Title: NO INTRACEREBRAL STEAL DURING HYPERCARBIC HYPOTHERMIC CARDIOPULMONARY BYPASS

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The pH-stat method of CO2 Introduction. management during hypothermic cardiopulmonary bypass (CPB) (temperature corrected PaCO, maintained near 40 mmHg) increases PaCO₂ and cerebral blood flow (CBF)^{1,2} and decreases cerebral oxygen consumption (CMRO₂)¹ relative to the alpha-stat method (PaCO₂ maintained at 40 mmHg uncorrected for body temperature). These effects are desirable if they reduce ischemia in focally diseased brain regions in patients with cerebrovascular disease (CVD). However, in patients undergoing carotid endarterectomy, hypercarbia causes a redistribution of CBF from poorly perfused to well-perfused areas. We studied patients with CVD undergoing CPB to determine whether pH-stat management also produces intracerebral stéals.

 $\underline{Methods}.$ In a study approved by the institutional Clinical Research Practices Committee, we obtained informed consent and measured CBF in 9 patients undergoing hypothermic CPB for treatment of coronary artery disease. Inclusion criteria consisted of clinical or angiographic evidence of occlusive cerebrovascular disease, including previous stroke or highgrade extracranial obstruction. No patients with recent stroke (< 3 months previously) were studied. Controls consisted of 9 concurrently studied patients without CVD. Drugs for premedication were lorazepam 50 $\mu g/kg$ p.o. and morphine 0.1 mg/kg im; for induction, intubation and maintenance, fentanyl 75 µg/kg iv and pancuronium 0.1 mg/kg iv. No additional agents other than enflurane (< 0.5% pre-bypass) were administered until measurements were completed.

During hypothermic, nonpulsatile regional CBF was determined by the clearance of Xenon, dissolved in saline and injected into the arterial line of the pump-oxygenator. CBF was calculated from the 133 Xenon clearance curves from 12 - 16 brain regions using the CBF₁₅ technique. PaCO₂ was varied in random order from a higher (pH-stat) to lower (alpha-stat) or from a lower to a higher level, once a stable nasopharyngeal temperature (NPT) was attained. NPT, perfusion flow rate (Q), mean arterial pressure (MAP), and temperature (Temp) were kept within narrow limits (Table).

Data were analyzed to answer questions: 1. Are intracerebral steals produced by hypercarbia? 2. Do low and high CBF regions respond differently to increased PaCO2? 3. Does CBF respond differently to changes in PaCO, in patients with and without cerebrovascular Paired two-tailed Student's "t" tests disease? (p < 0.05 considered significant) were used to evaluate the latter two questions.

 $\underline{\text{Results}}$. No demonstrable intracerebral steal occurred in any brain region in any

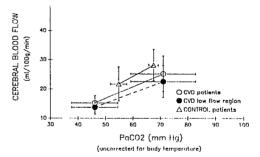
In the regions demonstrating the lowest flow at the lower PaCO, the CBF response to changes in PaCO, was statistically similar to the response of the brain as a whole (Fig). The average CO₂ response of patients with CVD was similar to the response of patients without clinical CVD. CBF increased an average of 3.40 \pm 2.32 ml/100g/min per mmHg PaCO in the CVD group and an average of 2.79 \pm 2.61 ml per 100g/min per mmHg in patients without CVD.

Discussion. No data clearly demonstrate which CO₂ management method is clinically preferable in patients with CVD undergoing CPB. Although the alpha-stat method appears to be more physiologic in patients with normal cerebral vasculature, the pH-stat method could cerebral vasculature, the pH-stat method could protect the brain by increasing CBF and decreasing CMRO₂. But, because the pH-stat method may be equivalent to hypercarbia, it creates a potential for intracerebral steals in CVD patients. In this study, however, the pH-stat approach increased CBF without inducing steals.

Table. Cerebrovascular Disease Patients

	Lower PaCO ₂	Higher PaCO ₂
PaCO ₂ (mmHg)*	46.1 <u>+</u> 8.4	71.1 <u>+</u> 11.8
PaCO2 (mmHg)**	29.7 ± 7.5	46.4 ± 8.9
NPT (degrees C)	28.0 ± 3.0	26.8 ± 2.2
MAP (mmHg)	66.5 ± 6.3	68.4 ± 9.1
Q (L/min/m²)	2.2 ± 0.7	2.1 ± 0.7
Hct (%)	23.4 ± 3.9	23.2 ± 3.7
CBF (m1/100g/min)	15.2 ± 2.5	25.3 ± 6.1
CBF (low flow region)		22.7 ± 5.6

*temp-uncorrected; **temp-corrrected; mean + SEM



References.

- Rogers AT, Prough DS, Stump DA,
- Anesth Analg 67:S187, 1988
 2. Murkin JM, Farrar JK, Tweed WA, et al: Anesth Analg 66:825-32, 1987
- 3. Boysen G, Ladegaard-Pedersen HJ, Henriksen H, et al: Anesthesiology 35:286-300, 1971.