POSTER SESSION XVI

A165

TITLE: HEMODYNAMIC CHANGES DURING CEMENTED NON HINGED TOTAL KNEE PROSTHESIS INSERTION: INFLUENCE OF TOURNIQUET UTILIZATION

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Insertion of hinged total knee prosthesis with bone cement induces major pulmonary and systemic hemodynamic changes (1), which are reduced if the tourniquet is maintained during bone cement insertion (2). As hemodynamic changes are less important with non hinged total knee prosthesis (3), the maintenance of a tourniquet can be questionned. We therefore evaluated the hemodynamic consequences of tourniquet utilization during cemented non hinged total knee prosthesis insertion.

After institutional approval and informed consent, 61 patients were randomly divided in two groups: in group 1 (n=24), prosthesis was cemented after the tourniquet had been released; while in group 2 (n=37), tourniquet was maintained during cement insertion. Anesthesia was induced with thiopental (3-5 $mg.kg^{-1}$) and vecuronium (0.1 $mg.kg^{-1}$), then maintained with 50% N_2O in O_2, and enflurane and fentanyl as required. Mechanical ventilation was adjusted to maintain $PetCO_2$ in a normal range. Mean arterial pressure (MAP, mmHg) was continuously recorded using a radial artery catheter. Pulmonary arterial pressure (PAP, mmHg) and cardiac output were measured using a Swan-Ganz catheter. Cardiac index (CI, $l.min^{-1}.m^{-2}$) and systemic vascular resistance (SVR, $dyne.s.cm^{-5}$) were calculated. Hemodynamic profiles were recorded: before tourniquet installation (Control); under tourniquet just before its release (Tourniquet) and three min after the release of the tourniquet (Release); three min after cement insertion (Cement). Results (mean ± SEM) were compared using ANOVA and modified t-test.

	Gr	Control	Tourniquet	Release (Gr1)	Cement	Release (Gr2)
MAP	1	77±2	90±3 *	72±4	83±3 *	
	2	72±2	86±2 *		92±3 **	67±2
CI	1	2.4±0.1	2.2±0.1	2.4±0.2	2.3±0.1	
	2	2.1±0.1	2.1±0.1		2.3±0.1	2.1±0.1
PAP	1	14±1	18±1 *	16±1	17±1 *	
	2	13±1	19±1 *		20±1 **	16±1
SVR	1	2379±162	2992±180*	2252±165	2689±191*	
	2	2709±181	2969±118*		3135±161*	2316±121

* $P < 0.05$ vs group 1. *$P < 0.05$ vs control values.

In both groups, MAP and PAP increased significantly after cement insertion, as compared to control values, while CI remained stable throughout the procedure. In both groups, MAP significantly decreased after tourniquet release, as compared to its previous value. After cement insertion, PAP and MAP were significantly higher in group 2 than in group 1.

Hemodynamic changes were moderate during cemented insertion of non hinged total knee prosthesis. The maintenance of the tourniquet during cement insertion had no influence on these changes. Tourniquet release, whenever it was performed, induced more pronounced hemodynamic changes than cement insertion itself.

References:
(1) Anesthesiology 52: 271-3,1980
(2) Anesthesiology 50: 239-41,1979
(3) Anesthesiology 67: A106,1987

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TITLE: INFLUENCE OF MODERATE HYPOCAPNIA ON PULMONARY VASCULAR TONE FOLLOWING MITRAL VALVE REPLACEMENT

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Mitral stenosis is almost always associated with increased pulmonary vascular resistance (PVR). Although hypocapnic alkalosis has been shown to be effective in decreasing PVR in infants, it is unclear whether this effect extends to adults with mitral disease. The present study was designed to investigate the effect of moderate hypocapnia on pulmonary vascular tone after mitral valve replacement in adults. To avoid the influence of changes in airway pressure on hemodynamics, hypocapnia was induced by removal of previously added deadspace.

After approval by the Institutional Review Board, informed consents were obtained from eight patients, with mitral stenosis, scheduled for elective mitral valve replacement. Postoperative mechanical ventilation was maintained with a tidal volume of 10 to 15 ml/kg, a rate of 10 breaths/min, and an FIO_2 of 0.6 to 1.0. The ventilator was initially adjusted to obtain a $PaCO_2$ of 37 to 44 mm Hg. At least four hours after surgery and two hours following cessation of any drug which might have affected cardiovascular parameters, the study commenced. Deadspace (corrugated breathing circuit tubing) equalling a volume of 200 to 400 cc was added to the ventilator circuit proximal to the Y piece, the tidal volume was increased by an equal amount, and the FIO_2 increased to 1.0. After a 30 minute equilibration period, complete hemodynamic and respiratory parameters, ventilator settings, and arterial and mixed venous blood gases were recorded (pre-hypocapnia). After initial measurements were obtained, the deadspace tubing was removed and mechanical ventilation was maintained for 30 minutes to produce moderate (hypocapnia). Following measurements , the deadspace was added again for 30 minutes and a final set measurements were obtained after 30 minutes (post-hypocapnia). Following the conclusion of the study, the deadspace was removed, and tidal volume and FIO_2 were returned the prestudy settings. All data was analyzed by an analysis of variance for repeated measures and the Student Newman-Keuls test. Statistical significance was accepted when $P < 0.05$.

	Pre Hypocapnia	Hypocapnia	Post Hypocapnia
pHa	7.38 ± 0.02	7.46 ± 0.02*	7.34 ± 0.02
$PaCO_2$	42.0 ± 2.1	33.3 ± 1.1*	47.7 ± 1.9
PaO_2	308 ± 43	305 ± 40	361 ± 52
CI	2.70 ± 0.17	2.70 ± 0.20	2.53 ± 0.17
HR	72 ± 3	76 ± 6	68 ± 9
MAP	79 ± 3	77 ± 1	76 ± 5
CVP	12 ± 1	12 ± 1	12 ± 1
PVRI	435 ± 61	352 ± 52*	478 ± 108
Qs/Qt	17.4 ± 1.9	15.7 ± 2.8	18.6 ± 2.7

† all data is presented as a mean ± the standard error of the mean
* Statistically significant from pre and post hypocapnia

Moderate hypocapnia ($PaCO2 = 33$ mm Hg) resulted in a statistically significant decrease in pulmonary vascular resistance index. There were no other statistically significant changes in hemodynamic or respiratory parameters measured except in pHa and $PaCO_2$ during hypocapnia.

The present study demonstrates that moderate hypocapnia is effective in decreasing pulmonary vascular tone in adults following mitral valve replacement. This may prove to be a useful therapeutic maneuver in the management of patients with pulmonary hypertension following mitral valve replacement in the postoperative period.