

**SCA 131**  
**HEMODILUTION DURING CARDIOPULMONARY BYPASS INCREASES CEREBRAL INFARCT VOLUME FOLLOWING MIDDLE CEREBRAL ARTERY OCCLUSION IN RATS**

Homi HM<sup>1</sup>, Grocott HP<sup>1</sup>, Pearlstein RD<sup>2</sup>, Yang H<sup>1</sup>  
*Departments of Anesthesiology<sup>1</sup> and Surgery<sup>2</sup>, Duke University Medical Center, Durham, NC*

**Introduction:** Hemodilution occurs commonly during cardiopulmonary bypass (CPB), either spontaneously or as a result of planned acute normovolemic hemodilution (ANH). Although the optimal hematocrit (Hct) during CPB is not known, excessive hemodilution may lead to organ ischemia via a reduction in oxygen carrying capacity uncompensated by autoregulatory and/or rheologic increases in organ blood flow.<sup>1,2</sup> As a result, the consequences of hemodilution in patients at risk for cerebral ischemia are not clearly understood. This study was designed to evaluate the effects of hemodilution in the setting of focal cerebral ischemia during CPB.

**Methods:** Fasted male Wistar rats (290-340g) were anesthetized, intubated, ventilated, and surgically prepared for CPB. Cannulae were placed in the superficial epigastric artery for mean arterial pressure (MAP) monitoring, tail artery for the CPB circuit arterial inflow, and external jugular vein for venous return.<sup>3</sup> The animals were randomized into a LOW Hb group (target on-CPB Hb = 6.0 g/dL, obtained by ANH) and a HIGH Hb group (target Hb = 12 g/dL) following which the right carotid artery was prepared for middle cerebral artery occlusion (MCAO).<sup>4</sup> Immediately after the onset of MCAO (which was maintained for 90 min), hypothermic (28°C) CPB (flow rate: 150-170 ml/kg/min) was initiated utilizing a modified neonatal membrane oxygenator and a roller pump. The CPB time was 65 min which included rewarming to 37.5°C by the end of CPB. The LOW Hb group was transfused to 11 g/dL in the early post-CPB period. 24 hours later, functional neurological testing (maximum Neuroscore 18 = normal behavior) was performed following which the brains were harvested, sliced and stained with 2% 2,3,5-Triphenyltetrazolium chloride, thereby allowing cerebral infarct volumes to be measured. Data were compared between groups using ANOVA or the Mann-Whitney U test, as appropriate. Statistical significance was considered when P<0.05.

**Results:** The data are summarized in the table below. Values are expressed as the mean ± SD except for Neuroscore (median ± IQR). Compared to the HIGH Hb group, the rats in the LOW Hb group had significantly worse functional and histologic outcomes. \*

Parameters	LOW Hb (n=9)	HIGH Hb (n=8)	P value
Hb- baseline (g/dL)	15.6 ± 1.2	15.2 ± 1.0	NS
Hb- during CPB (g/dL)	6.0 ± 0.3	11.3 ± 0.6	0.0005
Hb- 120 min post CPB (g/dL)	10.1 ± 1.7	11.3 ± 0.8	NS
MAP during CPB (mmHg)	90 ± 5	91 ± 5	NS
Glucose during CPB(mg/dL)	159 ± 27	153 ± 33	NS
Neuroscore	8 ± 2.2	10 ± 2.0	0.0304
Total Infarct volume (mm <sup>3</sup> )	182 ± 84	103 ± 58	0.0433
Cortical infarct volume (mm <sup>3</sup> )	171 ± 26	77 ± 43	0.0043
Subcortical infarct volume (mm <sup>3</sup> )	49 ± 14	45 ± 12	NS

**Conclusion:** In this experimental model combining CPB with reversible MCAO-induced focal cerebral ischemia, hemodilution worsened neurological function and increased cerebral infarct volume. We speculate that this worsening of outcome was related to the inadequate delivery of hemoglobin-bound oxygen to the penumbra area. Although hemodilution has several advantages, including a reduction in the need for blood transfusion, its benefits must be weighed against its potential risks. Further work is needed in order to determine the optimal level of hemodilution during CPB, particularly in the setting of cerebral ischemia.

References:

1. J Cardiothorac Vasc Anesth 1996; 10: 54-65
2. Anesth Analg 1999; 89: 1078-83
3. Anesthesiology 2001; 95: 1485-91
4. Stroke 1989; 20: 84-91