Technique

Combined Hypothermic Circulatory Arrest and Warm Blood Cardioplegia for Aortic Surgery

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ABSTRACT

This is a case of an 87-year-old male who underwent successful repair of an acute ascending aortic dissection (Stanford Type A). Hypothermic circulatory arrest was used to provide cerebral protection during the performance of the "open" distal anastomosis. Continuous normothermic retrograde blood cardioplegia was used as a means for myocardial protection. To our knowledge, this is the first report of combining warm (aerobic) cardiac preservation with hypothermic circulatory arrest. We describe the perfusion management and discuss the rationale for the use of normothermic blood cardioplegia. We advocate the use of warm aerobic myocardial arrest for all procedures requiring myocardial protection regardless of systemic temperature.

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INTRODUCTION

Ascending, transverse and some descending aortic aneurysms and dissections frequently require arrest of the circulation to adequately repair or replace diseased segments. This provides excellent exposure, can avoid clamping of a diseased aorta and in general, greatly facilitates aortic surgery. Deep hypothermia was developed as a means of cerebral protection during circulatory arrest. (1) Cerebral protection, along with other organ preservation, has been shown to be excellent (within time constraints) with this technique. (2)

In the past, hypothermia, with or without cardioplegic infusions, has been the mainstay of myocardial protection. Normothermic continuous retrograde blood cardioplegia has recently been added in an attempt to improve myocardial protection during cardiac procedures. This case report describes the use of warm aerobic myocardial arrest combined with systemic hypothermic circulatory arrest.

Table 1
Components of Crystalloid Cardioplegia (mixed 1:4 with blood)

High Potassium		Low Potassium	
Dextrose 50%	30ml	Dextrose 50%	30 ml
KCl	128 mEq	KCl	25 mEq
$NaHCO_3$ 7.5%	75 ml	$NaHCO_3$ 7.5%	75ml
Plasma-Lyte A	1000 ml	Plasma-Lyte A	1000 ml

CASE REPORT

An 87-year-old male initially presented to another hospital with a 5 hour history of chest and interscapular pain. In the emergency room, echocardiography revealed cardiac tamponade during which time he suffered cardiopulmonary arrest. He was successfully intubated, resuscitated, and pericardiocentesis was done. He was stabilized and underwent computerized tomography scan which revealed dissection of the ascending aorta, and was transferred to our facility. He was emergently operated and underwent repair (resection and replacement with 24 mm Hemashield dacron graft[a]) of a Stanford A dissection. Findings at surgery included a 200-250 ml hemopericardium and the presence of a 3 cm transverse intimal disruption on the posterior medial aspect of the distal ascending aorta at the level of the takeoff of the innominate artery. There was no evidence of retrograde dissection or aortic valve pathology. Under conditions of hypothermic circulatory arrest the intimal disruption was excluded and false lumen obliterated or "sandwiched" between 2 teflon felt strips, before prosthetic graft replacement.

Details of perfusion and cardioplegia delivery are now described. Venous drainage was established using #32 and #34 French caval cannulae. Arterial return was established by cannulation of the right common femoral artery with a #22F cannula. A membrane oxygenator[b] with an integrated reservoir was used. An accessory recirculating (shunt) line extending from the arterial line to the reservoir was attached. There are 2 "Y-connectors" also attached to the shunt line: the first, to supply the blood component of the cardioplegia set-up; the second, to supply blood to a hemoconcentrator[c].

A commercially available blood[d] cardioplegia set was used. The setup is designed to automatically deliver a 4:1 ratio of blood to crystalloid cardioplegia solution. Blood is drawn from the recirculating (shunt) line by means of a roller head pump[e], mixed at the pump head, delivered through a heat exchanger, through a vented pressure and temperature monitor and finally delivered to the coronary sinus via a retrograde cannula[f].

Cardiac asystole was induced with 1000 ml of high potassium (28 mEq/L final delivery), warm 37°C blood cardioplegia via the coronary sinus. This was maintained with low potassium (8 mEq/L after 4:1 dilution), warm blood cardioplegia via the coronary sinus, throughout the remainder of the aortic cross clamp and circulatory arrest period.

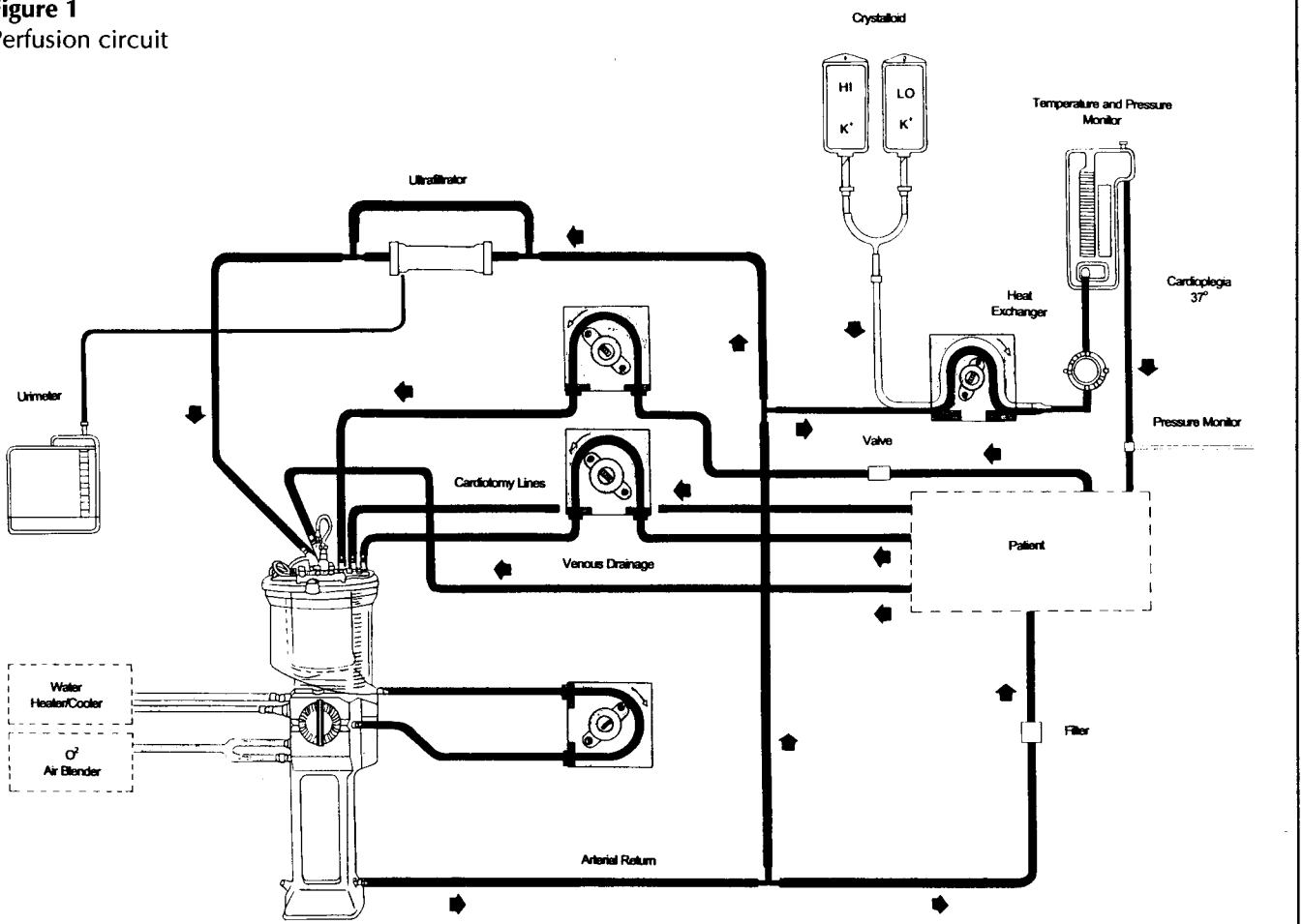
During periods of total circulatory arrest the heart was perfused with warm blood cardioplegia at a temperature of 37°C. To accomplish this, the arterial pump must be delivering at a rate slightly above the rate of the cardioplegia pump (in order to avoid cavitation and/or outgassing at the membrane oxygenator). The arterial line must be clamped on the patient's side of the arterial filter to freely recirculate and observe any changes in resistance in the recirculating circuit. Ventilating gas is maintained to the membrane oxygenator throughout this period.

In order to perfuse the heart with warm blood during periods of total circulatory arrest, this blood must be returned to the reservoir from the heart. This was accomplished with the use of a left ventricular vent, inserted via the right superior pulmonary vein. The perfusion circuit is shown in Figure 1. The pump prime consisted of Plasma-Lyte A[g] 2000 ml with 89.20 mEq $NaHCO_3$, 12.5 g mannitol, and 1 g methylprednisolone. Heparin and protamine were administered in the following fashion: 400 u/kg heparin to maintain ACT > 480 and 1 mg of protamine to 100 units of heparin. Components of crystalloid cardioplegia are shown in Table 1. Blood cardioplegia was given at a rate of 100-150 ml/min with a coronary sinus pressure of less than 40 mmHg.

Additional cerebral protection techniques included ice bags to the head and thiopental, 30 mg/kg bolus. The patient was weaned off cardiopulmonary bypass and left the operating room

a Meadox Medicals, Oakland, NJ
b Cobe CMS30-HVRF, Cobe Laboratories Inc., Lakewood, CO 80215
c Cobe Model 1200, Cobe Laboratories Inc., Lakewood, CO 80215
d Sarns Conducer MP4, 3M Healthcare, Ann Arbor, MI
e Cobe Laboratories Inc. Lakewood, CO 80215
f Gundry 94115 NCSP Retroplegia, DLP Inc., Grand Rapids, MI 49501
g Baxter Healthcare Corporation, Deerfield, IL 60015

Figure 1
Perfusion circuit



without requiring inotropic support. The cardiopulmonary bypass (maintained at 2.5 L/min/m^2 with a mean arterial pressure 70 mmHg) time was 130 minutes; circulatory arrest time was 28 minutes at 17°C nasopharyngeal temperature and the aortic cross clamp time was 69 minutes.

The postoperative course was smooth other than a short episode of atrial fibrillation/flutter and the patient was discharged to home on the ninth postoperative day. Final pathology revealed fragments of fibro-atheromatous plaque and foci of intramural hemorrhage of the aortic wall.

DISCUSSION

During operations using hypothermic circulatory arrest, most concerns have been about cerebral protection rather than myocardial protection. (3) Although the brain is more sensitive to anoxia than any other organ, we feel the method of cerebroprotection should not influence (or be influenced by) the method of myocardial preservation. Abundant literature has been published concerning myocardial protection with normothermic vs. hypothermic cardioplegia. (4-10)

Gott, et al, were the first to report retrograde perfusion of the coronary sinus for direct vision-open aortic valve surgery. (11-12) Salerno, et al, reported the first large clinical series of patients receiving continuous warm blood cardioplegia through the coronary sinus (13). In his group of high risk patients (over half required urgent revascularization) excellent results (only 3% mortality) were achieved. We have been extremely impressed with the results of warm aerobic arrest and have been using this method exclusively for myocardial protection in all cases. Since adopting this routine almost one year ago we have had only two patients with post-operative low cardiac output syndrome. Lichtenstein, et al, reported a significantly lower incidence of peri-operative myocardial infarction, need for intra-aortic balloon pump, with cardiac output higher than pre-operation, and almost all (99.2%) hearts spontaneously reverting to normal sinus rhythm with normothermic cardiac arrest (14). These authors contend the difference between oxygen requirements of a hypothermic arrested heart and a normothermic arrested heart is only 5%. However, oxygen delivery (due to a leftward shift of

the oxy-hemoglobin curve) is dramatically reduced during the hypothermia, possibly stimulating anaerobic metabolism.

McGovern, et al, compared blood cardioplegia infusions at 20°C, 10°C and 4°C. They found that there was, compared to 20°C, no benefit of 10°C infusions due to a dramatic decrease in oxygen delivery, as evidenced by decreased myocardial oxygen tensions in this group (15). In fact, further decrease to 4°C was actually deleterious as left ventricle recovery was decreased, left ventricular end diastolic pressure higher, myocardial edema was increased and electron microscopy showed increased myocardial structural damage at this infusion temperature. The concept of normothermic aerobic versus hypothermic anaerobic arrest with blood cardioplegia was further elucidated by Menasché, et al (16). These authors showed that normothermic blood cardioplegia keeps the heart in an aerobic state by measuring markers of anaerobic metabolism (myocardial blood gases, lactates) as well as leukocyte activation (elastase) and lipid perioxidation (malondaldehyde, Vitamin E). Yau, et al, found that normothermic blood cardioplegia allows for generation of adenosine triphosphate and increased myocardial oxygen consumption; while hypothermic cardioplegia caused a great increase in adenosine di- and mono-phosphate (17). They also found the optimal combination was a flow rate greater than 80ml/min and hemoglobin concentrations greater than 8 gm Hb/100ml, as evidenced by improved myocardial performance post-operatively. We used a flow of 100 - 150 ml/min as a safety factor and employ diuretics, hemoconcentration and measure frequent potassium levels as a means to deal with the excess potassium and crystalloid load.

Theoretical concerns about right ventricular protection with warm retrograde blood cardioplegia have not been borne out in clinical practice. Indeed, warm heart surgery has been shown to give excellent myocardial protection in spite of long cross clamp times, pulmonary hypertension and left and/or right ventricular hypertrophy. The efficacy of warm blood cardioplegia, even in these high risk situations, was independent of any delivery of hypothermia (18).

We do feel however, as do others, (19) that the protection afforded by warm blood cardioplegia is compromised when it is interrupted for any length of time. This is usually not necessary for aortic surgery and valvular procedures as coronary flow does not obstruct exposure. Coronary blood flow may hinder exposure in coronary revascularization procedures, however, it can be minimized by using various occluders, probes and vessel tapes. We have not found it necessary to interrupt the cardioplegia using these techniques for coronary procedures.

It is not the intent of this paper to compare different types of cerebral protection for aortic repairs and replacements. However, certain points deserve mentioning. First, the safe period of ischemic tolerance of the central nervous system is frequently debated, but is generally considered to be three to five minutes at normothermia. For each additional 5° C drop in temperature it approximately doubles, i.e., 10 min at 32°C, 20 min at 27°C and 40 min at 22°C (20). Although great care must be used when reinstituting flow because of the threat of air embolism, we feel hypothermic circulatory arrest is much safer than selective cerebral perfusion. This later technique involves additional dissection, cannulation and perfusion techniques. These procedures can be time consuming and have their own complications which we feel are more dangerous than those associated with hypothermic arrest, provided the previously mentioned time constraints and care to prevent embolism are taken.

Recently, Yasuura, et al, described a technique of retrograde (cold blood) cerebroplegia via the superior vena cava as an alternative to hypothermic circulatory arrest for cerebral protection during aortic surgery (21). They reported excellent results (no neurologic sequelae) with a mean "systemic" circulatory arrest time of 82 minutes (range 32 to 110 minutes). We have had similar excellent results (re: neurologic sequelae) in the past with hypothermic circulatory arrest, probably due to the fact we have rarely found it necessary to prolong circulatory arrest time greater than 45 minutes and usually keep it less than 30 minutes. Davis, et al (22), found the incidence of neurologic complications to be independent of circulatory arrest time as long as this time is kept under 30 minutes. Incidentally, this same cannulation and warm cardioplegia system could be easily adapted to provide retrograde (continuous or intermittent) hypothermic cerebral perfusion if the surgeon anticipates, or needs, prolonged circulatory arrest, such as with total arch replacements.

We have, however, occasionally encountered low cardiac output states, as evidenced by hemodynamic instability requiring inotropic support postoperatively using cold cardioplegic techniques during aortic surgery. This prompted the transition to warm continuous cardioplegia, in an attempt to improve myocardial protection.

The heart, like any muscle, may be chemically arrested by potassium infusion, which results in a 90% reduction of oxygen requirements at normothermia. (18) There is no analogous means to lower the metabolism of the brain to such a profound extent at normothermia, although thiopental, ketamine and phenytoin are often used as adjunctive protection. For this reason we, and others, feel hypothermic circulatory arrest is the safest method of cerebral protection and best facilitates performance of an "open anastomosis" during aortic surgery.

SUMMARY

This case supports the contention that normothermic blood cardioplegia can be safely combined with hypothermic circulatory arrest. Normothermic aerobic arrest has been shown to give excellent results even in high-risk patients. The great majority of reduction of oxygen demands by the myocardium is obtained by electro-mechanical arrest; hypothermia adds little more. When continuous perfusion with oxygenated blood is added to prevent anoxia, an aerobic state may be maintained with normothermic conditions. Controversy exists concerning whether or not decreasing the temperature of blood cardioplegia increases its efficacy, although historically the synergy of hypothermia and hyperkalemic arrest is well established. The critical question,

whether continuous normothermic blood cardioplegia is superior to hypothermic cardioplegia arrest, will require large randomized clinical trials. The perfusion circuit described is simple to use, allows hypothermic circulatory arrest during normothermic retrograde cardioplegia, and is easily adaptable to retrograde hypothermic caval cerebroplegia delivery if prolonged circulatory arrest time is expected or encountered. Although it is difficult or nearly impossible to perform randomized clinical trials in this small group of patients, this technique may offer promise to these challenging patients in the future, particularly those with compromised myocardial function preoperatively.

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