CASE REPORTS

References


8. Goldfarb G, Debaene B, Ang ET, Roulot D, Jolis P, Lebrec D:


Immediate Detection of Carotid Arterial Thrombosis by Transcranial Doppler Monitoring

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Since the introduction of Doppler ultrasound for intracranial applications by Aaslid in 1982,1 the transcranial Doppler (TCD) has been used by several investigators to monitor cerebral blood flow velocity during surgery for carotid endarterectomy (CEA). Transcranial Doppler velocity as a monitoring technique has been compared to regional cerebral blood flow (rCBF),2,4,5 EEG,6,7 and carotid artery stump pressure.8 Cerebral autoregulation with respect to change in blood pressure has been demonstrated in human using the TCD.9 In addition to noninvasive, continuous monitoring of middle cerebral artery blood flow velocity (MCAV) during CEA, TCD monitoring also permits the detection of embolic events, shunt malfunction, vasospasm, and the assessment of collateral cerebral perfusion during cross clamping of the carotid.2,4,6,8 The following case report serves as an example of immediate detection of internal carotid artery thrombosis by the TCD in the absence of EEG change.

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Case Report

A 69-year-old, 86-kg man was admitted for right CEA 1 month after successful, uncomplicated left carotid endarterectomy for high-grade stenosis. His medical history was significant for insulin-dependent diabetes mellitus, hypertension, atherosclerotic vascular disease, and rheumatoid arthritis. He denied any medication allergies.

After application of standard noninvasive monitors and EEG cup electrodes, and the insertion of a radial arterial catheter, the Transpect Transcranial Doppler (Medasonics, Fremont, CA) was secured above the zygomatic arch. The patient had a good Doppler monitoring window in the temporal bone, allowing insonation of the middle cerebral artery at a depth of 50 mm from the temporal scalp.Baseline preinduction blood pressure was 123/67 mmHg (mean 86 mmHg).

After sedation with 2 mg intravenous midazolam, anesthesia was induced with sodium thiopental (500 mg total). Middle cerebral artery blood flow velocity decreased with thiopental on induction, and subsequently increased after tracheal intubation when the blood pressure reached 173/83 mmHg (mean 113 mmHg). The corresponding MCAV was 36/15 cm/s (mean 21 cm/s). Anesthesia was maintained with isoflurane and intermittent intravenous fentanyl. The end-tidal isoflurane concentration ranged from 0.57 to 0.7%, and a total of 500 μg fentanyl was required for the case. End-tidal PaO2 ranged from 29 to 33 mmHg, with an arterial end-tidal gradient of 6 mmHg or less. Several measurements of MCAV and blood pressure were obtained after anesthetic induction and before test occlusion of the common carotid. During this period, the range of mean MCAV was from 16 to 21 cm/s, with only one value less than 20 cm/s. The immediate preocclusion velocity was 35/15 cm/s (mean 21 cm/s) at a blood pressure of 135/42 mmHg (mean 75 mmHg).

After anticoagulation with heparin, the common carotid artery was occluded, resulting in a decrease in MCAV to 18/10 cm/s (mean 13 cm/s) at a blood pressure of 133/90 mmHg (mean 104 mmHg). Mean arterial pressure was not increased empirically at this point, because there was no critical change in TCD or EEG. No EEG changes were noted after cross clamping. No intracarotid shunt was placed. Back bleeding was brisk, and the mean carotid artery stump pressure was 66 mmHg, with a mean radial artery pressure of 96 mmHg.

During the 29 min of carotid cross-clamp time, the mean MCAV increased progressively from 15 to 19 cm/s. At the conclusion of the endarterectomy, the cross clamp was released and the MCAV increased to 39/20 cm/s (mean 26 cm/s). Within 2 min after the slow administration of a 50-mg dose of intravenous protamine sulfate, the patient became hypertensive with blood pressures ranging from 52/39 to 83/65 mmHg, and mean MCAV in the range of 5 to 15 cm/s. This hypertensive period was relatively refractory to treatment with epinephrine in steadily increasing bolus doses, lasted 9 min, and eventually resolved with restoration of blood pressure to the levels measured before the administration of protamine. Within 5 min of restoration of blood pressure, the MCAV was determined to be to 15/12 cm/s (mean 13 cm/s) at a blood pressure of 147/81 mmHg (mean 105 mmHg). A mean MCAV of 15 cm/s was observed at the time of the initial cross clamping of the common carotid artery, and was less than expected at this point in the case.

The surgeon was notified of the decreased mean MCAV, and a thrombectomy of a totally occluded, thrombosed proximal internal carotid artery was performed. Thrombus length was 1 cm with no extension found to the distal artery. No technical defect was found; therefore, thrombosis was presumed because of hypotension secondary to protamine. Low molecular weight dextran was given for antithrombotic effect. When blood flow was reestablished, the MCAV returned to expected values of 46/19 cm/s (mean 28 cm/s) at a blood pressure of 125/65 mmHg (mean 85 mmHg). There was no change in the ten-channel EEG during the anesthetic. No attempt was made to insinuate other vessels, such as the anterior or posterior cerebral arteries, to document flow reversal. To do so is difficult during carotid surgery without disrupting the sterile field; TCD monitoring would have been interrupted during a critical period of this case; and there would be no guarantee that the exact angle of insination could be reproduced when the middle cerebral artery was relocalized. The patient awakened without neurologic deficit.

Discussion

The application of TCD monitoring to carotid artery surgery has been conducted in our institution as an adjunct to ten-channel EEG monitoring for the past 6 yr. Although absolute TCD velocity does not correlate well with rCBF, changes in TCD velocity have been shown to correlate well with changes in rCBF. The major strengths of the technique are that it is noninvasive and provides continuous monitoring. This is an advantage over rCBF measurement techniques, particularly during a surgical procedure in which dynamic changes in physiologic parameters and surgical manipulation preclude steady state conditions. In patients with limited vasomotor reserve and poor collateral cerebral circulation, TCD permits immediate detection of failure of collateral blood flow to compensate for common carotid artery occlusion, thus allowing the surgeon to adjust the surgical technique and assess the intervention.

The blood flow velocity changes at the time of occlusion of the common carotid artery are used by our surgeons to selectively shunt around the temporary carotid occlusion for endarterectomy. The need for intracarotid shunting depends, in part, on the adequacy of collateral blood supply to the hemisphere during the period of occlusion. During cross clamping of the common carotid, blood may be supplied to the ipsilateral hemisphere via reversed flow in the anterior and posterior cerebral arteries, via the leptomeningeal anastomoses with the anterior and posterior cerebral arteries, the vertebrobasilar system via the posterior communicating artery, or the external carotid via orbital collaterals.

Although the criteria for shunting in our institution ultimately depends on surgical judgment, intracarotid
shunting is usually performed if the mean MCAV falls to less than 15−20% of the preclosure mean MCAV. This velocity threshold, which was not reached by our patient, is based on the data of Halsey and McDowell. There is, however, no uniform agreement on the velocity threshold for shunting at this time. For example, Spencer et al. recommend shunting for mean MCAV during cross clamping less than 40% of baseline. The use of a shunt without indications may be detrimental, because there was a 17% incidence of stroke in those shunted with a mean MCAV ≥ 41% of baseline and a 1.1% incidence in those not shunted. The increased incidence of stroke after carotid shunting could be the result of embolism, intimal injury and thrombosis, or carotid dissection.

There appear to be velocity and cerebral blood flow thresholds below which EEG changes are seen during occlusion of the carotid. Halsey et al. found that, in 65% of patients, MCAV postocclusion velocity between 0 and 15% of the preclosure value precedes ischemic EEG changes, such as loss of amplitude. In 31 patients undergoing carotid endarterectomy, EEG suppression occurred when rCBF decreased to less than 9 ml·min⁻¹·100 g⁻¹ and mean MCAV was less than 15 cm/s. In that study, there were six patients with EEG changes; four had zero velocity at carotid cross clamping, and the others had velocities of 9 and 14 cm/s. Based on Halsey’s data, CBF in our patient may have been near the threshold for EEG changes. In addition, it has been proposed that the EEG predominately monitors cortical blood flow, whereas the TCD may be more sensitive to blood flow to the basal ganglia and internal capsule. It was also stressed that the monitoring methods, EEG and TCD, complement one another for this very reason.

As seen in table 1, MCAV, after release of the cross clamp, was 39/20 cm/s (mean 26 cm/s). Although higher than the preclosure value of 35/15 cm/s (mean 21 cm/s), the higher mean MCAV does not indicate a degree of hyperperfusion requiring special treatment or intervention. This is not always the case, however. If significant hyperemia is present after reperfusion, increased attention to blood pressure control may be important in the postoperative period to prevent intracerebral hemorrhage.

During the hypotensive period, which was probably the result of anaphylaxis to protamine, the blood pressure of 52/39 mmHg (mean 43 mmHg; table 1) is less than the accepted threshold for cerebral blood flow pressure autoregulation. For this reason, the low mean MCAV during hypotension was not necessarily surprising. There was evidence of occlusion distal to the endarterectomy site after restoration of blood pressure, which was alarming. The mean MCAV failed to return to the expected value (near or above preclosure), and there was a decrease in the pulsatility index (PI; the difference between systolic and diastolic velocities divided by the mean velocity) from 0.95 to 0.23 after the formation of the thrombus (table 1). This is believed to reflect the reduced distal vascular resistance after occlusion, is seen with carotid cross clamping, and may be more sensitive than mean MCAV changes during carotid cross clamping.

After restoration of blood pressure, the MCAV of 15/12 cm/s (mean 13 cm/s) and a lower PI than expected prompted a search for the etiology of these changes. A thrombectomy was performed, and clot was recovered. After thrombectomy and restoration of blood flow, the MCAV increased to 46/20 cm/s (mean 28 cm/s) and the PI increased to 0.96 at a BP of 125/65 mmHg (mean 85 mmHg; table 1). It is likely that, during the 9-min period of hypotension, blood flow was low enough to allow clot formation in the freshly endarterectomized carotid artery.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Preclosure</th>
<th>Carotid Occluded</th>
<th>Carotid Released</th>
<th>Hypotensive Period</th>
<th>Pressure Restored</th>
<th>Postthrombectomy</th>
</tr>
</thead>
<tbody>
<tr>
<td>BP (mmHg)</td>
<td>135/42</td>
<td>133/90</td>
<td>165/87</td>
<td>52/39</td>
<td>147/81</td>
<td>125/65</td>
</tr>
<tr>
<td>MAP (mmHg)</td>
<td>73</td>
<td>104</td>
<td>113</td>
<td>43</td>
<td>103</td>
<td>85</td>
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<td>MCAV (cm/s)</td>
<td>35/15</td>
<td>18/10</td>
<td>39/20</td>
<td>17/14</td>
<td>15/12</td>
<td>46/20</td>
</tr>
<tr>
<td>Mean MCAV (cm/s)</td>
<td>21</td>
<td>13</td>
<td>26</td>
<td>15</td>
<td>13</td>
<td>28</td>
</tr>
<tr>
<td>PI</td>
<td>0.95</td>
<td>0.62</td>
<td>0.74</td>
<td>0.20</td>
<td>0.23</td>
<td>0.92</td>
</tr>
</tbody>
</table>

After release of the common carotid cross clamp, hypotension due to protamine occurred, resulting in thrombosis of the internal carotid artery. Data in the last column are postthrombectomy with flow restored.

BP = systolic/diastolic blood pressure; MAP = mean arterial pressure; MCAV = systolic/diastolic middle cerebral artery blood flow velocity; PI = pulsatility index of MCAV [(systolic − diastolic velocity)/mean velocity].

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Without the Doppler monitoring, and in the absence of EEG changes, our patient probably would have been awakened and transported to recovery and then on to his hospital room without discovery of the thrombus. At some point in the postoperative course of this patient, undetected persistent low blood flow may have resulted in a cerebrovascular accident. It is also possible that extension of the common carotid artery clot causing occlusion of the middle cerebral artery (MCA), or collaterals to the MCA may have resulted in stroke. Cerebrovascular accident may also have resulted from the embolism of thrombus in the postoperative period. Early detection, using the TCD, permitted early thrombectomy and assessment of the results of the thrombectomy.

References


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