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BRIEF COMMUNICATIONS

CEREBRAL HYPERTHERMIA DURING CARDIOPULMONARY BYPASS IN ADULTS

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In this report, we document that cerebral hyperthermia occurs regularly during rewarming from hypothermic cardiopulmonary bypass (CPB). These high brain temperatures are not adequately reflected by nasopharyngeal (NP) temperature and may contribute to neurologic morbidity. Stroke and neurocognitive injury remain significant components of perioperative morbidity associated with cardiac surgery. Hypothermic CPB is used in part for its cerebral protective effects. Recent reports, however, have indicated that the rewarming phase of hypothermic CPB is associated with a significant incidence of decreased cerebral venous oxygen saturation ($Sjvo_2$; ≤50%).[1,2] $Sjvo_2$ is thought to be a general index of the adequacy of the matching of cerebral oxygen supply with demand.

Early CPB studies demonstrated that significant temperature gradients exist during the cooling phase of CPB and that NP temperature may not be an adequate indicator of either brain or whole-body temperature.[3] We therefore undertook this study to examine changes in cerebral venous temperature and $Sjvo_2$ during rewarming from hypothermic CPB.

Methods

After obtaining approval from the Institutional Review Board and written, informed patient consent, we studied 10 adults undergoing hypothermic (27° C) CPB for combined valvular or valve-coronary procedures. A 5.5F oximetric Swan-Ganz catheter (Abbott Critical Care, Chicago, Ill.) was placed retrogradely into the right jugular bulb with

fluoroscopy. NP and cerebral venous temperature at the jugular bulb were recorded throughout rewarming. Anesthesia consisted of fentanyl and midazolam. The pH management was alpha-stat. The maximum heat exchanger–blood temperature gradient never exceeded 10º C, and water temperature never exceeded 41º C. Otherwise, the management of rewarming was according to the discretion of the perfusionist, who was kept unaware of cerebral venous temperature. Adequate rewarming was considered to be reflected by NP temperatures of at least 37.5º C and temperatures of the venous return to the CPB pump of at least 37.2º C. The ascending aorta was cannulated in all patients. The oximetric catheter was calibrated before insertion and again in vivo if necessary. The oximetrically measured $Sjvo_2$ was recorded continuously. Systemic venous oxygen saturation was also continuously measured (CDI 100; Cardiovascular Devices, Inc., Irvine, Calif.).

Results

In this group of 10 patients, mean age was 69 years, mean weight was 78 kg, mean crossclamp time was 81 minutes, and mean total CPB time was 133 minutes. All patients were cooled to NP temperatures of 27º C. Cerebral temperature rises extremely quickly during rewarming (Fig. 1). In the 10 patients studied, cerebral venous temperature at the jugular bulb reached 37º C in a mean of 10 minutes, whereas mean NP temperature at this time was still 34º ± 2.9º C. When NP temperature was 37º C (mean 18 minutes of rewarming), mean cerebral venous temperature was 38.2º ± 1.1º C. All 10 patients had peak cerebral venous temperatures of at least 39º C before termination of CPB, and the average duration that cerebral venous temperature exceeded 39º C was 15 minutes. $Sjvo_2$ decreased with rewarming in all patients, and five of 10 patients demonstrated $Sjvo_2$ values lower than 50%. In these patients, desaturation occurred at a mean of 14 ± 7 minutes of rewarming, when mean jugular bulb and NP temperatures were 38º and 34.8º C, respectively (Fig. 1). Systemic venous oxygen saturation was greater than 60% in all patients when cerebral venous desaturation occurred. The mean peak gradient between jugular bulb and NP temperature was 4.9º C; this occurred at 7.4 minutes of rewarming (Fig. 1). The sample size is too small to determine whether desaturation could be attributed to differences in age, body weight, hemoglobin concentration, or rapidity of rewarming, although the patients who demonstrated desaturation do appear to have been rewarmed more rapidly (Table I). None of the 10 patients demonstrated gross focal neurologic deficit after operation, although no formal neurologic or neurocognitive testing was conducted.

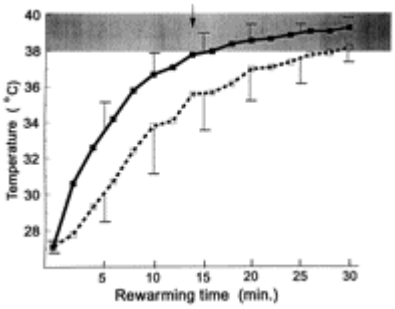

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Fig. 1. Mean NP (❑) and cerebral venous (■) temperatures during the first 30 minutes of rewarming from hypothermic CPB (n = 10). Standard deviations are shown every 5 minutes. *Down arrow* designates mean time of the cerebral venous desaturation occuring in five patients.

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Table I. Demographic and physiologic variables in patients with and without cerebral venous oxygen desaturation

Discussion

Our institutional perfusion practice is conventional. Excessive gradients between heat exchanger, perfusate, and NP temperatures are avoided, and body and blood temperature are monitored at standard sites (NP, perfusate, venous return). Regardless, cerebral hyperthermia (cerebral venous blood temperature >38° C) occurred in all of our patients; in half, this appeared to be associated with oxygenation stress.

Cerebral hyperthermia probably occurs during rewarming as a result of high cerebral blood flow and the proximity of carotid origins to the aortic cannulation site. Previously in this Journal,[1] we speculated that rapid rewarming may be responsible for low $Sjvo_2$ during rewarming. Hyperthermia increases brain oxygen demand and may accentuate the ischemia-related injury cascade. It has been established that elevated temperature after ischemic insult worsens neurologic outcome,[4,5] so cerebral hyperthermia may contribute to post-CPB confusion, encephalopathy, and neurologic or neurocognitive injury. Systematic evaluation of rewarming strategies that can achieve adequate body rewarming while avoiding cerebral hyperthermia must be undertaken.

Footnotes

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