Extracorporeal Circulation During Treatment of Aneurysms of the Ascending Aorta

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Abstract

Extracorporeal circulation in the treatment of aneurysms of the ascending aorta and transverse arch present both the surgeon and perfusionist with a formidable challenge. Thirty-one patients presented with either of these anomalies and have been treated in a two-year period from August 1987 to December 1989 at the University of Michigan. Of these, 11 had lesions which involved one or more arch vessels and necessitated either a period of circulatory arrest (7 patients) or brachiocephalic perfusion (four patients). Mean circulatory arrest periods were 44.8 ± 9.0 minutes (Mean ± SD), while the brachiocephalic perfusion patients had one or more arch vessels cannulated for a mean length of 64.3 ± 19.7 minutes. Postoperative complications in both groups included pulmonary and renal insufficiency, coagulopathies, and neurological deficits. The circulatory arrest group experienced encephalopathies (5/7 patients) and high perioperative mortality (4/7 patients). In the brachiocephalic perfusion group only one patient experienced postoperative neurological complications and there were no immediate postoperative deaths. Lesions of the ascending and transverse aorta are often associated with high morbidity and mortality. Although these population groups are small we believe that the technique of cerebral perfusion via cannulation of the brachiocephalic vessels provides a safe alternative to that of hypothermic circulatory arrest.

Introduction

Lesions which involve the ascending aorta and the transverse arch present the surgeon and perfusionist with the formidable challenge of cerebral preservation during interrupted flow. Early experience in treating these patients included cannulation of the brachiocephalic vessels, while systemic extracorporeal circulation (ECC) was either reduced or altogether stopped (1-4). As the clinical use of hypothermia and its physiochemical characteristics became better established in thoracic surgery, several individuals examined these effects on the protection of the central nervous system during a period of circulatory arrest (5-10). The results were encouraging and this method of ECC during repair was generally accepted. However, perioperative complications were evident and included bleeding diathesis, neurological complications, pulmonary insufficiency, and multiple organ failure (5 - 9). The mortality figures varied greatly between institutions ranging from 0 to 34% (8, 10 - 12), and a finite time for circulatory arrest was suggested to be no longer than 45 minutes (6, 8, 15), although arrest times of 75 minutes without neurological deficits have been reported (10). Indeed, in a discussion of the results of 30 patients treated by Crawford and Snyder with circulatory arrest, Miller stated, "the results are absolutely spectacular," and "(they) may never be equaled" (10).

Unequivocally, the primary concern during ECC is the protection of the central nervous system, with the major emphasis of either treatment directed towards cerebral protection. Several investigators have questioned the use of cerebral perfusion because of the complexity involved in circuit design (3,6,13,15). The various results obtained by utilizing brachiocephalic perfusion may reflect the lack of consensus on appropriate cerebral flows and pressures during ECC (1-4, 11-15). The benefits of brachiocephalic perfusion during surgical repair of these arch lesions include; one, a continuous supply of nutritive oxygenated perfusate provided to the still metabolically active tissue, two, reduction in the level of systemic hypothermia which may limit postoperative coagulopathies, and three, time extension for surgical repair. In addition, the lower levels of hypothermia required by selective cerebral perfusion may reduce overall ECC time limiting the associated pathophysiology.

This study was undertaken to examine the clinical outcome of both treatment modalities in patients with ascending aorta and transverse arch lesions. Each method of ECC will be described and the clinical results will be presented.

Materials and Methods

Between August 1987 and December 1989, 31 patients were seen with lesions involving the ascending aorta. A subgroup of 11 patients had coinciding lesions of the transverse aortic arch. These individuals were treated with profound hypothermia, and either circulatory arrest (CA) (seven patients), or brachiocephalic perfusion (BP) (four patients) of one or more

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arch vessels.

There were three males and eight females ranging in age from 29 to 78 years, mean of 63.9±16.6 SD (standard deviation). Six patients presented emergently with acute Type 1 aortic dissections (Debakey classification). Of the five remaining patients, three had atherosclerotic type lesions, one had focal granulomatous mesoatriitis, and one had a large sacciform aneurysm involving the ascending aorta and innominate artery. Associated disease states included coronary artery disease in one patient and severe aortic regurgitation in two patients. Neurological function was grossly assessed during preoperative evaluation, with no obvious deficiencies noted in any patient. No patient had discernible carotid bruits or previous history of cerebrovascular accident. All patients except for one were heavy smokers.

Table 1 lists the distribution of onsetting symptoms as well as type of aneurysm or dissection. Treatment selection was individualized for each patient according to angiographic interpretation of the location of the lesions. The ultimate decision to use either BP or CA was made by the surgeon (MMK).

Operative Technique

Induction of anesthesia was achieved in all patients by intravenous administration of diazepam, pancuronium, and fentanyl. Prior to aortic cannulation, each patient had their heads packed in ice and received a dose of thiamylal ranging from 6.3 to 12.3 mg Kg⁻¹ body weight. Each patient received the steroid methylprednisolone (one gram) concurrently with aortic cannulation.

All patients had portions of the ascending aorta and transverse arch replaced with low porosity woven dacron grafts (a). Each graft had been prebaked in albumin for 10 minutes at autoclave temperatures of 270°F. In treating dissections both the origin and the reentry point of the intimal tear was identified, with the prosthetic graft placed to incorporate both. In two patients the distal entry point was located at the level of mesenteric artery and it was decided to stage the repair. Severe aortic annulus dilatation necessitated concomitant valve replacement in two patients. Both distal and proximal anastomosis were prepared by reinforcing the site with felt strips using continuous running sutures. In most cases the ascending aorta was reinforced first with the cross clamp placed just proximal to the innominate artery (INA). When the target nasopharyngeal (NP) temperatures were reached, the perfusion flow rate was decreased, and a Foley catheter (b) was inserted and insufflated in the descending aorta to prevent retrograde cardiopulmonary bypass flow. The distal anastomosis and reimplantation of the arch vessels was performed. At that time the patient was placed in deep Trendelenberg position and retrograde perfusion begun. Deairing was carefully achieved by gentle manipulation of the arch vessels and prosthesis. Rewarming was begun at a rate not to exceed 1°C change in core temperature per three minutes, and the proximal anastomosis completed. The cross clamp was released and the patient's temperature was increased to 38°C before terminating bypass.

Cardiopulmonary Bypass

Components for the ECC circuit were chosen by the perfusionist and in all cases included a membrane oxygenator (c) with a closed system for venous return, and a constrained vortex pump (d) as the arterial drive system. Arterial cannulation was achieved via the femoral artery with a stainless steel 5.5 or 6.0 mm cannula, while venous return was accomplished either through a dual stage catheter, or with bicaval cannulas. All patients were fitted with left ventricular vents placed through the right superior pulmonary vein. Myocardial preservation was accomplished with cold (4°C) blood cardioplegia delivered either through the coronary sinus or by individual coronary ostia cannulas. Ventricular septal temperatures were continuously monitored and kept below 15°C in all patients. Additional topical cooling of the pericardial cavity was completed either with slush solution or a continuous cold saline flush. At the termination of cross clamping all patients received a bolus of aspartate/glutamate (14mMol L⁻¹) enriched blood cardioplegia warmed to 37°C. Patients receiving

FIGURE 1: Extracorporeal circuit used for combined perfusion of the systemic and cerebral circulations

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a. C.R. Bard Co., Inc., Billerica, MA
b. C.R. Bard Co., Inc., Billerica, MA
c. Scimed Life Systems, Inc., Minneapolis, MN
d. Biomedicus, Eden Prairie, MN
e. Cinco Cardiovascular Co., Wakefield, MA
f. Sarns 3M Health Care Systems, Ann Arbor, MI
g. Electromedics, Inc., Englewood, CO
h. Healthdyne Cardiovascular, Inc., Irvine, CA
i. Abbott Laboratories, Chicago, IL
BP were also fitted with additional 10F (c) or 12F (c) cannulas placed through purse string sutures in the left common carotid (LCC) and INA arteries, respectively. Two patients had both LCC and INA perfusion while two had only INA perfusion.

The perfusion circuit for BP is outlined in Figure 1 and consists of 1/4 inch internal diameter tubing attached to the recirculation line of the oxygenator, and placed through a roller pump (f). Oxygenated blood passes through a heat exchanger/bubble trap (g) and arterial line filter (h). The circuit recirculates constantly by incorporating a 1/4 "Y" connector at the field with return directed to the cardiotomy via a clamp. If both vessels are to be cannulated an additional 1/4 "Y" is connected to the stub of the perfusion line (Figure 2) with the bifurcation used to feed each cannula. Perfusion pressure is measured by an aneroid manometer located on the outflow port of the heat exchanger. A purge line is placed on the arterial line filter and used only during the priming and recirculation periods.

The pump prime solution consists of 2500 cc of Normosol (i) to which 50 mEq of NaHCO₃, 37.5 grams albumin, 25 grams mannitol, and 5,000 units heparin have been added. Mannitol was used to optimize the osmolality of the perfusate, decreasing intracranial pressure by minimizing extravascularization of fluid into the cerebral interstitium. The circuit hematocrit is calculated and at the onset of ECC is kept between 18-20%, decreasing the negative effects of viscosity changes experienced during cooling. ECC is instituted at an index of 2.4 L min⁻¹ M⁻¹ and cooling is carried out gradually. Acid base balance on CPB is maintained according to the principles of alpha stat regulation. Upon fibillation of the heart rapid systemic cooling is performed, and the aorta is cross clamped and cardioplegia administered. Temperatures vary according to the treatment plan with CA patients cooled to 15°C core, and when the NP temperature reaches 17°C, the pump is turned off and the patient partially exsanguinated. For BP patients, core temperatures are dropped to 20°C with the NP temperature falling to around 22°C. ECC systemic flows are reduced to 1 to 1.2 L min⁻¹ M⁻¹ while the distal anastomosis is completed. BP flow rates are controlled between 250 and 300 cc/min per head vessel cannulated, with pressure readings on the aneroid manometer not exceeding 125 to 150 mmHg.

Upon completion of both the distal anastomosis and brachiocephalic vessels, if accomplished en bloc, BP is stopped with flow to either the LCC or INA reinstated by the anastomosis. Rewarming times ranged between 50 and 75 minutes depending upon the depth of hypothermia reached. The patients hematocrit is brought up gradually to approximately 30%, and any deficiencies in acid base balance corrected. All patients were treated prophylactically for coagulopathies with fresh frozen plasma and platelets administered following reversal of heparin with protamine.

Statistical analysis was performed on a Macintosh SE computer utilizing the Statview SE (k) software package. A Student's two-tailed t-test was performed when evaluating data between the two study groups with statistical significance accepted at the p<.05 level. All data are expressed as mean ± standard deviation of the mean.

### TABLE 1: Aortic arch aneurysms and respective treatments

<table>
<thead>
<tr>
<th>Operation</th>
<th>Date</th>
<th>Disease</th>
<th>Symptoms</th>
<th>Extracorporeal Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>10/13/87</td>
<td>Type 1 Dissection *</td>
<td>Acute</td>
<td>Circulatory Arrest</td>
</tr>
<tr>
<td>2.</td>
<td>1/22/88</td>
<td>Type 1 Dissection</td>
<td>Acute</td>
<td>Circulatory Arrest</td>
</tr>
<tr>
<td>3.</td>
<td>1/28/88</td>
<td>Type C Aneurysm**</td>
<td>Non-Acute</td>
<td>Brach. Perfusion+</td>
</tr>
<tr>
<td>4.</td>
<td>4/22/88</td>
<td>Type 1 Dissection</td>
<td>Acute</td>
<td>Brach. Perfusion+</td>
</tr>
<tr>
<td>5.</td>
<td>4/25/88</td>
<td>Type C Aneurysm**</td>
<td>Non-Acute</td>
<td>Brach. Perfusion+</td>
</tr>
<tr>
<td>6.</td>
<td>11/18/88</td>
<td>Type 1 Dissection</td>
<td>Acute</td>
<td>Circulatory Arrest</td>
</tr>
<tr>
<td>7.</td>
<td>6/7/89</td>
<td>Type C Aneurysm**</td>
<td>Non-Acute</td>
<td>Circulatory Arrest</td>
</tr>
<tr>
<td>8.</td>
<td>6/20/89</td>
<td>Type A Aneurysm</td>
<td>Non-Acute</td>
<td>Brach. Perfusion#</td>
</tr>
<tr>
<td>9.</td>
<td>11/7/89</td>
<td>Type 1 Dissection</td>
<td>Acute</td>
<td>Circulatory Arrest</td>
</tr>
<tr>
<td>10.</td>
<td>12/1/89</td>
<td>Type C Aneurysm</td>
<td>Acute</td>
<td>Circulatory Arrest</td>
</tr>
<tr>
<td>11.</td>
<td>12/8/89</td>
<td>Type 1 Dissection</td>
<td>Non-Acute</td>
<td>Circulatory Arrest</td>
</tr>
<tr>
<td>12.</td>
<td>12/8/89</td>
<td>Type 1 Dissection</td>
<td>Non-Acute</td>
<td>Circulatory Arrest</td>
</tr>
</tbody>
</table>

Legend: *DeBakey classification of aortic dissections. **Cooley classification of aortic aneurysms. #=Brachiocephalic, cannulation of both innominate and left common carotid arteries. +=Cannulation of left common carotid only.

### Results

All patients survived the operation and were admitted to the thoracic intensive care unit. The distribution of patients receiving either CA or BP, the type of anatomic pathology, and the urgency of their operation is listed in Table 1. All patients had portions of their thoracic aorta replaced with dacron low porosity grafts that had been preclotted in albumin and baked in an autoclave for 10 minutes. CPB times and temperatures are listed in Table 2 where Group I patients are those that have received CA, while Group II patients were treated with BP. Mean CPB and cross clamp (CC) times did not vary between groups. The mean CA time was 44.8 minutes±9.0 (SD), while the mean BP time was 64.3 minutes±19.7 (p<.05) (Figure 3).

j. Apple Computer, Inc., Cupertino, CA
k. BrainPower, Inc., Calabasas, CA
Perfusate and bladder temperatures were also significantly lower in Group I compared to Group II (Table 1) (Figure 4).

Both groups had substantial postoperative morbidity associated with repair of their aneurysms (Table 2). Bleeding was the most frequently encountered non-neurological complication appearing in 43% of the patients in Group I and 25% of those in Group II. Other complications included pulmonary insufficiency (two patients in each group), acute tubular necrosis (two patients in Group I and one in Group II), and multiple organ failure in two patients from Group I. Decreased cardiac output in two patients from the BP group necessitated intraoperative placement of an intraortic balloon pump. Both patients were able to be weaned off the ventricular assist device on the second and fourth postoperative days (POD).

Mortality was substantial in the CA group with four out of seven patients expiring; two patients died within the first 10 POD. One patient developed sepsis and renal failure and died on POD seven, while the other patient exhibited multifocal myoclonus secondary to anoxic injury received during the CA period of 57 minutes. The third patient expired on POD 48 from multiple complications. This patient had been on long term steroid therapy as treatment for systemic lupus erythemosis as well as being diagnosed with uncontrolled hypertension. On POD three this patient exhibited right sided weakness and a neurology consult demonstrated a left cerebrovascular accident of the anterior cerebral distribution. It was felt that this may well have been a result of the long standing steroid therapy, although the period of CA could not be ruled out in precipitating this event. The pathological summary of the aneurysm specimen indicated cystic medial necrosis as the primary lesion with the etiology most likely attributed to steroid usage. The fourth patient was diagnosed on POD two with massive cerebral deficit as a result of bilateral hemispheric infarcts resulting in the patient failing to regain consciousness, and expiring of multiple organ failure on POD 35. All hospital deaths occurred in patients experiencing CA times greater than 40 minutes (mean 52.3 ± 10 min). Both patients who failed to regain consciousness following the procedure had arrest periods

![Figure 3: Comparison of bypass, cross clamp, and circulatory arrest vs. brachiocephalic perfusion times. All data are mean ± SD.](image)

![Figure 4: Perfusate, bladder, and nasopharyngeal temperatures between circulatory arrest and brachiocephalic perfusion group. All data are mean ± SD.](image)
of 57 and 64 minutes. In group II only one patient exhibited neurological dysfunction manifested as confusion and disorientation on POD 5. His initial postoperative course was unremarkable. He was able to respond to commands on POD 1, and extubated on POD 2. Neurological evaluation of his late cerebral event was thought to be secondary to a metabolic encephalopathy, and a CT scan of the head revealed no evidence of focal infarcts. He had acute tubular necrosis with BUN and creatinine levels peaking at 90 mg/dl and 4.4 mg/dl respectively. This patient's convalescence was further complicated by respiratory distress secondary to aspiration. He was discharged on POD 31 and continues to do well today. Postoperative followup averages 14.4±9 months with a range of one to twenty-four months. There was one late death in the BP group resulting from a second stage operation for a thoracoabdominal dissection following a Type 1 repair. This patient died on the operating table as a result of massive coagulopathy. All other patients are alive and well as of this time.

<table>
<thead>
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<th>TABLE 3: Complications following surgery for aortic arch aneurysms.</th>
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<tr>
<td><strong>Group 1</strong></td>
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<tr>
<td><strong>Patient</strong></td>
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<tr>
<td>1.</td>
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<td>2.</td>
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<td>6.</td>
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<td>7.</td>
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</tbody>
</table>

| **Group 2** | Complications | Neurological Deficit | Survival |
|---------------------------------------------------------------|
| **Patient** | | | |
| 1. | Renal insuff., low output, IABP # | Late confusion and disorientation | Discharged (POD 31) |
| 2. | Pulmonary insuff. | Normal | Discharged (POD 19) |
| 3. | Low cardiac output, IABP | Normal | Discharged (POD 11) |
| 4. | Reexploration for bleeding, pulmonary insuff. | Normal | Discharged (POD 28) |

Legend: *POD = Postoperative day. + = Patient in motor vehicle accident. # IABP = Intraaortic balloon pump placed in operating room

Discussion

Aortic aneurysms and dissections which involve the ascending and transverse arch are not uncommon (11), with untreated aortic dissections resulting in mortality rates of 50% at 48 hours and 90% at one month (15). The first reported correction of a ascending thoracic aneurysm occurred over 30 years ago (16), yet the challenge in treating these patients remains substantial. The development of graft materials of low porosity that permitted rapid tissue in growth facilitated an era of increasing success in treating these vascular defects.

The incorporation of extracorporeal circulation as a means of redirecting blood away from the operation site, coupled with the advent of myocardial preservation strategies, provided an environment which enabled surgical access with unobstructed correction. In spite of all the technological sophistications the issue of paramount importance continues to involve the ability of the nervous system to withstand any imbalances in nutritive delivery.

The extracorporeal techniques of circulatory arrest and brachiocephalic perfusion create a non-physiologic environs which greatly affects cerebral metabolism. The occurrence of neurological deficits following treatment of these aneurysms with either modality remains high, ranging from 0 to 35% (1,2,4,11). Therefore, this paper was undertaken for two reasons; one, to present our results utilizing both methods of extracorporeal circulation in treating these select patients, and two, to increase our understanding of cerebral hypoperfusion and cephaloplegia.

Routine open heart surgery results in significantly higher brain injury than other types of surgery (17), with embolic phenomena (18) and hypoperfusion often indicated as etiological factors. The minimization of these events does not preclude neurological injury from developing which may be related to the diversity of perfusion hemodynamic techniques, cerebral metabolic requirements at various temperatures, methods of acid base management, and the level of anesthesia.

Brain perfusion during extracorporeal circulation is affected by cerebral autoregulation which itself is a multifactorial phenomena, dependent upon alterations in hemodynamic and respiratory parameters. It is defined as the "ability of a vascular bed to alter its resistance in response to a pressure change whereby flow remains constant" (10).

During cardiopulmonary bypass preferential perfusion of the cerebral vasculature occurs between a mean arterial pressure of 30 to 100 mm Hg, with the redirection of flow away from the systemic system (10). We chose to maintain our indirect cerebral perfusion pressure between 75 and 100 mm Hg. In the well controlled study of Matsuda, et al. (13) the authors measured cerebral perfusion pressures during brachiocephalic perfusion by cannulating the bilateral superficial temporal arteries. They maintained pressures initially between 50 and 75 mm Hg by direct measurement and reported a 10% occurrence of neurological disturbances over 30 patients (13). They also reported flow rates of between 4.5 and 12.9 ml/min/kg depending upon number of arch vessels cannulated, and monitored cerebral venous saturations via the internal jugular and superior vena cava cannula, keeping their levels greater than 90%. Our flow rates were similar and averaged 9.2 ml min⁻¹ kg⁻¹ and although we did not monitor in line cerebral venous saturations, our venous saturation monitor for the ECC circuit did not fall below 90% during BP. Soma et al (14) cannulated the right axillary, both right and left carotids, and the left subclavian arteries in a group of 10 patients, and maintained flow rates of 830±278.2 ml min⁻¹. In one study (2) where BP flows of 350 ml min⁻¹ vessel⁻¹ with perfusion pressures kept
between 150 and 200 mm Hg, there was a high incidence of hyperpyrexia and lesions of the cerebrovascularity, suggesting arterial injury. No mention was made of patient size or indexed flows, or of the method of acid base maintenance during ECC in this study. Michenfelder has stated (20) that chronic hypertensive patients may experience better brain protection if their cerebral perfusion pressures are maintained higher than normotensive patients. This suggests that each protocol for flow and pressures during BP must be individualized for each patient. Others have reported cerebral flow rates during BP of 200 to 300 ml/min\textsuperscript{1} (15,16) with the absence of neurological problems.

Although optimum flow requirements of the brain have not been established it has been reported that cerebral blood flow for normothermic, normotensive patients is approximately 55 ml 100 gm\textsuperscript{-1} min\textsuperscript{-1} (21). The adult human brain weighs approximately 1,400 gm, which normally receives 15% of the cardiac output. However, caution must be exerted in maintaining this volume of flow during selective BP. These values were obtained from individuals with normal hemoglobin levels at normothermic temperatures which differ greatly from patients who are anesthetized, profoundly hypothermic, and hemodiluted.

Some of the factors effecting cerebral flow must be considered in establishing a protocol for BP. It is known from the Q10 value (the ratio of the rate of a reaction of one temperature to that observed with a 10°C change in temperature) that the cerebral metabolic rate decreases with temperature so that at 27°C, metabolic needs are reduced by approximately 50%. The resistance of the cerebral vessels is also effected by blood viscosity. Since hematocrit is the major determinant of blood viscosity, and since viscous changes in blood are inversely related to temperature, the profound hypothermia alone would decrease cerebral flow. However, this effect on cerebrovascular resistance is offset by the level of hemodilution employed during ECC when hematocrit levels are kept between 18 and 20%. In our experience these values afforded adequate oxygen delivery to the brain evidenced by mixed venous saturation levels never falling below 90%.

The utilization of profound hypothermia results in metabolic deregulation and a loss of membrane integrity (28). Hemorrhagic complications induced by profound hypothermia are common with the majority of our patients experiencing some coagulation complication, necessitating multiple transfusions which included fresh frozen plasma with platelet therapy.

It is also known that autoregulation of the brain may be impaired by anesthetics, hypocapnia, and cerebrovascular disease. Therefore, the assumption cannot be made that functional characteristics based on autoregulatory principles will remain intact. Fox and associates (22) have shown that when the cerebral temperatures of monkeys are cooled to 20°C the whole brain blood flow is decreased as systemic flow drops, but that the proportion of total flow to the brain is increased. Furthermore, brain O\textsubscript{2} extraction increased as perfusion flow declined, which was unlike other tissue beds in the body (22).

Acid base management during hypothermia has long been debated in the perfusion literature. We maintained our ventilating gases, and hence perfuse blood gas composition, to maintain constant PaCO\textsubscript{2} levels around 40 mmHg (temperature uncorrected) reflecting alpha stat methodology (23). The partial pressure of CO\textsubscript{2} in arterial blood has a profound effect of cerebral blood flow in an indirect linear relationship. Gover et al (24) studied several factors and their relationship to CBF during hypothermic CPB, and found a significant direct correlation between PaCO\textsubscript{2} levels and CBF. They advocate maintaining a normal PaCO\textsubscript{2} (temperature uncorrected) during hypothermic CPB (24). In a study by Murkin (25), 33 patients undergoing routine coronary artery bypass grafting were treated with either pH or alpha stat CPB management strategies. In the pH stat treated patients, during hypothermia, cerebral O\textsubscript{2} consumption fell sharply while CBF remained essentially unchanged resulting in cerebral hyperperfusion or hyperemia. This PaCO\textsubscript{2} dilatation may jeopardize areas of the brain dependent upon flow through critically stenosed vessels (26). The alpha stat group had similar declines in cerebral O\textsubscript{2} consumption but paralleled a decline in CBF to approximately half that seen in the pH stat group.

The reduction in cerebral basal metabolic rate with temperature facilitates the ECC decreased flow rates necessary for correction of brachiocephalic lesions. Although cerebral O\textsubscript{2} requirements decrease by an estimated 5 to 7% °C\textsuperscript{-1}, the linearity of the predicted value does not persist with profound hypothermia. Even with nasophynageal temperatures of 15°C, once circulatory arrest ensues severe cerebral hypoxia quickly develops (27).

The use of barbituates in providingadded cerebral protection has not been clearly defined, and their benefits in open heart surgery may only be evident when they are administered in the immediate perioperative period (29). Barbituates may provide protection by reducing intracranial blood volume, pressure and edema, as well as acting as oxygen free radical scavengers (29).

Little has been published on barbiturate dosing of patients undergoing CA or CPB utilizing profound hypothermia. In one study the authors stated that barbiturate maximum protection was seen at doses which abolished cortical electrical activity, which was achieved with a mean thiopental dose of 39.5 mg kg\textsuperscript{-1} (29). Concomitantly, however, these patients had depressed myocardial contractility and required longer pulmonary recovery periods for extubation (29). It is not known whether barbituates and hypothermia have an additive effect on cephaloplegia, but profound cerebral depressant effects are seen during CPB with thiopental doses of 8 to 24 mg/kg\textsuperscript{-1} at 25 to 30°C hypothermia (30). We have utilized a dose of 12.5 mg/kg\textsuperscript{-1} of thiopental in all patients undergoing CA or BP.

Summary

The treatment of patients with lesions involving the ascending aorta and transverse arch with extracorporeal circulation involving circulatory arrest or brachiocephalic perfusion remains a formidable challenge. Circulatory arrest optimizes a bloodless operative field maximizing exposure, is un-
complicated by arch vessel cannulas, and allows total visualization of the lesion. However, the level of profound hypothermia necessary for cerebral protection, predisposes the patient to increased coagulopathies, severe cerebral hypoxia, and forces the surgeon to complete the repair and restore cerebral circulation within a short finite time period. Brachiocephalic perfusion succeeds in providing nutritive perfusate to the brain during the period of surgical repair, as well as removing metabolic intermediates, increasing the time allotment for surgical reconstruction, and decreasing the level of systemic hypothermia. This method requires the placement of peripheral cannula(s) and associated circuitry, necessitating a constant vigilance on the hemodynamic events involving the cerebral circulation. In our experience the method of brachiocephalic perfusion has proven more efficacious than circulatory arrest in treating these select patients.

References