Seizures Following Carotid Endarterectomy in Patients with Severely Compromised Cerebral Circulation

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Objectives: To determine the incidence of postoperative neurological complications following carotid endarterectomy in patients with severely compromised cerebral circulation.

Design: Prospective open clinical study.

Setting: Department of Vascular Surgery, University Hospital.

Materials and Methods: We determined the incidence of postendarterectomy seizures related to haemodynamic impairment in terms of intraoperatively measured perfusion pressure in 151 patients undergoing 153 carotid endarterectomies.

Main Results: Cerebral perfusion pressure index (ICA/CCA pressure ratio) was significantly reduced (25% or more) in 47% (55/118) of patients with 70-99% stenosis compared to 6% (2/35) of patients with 30-69% stenosis (p < 0.00005). Among the 57 haemodynamically compromised patients five developed seizures considered due to cerebral hyperperfusion five to seven days after surgery. The seizures were associated with headache in two, focal neurological deficits in four and hypertensive episodes in all cases. The symptoms remitted within 2 weeks and no patients suffered cerebral haemorrhage.

Conclusions: Seizures following correction of high grade stenoses causing severe pressure reduction may be more common than previously assumed. Patients at risk should be identified and postoperative blood pressure controlled meticulously.

Key Words: Carotid endarterectomy; Cerebral hyperperfusion; Seizures.

Introduction

Cerebral perfusion pressure distal to a tight stenosis of the internal carotid artery (ICA) is reduced if collateral blood supply is impaired. Under such circumstances carotid endarterectomy may lead to transient cerebral hyperperfusion postoperatively, presumably due to failure of autoregulation. The risk of neurological complications in relation to postendarterectomy hyperperfusion was first acknowledged by Sundt. This hyperperfusion syndrome is characterised by unilateral headache, followed by seizures and, in worst cases, intracerebral haemorrhage, which may be fatal. The incidence of cerebral haemorrhage following carotid endarterectomy has previously been reported to be 0.4–1.0%. Recently, however, it has been established that carotid surgery should be restricted to patients with high grade stenosis. Since the risk of haemodynamic impairment increases with the degree of ICA stenosis, candidates for carotid surgery according to currently accepted criteria could be expected to carry a higher risk of hyperperfusion syndrome. We therefore found it of interest to determine the incidence of postoperative neurological complications related to degree of haemodynamic impairment, assessed by intraoperative pressure measurements.

Materials and Methods

From 1 January 1990 to 31 March 1993 a total of 158 patients underwent 160 carotid endarterectomies at the Vascular Service, Rigshospitalet. In seven patients intraoperative pressure measurements were not recorded and these were excluded from the study. The median age of the remaining 97 males and 54 females was 62 (range 39–77) years. The indication for surgery was focal neurological events referable to the relevant hemisphere in all but one patient. Thirty-eight (25%) patients presented with amaurosis fugax, 55 (36%)
with transient ischaemic attacks (TIA) while 59 (39%) had suffered a stroke with no or only minor residual symptoms. Any focal neurological deficit lasting more than 24 hours was considered a stroke. Finally there was one patient with an asymptomatic intimal lesion, that had developed in relation to angiography. The diameter reduction of the relevant ICA, determined by arteriography and ultrasound Duplex scanning, was 30–69% in 35 (23%) cases and 70–99% in 118 (77%). Seventy-nine (52%) patients had 0–29% contralateral ICA-stenosis, 38 (25%) 30–69% stenosis, 19 (12%) 70–99% stenosis and the remaining 17 (11%) occlusion of the contralateral ICA. Preoperative computer-tomography (CT-scanning), carried out in 135 (88%) patients, revealed hypodense areas indicating infarction in the relevant hemisphere in 38 (28%) and in the contralateral hemisphere in 10 (7%). Complicating diseases were common as 38% had hypertension, 26% cardiac disease and 11% diabetes mellitus.

Surgery was performed under halothane anaesthesia and moderate hypocapnia using EEG-monitoring. Following exposure of the carotid bifurcation mean arterial blood pressure in the common carotid artery (CCA) and the ICA distal to the stenosis were measured directly by means of a 21G cannula connected to a pressure transducer. Cerebral perfusion pressure index (CPPI) was calculated as the ratio of ICA and CCA mean pressures.2 A CPPI of 0.75 or less was considered a marked pressure reduction. Mean blood pressure in the ICA during clamping (stump pressure) was also determined. A temporary shunt was inserted in 21 cases based on continuous EEG-monitoring and stump pressure determination.

Postoperatively systolic blood pressure was kept at normal to slightly subnormal levels in patients with marked pressure reduction across the operated ICA stenosis. Labetalol, a combined alpha and beta blocking agent, was the drug of choice for controlling hypertensive episodes. Postoperative Duplex scan of the carotid bifurcation prior to discharge has been performed routinely since June 1992.

Fischer’s exact test was used for comparison of two proportions and rank sum test for comparison of three proportions. A confidence level of < 5% was regarded as significant.

Results

Preoperative CPPI was low (< 0.75) in two of 35 (6%) patients with 30–69% stenosis compared to 55 of 118 (47%) patients with 70–99% stenosis (p < 0.00005). Scatter of CPPI was considerable and increased with increasing degree of stenosis (Fig. 1). Among patients presenting 70–99% ipsilateral stenosis, 69% (11/16) with contralateral occlusion had low CPPI as opposed to 50% (8/16) with 70–99% stenosis and 42% (36/86) with less than 70% contralateral ICA stenosis (p = 0.07) (Fig. 2). Cerebral infarctions in the relevant hemisphere were demonstrated by preoperative CT-scan in 37% (19/51) of patients with low CPPI which was not significantly different from an incidence of 23% (19/84) in patients with normal or only modestly reduced CPPI (p = 0.1).

Seizures, local or generalised, developed in five (3%) patients. In all cases the seizures presented 5 to 7 days following surgery accompanied by hypertensive episodes. In two cases the seizures were preceded by
Seizures Following Carotid Endarterectomy

Table 1. Clinical and haemodynamic data in five patients with postendarterectomy seizures

<table>
<thead>
<tr>
<th>Patient</th>
<th>Indication for surgery</th>
<th>Preoperative CT-scan</th>
<th>Ipsilateral ICA stenosis</th>
<th>Contralateral ICA stenosis</th>
<th>CPPI (ICA/CCA pressure ratio)</th>
<th>Stump pressure (mmHg)</th>
<th>Shunt</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>stroke</td>
<td>infarct</td>
<td>80%</td>
<td>100%</td>
<td>0.52</td>
<td>43</td>
<td>+</td>
</tr>
<tr>
<td>2</td>
<td>AF</td>
<td>normal</td>
<td>85%</td>
<td>65%</td>
<td>0.47</td>
<td>32</td>
<td>-</td>
</tr>
<tr>
<td>3</td>
<td>stroke</td>
<td>infarct</td>
<td>90%</td>
<td>0%</td>
<td>0.50</td>
<td>30</td>
<td>-</td>
</tr>
<tr>
<td>4</td>
<td>TIA</td>
<td>normal</td>
<td>85%</td>
<td>0%</td>
<td>0.58</td>
<td>20</td>
<td>+</td>
</tr>
<tr>
<td>5</td>
<td>stroke</td>
<td>normal</td>
<td>95%</td>
<td>100%</td>
<td>0.64</td>
<td>30</td>
<td>+</td>
</tr>
</tbody>
</table>

Table 2. Symptoms and findings in five patients with postendarterectomy seizures

<table>
<thead>
<tr>
<th>Patient</th>
<th>Hypertension</th>
<th>Headache</th>
<th>Seizures</th>
<th>Neurological deficit</th>
<th>Impaired consciousness</th>
<th>Time from surgery to seizures (days)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>+</td>
<td>+</td>
<td>universal</td>
<td>none</td>
<td>+</td>
<td>5</td>
</tr>
<tr>
<td>2</td>
<td>+</td>
<td>-</td>
<td>focal</td>
<td>hemiparesis</td>
<td>+</td>
<td>7</td>
</tr>
<tr>
<td>3</td>
<td>+</td>
<td>-</td>
<td>focal</td>
<td>hemiparesis</td>
<td>-</td>
<td>6</td>
</tr>
<tr>
<td>4</td>
<td>+</td>
<td>-</td>
<td>focal</td>
<td>upper extremity paresis + aphasia</td>
<td>+</td>
<td>6</td>
</tr>
<tr>
<td>5</td>
<td>+</td>
<td>+</td>
<td>universal</td>
<td>upper extremity + facial paresis</td>
<td>+</td>
<td>5</td>
</tr>
</tbody>
</table>

headache. Four of the five patients had transient focal neurological symptoms referable to the relevant hemisphere. The neurological symptoms usually evolved over hours, varied in intensity and resolved completely within 2 weeks. A CT-scan was performed in all cases, none indicated cerebral haemorrhage. In one patient the immediate CT-scan showed hypodense areas compatible with lacunar infarctions, but a repeated scan 12 days later did not confirm this finding. Patients suffering seizures were characterised by tight stenoses of the operated ICA and two had occlusion of the contralateral ICA. CPPI ranged from 0.47 to 0.64 and stump pressure ranged from 20 to 42 mmHg. Clinical and haemodynamic data are summarised in Table 1 and postoperative events in Table 2. Elevations in blood pressure were controlled by labetalol and seizures were treated with diazepam and phenytoin. Duplex scanning confirmed ICA patency in all cases and in two a markedly increased flow velocity was found in the operated ICA (200 cm/s in both cases).

New neurological symptoms referable to the relevant hemisphere developed in nine (6%) patients, not counting the seizures. All events occurred within 2 days after surgery. In three cases the deficits were caused by thrombosis of the operated ICA, as determined by Duplex scanning. In the remaining six patients the reconstructions were patent and of these six the deficits were present immediately after recovery from anaesthesia in three, of which two had developed transient EEG-flattening during clamping, while in three cases there was a lucid interval from surgery to the occurrence of symptoms. These new neurological events resulted in significant residual deficits (major stroke) in four (3%) cases, whereas the deficits were minor in three (2%) and transient in two (1%) patients. Finally one patient, who underwent a left carotid endarterectomy experienced a transient paresis of the left leg associated with headache and

Table 3. Incidence of hyperperfusion syndrome and new neurological events not related to ICA occlusion according to haemodynamic risk factors

<table>
<thead>
<tr>
<th>Risk Factor</th>
<th>Hyperperfusion TIA/Stroke (n = 5)</th>
<th>New Neurological Events (n = 6)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ipsilateral ICA stenosis</td>
<td>30-69% (n = 35)</td>
<td>3(12%)</td>
</tr>
<tr>
<td>Contralateral ICA patency</td>
<td>Patent (n = 136)</td>
<td>5(4%)</td>
</tr>
<tr>
<td>CIPPI (ICA/CCA pressure ratio)</td>
<td>&lt; 0.75 (n = 96)</td>
<td>5(5%)</td>
</tr>
<tr>
<td>Stump pressure</td>
<td>&lt; 35 mmHg (n = 107)</td>
<td>4(9%)</td>
</tr>
</tbody>
</table>

* = p = 0.01
hypertension on the third postoperative day. The perioperative (< 30 days) mortality was zero. Table 3 shows the incidence of seizures and new neurological events unrelated to ICA occlusion — according to haemodynamic risk factors. Only perfusion pressure index had significant effect. The rate of neurological complications was neither correlated to age, hypertension, cardiac disease, preoperative neurological status nor to outcome of CT-scanning.

Discussion

In the presence of high grade carotid artery stenoses and reduced cerebral perfusion pressure carotid endarterectomy may induce temporary cerebral hyperperfusion associated with a risk of oedema and intracerebral haemorrhage.1 4-8 With the aim of correlating surgical risk to the degree of haemodynamic impairment, we assessed cerebral haemodynamics by intraoperative pressure measurements and related the pressure parameters to postoperative neurological complications in patients undergoing carotid endarterectomy.

Among patients with only moderate stenoses a significant reduction of CPPI was found in 6% as opposed to 47% in patients with severe ICA stenoses (p < 0.00005). Though CPPI was correlated to luminal diameter reduction of the ipsilateral ICA there was considerable inter-individual variation (Fig. 1) and assessment of the degree of ipsilateral as well as contralateral stenosis did not predict the haemodynamic state of the brain in individuals (Fig. 2).

Postoperatively five (3%) patients developed seizures compatible with hyperperfusion syndrome. The seizures occurred 5 to 7 days following uncomplicated surgery. They were heralded by headache in two and associated with hypertensive episodes and fluctuating focal neurological deficits. In contrast, the clinical course of the nine patients (6%) experiencing new neurological events due to thrombosis or embolism was characterised by an early (within 2 days), often abrupt onset with gradual recovery and by the absence of hypertension.

We have previously observed a marked hyperaemia following correction of high grade ICA stenoses causing severe pressure reduction.1 2 In the present study postendarterectomy seizures exclusively occurred in patients who preoperatively had reduced CPPI making cerebral hyperperfusion a likely cause. In support of this, Duplex scanning revealed a significantly increased velocity in the operated ICA in two cases. Similar cases of seizures occurring several days after correction of high grade stenosis have been described by others.5 13 Reigel et al. reported on 10 cases of seizures in a series of 2439 patients undergoing carotid surgery.14 Cerebral blood flow (CBF) measurements carried out in seven patients revealed a significant increase in flow immediately following surgery and the authors concluded that the events were part of a hyperperfusion syndrome.

Hyperperfusion syndrome is considered to be an uncommon complication, previously reported in 1% of patients undergoing carotid endarterectomy.5 14 In recent years, however, multicentre trials have established that surgery should be restricted to patients with symptomatic stenoses exceeding 70%. According to our results reduced CPPI can be expected in about half of these cases and hyperperfusion related complications appear to be a substantial risk. In this study, intraoperative pressure measurements identified 57 haemodynamically compromised patients, of whom five developed symptoms compatible with hyperperfusion five to seven days after surgery. Without surgical intervention patients with severely compromised cerebral circulation would be expected to carry a high risk of embolic15 and haemodynamic strokes.16 Therefore, the benefits of carotid endarterectomy and restoration of cerebral perfusion pressure seem to outweigh the increased surgical risk in this group.

A devastating complication of postendarterectomy hyperperfusion is intracerebral haemorrhage. As cerebral blood flow seems to be pressure dependent until autoregulation is regained, blood pressure control has been recommended as a means of reducing the risk of haemorrhage in haemodynamically compromised patients.3 8 14 17 Analysis of ultrasound Doppler waveforms, obtained distal to the stenosis may identify patients with severe pressure reduction.18 In our institution, patients found to be at risk remain in the intensive care unit for intraarterial blood pressure monitoring the first 24 hours and blood pressure is kept at normal to slightly subnormal levels. Using this regime no cases of haemorrhage were observed among the five patients who presented symptoms of hyperperfusion.

In conclusion, seizures as part of the hyperperfusion syndrome is not a rare complication in modern carotid surgery. We recommend preoperative assessment of cerebral perfusion pressure followed by meticulous blood pressure control throughout the first week after surgery in patients with compromised cerebral circulation.
References


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