pH MANAGEMENT DURING HYPOTHERMIC CARDIOPULMONARY BYPASS DOES NOT INFLUENCE CEREBRAL OXYGEN CONSUMPTION

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Controversy regarding optimal pH management techniques during hypothermic cardiopulmonary bypass (CPB), includes reports of disproportionate decreases in cerebral metabolic rate for oxygen ($CMRO_2$) during pH-stat management at 27°C,[1] vs preservation of cerebral flow/metabolism coupling with proportionate decreases in cerebral blood flow (CBF) reported during alpha-stat pH management.[2] The following study was designed to prospectively assess the influence of pH management on CBF and $CMRO_2$ in patients during hypothermic CPB.

Methods: After obtaining institutional ethics committee approval and written informed consent, 5 patients, mean age 58±14 yr undergoing hypothermic CPB, had CBF measured using ^{133}Xe clearance. Using a jugular catheter for sampling effluent cerebral venous blood, $CMRO_2$ was determined as the product of CBF and cerebral arterial-venous oxygen content difference. Once a stable nasopharyngeal temperature had been obtained during CPB, patients were randomly assigned to either alpha-stat or pH-stat management techniques and CBF and $CMRO_2$ were measured. Following this, the alternate pH management technique was employed and CBF and $CMRO_2$ were remeasured after a minimum 5 min equilibration period. Data were analyzed using a paired t-test with $p < 0.05$ required for significance.

Results: There were no significant differences in temperature or mean arterial pressure between the two measurement periods. Mean temperature corrected $PaCO_2$ was 31.6±2.6 mmHg during alpha-stat and 40.8±3.8 mmHg during pH-stat pH management ($p<0.05$). Mean CBF was significantly higher in the pH-stat 32.9±5.2 $ml.100g^{-1}.min^{-1}$) vs a significant alpha-stat (19±2.2 $ml.100g^{-1}.min^{-1}$) groups ($p<0.05$). However, there was no significant difference in $CMRO_2$ between the two groups (0.61±0.17 vs 0.57±0.27 $ml.100g^{-1}.min^{-1}$, respectively).

Discussion: This study is consistent with reports demonstrating alterations in CBF, but no differences in $CMRO_2$, during alpha-stat vs pH-stat pH management,[2] but does not support the concept of decreases in $CMRO_2$ resulting from differences in pH management[1] over this range of $PaCO_2$ values.

References: 1.Rogers et al. Anesth Analg 67:S187, 1988. 2.Murkin et al. Anesth Analg 66:825-32,1987.

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Title: PHOSPHOLIPASE A_2 ACTIVITY AND PROSTAGLANDIN LEVELS DURING CARDIAC SURGERY.

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Prostaglandin levels (TXB_2,6-Keto) are elevated during CPB.[1] Phospholipase A_2 (PLA_2) is an enzyme involved in release of free fatty acids necessary for prostaglandin production.[2] This study examined PLA_2 activity and its relationship to prostaglandin levels during cardiac surgery.

Twelve adult patients undergoing CABG were studied with institutional approval and informed consent. Samples for measurement of PLA_2, TxB_2 and 6-Keto were obtained before induction, after incision, before and after heparin (3 mg/kg), at 15,30 and 60 min. of CPB, before and after protamine, and at end of operation. PLA_2 was measured by the method of Ballou[2]; TxB_2 and 6-Keto by radioimmunoassay.

No significant changes were detected until heparin administration. With this, PLA_2 activity rose significantly (0.13±0.02 to 0.46±0.09 pmol/min/mg - $p < 0.05$) and was accompanied by a significant rise in 6-Keto (96±28 to 454±92 pg/ml - $p < 0.05$). These remained elevated until after CPB. Protamine administration produced significant decreases (0.50±0.07 to 0.23±0.05 p.mol/min/mg - $p < 0.05$ and 370±100 to 200±47 pg/ml - $p < 0.05$ respectively). TxB_2 levels did not increase until CPB (124±20 to 197±36 pg/ml - $p < 0.05$), but remained elevated after protamine (195±32 to 240±49 pg/ml) reversing the TxB_2/6-Keto ratio (0.94±0.29 to 1.85±0.57).

These data demonstrate that heparin administration produces significant increases in PLA_2 activity associated with increases in 6-Keto but not TxB_2 levels. The increase in TxB_2 occurs with CPB and may be due to a number of factors (cellular destruction, etc.). While protamine administration reduces PLA_2 activity and returns 6-Keto toward control levels, it has no effect on TxB_2 levels. The reversal of the TxB_2/6-Keto ratio by protamine may be a factor in the deleterious effects sometimes associated with its administration.

References
1. J Thorac Cardiovasc Surg 84:250-256,1982
2. Proc Nat'l Acad Sci USA 80:5203-52, 1983