Cerebral Blood Flow, Metabolism and Mean Arterial Pressure Changes Following Unilateral Internal Carotid Endarterectomy: Cerebral Ischemia and Elevated Systemic Arterial Pressure

BY F. HAVEN JONES, M.D.,* MARK L. DYKEN, M.D.,* AND ROBERT KING, M.D.†

Abstract:
Cerebral blood flow and metabolism studies were performed on 13 patients before and after unilateral carotid endarterectomy for severe atheromatous occlusive disease. Mean brachial arterial pressure was monitored during these studies. Postoperative studies compared to a control group revealed a rise in cerebral blood flow and a fall in mean arterial pressure and cerebral vascular resistance. The clinically recorded blood pressure in the study group compared to closely matched control patients with cerebral vascular disease showed a similar but not significant difference. Ten patients studied following ligation of a common carotid artery had a small but significant increase in mean systemic arterial pressure. These results suggest a relationship between systemic arterial pressure and cerebral ischemia. Some possibilities for this relationship are discussed.

Additional Key Words
intracranial aneurysms angiography common carotid artery ligation ulcerated plaques transient ischemic attacks

The purpose of this communication is to report the changes in mean arterial blood pressure (MAP), cerebral blood flow (CBF) and metabolism in 13 patients who have undergone internal carotid endarterectomy for severe atheromatous occlusive disease and to compare these changes to 11 control patients with cerebral vascular disease without operation. In addition, the changes in MAP were compared (1) to a matched group of 13 patients, and (2) to ten patients who had common carotid artery ligation for bleeding intracranial aneurysms.

Methods
Thirteen patients who had cerebral blood flow studies before and after unilateral internal carotid endarterectomy were studied. This group consisted of 12 men and one woman, with a mean age of 61.2 ± 7.4 years. The degree of carotid occlusion ranged from 50% to 99% (nine patients over 70%). All had ulcerated or irregular plaques documented angiographically. Three patients had old cerebral infarctions, eight had transient ischemic attacks, one had disseminated cerebrovascular disease, and one had greater than 99% occlusion of the left internal carotid artery and dementia. Significant associated diseases included five cases of diabetes mellitus and three established abnormalities of lipid metabolism.

All patients were extensively evaluated. This workup included routine medical laboratory and radiographical procedures, ECG, EEG, brain scan, skull films, cerebrospinal fluid examination, serum lipids and lipoprotein electrophoresis,
neuropsychological testing, four-vessel angiography, ophthalmosonometry, and average cerebral blood flow studies. Postoperatively, each patient had angiographical visualization of the carotid artery system on the side of surgery and a second cerebral blood flow and metabolism study. The latter was performed four to 15 days postoperatively (mean 7.7 days).

Cerebral blood flow (CBF), cerebral oxygen utilization (CMRO₂), and cerebral vascular resistance (CVR) were determined by the krypton 85 desaturation technique of McHenry1 or the argon desaturation technique utilizing the clinical mass spectrometer.2 The mean arterial pressure (MAP) was continuously monitored via a brachial artery catheter. The responses of CBF, CMRO₂, CVR, and MAP to the inhalation of 5% CO₂ and 100% O₂ were recorded in each patient.

In the first part of this study, these parameters were compared with those obtained from a group of control patients with cerebral vascular disease, each of whom had two cerebral blood flow studies performed during hospitalization without intervening surgery. This control group consisted of 11 patients, five men and six women, with a mean age of 58.4 ± 9.7 years. Of these, eight had acute cerebral infarction; one had intracerebral hemorrhage; one had transient ischemic attacks; and one had an old cerebral infarction and occlusion of one carotid artery. The average time between CBF studies in the study group was 16.4 ± 5.8 days, in the control group 31.7 ± 22.3 days.

In the second part of this study, the surgically treated patients were compared with a second control group of patients with CVD with respect to change in blood pressure during hospitalization, the blood pressure being measured by the standard sphygmomanometer. This control group was matched with the study group as closely as possible for age, sex, and clinical diagnosis. The recorded blood pressures of each subject in the two groups were averaged over (a) the first two hospital days on which at least two blood pressures were recorded and (b) the last two hospital days on which at least two blood pressures were recorded. For each subject, a clinical MAP was calculated for time segments (a) and (b) according to the formula MAP equals mean diastolic pressure plus one-third mean pulse pressure. The changes in blood pressure in the two groups during hospitalization were then calculated and compared.

A third control group consisted of the last ten patients in our institution to have common carotid artery ligation without permanent neurological deficit for bleeding intracranial aneurysms. The group consisted of eight women and two men, with a mean age of 44.4 ± 11.9. All recorded arterial blood pressures on each day of hospitalization were averaged. From this the average mean pressures were calculated. Two comparisons were made: (1) The MAPs were averaged for each three-day interval before and after the day of the surgical procedure. The mean of the means of the three days preceding surgery was compared to the mean of each three-day interval after the day of surgery. (2) The MAP for one day before surgery was compared to that of a day approximately 16 days later after surgery to match the time interval between blood flow measurements in the study group. The significance of the change was calculated by a t test for nonindependent samples and the difference between the two groups by a test for independent samples.

Results
The CBF and metabolism data in the study and control groups are displayed in table 1. The study group showed a significant increase in CBF (P < 0.05), a decrease in MAP (P < 0.05), and a decrease in CVR (P < 0.005) postoperatively. CMRO₂ did not change significantly. No significant changes occurred in the response of CBF, CMRO₂, CVR, or MAP to inhalation of 5% CO₂. Data in the control group were insufficient to allow analysis of the responses to 100% O₂ inhalation.

Comparison of the clinically calculated MAP revealed an 8.5 ± 10.2% decrease in the surgically treated group and only 2.9 ± 6.5% decrease in the CVD control group. The difference is not statistically significant.

Those patients who had common carotid artery ligation had an average MAP percentage increase for all three-day intervals until the ninth (see fig. 1). The increase of MAP of 7.4% for the first three days was significant (P < 0.005), as was the increase of 5.8% for the second three-day period (P < 0.05). In no other group did the significance of the differences of the means reach the 0.05 level.

The MAP was selected at random for a day previous to the operation and taken as near as possible to 17 days following the first. The first MAP was taken 6.0 ± 2.7 days before surgery and the second MAP was taken 10.2 ± 3.0 days after surgery. The total time between the two determinations was 16.2 ± 1.2. The MAP increase of 4.9% was significant.
CEREBRAL ISCHEMIA AND ELEVATED SYSTEMIC ARTERIAL PRESSURE

Discussion

We have noted a significant rise in CBF and fall in MAP and CVR following surgery in a group of patients undergoing internal carotid endarterectomy for symptomatic atherosclerotic occlusive disease at the carotid bifurcation. To our knowledge, this is the first study of the effect of carotid endarterectomy on cerebral blood flow and metabolism which utilizes a control series. Unfortunately, the surgically treated group and the control group were poorly matched. When the clinically measured MAP changes in the surgically treated group were compared with a well-matched group with CVD, a similar trend was demonstrated but this was not statistically valid.

In animals, chronic hypertension cannot be caused by cerebral ischemia unless the carotid sinus nerves are disrupted. It has been reported that, because of the relatively low incidence of carotid artery occlusion in routine series of hypertensive patients and the variability of hypertension in patients with carotid artery occlusion, cerebral ischemia is probably never a cause of chronic hypertension among conscious ambulant patients. Although a rise in mean systemic arterial blood pressure following common carotid artery ligation has been noted in small series, larger studies have not reported this.

Our ten patients who had ligation of the common carotid artery demonstrated a statistically valid rise in MAP in the first and the second three-day time period following occlusion. Although the mean pressure remained elevated during subsequent three-day time periods until the ninth (25 days postocclusion), in no other three-day period was the difference significant. A tendency for a gradual decrease in the elevation occurred until during the last two time periods the mean pressure was actually less than the preoperative measurements. Of course, after the fourth time period the total number of patients is too small to be of any value other than to suggest a trend. One is tempted to postulate that initial cerebral ischemia produced a hypertensive response which in the presence of intact carotid sinuses was overridden by baroceptors.

(P < 0.005). The difference between this increase and the mean decrease of 8.5% in the postendarterectomy group was also significant (table 2).

Discussion

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Adams et al.\(^8\) showed no significant postendarterectomy changes in CBF, CMRO₂, CVR, MAP, or cerebral metabolic rate in a group of patients with stenotic carotid bifurcation lesions, but their study included no control series. Furthermore, the degree of internal carotid artery occlusion in these patients was not specified. This may be important inasmuch as the most likely explanation for the postoperative increase in CBF observed in our

![Figure 1](image-url)

**TABLE 2**

**MAP Changes Following Endarterectomy Versus Carotid Ligation**

<table>
<thead>
<tr>
<th></th>
<th>Age</th>
<th>Days prep</th>
<th>Days postop</th>
<th>Days 1st to 2nd</th>
<th>1st MAP</th>
<th>2nd MAP</th>
<th>% Diff</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Endarterectomy</strong></td>
<td>Mean</td>
<td>61.2</td>
<td>8.7</td>
<td>7.7</td>
<td>16.4</td>
<td>106</td>
<td>96</td>
</tr>
<tr>
<td></td>
<td>S.D.</td>
<td>7.4</td>
<td>5.1</td>
<td>3.1</td>
<td>5.8</td>
<td>15</td>
<td>15</td>
</tr>
<tr>
<td></td>
<td>N</td>
<td>13</td>
<td>13</td>
<td>13</td>
<td>13</td>
<td>13</td>
<td>13</td>
</tr>
<tr>
<td><strong>Ligation</strong></td>
<td>Mean</td>
<td>44</td>
<td>6.0</td>
<td>10.2</td>
<td>16.2</td>
<td>86</td>
<td>90</td>
</tr>
<tr>
<td></td>
<td>S.D.</td>
<td>11.9</td>
<td>2.7</td>
<td>3.0</td>
<td>1.2</td>
<td>7.9</td>
<td>9.2</td>
</tr>
<tr>
<td></td>
<td>N</td>
<td>10</td>
<td>10</td>
<td>10</td>
<td>10</td>
<td>10</td>
<td>10</td>
</tr>
<tr>
<td><strong>Significance difference</strong></td>
<td>P &lt; 0.001</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

MAP = mean arterial pressure. % Diff. calculated from total of individual differences.
study group would seem to be the alleviation of mechanically obstructed flow in the diseased internal carotid artery. Because at least a 70% attenuation of a single carotid lumen is required to alter blood flow significantly, a difference in the extent of atherosclerotic disease in the two patient groups could account for the discrepancy between our observations and those of Adams et al.

We assume that the observed decrease in blood pressure in our patient group represents a decrease in response to the stress of the cerebral blood flow procedure, and further that this decrease in stress response is related to the fact of surgery. One possible explanation for this is that carotid stenosis and consequent impairment of cerebral blood flow induces a hypertensive response, mediated chemically in a manner similar to the mechanism inherent in the hypertensive response to renal ischemia, and that relief of the stenosis changes this process. Severe atherosclerosis was frequently present in both internal carotid artery bulbs, and this process may interfere with the overriding effect of the carotid sinus.

A second possibility is that the change in stress response is an artifactual, psychological phenomenon, incumbent upon the fact of major surgery, without being specifically related to carotid surgery. Although ligating a common carotid artery is not an ideal operative procedure to use as a control, if indeed the changes were related only to a nonspecific surgical stress phenomenon, the changes would be expected to be similar in both groups. In actuality the increase in MAP following ligation was significantly different from the decrease in MAP following endarterectomy and would tend to disprove this hypothesis (table 2).

Summary

Cerebral blood flow and metabolism studies were performed on 13 patients before and after unilateral carotid endarterectomy for severe atheromatous occlusive disease. Mean brachial arterial pressure was monitored during these studies. Postoperative studies compared to a control group revealed a rise in cerebral blood flow and a fall in mean arterial pressure and cerebral vascular resistance. The clinically recorded blood pressure in the study group compared to closely matched control patients with cerebral vascular disease showed a similar but not significant difference. Ten patients studied following ligation of a common carotid artery had a small but significant increase in mean systemic arterial pressure. These results suggest a relationship between cerebral ischemia and systemic arterial pressure.

References


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