

Title: CEREBROVASCULAR RESPONSIVENESS TO PaO₂ IS PRESERVED DURING HYPOTHERMIC CARDIOPULMONARY BYPASS

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Introduction:

In awake volunteers, cerebral blood flow (CBF) changes in response to alterations in arterial oxygen tension (PaO₂)^{1,2}. We report CBF changes induced by specifically varying PaO₂ during hypothermic cardiopulmonary bypass (CPB).

Methods:

We studied eight patients undergoing elective cardiac surgery, with the approval of the Clinical Research Practices Committee. All gave informed written consent. Subjects were excluded for chronic hypertension and known cerebrovascular disease. After premedication with lorazepam po and morphine im, we induced narcosis with fentanyl 75 mg.kg⁻¹, while pancuronium and/or metocurine produced muscle relaxation. No additional drugs other than O₂ were given until completion of measurements.

During hypothermic nonpulsatile CPB, we determined regional CBF by clearance of ¹³³Xenon injected via the arterial inflow cannula. Since regional variance was minimal, mean global CBF for each patient was calculated by averaging regional values from 16 cadmium telluride gamma detectors. We utilized the CBF₁₅ computation technique, corrected for decreases in nasopharyngeal temperature (NPT) and hematocrit (Hct). PaCO₂ was held constant at approximately 40 mm Hg, uncorrected for NPT. Pump flow (Q), Hct, and mean arterial pressure (MAP) were maintained within narrow limits throughout. During stable hypothermia (NPT = 28°C), baseline CBF was measured and then repeated after specific alteration of PaO₂, achieved by varying the FiO₂ of gas flow to the membrane oxygenator. Because CBF declines spontaneously with time during CPB, we randomized the direction of PaO₂ change. In the other 4 patients, we increased PaO₂ from the first to the second measurement. In 4 patients we decreased PaO₂ from the first to the second measurement. Data from all 8 patients were combined for statistical analysis. Differences in CBF were compared using paired two-tailed t-tests, significant at p<0.05. All values are expressed as mean ± standard deviation (SD).

Results:

Controlled variables remained similar during both measurement intervals (Table). The lower PaO₂ in each patient averaged 128±50 mmHg; the higher PaO₂ averaged 291±46 mmHg. This was associated with a 14% change in CBF from 13.9 ± 3.9 to 12.0 ± 3.2 ml.100g⁻¹.min⁻¹ (p<0.02)(Figure).

Discussion:

In normal awake persons, hypoxemia (FiO₂ = .1) increases CBF by 35%, while hyperoxia (FiO₂>.85) decreases CBF by approximately 15%^{1,2}. Cerebral oxygen utilization remains unchanged. This study examined the response of CBF to changes in PaO₂ within a range acceptable during clinical CPB. Our results demonstrate that cerebrovascular responsiveness to PaO₂ is preserved during hypothermic nonpulsatile cardiopulmonary bypass.

Table Controlled Variables (mean ± SD)

PaO ₂ (mmHg)	NPT (°C)	Q (L/min/m ²)	PaCO ₂ * (mm Hg)	Hct (vol%)	MAP (mmHg)
128±50	27.2±.7	1.95±.5	41±1.4	22±3	70±12
291±46	27.3±.7	2.00±.6	40±1.9	22±3	72±10

* Uncorrected for body temperature

References:

- Lassen NA: Cerebral blood flow and oxygen consumption in man. *Physiol Rev* 39:183-238,1959
- Kety S, Schmidt CF: The effects of altered arterial tensions of carbon dioxide and oxygen on cerebral blood flow and cerebral oxygen consumption of normal young men. *J Clin Invest* 27:484-492,1948

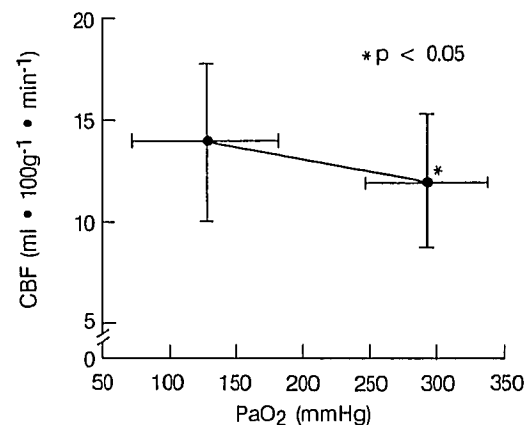


Figure. Correlation of CBF with PaO₂ during CPB