Transcranial Doppler Ultrasonography During Cardiopulmonary Bypass in Patients With Severe Carotid Stenosis or Occlusion

Gerhard-Michael von Reutern, MD, Andreas Hetzel, MD, Dietrich Birnbaum, MD, and Volker Schlosser, MD

Blood flow velocity of the middle cerebral artery was monitored during cardiopulmonary bypass procedures by means of transcranial Doppler ultrasonography. Our investigation was carried out in a group of 16 patients with severe carotid stenosis or occlusion and in a control group of 42 patients with no or stenosis of <50% local diameter reduction. After onset of cardiopulmonary bypass, both groups showed a short unstable phase followed by increased blood flow velocity (10% increase ipsilateral to the obstruction, 27% increase in the control group). Just before rewarming, blood flow velocity was still comparable to (control group -3%) or higher than (ipsilateral to obstructions +14%) prebypass values. Analysis of three patients with postoperative diffuse encephalopathy did not reveal reduced blood flow during cardiopulmonary bypass as a relevant factor. Two of the three showed luxury perfusion. Reduced perfusion due to carotid obstruction was not observed during cardiopulmonary bypass and therefore cannot be considered a significant risk factor for the development of intraoperative stroke. (Stroke 1988;19:674-680)

Neurologic and psychiatric disorders associated with cardiopulmonary bypass (CPB) are serious complications. Four percent (2-12%) of nonfocal and 2% (1-6%) of focal neurologic deficits1-2 have been reported to occur after coronary artery bypass grafting (CABG). Cerebrovascular disease especially seems to pose a risk for the development of focal cerebral deficits.3 Low-pressure phases of CPB are one possible factor for this danger, but, if the results of several publications are combined,2-4 no significant differences can be seen in the stroke rate after CABG in patients with or without carotid obstructions of ≥50% local diameter reduction. The purpose of our study with transcranial Doppler ultrasonography (TCD) monitoring was to determine the changes in middle cerebral artery (MCA) blood flow during nonpulsatile CPB in patients with severe stenosis or occlusion of the internal carotid artery (ICA).

In several studies cerebral blood flow was measured during CPB in patients without carotid lesions. The xenon-133 washout technique was used in most studies.2-9 Diverging results of changes in regional cerebral blood flow during CPB were obtained. The inferior time resolution is a disadvantage of this technique. In contrast, TCD offers a method for continuous evaluation of the cerebral circulation.

Subjects and Methods

Between April 1985 and June 1986 TCD monitoring was done during 69 operations (64 CABGs, 3 CABGs with valve replacement, 2 valve replacements) with extracorporeal circulation. Patients were classified into three groups based on prebypass extracranial carotid artery findings: Group 1, with unilateral severe stenosis (n = 6) or occlusion (n = 12); Group 2, with unilateral moderate stenosis (n = 7); or Group 3, with no (n = 37) or mild stenosis (n = 7). To obtain clear-cut groups we eliminated Group 2 from the final analysis.

In nine cases in Group 1, carotid and cardiac operations were performed in a single procedure (eight before sternotomy, one before cannulation of the heart). The patients were classified in accordance with the postendarterectomy status of their extracranial brain vessels; contralateral to the operated side we found four occlusions (Group 1) and five mild stenoses of the carotid artery (Group 3). In six of the nine patients, history and preoperative examination revealed ipsilateral transient ischemic attacks (TIAs) (n = 5) or amaurosis fugax (n = 1).

Carotid reconstruction could not be performed in two symptomatic patients, who had an occlusion and an unoperable severe ICA siphon stenosis, respectively (Group 1). Four (two in Group 1, two in Group 3) of the 69 patients were excluded from the final analysis; TCD examination was either incomplete or impossible, mainly due to difficulties in transmitting sound waves through the temporal bone.

Preoperative Examinations

Each operation was preceded by an examination of the extracranial arteries with a 4-MHz continuous-wave Doppler device (Sonotechnik, München, F.R.G.). In the case of a pathologic result, a duplex
scan examination (Ultramark 8, ATL, Bellevue, Washington) and TCD (prototype, EME, Ueberlingen, F.R.G.) were also performed.

Carotid artery stenosis was quantified by continuous-wave Doppler ultrasonography and duplex scanning using direct and indirect criteria including systolic maximum frequency measurements with spectral analysis\(^{10}\) (Sonacolor CD spectrum analyzer, Carolina Medical Electronics, King, North Carolina). Local narrowing of the vessel diameter was categorized as mild (\(\leq 50\%\)), moderate (\(<80\%\)), or severe (\(\geq 80\%\)).

Selective transfemoral catheter angiography was performed in Group 1 patients to document the lesion. Ultrasound diagnosis was confirmed in every case.

**Operative Monitoring**

Doppler frequency shift was obtained from the MCA using a transcranial pulsed-wave Doppler device (EME, TC2-64, range-gated, 2-MHz emission frequency). The method has been described.\(^{11,12}\) The Doppler probe was positioned freehand on the relatively thin temporal bone above the zygomatic arch and, in most cases, just anterior to the ear. Blood flow in the MCA is directed toward the probe; the anterior cerebral artery (ACA) shows blood flow away from the probe. To confirm insonation of the MCA, the signal must be shown from gate position 40 to 50 mm. For monitoring purposes a position of the sample volume was chosen, which showed MCA signals as close as possible to the origin of the ACA. Measurements were performed at short intervals of about 6 minutes during stable phases but continuously during critical maneuvers.

In all but two patients with at least moderate carotid stenoses or occlusion, the left and right MCA were recorded; in Group 3 only one side was examined. Simultaneous bilateral recording was not available. The side-to-side comparison was achieved by examining sequentially the right and left sides, which was in general possible within 2 minutes. Recordings of the different sides were performed at equal blood pressures. A rigid protocol could not be applied because of some artifacts producing surgical interventions (e.g., electrocautery).

The Doppler signals were documented by stereo tape-recording and evaluated off-line with a frequency spectrum analyzer (Sonacolor CD). The time-mean of the envelope curve of the Doppler spectrum of the MCA over 10 seconds was calculated. We defined six phases: I, before anesthesia; II, during anesthesia under conditions of stable circulation; III, at beginning of CPB, first steady state; IV, during CPB just before rewarmin; V, last recording during CPB; and VI, after CPB under conditions of stable circulation. Phase II was taken as the prebypass reference, and results of frequency measurements during the later phases were expressed as percent of the reference value.

Microembolic events can be detected by ultrasound.\(^{13,14}\) Short, high-intensity (overmodulated) signals within the Doppler shift spectrum were classified as microembolic events. Oxygen bubbles, however, cannot be differentiated from solid particles.

Doppler results were correlated with mean arterial pressure minus central venous pressure (MAP – CVP), measured with fluid pressure transducers; temperature-corrected Paco\(_2\) and venous oxygen with saturation (Pvo\(_2\)/sat) and hemoglobin, measured with microelectrodes (Radiometer, Copenhagen, Denmark); and rectal temperature.

Anesthesia was induced and maintained with flunitrazepam, fentanyl, pancuronium, and nitrous oxide. Bubble or membrane oxygenators of various types were...
FIGURE 1. Mean values + SD of transcranial Doppler monitoring in controls. Group 3 patients with carotid stenosis of ≤50%. For definition of phases see "Subjects and Methods." MAP–CVP, mean arterial pressure minus central venous pressure.

used. The regimens for nonpulsatile CPB were: priming volume free of blood, arterial and venous filters, moderate hypothermia, flow rate between 2.2 and 2.4 l/min/m², mean arterial pressure >60 mm Hg (especially in Group 1 patients) Paco₂ between 35 and 45 mm Hg, Pvo₂ >40 mm Hg, and venous saturation >70%.

Statistics

After Levene's test for equal variances, the paired t test was used to compare those variables within a group; between groups the unpaired t test was employed.

Data on age, sex, duration of CPB, and postoperative outcome are summarized in Table 1.

Results

Transcranial Doppler Monitoring During Stable Phases of Cardiopulmonary Bypass

The average finding in the control group (Figure 1) was a 27% increase in frequencies of the MCA during Phase III compared with Phase II. During Phase IV a 3% reduction, during Phase V a 23% increase, and during Phase VI a 20% increase were found. The differences in single variables were significant between Phase II and Phases III, V, and VI (Table 2).

In Group 1, the average MCA Doppler shift ipsilateral to a high-grade extracranial obstruction also increased during all phases with hemodilution (Phase III, 10%; Phase IV, 14%; Phase V, 18%; and Phase VI, 20%). A significant difference between MCA blood velocities on the ipsilateral side in Group 1 and Group 3 was found only during Phase III (10% vs. 27%, p < 0.01). A marked reduction of blood flow velocities in the MCA was never observed, even in the presence of ICA occlusion. Contralateral to the extracranial obstruction, the increase of blood flow velocity during CPB was more pronounced (Phase III, 19%; Phase IV, 16%; Phase V, 31%; and Phase VI, 22%) (Figures 2 and 3; Table 3).

Changes in Cerebral Perfusion During Cardiopulmonary Bypass

The following deals especially with observations made during short phases of lowered blood pressure, which occurred only with the initiation of CPB. There

<table>
<thead>
<tr>
<th>Table 2. Transcranial Doppler Monitoring in Patients With Carotid Stenosis of ≤50% Undergoing Cardiopulmonary Bypass</th>
</tr>
</thead>
<tbody>
<tr>
<td>Phase of operation</td>
</tr>
<tr>
<td>Number</td>
</tr>
<tr>
<td>MAP–CVP (mm Hg)</td>
</tr>
<tr>
<td>Doppler frequency time-mean (kHz)</td>
</tr>
<tr>
<td>Rectal temperature (°C)</td>
</tr>
</tbody>
</table>

Values are mean±SD. MAP–CVP, mean arterial pressure minus mean central venous pressure.

*†p<0.001, p<0.01, respectively, different from Phase II by Student's t test (differences in single variables).
was a wide range of pressure decrease during the first 5–10 minutes, in addition to the decrease of blood pressure caused by the initiation of CPB. This is a prominent feature due to hemodilution resulting from the use of nonblood pump prime. In half the patients such a decrease occurred a few minutes after the onset of CPB, almost simultaneous with cross-clamping of the aorta. In both Groups 3 and 1, a pressure decrease to < 50 mm Hg was observed with the same frequency (43% vs. 44%) and duration (mean approximately 3 minutes).

During such pressure decreases there were no differences in variation of MCA blood flow velocities between Groups 1 and 3 (Figure 4).

Correlation of Intraoperative Doppler Shift With Other Parameters

Several factors, such as hemodilution, hypothermia, hypocapnia, and relative pump flow, influence the blood flow velocity of the MCA. These parameters were recorded in Group 3 as well as in Group 1. We found only minor differences in these factors in both groups (Table 4).

Results in Patients With Poor Outcome

In three patients postoperative neurologic deficits were observed. Longer intraoperative hypotension or reduction of blood flow velocities in the MCA was not seen in these patients.

In one patient with bilateral obstructions (moderate stenosis on one side, occlusion on the other) a postoperative transient nonfocal neuropsychiatric deficit occurred. In this case coronary revascularization and valve replacement were performed, with the longest pump time and aortic cross-clamp time of all the patients (176 and 103 minutes, respectively). In addition, a period of hypotension (80/50 mm Hg) before the onset of CPB was registered. The MCA Doppler shift during CPB was equal to or slightly higher than that during Phase II.
In the remaining two cases, we saw clearly increased Doppler shift values and frequent microemboli during the entire CPB. One patient without extracranial lesions showed transient nonfocal neuropsychiatric complications with agitation and anxiety; the other, with mild carotid stenosis on the left and moderate stenosis on the right (Group 2, not included in Table 1) suffered severe diffuse neurologic deficit and died postoperatively. Figure 5 shows the results of TCD monitoring in this patient on the side of moderate stenosis.

Discussion

The risk of stroke posed by carotid obstructions could be due to diminished blood supply in the territory of the diseased artery during CPB. Therefore, we monitored MCA blood velocity during CPB in patients with and without extracranial carotid disease.

TCD does not measure cerebral blood flow quantitatively, but changes in blood flow reflected by changes in flow velocity can be recorded reliably because the diameter of the basal cerebral arteries does not change qualitatively, but changes in blood flow reflected by changes in Doppier shift of the MCA may occur ipsilateral or contralateral to the occlusion or in the ICA. A close relation was observed between the two. Therefore, variations in Doppler shift of the MCA reflect variations in blood flow volume.

Marked reduction of blood flow ipsilateral to severe carotid obstruction was not observed in our patients. In half the patients a drop of arterial pressure to <50 mm Hg was seen during the first 5–10 minutes of CPB. Only then did transiently diminished cerebral perfusion occur ipsilateral or contralateral to the occlusion or in Group 3. Reduction of Doppler shift in the MCA to half the Phase II values during this critical phase was observed only once in Group 1 and three times in Group 3. It can hardly be assumed that these reductions of blood flow lasting only a few minutes cause a focal ischemic deficit.

During Phase III, blood flow was greater than during Phase II in both Groups 1 and 3 despite the decrease of pressure to a range of 37–80 mm Hg. This increase, however, was less on the side of the stenosed or occluded carotid artery. Increased blood flow during cooling can be considered luxury perfusion.

Lundar et al performed simultaneous recordings of MCA blood flow velocity with TCD, and ipsilaterally and contralaterally they electromagnetically measured blood flow in the ICA. A close relation was observed between the two. Therefore, variations in Doppler shift of the MCA reflect variations in blood flow volume.

Table 3. Transcranial Doppler Monitoring in Patients With Carotid Stenosis of ≥80% or Occlusion Undergoing Cardiopulmonary Bypass

<table>
<thead>
<tr>
<th>Phase of operation</th>
<th>II</th>
<th>III</th>
<th>IV</th>
<th>V</th>
<th>VI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number</td>
<td>16</td>
<td>16</td>
<td>16</td>
<td>16</td>
<td>16</td>
</tr>
<tr>
<td>MAP−CVP (mm Hg)</td>
<td>76.8 ± 17.4</td>
<td>58.8 ± 18.8</td>
<td>80.1 ± 18.6</td>
<td>65.1 ± 13.2</td>
<td>72.1 ± 13.2</td>
</tr>
<tr>
<td>Doppler frequency-time-mean (kHz)</td>
<td>0.98 ± 0.21</td>
<td>1.07 ± 0.33</td>
<td>1.10 ± 0.47</td>
<td>1.14 ± 0.36</td>
<td>1.16 ± 0.39</td>
</tr>
<tr>
<td>Ipsilateral</td>
<td>1.15 ± 0.22</td>
<td>1.37 ± 0.40</td>
<td>1.32 ± 0.51</td>
<td>1.48 ± 0.28</td>
<td>1.39 ± 0.34</td>
</tr>
<tr>
<td>Contralateral</td>
<td>35.9 ± 0.5</td>
<td>33.8 ± 0.8*</td>
<td>30.8 ± 1.3</td>
<td>34.9 ± 1.3</td>
<td>36.1 ± 0.5</td>
</tr>
</tbody>
</table>

Values are mean ± SD. MAP−CVP, mean arterial pressure minus mean central venous pressure.

Table 4. Parameters Influencing Middle Cerebral Artery Blood Flow in Patients Undergoing Cardiopulmonary Bypass

<table>
<thead>
<tr>
<th>Phase of operation</th>
<th>II</th>
<th>III</th>
<th>IV</th>
<th>V</th>
<th>VI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group 3, carotid stenosis of ≥50%</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hb (g/dl)</td>
<td>12.1 ± 1.9</td>
<td>10.2 ± 1.7*</td>
<td>9.8 ± 1.3*</td>
<td>10.3 ± 1.5*</td>
<td>9.9 ± 1.5</td>
</tr>
<tr>
<td>Pao2 (mm Hg)</td>
<td>33.9 ± 3.9</td>
<td>37.2 ± 6.8*</td>
<td>33.4 ± 6.3</td>
<td>36.8 ± 4.4*</td>
<td>35.8 ± 4.7</td>
</tr>
<tr>
<td>Pvo2 mm Hg</td>
<td>44.1 ± 11.2</td>
<td>36.4 ± 7.8</td>
<td>39.7 ± 7.8</td>
<td>42.1 ± 8.4</td>
<td>42.3 ± 8.4</td>
</tr>
<tr>
<td>% saturation</td>
<td>86.1 ± 8.3</td>
<td>81.5 ± 6.7</td>
<td>72.4 ± 8.4</td>
<td>42</td>
<td>42.3 ± 8.4</td>
</tr>
<tr>
<td>Relative flow (l/min/m²)</td>
<td>2.4 ± 0.3</td>
<td>2.3 ± 0.2</td>
<td>2.3 ± 0.2</td>
<td>2.3 ± 0.2</td>
<td>2.3 ± 0.2</td>
</tr>
<tr>
<td>Oxygen microemboli</td>
<td>15/42</td>
<td>10/42</td>
<td>19/42</td>
<td>42</td>
<td>42</td>
</tr>
<tr>
<td>Group 1, carotid stenosis of ≥80%</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hb (g/dl)</td>
<td>11.6 ± 1.4</td>
<td>8.8 ± 1.3*</td>
<td>9.1 ± 1.9</td>
<td>9.3 ± 1.5</td>
<td>8.3 ± 0.7</td>
</tr>
<tr>
<td>Pao2 (mm Hg)</td>
<td>34.1 ± 7.2</td>
<td>34.4 ± 5.9</td>
<td>33.0 ± 7.0</td>
<td>35.9 ± 5.4</td>
<td>36.1 ± 3.1</td>
</tr>
<tr>
<td>Pvo2 mm Hg</td>
<td>52.9 ± 9.7</td>
<td>37.8 ± 6.3</td>
<td>41.4 ± 4.7</td>
<td>73.0 ± 6.1</td>
<td>14</td>
</tr>
<tr>
<td>% saturation</td>
<td>92.3 ± 3.1*</td>
<td>85.0 ± 4.4</td>
<td>92.3 ± 3.1</td>
<td>16</td>
<td>16.4 ± 0.4</td>
</tr>
<tr>
<td>Relative flow (l/min/m²)</td>
<td>2.6 ± 0.4</td>
<td>2.4 ± 0.3</td>
<td>2.4 ± 0.4</td>
<td>2.4 ± 0.4</td>
<td>2.4 ± 0.4</td>
</tr>
<tr>
<td>Oxygen microemboli</td>
<td>8/16</td>
<td>6/16</td>
<td>10/16</td>
<td>16</td>
<td>10</td>
</tr>
</tbody>
</table>

*† p<0.0001, p<0.01, p<0.05, respectively, different from Phase II by Student's t test (differences in single variables).
§‖ p<0.01, p<0.05, respectively, different from Group 3 by paired t test.
A.L. 65y, 70% sten. R-ACI

FIGURE 5. Results of transcranial Doppler monitoring in patient with mild left carotid artery stenosis and moderate right stenosis. Increased transcranial Doppler shift was found. Factors such as hemoglobin concentration, pump flow, $P_{aco_2}$, and blood pressure were not responsible for clearly increased middle cerebral artery (MCA) blood velocity. Frequent embolic events were detected during Phases III and IV. Severe diffuse neurologic deficit was seen postoperatively with subsequent death. $T_{rect}$, rectal temperature in °C; BP, blood pressure in mm Hg; CVP, mean central venous pressure in mm Hg; II–VI, phases of operation (see "Subjects and Methods"); R-MCA, transcranial Doppler shift of right MCA in kHz; card pulm. bypass, steps of cardiopulmonary bypass: 1, partial bypass; 2, total bypass; 3, cross-clamped aorta; h, hours of anesthesia.

xenon-133 washout technique, also saw an increase of cerebral blood flow (67%) during the stable phase of CPB. On the other hand, Govier et al.7 and McKay et al.9 also using the xenon-133 washout technique, found significantly reduced regional cerebral blood flow during CPB. There is no explanation for these diverging results.

Postoperative clinical evaluation of our patients did not reveal a focal neurologic deficit, but three patients had diffuse encephalopathy. Only one of the three had severe carotid disease. Definite conclusions cannot be drawn from such a small number. The analysis of these three complications, however, indicates that it is unlikely that reduced blood flow was a factor. A transient deficit occurred once with an especially long procedure, which is a well-known risk factor.10,11 The other two patients with diffuse encephalopathy showed increased Doppler shift values during CPB (luxury perfusion) and frequent microembolic events as a parallel finding. Luxury perfusion also was seen by Henriksen et al.8 in patients with postoperative neurologic deficit.

We conclude that unilateral severe carotid stenosis or occlusion does not reduce cerebral blood flow during CPB. This furthermore supports clinical observations that the stroke rate is not significantly increased in the presence of carotid obstructions.1,2,4-6,8 Even if cerebrovascular arteriosclerosis increases the perioperative risk for stroke,7 other factors besides pressure-related reduction of blood flow may be responsible. Carotid endarterectomy, with the objective to increase cerebral perfusion during CPB, does not seem justified. Prophylactic carotid endarterectomy should therefore be confined to widely accepted indications, for the time being to patients with a history of TIA ipsilateral to carotid stenosis.

References

KEY WORDS • cardiopulmonary bypass • carotid artery diseases • ultrasonics
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