Cerebral monitoring with transcranial Doppler ultrasonography improves neurologic outcome during repairs of acute type A aortic dissection

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Objective: Neurologic complications after repair of acute type A aortic dissection remain significant. The use of power M-mode transcranial Doppler monitoring to verify cerebral blood flow during these repairs might decrease cerebral ischemia by correcting malperfusion. The purpose of this study was to analyze the use of power M-mode transcranial Doppler monitoring during repairs of acute type A dissection with regard to neurologic outcome.

Methods: We performed a prospective study of patients undergoing repairs of acute type A aortic dissection. Repairs included profound hypothermic circulatory arrest and retrograde cerebral perfusion. Patients in whom transcranial Doppler monitoring was used to monitor cerebral blood flow and modify operative technique during repair (study group) were compared with those without monitoring and modification (control group).

Results: Between September 2001 and October 2003, we repaired 56 cases of acute type A dissection. Power M-mode transcranial Doppler monitoring was used in 50% (28/56) of cases. Power M-mode transcranial Doppler monitoring altered operative cannulation and guided retrograde cerebral perfusion flow in 28.5% (8/28) and 78.6% (22/28) of cases, respectively. Two patients presented with preoperative stroke, one in each group. One operative death occurred in each group. In-hospital mortality and the occurrence of new stroke were not significantly different between the 2 groups. Temporary neurologic dysfunction occurred less often in the study group (14.8% [4/27] vs 51.8% [14/27], $P = .008$).

Conclusions: Identification of cerebral malperfusion requires cerebral monitoring. By ensuring cerebral blood flow by using power M-mode transcranial Doppler monitoring and correcting cerebral malperfusion by modifying operative technique, neurologic outcome was improved during repairs of acute type A aortic dissection.

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Neurologic injury remains a significant problem during repairs of acute type A aortic dissection. The incidence of permanent neurologic injury (stroke) during these repairs ranges from 1% to 11% and is associated with increased early and late mortality.[1-7] Postoperative confusion, agitation, or delirium, collectively known as temporary neurologic dysfunction (TND), varies in incidence from 9% to 32%.[6-8] Although previously thought to be associated with minimal disability, TND has been demonstrated to have detrimental long-term consequences.[9] For this reason, differing techniques of cerebral protection have been devised for these complex aortic repairs.[5,10-13]

Prolonged focal ischemia caused either by cerebral emboli or malperfusion is responsible for stroke. The specific cause of TND is unclear but is likely associated with subclinical microemboli or generalized cerebral malperfusion. In a study evaluating profound hypothermic circulatory arrest (PHCA) during transverse aortic arch repair, the incidence of TND was directly related to the duration of cerebral ischemia, suggesting cerebral malperfusion and suboptimal cerebral protection.[14]

Malperfusion syndromes are common with acute aortic dissection.[3,4] With progression of the dissection flap, any aortic branch vessel can be involved. Furthermore, dissection flap occlusion or progression of dissection into the cerebral vessels can lead to cerebral malperfusion and ultimate neurologic injury. Differing methods of cerebral monitoring during complex aortic repairs have included 2-channel TCD, near-infrared spectroscopy (NIRS), electroencephalography (EEG), and jugular venous oxygenation monitoring.[12,15-19] We have recently reported our experience with power M-mode transcranial Doppler ultrasonography (PM-TCD) and its monitoring advantages during repairs of the ascending and transverse aortic arch.[20] We hypothesize that by monitoring cerebral blood flow with PM-TCD to identify and correct cerebral malperfusion, we can improve neurologic outcome during repairs of acute type A aortic dissection.

Materials and Methods

Study Cohort

The protocol of using PM-TCD for patients requiring any repair of the ascending and transverse aortic arch was approved by the University of Texas Houston and Memorial Hermann Hospital Committee for the Protection of Human Subjects. From September 2001 through October 2003, we repaired 56 consecutive cases of acute type A aortic dissection. Acute type A aortic dissection was defined as dissection involving the ascending aorta with the onset of chest pain within 2 weeks of repair. Diagnosis was made with either contrast computed tomographic angiography or transesophageal echocardiography. Once given diagnoses, all patients were offered urgent surgical intervention, and informed consent was obtained.

The use PM-TCD was determined on the basis of the availability of the device and the ultrasonographer (ZG). Twenty-eight patients underwent repair with PM-TCD monitoring (study group), and 28 patients were repaired without the use of PM-TCD monitoring (control group). EEG and NIRS were monitored in all patients.

Anesthetic Management

Anesthetic induction involved fentanyl (10-15 μg/kg), midazolam (0.05 mg/kg), and pancuronium (0.1 mg/kg). 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 0.5% to 1.0% isoflurane and an oxygen-air mixture. Transesophageal echocardiography was used, and hemodynamics were controlled to achieve a cardiac index of between 2.0 and 3.0 $L \cdot min^{-1} \cdot m^{-2}$. Serial arterial blood gas measurements were obtained, and the hematocrit level 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. All patients were monitored with 5-lead electrocardiography, peripheral pulse oximetry, end-tidal carbon dioxide measurement, temperature probes (nasopharyngeal, bladder, and blood), arterial line placement, and a pulmonary artery catheter.

Cerebral Monitoring

Patients were monitored with bilateral NIRS-INVOS (Somanetics, Troy, Mich), and a 10-lead EEG was used to monitor cerebral function. PM-TCD was performed by a neuroultrasonographer (ZG). Shortly after induction, the patient's head was fitted with a probe

TABLE 1. Cerebral malperfusion* and operative maneuvers performed

Stage of repair	Operative maneuver	Flow altered	Cases
Initiation of CPB	Alteration of arterial cannulation	Arterial	
	Contralateral femoral artery		2
	Axillary artery		1
Cooling-CPB	Warm arrest; fenestrate the dissecting membrane; reinitiate CPB	Arterial	2
PHCA-RCP	PM-TCD–guided RCP flow with altering of RCP perfusion	RCP	22
Warming-CPB	Deairing of the arch graft	Arterial	1
	Reposition of transverse arch arterial cannula (see Figure 1)		2

CPB, Cardiopulmonary bypass; *PHCA*, profound hypothermic circulatory arrest; *RCP*, retrograde cerebral perfusion; *PM-TCD*, power M-mode transcranial Doppler ultrasonography.

*Cerebral malperfusion is defined as a reduction in middle cerebral blood flow velocity, as determined by means of PM-TCD of 50% baseline.

TABLE 2. Patient variables*

Variable	Control group n = 28	Control group SD or %	Study group n = 28	Study group SD or %
Age (y)†	61	±12	58	±12
Male sex	21	75%	22	79%
Surgical start time‡				
Morning (6 AM-6 PM)	21	75%	15	54%
Evening (6 PM-6 AM)	7	25%	13	46%
Preoperative stroke	1	4%	1	
Preoperative Paraplegia	0	0%	1	
Hypotension	11	39%	9	32%
Tamponade	7	25%	8	29%
Myocardial infarction–ischemia	4	14%	5	18%
Aortic insufficiency	22	79%	16	57%
Preoperative Rx β-blockers	18	64%	22	79%
MABP controlled	18	64%	17	61%
Ascending aortic size (cm)†	5.2	±0.81	5.2	±0.93
Rupture	7	25%	7	25%
Replace ascending aorta	28	100%	27	96%
Replace hemiarch	17	61%	17	61%
Replace total arch	0	0%	0	
AV resuspension	27	96%	26	93%
AVR	1	4%	2	
Aortic root replac	0	0%	2	
RCP	28	100%	28	100%
IABP	2	7%	1	
PHCA time (min)	34.4	±10.1	33.0	±8.1
Cooling time (min)	26.6	±13.7	25.4	±8.5
Warming time (min)	83.0	±21.4	82.2	±16.5
High RCP flow†	0.56 L/min	±11	0.56 L/min	±0.9
Low RCP flow†	0.49 L/min	±0.05	0.49 L/min	±0.06
High RCP pressure†	26.7 mm Hg	±10.6	33.3 mm Hg	±7.1
Low RCP pressure†	23.1 mm Hg	±9.1	26.6 mm Hg	±5.3
RCP time (min)†	34.4	±10.2	33.0	±8.1
CPB time (min)†	159	±49	165	±61
AXC (min)†	98	±27	96	±26
PRBC (units)†	5.7	±5.3	6.5	±5.4
FFP (units)†	6.1	±4.5	7.6	±7.5
Cryo (units)†	3.5	±7.8	6.1	±9.9
Platelets (units)†	11.9	±7.2	12.6	±12.9
Lowest nasopharyngeal temperature†	17.7 (°C)	±2.0	16.7 (°C)	±2.1

MABP, Mean arterial blood pressure (<90 mm Hg); *AV*, aortic valve; *AVR*, aortic valve replacement; *RCP*, retrograde cerebral perfusion; *IABP*, intra-aortic balloon pump; *PHCA*, profound hypothermic circulatory arrest; *CPB*, cardiopulmonary bypass; *AXC*, aortic crossclamp time; *PRBC*, packed red blood cells; *FFP*, fresh frozen plasma; *Cryo*, cryoprecipitate.

*No significant differences between groups were noted, except with the RCP pressure high, with a *P* value of .008.

†Expressed as means ± SD. All other data are presented as raw counts and percentages.

‡The operations start in the morning between 6 AM and 6 PM or in the evening between 6 PM and 6 AM.

fixation head frame with a hands-free, standard, 2-MHz pulsed-wave TCD transducer (Spencer Technologies, Seattle, Wash) positioned on the temporal bone window for monitoring the middle cerebral artery (MCA) blood flow velocity (in centimeters per second). The mean flow velocities and pulsatility indices were obtained by using a single-channel spectral display at assumed zero angle of insonation at the depths represented by a yellow line on the PM-TCD screen display. Bilateral MCA blood flow velocities were monitored continuously. Any reduction in PM-TCD velocity to less than 50% of baseline was reported to the operating surgeon.

Operative Procedure

After systemic anticoagulation, cardiopulmonary bypass (CPB) was established by means of either right or left femoral artery

cannulation, depending on the patient's pulse status. If no pulse was palpable, then axillary artery cannulation was performed (1 patient of the study group). Venous cannulation was obtained through the superior vena cava and inferior vena cava or femoral vein. In the study group, if cerebral malperfusion was observed, then operative maneuvers were performed to improve cerebral blood flow (Table 1). In the control group our established standards of repair were maintained, and no modifications were performed.

Snares were applied to both the inferior and superior vena cavae for total CPB. Systemic cooling was initiated, and the patient's temperature was monitored with both nasopharyngeal and bladder temperature probes. Myocardial protection was achieved by 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 saver and pump suction were used for blood salvage.

Once the EEG was isoelectric, which coincided with a nasopharyngeal temperature of 15°C to 20°C, CPB was discontinued, and circulation was arrested. Retrograde cerebral perfusion (RCP) was begun through the superior vena caval cannula by using a centrifugal pump. For the control group, we used a conventional maximum flow rate of 500 mL/min in the RCP circuit to maintain the superior vena caval line pressure at less than 25 mm Hg. In the study group, using PMD-TCD to directly monitor cerebral flow in both MCAs, we increased or decreased RCP flow and pressure as necessary to maintain reversed MCA flow. PM-TCD monitoring identified any reversal of flow during RCP and provided a guide for optimal RCP flow.[20]

After completion of the distal aortic reconstruction, RCP was discontinued, and a cannula was placed into the new aortic graft. With the patient in the Trendelenberg 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 completed with aortic root reconstruction and aortic valve resuspension 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. The patient was weaned from CPB, systemic anticoagulation was reversed, and the patient was decannulated. PM-TCD monitoring was discontinued at the end of the procedure.

Outcome Variables

Study end points included 30-day and in-hospital mortality, stroke, and TND. Thirty-day mortality refers to deaths that occurred within 30 days of surgical intervention. In-hospital mortality refers to deaths that occurred during hospitalization. Stroke was defined as any gross focal neurologic brain injury, either temporary or permanent, identified on neurologic examination by a neurology consultant and confirmed with computed tomography or magnetic resonance imaging. Temporary confusion, delirium, agitation, disorientation, or altered mental status denoted TND in all patients who survived the operation, as defined by Ergin and colleagues.[9] We modified this classification by waiting 24 hours after complete reversal of anesthesia before the pronouncement of TND. This was determined by an attending neurologist who was blinded to whether PM-TCD monitoring was used. Acute stroke was excluded by means of computed tomographic scanning or magnetic resonance imaging of the head in all patients suspected of having TND. All patients with TND were followed for the entire hospitalization for ultimate resolution of dysfunction. Significant reduction in cerebral blood flow was designated as a 50% decrease in PM-TCD velocity in either MCA.

Data Analysis

Data were collected from chart reviews done by a trained nurse abstractor and were entered into a dedicated Microsoft Access database. Data were exported to SAS for analysis, and all computations were performed by using SAS version 8.02 running on Windows 2000. Within-subject comparisons of means were performed by using paired *t* tests or Wilcoxon signed-rank tests as appropriate, depending on distributional assumptions. Correlation analysis was performed by using Pearson product moment for normal data or Spearman rank correlations for nonnormally distributed data. The null hypothesis was rejected at a *P* value of less than .05.

Results

The 56 patients who were included in the analysis were well matched for relevant prognostic and surgical variables (Table 2).

Outcomes

In-hospital and 30-day mortalities for the entire cohort were the same, 21.4%. Mortality in the control group (8/28 [28.6%]) was not significantly different from that in the study group (4/28 [14%], $P = .21$). New stroke in the control group (2/27 [7%]) was not significantly different from that in the study group (0/27 [0%], $P = .16$). The incidence of TND was significantly reduced in the study group (PM-TCD) compared with in the control group (4/27 [14.8%] vs 14/27 [51.8%], respectively; $P < .008$). One preoperative stroke and one operative death occurred in each group, and analysis was adjusted accordingly.

Cerebral Monitoring

PM-TCD monitoring identified MCA antegrade cerebral blood flow during CPB and retrograde cerebral blood flow during RCP in all the study cases (100% of 28 cases). Mean PM-TCD flow velocity was 34.1 ± 21.4 cm/s during antegrade CPB and 16.2 ± 11.2 cm/s during PHCA and RCP in the study group. During CPB in the study group, significant decreases in PM-TCD MCA velocities leading to adjustments in arterial cannulation were observed in 28.6% (8/28) of cases (Table 1). Alteration of either arterial or RCP flow is listed in Table 1.

PM-TCD–guided Operative Modifications

In 3 study cases common femoral artery cannulation was changed to either the contralateral femoral artery (2 cases)

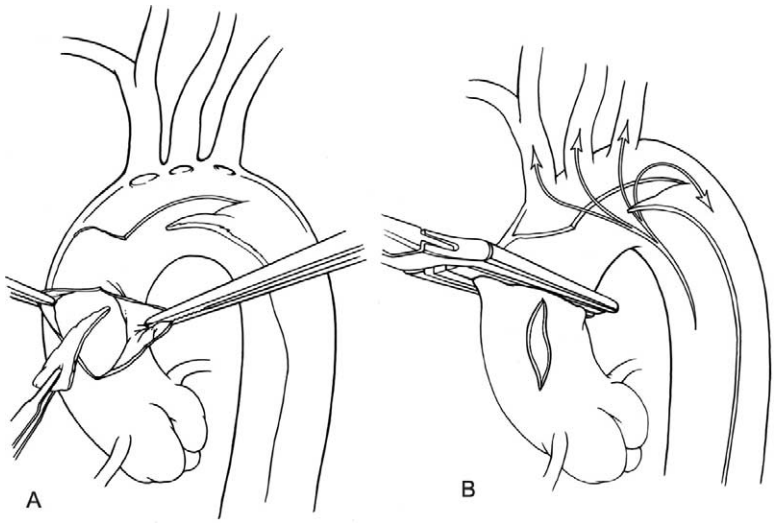

Figure 1. A, An open aortic fenestration is used after cooling is underway (25°C-30°C), and cerebral malperfusion is now identified. It is performed by briefly discontinuing CPB, placing the patient in the Trendelenberg position, performing a longitudinal aortotomy in the ascending aorta, and incising or fenestrating the dissecting membrane in the transverse aortic arch. B, The ascending aorta is clamped distal to the aortotomy, and CPB and systemic cooling are reinitiated. Retrograde arterial flow is now allowed to perfuse the cerebral vessels.

or the right axillary artery (1 case) at initiation of CPB when reduction in MCA blood flow velocity was noted by means of PM-TCD monitoring. Improvement of the MCA blood flow velocities was noted after the change in cannulation was performed. During the cooling phase of CPB, new episodes of cerebral malperfusion (not present at the initiation of CPB) were identified in 2 cases by means of PM-TCD monitoring that led to open aortic fenestration under brief moderate hypothermic circulatory arrest (Figure 1). A change in arterial cannulation could not be performed because the patient body temperature was 26°C and 28°C, respectively, in the 2 cases, and no cardiac output could be generated without CPB.

During the CPB warming phase of the procedure, after completion of the distal anastomosis, PM-TCD monitoring modified operative management in 3 cases. In one case excessive residual air in the transverse aortic arch graft was identified. This was identified on PM-TCD monitoring as a shower of high-intensity transient signals that were relieved by placing the patient in the Trendelenberg position and deairing the graft by briefly releasing the aortic crossclamp. In 2 cases direct cannulation of the transverse aortic arch graft led to right MCA malperfusion that was corrected by repositioning the arterial cannula. Because of the potential for cannula malpositioning, we ultimately converted to the branched arm polyester woven prosthetic grafts (Hemashield, Maple Grove, Minn) for transverse arch work.

During PHCA and RCP, 78.5% of the study group required modification of RCP flow to identify reversal of MCA blood flow. In addition, the RCP pressures in the study group were significantly higher than in the control group (33.3 $\pm$ 7.1 vs 26.7 $\pm$ 10.6, $P = .008$).

Multiple logistic regression analysis performed for TND revealed that the use of PM-TCD monitoring (adjusted odds ratio, 0.12; 95% confidence interval, 0.03-0.57; $P = .008$) and the preoperative use of β-blockers (anti-impulse therapy; adjusted odds ratio, 0.18; 95% confidence interval, 0.04-0.81; $P = .03$) were protective against TND. Rupture was the only independent factor predictive of TND (adjusted odds ratio, 6.30; 95% confidence interval, 1.12-35.32; $P = .04$).

Discussion

Cerebral malperfusion can occur at any point in the operative period during repairs of acute type A aortic dissection, leading to neurologic complications. The incidence of stroke and TND can be as high as 40%, with devastating long-term consequences.[1-8] It was hypothesized that identifying and correcting cerebral malperfusion would improve neurologic outcome during these repairs. In this study we demonstrated that during the course of operative repair, significant cerebral malperfusion occurred in 28.6% (8/28) of patients. In these 8 patients the operative procedure was modified to improve cerebral blood flow velocity, as determined by means of PM-TCD monitoring, and only one patient sustained a TND, with no patients experiencing a stroke in this subgroup. Because modifications were performed in this group, however, it is unclear what the outcome would have been if no maneuvers were performed. In addition, we defined a reduction of 50% of PM-TCD ve-

locity as a significant qualitative decrease in cerebral blood flow but were unable to quantify the actual blood flow. Regardless, the high incidence of cerebral malperfusion during repairs of acute type A aortic dissection remains concerning and might go undetected without any neurologic monitoring.

We recently reported our results with PM-TCD monitoring to guide RCP during repairs of the ascending and transverse aortic arch. In the previous study we identified that an opening RCP pressure was required to identify reversal of blood flow in the MCAs.[20] In addition, by using only standard RCP flow and pressure (0.5 L/min and <25 mm Hg), reversal of MCA blood flow was identified in only 20% of cases, and 80% of cases required some modification of RCP flow. Similar to this previous study, 78.5% of our study group required modification of RCP flow to identify reversal of MCA blood flow. In addition, the RCP pressures in the study group were significantly higher than in the control group (33.3 ± 7.1 vs 26.7 ± 10.6, $P = .008$). This higher pressure was the opening pressure required to identify reversal of MCA blood flow.[20] Maintenance of the RCP pressure at less than 25 mm Hg has been recommended because of the concern of cerebral edema that might be caused by higher pressures.[10] Despite the higher RCP pressures in the study group, worse neurologic outcomes were not observed.

TND is a term used to encompass a wide range of neurologic deficits encountered in the postoperative period. The incidence might vary from 9% to 40% for repairs of acute type A aortic dissection and is likely dependent on how the term is defined.[6-8,20] We have adopted the definition reported by Ergin and colleagues[9] that classifies dysfunction on the basis of type, duration, and time required to recover. Despite this classification system, identification and categorization of TND can still be quite subjective. For this reason, all patients with TND were examined by a neurologist, who used brain imaging to exclude stroke, bleed, or edema. We previously reported a TND incidence of 40% in cases of ascending and transverse aortic arch repair.[20] For the previous study, we declared any patient with confusion, agitation, or disorientation at any time during the hospitalization, including the early postoperative period, as sustaining TND. For this study, we have modified the definition by defining TND as any confusion, agitation, or disorientation 24 hours after complete reversal of anesthesia. With this definition, the incidence of TND in the control group remains high at 51% but was significantly reduced in the study group (14%, $P = .008$).

A previous study identified age, hypothermic circulatory arrest time, coronary artery disease, hypotension, and emergency procedure as predictors for TND.[6] In this study multiple logistic regression analysis revealed rupture ($P = .04$) as a predictor for TND and the use of preoperative β-blocker ($P = .03$) and PM-TCD monitoring ($P = .008$) as independent predictors protective against TND. The use of preoperative β-blockers delineated the group of patients started on anti-impulse therapy for the control of pain and blood pressure.

The benefits of anti-impulse therapy have been previously reported with aortic dissection.[21-23] The protective effect of anti-impulse therapy suggests the importance of preventing the progression of aortic dissection on outcome.

In addition to PM-TCD, 2-channel TCD monitoring and NIRS have been used for cerebral monitoring during complex aortic repairs.[12,15-18] The advantage of the multichannel PM-TCD monitoring is the larger window for detection compared with 2-channel TCD monitoring.[20,24] This allows for a higher sensitivity in the identification of cerebral blood flow. A limitation of this technology, however, is the necessary dependence on a skilled technician for the operation of the monitor. As with TCD in general, PM-TCD monitoring is also limited by patient variables. The use of PM-TCD monitoring assumes that an adequate temporal window is available. NIRS has also been used for cerebral monitoring during repairs requiring PHCA.[16,18] Most of these studies report use of NIRS to monitor cerebral oxygenation, with no attempts to modify operative techniques if a decrease is noted.[25] In this study we used NIRS to monitor bilateral cerebral oxygenation in both the control and study groups. For the purpose of this study, however, no operative modifications were made during the procedure on the basis of changes observed on NIRS. Analysis of NIRS data is ongoing.

In this study PM-TCD monitoring led to alterations in operative technique to resolve cerebral malperfusion. The maneuvers noted in Table 1 and described in Figure 1 have allowed us to respond to episodes of cerebral malperfusion and have ultimately modified our technique of repair. Although many have reported the feasibility and benefits of axillary cannulation for complex aortic repairs and recent reports have suggested a benefit of axillary cannulation over femoral cannulation,[26,27] the possibility of cerebral malperfusion still remains. This emphasizes the importance of cerebral monitoring and the verification of cerebral blood flow irrespective of the cannulation approach selected. Our experience with a malpositioned transverse arch arterial cannula leading to right-sided malperfusion has led us to adopt the use of the branched polyester woven grafts for most ascending and transverse arch repairs. We do admit that the clinical significance of this event is unknown, but the use of the presewn side arm graft has eliminated cerebral malperfusion from malpositioned arterial arch graft cannula, as identified by means of PM-TCD monitoring.

Our study should be viewed in light of certain limitations. The study design was nonrandomized and therefore subject to selection bias. As mentioned previously, we did

not enroll a true control group (ie, a group of patients who had PM-TCD monitoring but with no maneuvers performed). Thus the clinical significance of cerebral malperfusion as identified by PM-TCD monitoring is not entirely clear.

In addition, the use of PM-TCD monitoring was dependent on the availability of the neuroultrasonographer (ZG). To address the potential for bias, we compared all variables, including hypotension, rupture, tamponade, and the surgical start time, between groups and noted no significant differences (Table 2). Of interest, more evening repairs (6 PM to 6 AM) were performed in the study group than in the control group. Furthermore, multiple logistic regression analysis was performed for TND, and the use of PM-TCD monitoring was identified as an independent predictor protective against TND.

Because PM-TCD monitoring requires an ultrasonographer dedicated to monitoring, this technique is dependent on availability. To eliminate this limitation, we are currently training our anesthesiologist in PM-TCD monitoring. Other cerebral monitoring devices not dependent on the availability of a technician have been used (eg, NIRS), but further studies with regard to applicability are still required.

In conclusion, cerebral malperfusion can occur at any period during the repair of acute type A aortic dissection. Identification of cerebral malperfusion ultimately requires cerebral monitoring. By ensuring cerebral blood flow with PM-TCD monitoring and correcting cerebral malperfusion by modifying the operative technique, neurologic outcome was improved.

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Discussion

Dr Ross M. Ungerleider *(Portland, Ore).* This is an excellent study. It is a potentially useful modality for providing online

monitoring that can lead to interventions for patients with acute dissection, and it is certainly worth exploring the application of this. Although the data are intriguing, we do not have a true control group, that is, patients with similar PM-TCD findings but who receive no intervention. The placement into the control group was simply by availability of the device, and I think that should be noted. It does not detract, but it does raise questions for the future, and I think your NIRS data that are obtained in both groups of patients, those with and without interventions, will be useful and interesting.

The other feature that you have noticed is that it is a nonrandomized study. It is therefore open to selection bias, but you have adequately noted that, and we appreciate that.

I am wondering who determined the presence of transient neurologic deficits. Was that individual qualified to do this, and was he or she blinded to whether the patient was in the control or the study group?

Dr Estrera. The determination of TND was made by a neurologist who was blinded to whether we used TCD monitoring. The neurologist specifically who was part of the University of Texas neurology group identified the patient, examined the patient, performed a computed tomographic scan to rule out any obvious stroke, and then declared the occurrence of TND, assuming the patient did resolve before they were discharged.

Dr Ungerleider. I think it would be helpful to note that in the article because it does increase the validity of the findings.

I am curious why you used alpha-stat cooling. There have been increasing data correlating pH-stat cooling with improved brain protection, at least in infants and children, and I am wondering why in the adult population, where I do not have very much experience, alpha-stat continues to be the preferred cooling method.

Dr Estrera. I think the original concern about alpha-stat when compared with pH-stat was the increased cerebral blood flow (with pH-stat) and the potential increased risk of embolization in the adult population. Is that valid or not? I do not know. The work that Richard Jonas has done at Boston Children's Hospital in the pediatric population is very convincing, and we are actually investigating this approach at this time. I personally have some reservations about alpha-stat, although we still use alpha-stat because that is how I was trained by Dr Safi.

Dr Ungerleider. Your cannulation techniques that led to malperfusion are interesting because the decreased flow to the right cerebral artery with the cannula being perhaps too far into the aortic arch that you saw is something that we saw and have seen in most ductal-dependent systemic blood flow lesions, such as hypoplastic left heart syndrome. At Duke, my colleague, Bill Greeley, and I used to study several patients with that type of flow pattern by using radioactive xenon, and we did find that there was a diminishment of flow, but the effects of that really did not seem to make a difference in terms of the outcome for those patients. Therefore I am wondering, because TCD really measured velocity and not anything quantitative related to flow, can you really say anything about cerebral blood flow from these TCD data? It is interesting, but what does it mean?

Dr Estrera. I think one of the important points you brought up was the fact that we do not have a true control group. When performing this study, it was difficult for us to do nothing when we saw lack of blood flow to the brain with monitoring. What does it mean? Again, I stressed in our limitations that this type of monitoring is a qualitative study. It is something that I use in the operating room when we have a patient with acute type A dissection and I ask the ultrasonographer, Dr Garami, "Do I have blood flow?" Obviously knowing thin information treats me as a surgeon knowing that there is some cerebral blood flow, but what it means quantitatively, I really do not know, unless you do these other more elaborate studies.

Dr Ungerleider. You had very interesting observations regarding RCP. In fact, the majority of your intervention is almost 80% related to the alteration of your RCP techniques, and you suggest that effective RCP requires an adequate opening pressure to provide reversal of flow, as measured by means of TCD monitoring, and that makes sense. Now conventionally, this RCP pressure has been maintained at or around 25 mm Hg, and in your study group the mean high pressure that correlated with reversal of flow was 33 mm Hg compared with 26 mm Hg in the control group. Although it was impossible for the audience to read the flow rates, they were identical between the 2 groups, and I am wondering how you achieved the higher pressure in the study group at a similar flow rate.

Dr Estrera. What happens, and this was published in our study a couple of years ago using TCD monitoring for RCP, is that achievement of reversal of cerebral blood flow requires a higher opening flow pressure or the highest RCP pressure. Once this reversed flow is identified by means of TCD, then we actually dial down our RCP flows. The RCP flows that are listed are the averaged flow for the entire RCP period. Thus that is why the maintenance pressure ends up being about the same as your standard pressure of 25 mm Hg or flow of 500.

Dr Ungerleider. Then does this suggest that the conventional pressure of 25 mm Hg should be altered to 33 or 35 mm Hg, given the difficulty of performing PM-TCD monitoring related to the availability of trained personnel? It seems that the major contribution of your study is to liberalize the RCP high pressure limits for this group of patients.

Dr Estrera. You can liberalize the pressures, but what might be more important than the absolute pressure value is your ability to monitor cerebral blood flow. I remember you gave a talk at the Southern Thoracic last year about flying a plane with regard to safety in cardiac surgery, and again Dr Jonas had mentioned this in his discussion at the AATS. We do not want to fly a plane without any kind of monitor or fly it blindly, and that is the whole concept here. I am not saying that we should increase our pressures, but having some kind of device to verify that you have blood flow to the brain is what is most important, and therefore we do not fly blindly.

Dr Ungerleider. A final question. The other major finding of your study as I interpret this is that it confirms the limitations of RCP. There have been good data from Randy Griepp's group, as well as from our group and several others, that suggest that antegrade cerebral perfusion might be more effective in protecting the brain during periods of hypothermic circulatory arrest than techniques of either RCP or no perfusion at all, and therefore your use of PM-TCD monitoring allowed you to make RCP more effective, but would it even be useful if all you simply used was selective antegrade cerebral perfusion during that period of hypothermic arrest?

Dr Estrera. I do not know that answer specifically, but I think it could make antegrade cerebral perfusion more effective. I do not disagree with the work of Dr Griepp, your group, and a number of other groups who have used antegrade cerebral protection. Because our circulatory arrest times are respectively short, at about 30 to 33 minutes, our results are acceptable. If we extend that time beyond that, we might show the benefit of antegrade perfusion over RCP.

Dr R. Scott Mitchell *(Stanford, Calif).* I just have one quick comment. I think this is a great study, but I am really worried that you are missing one of the critical conclusions, and that is that femoral cannulation frequently cannot provide adequate cerebral circulation. I think there is a study by Dr Sabik coming out of the Cleveland Clinic now showing a very definite effect on improved outcomes with axillary artery cannulation in acute type A aortic dissections, which is quite compelling.

Dr Estrera. The point here, again as I concluded, is that whether you cannulate the groin, the axillary artery, or the ascending aorta, which I saw at the aortic symposium this past year from a group from Germany, what is most important is you have some kind of device to verify blood flow to the brain. I think that is most important because if you cannulate the axillary artery, there are still 25% to 33% of patients who do not have a complete circle of Willis, and therefore you might not perfuse the contralateral side of the brain if you cannulate the right axillary artery. You have to have some kind of monitoring device to verify blood flow to the brain in my opinion. Whether you do axillary or femoral cannulation might not matter as much.