significantly greater both within 3 minutes and at 5 minutes (Fig. 5; p < 0.05). The changes in T₂B₂ concentrations in the no lavage group were not significantly greater than those observed in the lavage group.

Pathological results. Quantitative morphometry of post-mortem lung tissue showed that the mean number of pulmonary fat emboli per 60 mm² of lung tissue was very significantly different in the two groups (816 ± 80 in no
lavage dogs; 64 ± 15 in lavage; p < 0.00005). The volume proportion of lung tissue occluded by fat was also significantly greater (1.86 ± 0.1%; 0.2 ± 0.06%; p < 0.0005). The size of vessels occluded by fat ranged from 8.2 to 12.6 μm; there was no significant difference between groups in the size distribution of occluded pulmonary vessels.

DISCUSSION

Our study corroborates previous reports that pulsatile lavage of the medullary cavity before the cementing of joint replacements reduces acute cardiopulmonary instability (Sherman et al 1983; Byrick et al 1989). Lavage reduced both the number of emboli and the measured volume of lung tissue occluded by fat to approximately one-tenth of that observed without lavage. In the lavage group there was also a reduction in the degree of hypotension with no change in cardiac output, and reduction of pulmonary hypertension. Our important new finding was that a transient decrease in cardiac output in the no lavage group was abolished by intramedullary lavage before cementing and prosthesis insertion. The generation of arachidonic acid metabolites, particularly prostacyclin, was also significantly reduced by pulsatile lavage.

There are many reports of clinical hypoxaemia and transient hypotension during joint replacement surgery (Sevitt 1972; Herndon et al 1974; Kallos 1975; Modig et al 1975; Alexander and Barron 1979; Mebius and Hedenstierna 1982; Byrick, Forbes and Waddell 1986; Orsini, Richards and Mullen 1986), but the underlying pathophysiological mechanisms have been poorly understood.

Explanation of the association between fat embolism and acute haemodynamic instability requires a quantitative estimate of pulmonary fat embolism (McIntyre and Sasahara 1977). This cannot be achieved accurately in the clinical situation, although echocardiography has detected embolic material in patients (Heinrich et al 1985; Svarling 1988). A reliable animal model is needed and we have found that, in the SBCA model, the embolic load can be maximised by performing bilateral procedures simultaneously (Byrick et al 1987). It has been shown that the degree of pulmonary fat embolism is directly related to the magnitude of the intramedullary pressure at the time of cement and prosthesis insertion (Orsini et al 1987; Watson and Stulberg 1989), when reproducible and transient haemodynamic changes are created.

We continuously monitored aortic flow, acute changes in cardiac output and the relationship to changes in arterial BP. The results confirm our previous report of a transient decrease in cardiac output in association with a decrease in BP within 3 minutes of SBCA (Byrick et al 1992). These changes in cardiac output were prevented by prior pulsatile lavage of the medullary cavity. Since the heart rate did not change, the decreased cardiac output was due to a 21% decrease in stroke volume, which suggests that the BP changes are caused by either the mechanical effects of emboli or the activation of chemical mediators. The decrease in cardiac output after SBCA is not caused by PMMA, since bone cement was used in equal quantities in both groups and there was no decrease in the lavage group. Our results strongly implicate pulmonary fat and marrow embolism as the major factor and, if the animal model is relevant to the clinical situation, the rare but sometimes catastrophic circulatory collapse during cemented joint replacement is probably related to massive fat embolism.

Mild, transient hypotension is a more common observation at the time of cementing and prosthesis insertion. The experimental results in our lavage group suggest that this may be due to systemic vasodilatation, in the absence of a large embolic load. We found a significant decrease in SVR within 3 minutes of SBCA in both groups of dogs. In the no lavage group, when massive pulmonary fat embolism occurred, there was a marked reduction in BP, associated with both vasodilatation and reduced cardiac output. The normal homeostatic response to a reduced cardiac output would be vasoconstriction, but in our model, vasodilatation accentuated the hypotension. In the lavage group, transient hypotension occurred despite the lack of change in cardiac output and therefore the hypotension probably resulted from transient peripheral vasodilatation alone.

Other mechanisms have been proposed to explain the haemodynamic instability after cemented arthroplasty. These include methylmethacrylate monomer toxicity which causes either peripheral vasodilatation (Peebles et al 1972; Kim and Ritter 1972; McMaster et al 1974) or direct myocardial depression (Mir et al 1973). The dose of monomer, however, necessary to cause vasodilatation in dogs (Peebles et al 1972) greatly exceeds that liberated in man during cemented arthroplasty (McLaughlin et al 1973) and other workers have concluded that changes in BP were independent of circulating cement monomer and its metabolites (Breed 1974; Crout et al 1979). If monomer was a major contributor to the hypotension and reduced cardiac output, we would have expected to observe severe hypotension in the lavage group. We therefore conclude that monomer does not directly cause the decrease in cardiac output after cemented arthroplasty, although cement contributes physically to the increase in medullary pressure.

In our young, healthy dogs there was no change in RAP or LAP in either group, suggesting that neither intravascular volume depletion nor ventricular dysfunction was a major cause of the haemodynamic changes. Most of the patients having cemented arthroplasties are elderly, and many have cardiopulmonary disease. This myocardial ischaemia and dysfunction may complicate the development of hypotension. Paradoxical fat emboli
have been shown to enter the coronary circulation during total hip arthroplasty, giving rise to embolic myocardial ischaemia and cardiac arrest (Camann et al 1991). In our model the possibility of a patent foramen ovale was excluded by post-mortem examination of every dog.

Another proposed mechanism is a reflex autonomic response (Rudigier and Ritter 1983), and in our study, either monomer-induced vasodilatation or a reflex autonomic mechanism could have been responsible for the decrease in SVR in the lavage group. Primary vasodilatation alone cannot explain the decrease in cardiac output which accompanies the hypotension in the no lavage group.

We also investigated the role of prostaglandin release in haemodynamic changes. Prostaglandins are known to alter the vasoactive properties of both the pulmonary (Malik 1983) and systemic (Hudson et al 1990) circulations. Acute fat and marrow embolism at cemented arthroplasty has been shown to be associated with increased levels of vasoactive products of arachidonic acid metabolism (Byrick et al 1991) TxB2 (the stable metabolite of thromboxane A2), a potent pulmonary vasoconstrictor and 6-keto-PGF1α (the stable metabolite of prostacyclin, a known systemic vasodilator) were both transiently elevated in the no lavage group. 6-keto-PGF1α has been shown to be produced by the lungs in response to acute pulmonary fat embolism (Winn, Maunder and Harlan 1987; Byrick et al 1991). In this study pulsatile lavage was associated with a reduction in pulmonary emboli and a significantly reduced production of 6-keto PGF1α. Prostacyclin production alone could produce systemic vasodilatation, and could have produced the decrease in SVR in the lavage group. The inhibition of prostaglandin release by using ibuprofen does not appear to have a major role in preventing the acute haemodynamic instability seen after fat and marrow microembolism (Byrick et al 1992).

Conclusions. We have demonstrated two mechanisms which explain the haemodynamic instability during simultaneous bilateral cemented arthroplasty. In the lavage group cement and prosthesis insertion results in transient vasodilatation, causing mild hypotension with the maintenance of cardiac output, which explains the clinical finding that such hypotension is usually self-limiting. When there is significant embolism during cement and prosthesis insertion, however, the increase in PVR and right ventricular afterload causes an acute decrease in cardiac output (falling by 20% to 25% in our animal model). This leads to severe hypotension, aggravated by the superimposed systemic vasodilatation.

Our results suggest that the major clinical emphasis should be on the prevention of the early physiological effects of pulmonary fat and marrow microembolism. Evacuation of the contents of the medullary canal before cement pressurisation is strongly recommended and may be achieved by lavage (Sherman et al 1983; Byrick et al 1989) and venting (Breed 1974; Herndon et al 1974). An alternative is to avoid pressurisation by using a non-cemented arthroplasty (Orsini et al 1987; Watson and Stulberg 1989). The anaesthetist should seek to maintain cardiovascular stability and cardiac output by attention to intravascular fluid volume (McMaster et al 1974), the prevention of hypoxaemia, and the early aggressive treatment of hypotension, using a vasoconstrictor catecholamine to maintain systemic vascular resistance.

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REFERENCES


