Cerebral Blood Flow during Carotid Endarterectomy

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SUMMARY
Cerebral blood flow (CBF) was measured during 28 carotid endarterectomies in 25 patients, by injection of $^{133}$Xe into the exposed internal carotid artery (ICA). Twenty-three patients had episodic cerebral or retinal ischemia and five had neurologic deficits before operation. Initial CBF values were variable. Increases of $P_aCO_2$ caused CBF to increase in four of six patients, to increase slightly in one, and to decrease in one. In each of 19 procedures, CBF decreased during surgical occlusion of the ICA, 11 times to less than 30 ml/100 g/min, but absence of postoperative worsening indicated that decreased ICA blood flow is not a major risk of the procedure; embolization from the site of operation may be a greater threat to the patient. CBF increased after 14 endarterectomies, perhaps due to failure of autoregulation or to reactive hyperemia. Measurements of jugular $P_{VO}_2$ and lactate concentration were of little value.

Additional Indexing Words:
Internal carotid artery occlusion Autoregulation Cerebral emboli

Despite the frequent use of internal carotid endarterectomy for the surgical treatment of cerebral vascular disease, the effects of the procedure on the cerebral circulation are largely unknown. Studies of blood pressure and blood flow in the internal carotid artery (ICA) during endarterectomy are difficult to interpret because of the many distal arterial anastomoses. Measurements of the oxygenation of jugular venous blood often provide an inaccurate assessment of the status of the cerebral circulation. The blood vessels that regulate cerebral blood flow (CBF) may not react normally during carotid occlusion or ischemia, and technics for study of the circulation of the entire brain or even of one hemisphere may not detect disturbances or changes of CBF in ischemic regions. Thus, we and others have measured regional CBF during carotid endarterectomy to determine the effects of the surgical procedure on blood flow in brain tissue, to determine whether information about CBF is useful during the operation, and to relate changes of CBF to postoperative neurologic deficits.

Material and Methods
CBF was measured 86 times during 28 carotid endarterectomies in 25 patients whose ages ranged from 42 to 75 years; 20 were men. Twelve patients were hypertensive and four had diabetes. Twenty-three had episodes of transient cerebral or retinal ischemia before operation, and three of these had superimposed cerebral infarcts. The two other patients had cerebral infarcts without transient ischemic episodes but with severe ICA stenosis. Nineteen patients had carotid bruises, and retinal artery pressures were asymmetric in 16. Emboli were noted in the retinal arterial vessels of six patients on preoperative funduscopic examination.
Twenty procedures were done for severe atherosclerotic stenosis of the ICA (greater than 75% decrease of the diameter of the lumen of the vessel, demonstrated roentgenographically); bilateral procedures were done on one patient, several weeks apart. Four procedures were done for moderate stenosis (approximately 50% decrease in diameter); bilateral procedures were done on one patient, and in this patient the stenotic lesions appeared to be ulcerated. Stenosis of the remaining four arteries was minimal but the atherosclerotic plaques appeared to be ulcerated. Occasionally there were associated lesions of other cervical or intracranial arterial vessels. Most procedures were done with moderate induced hypercapnia and hypertension. A bypass shunt was used only once, for a patient who had an occluded ICA on the side opposite the endarterectomy. One patient had transient incoordination of the opposite hand after endarterectomy; there were no other cerebral deficits related to the operations.

CBF was measured by the rapid injection of approximately 300 μCi of 133Xenon, in 0.3–0.6 ml of isotonic saline through a 27-gauge needle into the exposed ICA distal to the stenosis.10 A scintillation detector was located over the regions of brain in the distribution of the middle cerebral artery. After discrimination at a lower level of 75 keV, pulses were recorded on magnetic tape and monitored with a rate meter and an analog chart recorder. At the time of operation, CBF was calculated from the half-times of the first parts of the clearance curves of 133Xe recorded on the analog recorder;10 these values were available within 2–4 min of the injection of the indicator. Subsequently, pulses were counted with a digital scaler and high-speed printer, and CBF values were calculated by the kinetic ("H/A") method and by exponential analysis for two rate constants, using a digital computer.5,7,10,11 Each method provided values for CBF in ml/100 g/min; there was reasonably close correspondence among the values obtained by the different methods of calculation.11

CBF was measured before and after each endarterectomy, while the ICA was exposed. In addition, in six procedures CBF was measured before and after increases of PaCO2 for the endarterectomy. CBF was measured during surgical occlusion of the ICA in 19 procedures on 16 patients, by injecting the indicator and then immediately applying a Scoville clip to the artery for the endarterectomy. The times of occlusion ranged from 14 to 33 min.

Jugular venous blood oxygen tension (PvO2), arterial blood oxygen tension (PaO2), and arterial and venous blood oxygen content (calculated from values for Po2, oxygen saturation, and hemoglobin concentration) were measured during 13 procedures (table 1). Arterial and jugular venous blood lactate concentrations were measured during nine procedures.

**Results**

**CBF before Endarterectomy.** There was considerable variability in the CBF values obtained before the 28 endarterectomies (fig. 1). At PaCO2 ranging from 34 to 48 torr (mean ± sd, 42.9 ± 3.7 torr), CBF ranged from 11.9 to 98.5 ml/100 g/min (mean ± sd, 58.9 ± 24.6 ml/100 g/min). The lowest value was obtained from a patient with recent onset of a severe neurologic deficit and a nearly occluded ICA. Three other patients with neurologic deficits had CBF values lower than the mean, but there was no consistent relationship between CBF and the presence of a deficit. Seven patients had CBF less than 40 ml/100 g/min; two of these had neurologic deficits.

**PaCO2 and CBF.** Increases of PaCO2 before endarterectomy caused appropriate increases of CBF in three patients who did not have preoperative neurologic deficits, including the one patient who had a transient postoperative deficit (fig. 2). However, an increase of PaCO2 caused an appropriate increase of CBF in only

| Table 1 |
|-------------|-------------|-------------|-------------|
| **Effects of Occlusion of Internal Carotid Artery** |
| Occlusion | C(a – v)O2 (ml/100 ml) | Jugular PVO2 (torr) | Jugular lactate (mEq/liter) |
| Mean | sd | N | Mean | sd | N | Mean | sd | N |
| Before | 2.65 | 0.99 | 13 | 60.6 | 13.4 | 13 | 1.00 | 0.28 | 9 |
| During | 3.02 | 1.28 | 13 | 58.5 | 14.7 | 13 | 0.99 | 0.43 | 9 |

Abbreviations: C(a – v)O2 = calculated difference between arterial and venous blood oxygen contents; PVO2 = venous blood oxygen tension.
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Figure 1

(Left) Cerebral blood flow (CBF) values were obtained before and after 28 endarterectomies, at similar PaCO₂. Heavier lines indicate values from one patient with a transient postoperative neurologic deficit (CBF: 48.9 before, 54.5 after) and five patients with preoperative neurologic deficits. (Right) Means of CBF values obtained before and after endarterectomies were not significantly different.

one of three patients with neurologic deficits; of the two others, one had an impaired response and one (who had a recent onset of a severe neurologic deficit) had a paradoxical reaction (decrease of CBF after the increase of PaCO₂).

Surgical Occlusion of ICA. In each of 19 procedures, CBF decreased during occlusion of the ICA for the endarterectomy (fig. 3). The magnitude of the decrease was not related to the degree of carotid stenosis. In 11 instances, CBF decreased to less than 30 ml/100 g/min. The mean (±SE) of the CBF values (at similar PaCO₂ before, during, and after occlusion) was 67.5 ± 21.2 ml/100 g/min before occlusion, 33.3 ± 14.2 during occlusion, and 67.3 ± 21.9 after occlusion. The mean of the values obtained during occlusion was significantly lower than the other means (P < 0.001). There was no worsening of the patient's neurologic status after any of these 19 procedures.

After endarterectomy and removal of the clip from the ICA, CBF usually increased to a value near that obtained before occlusion. However, in several patients there was a remarkable increase of CBF, and in one

Figure 2

Effects of increases of PaCO₂ on CBF in six patients before carotid endarterectomy.
patient CBF was lower after release of the clip than during occlusion (fig. 3).

**CBF before and after Endarterectomy.** For 14 procedures the CBF values obtained after endarterectomy were greater than those obtained before; for the 14 other procedures there was no change or CBF was slightly less after endarterectomy (fig. 1). There was no significant difference between the means of the values (mean ± sd: before, 58.9 ± 24.6 ml/100 g/min; after, 64.1 ± 23.4). In the one patient with a transient neurologic deficit related to the operation the value for CBF was greater after endarterectomy than before.

**Discussion**

The variability of the CBF values obtained before endarterectomy was expected, because of differences of Paco2, anesthesia, perfusion pressure, and blood constituents (such as hemoglobin concentration), and because of the natural variability of CBF from one person to another. The initial CBF values did not reflect the presence or absence of a neurologic deficit, but those patients with deficits and relatively normal CBF values may have had decreased CBF earlier, at the time of cerebral infarction. In studies done by others, CBF values have not always reflected the presence of a neurologic deficit, even without the artificial circumstances of anesthesia and surgery.

Despite recent evidence that blood vessels of ischemic brain may not always respond to stimuli in the same way as do blood vessels of normal brain, most surgeons perform endarterectomies with induced hypertension and hypercapnia. The dangers of the indiscriminate use of hypercapnia are exemplified...
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by the patient who had the recent onset of a severe neurologic deficit: with hypercapnia, CBF decreased. Paradoxic reactions of this sort ("intracerebral steal") have been demonstrated repeatedly in animals and humans,

although they are unusual, transient, and generally occur only shortly after the onset of severe ischemia. With the rapid method for calculating CBF from the initial slope of the clearance curve of \(^{133}\text{Xe}\),

paradoxic reactions can be recognized during the surgical procedure and attempts to modify \(P_{\text{ACO}_2}\) can be abandoned. The effects of changes of other anesthetic and systemic factors likewise can be determined, so that the circumstances can be modified if indicated.

Methods other than those that measure volume flow of blood in brain tissue are of doubtful value for assessment of the cerebral circulation. For example, in the cases reported here, jugular \(P_{\text{VO}_2}\) and lactate concentrations did not change appreciably during surgical occlusion of the ICA despite decreases of CBF. Lactate does not readily cross the blood-brain barrier, and measurements of jugular \(P_{\text{VO}_2}\) reflect the average oxygenation of the entire brain or, at best, one hemisphere; thus the effects of regional decreases of CBF may not be detected.

Despite distal arterial anastomoses, such as those of the circle of Willis, surgical occlusion of the ICA invariably caused decreases of regional CBF. However, the decreased perfusion did not cause postoperative neurologic worsening even in the patients who had decreases of CBF to very low values (less than 30 ml/100 g/min). These results differ from those of others who found postoperative neurologic deficits to be much more frequent and indicate that embolization from the site of operation is probably the major risk of the procedure rather than a decrease of the flow of blood through the ICA.

The absence of postoperative neurologic worsening in patients with low CBF values and moderately long periods of carotid occlusion (20–25 min) is in contrast to situations that occur with respiratory arrest, cardiac arrest, or total circulatory arrest, with which irreversible neurologic deficits frequently develop within 4–5 min. The present data provide additional evidence that neuronal function can be preserved for a much longer time if there is even minimal flow of oxygen-containing blood to ischemic regions of brain than if the flow of blood stops completely.

No conclusions about the long-term effects of carotid endarterectomy on CBF can be made from these data. Although increases of CBF were found after half the endarterectomies, the measurements were made shortly after the removal of an occluding clip from the ICA, and the increases of CBF may have represented reactive hyperemia in response to the preexisting ischemia or an inability of the regulatory arterial vessels to constrict to maintain CBF relatively constant despite the increase of perfusion pressure.

Decreases of CBF after endarterectomy may have been related to hyperreactivity of ischemic regulatory vessels to the sudden increase of perfusion pressure, to an influx of accumulated endogenous vasoactive agents, to changes of blood volume associated with the surgical procedure, or to other factors as yet unknown. Studies of CBF before and weeks to months after endarterectomy are needed.

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