Changes in Blood Flow Velocity in the Middle Cerebral Artery During Nonpulsatile Hypothermic Cardiopulmonary Bypass

Hiroshi Endoh, MD; Koki Shimoji, MD

Background and Purpose We evaluated the utility of blood flow velocity measurements by transcranial Doppler ultrasonography as a tool to indirectly measure cerebral perfusion during cardiopulmonary bypass.

Methods We simultaneously measured blood flow velocity in the middle cerebral artery and physiological variables in 18 patients undergoing cardiac surgery under hypothermic cardiopulmonary bypass in which pH and PaCO2 were managed with the alpha-stat acid-base strategy. We expressed blood flow velocity as a relative value of control obtained under normothermia and normocarbia before bypass. We also developed an original index, modified cerebral metabolic rate for oxygen, to estimate cerebral metabolic rate for oxygen, to estimate cerebral metabolic rate for oxygen.

Results Relative velocity was significantly (P<0.01) reduced during stable aortic cross-clamp compared with before bypass and was significantly (P<0.01) increased during rewarming compared with at aortic cross-clamp. Modified cerebral metabolic rate for oxygen significantly correlated with nasopharyngeal temperature during cooling, aortic cross-clamp, and rewarming (r=.756, P<.0001; r=.4, P<.01; r=.725, P<.0005, respectively). Calculated temperature coefficient for modified cerebral metabolic rate of oxygen was 2.7±1.4 (mean±SD, n=10) during cooling. Only nasopharyngeal temperature and PaCO2 were significant determinants of relative velocity during aortic cross-clamp.

Conclusions We can monitor cerebral perfusion and metabolism by measurements of relative velocity and modified cerebral metabolic rate for oxygen during hypothermic cardiopulmonary bypass. (Stroke. 1994;25:403-407.)

Key Words • blood flow velocity • cardiopulmonary bypass • cerebral metabolism • ultrasonics

Brain injury is one of the most serious complications of cardiac surgery under cardiopulmonary bypass (CPB). Continuous monitoring of cerebral blood flow (CBF) during CPB seems to be of great value since cerebral hypoperfusion or hyperperfusion is suggested as a possible etiologic factor in brain injury. However, measurement of CBF by the isotope washout technique is too invasive, expensive, and time-consuming to become a popular monitoring method during CPB.

Transcranial Doppler ultrasonography (TCD) can measure the blood flow velocity in intracranial basal vessels by using a 2-MHz pulsed wave that allows sufficient ultrasound to penetrate the cranial bone. Several reports have indicated a close relation between changes in CBF and changes in blood velocity measured by TCD in humans or animals. Thus, changes in blood flow velocity continuously measured by TCD have been shown to help in evaluating CBF and maintaining an adequate cerebral perfusion in some situations in which other clinical parameters cannot be assessed.

In the present study, simultaneous measurements of blood flow velocity in the middle cerebral artery (MCA velocity) by TCD and physiological variables during hypothermic CPB were performed to evaluate cerebral perfusion and metabolism in hypothermic nonpulsatile CPB.

Subjects and Methods

Patients and Anesthesia

After approval from the Hospital Committee on Human Research and after obtaining consent, 18 patients (mean±SD age, 49.8±18.2 years) undergoing elective open heart procedures were subjected to this study. Patients with clinical evidence of cerebrovascular disease or uncontrolled hypertension were excluded from this study. All patients were premedicated with morphine sulfate 0.1 to 0.2 mg/kg IM and scopolamine 0.015 mg/kg IM 90 minutes before the start of the operation. Anesthesia was induced and maintained with fentanyl 80±15 µg/kg, and ventilation was controlled with an air/oxygen mixture to maintain normocarbia. Diazepam 0.2 mg/kg was administered immediately before starting CPB. No anesthetics were administered during CPB.

After induction, a 4F, 13-cm-long, double-lumen catheter (Arrow International Inc, Reading, Pa) was inserted into the right jugular venous bulb for blood sampling and continuous monitoring of jugular venous bulb pressure. Postoperative confirmation of the appropriate location of the catheter tip was obtained by a plain skull x-ray. Rectal, esophageal, and nasopharyngeal temperatures were continuously monitored.

Management of Cardiopulmonary Bypass

After anticoagulation with 250 IU/kg heparin, nonpulsatile hypothermic CPB was carried out through an ascending aortic cannula using a standard roller pump (PEMCO Inc, Cleveland, Ohio), a hollow-fiber membrane oxygenator (Capiox II-36 or II-50, Terumo, Tokyo, Japan), and a 40-µm arterial filter (AF-1040C, Bentley, Irvine, Calif.). An activated clotting time was maintained at greater than 400 seconds by additional administration of heparin. Pump flow varied between 2.2 and

Received August 6, 1993; final revision received October 8, 1993; accepted October 20, 1993.

From the Department of Anesthesiology, Niigata City General Hospital (H.E.) and Niigata University School of Medicine (K.S.), Niigata, Japan.

Correspondence to Hiroshi Endoh, MD, Niigata City General Hospital, #2-6-1 Sitikuyama, Niigata 950, Japan.
2.4 L/min per square meter as determined by venous return. Mean arterial blood pressure (MABP) ranged between 50 and 100 mm Hg.

During hypothermic CPB, pH and Paco₂ were maintained at 7.40 and 40 mm Hg, respectively, as measured in a blood gas analyzer at 37°C (alpha-stat strategy). Blood samples were analyzed with a blood gas analyzer (ABL 300, Radiometer, Copenhagen, Denmark) and a spectrophotometer (OSM3, Radiometer, Copenhagen). Sodium bicarbonate was given to maintain a base deficit of not greater than 4 meq/L measured at 37°C.

Measurement of Middle Cerebral Artery Velocity

The TCD Transit II probe (Medasonics, Mountainview, Calif) was positioned over the right temporal bone window and anchored using a head harness so that the angle of insonation remained constant throughout the study. Doppler signals from the right middle cerebral artery (MCA) were identified and measured at a depth of 40 to 55 mm. Confirmation of signals from the MCA was performed by gate depth, direction of signals, and compression tests. The shift frequency spectra of the Doppler signals were converted into velocity by fast Fourier transformation and displayed on a video monitor. The time/mean velocity of MCA blood flow for 4.27 seconds was continuously calculated by the equipment. However, some artifacts generated from electric instruments in the operating department made automatic, continuous measurements of time/mean velocity inaccurate, and the mean MCA velocity was therefore calculated from systolic and diastolic velocities using the following formula: Mean MCA Velocity=Diastolic Velocity+(Systolic Velocity-Diastolic Velocity)/3.15

Before skin incision, after establishment of steady-state ventilation, three consecutive baseline measurements of the time/mean velocity in MCA (MCA velocity) were performed over 3 minutes at a Paco₂ of 38 to 42 mm Hg. The average of these three baseline measurements of MCA velocity was taken to represent the control value.

Measured Variables

Middle cerebral artery velocity, cerebral perfusion pressure (defined as MABP minus jugular bulb venous pressure), pump flow, and nasopharyngeal temperature were measured every 15 minutes. Arterial oxygen tension, Paco₂, arterial oxygen content, arterial hematocrit, jugular bulb venous oxygen tension, carbon dioxide tension, and oxygen content were measured at intervals of 30 to 60 minutes during the study. Because MCA velocity varied considerably from patient to patient, MCA velocity was also expressed as a relative value to the control (relative MCA velocity). Cerebral oxygen extraction ratio (defined as difference between arterial and jugular bulb venous oxygen content divided by arterial oxygen content) and an original index of cerebral metabolic rate for oxygen, the modified cerebral metabolic rate for oxygen (modified CMRO₂, defined as difference between arterial and jugular bulb venous oxygen content multiplied by relative MCA velocity), were calculated.

Statistical Analysis

During the study there were 566 measurements of MCA velocity and physiological variables, 157 paired analyses of arterial and jugular venous bulb blood gas, and 60 analyses of arterial blood gas. Control MCA velocity was obtained at a Paco₂ of 39.7±2.7 mm Hg (mean±SD).

All variables obtained throughout the study were divided into the following seven periods according to sampling time: prebypass, partial bypass (before aortic cross-clamp), total bypass (before aortic cross-clamp), aortic cross-clamp, total bypass (after aortic cross-clamp), partial bypass (after aortic cross-clamp), and postbypass. Variables during the postbypass period were obtained within 30 minutes after weaning from CPB. Multiple comparisons between each period were made using a one-way ANOVA with post hoc Fisher's protected least significant difference test. A simple linear regression technique was performed between variables. Further, in stable hypothermic CPB (during aortic cross-clamp), multiple stepwise linear regression analysis using the methods of ordinary least squares was regressed on 10 variables (cerebral perfusion pressure, pump flow, nasopharyngeal temperature, hematocrit, Paco₂, jugular bulb venous oxygen tension, difference between arterial and jugular bulb venous oxygen content, modified CMRO₂, cerebral oxygen extraction ratio, and duration of aortic cross-clamp) against relative MCA velocity for the identification of significant variables. Significance was assumed for a value of P<.05 in all comparisons. All data were expressed as mean±SD.

Results

All patients survived the operation without clinical evidence of cerebral complications. Table 1 shows surgical procedures and demographic and CPB data. There was a significant (r=.576, P<.0005) linear relation between relative MCA velocity and nasopharyngeal temperature during surgery (Fig 1). There was a highly significant (r=.756, P<.0005) exponential relation between modified CMRO₂ and nasopharyngeal temperature during cooling (Fig 2). The temperature coefficient (Q₁₀) for modified CMRO₂ between 37°C and 27°C was 2.7±1.4 (mean±SD, n=10) during cooling. There was a highly significant linear relation (r=.639, P<.0005) between jugular bulb venous oxygen tension and modified CMRO₂ during surgery (Fig 3). Table 2 summarizes nasopharyngeal temperature, MCA velocity (expressed in centimeters per second), relative MCA velocity, modified CMRO₂, cerebral perfusion pressure, hematocrit, Paco₂, jugular bulb venous oxygen tension, and cerebral oxygen extraction ratio in each period. During the aortic cross-clamp at 26.5±2.5°C of nasopharyngeal temperature, MCA velocity, relative MCA velocity, modified CMRO₂, and cerebral oxygen extraction ratio were significantly (P<.01) increased compared with prebypass values. On rewarming (during the partial bypass period), at 36.7±1.0°C of nasopharyngeal temperature, MCA velocity, relative MCA velocity, modified CMRO₂, cerebral oxygen extraction ratio were significantly (P<.01) increased, whereas jugular bulb venous oxygen tension was significantly (P<.01) reduced compared with aortic cross-clamp values. Relative MCA velocity significantly correlated with modified CMRO₂ during aortic cross-clamp (r=.406, P<.005) and on rewarming (r=.725, P<.0005).

During stable hypothermic CPB (during aortic cross-clamp), there was a significant linear relation between relative MCA velocity and nasopharyngeal temperature (r=.4, P<.0005), between relative MCA velocity and Paco₂ (r=.3, P<.005), and between relative MCA velocity and hematocrit (r=.3, P<.01). On the other hand, there was a poor relation between relative MCA velocity and cerebral perfusion pressure (r=.1, P=.1082) and between relative MCA velocity and duration of aortic cross-clamp (r=.1, P=.2112). Only nasopharyngeal temperature and Paco₂ were significant (P<.01) determinants for relative MCA velocity during aortic cross-clamp as determined by multiple stepwise linear regression analysis.

Discussion

Transcranial Doppler ultrasonography cannot measure flow in a vessel but can measure blood flow...
TABLE 1. Demographic Profiles, Surgical Procedures, and Cardiopulmonary Bypass Data

<table>
<thead>
<tr>
<th>Pt No.</th>
<th>Age, y</th>
<th>Sex</th>
<th>Surgical Procedures</th>
<th>Duration of CPB, min</th>
<th>Duration of ACC, min</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>38</td>
<td>M</td>
<td>ASD closure, MAP, TAP</td>
<td>154</td>
<td>89</td>
</tr>
<tr>
<td>2</td>
<td>34</td>
<td>F</td>
<td>ASD and VSD closure</td>
<td>116</td>
<td>64</td>
</tr>
<tr>
<td>3</td>
<td>39</td>
<td>F</td>
<td>ASD closure, TAP</td>
<td>79</td>
<td>19</td>
</tr>
<tr>
<td>4</td>
<td>79</td>
<td>F</td>
<td>AVR</td>
<td>224</td>
<td>123</td>
</tr>
<tr>
<td>5</td>
<td>59</td>
<td>M</td>
<td>AVR, MAP</td>
<td>272</td>
<td>155</td>
</tr>
<tr>
<td>6</td>
<td>49</td>
<td>M</td>
<td>MVR</td>
<td>254</td>
<td>155</td>
</tr>
<tr>
<td>7</td>
<td>66</td>
<td>M</td>
<td>CABG</td>
<td>211</td>
<td>88</td>
</tr>
<tr>
<td>8</td>
<td>52</td>
<td>M</td>
<td>AVR</td>
<td>175</td>
<td>95</td>
</tr>
<tr>
<td>9</td>
<td>33</td>
<td>M</td>
<td>VSD closure</td>
<td>111</td>
<td>54</td>
</tr>
<tr>
<td>10</td>
<td>54</td>
<td>F</td>
<td>AVR, MVR, TAP</td>
<td>278</td>
<td>192</td>
</tr>
<tr>
<td>11</td>
<td>62</td>
<td>M</td>
<td>ASD closure</td>
<td>100</td>
<td>30</td>
</tr>
<tr>
<td>12</td>
<td>59</td>
<td>M</td>
<td>CABG</td>
<td>189</td>
<td>110</td>
</tr>
<tr>
<td>13</td>
<td>65</td>
<td>F</td>
<td>CABG</td>
<td>268</td>
<td>133</td>
</tr>
<tr>
<td>14</td>
<td>10</td>
<td>F</td>
<td>VSD closure</td>
<td>65</td>
<td>30</td>
</tr>
<tr>
<td>15</td>
<td>58</td>
<td>M</td>
<td>CABG</td>
<td>261</td>
<td>140</td>
</tr>
<tr>
<td>16</td>
<td>65</td>
<td>M</td>
<td>CABG</td>
<td>252</td>
<td>140</td>
</tr>
<tr>
<td>17</td>
<td>59</td>
<td>M</td>
<td>CABG</td>
<td>219</td>
<td>103</td>
</tr>
<tr>
<td>18</td>
<td>15</td>
<td>F</td>
<td>PAPVR repair</td>
<td>228</td>
<td>117</td>
</tr>
</tbody>
</table>

Mean±SD 49.8±18.2 197±71.4 102.1±48.3

Pt indicates patient; CPB, cardiopulmonary bypass; ACC, aortic cross-clamp; ASD, atrial septal defect; MAP, mitral annuloplasty; TAP, tricuspid annuloplasty; VSD, ventricular septal defect; AVR, aortic valve replacement; MVR, mitral valve replacement; CABG, coronary-aortic bypass graft; and PAPVR, partial anomalous pulmonary venous return.

velocity. Because blood flow through a vessel is equal to the mean velocity multiplied by the cross-sectional area of the vessels, measurements of both velocity and segment vessel diameter are essentially needed to estimate blood flow. Estimation of CBF based on blood flow velocity measurements alone, therefore, represents a risky shortcut. However, several lines of evidence have shown that cerebral basal arteries are relatively constant in their diameter during changes in blood pressure, and that small arterioles play an important role in keeping CBF (pressure-flow autoregulation) constant by changing their calibers through the vasomotor action of smooth muscle. Experimental studies have shown that the brain parenchymal small arterioles (with diameters <40 μm) mainly dilate against changes in blood pressure, whereas arterioles with diameters of 322 μm increase their calibers only minimally. If these findings are extrapolated to the human MCA, which is larger than 1000 μm in diameter, it is conceivable that...
the MCA may not change its diameter during changes in blood pressure. Thus, we can estimate the changes in CBF by measuring changes in MCA velocity (relative MCA velocity) without simultaneous measurement of cross-sectional area.

Hypocarbia is well known to cause cerebral vasospasm, while hypercarbia causes vasodilatation. However, Huber and Handa20 showed that the calibers of the basal large arteries remain constant during changes in Paco2. Moreover, Markwalder et al21 found that the same CO2 reactivity in changes of MCA velocity and CBF in response to changes of end-tidal Pco2 in the range of 22 to 50 mm Hg. They indicated that the vasomotor action is confined to small arterioles but not to cerebral basal arteries. These findings demonstrate the relatively constant diameter of the MCA during changes in Pco2.

Furthermore, several studies indicate that changes in blood flow velocity are well correlated with changes in CBF in humans or animals. So far, a significant correlation between them has been shown by Bishop et al15 (r=0.89), Hansen et al16 (r=0.86), Werner et al17 (r=0.82), Lindegaard et al18 (r=0.94), Stump et al19 (r=0.90), Dahl et al20 (r=0.63), Hasley et al11 (r=0.69), and Werner et al12 (r=0.68). Thus, these observations support the validity of measurement of MCA velocity as an index of changes in CBF.

There have been two options for blood gas management during hypothermic CPB. One option, termed pH-stat strategy, maintains Paco2 and pH at 40 mm Hg and 7.50, respectively, after correction for body temperature (temperature corrected). The alternative, alpha-stat strategy, maintains Paco2 and pH at 40 mm Hg and 7.40, respectively, as measured in the blood gas analyzer at 37°C (temperature noncorrected). Blood gas management with pH-stat strategy results in increased Paco2 and CBF over cerebral metabolic demand. Therefore, both the coupling of CBF/CMRO2 (metabolic autoregulation) and flow-pressure autoregulation might not be preserved in pH-stat. With alpha-stat strategy, CBF is dependent on CMRO2, but not cerebral perfusion pressure. Both metabolic and pressure-flow autoregulation are preserved during moderate hypothermic CPB.22

In the present study both MCA velocity and relative MCA velocity significantly decreased in correlation with nasopharyngeal temperature during hypothermic CPB. This observation is consistent with the recent study by Hillier et al23 who reported a close correlation (r=0.78) between relative blood flow velocity and temperature in neonates or infants undergoing cardiac surgery. In contrast, Lundar et al24 reported that MCA velocity measured by TCD markedly increased in 10 of 11 patients (range, 80% to 300% of the prebypass values) during stable hypothermic CPB. Von Reutern et al25 also reported an increase in MCA velocity during stable CPB (127% of the prebypass values). These quite divergent observations of changes in MCA velocity may be explained on the basis of different blood gas management during hypothermic CPB. The alpha-stat strategy was applied in both Hillier et al23 and the present study, whereas Lundar et al24 and Von Reutern et al25 applied

![Graph showing relation between modified cerebral metabolic rate of oxygen (CMRO2) and jugular bulb venous oxygen tension throughout surgery.](image)

**FIG 3.** Scatterplot shows relation between modified cerebral metabolic rate for oxygen (CMRO2) and jugular bulb venous oxygen tension during surgery. Note that a linear relation was found between modified CMRO2 and jugular bulb venous oxygen tension throughout surgery.

### TABLE 2. Changes in Blood Flow Velocity in Middle Cerebral Artery and Physiological Variables During Surgery

<table>
<thead>
<tr>
<th>Variables</th>
<th>Prebypass</th>
<th>Partial Bypass*</th>
<th>Total Bypass*</th>
<th>ACC</th>
<th>Total Bypass*</th>
<th>Partial Bypass*</th>
<th>Postbypass</th>
</tr>
</thead>
<tbody>
<tr>
<td>NPT, °C</td>
<td>35.6±0.8</td>
<td>33.3±2.5†</td>
<td>30.3±1.5‡</td>
<td>28.5±2.5‡</td>
<td>33.7±3.1†‡</td>
<td>36.7±1.0‡</td>
<td>36.4±0.7†‡</td>
</tr>
<tr>
<td>MCA velocity, cm/s</td>
<td>46.4±19.5</td>
<td>51.9±23.5§</td>
<td>46.2±19.1‡‡</td>
<td>34.3±12.4‡</td>
<td>48.5±21.3‡‡</td>
<td>61.9±27.4‡‡</td>
<td>55.4±32.2‡‡</td>
</tr>
<tr>
<td>Relative MCA velocity</td>
<td>1.0±0.2</td>
<td>1.0±0.3‡</td>
<td>0.9±0.2‡</td>
<td>0.7±0.2‡</td>
<td>1.0±0.3‡</td>
<td>1.2±0.3‡</td>
<td>1.1±0.3‡</td>
</tr>
<tr>
<td>Modified CMRO2</td>
<td>5±1.5</td>
<td>2.6±1.4†</td>
<td>2.2±1.6†</td>
<td>1.8±1.1†</td>
<td>3.9±2.6†</td>
<td>5.1±2.3‡</td>
<td>4.3±2.0†</td>
</tr>
<tr>
<td>Cerebral perfusion pressure, mm Hg</td>
<td>76.7±11.4</td>
<td>58.1±14.4†</td>
<td>63.3±10.8‡‡</td>
<td>56.0±15.3‡</td>
<td>50.3±14.1‡‡</td>
<td>52.7±13.4‡‡</td>
<td>61.0±14.6‡‡</td>
</tr>
<tr>
<td>Hematocrit, %</td>
<td>33.3±4.0</td>
<td>22.8±3.9†</td>
<td>22.4±3.4†</td>
<td>22.5±3.5†</td>
<td>20.4±4.1†</td>
<td>20.7±3.6†</td>
<td>27.0±5.1†</td>
</tr>
<tr>
<td>Paco2, mm Hg</td>
<td>38.3±6.8</td>
<td>34.3±4.8‡</td>
<td>35.3±3.8‡</td>
<td>37.3±4.0</td>
<td>37.0±3.8‡</td>
<td>34.2±3.1‡</td>
<td>40.5±5.7‡</td>
</tr>
<tr>
<td>Jugular venous oxygen tension, mm Hg</td>
<td>40.9±6.5</td>
<td>43.1±11.7</td>
<td>45.8±9.5</td>
<td>47.6±12.3†</td>
<td>38.1±10.4‡</td>
<td>33.1±6.1‡</td>
<td>41.5±11.9‡</td>
</tr>
<tr>
<td>Cerebral oxygen extraction ratio</td>
<td>0.3±0.1</td>
<td>0.3±0.1</td>
<td>0.2±0.1</td>
<td>0.2±0.1</td>
<td>0.3±0.1</td>
<td>0.4±0.1</td>
<td>0.3±0.1</td>
</tr>
</tbody>
</table>

Values are mean±SD. B or A indicates before and after aortic cross-clamp (ACC), respectively; NPT, nasopharyngeal temperature; MCA, middle cerebral artery; and CMRO2, cerebral metabolic rate of oxygen.

*P<.05, †P<.01 significantly different from prebypass values.

‡P<.05, §P<.01 significantly different from values during ACC.
the pH-stat strategy. Furthermore, we demonstrated that relative MCA velocity significantly correlated with modified CMRO₂ but not with cerebral perfusion pressure in hypothermic CPB. This finding is consistent with the observation that both metabolic and pressure-flow auto-regulation are preserved under the alpha-stat strategy during moderate hypothermic CPB.

An index of modified CMRO₂ (defined as difference between arterial and jugular bulb venous oxygen contents multiplied by relative MCA velocity) was calculated to estimate CMRO₂. There was a close correlation (r=0.756) between modified CMRO₂ and nasopharyngeal temperature during cooling. There was also a close relation (r=0.639) between modified CMRO₂ and jugular bulb venous oxygen tension during surgery. The mean Q₀ in the present study (2.7) is also comparable to that (2.8) in the study of Kent and Peirce. These findings suggest that modified CMRO₂ is a valuable index for evaluation of changes in CMRO₂.

In regard to variables affecting relative MCA velocity during stable hypothermic CPB, nasopharyngeal temperature and Paco₂ were found to be significant determinants for relative MCA velocity as demonstrated by multiple stepwise linear regression technique in the present study. These two determinants are consistent with the study by Govier et al, who found that nasopharyngeal temperature and Paco₂ were the only factors that significantly influenced regional CBF measured by the xenon-131 clearance method during nonpulsatile hypothermic CPB. These findings indicate that the physiological variables that correlate with regional CBF are the same physiological variables that also correlate with relative MCA velocity during hypothermic CPB.

In summary, we demonstrated that relative MCA velocity correlated well with nasopharyngeal temperature but not with cerebral perfusion pressure, and that the coupling of relative MCA velocity and modified CMRO₂ remained intact under blood gas management with the study by Govier et al. These two determinants are consistent with the present study. These two determinants are consistent with the study by Govier et al, who found that nasopharyngeal temperature and Paco₂ were the only factors that significantly influenced regional CBF measured by the xenon-131 clearance method during nonpulsatile hypothermic CPB. These findings indicate that the physiological variables that correlate with regional CBF are the same physiological variables that also correlate with relative MCA velocity during hypothermic CPB.

Thus, changes in CBF and CMRO₂ during hypothermic CPB can be evaluated by the measurement of relative MCA velocity and modified CMRO₂ with simplicity and minimum invasiveness.

References

Changes in blood flow velocity in the middle cerebral artery during nonpulsatile hypothermic cardiopulmonary bypass.
H Endoh and K Shimoji

Stroke. 1994;25:403-407
doi: 10.1161/01.STR.25.2.403

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://stroke.ahajournals.org/content/25/2/403

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Stroke can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Stroke is online at:
http://stroke.ahajournals.org//subscriptions/