Cerebral Protection During Carotid Endarterectomy

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Abstract: Cerebral Protection During Carotid Endarterectomy

Carotid endarterectomy was performed on 73 occasions using the following regimen during the operative period: (1) general anesthesia with normothermia, (2) maintaining a normal or slightly elevated arterial oxygen tension (\(P_{\text{aO}_2}\)), (3) maintaining a normal or low arterial carbon dioxide tension (\(P_{\text{aCO}_2}\)), (4) maintaining systemic arterial pressure (SAP) at normal or slightly elevated levels, (5) employing a bypass shunt in all cases, and (6) utilizing transient systemic anticoagulation with heparin. The rationale for this regimen is explained in terms of current concepts of cerebral blood flow and metabolism. No new neurological deficit has appeared after operation using these techniques.

Additional Key Words: autoregulation of cerebral blood flow, anticoagulation, barbiturates, halothane anesthesia

The goals of carotid endarterectomy are to prevent strokes and to relieve the troublesome symptomatology associated with transient cerebral ischemia. Interest in the operation has grown enormously in recent years, and several large series of patients have been reported. As experience with the operation has accumulated, patient selection and operative indications have become more refined. As with every operation, it is important that the benefits from operation exceed the risks inherent in the procedure. If carotid endarterectomy is to be performed on patients with extracranial occlusive cerebrovascular disease who have not had strokes, then every effort must be made to prevent ischemic brain damage during the operative period.

During the past four years we have incorporated some newer concepts of cerebral blood flow and metabolism into a regimen designed to protect the brain from ischemia during the operative period. This report describes the method used on a series of patients.

Methods

Patients were selected for operation who had clinical pictures compatible with transient cerebral ischemia or a previous cerebrovascular accident from which they had recovered or that had stabilized. The presence of occlusive vascular disease at the carotid bifurcation was demonstrated by arch aortography and selective common carotid arteriography. Pancerebral angiography was performed in every case. Ophthalmodynamometry, radioisotope flow studies, brain scanning, and electroencephalography were employed when necessary for diagnosis. Using these criteria, 73 operations were performed in 62 patients over a four-year period.

The regimen used to protect the brain during the operative period consisted of: (1) general anesthesia with normothermia, (2) maintaining a normal or slightly elevated \(P_{\text{aO}_2}\), (3) maintaining a normal or low \(P_{\text{aCO}_2}\), (4) maintaining SAP at normal or slightly elevated levels, (5) employing a bypass shunt in all cases and (6) transient systemic heparinization.

Results

Since instituting this regimen, we have had no increased neurological deficit in the immediate postoperative period. The duration of follow-up is too short to draw conclusions about the long-term effect of preventing a stroke. Only one of the operated patients has had a thrombotic stroke at the time of this writing, and it occurred in the distribution of the nonoperated carotid artery. One patient required reoperation eight hours postoperatively. Clinical evaluation in this case suggested occlusion of the operated carotid artery and angiography confirmed the complication. The artery was reopened and systemic anticoagulation with heparin was continued for several days.

Because of our reluctance to reverse the heparin totally with protamine, several hematomas have occurred at operative sites. It has not been necessary to drain these collections, and no infectious complications have resulted.

Labile blood pressure in the immediate postoperative period has occasionally been troublesome. We have not had instances of hypertension that did not respond to simply elevating the head of the bed. Postoperative hypotension has generally been responsive to the administration of intravenous medication.
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fluids, colloid, or blood. On one occasion it was necessary to infuse neosynephrine for a short period of time.

Postoperative angiography was routinely employed early in the series. The studies generally demonstrated dilatation of the operated arterial segment in those instances where the intravascular diameter varied from normal (fig. 1).

Only one of the patients has been less able to function following operation than he was prior to it, although he is able to care for himself. Several patients who were not working prior to operation have returned to work following amelioration of their transient ischemic attacks.

Discussion

Local anesthesia has been widely used for carotid endarterectomy in the past. The theoretical advantages of local anesthesia are minimal alteration of normal cardiovascular and respiratory dynamics, and the ability to monitor the patient's level of consciousness and neurological status. We believe general anesthesia has proved to be more suitable for a variety of reasons. Incidental advantages include the ability to control the patient's respirations and maintain the arterial Po2 and Pco2 at optimal levels. The potential hazards of airway obstruction or agitation in the demented, uncooperative, or otherwise debilitated patient are avoided. Of perhaps greater importance is the significant cerebral protection afforded by general anesthesia per se.

There is a decrease in energy-yielding reactions in the brain during anesthesia induced by barbiturates and other general depressants. Current evidence suggests that this is the result of reduced neuronal activity rather than the cause. Cerebral oxygen uptake, heat production, and lactic acid production are correspondingly diminished, the latter tending to eliminate the deleterious effects of cerebral acidosis. The result would seem to be better tolerance to periods of hypoxia during carotid endarterectomy when general anesthesia is used. Even anesthetic doses of barbiturates do not seem to exert any specific direct action on cerebral vessels. In those cases where CBF is briefly reduced by the barbiturate, it does not fall as much as the CMRO2, so adequate oxygen is available to the brain when other variables are controlled.

Halothane and most volatile general anesthetics dilate cerebral blood vessels and increase total CBF in man. In general, there is a corresponding increase in ventricular pressure, which is thought to be secondary to a generalized increase in venous pressure. Cerebrovascular responses to change in Paco2 are not altered. This dilatation of cerebral vessels and resultant increased CBF is of questionable value, and it may be that a decreased CMRO2 is the major advantage of general anesthesia during carotid endarterectomy.

When general anesthesia is instituted via endotracheal tube, the PaO2 can be maintained at normal or slightly elevated levels. By this method adequate saturation of blood going to the brain is insured. In addition, the potentially deleterious effect of diminished CBF from an elevated Paco2 is avoided. Several papers have appeared recently which continue to advocate the inhalation of 5% CO2 during the operative period in order to maximally dilate cerebral blood vessels. The value of this technique is questionable in light of evidence that an elevated Paco2 may produce a considerable increase in blood flow through normal brain and a diminution of flow through adjacent areas of ischemic brain. When significant arterial disease exists, maximal arterial dilatation in diseased segments most likely exists chronically. There is experimental evidence to suggest an elevated Paco2 acts to dilate normal vessels and "steal" blood from ischemic areas. These regional variations in blood flow, as well as the variable patterns of intracranial circulation, make jugular venous gas determinations of uncertain value as an
adjunct to operation. A normal or elevated Po₂ in jugular venous blood is no assurance of adequate oxygenation of ischemic areas of brain. Any increase in blood flow through diseased arterial segments following CO₂ administration would seem to be secondary to the pressor effect of CO₂ and not to vasodilatation. As we do not use CO₂ inhalation, the potential hazards of cardiac depression and dysrhythmia have been avoided.

It has been our practice to maintain SAP at normal or slightly elevated levels. CBF varies directly with the blood pressure and inversely with the cerebrovascular resistance. Resistance through diseased segments would seem to be fixed at a constant level as a result of decreased vascular compliance. In normal arterial segments the vascular resistance rises in response to elevations in SAP under the principle of autoregulation of CBF. It therefore seems important to maintain the other variable of SAP at normal or slightly elevated levels in order to avoid transiently decreasing CBF. This precaution is particularly important during the anesthetic induction period.

During an earlier experiment one carotid artery was occluded in the goat, an animal that has no significant CBF supplied by the vertebrobasilar system, and blood flow was measured in the contralateral carotid artery at various values of SAP. When one vessel was occluded, autoregulation did not occur until nearly normal total CBF was attained through the patent carotid artery. Until that value was reached, flow through the open carotid artery varied linearly with SAP. These data suggest that elevations in SAP may increase total CBF in situations where it has been chronically reduced by extracranial occlusive vascular disease.

A recent study by Fourcade et al. included measurements of carotid stump and ipsilateral venous sinus pressure measurements at various levels of SAP and Paco₂. The data suggested that an area of ischemia may exist within the distribution of the occluded carotid artery and, because of regional vasomotor paralysis, blood flow to this area is increased by hypocarbia and systemic hypertension. On the other hand, total CBF is probably not markedly decreased by even occlusion of a single carotid artery. Cautious use of mild induced hypertension might be of value during the operative period in patients with combined carotid and vertebrobasilar insufficiency, although a recent study tends not to support that conclusion.

We advocate the use of a bypass shunt in all cases. For this reason we do not feel that induced hypertension is necessary or even safe. There is an acute increase in blood flow past diseased arterial segments when the shunt is put in place, and the added impetus of elevated SAP would seem to increase the risk of hemorrhage into ischemic brain.

The theoretical disadvantages of using a bypass shunt are increased duration of the operative period and inability to do an adequate endarterectomy with the shunt in place. The former objection is eliminated by using general anesthesia. The few minutes added to the operation do not add significant risk to the operation and the period of cerebral ischemia is actually reduced. We have found it necessary to suture the distal intimal flap to the vessel wall in some cases, a maneuver which is not always possible when a shunt is not used. Patch grafting has been unnecessary in this series of patients since enlarged lumen diameter at the site of the endarterectomy has been the rule (fig. 2).

Systemic heparin is used routinely, in doses of 10,000 units. There is some evidence that heparin affords an added measure of cerebral protection which is not related to decreased blood viscosity. Microvascular sludging and stasis appear to be the in-

FIGURE 2
Postoperative arteriogram of the same patient. Note the diameter of comparable areas opposite the black arrow.
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Initiating events in thrombosis leading to cerebral infarction. These events may be delayed when sluggish flow occurs after systemic heparin has been given. We have not employed acetazolamide during the operative period as has been recommended by others.\(^{18}\) We attempt to preserve the carotid body in every instance in order to reduce the lability of SAP in the postoperative period.\(^{18}\)

A recent report by McDowell\(^ {20}\) describes a similar regimen employed in 440 carotid endarterectomies with neurological worsening in 2.2% of patients. An internal shunt was not employed in most of his cases. Another series of 103 cases reported by DeWeese et al.\(^ {32}\) indicated a 6% incidence of increased neurological deficit following operation, including one death from stroke. Others have employed various aspects of this regimen with success.\(^ {22,23}\)

Patient selection variables make comparisons among the reported series difficult. Continued improvement in surgical technique and perioperative care are essential to prevent neurological deficit from carotid endarterectomy. The above regimen appears to be rational, is based on current concepts of cerebrovascular physiology, and has been employed with modest success.

References


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