Use of deep hypothermic circulatory arrest following ventricular laceration: A case report

Ken Spain, CRNA, MSEd
Hutchinson, Kansas

Deep hypothermic circulatory arrest (DHCA) is usually a planned approach to certain types of cardiovascular surgery. This case report is an example of DHCA used to provide cerebral protection for a patient whose right ventricle was lacerated by an oscillating saw during sternotomy. The surgical case was a redo coronary artery bypass graft surgery. Anesthetic intervention with specific drug administration and steps taken for neuroprotection are discussed.

Before DHCA, anesthetic interventions are used to reduce brain metabolism and, thus, oxygen consumption for the 30- to 60-minute window of complete circulatory arrest.

Although not having an electroencephalographic monitor for the case, an interesting finding was that the bispectral monitor (or BIS monitor) was at the zero level for the entire circulatory arrest and about 30 minutes after rewarming.

Key words: **Cerebral protection, deep hypothermic circulatory arrest, ventricular laceration.**

Deep hypothermic circulatory arrest (DHCA) is used most frequently in pediatric cardiac surgery[1] and during resection of thoracic aneurysms in adults. Most often, DHCA is used for cases that necessitate stopping perfusion to the brain for the surgeon to complete the surgical procedure. Deep hypothermic circulatory arrest offers brain protection for a period of about 30 to 60 minutes and is usually a planned surgical anesthetic event.[2-4] Deep hypothermia reduces tissue metabolism and the cerebral rate of oxygen consumption and is mandatory for total body arrest.[5,6] By establishing conditions that make the patient "clinically dead," the surgeon is given valuable time to perform procedures that would be impossible during normothermic circulation.

Most protocols involving DHCA involve the following[7,8]: (1) administration of barbiturates, usually thiopental or longer acting barbiturates such as secobarbital, for the reduction of cerebral metabolism; (2) mannitol to help reduce potential increased intracranial pressure and to reduce free radicals; (3) steroids to promote cell membrane integrity and reduce brain swelling; and (4) deep hypothermia via the extracorporeal membrane oxygenator, with ice packing to the head. The anesthetic and surgical plan involves mutual goals of tissue and organ protection by decreasing cellular metabolism and substrate delivery during the absence of perfusion.

This case report represents the emergency and unplanned use of DHCA to repair a large laceration in the right ventricle of a patient who likely would have sustained grave sequelae without rapid intervention and the use of the DHCA surgical technique and anesthetic protocols. The size of the laceration, 10 cm, mandated DHCA because opening the sternum and attempting to rapidly suture such a large laceration likely would have resulted in the insufflation of large quantities of air.

Case summary

A 72-year-old man was admitted for redo coronary artery bypass surgery, 25 years after the first operation. Recently, the patient had experienced congestive heart failure, and, preoperatively, a chest radiograph revealed perihilar congestion, edema, and vascular congestion. This patient also had previous findings of chronic atrial fibrillation with ventricular pacing, hypertension, type 2 diabetes mellitus, obesity, and recurrent coronary artery disease with angina. Cardiac catheterization data documented a 50% ejection fraction, 80% stenosis of the left anterior descending artery, a totally occluded right coronary artery, and obtuse marginal artery. Duplex studies revealed 40% stenosis of both carotid arteries.

While in the preanesthesia care unit, the patient was sedated with 4 mg of midazolam, and a right internal jugular introducer and a right subclavian central venous catheter were inserted. A Swan-Ganz catheter was inserted through the introducer and guided 55 cm with a good pulmonary artery wave form (28/22 mm Hg) and wedge pressure (19 mm Hg). A right radial artery catheter also was inserted.

Next, the patient was transported to the operative suite and prepared for induction and preoxygenated with 100% oxygen for about 4 minutes before induc-

tion. The patient was induced with 20 µg of sufentanil, 20 mg of etomidate, and 60 mg of rocuronium. After intubation and securing the endotracheal tube, the anesthetic was maintained with 1.5% to 3% sevoflurane with a rocuronium infusion of 4 µg/kg per minute.

Before sternotomy, the operative course was uneventful. At the point of sternotomy, 2 U of packed red blood cells were readied at the anesthesia cart, which is a standard procedure for all redo sternotomies at this institution. On attempting the sternotomy with the oscillating saw, the surgeon realized, due to the excessive volume of dark blood exiting the opening, that the ventricle had been lacerated by the saw. One of the packed cell units was infused rapidly to maintain volume status as the hemorrhage continued.

Rather than continue with sternotomy and cause a grave insufflation of ambient air, the surgeon clamped the sternum and began to cannulate the femoral artery and vein for bypass surgery. During cannulation, the surgeon asked whether the patient could be prepared for DHCA to avoid massive air emboli caused by opening the chest for repair of the beating heart.

Preparation for DHCA was instituted immediately and the following drugs were given intravenously: 1 g of thiopental, 5 mg of midazolam, and 10 mg of dexamethasone. Mannitol, 12.5 g, was added to the pump prime, and the patient's head was packed in ice. Five minutes were allowed to elapse to circulate the medications at an average pulsatile pressure of 90 mm Hg and to ensure perfusion of the vessel-rich organs: brain, liver, and kidneys. The patient then was placed immediately on bypass, and cardioplegic drugs were infused to arrest the heart. As soon as asystole was established, the pump was turned off. The bispectral index reading went from the low 40s to zero after protocols were initiated, indicating the efficacy of the drugs used.

As soon as total circulatory arrest was established, the chest was completely opened, revealing a laceration through most of the front wall of the right ventricle, about 10 cm long. From the size of the laceration it was evident that opening the chest during normal pumping of the heart would have been disastrous to the patient. The surgeon repaired the wound, and the perfusion pump was turned on for normal bypass perfusion. The total time of nonperfusion or DHCA was 22 minutes at a core body temperature of 25°C. The patient was slowly warmed to 36°C, and the operation was uneventful from that point on. Extubation occurred several hours later, and the patient had no observed sequelae: he exhibited preoperative levels of cognition according to the patient, family, family physician, and surgeon; no motor dysfunction or paresthesias were elicited. Deep hypothermic circulatory arrest has been associated with early postoperative sequelae, but the patient did not exhibit any defects despite 40% bilateral carotid stenosis.[9]

Discussion

This case report represents a combined effort involving communication and timing of anesthetic interventions in response to a potentially grave complication. The surgeon and the anesthetist had to time their interventions in a precise window of time to protect the patient. Barbiturates had to be administered about 5 minutes before circulatory arrest to ensure arrival to the vessel-rich groups, especially the brain, and to maximize protection. Redistribution could have diluted barbiturate levels if too much time elapsed before total arrest.

The fact that the patient survived without any observed complications was probably due to the combined protective effects of the following: (1) surgeon response, skill, and the ability to adapt to the situation; (2) proper timing as mentioned; (3) packing the patient's head in ice; and (4) the administration of pharmacological agents (thiopental, midazolam, sufentanil, dexamethasone, and mannitol). Barbiturates, sufentanil, and midazolam have been shown to reduce the cerebral rate of oxygen consumption, protecting the brain during total lack of perfusion.[10,11] Steroids reduce inflammatory response and promote cell membrane integrity and mannitol decreases intracranial pressure and free radicals, and they were used for brain protection in this circumstance.[12]

Finally, all of the aforementioned techniques and protocols were thought to be instrumental in the patient surviving intact and at his preoperative level of functioning. This case represents a situation validating how training, experience, and education are important in responding to situations that we as anesthetists do not face on a day-to-day basis. Nurse anesthetists need to react and adapt to the many vicissitudes of any given anesthetic situation. This case report also validates that we as nurse anesthetists can be instrumental in the survival of our patients outside of tertiary centers.

REFERENCES

1. Bellinger DC, Jonas RA, Rappaport LA, et al. Developmental and neurologic status of children after heart surgery with hypothermic circulatory arrest or low-flow cardiopulmonary bypass. *N Engl J Med.* 1995;332:549-555.
2. Swain JA, McDonald TJ Jr, Griffith PK, Balaban RS, Clark RE, Ceckler T. Low-flow hypothermic cardiopulmonary bypass protects the brain. *J Thorac Cardiovasc Surg.* 1991;102:76-84.
3. Tinker JH. Cardiopulmonary bypass: technical aspects. In: Thomas SJ, ed. *Manual of Cardiac Anesthesia.* New York, NY: Churchill Livingstone; 1984:380.
4. Kwitka G, Roseberg J, Nugent M. Thoracic aortic disease. In:

Kaplan J, ed. *Cardiac Anesthesia.* Philadelphia, Pa: WB Saunders Co; 1993:763.

5. Fox L, Blackstone E, Kirklin J, et al. Relationship of brain blood flow and oxygen consumption to perfusion flow rate during profoundly hypothermic cardiopulmonary bypass. *J Thorac Cardiovasc Surg.* 1984;83:239-248.
6. Greeley WJ, Bracey VA, Ungerleider RM, et al. Recovery of cerebral metabolism and mitochondrial oxidation state is delayed after hypothermic circulatory arrest. *Circulation.* 1991;84(5 suppl): III400-III406.
7. Nemoto EM, Klementavicius R, Melick JA, et al. Suppression of cerebral metabolic rater for oxygen (CMRO2) by mild hypothermia compared with thiopental. *J Neurosurg Anesthesiol.* 1996;8:52-59.
8. Michenfelder JD, Milde JH, Sundt TM. Cerebral protection by barbiturate anesthesia: use after middle cerebral artery occlusion in Java monkeys. *Arch Neurol.* 1976;33:345-350.
9. Newburger, Jonas RA, Wernovsky G, et al. A comparison of the perioperative neurologic effects of hypothermic circulatory arrest versus low-flow cardiopulmonary bypass in infant heart surgery. *N Engl J Med.* 1993;329:1057-1064.
10. Baughman VL, Hoffman WE, Albrecht RF, et al. Cerebral vascular and metabolic effects of fentanyl and midazolam in young and aged rats. *Anesthesiology.* 1987;67:314-319.
11. Nugent M, Artru A, Michenfelder JD. Cerebral metabolic, vascular and protective effects of midazolam maleate. *Anesthesiology.* 1982; 56:172-176.
12. Banoub M. Anesthesia for thoracic aortic surgery. In: Kaplan J, ed. *Cardiac Anesthesia: Principles & Clinical Practice.* 2nd ed. Philadelphia, Pa: Lippincott Williams & Wilkins; 2001:806.

AUTHOR

Ken Spain, CRNA, MSEd, is a self-employed, independent practitioner at Hutchinson Hospital, Hutchinson, Kan.