Relation Between Middle Cerebral Artery Blood Flow Velocity and Stump Pressure During Carotid Endarterectomy

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Background and Purpose: Many patient monitoring techniques have been used for detecting cerebral hypoperfusion during carotid endarterectomy. We compared middle cerebral artery blood flow velocities with carotid artery stump pressures to evaluate the indications for common carotid artery cross-clamp shunting and the probable hemodynamic causes of cerebrovascular complications.

Methods: Blood flow velocities were monitored with transcranial Doppler ultrasound and carotid stump pressures were measured at the time of common carotid artery cross-clamping during 97 carotid endarterectomy procedures. Stump pressures measured with the gauge zero reference at the common carotid artery level were correlated with the percentage change of velocities.

Results: Middle cerebral artery blood flow velocities usually decreased upon common carotid artery cross-clamping, depending on collateral availability and the autoregulation response. The best fit of the data was to an exponential function concave to the pressure axis, with velocity as a percentage of the pre-cross-clamp value reaching zero at 15 mm Hg stump pressure ($r=0.85$ and $p<0.001$).

Conclusions: There is a less critical margin of error with percentage middle cerebral artery blood flow velocity decreases than with stump pressure measurements. This relation establishes changes in middle cerebral artery blood flow velocities as a reliable parameter for judging the effects of carotid cross-clamping on cerebral blood flow and providing an excellent indicator as to the necessity for shunting. (Stroke 1992;23:1439-1445)

KEY WORDS • blood flow velocity • carotid endarterectomy • ultrasonics

Surgical operations requiring cross-clamping of the carotid artery threaten the cerebral circulation and the brain if regional hypoperfusion, embolization, or hyperperfusion at clamp release are produced. If, through improved monitoring, these threats can be reduced, then carotid endarterectomy and other operations affecting cerebral perfusion may become more universally accepted with extended clinical indications. Various modalities used to monitor and determine the vulnerability of the brain during carotid cross-clamping have included: 1) measurement of the distal internal carotid artery (ICA) pressure response upon common carotid artery (CCA) clamping (i.e., stump pressure),1-13 2) patient response monitoring under local anesthesia, 3) electroencephalographic (EEG) monitoring, and 4) jugular venous oxygen saturation.14 Jugular venous oxygen saturation changes represent more territory than that perfused by the ICA and thus may underestimate changes in oxygen extraction of the threatened territory. Serious reduction in cortical perfusion is more directly indicated by EEG or patient response monitoring and is widely used today for that reason. Operating on a conscious patient, however, sometimes increases the technical difficulty of the operation, and adverse EEG changes or patient responses are often somewhat delayed compared with the responses of stump pressure or middle cerebral artery (MCA) blood flow velocity. Stump pressure measurements represent the threatened territory but do not recognize embolization and may be incorrect because of inconsistent zero referencing of the pressure gauge.

Recently transcranial Doppler ultrasonography (TCD) has evolved as a method of monitoring blood flow velocities in the MCA of patients undergoing carotid surgery, being first reported in 1986.15 Through a series of subsequent reports comparing TCD with EEG16-20 and stump pressures21-23 the ability of TCD to assess hemodynamic and embolic phenomena developed. Additional disclosures24-31 of the value of TCD monitoring during carotid endarterectomy have added to our knowledge. TCD provides both hemodynamic and embolic information relating to perfusion of the threatened territory.

The purpose of this report is to demonstrate the relation between MCA blood flow velocity changes and the carotid back (stump) pressures found immediately following cross-clamping of the CCA and to relate this information to previous studies on stump pressure,
MCA blood flow velocities, and regional cerebral blood flow (rCBF) regarding indications for selective shunting and to the causes of cerebrovascular complications from the surgery.

Subjects and Methods

A 2-MHz pulsed Doppler ultrasound device was used to access the MCA or one of its branches through the transtemporal approach during cross-clamping of the CCA in carotid endarterectomy operations. The length of the ultrasonic sample volume was 12 mm, centered at a depth of either 4.5 or 5 cm from the crystal face. A depth of 4.5 cm was preferred and used in 77% of the cases to avoid the overlapping of collateral effects from the anterior cerebral artery or posterior cerebral artery sometimes occurring at a depth of 5 cm. The average MCA blood flow velocity (vMCA) recorded at the two depths was identical at 38 cm/sec. The maximum ultrasound power used was 900 mW/cm², adjusted downward when good signals were obtained, to produce an audio tape record level of −5 to −10 dB. A specially designed headband (Transpect, MedaSonic, Fremont, Calif.) fixed the probe to the patient's head with provisions for adjusting the probe position and angle. When MCA velocities dropped to immeasurable levels, below 5 cm/sec (i.e., below the equipment's high-pass cutoff frequency of 200 Hz), the systolic and diastolic velocities were entered as 1 cm/sec to avoid ambiguities in the data base formulas. For the baseline values, velocities and pressures were averaged over one half of a respiratory cycle before cross-clamping. Mean velocity for the first beat after cross-clamping was measured if a rapid clamping took place. The velocities and pressures during a 10–15-second interval after cross-clamping were also measured. Figure 1 illustrates the three periods of measurement. Assurance that the monitoring was performed on the MCA or a branch thereof was usually confirmed by oscillations of the carotid artery incurred spontaneously during dissection or upon deliberate finger oscillations of the carotid arteries by the surgeon. The oscillations were transmitted in the blood column to the MCA and were visible on the Doppler spectrum.

The stump pressure was measured, by means of a strain gauge manometer connected through saline-filled tubing to a needle puncturing the CCA, at the time of cross-clamping of the CCA and after cross-clamping of the external carotid artery and its immediate branches. The 97 cases reported here were selected from a larger data base using only those that had accurate stump pressures measured with a method for consistent gauge zero referencing. The zero reference level of the pressure measurement was set at the CCA and was indicated automatically at the moment just prior to insertion of the needle into the artery on the analog tracing of a strip chart recorder. This technique assured an accurate and consistent zero reference and documented pressure measurements for later review. In 85 of the 97 cases the routine stump pressure reported by the anesthesiologist used a gauge zero reference level variably estimated by the level of the heart. These measurements were not used in our quantitative analysis because of the uncertainty of the zero reference. When both measurements were compared (Figure 2), the heart-referenced measurements averaged 6.4 mm higher and one third exceeded the CCA-referenced measurements by >10 mm Hg. The stump pressures reported hereafter are only those measured from the strip chart recordings using the CCA as the gauge zero reference. A resistance index was calculated as mean pressure divided by mean velocity to correct for changes in velocity incident to simultaneous changes in pressure.

All signals, including the Doppler spectrum, stereo-separated directional Doppler signals, voice annotations, and arterial pressures, were recorded on a four-channel audio/video recorder. Arterial pressures were recorded on one audio channel by means of a voltage-controlled oscillator in addition to the strip chart recordings. By this means, the data could be replayed and validated after the operation to assure simultaneity and accuracy. Systolic and diastolic velocities and pressures were entered into an analytic data base (REFLEX), and mean values calculated as 0.4 × (systolic−diastolic)+diastolic. The factor 0.4 was determined by taking a sample of data from our patient population and estimating, with a cursor, the true mean velocity and mean pressure just before cross-
while hospitalized were graded on a scale of 1 to 5, with 1 representing a transient ischemic attack, 2 a persistent deficit not limiting ordinary activity, 3 a deficit limiting activity but patient able to care for self, 4 a patient needing help with personal care, confined to wheelchair, or walking with assistance, and 5 a bedridden patient.

Results
Immediately upon cross-clamping, MCA velocities usually diminished (Figure 3). First-beat velocities averaged 53 ± 24% (mean ± SD) of the baseline values, ranging from 100% to 3%. After the first beat velocities increased, on average 4 cm/sec toward baseline values, when measured at the 10–15-second interval. The variation in return depended on blood pressure, which during this phase usually diminished and averaged 6 mm Hg lower than at the first beat. The resistance index calculated at the first beat and at the 10–15-second interval were compared. In 65 patients in whom both sets of data were available and velocities did not drop below 5 cm/sec, there was a decrease in resistance after cross-clamping averaging from 2.5 ± 1.0 to 1.8 ± 0.6 mm Hg/cm/sec. This implied vasodilatation is assumed to represent autoregulation and occurred in all but two patients whose resistance index did not change after cross-clamping.

The combined relation between vMCA both before and after cross-clamping and stump pressure (p) both before and after cross-clamping fit an exponential function with r = 0.63 and p < 0.001; the pressure intercept of this velocity function was 20 mm Hg. The relation between MCA velocity after cross-clamping calculated as a percentage of the baseline value (vMCA%) and stump pressure (p) both before and after cross-clamping fit an elliptical function with r = 0.55, p < 0.001 (Figure 3) and T = 0.85.

The data were obtained from the operations of six surgeons who used selective shunting based on the anesthesiologist's stump pressures as well as MCA velocities. Cross-clamp times were recorded in minutes as a single number if not shunted and as two numbers representing the times required for insertion and removal if shunted. Microemboli detected in the anesthesiologist's stump pressures as well as MCA artery pressures and middle cerebral artery (MCA) mean blood flow velocity as percentage of value before cross-clamping averaging from 2.5 ± 1.0 to 1.8 ± 0.6 mm Hg/cm/sec. This implied vasodilatation is assumed to represent autoregulation and occurred in all but two patients whose resistance index did not change after cross-clamping.

The combined relation between vMCA both before and after cross-clamping and stump pressure (p) both before and after cross-clamping fit an elliptical function with r = 0.63 and p < 0.001; the pressure intercept of this velocity function was 20 mm Hg. The relation between MCA velocity after cross-clamping calculated as a percentage of the baseline value (vMCA%) and stump pressure also fit an elliptical function with r = 0.82 and p < 0.001; the pressure axis intercept of this function was 19 mm Hg. Both relations also fit exponential functions with slightly better correlation coefficients: vMCA = 40 \left[1 - e^{-0.035p+6}\right], r = 0.64, p < 0.001 (Figure 3) and vMCA% = 100 \left[1 - e^{-0.026p+15}\right], r = 0.85, p < 0.001 (Figure 4). The pressure axis intercepts of these functions were 9 and 15 mm Hg, respectively. Comparing the relation

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Average</th>
<th>Maximum</th>
<th>Minimum</th>
<th>Standard deviation</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>O</td>
<td>P</td>
<td>O</td>
<td>P</td>
</tr>
<tr>
<td>Stump pressure (mm Hg)</td>
<td>2</td>
<td>40</td>
<td>63</td>
<td>80</td>
</tr>
<tr>
<td>Blood flow velocity (% baseline)</td>
<td>42</td>
<td>67</td>
<td>93</td>
<td>100</td>
</tr>
</tbody>
</table>

ICA, internal carotid artery; O, 12 patients with occlusion of contralateral ICA; P, 78 patients with patent contralateral ICA.

References

FIGURE 3. Scatter plot of relation between common carotid artery (CCA) pressures and middle cerebral artery (MCA) blood flow velocities before (+) and 10–15 seconds after (x) cross-clamping. x, best fit of exponential function with r = 0.64.

FIGURE 4. Scatter plot of relation between middle cerebral artery (MCA) mean blood flow velocity as percentage of value before cross-clamping and mean common carotid artery (CCA) stump pressure x, best fit of exponential function with r = 0.85.
between vMCA% and percentage pressure change, the correlation coefficient for an exponential function was 0.85. There were 17 patients with a <10% decrease in vMCA upon cross-clamping. Among the 13 who underwent preoperative angiography, 10 had a >89% stenosis and in four of these "slow flow" or a "string sign" was demonstrated. Five with stenosis of >50% apparently had very efficient collateral systems. However, tight lesions (>95% area stenosis) did not predict a high clamp vMCA%; rather, patients with >90% stenosis were well distributed over the range of vMCA%.

The lowest stump pressure recorded was 8 mm Hg; this patient was one of five with stump pressures averaging 19 (range 8-25) mm Hg in whom vMCA dropped below 5 cm/sec. This patient demonstrated preoperative contralateral occlusion of the ICA. However, contralateral occlusion did not predict routinely lower stump pressures or routinely lower cross-clamp velocities, but patients with contralateral occlusion demonstrated lower average stump pressures and velocities (Table 1).

Cerebral complications were observed in the immediately postoperative period in seven patients, two among the 64 nonshunted and five among the 33 shunted surgeries. Table 2 lists all patients with complications in decreasing order of vMCA% and indicates the probable cause or causes of the complication concluded from the available data. None of the seven demonstrated postoperative occlusion of the ICA. The complications in patients 200, 211, and 55 were concluded to be caused by embolization because 25 other nonshunted patients who had no complications and sustained more adverse hemodynamic conditions with lower stump pressure, lower vMCA%, and longer cross-clamp time had no complications. A high level of microembolization was found (14 during 18 minutes) in the recovery room in patient 211, indicating a significant thrombus source at the operative site. Patient 55 was monitored for only 1 minute in the recovery room to make a determination regarding microembolization.

Shunted patient 200 with a high stump pressure and a high vMCA% sustained a transient worsening of the postoperative symptoms (weakness in one arm caused by a previous infarction). Shunt insertion was delayed for 58 minutes because of the high stump pressure. Baseline MCA velocity was only 17 cm/sec, dropping to 16 cm/sec upon cross-clamping, presumably due to a 99% stenosis with poor collateralization. A shunt was eventually used because the surgeon worried about these persistent low velocities. Postoperatively, the operated ICA was found to be patent by means of duplex Doppler examination. The cause of this minor complication was probably embolization, but the complication also points up previous recommendations for always shunting patients with previous infarctions. Microembolization was detected in all shunted and nonshunted patients with cerebrovascular complications occurring in the operating room and was concluded to be a possible contributing cause in all complications.

Patients 79 and 38, both with poor hemodynamic parameters but brief cross-clamp times for shunt insertion and removal, sustained severe strokes. The primary cause of stroke in patient 79 was probably hypoperfusion, but an embolus may have been produced when the shunt was forced to achieve placement. The microembolization monitoring data at that time were, however, not available, beyond 1 minute, to prove this. The primary cause of stroke in patient 38 was concluded to be hyperperfusion occurring when MCA velocities increased after release of the clamp to more than twice the baseline value. A postoperative cerebral computed tomogram demonstrated hemorrhagic infarction in both hemispheres. The contralateral ICA was occluded preoperatively, and there was anterior cerebral artery collateral crossover from the operated side.

In patients 267 and 173 cerebral complications were clearly due to hypoperfusion. Both patients demonstrated low MCA velocities and low stump pressure. Patient 267 required an 11-minute cross-clamp time due to difficulties in removing the shunt. Patient 173 required brief cross-clamp times for the initial insertion and removal of the shunt, but an additional cross-clamping was necessary when MCA velocities dropped to zero soon after release of the carotid clamp. Upon reopening the artery an occluding intimal flap was found. Fifteen minutes of additional cross-clamping was required to repair the flap, during which time MCA velocities were maintained, by elevation of the arterial pressure, at 12 cm/sec (vMCA% = 32). This patient luckily sustained only a transient postoperative weakness in the left arm. The complication was probably caused by hypoperfusion during the 15 minutes of low

**Table 2. Cerebrovascular Complications in Seven Patients With Carotid Endarterectomy**

<table>
<thead>
<tr>
<th>Pt No</th>
<th>Homolateral</th>
<th>Contralateral</th>
<th>ICA stenosis (%)</th>
<th>Stump pressure (mm Hg)</th>
<th>Shunt</th>
<th>Clamp time (min)</th>
<th>Microemboli (no./min)</th>
<th>Complication grade</th>
<th>Cause</th>
<th>Computed tomogram</th>
</tr>
</thead>
<tbody>
<tr>
<td>200</td>
<td>T</td>
<td>99</td>
<td>0</td>
<td>94</td>
<td>50</td>
<td>N/Y</td>
<td>58±4</td>
<td>44/45</td>
<td>1</td>
<td>E</td>
</tr>
<tr>
<td>211</td>
<td>D</td>
<td>85</td>
<td>0</td>
<td>90</td>
<td>62</td>
<td>No</td>
<td>31</td>
<td>14/18</td>
<td>1</td>
<td>E+HT</td>
</tr>
<tr>
<td>55</td>
<td>T</td>
<td>47</td>
<td>0</td>
<td>83</td>
<td>54</td>
<td>No</td>
<td>28</td>
<td>0/1</td>
<td>2</td>
<td>E in OR</td>
</tr>
<tr>
<td>79</td>
<td>A</td>
<td>95</td>
<td>75</td>
<td>46</td>
<td>20</td>
<td>Yes</td>
<td>5±6</td>
<td>0/1</td>
<td>3</td>
<td>Hypo</td>
</tr>
<tr>
<td>38</td>
<td>T</td>
<td>99</td>
<td>100</td>
<td>38</td>
<td>19</td>
<td>Yes</td>
<td>4±2</td>
<td>7/2</td>
<td>5</td>
<td>Hyper+E+Hypo</td>
</tr>
<tr>
<td>267</td>
<td>A</td>
<td>80</td>
<td>50</td>
<td>33</td>
<td>23</td>
<td>N/Y</td>
<td>3±11</td>
<td>0/5</td>
<td>4</td>
<td>Hypo+E</td>
</tr>
<tr>
<td>173</td>
<td>D</td>
<td>80</td>
<td>100</td>
<td>3</td>
<td>8</td>
<td>N/Y</td>
<td>4±3+15</td>
<td>97/37</td>
<td>1</td>
<td>Hypo+E</td>
</tr>
</tbody>
</table>

ICA, internal carotid artery; Sx, preoperative symptom; T, transient ischemic attack; D, dizziness; A, asymptomatic; vMCA%, middle cerebral artery blood flow velocity after cross-clamping as percent of baseline; no./min, number of microemboli detected in recovery room/minutes monitored; N/Y, prolonged insertion or removal time; E, embolus; HT, hypertensive episode in recovery room; OR, operating room; Hypo, hypoperfusion; Hyper, hyperperfusion; *no change from preoperative atrophy; toxocapitoparietal infarct; §hemorrhagic infarcts in both hemispheres with diffuse cerebral edema; ‡left frontoparietal infarct.
blood flow, but the many microemboli detected in the recovery room may have contributed.

Discussion

The relation between vMCA and stump pressure has been reported to be linear.15,22,23,28,31 The correlation coefficients of these studies using 28–49 patients varied from 0.46 to 0.84. The linear analyses projected to zero pressure or to an intercept on the velocity axis. Careful analysis of these reports lead to two major criticisms. 1) The relations are probably not linear because an intercept on the pressure axis is to be expected, requiring some critical closing (flow cessation) pressure so that flow and velocity will reach zero with some residual pressure. Moreover, any cortical collateral circulation will tend to produce some MCA back pressure, causing blood flow in the MCA to be zero with some residual cortical collateral pressure. 2) The zero reference levels of the pressure measurements were not indicated in any of these reports and were probably mostly the heart level and thus were measured falsely high and with great variability. When we eliminated, in our studies, all uncertain zero pressure references, used only stump pressures measured with the CCA as the gauge zero reference, and assumed a residual pressure at a flow of zero and a curvilinear relation, a more likely result (a pressure axis intercept) was found. In addition, when percentage change in MCA velocities rather than the absolute value was used, the problems of using signals from an MCA branch as well as those resulting from variable diameters of the MCA and variations in the Doppler angle are ameliorated and the relation is further improved.

The combined effect of collateralization and autoregulation is inherent in the 10–15-second cross-clamp pressure/velocity relation. In those patients with a good collateral circulation and an active autoregulation, a disproportionately greater drop in stump pressure was necessary to produce a perceptible change in MCA velocity. In patients with a poor collateral circulation, the decrease in stump pressure caused a disproportional decrease in velocity, even with normal autoregulation. The curved pressure/velocity relation, with concavity toward the pressure axis, demonstrates greater reliability of vMCA% over stump pressure as an indicator for selective shunting. When choosing a critical level for stump pressures (e.g., 25 or 30 mm Hg), a small overestimation can cause a great underestimation of cerebral perfusion while errors in the vMCA% measurements are far less critical. This is even more important if stump pressures are measured when the gauge zero reference is estimated at the heart level. The proponents and users of carotid back (stump) pressure have usually not indicated the zero reference level to be used. Only one article11 in our review has mentioned the need to reference the pressure zero "at the level of the carotid artery." It should be noted that there are no zero reference problems with Doppler-determined velocities.

In patients with lower stump pressures, arterial blood flow velocity falls precipitously with small changes in pressure, requiring a high shunt decision level and producing either unnecessary shunts or false conclusions of adequate perfusion. Shunting decisions, in this series, were initially based on the stump pressure, with the criterion that a shunt was placed if the pressure fell below 30 mm Hg. After experience with TCD monitoring, greater reliance was placed on MCA velocities to the gradual exclusion of stump pressure as a decision criterion. Experience with nonshunted patients without complications indicated that a vMCA% of >40 (corresponding to a stump pressure of 25 mm Hg) provides adequate brain perfusion.

Archie10 reported carefully measured stump pressures that are quite consistent with our findings when comparing values uncorrected for venous pressure. Venous pressure used to calculate perfusion pressure serves a valid concept; however, venous pressure averaged only 6.2±3.9 mm Hg. We believe venous pressure measurement is a minor correction to be made considering the relatively greater importance of arterial pressure in determining cerebral perfusion. Archie10 considers the major error in stump pressure measurements to be incomplete occlusion of the carotid arteries, resulting in erroneously high stump pressures. This may have occurred in some of our cases because no confirmatory test was performed. This may account for some of the variability of our data but probably was not frequent because the percentage of our patients with a stump pressure of <25 mm Hg was 20% compared with Archie's 16.2%.10 The high correlation between stump pressure and vMCA% strongly suggests that changes in blood flow velocity represent changes in volumetric flow in the MCA. This conclusion is in agreement with other investigations.29,31

The rCBF has been poorly correlated with stump pressure in previous studies (r=0.62,4 r=0.51,6 and r=0.5331). The difference in these correlations may be due to technical problems with rCBF measurements or problems with pressure zero references. MCA blood flow velocity has been compared with rCBF during carotid endarterectomy. The correlation was strong if rCBF was <20 ml/100 g/min but weak at higher levels. The rCBF was more specific than vMCA for EEG change. At cross-clamping, the rCBF/vMCA relation intercepted the pressure axis between 5 and 10 ml/100 g/min, indicating some cortical perfusion when vMCA is zero. Also, the rCBF/stump pressure relation* intersects the pressure axis between 9 and 16 mm Hg. It appears from these studies and the present study that both cerebral perfusion and MCA velocity cease at some positive arterial pressure between 8 and 20 mm Hg and that MCA velocity may reach zero before cortical perfusion ceases. If the critical perfusion levels below which the brain suffers irreversible damage are 20–30 ml/100 g/min, the corresponding critical vMCA% level will be 40–60. In our experience no hypoperfusion-related cerebral complications have occurred with vMCA% of >60. Worsening of symptoms from previous infarctions has occurred in the vMCA% range of 40–60, and all clearly hypoperfusion-related complications have occurred in patients with a vMCA% of <40. We believe a vMCA% of >40 and a stump pressure of >25 mm Hg represent values above which shunting may not be necessary to prevent hypoperfusion. This belief that a vMCA% of 40 represents a level below which complications are prone to occur is confirmed by a recent publication30 comparing the ratio of pre- to post-cross-clamp MCA velocities in patients with EEG flattening and an rCBF of <20 ml/100 g/min.
The major problems in TCD monitoring of the MCA revolve around difficulty in finding and holding the probe over an ultrasonic window, problems requiring continuous surveillance of the signal. Technologist dependency is possibly not more of a problem than with monitoring rCBF or EEG, or even with surgery under local anesthesia. The cost of equipment for TCD monitoring is modest compared with that for rCBF and EEG monitoring. Stump pressure and rCBF measurements provide only intermittent values and do not continuously follow changes throughout the procedure as does TCD. TCD offers an additional unique advantage in providing on-line detection of active microembolization. For those cases in which the temporal bone ultrasonic window is not available, alternative use of stump pressure, or cortical function monitoring with EEG or patient response, appears advisable. Where feasible, a combination of cortical perfusion and MCA velocity monitoring techniques may be optimal.

Conclusions

1. TCD monitoring of the MCA signals in patients undergoing carotid endarterectomy provides hemodynamic data that can be used to prevent hypoperfusion during carotid endarterectomy.
2. Confirmation of appropriate monitoring of the MCA or a branch thereof can be assured by observation of oscillations in the Doppler velocity spectrum incurred during surgical manipulations or by direct finger oscillations of the CCA.
3. Following cross-clamping of the CCA, the MCA blood flow velocities decrease depending on the collateral availability and the autoregulation response. In cases of severe ICA stenosis or where great collateralization between the basal cerebral arteries is available, MCA velocities may not decrease upon cross-clamping.
4. From the first beat immediately after carotid cross-clamping, the cerebrovascular resistance progressively decreases over a 10–15-second period representing the autoregulation response. The first-beat stump pressure or vMCA% reflects collateralization available; changes over the subsequent 10–15-second interval reflect the autoregulation response. Decision for shunting based on the autonomic criteria could be made after the autoregulation response is complete.
5. The relation between stump pressure and vMCA% follows defined curvilinear functions with correlation coefficients of >80%. The vMCA% is a better index of change in perfusion than is residual velocity or the absolute difference in velocity.
6. Because the stump pressure/vMCA% relation is concave toward the pressure axis, TCD velocity changes provide a greater margin of safety for shunting decisions than do stump pressure measurements.
7. When stump pressures are measured for shunting decisions, the zero reference level for the strain gauge should be carefully set at the level of the CCA to prevent overestimation and subsequent danger of hypoperfusion during carotid cross-clamping without shunting. Doppler velocity measurements have no zero reference ambiguities in monitoring MCA signals during carotid endarterectomies.
8. More than 75% of patients have sufficient intracranial collateral circulation and autoregulation response to allow cross-clamping of the carotid arteries in the neck without the use of a shunt. A reasonable MCA velocity below which shunting should be performed in anesthetized patients is <40% of the baseline level.
9. MCA velocity changes can serve as the sole hemodynamic decision-making parameter, but in cases in which the Doppler signal cannot be found stump pressure, EEG, and other parameters can be used.

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