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 (CMRO₂) 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. The following study was designed to prospectively assess the influence of pH management on CBF and CMRO2 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 ¹³³Xe clearance. Using a jugular catheter for sampling effluent cerebral venous blood, CMRO2 was determined as the product of CBF and cerebral arterial-

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PHOSPHOLIPASE A ACTIVITY AND PROSTAGLANDIN LEVELS DURING Title:

CARDIAC SURGERY.

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Prostaglandin levels (TXB, 6-Keto) are elevated during CPB. Phospholipase A (PLA) is an enzyme involved in release of free fatty acids necessary for prostaglandin production. This study examined PLA 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, TxB, 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, was measured by the method of Ballou; TxB, and 6-Keto by radioimmunoassay.

No significant changes were detected until heparin administration. With this, 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 CMRO₂ were measured. Following this, the alternate pH management technique was employed and CBF and CMRO₂ 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 PaCO2 periods. Mean temperature corrected PaCO₂ 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 CMRO₂ between the two groups (0.61±0.17 vs. 0.57±0.27 the two groups (0.61±0.17 vs 0.57±0.27 ml.100g⁻¹.min⁻¹, respectively). ml.100g⁻¹.min⁻¹, respectively).

<u>Discussion:</u> This study is consistent with reports demonstrating alterations in CBF, but no differences in CMRO2, during alphastat vs pH-stat pH management, but does not support the concept of decreases in CMRO2 resulting from differences in pH management 1 over this range of PaCO2 values. References: 1.Rogers et al. Anesth Analg 67:S187 1988. 2.Murkin et al. Anesth Analg 66:825-32,1987.

PLA activity rose significantly $(0.13\pm0.02$ to 0.46 ± 0.09 pmol/min/mg - p (0.05) and was accompanied by a significant rise in 6-Keto $(96\pm28 \text{ to } 454\pm92 \text{ pg/ml} - \text{p } (0.05).$ remained elevated until after CPB. Protamine administration produced significant decreases $(0.50\pm0.07 \text{ to } 0.23\pm0.05)$ p.mol/min/mg - p <0.05 and 370 ± 100 to 200 ± 47 pg/ml - p <0.05 respectively). Txl levels did not increase until CPB (124 \pm 20 to 197+36 pg/ml - p (0.05), but remained elevated after protamine $(195\pm32 \text{ to } 240\pm49)$ 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 PLA2 activity associated with increases in 62Keto but not TxB, levels. The increase in TxB, occurs with CPB and may be due to a number of factors (cellular destruction, etc.). While protamine administration reduces PLA activity and returns 6-Keto toward control levels, it has no effect on TxB levels. The reversal of the TxB /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