

Management of Temperature During and After Cardiac Surgery

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Neurologic injury is a devastating complication of cardiac surgery. Cerebral cooling is an important aspect of hypothermic cardiopulmonary bypass in some patients, because hypothermia is the only reliable method of neuroprotection against injuries related to cerebral ischemia. Hypothermia may afford neuroprotection by a variety of mechanisms, including reduction in cerebral metabolic rate, decreased excitatory transmitter release, reduced ion influx, and reduced vascular permeability.

Conversely, hyperthermia, even if mild (2–3 °C), is harmful; it aggravates ischemic neuronal injury and accelerates neuronal death. In patients with acute strokes, hyperthermia worsens prognosis with respect to stroke severity, infarct size, mortality, and outcome in survivors.

The degree of temperature discrepancy among standard monitoring sites in individual patients is often striking. The differences between jugular bulb temperature and rectal or bladder temperature are particularly large. Blood temperature in the arterial line leading from the oxygenator may be the most consistently accurate indicator of cerebral temperature.

When hypothermia is used to protect vital organs during cardiopulmonary bypass, the cooling phase should be adequate, and the rewarming phase must be carefully managed. Hyperthermia may be as hazardous during the postoperative period as during surgery, exacerbating the extent of tissue injury if an overt stroke has occurred. Postoperative hyperthermia correlates with a greater degree of cognitive dysfunction measured 6 weeks after cardiac surgery.

In conclusion, cardiac anesthesiologists can reduce the risk of inadvertent hyperthermia by selecting the best sites for temperature monitoring, carefully controlling the rewarming process, and continuing temperature monitoring during the postoperative period. (*Tex Heart Inst J* 2005;32:472-6)

For decades, anesthesiologists and other specialists have studied the effects of cerebral hypoxia and ischemia and have developed methods to protect the brain under these conditions.^{1,2} Cerebral cooling is an important aspect of hypothermic cardiopulmonary bypass (CPB), because deliberate hypothermia reliably protects the brain from ischemic injury.^{1,3-5} Even mild hypothermia—as little as 1 °C—will diminish the severity of cerebral ischemia. For every 1 °C that brain temperature is decreased, cerebral metabolic rate decreases by 7%.⁶

Benefits of Hypothermia

Hypothermia provides neuroprotection against ischemia by several mechanisms. First, it creates a favorable balance between oxygen supply and demand. Second, it decreases excitotoxic neurotransmitter release, which is believed to play an important role in delayed neuronal death.^{1,2} Third, hypothermia is a crucial factor in preventing blood-brain barrier dysfunction and decreasing permeability in brain arterioles.^{1,7} Fourth, hypothermia appears to decrease polymorphonuclear leukocyte adhesion in the damaged region.⁸

Harmful Effects of Hyperthermia

In contrast, even mild hyperthermia (1–2 °C) can be harmful. Increased cerebral temperature significantly aggravates ischemic neuronal injury and accelerates neuronal death.^{4,9} Thus, increased brain temperature exacerbates the effects of cerebral ischemia, including the histopathologic consequences, stroke severity, and risk of death. This is so for both intra-ischemic mild hyperthermia (that is, hyperthermia that is present when the injury occurs) and for delayed, postischemic hyper-

thermia.¹⁰ In addition, hyperthermia delays neuronal metabolic recovery⁹ and increases excitotoxic neurotransmitter release,¹¹ oxygen free-radical production,¹² intracellular acidosis,⁹ and blood-brain barrier permeability.⁷ Hyperthermia also modulates protein kinase activity¹³ and destabilizes the cytoskeleton.¹⁴

Several clinical studies have shown a strong association between body temperature and outcome after stroke. Reith and colleagues¹⁵ reported that in patients with acute strokes, low body temperature at hospital admission was independently and significantly related to lower initial stroke severity, lesion size, and mortality; and with a better outcome in survivors. An increase in body temperature of only 1 °C was associated with a 4-point difference in patients' Scandinavian stroke severity scores (SSS)¹⁶ at discharge, a 15-mm difference in infarct size, and an 80% difference in mortality rate. Azzimondi and coworkers¹⁷ showed that a fever of at least 37.9 °C, regardless of the cause, indicated a greater likelihood of death within 30 days. After an initial feasibility study was performed,¹⁸ a large, randomized clinical trial—the Copenhagen Stroke Study—showed that body temperature at admission predicts long-term mortality after acute stroke.¹⁹

Neuroprotection with Hypothermia

Because it affects the outcome of cerebral ischemic events, brain temperature may influence the extent of neurologic injury during and after cardiac surgery with CPB.^{20–22} Therefore, when hypothermia is used to protect vital organs, the cooling phase should be adequate and rewarming must be carefully managed. More than half of the overt strokes recognized after cardiac surgery probably occur during surgery itself.²³ Salazar and colleagues²³ reported that in the Johns Hopkins Hospital cardiac surgery database, 74% of strokes were identified on the day of surgery and 91% were identified within the first 3 postoperative days. The presence of cerebral hyperthermia during surgery can only aggravate the tissue injury that ensues. Furthermore, intraoperative hyperthermia has been associated with significantly worse postoperative neuropsychological performance.^{20,24}

During hypothermic CPB, hypothermia is always initiated after aortic cannulation and the onset of bypass. Patients are rewarmed before bypass is terminated, usually before the aortic cross-clamp is removed. Cerebral embolism is unlikely during the hypothermic period because the heart is excluded from the circulation by the aortic cross-clamp throughout this period. Cerebral embolism most often occurs during periods when the brain is warm or warming, particularly during and immediately after removal of the aortic cross-clamp.²⁵ Therefore, too aggressive rewarming in an attempt to avoid the “afterdrop” in temperature that usually occurs after discontinuation of CPB may cause

cerebral hyperthermia during a period when cerebral embolism is likely. Although some investigators advocate the development of a pharmacologic agent to provide neuroprotection during these vulnerable periods, no such magic bullet has yet been discovered.²⁶ Regardless of any agent that may be used or developed, clinicians should continue to avoid inducing hyperthermia.

The site of temperature monitoring during hypothermic CPB is crucial, because sites vary substantially in the readings they produce.^{27–29} Monitoring bladder or rectal temperatures is standard practice in many institutions, and many clinicians try to normalize temperature at these sites by aggressive rewarming before CPB is terminated. However, when deep hypothermia is rapidly induced, relatively noninvasive temperature measurements made at standard body monitoring sites do not approximate intracerebral temperature well.²⁷ We and others^{27,29–32} have shown that, during rewarming from hypothermic CPB, temperatures measured in the nasopharynx, esophagus, and rectum differ substantially from those measured in the jugular bulb. In a recent study at the Texas Heart Institute*, we compared jugular bulb temperature—the best in vivo indicator of cerebral cortical temperature—throughout the rewarming phase of CPB in 16 patients undergoing hypothermic CPB for coronary artery bypass grafting. A 6-channel monitor continuously and simultaneously recorded all temperatures. We found that the bladder temperature was more than 4 °C lower than the jugular bulb temperature during some periods of rewarming, and nasopharyngeal and esophageal temperatures differed from jugular bulb temperature by as much as 2 °C during much of the rewarming phase (Fig. 1).

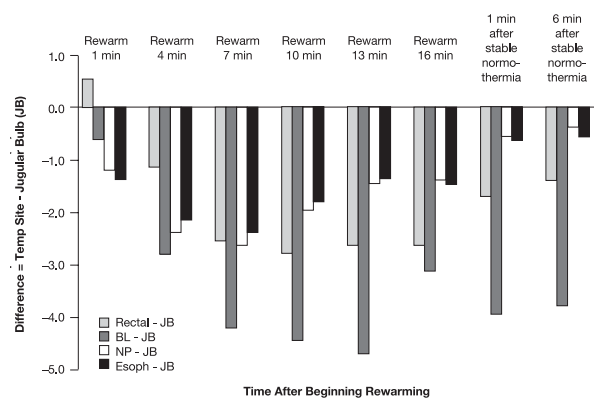


Fig. 1 Temperature differences between the jugular bulb (JB) site and the rectal, bladder (BL), nasopharyngeal (NP), and esophageal (Esoph) sites during rewarming in 16 patients undergoing cardiopulmonary bypass during cardiac surgery.

*Unpublished data; 2001–2002.

These findings have led us to modify our practice at the Texas Heart Institute. During CPB, we do not allow the nasopharyngeal temperature to go above 37 °C. We also start rewarming the patient much earlier and rewarm more slowly than we did previously. We never set the water bath above 38 °C. Importantly, we do not monitor bladder or rectal temperatures, because they do not accurately indicate cerebral temperature.

Our alterations in practice have improved our ability to avoid hyperthermia. In subsequent studies, the temperature difference between the jugular bulb and the bladder and rectum varied by about 3 °C during rewarming, which is better than the 5 °C difference we saw before these changes were put into practice.²⁹ There is still a 1 to 2 °C gradient for nasopharyngeal and esophageal temperature, but these eventually equilibrate. The mean rewarming time is 38 minutes, which is a bit longer than it was before we instituted these changes; the rewarming rate is 0.2 °C per minute.²⁹

Blood temperature in the arterial line leading from the oxygenator may be the most consistently accurate indicator of cerebral temperature.²⁹ When we recorded pump outflow temperature with a temperature probe, we found that it closely approximated jugular bulb temperature within approximately 10 minutes (Fig. 2), leading us to use the arterial line temperature as our current gold standard monitor.

Postoperative Hyperthermia

Hyperthermia that develops after surgery may be just as hazardous as intraoperative hyperthermia, and avoiding it requires fastidious attention. Postoperative hyperthermia is known to be correlated with a greater degree of cognitive dysfunction 6 weeks after cardiac surgery³³ (Fig. 3). Temperatures exceeding 38.5 °C are common during the first 48 hours after cardiac sur-

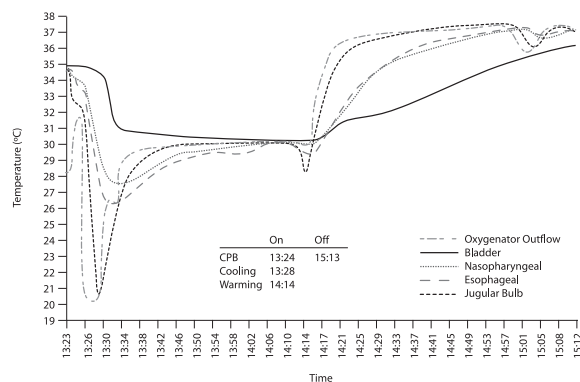


Fig. 2 Temperature monitoring during cardiopulmonary bypass (CPB) and rewarming in a patient undergoing cardiac surgery. Temperatures in the jugular bulb and oxygenator outflow are similar throughout and are virtually superimposable during rewarming.

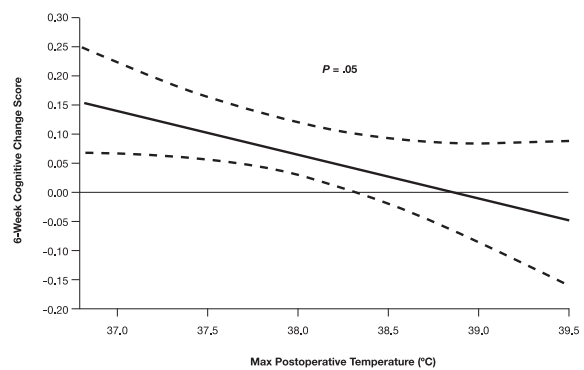


Fig. 3 The association between maximum postoperative temperature and cognitive change score 6 weeks after coronary artery bypass grafting.

(From: Grocott HP, Mackensen GB, Grigore AM, Mathew J, Reves JG, Phillips-Bute B, et al. Postoperative hyperthermia is associated with cognitive dysfunction after coronary artery bypass graft surgery. *Stroke* 2002;33:537-41. Reproduced with permission from Lippincott Williams & Wilkins.)

gery, occurring in nearly 40% of patients.³⁴ Furthermore, fever can exacerbate the extent of tissue injury if an overt stroke occurs. Salazar's group²³ and Hogue and co-authors³⁵ have noted that approximately one fourth of strokes that occur after CPB occur during the postoperative period, usually in patients who initially had an uneventful recovery. These strokes may not be directly related to the CPB; other potential causes include circulatory failure or atrial fibrillation.^{35,36}

These findings have led some clinicians to reduce the temperature at which they wean patients from bypass. In a randomized controlled study, Nathan and colleagues³⁷ showed that patients rewarmed to a lower temperature (34 °C) before weaning were significantly less likely to have postoperative cognitive deficits than were patients rewarmed to 37 °C (48% vs 62% at 1 week, with the trend continuing to 3 months). It is important to note that this lower post-bypass temperature has the potential to cause hemodynamic instability or coagulopathy;⁵ the risks and benefits must therefore be weighed for each individual patient.

Summary

Hypothermia (33–35 °C) during cardiopulmonary bypass has well-documented neuroprotective benefits. Evidence suggests that hypothermia improves neurologic outcome even when induced after a cerebral ischemic event. Rewarming must proceed slowly, and the clinician may consider weaning at temperatures slightly below 37 °C in patients at high risk of an ischemic event. Hyperthermia—even if mild—can exacerbate any neurologic injury that occurs during surgery, so it is critical to avoid overheating of the brain

during rewarming. Finally, physicians should continue temperature management into the postoperative period.

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