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Objectives: to examine the relationship between the degree of extracranial internal carotid artery (ICA) stenosis and changes in the ipsilateral ICA blood flow after carotid endarterectomy (CEA).

Material and Methods: in a prospective study we studied 51 patients with unilateral 60–99% ICA stenosis (median degree 84%, asymptomatic stenosis n = 13, symptomatic stenosis n = 38). The degree of ICA diameter stenosis was determined by ex-vivo plastination of the surgically removed atherosclerotic specimen and video-assessed planimetry. Intraoperative transit time ultrasound flow measurements of the carotid arteries were performed before and after CEA. Blood flow changes were assessed by mathematical approximations. Statistics were done by use of the Wilcoxon signed Rank test.

Results: common carotid artery (CCA) and ICA median blood flow increased after CEA from 370 and 130 ml/min to 450 and 282 ml/min, respectively (p < 0.001). The relative increase of ICA blood flow was 5% and 18% for 60–69% and 70–79% ICA stenosis (n.s.) but 70% and 247% for 80–89% and 90–99% stenosis (p < 0.001 each). Mathematical evaluation (fourth-polynomal function) determined a significant increase of carotid blood flow after CEA in ICA stenosis of ≥82.3%.

Conclusions: in the absence of severe contralateral ICA occlusive disease a significant increase of ipsilateral ICA blood flow by CEA can be expected in patients with an ICA stenosis of ≥82.3% (linear degree of stenosis, ECST criteria).

Key Words: Carotid endarterectomy; Blood flow; Carotid stenosis; Carotid specimen.

Introduction

A hemodynamically impaired cerebral perfusion represents an increased risk for cerebral ischemia.1–3 Besides removal of the potential embolic atheromatous plaque restoration of cerebral blood flow may be also important in patients with high-grade stenosis of the internal carotid artery.4–14

Transit time ultrasound flow measurement is an easy-to-use method to quantify blood flow intraoperatively. Compared to electromagnetic flowmeters it is more accurate and relatively insensitive to discrepancies between probe and vessel diameter and hemoglobin concentration.15,16

Although selective angiography is considered to be the gold standard for quantification of ICA stenosis, measurements of the surgically removed atherosclerotic specimens may be even more accurate.17–22 The present analysis is based on specimen quantifications of ICA stenosis after eversion CEA.

The aim of this study was to evaluate the degree of ICA stenosis in which CEA may lead to significant improvement in carotid blood flow measured with transit time ultrasound flowmeter.

Patients and Methods

Between September 1998 and July 1999, we prospectively studied 51 consecutive patients (mean age 68 years, range 48–93) with angiographically proven unilateral 60–99% extracranial stenosis of the ICA who were operated on for asymptomatic stenosis (n = 13), amaurosis fugax (n = 11), reversible cerebral ischemia (n = 21) and minor stroke (n = 6). All CEAs
were performed under general anaesthesia. Neuro-monitoring was performed by use of somato-sensory evoked potentials. Shunt insertion led to exclusion of the study (n = 3). The median clamping time was 21 min (range 15–31 min).

Ultrasound blood flow measurements

Absolute flow data of the internal carotid artery (ICA), the common carotid artery (CCA) and the external carotid artery (ECA) were measured intraoperatively before CEA and 15 min after declamping using a transit-time ultrasound flowmeter (Transonic Systems Inc. Ithaca, NY, U.S.A.). After exposition of the carotid bifurcation a 8 mm probe cuff was selected to measure the CCA blood flow. ICA blood flow was obtained without changing the position of the probe by clamping the ECA and the superior thyriod artery. During flow measurements the patient’s systolic blood pressure, pulse rate and pCO₂ were protocolled by the anesthesiologist to ensure that local blood flow was not influenced by general hemodynamic changes or metabolically caused hyperemia. Patients with differences of the CO₂ greater than 3% before and after CEA and changes of the systolic blood pressure of more than 40 mmHg respectively were excluded from the study (n = 4).

Relative flow data before and after CEA were assessed by the ratios \( Q_{\text{pre}} = \frac{\text{ICA}_{\text{flow pre}}}{\text{CCA}_{\text{flow pre}}} \) and \( Q_{\text{post}} = \frac{\text{ICA}_{\text{flow post}}}{\text{CCA}_{\text{flow post}}} \). Since general hyperemic effects might influence ICA flow and CCA flow equally changes in this ratio after CEA were documented as \( \Delta Q\% = \frac{Q_{\text{post}} - Q_{\text{pre}}}{Q_{\text{pre}}} \times 100 \). \( \Delta Q\% \) was used as a parameter to characterize relative blood flow changes after CEA.

Determination of the degree of stenosis

In all 51 patients an intact atherosclerotic specimen was obtained by eversion CEA. The eversion specimen were imbedded in a predilated PTFE prosthesis, fixated with an histo-acryl adhesive and filled with a liquid acrylat, which is frequently used in dental medicine (Palavit M®, Co. Heraeus Kulzer, Wertheim, Germany). Pressure of 120 mmHg was applied, controlled by a manometer. After the acrylat had hardened, the specimen were incisioned and removed (Fig. 1). The obtained acrylic specimen were transversely divided at the level of the maximum stenosis and the resulting surface was stained with black ink. This surface was printed onto a white sheet of paper. We measured the minimal diameter of the surface print of the acrylic specimen. The original diameter (Do) at the level of the stenosis was assessed by measuring the outer diameter (Dspec) of the eversion specimen. Since the acrylic specimen were obtained by filling the endarterectomised specimen (which includes an intimal layer of about 0.5 mm) the original diameter (Do) could have been overestimated. As a consequence 2×0.5 mm (for both sides) were substracted from the measured outer diameter of the eversion specimen (formula: Dspec − 1 mm). In subtotal occlusions the degree of stenosis was assumed to

Fig. 1. Absolute flow distribution in the common (CCA), internal (ICA) and external carotid artery (ECA) before CEA for 60–69%, 70–79%, 80–89% and ≥90% linear specimen stenosis of the internal carotid artery.
be 99%. These measurements correspond to the criteria of the European Carotid Surgery Trial (ECST).\textsuperscript{20–22}

Mathematical and statistical evaluation

Absolute flow data in the CCA, ICA and ECA and absolute flow increase are assessed for the following groups of ICA specimen stenosis: 60–69%, 70–79%, 80–89% and \geq 90%. The degree of the ICA stenosis (specimen) and the relative blood flow changes (\(\Delta Q\%\)) were figured graphically in plot diagrams. These plots were fitted by exponential and polynomial functions. According to Hagen-Poiseuille’s (Eq. 1) and Ohm’s (Eq. 2) law blood flow is inversely proportional to the fourth power of the radius of a stenosis. Mathematically the relation between blood flow and lumen diameter can assessed by the following equations:

\[
R = (8 \times l \times \eta) / (\pi \times r^4)
\]

(1)

(\(R\) = Resistance, \(l\) = length of the vessel, \(\eta\) = blood viscosity, \(r\) = lumen radius)

\[
\Delta Q = V' \times R
\]

(2)

(\(\Delta Q\) = difference of blood pressure over an ideal vessel, \(v'\) = blood volume flow)

\[
\Delta P = V' \times 8 \times l \times \eta / (\pi \times R^4)
\]

(3)

Equation (5) shows that \(v'\) is directly correlated with the lumen radius \(r\):

\[
v' \sim r^4
\]

(5)

The degree of carotid stenosis \(X\) according to the criteria of the European Carotid Surgery Trial (ECST)\textsuperscript{20,21} and the relative changes of blood flow \(\Delta Q\%\) were calculated according to the following equations (6) and (7):

\[
X = (1 - r/D) \times 100 \Rightarrow X \sim (-r)
\]

(6)

(\(X\) = diameter degree of ICA stenosis, \(r\) = lumen radius, \(D\) = external vessel radius at the site of the stenosis)

\[
\Delta Q\% = (Q_{\text{post}} - Q_{\text{pre}}) / Q_{\text{pre}} \Rightarrow \Delta Q\% \sim 1/r^4
\]

(7)

\(Q_{\text{post}}\) = volume flow after CEA, \(Q_{\text{pre}}\) = volume flow before CEA, \(r\) = lumen radius of the vessel

Combining Eqs (6) and (7) the following relation can be found:

\[
\Delta Q\% \sim 1/X^4
\]

(8)

This equation indicates that the relative flow change \(\Delta Q\%\) is related to the degree of stenosis to \(X\) in a fourth-polynomial way. As a consequence fourth-polynomial function was applied. The turning point obtained by use of differential analysis were considered as the degree of stenosis with a significant blood flow change.

For all statistic evaluations the Wilcoxon Signed Rank test was used. A \(p\) value < 0.01 was considered to be statistically significant.

Results

The median degree of the specimen stenosis of the ICA was 83.6% (61.3–99%). The degree of stenosis was 60–69%, 70–79%, 80–89% and \geq 90% in 6, 10, 23 and 12 patients respectively.

The absolute median blood flow in the CCA, ICA and ECA was 370, 130 and 222 ml/min before CEA and 450, 282 and 150 ml/min after CEA (Table 1). Median flow increased in the CCA and the ICA (90 and 140 ml/min respectively), and decreased in the ECA (\(- 46\) ml/min). These flow changes were statistically significant (Table 2).

Table 1. Absolute blood flow in the common (CCA), internal (ICA) and external carotid artery (ECA) before and 15 min after CEA for 60–69%, 70–79%, 80–89% and \geq 90% linear specimen stenosis of the internal carotid artery. All values are given as median and range.

<table>
<thead>
<tr>
<th>Degree of ICA stenosis (%)</th>
<th>(n)</th>
<th>Absolute blood flow before CEA (ml/min)</th>
<th>Absolute blood flow after CEA (ml/min)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>CCA</td>
<td>ICA</td>
</tr>
<tr>
<td>80–89</td>
<td>23</td>
<td>370 (120–695)</td>
<td>130 (18–320)</td>
</tr>
<tr>
<td>\geq 90</td>
<td>12</td>
<td>258 (120–720)</td>
<td>35 (6–230)</td>
</tr>
<tr>
<td>60–99 (all patients)</td>
<td>51</td>
<td>370 (120–850)</td>
<td>130 (6–600)</td>
</tr>
</tbody>
</table>

Eur J Vasc Endovasc Surg Vol 25, February 2003
respectively, Table 1, Fig. 1). After CEA the median blood flow increased significantly in the CCA and the ICA in 80–89% ICA stenosis (+90 and +176 ml/min respectively) and in ≥90% ICA stenosis (+160 and +193 ml/min respectively, Table 2, Fig. 2).

The relative increase of ICA-blood flow (considering the ratio ICA/CCA blood flow) depended on the degree of ICA-stenosis and varied from 5% for 60–69% stenosis to 247% for ≥90% ICA stenosis. This increase was statistically significant for ICA stenosis ≥80%. For all 51 patients the relative blood flow improvement was 63.6% (Table 3).

Figures 3 and 4 illustrate the relation between the degree of ICA stenosis (diameter) and the relative blood flow change ΔQ%. Plots were fitted by an exponential function and by a fourth-polynomal function.
Figure 3 indicates that a significant blood flow improvement can be found only in ICA stenosis ≥80%. The differential analysis of the fourth-polynomial function (Fig. 4) delivered a turning-point on $x = 82.3\%$ (diameter stenosis). This turning-point indicates a degree of ICA stenosis in which CEA led to a mathematically significant increase of blood flow in the ICA.
Critical Degree of Carotid Stenosis

Discussion

Blood flow measurements of the carotid arteries using intraoperative ultrasound were performed only in a few number of studies. Most authors report a postoperative ICA blood flow between 200 and 400 ml/min, which is equivalent to normal blood flow data in healthy individuals.Magee et al. analyzed the influence of the angiographically measured degree of ICA stenosis on carotid blood flow before CEA. The mean ICA flow decreased in relation to the degree of stenosis from 271 ml/min with 71–80% stenosis to 208 ml/min with 81–90% stenosis. With <90% ICA stenosis a mean blood flow of 141 ml/min could be measured. We measured a median ICA flow of 160, 130 and 33 ml/min respectively (median ICA blood flow of all patients 130 ml/min). These data are lower than in the study of Magee but higher compared to the results of Gordon et al. who found a mean blood flow of only 87 ml/min for all ICA stenosis ≥70%. It remains speculative in how far these differences are caused by relevant contralateral stenosis or by differences in the determination of the degree of stenosis (NASCET criteria vs ECST criteria). Our data confirm that blood flow in the carotid arteries depends highly on the degree of ICA stenosis. This observation is true for the ICA and the CCA.

To better define the real increase of blood flow by CEA we analyzed the percentage change of the ratio ICA/CCA after CEA (ΔQ%) to make sure that there are only minimal side effects caused by reactive hyperemia or changes in cardiac output. The statistical analysis showed there is a significant blood flow increase after CEA of an ≥80% ICA stenosis. The mathematical analysis of the fourth polynomial function showed a turning point of 82.3% (diameter stenosis). These results indicate that CEA of an unilateral <80% ICA stenosis (diameter stenosis) will not result in a significant increase of blood flow. Our data confirm former results that changes in pressure and flow do not occur until the cross-sectional area of a vessel has been reduced by more than 75%. Since we did not include patients with a ≥90% stenosis or an occlusion of the contralateral ICA these results may be true only for unilateral disease. The impact of multi-vessel-disease has to be analyzed in a further series.

The hemodynamic effects of CEA have also been assessed with MRI examinations. In a recent MR-study we found a significant increase of cerebral blood volume after CEA in patients with ≥80% ICA stenosis. In other MRI studies an increase of the regional cerebral blood flow and the mean transit time after CEA were found only in patients with contralateral ICA occlusion but not in patients with a severe ipsilateral ICA stenosis. The differences in these studies may be due to differences in methodology. Furthermore different degrees of ICA stenosis were not stratified which may have led to different results especially in patients with >80% ICA stenosis.

Nevertheless our data confirm that CEA of an unilateral ICA stenosis with a diameter reduction ≥80% will result in a significant increase of ipsilateral carotid blood flow. The fourth-polynomial function of blood flow changes after CEA indicate that a diameter reduction of 82.3% (which is equivalent to an area reduction of about 90%) can be considered as the hemodynamically critical degree of an ICA stenosis. This result may help to identify patients who might benefit hemodynamically from CEA. Further studies are necessary to evaluate the clinical benefit of CEA in these patients in terms of stroke prevention especially in asymptomatic patients.

References


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