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Hemodynamic Changes during Total Knee Replacement Surgery with Total Condylar Prosthesis

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Total knee replacement surgery with cemented Guepar prosthesis may induce complications such as hypotension, cardiac arrest, and fat embolism.[1–3] This prosthesis induces increases in pulmonary arterial pressure and pulmonary vascular resistance which were not observed when it was inserted without cement.[4,5] Total knee replacement surgery with total condylar prosthesis should avoid the above complications.[6] However, relevant hemodynamic data are not available. We sought to obtain hemodynamic data during total knee replacement surgery performed with a total condylar prosthesis inserted with or without the use of cement, inserted either during or after release of the tourniquet.

MATERIALS AND METHODS

Twenty-nine patients with no history or objective evidence of cardiopulmonary disease were studied after informed consent and approval by our Institutional Ethical Committee. All the patients were ASA Physical Status I or II. The patients were randomly divided into three groups. Group I consisted of six patients who underwent knee replacement without the use of bone cement (Freeman prosthesis). Groups II and III consisted of ten and 13 patients, respectively, who underwent knee replacement surgery with use of bone cement inserted after and before release of the tourniquet, respectively. The three groups were similar in respect to sex ratio and age (table 1). All patients were subjected to similar anesthetic techniques. Premedication consisted of hydroxyzine 100 mg po. Induction of anesthesia was performed with fentanyl 15 μg/kg iv, and endotracheal intubation facilitated by iv pancuronium 0.1 mg/kg. Anesthesia was maintained using controlled ventilation with N_2O 50% in oxygen and fentanyl 10 $\mu g \cdot kg^{-1} \cdot hr^{-1}$ iv. The limb was exsanguinated with an Esmarch bandage, and the pneumatic cuff was inflated to 500 mmHg. Intravascular volume was maintained with saline and red packed blood cells to maintain pulmonary artery wedge pressure (PWP) between 10 and 15 mmHg, and hematocrit between 30 and 40%. Central venous pressure (CVP), PWP, and pulmonary arterial pressure (PAP) were measured using a flow-directed triple-lumen pulmonary artery catheter, which was inserted *via* the internal jugular vein. Mean arterial pressure (MAP) was measured through a radial artery catheter. Heart rate (HR) was determined from a standard ECG lead. Cardiac output (CO) was measured in triplicate by the thermal dilution method with a computer (Edwards). Derived variables were:

Cardiac index (CI) = CO/body surface area

$(l/min/m^2)$

Systemic vascular resistance (SVR)

$= (MAP\text{-}CVP)/CI\ (mmHg/l/min/m^2)$

Pulmonary vascular resistance (PVR)

$= (PAP\text{-}PWP)/CI\ (mmHg/l/min/m^2)$

The quantities of the monomeric methylmethacrylate in expired gases sampled during 5 min were measured

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using a previously described technique.[7,8] Methylmethacrylate was collected during 5 min by absorption on 500 mg activated charcoal (Merck ref. 9624) on the expiratory circuit of a respirator which had a minute expired volume of 8 l/min. Desorption was performed with carbon disulphide. The solution was then analyzed by gas chromatography with a GIRA GC81 chromatograph, and data are given in μg for 500 mg activated charcoal at 8 l/min. The control hemodynamic measurements were performed 10 min after induction of anesthesia and before skin incision. The sequence of hemodynamic and methylmethacrylate measurements are shown in tables 2–4. In groups I and III, which had prosthesis insertion under tourniquet, measurements were performed just before and after release of the tourniquet. In group II, measurements were performed before and after release of the tourniquet and before and after prosthesis insertion. Statistical analysis was performed using ANOVA and Student's *t* test. Data are expressed as mean values ± SEM, and changes are considered statistically significant if $P < 0.05$.

Results

Statistical analysis did not show any difference between the control hemodynamic data of the three groups of patients studied (tables 2–4). Table 2 summarizes the hemodynamic variables measured in group I, in which no cement was used. Significant increases from the control values were observed in MAP and SVR before release of the tourniquet. These variables returned toward their control values after release of the tourniquet. Table 3 summarizes the hemodynamic changes observed in group II, in whom a cemented prosthesis was inserted after release of the tourniquet. Significant increases from the control values were observed in MAP and SVR before release of the tourniquet. These variables decreased after release of the tourniquet.

TABLE 1. Demographic Data

	Without Cement	With Cement	
	Group I Insertion Before Release of the Tourniquet	Group II Insertion After Release of the Tourniquet	Group III Insertion Before Release of the Tourniquet
Sex (M/F)	1/5	2/8	1/12
Age (yr)	69 ± 2*	75 ± 3	63 ± 3
Total patients (n)	6	10	13

M = Male; F = Female.
* Mean values ± SEM; no significant differences between groups.

Prosthesis insertion was followed by an increased PAP when compared to the control value. This variable was, however, not significantly increased from the pre-prosthesis insertion value. Table 4 summarizes the hemodynamic changes observed in group III, in which the cemented prosthesis was inserted during the tourniquet application. Before release of the tourniquet, significant increases from the control values were observed in MAP and PAP. Release of the tourniquet was followed by significant decreases in MAP and SVR. Tables 3 and 4 show that the monomeric methylmethacrylate concentrations in expired gases were low in both cemented groups.

Discussion

The hemodynamic changes observed during surgery in the three groups were similar, consisting of an increase in MAP during tourniquet application and a decrease in this variable after release. The hemodynamic changes observed in the three groups are probably only due to the tourniquet. Indeed, tourniquet pain is able, *per se*, to explain increased SVR and MAP which return to normal after its release. Group III showed significant

TABLE 2. Hemodynamic Data of Group I: Operation Without Cement (N = 6)

	Control	Inflation of the Tourniquet and Insertion of the Prosthesis +80 Min	Release of the Tourniquet	
			+86 Min	+100 Min
HR min^{-1}	60 ± 3	63 ± 2	61 ± 1	60 ± 2
MAP mmHg	92.3 ± 3.1	121.5 ± 9.9*	103.5 ± 10.8	104.3 ± 5.1
PAP mmHg	14.8 ± 3	16.8 ± 2.5	13 ± 2.7	12.8 ± 2.8
CVP mmHg	8.6 ± 2.6	10 ± 2.5	10 ± 2.9	7.8 ± 1.6
PWP mmHg	10 ± 1.8	12.1 ± 3	8.7 ± 2.7	9 ± 2.3
CI $l/min/m^2$	2 ± 0.2	2.2 ± 0.2	2 ± 0.2	1.9 ± 0.2
SVR $mmHg/l/min/m^2$	42.0 ± 2.9	57.7 ± 3.5*	48.4 ± 2.2	54.4 ± 5.8
PVR $mmHg/l/min/m^2$	2.4 ± 0.4	2.3 ± 0.5	2.1 ± 0.6	2.1 ± 0.5

Mean values ± SEM. HR = heart rate; MAP = mean arterial pressure; PAP = pulmonary arterial pressure; CVP = central venous pressure; PWP = pulmonary wedge pressure; CI = cardiac index; SVR = systemic vascular resistance; PVR = pulmonary vascular resistance.
* Statistically significant difference from control.

TABLE 3. Hemodynamic Data of Group II: Prosthesis Insertion with Cement After Release of the Tourniquet (N = 10)

	Control	Inflation of the Tourniquet +45 Min	Release of the Tourniquet		Insertion of the Prosthesis		
			+46 Min	+80 Min	+86 Min	+100 Min	+115 Min
HR min^{-1}	65 ± 4	62 ± 7	66 ± 5	70 ± 7	81 ± 4	65 ± 4	
MAP mmHg	87 ± 6.3	105 ± 9*	89 ± 3.7†	95 ± 5	96 ± 7	83 ± 5.3	
PAP mmHg	15.5 ± 1.8	20 ± 2.8	19 ± 2.8	20.7 ± 2.5	25.1 ± 3.7*	18.8 ± 2.5	
CVP mmHg	6.6 ± 0.9	8.6 ± 1.2	6.7 ± 1.3	7.6 ± 1.3	10.7 ± 1.6	7.2 ± 1.5	
PWP mmHg	11 ± 1.3	14 ± 2.2	12 ± 2.2	14 ± 4.4	17 ± 3.1	12 ± 2.2	
CI $l/min/m^2$	2.5 ± 0.2	2.4 ± 0.3	2.6 ± 0.2	2.8 ± 0.3	2.8 ± 0.2	2.7 ± 0.2	
SVR $mmHg/l/min/m^2$	34.1 ± 3.5	41.9 ± 5.4*	33.4 ± 2.3†	32.8 ± 3	30.8 ± 2.7	39.6 ± 3	
PVR $mmHg/l/min/m^2$	2.1 ± 0.3	2.2 ± 0.2	2.7 ± 0.7	2.5 ± 0.4	3.1 ± 0.6	2.7 ± 0.3	
Quantity of acrylic cement monomer absorbed μg					2.7 ± 2.4	6.4 ± 5.5	0

Mean values ± SEM. Abbreviations are as listed in Table 2.
* Statistically significant difference from control.
† Statistically significant difference from +45 min.

decreases in MAP and SVR after release of the tourniquet following prosthesis insertion. However, the decrease in MAP did not lead to systemic hypotension in this group. On the other hand, PAP, which was significantly increased after prosthesis insertion in group II, was not significantly increased from its value measured just before the prosthesis insertion. This increase in PAP which was not combined to an increase in PVR is, thus, not the effect of the acrylic cement monomer, but probably due to an increased preload as suggested by the trend, however insignificant, toward increased PWP. Thus, neither systemic hypotension nor pulmonary vasoconstriction were observed after total condylar prosthesis insertion.

Samii *et al.*[4] reported constant and highly significant increases in PAP and PVR after total knee replacement surgery using cemented Guepar prosthesis, whereas no change was observed when a non-cemented prosthesis was used. They also observed an acute systemic hypotension after release of the tourniquet when a cemented Guepar prosthesis insertion was performed.[5] These findings are in accordance with the frequent occurrence of complications observed after Guepar prosthesis surgery.[3] Total condylar prosthesis has been considered a safe total knee replacement procedure.[6] Two cases of fat embolism have been reported following this operation.[9] However, these cases were observed after bilateral total knee replacement was performed during the same surgical procedure. The low rate of complications after total condylar prosthesis surgery is probably due to the small area of contact with the bone and the small quantity of cement used. This is suggested by our study showing very low monomeric methylmethacrylate concentrations. Using the same dosage of monomeric methylmethacrylate, Tordjmann *et al.*[8] found values of 25 ± 10 μg and 264 ± 396 μg after acetabular and femoral prosthesis insertions, respectively, during total hip replacement surgery. The absence of acute hemo-

TABLE 4. Hemodynamic Data of Group III: Prosthesis Insertion with Cement Under Tourniquet (N = 13)

	Control	Inflation of the Tourniquet and Insertion of the Prosthesis +80 Min	Release of the Tourniquet		
			+86 Min	+100 Min	+115 Min
HR min^{-1}	55 ± 5.8	68 ± 5.8	67 ± 0.5	66 ± 4.7	
MAP mmHg	91 ± 6.4	116 ± 6.9*	84 ± 7.5†	92 ± 5.5	
PAP mmHg	16 ± 0.9	24 ± 1.9*	23 ± 2.5	22 ± 1.5	
CVP mmHg	9.5 ± 1.2	12.7 ± 1.2	10.5 ± 1.5	12 ± 1.2	
PWP mmHg	12 ± 1.2	18.1 ± 1.3	15.5 ± 1.6	16.1 ± 1.2	
CI $l/min/m^2$	2.0 ± 0.2	2.4 ± 0.2	2.7 ± 0.2	2.8 ± 0.6	
SVR $mmHg/l/min/m^2$	42.7 ± 3.6	44.5 ± 3	35.9 ± 4.1†	38.7 ± 5.2	
PVR $mmHg/l/min/m^2$	2.2 ± 0.4	2.7 ± 0.3	2.9 ± 0.8	2.2 ± 0.6	
Quantity of acrylic cement monomer absorbed μg		0	16.6 ± 7.2	7.2 ± 2.6	7.7 ± 4.4

Mean values ± SEM. Abbreviations are as listed in Table 2.
* Statistically significant difference from control.
† Statistically significant difference from +80 min.

dynamic changes and of high monomeric methylmethacrylate concentrations after cemented total condylar knee prosthesis in our small groups of patients does not, however, eliminate the potential risk of complications. A beta (type II) error is possible. Indeed, Svartling *et al.*,[10] studying the blood levels of monomeric methylmethacrylate in nine patients after total condylar knee prosthesis inserted with cement, observed low levels ranging between 0.1–1.44 μg/ml and a high level of 119.8 μg/ml in one patient who presented ventricular extrasystoles after release of the tourniquet.

In conclusion, our prospective study showed that total replacement surgery with total condylar prosthesis is followed only by acute systemic hemodynamic changes related to inflation and release of the tourniquet.

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The Use of Ventilation/perfusion Lung Scans to Predict Oxygenation during One-lung Anesthesia

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Selective ventilation of one lung provides a quiet surgical field and isolation of the operative bronchus during thoracic surgery.[1] Ten to forty per cent of patients undergoing this technique develop significant intraoperative hypoxemia.[2–4] It is difficult to anticipate, however, which patients will develop hypoxemia during surgery.

Kerr *et al.*[4] reported that patients undergoing one-lung ventilation for non-pulmonary surgery had a lower mean Pa_{O_2} than patients who underwent one-lung ventilation for a pulmonary resection. They postulated that the perfusion to the unventilated operative lung of patients undergoing a pulmonary resection may have been reduced chronically. This increase of pulmonary vascular resistance might protect against the development of a large pulmonary shunt and hypoxemia during one-lung ventilation. Subsequently, others have shown that, when blood flow to the operative lung is limited by intraoperative occlusion of the pulmonary artery, the Pa_{O_2} during one-lung anesthesia improves.[2,6,7] The influence of the preoperative distribution of pulmonary vascular resistance and, therefore, blood flow upon oxygenation during one-lung anesthesia has not been systematically studied.

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