Direct Splanchnic Perfusion Safely Avoids Deep Hypothermia

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Deep hypothermia for the operative correction of congenital cardiac lesions protects hypoperfused organs, mostly because of its effect on lowering metabolic demand and oxygen requirement. Deleterious cerebral and extracranial side effects of deep hypothermia itself calls for a reexamination of the therapeutic value of hypothermia, and has led to the development of alternative perfusion strategies. Here we describe the potential advantages of milder hypothermia over deep hypothermia and our method of a practical and reproducible implementation of multisite perfusion under mild hypothermia (32°C).
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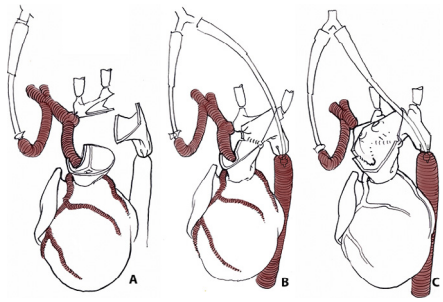
Introduction

The protective effects of hypothermia, recognized since the 1940s, make hypothermia a mainstay of management for patients with cardiac arrest, traumatic brain injury and operative procedures on the aorta and the heart.[1,2] The protective effects of hypothermia, presumed to be due to a temperature-dependent reduction in metabolic demand, allows recovery of tissues after periods of hypoperfusion, oxygen and metabolite deprivation. The therapeutic value of hypothermia is however tempered by important cerebral and extracranial side effects, especially of deep hypothermia (< 30°C), that include coagulopathy, seizures, adverse neurological outcomes and kidney dysfunction.[1,3] Various strategies of low flow regional perfusion during periods of deep hypothermia have been developed to extend the safe time for hypoperfusion to the most vulnerable organs and to mitigate some of the negative effects. Irrespective of perfusion strategy, cellular and vascular homeostasis is perturbed by hypothermia itself. Extracorporeal perfusion at deep hypothermic temperatures is more complicated and less predictable than perfusion at normothermia or mild hypothermia. Strategies that can safely avoid deep hypothermia are emerging.



Three region perfusion technique: with the descending aorta cross clamped, distal aortic arch reconstruction is begun while perfusing the coronary and cerebral circulations (1A). The cross clamp is removed and descending aortic perfusion continued through an 8-10 Fr cannula, with continued perfusion of the coronary and cerebral circulations (1B). Antegrade cardioplegia is administered for Damus-Kaye-Stansel and proximal arch reconstruction, with continued perfusion of the cerebral and splanchnic circulations (1C).

Central Message

Important cerebral and extracranial side effects may counter the protective effects of deep hypothermia. We describe the potential advantages of avoiding deep hypothermia using multisite perfusion.

Systemic and Regional Effects of Deep Hypothermia

Brain

Cerebral metabolism decreases by 6%-10% for every degree drop in temperature.[1] Gains in neurologic recovery after an ischemic insult are achieved with mild hypothermia (31-35°C), and the incremental added protection from deep hypothermia (18-25°C) may be countered by systemic complications, adverse effects of profound hypothermia on neuronal health, electrical activity and vascular autoregulation.[1,3-5] Regulating the flow rate on cardiopulmonary bypass to achieve appropriate oxygen delivery in a dysregulated vascular bed is complicated, and studies have questioned the adequacy of flow rates in patients undergoing antegrade cerebral perfusion (ACP) with hypothermia.[6-9]

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The loss of the brain's ability to maintain constant blood flow over varying perfusion pressures is associated with higher stroke rates in patients with deregulated cerebral circulation who undergo cardiopulmonary bypass.[10] The uncoupling of metabolic rate and flow at hypothermic temperatures may additionally result in tissue hyperoxia, contributing to oxygen radical production, and reperfusion injury.[10-12] Further, there is evidence that the elevation in cerebral resistance caused by hypothermia persists during rewarming, calling into question the effectiveness of cerebral blood flow even after hypothermia.[10]

Variability in collateral vasculature renders the distribution of flow from ACP to the extracranial circulation unpredictable.[6] Logically, for a patient with aortopulmonary collaterals and cerebral resistance elevated by hypothermia, ACP flow might suboptimally perfuse the brain, with flow instead distributing preferentially to the right arm and lungs, whereas a patient with poor collateralization to the lower compartment might experience hyperemic cerebral perfusion at the same ACP flow rate.

Complicating the measure of outcomes is evidence that, while DHCA has clear neurodevelopmental consequences, differences in neurodevelopmental outcomes between low flow bypass or ACP and DHCA narrow on late follow up testing. Cerebral protection by various combinations of ACP and hypothermia has been the subject of numerous and conflicting studies.[6,13-16] Recognition of the insult inflicted by circulatory arrest during surgery has prompted the development of numerous perfusion strategies that include low flow bypass with intermittent arrest, antegrade cerebral perfusion, and multi-site perfusion.

Coagulation

Hypothermia promotes coagulopathy, with platelet dysfunction and thrombocytopenia starting at temperatures of 35°C. Temperatures lower than 33°C can additionally alter the kinetics of the clotting cascade and synthesis of multiple coagulation factors.[1] PT and PTT have also been shown to increase with the degree of hypothermia, further highlighting the wide-ranging effects of hypothermia on the entirety of the coagulation cascade.[17]

Inflammation and Infection

Hypothermia inhibits some pro-inflammatory cytokines.[1] While the anti-inflammatory effect of hypothermia has some advantages in organ preservation, it may also lead to an increased risk of infection. At temperatures less than 32°C, circulating WBC count is lowered and macrophage and neutrophil function is impaired. Hypothermia promotes microvascular vasoconstriction, impaired WBC function, and has been linked to surgical site infection.[1] Cardiopulmonary bypass with moderate hypothermia and an increased duration of bypass have also been associated with increased pro-inflammatory IL-8 levels and a decrease in the levels of the anti-inflammatory cytokine IL-10 and COX 2 expression.[18-20] Measuring the effects of hypothermia on inflammation is challenged by the ongoing systemic inflammatory response secondary to cardiopulmonary bypass, and further study is required.

Capillary Leak

Fluid extravasation and increased capillary permeability are known deleterious effects of hypothermia. Interstitial fluid shift secondary to a hypothermia-induced increase in capillary permeability affects soft tissues, especially the lungs, and contributes to the morbidity associated with cardiopulmonary bypass. Animal models show a direct relationship between the degree of hypothermia and increasing fluid extravasation rate, plasma volume, a greater need for fluid supplementation, a net positive fluid balance and higher levels of bradykinin during hypothermic cardiopulmonary bypass.[21-23] Clinical studies show that cardiopulmonary bypass at temperatures > 24°C result in less postoperative fluid accumulation as compared to deeper hypothermia.[24]

Kidney

During arch repair, ACP provides direct cerebral perfusion while splanchnic organ protection relies on hypothermia and variable collateral-dependent flow. In conjunction with a loss of vascular autoregulation at hypothermic temperatures, this results in uncertain protection of the splanchnic organs. Acute renal injury is a known complication of aortic procedures under CPB with rates of acute renal failure reported between 5%-14% and conversion to dialysis reported at 7%.[25,26] In addition, studies comparing the effect of DHCA with ACP have shown renal dysfunction more commonly associated with DHCA.[27]

Heart

The disruptive effects of hypothermia on myocardial contractility, elastance, ion channel hemostasis and the propagation of arrhythmias are described.[1,28] There is evidence of higher postoperative troponin leak in adult patients undergoing coronary bypass grafting under hypothermia compared with normothermia.[29] Infants undergoing the arterial switch operation at normothermic temperature show a more rapid normalization of troponin compared with those repaired with hypothermia.[18,24,30]

Regional Perfusion Strategies

Antegrade cerebral perfusion (ACP), used in conjunction with deep hypothermia, has gained widespread adoption for neuroprotection during aortic arch reconstruction in neonates, though its superiority over deep hypothermic circulatory arrest (DHCA) remains controversial.[6,13] Antegrade cerebral perfusion, even if effective in protecting the brain, usually still obligates hypothermia to protect vulnerable extracranial organs, notably the kidneys.

Perioperative acute kidney injury, gut malperfusion and liver dysfunction are important postoperative complications of neonatal heart surgery, and have prompted questions about strategies that best support the recovery of extracranial organs.

While there are many studies of ACP, few have examined direct splanchnic perfusion (DSP) during aortic arch surgery. Described methods include separate cannulation sites at the descending aorta or femoral artery to provide direct circulatory support for the splanchnic circulation.[31-33] Kidney function, an important determinant of overall recovery and length of