Anaesthesia for aortic arch aneurysm repair: experience with 17 patients

Pierre A. Casthely MD, Phillip N. Fyman MD,
Lawrence M. Abrams MD, Randall B. Griep MD,
M. Arisan Ergin MD

Mortality and morbidity during aortic arch aneurysm repair is high despite improvements in surgical technique which attempt to assure brain protection during surgery. We successfully managed 17 patients using deep hypothermia and circulatory arrest. Anaesthesia consisted of pancuronium, fentanyl, plus isoflurane or halothane if needed. Pulmonary artery and arterial catheters were inserted. Surface cooling was performed followed by core cooling on cardiopulmonary bypass, using a heat exchanger. Total circulatory arrest was performed when esophageal temperature reached 12–14 °C after previous administration of thiopentone 30 mg·kg⁻¹, methylprednisolone 2 gm, furosemide 40 mg and mannitol 25 gm. At that time the head was packed in ice and surgical correction performed. Mean arrest time was 36.5 ± 13 minutes at a mean oesophageal temperature of 12.5 ± 0.75° C. No serious, permanent neurological deficit was found. Tracheostomy was required in five patients of whom two had chronic obstructive pulmonary disease (COPD). Two of these patients died of adult respiratory distress syndrome (ARDS) and renal failure. The reported technique is safe and can be easily used in patients undergoing aortic arch aneurysm repair.

Key words

SURGERY: aortic arch aneurysm repair; ANAESTHETIC TECHNIQUE: hypothermia, circulatory arrest.

Surgical repair of aortic arch aneurysms presents a challenge for the anaesthetist and the surgeon because of complex technical and physiological problems.^{1,2} Despite an improvement in the surgical technique this procedure is not frequently attempted because of the associated high mortality ranging from 10 to 25 per cent. Profound hypothermia is a technique which has long been used during surgical repair of congenital malformation in infancy.³ We report the successful management of 17 patients who underwent complete aortic arch repair during profound hypothermia and circulatory arrest.

Methods

We report our experience with 17 patients, ages 25–79 years, from 1980 to 1983. Thirteen were male, four female. The operation was performed as an emergency in three patients, two of whom had acute dissection unresponsive to conservative medical management and one who had suffered a gunshot wound to the chest. Premedication consisted of morphine 0.1 mg·kg⁻¹ and atropine 0.4 mg given one hour prior to surgery. A #18 gauge canula was inserted into a radial artery and a #7 French pulmonary artery catheter was inserted into the pulmonary artery via the right internal jugular vein, prior to induction of general anaesthesia. Anaesthesia was slowly induced with fentanyl 30 µg·kg⁻¹, diazepam, oxygen and pancuronium after which tracheal intubation was performed. Lidocaine 1.5 mg·kg⁻¹ was administered intravenously prior to tracheal intubation. Maintenance of anaesthesia consisted of fentanyl, pancuronium and a low concentration of an inhalational agent (halothane in nine, isoflurane in eight patients). Sodium nitroprusside was used as needed to control blood pressure. Propranolol was given concomitantly with sodium nitroprusside in three patients under-

From the Departments of Anesthesiology and Cardiovascular Surgery, State University Hospital, Downstate Medical Center, New York.

Address correspondence to: Dr. Pierre A. Casthely, Department of Anesthesiology, State University Hospital, Downstate Medical Center, 450 Clarkson Avenue, Box 6, Brooklyn, New York 11203.

going emergency repair in an attempt to decrease myocardial contractility, thereby preventing an increase of shear forces in the false lumen. Rectal and oesophageal temperatures and urinary output were monitored. A Beckman digital analyzer was used to monitor end tidal CO_2 .

Profound hypothermia was achieved by surface cooling and core cooling. Surface cooling was induced by placing the patient on a cooling blanket unit set at 27° F. The entire body including the head and neck was packed with small plastic bags filled with ice chips. The genital organs, the nose, ears, eyes, toes and fingers were protected with gauze pads, to avoid thermal injuries. Haemodilution to facilitate cooling was not performed in any patient. Arterial blood gases were measured every 15 minutes during surface cooling. When the oesophageal temperature reached 30° C the ice was removed, the patient draped, and a median sternotomy performed. The incision was extended superiorly along the medial border of the left sternomastoid muscle to assure good visualization of the carotid vessels. The pericardium was opened and cannulation of the superior and inferior vena cava was carried out via the right atrium. Simultaneously the left common femoral artery was exposed and a #20 arterial perfusion catheter inserted. All the patients were exsanguinated just prior to circulatory arrest and the blood was used for volume replacement after reversing the heparin with protamine.

Cardiopulmonary bypass with cooling was begun at a flow rate averaging 3 to 3.5 $\text{L} \cdot \text{min}^{-1}$ using a disposable bubble through oxygenator and an auxiliary heat exchanger. Myocardial preservation was provided by perfusing the coronary artery with a cold prime solution averaging a temperature of 7–10° C in which 50 mEq of KCl per 1500 cc was added to the pump prime and continuous topical hypothermia. The cooling continued until an oesophageal temperature of 12–14° C was reached. At this point circulatory arrest was performed and the aortic arch vessels were individually clamped. The head was packed with ice during the period of circulatory arrest. Five minutes before circulatory arrest was induced, thiopentone 30 $\text{mg} \cdot \text{kg}^{-1}$, methylprednisolone 2 gm, furosemide 40 mg and mannitol 25 gms were administered. The entire arch or appropriate portion of it including the tear was then replaced during a single period of circulatory arrest.^{4,5} Following the replacement of the

arch, cardiopulmonary bypass was reinstated and utilized to warm the patient and resuscitate the heart. The patients were subsequently warmed to an oesophageal temperature of 37° C and a rectal temperature of 32.5° C and weaned off cardiopulmonary bypass. The average time of cardiopulmonary bypass was 135 ± 30 minutes. At the termination of cardiopulmonary bypass and protamine infusion the patients were given fresh frozen plasma concentrate and platelets. An intravenous dose of 1 gm of methylprednisolone was also given following discontinuation of cardiopulmonary bypass. Mannitol and furosemide were administered to increase urine output when needed. Following the procedure each patient was evaluated carefully for seizure activity, abnormal responses to verbal commands, as well as abnormalities in motor ability, speech, and behaviour.

Results

There was a progressive decrease of cardiac index during cooling. Cardiac index decreased from $2.85 \pm 0.51 \text{ L} \cdot \text{min}^{-1} \cdot \text{m}^{-2}$ at 35° C to 2.69 ± 0.55 at 33° C, 2.1 ± 0.4 at 30° C and 1.75 ± 0.35 at 28° C. This was accompanied by a progressive decrease in mean arterial pressure and an increase in systemic and pulmonary artery resistance (Table I). Heart rate decreased during cooling accompanied by a progressive widening of QRS complex which was also seen.

There was a progressive decrease in PCO_2 and PO_2 during cooling. Ph decreased from 7.54 ± 0.05 at 30° C to 7.35 ± 0.03 at 28° C (Table II).

The mean surface cooling time was 82.5 ± 7.5 minutes. Mean arrest time was 36.5 ± 13 minutes with a range of 40 to 60 minutes and mean oesophageal temperature during the period of circulatory arrest 12.5 ± 0.75 ° C. The average time of cardiopulmonary bypass was 135 ± 30 minutes. The average core cooling to complete circulatory arrest was 27.5 ± 0.5 minutes.

Bleeding following protamine administration usually responded to conservative management. Four patients required a conduit between the natural aortic sleeve covering the graft and the right atrium to treat bleeding from the aortic graft.

An average of seven units of FFP, 18 units platelets and 5.5 units of packed cells were transfused in the operating room after the protamine administration. Five patients had four units of

TABLE I Haemodynamic changes during hypothermia

	35° C	33° C	30° C	28° C
CI L·min ⁻¹ ·m ⁻²	2.85 ± 0.51	2.69 ± 0.55	2.1 ± 0.4	1.75 ± 0.35
HR (beats/min)	98 ± 12.5	80 ± 11.5	70 ± 10	60 ± 7.5
MAP (mmHg)	98 ± 9.5	87.5 ± 8	75.5 ± 7.5	70 ± 6.5
PAP (mmHg)	12 ± 1.5	14 ± 2	16.5 ± 4	19 ± 5
SVR dynes·sec·cm ⁻⁵	1639 ± 99.5	1712 ± 102	1850 ± 97.5	2057 ± 112.5
PVR dynes·sec·cm ⁻⁵	118.5 ± 11.5	170.2 ± 15.5	312.9 ± 20.1	457 ± 25

proplex (factors 2, 7, 9, 10) in the operating room. Only one patient was reoperated for bleeding.

No ventricular fibrillation occurred during surface cooling. Five patients developed atrial fibrillation during cannulation of the right atrium for which three required cardioversion and three developed nodal bradycardia unresponsive to atropine following the opening of the pericardium.

Postoperative ventricular arrhythmias (premature ventricular contractions and bigeminy) which responded to infusion of lidocaine occurred in two patients.

Prolonged intubation requiring tracheostomy was seen in five patients. Two patients who with chronic obstructive pulmonary disease (COPD) died of adult respiratory distress syndrome (ARDS) and renal failure. Both were dialyzed unsuccessfully in the postoperative period and had elevated BUN and creatinine prior to surgery. Surgery was performed on both patients as an emergency. Incentive spirometry was used on all the extubated patients in the postoperative period. Bronchodilators were used in those patients requiring prolonged intubation.

Three patients had psychotic behaviour for three days in the postoperative period which responded to conservative therapy. One had a seizure starting 12 hours after surgery which responded to dilantin and phenobarbitone and a focal neurological deficit which resolved completely after one week. No intraoperative deaths occurred.

TABLE II Arterial blood gases during hypothermia

	PH	PCO ₂	PO ₂
35° C	7.54 ± 0.05	30.5 ± 3	361 ± 95
33° C	7.49 ± 0.04	28.0 ± 2.65	300 ± 85.70
30° C	7.39 ± 0.035	25.5 ± 2.70	298 ± 80.5
28° C	7.35 ± 0.03	25 ± 2.5	285 ± 75.5

Discussion

The incidence of neurological complications associated with surgery for aortic arch repair is low if the period of total circulatory arrest is of short duration and if the peripheral circulation is maintained during cooling and rewarming. However, transient impairment of cerebral function after hypothermic circulatory arrest has been reported.^{6,7} The incidence of neurologic disorders increases with the duration of total circulatory arrest. Intellectual development of children subjected to prolonged circulatory arrest during hypothermic open heart surgery in infancy was found to be the same as the normal population.⁸ However, Wright found more developmental abnormalities in infants treated under hypothermic arrest.⁹ Morphologic alterations of the brain in seemingly normal patients following combined hypothermic circulatory arrest as well as following standard extracorporeal circulation has been documented by computerized tomographic scan.³ The incidence of neurologic complications ranges from one to three per cent.¹ Neurologic damage results mainly from the inadequacy of perfusion during cooling and rewarming rather than total circulatory arrest.¹⁰ Another important factor in avoiding neurological complications is length of time for total circulatory arrest.¹⁰ Our mean circulatory arrest time was only 36.5 minutes.

Postoperative central nervous system damage may result from various causes: (1) metabolic derangement, (2) embolism of air or thrombosis, microaggregates, and (3) hypoxia as a result of slow blood flow or circulatory arrest.

Even though hypothermia decreases CMRO₂ (cerebral metabolic rate of oxygen consumption) it is difficult to predict a safe time limit for circulatory arrest in humans. Circulatory arrest is said to be well tolerated for four minutes at 38° C, eight minutes at 30° C, 16 minutes at 22° C, and more than 30 minutes at 16° C.^{11,12} Embolism caused by

air or microaggregate is a rare cause of neurologic dysfunction in those patients. Brain protection is the main concern during complete circulatory arrest. Many drugs are given prior to complete circulatory arrest to provide protection. Corticosteroids are used to reduce cerebral oedema by affecting CSF dynamics, decreasing membrane permeability, stabilizing the brain's blood volume and improving intracranial compliance.¹³ Furosemide is administered to decrease the brain volume by systemic diuresis, decreased CSF production, reduced astroglial cellular fluid transport and capacitance relaxation.¹⁴ The use of thiopentone (barbiturates) is controversial because of its mechanism of action in brain protection. Barbiturates acts only by reducing cellular electrical activity.¹⁵ Since the EEG is flat within 20 seconds of complete circulatory arrest, their value may be limited. We administered routinely $30 \text{ mg} \cdot \text{kg}^{-1}$ thiopentone to our patients during cardiopulmonary bypass prior to complete circulatory arrest. This dose has been shown to reduce cerebral oedema in patients with head trauma.¹⁶ Barbiturates and hypothermia can profoundly decrease the CMRO₂ and prevent brain damage in those patients. Barbiturates will decrease the functional activity, and hypothermia will preserve the cellular integrity of the brain cell.

Although barbiturates may not be completely useful there has been a search for other drugs that may be. Two drugs, lidocaine and phenytoin, have been the centre of many studies. Lidocaine appears to have an effect on CMRO₂ even if the EEG is already flat. Phenytoin and lidocaine will both slow the release of potassium from ischaemic neurons.^{17,18} However, the large doses required experimentally to produce this effect make their use unpopular since clinical relevance has not been proven. The protective value of etomidate and flunarizine during surgery have also been studied, although their usefulness during complete circulatory arrest has not been reported.^{19,20}

Recently there has been great interest in the role of calcium in ischaemic brain injury and the possibility of protective effects of various calcium channel blockers. Since calcium is well known to be involved in the contraction of vascular smooth muscle, it can produce the increase in cerebrovascular resistance which occurs after a period of complete ischaemia. Other adverse reactions which

occur in the brain during ischaemia may also be due to the effect of calcium.

Infusion of nimodipine, a calcium channel blocker, has been shown to improve cerebral blood flow in the post-ischaemic hyperfusion period and neurologic recovery after complete cerebral ischaemia in the dog.²¹

Other calcium entry blockers have been found to be effective cerebral vasodilators. Nifedipine, verapamil and diltiazem caused a dose-related relaxation of spasm induced in canine cerebral arteries by potassium or prostaglandin.²² However, the doses required for that protective effect may produce complete heart block if used in patients undergoing aortic arch aneurysm resection under profound hypothermia and circulatory arrest, thereby compromising the cardiovascular system in the post-bypass period.

Because of decreased metabolism during profound hypothermia, carbon dioxide production decreases. It has been recommended that CO₂ be added to the inspiratory mixture to increase the PCO₂ and therefore correct the shift to the left of the hypothermia-induced oxygen haemoglobin dissociation curve. However, it is well known that hypercarbia increases the risk of arrhythmias.²³ We did not administer carbon dioxide to our patients during cooling or change minute ventilation.

The traditional presumption has been that the optimal value of blood pH at a body temperature of 37° C is also the optimal pH at a lower body temperature. More recently, however, it has been noted that the proper reference value may be electrochemical neutrality, where pH equals pOH.²⁴ With cooling the pH of the neutral point rises, with warming it falls.

We managed our hypothermic patients using uncorrected values of pH rather than correcting the pH for the temperature of 37° C. The concept of metabolic and respiratory acidosis and alkalosis are appropriate but uncorrected values for pH and PCO₂ should be used.²⁵ We did not correct acid base imbalance during surface hypothermia unless severe progressive acidosis developed.

Pulmonary complications occurred in 11 patients. The most common was atelectasis which responded to nasotracheal suctioning, incentive spirometry and aerosol administration. Five patients required tracheostomy for long-term venti-

latory support. Two of them with a history of COPD died from the adult respiratory disease syndrome (ARDS) and renal failure. One of these patients had previous radiation in the thorax for a malignancy and the other was a drug addict with elevated BUN and creatinine, prior to surgery. Both were dialyzed postoperatively and had the surgery performed as an emergency. We could not attribute those pulmonary complications to an increase in pulmonary artery (PA) pressure during CPB since a low PA pressure was always maintained in all of our patients. Other factors which may account for the high incidence of pulmonary complications include CHF and previous pulmonary disease.

We conclude that profound hypothermia with circulatory arrest is a safe and effective technique for repair of aortic arch aneurysms. In our experience the incidence of intraoperative and postoperative morbidity and mortality was much lower than that previously reported.

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References

- 1 Bailey LL, Takeuchi Y, Williams WG, Trusler GA, Mustard WT. Surgical management of congenital cardiovascular anomalies with the use of profound hypothermia and circulatory arrest. Analysis of 180 consecutive cases. *J Thorac Cardiovasc Surg* 1976; 71: 485-9.
- 2 Ergin MA, O'Connor J, Guinto R, Griep R. Experience with hypothermia and circulatory arrest in the treatment of aneurysms of the aortic arch. *J Thorac Cardiovasc Surg* 1982; 84: 649-55.
- 3 Muraoka R, Shirotani H. Open heart surgery in infants and small children utilizing profound hypothermia and limited cardiopulmonary bypass. *J Jap Surg Soc* 1977; 78: 1009-13.
- 4 Ergin MA, Griep RB. Progress in treatment of aneurysms of aortic arch. *World J Surg* 1980; 4: 535-43.
- 5 Griep RB, Stinson EB, Hollingsworth JF, Buehler D. Prosthetic replacement of the aortic arch. *J Thorac Cardiovasc Surg* 1975; 70: 1051-6.
- 6 Subramanian S, Wagner H, Vlad P, Lambert E. Surface induced deep hypothermia in cardiac surgery. *J Pediatr Surg* 1971; 6: 612-9.
- 7 Subramanian S, Wagner. Correction of transposition of the great vessels in infants under surface-induced deep hypothermia. *Ann Thorac Surg* 1973; 16: 391-7.
- 8 Stevenson JG, Stone EF, Dillard DH, Morgan BC. Intellectual development of children subjected to prolonged circulatory arrest during hypothermic open heart surgery in infancy. *Circulation* 1974; 49: 11-54.
- 9 Wright JS, Hicks RG, Newman DC. Deep hypothermic arrest: Observations on later development in children. *J Thorac Cardiovasc Surg* 1979; 77: 466-74.
- 10 Baba H, Wamoto I, Uchida J, Matsuo N, Hirota N, Kawashima N. Central nervous system consequences in children from cardiac surgery using simple deep hypothermia and circulatory arrest. Proceedings of the 10th Anniversary Meeting of Japan Society for Hypothermia 1979; 62: 64.
- 11 Hagerdal M, Harp JR, Nilsson L. The effect of hypothermia upon cerebral cortical blood flow and metabolism in the rat. *J Neurochemistry* (in press).
- 12 Prakash O, Johnson B, Bos E *et al.* Cardiorespiratory and metabolic effects of profound hypothermia. *Crit Care Med* 1978; 6: 165-71.
- 13 Miller SM, Cottrell JE, Turndorf H, Ransohoff J. Cerebral protection by barbiturates and loop diuretics in head trauma: possible modes of action. *Bull N.Y. Academy of Medicine* 1980; 56: 305-12.
- 14 Cottrell JE, Robustelli A, Post K *et al.* Furosemide and mannitol induced changes in intracranial pressure and serum osmolality and electrolytes. *Anesthesiology* 1977; 47: 28-30.
- 15 Michenfelder JD. The interdependency of cerebral functional and metabolic effects following massive doses of thiopental in the dog. *Anesthesiology* 1974; 41: 231-6.
- 16 Rockoff M, Marshall LF, Shapiro HM. High-dose barbiturate therapy in humans: a clinical review of 60 patients. *Ann Neurol* 1979; 6: 194-9.
- 17 Cullen JP, Aldrete JA, Jankowsky L. Protective action of phenytoin in cerebral ischemia. *Anesth Analg* 1979; 58: 165-9.
- 18 Astrup J, Moller-Sorensen P, Rahbek-Sorensen H. Inhibition of cerebral oxygen and glucose consumption in the dog by hypothermia, pentobarbital and lidocaine. *Anesthesiology* 1981; 55: 263-8.

- 19 Wauquier A. Brain protective properties of etomidate and flunarizine. *J Cereb Blood Flow Metab* 1982; 2: S53-S56.
- 20 Tulleken CAF, van Dieren A, Johnkan J, Kalenda Z. Clinical and experimental experience with etomidate as a brain protective agent. *J Cereb Blood Flow Metab* 1982; 2: S92-S97.
- 21 Steen PA, Newberg LA, Milde JH. Nemodipine improves cerebral blood flow and neurologic recovery after complete cerebral ischemia in the dog. *J Cereb Blood Flow Metab* 1983; 3: 38-43.
- 22 Allen GS, Bahr AL. Cerebral arterial spasm: Reversal of acute and chronic spasm in dogs with orally administered nifedipine. *J Neurosurg* 1979; 4: 43-7.
- 23 McDonald FM, Simonson T. Human electrocardiogram during and after inhalation of 30 per cent CO₂. *J Appl Physiol* 1953; 6: 304-11.
- 24 Rahn H, Reeves RB, Howell BJ. Hydrogen ion regulation, temperature and evolution. *Am Rev Respir Dis* 1975; 112: 165-72.
- 25 Ream AK, Reitz BA, Silverberg G. Temperature correction of PCO₂ and pH in estimating acid base status: An example of the emperor's new clothes. *Anesthesiology* 1982; 56: 41-4

Résumé

Le taux de mortalité et de morbidité durant la correction chirurgicale de l'anévrisme de l'arche de l'aorte est encore élevé malgré l'amélioration de la technique chirurgicale visant à assurer la protection du cerveau. Nous avons utilisé avec beaucoup de succès un système combinant l'hypothermie profonde et l'arrêt complet de la circulation. L'anesthésie administrée consistait en du pavulon, du fentanyl, de l'isoflurane ou de l'halothane si nécessaire. Le refroidissement de surface a précédé le refroidissement central pratiqué avec la machine cœur-poumon. L'arrêt complet de la circulation s'est produit quand la température de l'œsophage était de 12-14° C après l'administration de thiopentone de sodium (30 mgs·kg⁻¹), de méthylprednisolone 2 g, de furosemide 40 mgs et de mannitol 25 gs. A ce moment la tête a été complètement recouverte de sacs de glace et la correction chirurgicale pratiquée. Le temps moyen d'arrêt circulatoire était de 36.5 ± 13 minutes et la température de l'œsophage 12.5 ± 0.75° C. Nous ne rapportons aucune fatalité durant l'intervention chirurgicale. Nous n'avons constaté aucun déficit neurologique permanent chez nos patients.

La trachéotomie a été pratiquée chez cinq patients souffrant d'insuffisance respiratoire. Deux décès ont été rapportés des suites d'insuffisance rénale et respiratoire.

Cette technique est sûre et pratique et diminue la chance de complications durant la résection de l'anévrisme de l'arche de l'aorte.