Cerebral Blood Flow Measurement as a Safeguard During Carotid Endarterectomy

BY GUDRUN BOYSEN, M.D.

Abstract: Cerebral Blood Flow Measurement as a Safeguard During Carotid Endarterectomy

Intraoperative measurement of regional cerebral blood flow (rCBF) by the $^{133}$Xe intracarotid injection technique was performed in 17 patients operated on for stenosis of the internal carotid artery. Mean rCBF decreased from 51 ml/100 gm x minute before endarterectomy to 30 ml/100 gm x minute when the internal carotid artery was clamped, a reduction of 41%. Postoperative aggravation of neurological deficits was observed in five patients in whom rCBF was at or below 30 ml/100 gm x minute during the period of carotid clamping, indicating a critical lower limit of rCBF of this magnitude. Flow reduction below this limit was tolerated only if the occlusion time was short. A critical time/flow relation is suggested.

Distal internal carotid artery blood pressure during clamping (measured in eight cases) was poorly correlated to rCBF ($r = 0.62, 0.1 > P > 0.05$).

ADDITIONAL KEY WORDS

cerebrovascular disease
$^{133}$Xe
internal carotid artery pressure

During endarterectomy of the extracranial cerebral arteries it is essential to protect the brain against ischemia. Ischemic brain lesions following operation can result from critically low tissue perfusion during the period of carotid clamping or from emboli dislodged from the atheromatous plaque during manipulation of the artery. Insertion of a temporary bypass guards against the former, but even careful manipulation does not offer complete protection against emboli. To test the efficiency of the intracranial collaterals Jennett suggested intraoperative determination of regional cerebral blood flow (rCBF) during a test occlusion of the carotid artery. In this study rCBF during carotid occlusion and the length of occlusion time are related to the occurrence of postoperative neurological complications. The value of intraoperative rCBF measurement is discussed.

Methods

The series consists of 17 patients operated on for stenosis of the internal carotid artery. Four patients had occlusion and five had stenosis of the opposite internal carotid artery. All patients had had neurological symptoms from the hemisphere fed by the internal carotid artery operated on. Six patients presented with a history of one or more transient ischemic attacks and were neurologically intact at the time of operation. Two patients had recovered from episodes of cerebral ischemia with slight residual symptoms. Six patients had had...
completed strokes, and exhibited varying degrees of neurological deficits. Three patients were operated on immediately after admission because of progressing stroke.

All operations were performed under general anesthesia. After induction with a short-acting barbiturate, anesthesia was maintained by halothane in concentrations of ≤1.0% in a 50% O₂/N₂O mixture. The patients were ventilated by a respirator via a non-rebreathing system. Systemic blood pressure was continuously monitored through a catheter inserted into the radial artery and connected to a Statham pressure transducer. $P_{\text{aCO}_2}$ was measured at each rCBF measurement.

rCBF was measured by the $^{133}$Xe intracarotid injection technique with external detection by a single scintillation detector as described by Høedt-Rasmussen, Sveinsdottir and Lassen.² The method has been described elsewhere.³,⁴ rCBF was calculated by the initial slope method.⁵ However, the equation used by Paulson et al.⁵

$$rCBF_{10} = d_{\text{initial}}^{0.77} \times 141.1 \text{ ml/100 gm \times minute}$$

was found to overestimate rCBF₁₀ at high flow rates and to underestimate it at low flow rates. In a series of 72 cerebral blood flow measurements, performed on 31 patients undergoing carotid endarterectomy, rCBF₁₀ was calculated as

$$rCBF_{10} = \frac{H - H_{10}}{A_{10}} \times \lambda \times 100 \text{ ml/100 gm \times minute}$$

and correlated to $d_{\text{initial}}$, i.e., the fraction of a decade by which the semilogarithmic curve decreases per minute. The correlation was expressed by (fig. 1):

$$rCBF_{10} = d_{\text{initial}}^{0.77} \times 141.1 \text{ ml/100 gm \times minute}$$

In the following, rCBF is calculated according to equation 3.

PROCEDURE
After exposure of the internal carotid artery,
CEREBRAL BLOOD FLOW MEASUREMENTS

In eight cases distal pressure in the internal carotid artery was measured during the period of clamping. A 23-gauge needle with a three-way stopcock was connected to a syringe containing the tracer and to a pressure transducer. The internal carotid artery pressure was measured before and after the tracer injection.

Results

On clamping the internal carotid artery rCBF was reduced in 14 of 15 patients (fig. 2). The mean fall was 21 ml/100 gm · minute—from 51 to 30 ml/100 gm · minute (41%). The flow reduction depended on the $P_{\text{aCO}_2}$ level being greater in patients studied at hypercapnia (45%) than in those studied at normocapnia or hypocapnia (17%). In one case rCBF was unchanged. In two cases rCBF could not be measured before endarterectomy. Mean rCBF after endarterectomy was 51 ml/100 gm · minute.

In four cases where rCBF during test clamping ranged from 12 to 25 ml/100 gm · minute an internal bypass was used (table 1). The pattern of the washout curves in one of these cases is shown in figure 3. The occlusion time necessary for insertion and removal of the bypass varied from one and one-half to six minutes. In the 13 cases not having a shunt, occlusion time averaged 24 minutes (range 15 to 35 minutes).

The internal carotid artery pressure distal to the clamp was measured in eight cases. The correlation to rCBF was not statistically significant, $r = 0.62, 0.1 > P > 0.05$.

After allocating patients to groups according to the severity of neurological symptoms, it was found that patients with transient neurological attacks or with mild neurological symptoms (groups 1 and 2) had higher rCBF values than patients with completed or progressing strokes (groups 3 and 4) (table 2). In groups 1 and 2 carotid clamping reduced mean rCBF from 61.0 to 35.4 ml/100 gm · minute, $P < 0.01$. In groups 3 and 4 mean rCBF decreased from 40 to 26 ml/100 gm · minute, $P < 0.05$. However, the number of patients was small, and the differences between the two groups were not statistically significant.

Twelve patients had an uneventful postoperative course. Four patients had an aggravation of their preoperative neurological deficit, and one patient, with preoperative transient ischemic attacks, developed a transient hemi-

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**Figure 2**

rCBF before and after endarterectomy and during clamping of the carotid artery in 17 patients.

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Indicates cases with uncomplicated operation and postoperative course.

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Indicates cases where bypass was applied.

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Indicates postoperative neurological complication.

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Blood flow therein was measured by an electromagnetic flowmeter. The flowmeter probe was carefully placed well above the site of stenosis in order to avoid dislodging cerebral emboli. Simultaneously rCBF was measured after injecting the tracer into the internal carotid artery. Before endarterectomy a repeat $^{133}$Xe dose was injected, whereupon the artery was clamped and the washout curve recorded for two minutes. The clamp was then released, allowing time for calculation of rCBF. If a bypass was inserted, another tracer dose was given to test the bypass function. Otherwise, endarterectomy was undertaken without further delay. After completion of endarterectomy internal carotid artery flow and rCBF were measured again. There was an interval of at least ten minutes between two successive rCBF measurements. The results of the measurements before and after endarterectomy have previously been published.4 Patients investigated in both series have retained their original case numbers in the present paper.

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Boysen

Table 1: Clinical Observations and Hemodynamic Data During the Period of Carotid Clamping in 17 Patients Operated on for Stenosis of the Internal Carotid Artery.

<table>
<thead>
<tr>
<th>No.</th>
<th>Group</th>
<th>Left</th>
<th>Right</th>
<th>rCBF ml/100 gm/min</th>
<th>MABP radial artery</th>
<th>MABP internal carotid distal to clamp</th>
<th>PaCO₂ mm Hg</th>
<th>Duration of carotid occlusion (min)</th>
<th>Temporary neurologic bypass complications</th>
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<tbody>
<tr>
<td>1</td>
<td>3</td>
<td>ST</td>
<td>NS</td>
<td>24</td>
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<td>+</td>
</tr>
<tr>
<td>3</td>
<td>1</td>
<td>ST</td>
<td>NS</td>
<td>42</td>
<td>113</td>
<td>33</td>
<td>35</td>
<td>0</td>
<td>-</td>
</tr>
<tr>
<td>4</td>
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<td>35</td>
<td>0</td>
<td>-</td>
</tr>
<tr>
<td>5</td>
<td>1</td>
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<td></td>
<td>42</td>
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<td>14</td>
<td>0</td>
<td>-</td>
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<tr>
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<td>+</td>
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<tr>
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<td>ST</td>
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<td>2</td>
<td>NS</td>
<td>ST</td>
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<td>1½ + 2½</td>
<td>0</td>
<td>-</td>
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<tr>
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<td>1</td>
<td>NS</td>
<td>ST</td>
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<tr>
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<td>ST</td>
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<td>33</td>
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<td>29</td>
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<td>43</td>
<td>19</td>
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<tr>
<td>14</td>
<td>1</td>
<td>OC</td>
<td>ST</td>
<td>12</td>
<td>127</td>
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<td>1½ + 3</td>
<td>0</td>
<td>-</td>
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<tr>
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<td>3</td>
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<td>ST</td>
<td>30</td>
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<td>48</td>
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<tr>
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<td>3</td>
<td>ST</td>
<td>OC</td>
<td>25</td>
<td>108</td>
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<td>6 + 5</td>
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<tr>
<td>17</td>
<td>3</td>
<td>ST</td>
<td>ST</td>
<td>28</td>
<td>125</td>
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<td>15</td>
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<td>-</td>
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<td>NS</td>
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<td>85</td>
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<td>31</td>
<td>0</td>
<td>+</td>
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<tr>
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<td>4</td>
<td>ST</td>
<td></td>
<td>24</td>
<td>113</td>
<td>-</td>
<td>15</td>
<td>0</td>
<td>-</td>
</tr>
</tbody>
</table>

Group 1: Transient ischemic attacks.
Group 2: Cerebral ischemic attacks with slight neurological deficits.
Group 3: Completed strokes.
Group 4: Progressing strokes, acute operations.

rCBF = regional cerebral blood flow.
MABP = mean arterial blood pressure, mm Hg.
PaCO₂ = arterial carbon dioxide tension.
ST = stenosis of internal carotid artery.
OC = occlusion of internal carotid artery.
NS = no stenosis.

In these five cases paresis postoperatively. In these five cases rCBF ranged from 13 to 31 ml/100 gm • minute during the period of carotid clamping, ranging from five to 35 minutes. The ratio, rCBF/occlusion time, in these five patients averaged 1.33 (range 0.80 to 2.60). In seven patients without complications, where rCBF during clamping was below 30 ml/100 gm • minute, the ratio was 2.92 (range 1.52 to 5.60). This difference is significant, P < 0.05.

Table 2: Regional Cerebral Blood Flow in Patients with Transient Ischemic Attacks or Mild Neurological Symptoms Compared with that in Completed or Progressing Strokes

<table>
<thead>
<tr>
<th></th>
<th>Before endarterectomy Group</th>
<th>During carotid clamping Group</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1 + 2</td>
<td>3 + 4</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1 + 2</td>
</tr>
<tr>
<td>rCBF ml/100 gm/min</td>
<td>61 ± 23</td>
<td>40 ± 21</td>
</tr>
<tr>
<td>MABP, mm Hg</td>
<td>103 ± 10</td>
<td>* 99 ± 13</td>
</tr>
<tr>
<td>PaCO₂, mm Hg</td>
<td>51 ± 8</td>
<td>* 50 ± 13</td>
</tr>
</tbody>
</table>

*Difference not statistically significant (Wilcoxon's test for two samples).
CEREBRAL BLOOD FLOW MEASUREMENTS

COUNTS /SEC

\[ d_0 = 0.45 \]

\[ d_0 = 0.065 \]

\[ d_0 = 0.05 \]

\[ d_0 = 0.41 \]

PRE

TEST CLAMP

INSERTION of BY−PASS

POST

MIN

FIGURE 3

\[ ^{133} \text{Xe} \] washout curves in a patient, case 9, undergoing internal carotid endarterectomy. Before endarterectomy the semilogarithmic curve has a steep initial slope corresponding to an rCBF of 76/100 gm • minute. During test clamping the curve declines slowly corresponding to an rCBF of 14 ml/100 gm • minute, but as soon as the clamp is released the tracer rapidly washes out. The slope immediately after removal of the clamp, being even steeper than the preceding curve, indicates a cerebral vasodilatation caused by the short ischemia. An almost identical pattern was reproduced when the artery was clamped for insertion of the shunt which was functional after two and one-half minutes. After endarterectomy rCBF was 71 ml/100 gm • minute. PAO2 was 54 to 56 mm Hg during the whole procedure.

If the patients with temporary bypass are excluded from both groups the ratios become 1.02 and 1.67, \( P < 0.05 \). Based on these findings a critical time/flow relation is outlined (fig. 4).

The case histories and relevant examinations of the five patients in whom neurological complications occurred are as follows.

**Case Reports**

**CASE 1**

Nine months preoperatively, a 66-year-old woman had an aphasia of a few minutes' duration. Two months preoperatively a slowly progressing right-sided hemiparesis developed. This was accompanied by headache and visual disturbances (flashes of light). At the time of operation the right hemiparesis was mild in the arm and pronounced in the leg.

Angiography of the carotid arteries showed that the right internal carotid artery (ICA) was normal apart from some irregularity of the siphon contour. There was left ICA subtotal stenosis at the bifurcation and irregularity of the siphon. Intracranial arteries were normal on both sides. There was some spontaneous cross-filling of the left anterior cerebral artery from the right side.

Her blood pressure was 170/110 to 150/100, and the hemoglobin 13.4 gm %. The ECG and chest x-ray were normal.

During endarterectomy of the left ICA, rCBF decreased from 31 to 24 ml/100 gm • min-
of the right anterior and middle cerebral arteries. Angiography of the aortic arch was unsuccessful. Neurological examination was normal. The blood pressure was 165/80, and the hemoglobin was 15 gm %. The ECG and chest x-ray were normal.

Angiography had given suspicion of right ICA occlusion, and on exploration this was confirmed. Flow could not be restored. During endarterectomy of the left ICA ten days later, rCBF decreased from 33 to 28 ml/100 gm minute when the artery was clamped. Occlusion lasted 35 minutes. Postoperatively the patient had moderate paresis of the right arm and accentuated tendon reflexes in the right leg. Postoperative angiography was not performed.

Fifteen months postoperatively the paresis of the right arm had remitted and there had been no further cerebral episodes. He was able to go skiing and to ride a horse.

CASE 7
A 75-year-old-man with no previous history had an acute right-sided hemiparesis and aphasia three weeks preoperatively. On preoperative neurological examination he had slight intellectual impairment with receptive and expressive aphasia, and a right-sided hemiparesis mainly affecting the arm. Angiography showed occlusion of the left common carotid artery and subtotal stenosis of the right ICA with cross-filling of the left hemisphere. The intracranial arteries appeared normal.

The blood pressure was 210/120 to 170/100, hemoglobin was 13.6 gm %, and the chest x-ray was normal. The ECG showed a flattening of the T-wave in leads 3 and V6.

During endarterectomy of the right ICA, rCBF decreased from 24 to 13 ml/100 gm minute when the artery was clamped. The artery was clamped for five minutes while a bypass was inserted. Postoperatively the right-sided hemiparesis was worse. Two days later further deterioration was noted following a transient fall in blood pressure. At this time there was a transient paresis of the left arm.

Postoperative angiography showed that the stenosis of the right ICA was relieved. Intracranial arteries were normal with cross-filling of left anterior cerebral, and some of the branches of middle cerebral artery. The right external carotid artery was occluded.

Eight months postoperatively his condition was stable with slight aphasia and a spastic right-sided hemiparesis. He had been admitted to a nursing home.

CASE 8
A 65-year-old man for more than three years had had intermittent attacks of paraesthesia in the right arm lasting about 30 seconds, followed by
CEREBRAL BLOOD FLOW MEASUREMENTS

weakness of the arm. The frequency of attacks had steadily increased—at worst, six times within an hour. During the last month he had had attacks of left monocular obscurations. In the hospital, several attacks of paresis of the right arm were observed. Following angiography of the left carotid artery the paresis aggravated and the right leg was further involved.

Angiography showed an atheromatous plaque at the bifurcation of the right carotid. The proximal segments of the right anterior and middle cerebral arteries were irregular and the circulation time seemed prolonged. There was cross-filling of the left anterior cerebral artery. A left carotid angiogram showed a subtotal stenosis of ICA. The left middle cerebral artery was normal. The left anterior cerebral artery was not filled. Angiography of the aortic arch showed atherosclerosis of the right vertebral artery and a narrow, faintly outlined left vertebral artery.

The blood pressure was 140/100 and the hemoglobin was 16.7 gm %. The ECG showed a flattening of the T-wave in leads 2 and Vs. The chest x-ray showed ectasia of the ascending aorta.

Acute endarterectomy of the left ICA was performed. rCBF measurement before endarterectomy was unsuccessfully attempted. During clamping, which lasted 20 minutes, rCBF was 26 ml/100 gm . minute.

Postoperatively the pareses were unchanged and the patient had aphasia. Angiography of the left carotid artery showed relief of the stenosis. The intracranial arteries were normal. Six months postoperatively a mild right-sided hemiparesis and a slight aphasia persisted. There had been no further cerebral attacks. The patient lived alone and was able to care for himself.

CASE 18
A 69-year-old man had a three-month history of paresis of the right arm of variable severity and dysarthria. Angiography of the aortic arch showed stenosis of the left vertebral artery, and the right vertebral artery was not filled. Selective angiography of the left carotid artery showed stenosis of the ICA and normal intracranial arteries.

The blood pressure was 140/80 and the hemoglobin was 15.9 gm %. The ECG showed a left axis deviation. The chest x-ray showed ectasia of the ascending aorta.

During endarterectomy of the left carotid, rCBF decreased from 39 to 31 ml/100 . minute when the artery was clamped. The artery was occluded for 31 minutes.

Postoperatively the right monoparesis and the right facial paresis had increased. Angiography was not performed postoperatively. Admission to a nursing home was necessary.

Discussion

The marked reduction of rCBF during carotid clamping showed that these patients had a poor collateral function when acute alteration in blood flow was induced. Multiple arteriosclerotic lesions of intracranial as well as extracranial arteries probably account for this reduced collateral function. The present findings differ from those in experimental studies in monkeys, where acute occlusion of one extracranial artery causes only negligible change in total cerebral blood flow. Cerebral autoregulation may be lost during clamping of the carotid artery; cerebral vessels are unable to dilate in response to reduced perfusion pressure. This, related to induced hypercapnia, may account for some of the reduction of flow during carotid clamping.

Stagnant radioactive tracer in the segment of artery between the clamp and the circle of Willis could result in falsely low flow readings, but this was guarded against by clamping the artery only after the curve had reached its maximal height, i.e., after the total amount of tracer could be assumed to be distributed to the cerebral tissues. Further, rCBF might be low immediately after clamping but then increase as collateral circulation adjusted. If rCBF was gradually increasing during the measurement the slope of the semilogarithmic curve might be expected to increase during the first two minutes, but this was not observed.

In this series the incidence of neurological complications was high. Whether this was due to emboli or to reduced cerebral blood supply during the operation is debatable, because embolization can never be totally excluded. Postoperative angiography was performed in three of the five cases in whom neurological complications occurred. The internal carotid arteries were patent and intracranial arterial occlusion was not observed. However, Fieschi and Bozzao showed that emboli occluding intracranial arteries may later resolve. Thus, normal cerebral arteries on postoperative angiography performed after several weeks do not exclude that the symptoms were due to emboli.

The fact that the complications depended on insufficient cerebral perfusion cannot be proved but is suggested. In cases 1, 4, and 18, although rCBF during carotid clamping was not lower than in some of the cases where
complications did not occur, the time of clamping was longer, which will amplify the effect of a relative cerebral ischemia. In case 7 the reduction of rCBF to 13 ml/100 gm • minute for five minutes may have extended the apparent preoperative cerebral lesion. In case 8 neurological deterioration occurred some hours preoperatively. The immediately evident postoperative aphasia may have been a continuation of the preoperative process, a consequence of ischemia during clamping or of emboli. The ratio rCBF/occlusion time was significantly lower in the group with complications, which supports the theory of insufficient cerebral perfusion.

Though not statistically significant, there was a tendency to lower rCBF values in groups 3 and 4 than in groups 1 and 2. The higher frequency of complications in groups 3 and 4 in this as well as in other studies may depend on low rCBF values.

The critical lower level of rCBF being indeterminate, a temporary bypass was used only when rCBF was markedly reduced by the test clamping. The present findings may suggest that a bypass should be used when rCBF during test clamping falls below a level of 30 ml/100 gm • minute. This corresponds with the findings of Finnerty et al., who studied the level of cerebral blood flow at which signs of cerebral ischemia occurred in the awake man, when mean arterial blood pressure was reduced. For elderly normotensive subjects they found a critical lower level of 29 ml/100 gm • minute (range 19.6 to 35.6). The fact that lower flow values can be tolerated has been shown by Wollman et al. Under anesthesia, young men could tolerate a cerebral blood flow of 21 ml/100 gm • minute during extreme hypocapnia for periods over half an hour. Individual variations, variations of oxygen transport capacity and variations in cerebral metabolic rate, caused for instance by anesthesia or changes in body temperature, make it unreasonable to set any precise critical lower limit of cerebral blood flow. For practical purposes, however, the suggested level of about 30 ml/100 gm • minute may be useful.

The brain can tolerate total cessation of blood flow for a short period without irreversible damage, as known from patients with cardiac arrest. This suggests that when cerebral perfusion is below the level at which oxygen supply is sufficient for normal brain metabolism there will be a relation between the actual flow value and the time the brain can tolerate this reduced flow without damage. The present findings support this assumption, as rCBF values below 30 ml/100 gm • minute were tolerated if the occlusion time was short.

Various techniques have been applied to test the tolerance to carotid clamping. Maintenance of ipsilateral jugular venous oxygen saturation above 60% was advocated by Lyons et al. as a means of ensuring adequacy of cerebral oxygenation. This method could be based on the linear relationship between internal jugular oxygen tension and total cerebral blood flow. During carotid clamping, however, the reduction of cerebral perfusion may be predominantly regional, i.e., confined to the ipsilateral hemisphere, and the crucial point is to what extent the ipsilateral jugular vein actually drains the threatened region. Internal jugular blood generally is accepted as representative of mixed cerebral venous blood, although minor side-to-side differences have been demonstrated. If, though, the cortical perfusion in the territory of the internal carotid artery is decreased as a combined effect of carotid artery clamping and insufficiency of the circle of Willis, the venous contribution from the opposite hemisphere would increase relatively and thus minimize the decrease of oxygen tension in the mixed venous blood. Individual anatomical variations such as the superior sagittal sinus draining only to one lateral sinus might mean that jugular venous blood does not reflect localized cerebral hypoxia. Such factors may account for the findings of Larson et al., who reported five cases in whom unconsciousness occurred shortly after test clamping of the internal carotid artery although internal jugular oxygen saturation remained unchanged.

Some authors have found electroencephalographical monitoring useful in the identification of patients requiring internal bypass. It has been the experience in this laboratory, however, that electroencephalographical changes are difficult to interpret as the electroencephalogram is influenced not only by the cerebral blood flow but also by the depth of anesthesia and the $P_{A\text{O}_2}$ level.
CEREBRAL BLOOD FLOW MEASUREMENTS

Moore and Hall\(^1\) recently reported the application of distal internal carotid artery pressure during carotid clamping to test the adequacy of brain perfusion during endarterectomy. In a series of 48 operations they found a critical lower internal carotid artery occlusion pressure of 25 mm Hg. The measurements were performed under local anesthesia and all patients with occlusion pressure below 25 mm Hg immediately displayed signs of cerebral ischemia. These cases probably were comparable to two of the patients in this study in whom occlusion pressures below 20 mm Hg were associated with rCBF values below 15 ml/100 gm • minute. Ehrenfeld et al.\(^2\) have shown that internal carotid artery occlusion pressure decreases when \(P_{a\text{CO}_2}\) is raised from hypocapnic to normocapnic levels simultaneously with an increase of cerebral blood flow. The poor correlation between rCBF and occlusion pressure found in this study is presumably due to the wide scatter of \(P_{a\text{CO}_2}\) values. It may be that occlusion pressure can be taken as an index of rCBF in conditions of vasodilatation induced by hypercapnia, where the cerebral perfusion is directly dependent on blood pressure.\(^3\)

This study, in common with the other studies referred to, is concerned with the preservation of adequate brain oxygenation during internal carotid clamping. None of the methods measures directly the oxygen consumption in the affected hemisphere, but when the blood is normally oxygenated and rCBF does not fall below a critical point it is reasonable to assume that cerebral oxygenation is adequate. Measurement of the perfusion in that area of the brain, which normally is supplied by the internal carotid artery, but which during clamping is supplied from the collaterals, would seem to be a reliable method of detecting inadequate regional brain oxygenation.

References

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Cerebral Blood Flow Measurement as a Safeguard During Carotid Endarterectomy

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