Determination of Cerebral Blood Flow Dynamics During Retrograde Cerebral Perfusion Using Power M-Mode Transcranial Doppler

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Background. **Retrograde cerebral perfusion (RCP) during profound hypothermic circulatory arrest has been used as an adjunct for cerebral protection for repairs of the ascending and transverse aortic arch. Transcranial Doppler ultrasound has been used to monitor cerebral blood flow during RCP with varying success. The purpose of this study was to characterize cerebral blood flow dynamics during RCP using a new mode of monitoring known as transcranial power motion-mode (M-mode) Doppler ultrasound.**

Methods. **Data on pump-flow characteristics and patient outcomes were collected prospectively for patients undergoing ascending and transverse aortic arch repair. Retrograde cerebral perfusion during profound hypothermic circulatory arrest was used for all operations. Intraoperative cerebral blood flow dynamics were monitored and recorded using transcranial power M-mode Doppler ultrasound.**

Results. **Between August 2001 and March 2002, we used transcranial power M-mode Doppler ultrasound monitoring for 40 ascending and transverse aortic arch repairs during RCP. Mean RCP time was 32.2 ± 13.8 minutes. Mean RCP pump flow and RCP peak pressure for identification of cerebral blood flow were 0.66 ± 0.11 L/min and 31.8 ± 9.7 mm Hg, respectively. Retrograde cerebral blood flow during RCP was detected in 97.5% of cases (39 of 40 patients) with a mean transcranial power M-mode Doppler ultrasound flow velocity of 15.5 ± 12.3 cm/s. In the study group, 30-day mortality was 10.0% (4 of 40 patients). The incidence of stroke was 7.6% (3 of 40 patients); the incidence of temporary neurologic deficit was 35.0% (14 of 40 patients).**

Conclusions. **Transcranial power M-mode Doppler ultrasound consistently demonstrated retrograde middle cerebral artery blood flow during RCP. Transcranial power M-mode Doppler ultrasound can provide optimal RCP with individualized settings of pump flow.**

(Ann Thorac Surg 2003;76:704–10)

Shortly after retrograde cerebral perfusion (RCP) was first described for proximal aortic repairs [1], it became a standard adjunct for cerebral protection during profound hypothermic circulatory arrest (PHCA) at many centers. Some researchers, ourselves included, have used RCP and observed enhanced survival and outcome [2–8]. Others, however, have found no clinical improvements [9–14]. The cited benefits of RCP include ease of establishment, uniform cooling of the brain, flushing of atheromatous and gaseous debris, and possible provision of nutrient flow to the cerebral circulation, although the reliability of this last advantage is debatable. Several studies have reported the metabolic benefits of RCP during PHCA in animals [14–17], but demonstrating these benefits in humans has been difficult.

Part of the problem in determining the usefulness of RCP is the difficulty of detecting reversed cerebral blood flow to the brain. Many groups have experimented with transcranial Doppler (TCD) ultrasound for the purpose of monitoring cerebral blood flow [13, 18–20]. But inconsistencies in identification and monitoring cerebral blood flow have done little to clear up uncertainties. We recently adopted transcranial power motion-mode Doppler (PMD-TCD, or M-mode) invented by Moehring and Spencer, for monitoring middle cerebral artery blood flow during RCP [21]. Transcranial power M-mode Doppler displays flow intensity and direction for more than 5 cm of intracranial space simultaneously by means of a 33-sample gate. One of the advantages of this mode of insonation is that all flow signals obtainable along a given position and direction of the ultrasound beam may be displayed. Power M-mode TCD enables easier location of acoustic windows, when compared with a single-gate spectral TCD [22].

The purpose of this study was to use PMD-TCD to characterize the cerebral blood flow dynamics during

Presented at the Forty-ninth Annual Meeting of the Southern Thoracic Surgical Association, Miami Beach, FL, Nov 7–9, 2002.

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Published by Elsevier Inc

0003-4975/03/$30.00
PII S0003-4975(03)00552-6

Table 1. Demographics

Variable	Patients	Percent
Total	40	100.0
Female	19	47.5
Male	21	52.5
Acute dissection	10	25.0
Chronic dissection	10	25.0
Coronary artery disease	13	32.5
COPD	13	32.5
Hypertension	30	75.0
Cerebrovascular disease	2	5.0
Prior aneurysm repair	8	20.0
Prior stroke	2	5.0
Rupture	2	5.0

COPD = chronic obstructive pulmonary disease.

repairs of the ascending and transverse aortic arch using PHCA with RCP.

Material and Methods

During the study period of August 2001 and March 2002, we used PMD-TCD monitoring in 40 graft replacements of the ascending and arch aorta. Twenty-one patients (52.5%) who suffered from aortic insufficiency also had aortic valve replacement (7 patients) or valve resuspension (14 patients). All operations were performed using PHCA and RCP. Patient ages ranged from 32 years to 91 years, with a median age of 63.3 years. Table 1 shows basic patient demographics.

Operative Procedure

Operations varied depending on the cause of disease, but the basic features of our current technique for dissection or aneurysm of the distal ascending and proximal or transverse aortic arch include cardiopulmonary bypass (CPB), profound hypothermia, circulatory arrest, and RCP.

Anesthetic Management

All patients were monitored using 5-lead electrocardiogram, peripheral pulse oximetry, end tidal CO_2 measurement, temperature probes (nasopharyngeal, bladder, blood), arterial catheter placement, pulmonary artery catheter, and bilateral cerebral oximetry (Somanetics, Troy, MI). Transesophageal echocardiography was used, and PMD-TCD was performed by a neuro-ultrasonographer. Shortly after induction the patient's head was fitted with a probe-fixation head frame using a hand-free standard 2-MHz pulsed-wave TCD transducer (Spencer Technologies, Seattle, WA), positioned on the temporal bone window for monitoring the middle cerebral artery blood flow velocity (centimeters per second). The mean flow velocities and pulsatility indices were obtained using a single-channel spectral display at assumed zero angle of insonation at the depths represented by a yellow line on the PMD-TCD screen display. Pulsatility index was defined as the systolic velocity minus the diastolic velocity divided by the mean velocity. Bilateral middle cerebral artery blood flow velocities were recorded before the initiation of CPB, during CPB, during PHCA with RCP, at reinitiation of CPB, and at the conclusion of CPB. Figure 1 illustrates the relationships of pump flow, pressure, and PMD-TCD velocity during RCP.

Anesthetic induction was with fentanyl (10 to 15 μg/kg), midazolam (0.05 mg/kg), pancuronium (0.1 mg/kg), and propofol (40 to 70 mg). Aprotinin was administered as a 1 million–unit load followed by an infusion of 250,000 U/h. Maintenance of anesthesia was achieved with continuous administration of isoflurane 0.5% to 1.0% and an oxygen/air mixture. Hemodynamics were controlled to achieve a cardiac index between 2.0 and 3.0 $L \cdot min^{-1} \cdot m^{-2}$. Serial arterial blood gas measurements were obtained, and the hematocrit was kept at greater than 24% while the patient was warmed, and allowed to drift to no less than 18% when the patient was cooled. Alpha stat management was used for acid-base control throughout the procedure.

Depending on the degree of atheromatous plaque in the descending and ascending aorta as identified by transesophageal echocardiography or epiaortic ultrasound, CPB was established through the ascending aorta or the right common femoral artery and vein. The superior vena cava was cannulated through the right atrium, and snares were applied to both the inferior and superior vena cavae. Systemic cooling was initiated, and the patient's temperature was monitored using both nasopharyngeal and bladder temperature probes. Myocardial protection was achieved using continuous retrograde cold blood cardioplegia through the coronary sinus, supplemented with direct antegrade coronary ostia infusion once the aorta was opened. A left ventricular sump was inserted through the right superior pulmonary vein. Both cell-saving device and pump suction were used for blood salvage.

A 10-lead electroencephalogram monitored cerebral function. Once the electroencephalogram was isoelectric, which coincided with a nasopharyngeal temperature of 15°C to 20°C, CPB was discontinued and circulation was arrested. Retrograde cerebral perfusion was begun through the superior vena cava cannula using a centrifugal pump. In the past we used a conventional maximum flow rate of 500 mL/min in the RCP circuit to maintain the superior vena cava catheter pressure less than 25 mm Hg. Using PMD-TCD to directly monitor cerebral flow in both middle cerebral arteries, we were able to increase or decrease pressure as necessary to maintain middle cerebral artery flow. Power M-mode TCD identifies any reversal of flow during RCP and provides a guide for optimal RCP flow.

After completion of the distal reconstruction, RCP was discontinued, and a cannula was placed into the new aortic graft. With the patient in the Trendelenburg position, CPB flow was initiated through the femoral cannula until all debris was evacuated through the open aortic graft. Antegrade flow was established through the newly inserted graft cannula, the graft was clamped, and systemic warming was begun. Proximal reconstruction was

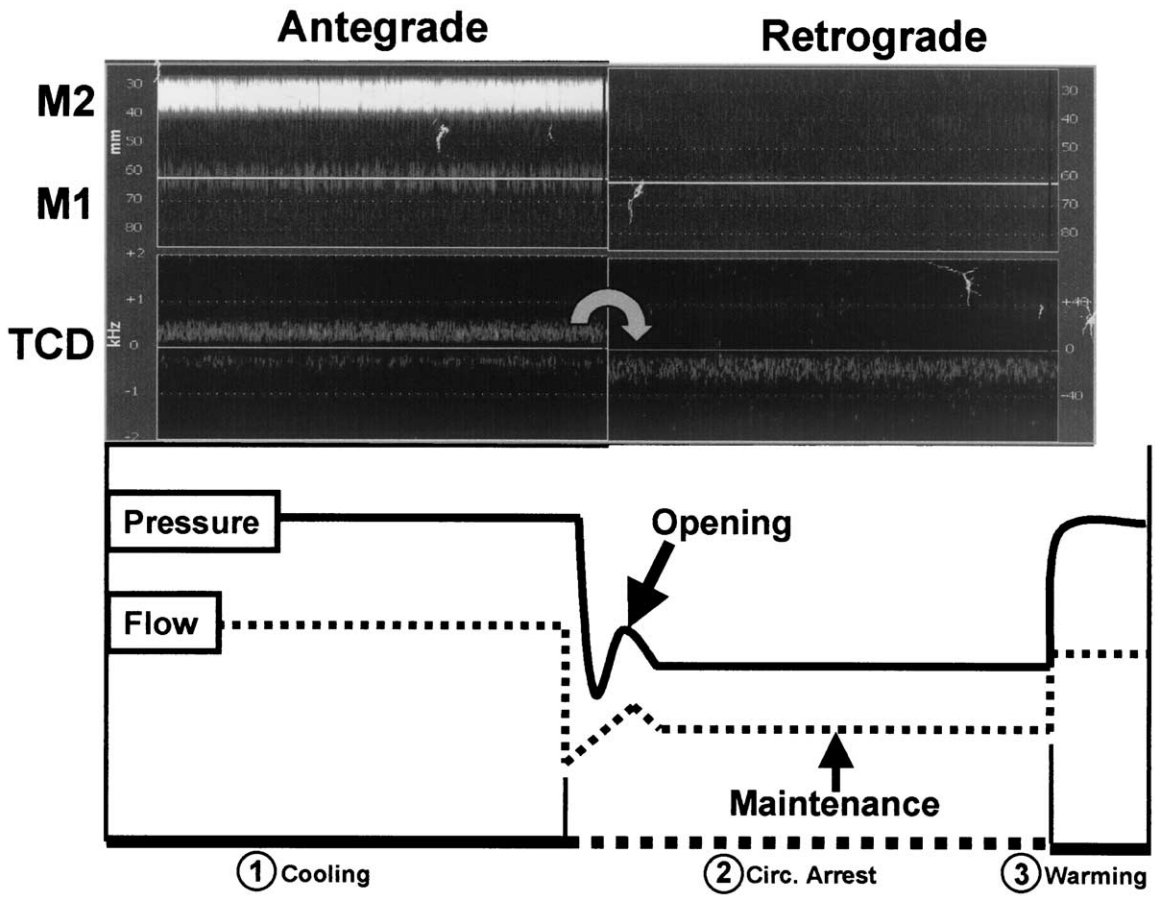

*Fig 1. Relationships of pump flow, pressure, and power M-mode transcranial Doppler (*TCD*) velocity during retrograde cerebral perfusion. (*1*) Cooling phase: relatively constant pump flow and systemic pressure; power M-mode transcranial Doppler velocity may decrease. (*2*) Circulatory (*Circ*) arrest phase: antegrade pump flow is discontinued, systemic pressure falls, power M-mode transcranial Doppler velocity disappears. Retrograde cerebral perfusion begins with increasing pump flow and cerebral venous pressure. Once reversed cerebral artery flow is identified with power M-mode transcranial Doppler, at the corresponding opening pressure retrograde cerebral perfusion pump flow is decreased, still maintaining reversed cerebral blood flow at a maintenance pressure. (*3*) Warming phase: antegrade pump flow is reinitiated; systemic circulation is restarted. (*M_1 = first branch of middle cerebral artery [horizontal segment]; M_2 = second branch of middle cerebral artery [sylvian segment]).*

completed while the patient was systemically warmed. Warming was continued until the patient's core body temperature reached 36°C. Blood and nasopharyngeal temperature never exceeded 37°C.

Outcome Variables

Mean arterial pressure, central venous pressure, hematocrit, pH, O_2, CO_2, pump flow (liters per minute), nasopharyngeal temperatures, and PMD-TCD middle cerebral artery blood flow velocities (centimeters per second) were recorded before CPB, at the initiation of CPB, for the duration of PHCA with RCP, at reinitiation of CPB, and just before weaning from CPB. Opening RCP flow and pressure were defined as the RCP flow (liters per minute) and RCP line pressure (millimeters of mercury) required to identify reversal of flow in the cerebral circulation. Maintenance flow and pressure was the flow (liters per minute) and pressure (millimeters of mercury) required to continually identify reversed cerebral blood flow after first identification.

Operative mortality refers to deaths that occurred within 30 days of surgery. In-hospital mortality refers to deaths that occurred during hospitalization. Neurologic injury was defined as either the occurrence of stroke or temporary neurologic deficit. Stroke was defined as any gross focal neurologic brain injury, either temporary or permanent, as identified by neurologic examination and confirmed with computed tomography or magnetic resonance imaging. Delirium, obtundation, disorientation, or altered mental status denoted temporary neurologic deficit. A history of transient ischemic attack, amaurosis fugax, cerebrovascular accident, or intervention for carotid artery disease characterized cerebrovascular disease. Repairs involving dissection were considered acute if surgery was performed in the 14-day period after onset of dissection, and chronic thereafter.

Data Analysis

Data were collected from chart reviews performed by a trained nurse abstractor, and were entered into a dedi-

Table 2. Operative Variables

Variable	Mean	SD
Clamp time (min)	91.6	31.6
Circulatory arrest time (min)	32.5	13.7
Pump time (min)	165.1	55.0
RCP time (min)	32.2	13.8
Cooling time (min)	19.6	6.3
Warming time (min)	78.3	24.4

RCP = retrograde cerebral perfusion.

cated Microsoft Access (Microsoft Corp, Redmond, WA) database. Data were exported to SAS for analysis, and all computations were performed using SAS version 8.02 (SAS Institute, Cary, NC) running under Windows 2000. Patients were followed until death or until follow-up reached the study end date (March 1, 2002). Within-subject comparisons of means were performed using paired Student's *t* tests or Wilcoxon sign-rank tests as appropriate, depending on distributional assumptions. Correlation analysis was performed using Pearson product-moment for normal data or Spearman rank correlations for nonnormally distributed data. The null hypothesis was rejected at p less than 0.05.

Results

Operative factors are listed in Table 2. Mean RCP pressure was 32.2 ± 13.8 mm Hg. Mean PHCA time was 32.6 ± 13.7 minutes. Pump flows, systemic and RCP pressures, and both antegrade and retrograde cerebral blood flow velocities are listed in Table 3. Mean opening RCP pump flow was 0.66 ± 0.11 L/min. and opening RCP pressure for identification of reversed cerebral blood flow was 31.8 ± 9.7 mm Hg. Mean RCP maintenance flow and pressure were 0.52 ± 0.08 L/min and 25.1 ± 8.7 mm Hg, respectively.

Retrograde cerebral blood flow during RCP was detected in 97.5% of cases (39 of 40 patients) with a mean PMD-TCD flow velocity of 15.5 ± 12.3 cm/s. In 1 patient flow was not detected because of unavailability of an adequate temporal window. This patient received RCP at the standard flow rate of 500 mL/min at 25 mm Hg. For the PMD-TCD flow velocity the standard deviation was high because of one value, recorded for the second patient who had an uncharacteristically high PMD-TCD velocity of 80 cm/s. Excluding this value, the range was 5 to 32 cm/s with a standard deviation of ± 6.23 cm/s. The mean pre-CPB and post-CPB pulsatility indexes were 1.11 ± 0.31 and 0.99 ± 0.14, respectively. This was not significantly different ($p > 0.05$). In addition, during periods when patients received standard RCP flows (ie, 500 mL/min) and RCP pressure less than 25 mm Hg, reversal of cerebral blood flow was identified in only 8 of 40 (20%) patients.

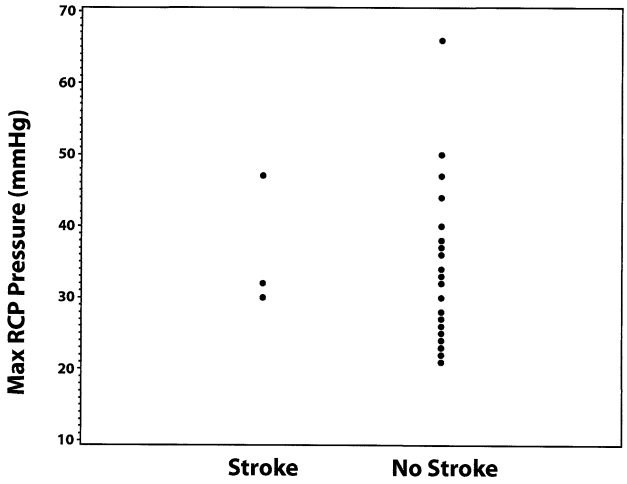

*Fig 2. Plot of highest retrograde cerebral perfusion (*Max RCP*) pressure in patients with stroke compared to those without. Pressures are not different between the groups.*

In the study group, 30-day mortality was 10.0% (4 of 40 patients), and hospital mortality 12.5% (5 of 40 patients). The incidence of stroke was 7.6% (3 of 40 patients); the incidence of temporary neurologic deficit was 35.0% (14 of 40 patients). No relationship between highest RCP pressure and stroke rate was observed (Fig 2). A neurologist evaluated any patient exhibiting signs of temporary neurologic deficit. In all instances cerebrovascular accident was ruled out by computed tomographic scan or magnetic resonance imaging. The incidence of 30-day mortality and stroke did not differ significantly from our previous series ($p = 0.70$ and $p = 0.17$, respectively) [6].

Table 3. Measurement of Mean Cardiopulmonary Retrograde Cerebral Perfusion Flow, Pressure, and Velocity During Operative Repair[a]

variable	Pre-CPB	Initial CPB	Pre-RCP	Open RCP	Maint RCP	Reinit CPB	End CPB
Flow (L/min)	NA	4.14 ± 0.73	3.99 ± 0.63	0.66 ± 0.11	0.52 ± 0.08	4.01 ± 0.68	4.41 ± 0.75
Systemic pressure (mm Hg)	79.4 ± 13.1	67.5 ± 8.8	66.5 ± 12.7	NA	NA	61.4 ± 16.2	74.4 ± 9.4
RCP pressure (mm Hg)	NA	NA	NA	31.8 ± 9.7	25.1 ± 8.7	NA	NA
Velocity (cm/s)	34.1 ± 12.8	28.9 ± 15.8	16.9 ± 4.5	15.5 ± 12.3	15.5 ± 12.3	22.4 ± 9.9	43.1 ± 17.4

[a] Values are mean ± standard deviation.

End CPB = off pump; Initial CPB = period at CPB initiation; Maint RCP = period during maintenance retrograde cerebral perfusion; NA = not available; Open RCP = period when flow reversal is first identified in middle cérebral artery by power M-mode transcranial Doppler; Pre-CPB = period immediately preceding cardiopulmonary bypass (CPB) initiation; Pre-RCP = period before CPB terminated and circulation arrested; Reinit RCP = period when retrograde cerebral perfusion is reinitiated.

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Comment

Although it has been demonstrated that increased RCP pump flow increases retrograde cerebral blood flow [23] and that higher RCP pressure may be required to identify reversed cerebral blood flow [20], the concept of an opening pressure for RCP has not been established. Like Ganzel and associates [20], we observed that a higher RCP pressure was required to immediately identify reversed cerebral blood flow (mean RCP pressure, 33.8 mm Hg), although our mean opening RCP flow was lower (0.67 L/min versus 1.2 L/min). Moreover, once reversed middle cerebral artery blood flow was identified by PMD-TCD, the RCP pump flow could be decreased to a maintenance level (0.53 L/min), leading to an RCP maintenance pressure of 25.1 mm Hg.

The requirement of a higher opening pressure (as opposed to maintenance pressure) may be related to an increase in cerebral-venous resistance as a result of the conversion from antegrade to retrograde perfusion or caused by the need to overcome competent venous valves [24]. The concept of an opening pressure has also been described for monitoring the adequacy of antegrade selective cerebral perfusion [25]. At any rate, standard RCP flows and pressures (0.5 L/min and 25 mm Hg) may not be adequate to achieve reversed cerebral perfusion during RCP. When Tanoue and colleagues [13] used TCD to identify cerebral blood flow during RCP, with RCP pressure limited to less than 20 mm Hg, reversed cerebral blood flow was observed in only 3 of 15 patients. Similarly, when patients received standard RCP pressure and flow, reversed cerebral blood flow was identified in only 20% of cases.

The standards for RCP pump flow and pressure were established early on [1, 23], with most centers regulating RCP at a pump flow of 500 mL/min with care to avoid RCP pressures more than 25 mm Hg. The concern for excessive RCP pressure has been related to the potential for increased cerebral edema leading to neurologic injury [26]. Although we did observe high opening RCP pressures, we did not observe PMD-TCD evidence of increased cerebral edema, which would have manifested as significantly different pulsatility indices and postbypass end-diastolic flow velocities [20, 27]. In addition, clinical evidence supplied by physical examination or radiographic evaluation demonstrated no increased cerebral edema. A recent animal study that evaluated increased RCP pressures (34 to 40 mm Hg) reported amplified cerebral tissue perfusion, but no rise in tissue edema at higher RCP pressures [28].

Lengthy periods of cerebral ischemia during PHCA without cerebral perfusion have been associated with an increased incidence of neurologic injury, both permanent and temporary [29]. This may be as high as 8% to10% for stroke and as high as 40% for temporary neurologic deficit [30]. In our RCP studies stroke rates have been comparatively lower. In our 1997 study, in which pressure was capped at 25 mm Hg, the stroke rate in the RCP group was 3 of 120 (2.5%) [6]. In the current series, where pressure was increased until reversal of flow was documented, stroke rate was 3 of 40 (7.5%). The odds ratio for this comparison is 3.16, but is not significant at $p < 0.17$. Whether the apparent increase in the incidence is related to increased pressure in this series is difficult to say, based on the small number of events. Internally to the current study population, no relationship between highest RCP pressure and stroke was observed ($p > 0.44$; Fig 2).

Data from cerebral circulation studies estimate that the amount of cerebral blood flow derived from RCP may range from 20% to 60% of antegrade cerebral blood flow during CPB [30]. The difference in cerebral blood flow is attributed to venous shunting during RCP. Although a significant amount of RCP flow may lead to venous shunting, it has been determined that the amount of RCP flow that reaches the cerebral circulation is directly dependent on the RCP pump flow [23, 31]. In the past, we used a conventional maximum flow rate of 500 mL/min in the RCP circuit and kept superior vena cava cannula pressure less than 25 mm Hg. Using PMD-TCD to directly monitor cerebral flow in both middle cerebral arteries, we were able to increase or decrease pressure as necessary to maintain middle cerebral artery flow in 97.5% of cases. Power M-mode TCD identifies any reversal of flow during RCP and provides a guide for optimal RCP flow.

Another advantage of the multichannel PMD-TCD is the larger window for detection. This allowed for a higher sensitivity in the identification of reversed cerebral blood flow. A limitation of this technology, however, is the necessary dependence on a skilled technician for operation of the monitor. As with TCD in general, PMD-TCD is also limited by patient variables. The use of PMD-TCD assumes that an adequate temporal window is available. Consistent with previous reports, our one case in which PMD-TCD failed to identify cerebral blood flow was an older female patient with no temporal window [13, 20]. (She received RCP at the standard flow rate. Postoperatively, she had a temporary neurologic deficit.)

This report represents our initial experience with PMD-TCD during repairs of the ascending aorta and transverse arch. Although we now use PMD-TCD for all proximal ascending and transverse aortic arch repairs, PMD-TCD was first used on the basis of the availability of the neuro-ultrasonographer (Z.G.) during the study period. Moreover, assessment of more subtle neurologic injury, ie, neurocognitive testing, was not performed. Randomized studies including fine neurocognitive testing will be required to validate the benefit of this monitoring device.

In conclusion, digital multichannel PMD-TCD ultrasound consistently demonstrated retrograde middle cerebral artery flow during RCP. Power M-mode TCD can provide optimal RCP with individualized pump flow settings.

We would like to thank Amy Newland Wirtz for her editorial assistance and Andrei Alexandrov, MD, for his professional guidance.

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DISCUSSION

DR CONSTANTINE MAVROUDIS (Chicago, IL): You said there was a 30-day mortality. Most of us would define operative mortality as that which would include deaths that occurred within 30 days of the operation in the event of intervening discharge or death occurring during the same hospitalization. This would include those patients who were in the hospital at the time of their death, which in a patient who has had a stroke or chronic infection could last maybe 6 months or so. Did that alter your mortality figures in this light? In other words, was what you said concerning a 30-day mortality in keeping with the accepted mortality of 30 days or during the same hospitalization?

DR ESTRERA: This is 30-day death. In our previous reports we have always used 30-day mortality in our series. The in-hospital mortality for this series was 12.5% (5 of 40 patients).

DR MAVROUDIS: Just for the record, there are standards about this, and the standard is 30 days within the time of the operation or within that hospitalization. I suggest that is probably what you ought to use.

DR ESTRERA: Thank you.

DR GEORGE CIMOCHOWSKI (Wilkes-Barre, PA): This is a beautifully presented paper and I think it is very important. My own feeling is I think circulatory arrest without cerebral monitoring has seen its day. To me, circulatory arrest alone without either retrograde or antegrade cerebral perfusion is sort of like driving your car and turning the lights out and hoping at the end of the day you are still on the road.

We had to resort, of course, to temporal artery monitoring because this was not available, but once you have monitoring of the temporal arteries and know there is flow, the risk of malperfusion syndrome goes down immensely. This, of course, precludes I think the need for cutting down the temporal arteries, this is a simpler approach, it can be used bilaterally, and for those people who want to use the right brachial or right axillary, it would be very useful to have this on the left side to see in fact that you have an intact circle of Willis.

I congratulate you. I think this should be standard. Thank you.

DR ESTRERA: Thank you for your kind comments. I appreciated the report you just presented. Of interest, this concept of observing an opening pressure to identify retrograde cerebral perfusion was actually first reported with antegrade cerebral perfusion in which it was observed that it required a higher flow to open the vessels followed by a lower flow for maintenance. But as far as I know, I have not seen this reported for retrograde cerebral perfusion.

In terms of your comments about neuromonitoring, I think that this is a very important point. The use of some type of neuromonitoring device to confirm adequate cerebral perfusion remains critical. In addition to transcranial Doppler ultrasound, we also use bilateral cerebral oximetry monitoring. Unlike your institution, which uses the tympanic membrane temperature monitoring, we have settled on nasopharyngeal temperature monitoring. But taking all of this into consideration, I think it is very important to make sure that we are protecting the cerebrum as best we possibly can.

DR CHRISTOPHER KNOTT-CRAIG (Oklahoma City, OK): Erle Austin presented some data at this meeting a few years ago that demonstrated that after circulatory arrest, if you reperfused at a low temperature for a few minutes before rewarming, there was a benefit in terms of neurologic recovery. Have you used this at all in the adult population with aortic arch reconstructions, and do you have any comments about that?

DR ESTRERA: Thank you for your question. We do not purposefully reperfuse at the lowest temperature. What we do feel is that the rate of rewarming is important. Thus, inherently, we will perfuse at the lower temperature while we are rewarming. The rate of rewarming in our series is more or less prolonged because we warm until both the nasopharyngeal and bladder temperature of 36°C is reached. We, however, have not prospectively correlated this strategy with neurologic outcome.