

**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.² 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-

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 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 ml.100g⁻¹.min⁻¹, respectively).

Discussion: This study is consistent with reports demonstrating alterations in CBF, but no differences in CMRO₂, during alpha-stat vs pH-stat pH management,² but does not support the concept of decreases in CMRO₂ resulting from differences in pH management¹ 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.

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Title: PHOSPHOLIPASE A₂ ACTIVITY AND
PROSTAGLANDIN LEVELS DURING
CARDIAC SURGERY.

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Prostaglandin levels (TxB₂, 6-Keto) are elevated during CPB.¹ Phospholipase A₂ (PLA₂) is an enzyme involved in release of free² fatty acids necessary for prostaglandin production. This study examined PLA₂ 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 PLA₂, TxB₂ and 6-Keto were obtained before induction,² 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.² PLA₂ was measured by the method of Ballou²; TxB₂ and 6-Keto by radioimmunoassay.

No significant changes were detected until heparin administration. With this,

PLA₂ 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). TxB₂ 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 TxB₂/6-Keto ratio (0.94±0.29 to 1.85±0.57)².

These data demonstrate that heparin administration produces significant increases in PLA₂ activity associated with increases in 6-Keto but not TxB₂ levels. The increase in TxB₂ occurs with CPB and may be due to a number of factors (cellular destruction, etc.). While protamine administration reduces PLA₂ activity and returns 6-Keto toward control levels, it has no effect on TxB₂ levels. The reversal of the TxB₂/6-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