Microsurgical endarterectomy under barbiturate protection: a prospective study

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Carotid endarterectomy has the potential to improve on the natural history of untreated carotid artery disease with respect to subsequent infarction in symptomatic patients with causative angiographic lesions. This benefit of a reduced risk of stroke can be realized only if the perioperative morbidity and mortality rates are kept low. An approach to symptomatic carotid artery bifurcation disease is outlined, with a defined protocol of microsurgical endarterectomy utilizing barbiturate protection during the period of potential focal temporary cerebral ischemia. This protocol includes preoperative antiplatelet therapy, barbiturate anesthesia, the avoidance of an internal shunt, the use of the operating microscope, and strict control of postoperative hypertension. A series of 200 consecutive endarterectomies performed within this protocol in 180 patients and the resultant combined permanent morbidity and mortality rate of 1.5% are reported.

KEY WORDS: endarterectomy • carotid artery • barbiturate • microsurgery

Several studies have demonstrated that patients with appropriate clinical symptoms who have significant ipsilateral angiographic disease of the carotid artery bifurcation experience a decreased risk of stroke following a "successful" carotid endarterectomy. Success in this case is defined by the absence of perioperative mortality or permanent neurological morbidity. The combined rate of perioperative stroke and death has ranged in various series from 1.5% to more than 20%. The benefit of carotid endarterectomy in stroke prevention is negated when complication rates are in the upper range. It is therefore incumbent on surgeons to make every effort to achieve and maintain complication rates that are at an irreducible minimum.

It is not clear how one can consistently achieve a "successful" endarterectomy. The literature is replete with articles on variations of surgical technique, intraoperative shunting, monitoring, anesthesia, and anti-thrombotic therapy intended to enhance the safety of the operation. After analyzing the relevant clinical and experimental data, we have established a protocol designed to take into consideration and thus avoid the causes of each type of major perioperative complication. This protocol includes: 1) barbiturate administration in every case to protect the brain during the period of ischemia accompanying carotid artery clamping; 2) the avoidance of an internal shunt in order to eliminate the possibility of trans-shunt embolization and shunt-related intimal injury; 3) use of the operating microscope to aid in the removal of atherosclerotic plaque and placement of fine sutures to close the arteriotomy without stenosis; 4) perioperative aspirin therapy and delayed heparin reversal to minimize thrombus formation at the endarterectomy site; and 5) strict control of postoperative blood pressure to avoid hypertension with its risk of intracerebral hemorrhage. This protocol was applied prospectively to 200 procedures in 180 patients who were managed by one of us (R.F.S.). The experimental and clinical data that support these tactics are discussed and the results of this protocol are reported.

Clinical Material and Methods

Patient Characteristics and Grading

The clinical characteristics of each patient and the lesions depicted on their preoperative angiographic studies were recorded prior to surgery on a cerebrovascular data sheet. Grading of the degree of surgical risk
TABLE 1

| Correlation between physical status and neurological classification* |
|------------------|------------------|------------------|------------------|
| Anesthesia Grade | Sundt Grade      | Total            |                  |
|                  | I                | II               | III              | IV               | Cases |
| ASA I            | 0                | 0                | 2                | 0                | 2     |
| ASA II           | 14               | 30               | 9                | 0                | 53    |
| ASA III          | 40               | 34               | 32               | 12               | 118   |
| ASA IV           | 0                | 4                | 6                | 17               | 27    |
| total            | 54               | 68               | 49               | 29               | 200   |

* For definitions of the American Society of Anesthesiologists (ASA) and the Sundt classifications see text.

was carried out according to the criteria of Sundt and associates, as follows: Group 1: neurologically stable patients with no major medical and no angiographically defined risks, with unilateral or bilateral ulcerative-stenotic carotid disease; Group 2: neurologically stable patients with no major medical risks, but with significant angiographically defined risks; Group 3: neurologically stable patients with major medical risks, with or without significant angiographically defined risks; and Group 4: neurologically unstable patients, with or without associated major medical or angiographically defined risks. The physical status for each patient was documented according to the classification system of the American Society of Anesthesiologists (ASA), as follows: ASA I: a normal healthy patient; ASA II: a patient with mild systemic disease that limits activity but is not incapacitating; ASA III: a patient with severe systemic disease that limits activity but is not incapacitating; ASA IV: a patient with an incapacitating systemic disease that is a constant threat to life; and ASA V: a moribund patient who is not expected to survive for 24 hours with or without an operation.

The 200 consecutive procedures were performed in 180 patients, 108 men and 72 women. The average age for the men was 62 years (range 40 to 83 years) and for the women it was 68 years (range 50 to 80 years). Associated medical illness included heart disease in 64% of patients, hypertension in 58%, and diabetes mellitus in 12%; 56% of the patients were smokers. The patients sought medical attention for the following reasons: transient ischemic attacks or minor strokes in 81% of cases, amaurosis fugax in 28%, and prophylaxis in 7%. Some patients had both visual and hemispheric symptoms.

Correlation of the ASA grade and Sundt classification in this series of patients is presented in Table 1, and the preoperative angiographic findings are summarized in Fig. 1. In general, the patients had more extensive cerebrovascular disease than was expected. Thirty-one of the patients were referred after they became hemiplegic and comatose within seconds of carotid artery occlusion under local anesthesia at the time of planned (then aborted) carotid endarterectomy.

**Preoperative Care**

The patients selected for endarterectomy were scheduled for surgery as soon as possible unless a major stroke, recent myocardial infarction, or other major medical illness necessitated a delay. All patients awaiting elective endarterectomies were given 325 mg aspirin three times daily for at least 2 but preferably 5 days prior to surgery. Patients with emergency endarterectomies were given aspirin at the time of neurosurgical consultation if they were not already taking it. Those receiving heparin because of fluctuating neurological deficits continued to do so until the conclusion of surgery.

**Anesthetic Technique**

General endotracheal anesthesia was used in all cases. Premedication of patients was dependent on preoperative cardiovascular and neurological status. Either diazepam (10 mg) and atropine (0.4 mg) or no premedication was given. Following preoxygenation, anesthesia was induced with thiopental sodium (3 to 5 mg/kg) and muscular paralysis was achieved using pancuronium or vecuronium bromide (0.1 mg/kg). Cardiovascular response to intubation was attenuated with either lidocaine (1.0 mg/kg) or fentanyl (0.05 to 0.1 mg). Anesthesia was maintained with a nitrous oxide-oxygen mixture (providing an inspired oxygen concentration of at least 40%) and isoflurane (0.5% to 1.5%). Respiration was controlled and end-tidal CO₂ was maintained between 35 and 38 torr. The patient was kept sufficiently hydrated with glucose-free intravenous fluids. Monitoring included direct arterial blood pressure measurement, end-tidal CO₂, arterial blood gas analysis, temperature, and electrocardiographic recording. Monitoring of cerebral electrical function was carried out...
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FIG. 2. Operative photograph and diagram showing excision of the proximal atherosclerotic plaque from the common carotid artery (CCA). ICA = internal carotid artery; ECA = external carotid artery.

by compressed spectral analysis of the raw electroencephalographic (EEG) data, using a two-channel online Neurotrac spectral analyzer. The dissection and proximal ICA is kept to a minimum in order to avoid dislodging embolic material from the atherosclerotic plaque. Meticulous attention to hemostasis is observed. The Shaw hemostatic scalpel† is used routinely. This heated blade facilitates the achievement of a completely dry operative field, even when the patient has been fully heparinized prior to and during surgery. The bifurcation of the carotid artery (at the carotid body) is not injected with a local anesthetic because we found no difference in blood pressure control whether lidocaine, saline, or no injection was utilized.

Prior to carotid artery clamping, heparin (100 U/kg) is given intravenously. Bolus doses of thiopental sodium (125 to 250 mg) are given until 15- to 30-second burst suppression is achieved, as indicated by the compressed spectral analysis of the EEG recording. Thiopental is continued by small bolus injections (if a short clamp time is anticipated) or by constant infusion at a rate necessary to maintain burst suppression. Phenylephrine infusion is occasionally required to maintain systemic pressure in the patient’s normal range. During and after barbiturate infusion, the isoflurane is reduced in concentration or is discontinued entirely. Thiopental is discontinued when flow is reestablished through the ICA.

A No. 9 French malleable multiperforated suction tube ‡ is placed adjacent to the common and internal carotid arteries, and fixed by stapling it to the surgical drapes. The distal ICA is occluded first, using an aneurysm clip. This is immediately followed by occlusion of the proximal common carotid artery with a vascular clamp and occlusion of the superior thyroid artery and the external carotid artery trunk with temporary aneurysm clips. The arteriotomy is begun in the common carotid artery and is extended beyond the termination of the atherosclerotic plaque in the ICA. The appropriate plane of dissection between the plaque and normal media is identified. The atherosclerotic plaque is separated from the vessel wall and carefully dissected first from the ICA and then from the external carotid artery. The proximal end of the common carotid artery is inverted and the plaque is cut circumferentially 1 to 2 cm below the end of the arteriotomy (Fig. 2). This allows plaque removal below the end of the arteriotomy incision, making closure of the vessel easier and avoiding proximal stenosis.

At this point the operating microscope is positioned such that both the surgeon and his assistant have unimpeded binocular vision. By employing a 300-mm lens and the mobility of a Contraves stand, the microscope is a welcome addition instead of a hindrance. The ability to appreciate the multiple tiny fragments of atherosclerotic material and abnormal intima that remain loosely adherent to the vessel wall after removal of the plaque has convinced us of the benefit of the microscope. We have not found the superior lighting and magnification of the microscope to be equaled by other techniques.

The loose filaments attached to the media are removed meticulously. This part of the dissection and

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† Shaw hemostatic scalpel manufactured by Oximetrix Inc., Mountain View, California.
‡ Multiperforated suction tube manufactured by Microvac, PMT, Inc., Hopkins, Minnesota.
§ Contraves stand manufactured by Carl Zeiss, Inc., Thornwood, New York.
TABLE 2
Temporary nonischemic postoperative complications

<table>
<thead>
<tr>
<th>Complication</th>
<th>Cases</th>
<th>Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>wound hematoma</td>
<td>3</td>
<td>1.5</td>
</tr>
<tr>
<td>recurrent laryngeal palsy</td>
<td>7</td>
<td>3.5</td>
</tr>
<tr>
<td>12th nerve paresis</td>
<td>3</td>
<td>1.5</td>
</tr>
<tr>
<td>infection</td>
<td>1</td>
<td>0.5</td>
</tr>
<tr>
<td>parotid fistula</td>
<td>2</td>
<td>1.0</td>
</tr>
<tr>
<td>total cases</td>
<td>16</td>
<td>8.0</td>
</tr>
</tbody>
</table>

Fig. 3. Postoperative angiogram showing enlargement of the vessel. The arrows denote the limits of the endarterectomized segment.

TABLE 3
Ischemic postoperative complications

<table>
<thead>
<tr>
<th>Complication</th>
<th>Cases</th>
<th>Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>transient ischemic attack</td>
<td>4</td>
<td>2.0</td>
</tr>
<tr>
<td>stroke</td>
<td>2</td>
<td>1.0</td>
</tr>
<tr>
<td>death (hypertensive bleed 4 days postop)</td>
<td>1</td>
<td>0.5</td>
</tr>
<tr>
<td>total cases</td>
<td>7</td>
<td>3.5</td>
</tr>
</tbody>
</table>

vessel closure is carried out under continuous heparinized saline irrigation, which is a constant task of the assistant. Irrigation fluid and blood are cleared from the wound by the Microvac suction tube. The distal ICA is carefully inspected, and any elevation of the intima beyond the end of the plaque is carefully trimmed with microscissors. If the step-off is abrupt and the intima is loosely adherent to the media, 7-0 monofilament tacking sutures are inserted. The artery is closed using a running 6-0 monofilament suture, starting at the distal end of the arteriotomy. Prior to final closure, back bleeding from all the vessels is permitted to expel air and debris from the lumen of the repaired segment. The clip on the superior thyroid artery is not replaced; this allows continuous back bleeding during placement of the final sutures and avoids air being trapped in the vessel. Small closely spaced stitches placed under the microscope produce a tight nonleaking suture line without narrowing the vessel. (Postoperative angiography routinely demonstrates enlargement of the vessel throughout the length of the arteriotomy (Fig. 3).) The arteries are reopened in the usual manner: external carotid artery, common carotid artery, brief reclosure of the common carotid artery, opening of the ICA, and reopening of the common carotid artery. This allows any potential embolic material to be washed into the external carotid artery system. Except for a few blood cells seeping from the needle holes, the arteriotomy should be completely dry. The neck incision is closed after complete hemostasis has been assured. A subcuticular closure technique is used and the skin edges are approximated with Steristrips. The heparin is usually not reversed except when wound hemostasis is difficult to achieve, in which case it is 50% reversed with protamine sulfate after a delay of at least 10 minutes following vessel reopening.

Postoperative Care

Many patients are obtunded in the initial recovery period and require continued intubation until they are alert (usually for 30 to 60 minutes, occasionally longer). Despite the obtundation, brain-stem function can be tested, and the patients will move their extremities in response to stimulation, permitting neurological evaluation immediately after arriving in the recovery room. Blood pressure is carefully monitored and hypertension immediately controlled with an infusion of sodium nitroprusside. Systolic pressure is maintained below 170 torr except in patients with long-standing severe hypertension. In these patients a slightly higher pressure is tolerated. Postoperative hypotension, reported in the past by other authors, has been unusual in our experience. The patient is monitored in the intensive care unit for 24 to 48 hours after surgery. As soon as the patient can tolerate liquids, aspirin is restarted and continued indefinitely.

Results

The postoperative complications included temporary nonischemic sequelae (Table 2) and both temporary and permanent ischemic complications (Table 3). There were no complications in Sundt Grade I and II patients (Table 4) and no physical complications despite the extensive use of barbiturates (Table 5). In particular, there were no recognized pre- or intraoperative myocardial infarctions, although temporary electrocardiographic changes with ST depression were seen on occasion.
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Seven patients presented with complete ICA occlusions on angiography. The recent onset of multiple transient symptoms and extensive angiographic retrograde flow into the ICA siphon suggested that the ICA occlusions were new. All seven patients underwent emergency endarterectomy. In two of these patients the ICA could not be successfully opened at surgery; in four patients long clots with proximal plaque formation were removed, and in the seventh patient (who presented with a stroke in evolution) the ICA was patent at the conclusion of surgery (although with very poor back flow). The fourth patient had a transient postoperative ICA occlusion; he underwent unsuccessful repeat surgery and his stroke progressed (described above). The overall angiographic patency of the endarterectomies, when the ICA was open at the completion of surgery, was 99.5% (153 of 154 cases).

Discussion

Carotid endarterectomy has a demonstrated capacity to reduce the incidence of stroke in patients with ischemic symptoms due to carotid artery atherosclerosis.4,29,82 These patients face a 5% to 7% yearly risk of stroke, and successful endarterectomy (uncomplicated by stroke or death) reduces this risk by approximately 65%.1,78,81,82 Neither platelet antiagregant nor anti-coagulant therapy has been shown to convey the same degree of protection from stroke that carotid endarterectomy has the potential to provide.22,20 However, in order for the potential benefits of surgical treatment for carotid artery atherosclerosis to be realized, the incidence of serious perioperative complications must be held to an absolute minimum.38 Recent surgical series from several large institutions have demonstrated that current anesthetic and surgical techniques provide the means to accomplish carotid endarterectomy with low rates of postoperative stroke and death.2,19,27,74

Because of the devastating nature of the complications of this procedure, surgical techniques and perioperative management must be continually reexamined and refined with the goal of reducing the risks of surgery to the lowest possible level. Operative and perioperative tactics must be developed that have the capacity to abort the pathophysiological events responsible for major complications. The neurological morbidity of carotid endarterectomy arises from: 1) intraoperative embolization; 2) cerebral ischemic injury during the period of carotid artery clamping; 3) postoperative embolization or thrombosis originating at the endarterectomized segment; and 4) postoperative intracerebral hemorrhage.6,12,19,21,41,52,55,72,74,76
Intraoperative Embolization

Embolization may occur during exposure of the carotid artery, presumably due to dislodgement of thrombotic or atherosclerotic material from the bifurcation during carotid artery clamping. Careful dissection that minimizes manipulation of the bifurcation and proximal ICA reduces the chance of embolization. In addition, a clip is first applied to the distal ICA, prior to clamping the common carotid artery. This avoids sending atherosclerotic debris from the site of common carotid artery clamping into the intracranial circulation. Embolization of air, debris, or thrombus from the operated segment of the vessel may also occur at the time of deocclusion. This is avoided by opening the vessels in a specific pattern. Back bleeding from the ICA and flushing of the carotid artery bifurcation before completion of the arteriotomy closure are carried out as described in the Operative Procedure section (above).

Cerebral Ischemia During Carotid Clamping

Ischemia during the period of carotid artery clamping is a potential cause of neurological complication that has received a great deal of attention. In fact, the occurrence of a critical degree of ischemia during operative carotid artery occlusion must be unusual, as attested to by the fact that several surgeons have achieved excellent results without taking any specific measures (such as shunting) to protect the brain during carotid artery clamping. However, a certain small group of patients will experience a profound and potentially injurious drop in cerebral blood flow with carotid artery clamping. In the large series studied at the Mayo Clinic, 8% of patients had occlusion flows of less than 10 ml/100 gm/min. On the basis of laboratory studies, it can reasonably be assumed that persistence of ischemia of this magnitude for more than a few minutes will cause significant cerebral damage in a large number of these cases. Ferguson and coworkers (unpublished data, 1985) have also identified a group of patients with a high risk of intraoperative hemodynamic stroke: namely, those patients who have both a major EEG change after clamping and an ICA stump pressure of less than 25 mm Hg. There seems little doubt that in certain patients hemispheric hypoperfusion due to temporary carotid artery occlusion will result in infarction. The temporary insertion of a vascular shunt has most commonly been employed to protect the brain during the period of occlusion. Some surgeons have used a shunt in every case in an effort to provide maximal protection while obviating the need for intraoperative brain monitoring. The use of a shunt is not without risks of its own, however. Shunt use exposes the patient to the risks of air embolization during insertion, and embolization of atherosclerotic material may occur through the shunt. The shunt may cause an intimal injury to the internal or common carotid artery and promote postoperative embolization or thrombosis. Finally, the presence of the shunt may limit exposure of the critically important distal end of the atherosclerotic plaque and impede precise completion of the endarterectomy.

In order to minimize the risks of shunting itself, some surgeons have used shunts only in selected patients, basing the decision on intraoperative measurements or monitoring (including carotid artery stump pressure, EEG, or cerebral blood flow). Unfortunately, the predictive value of these techniques is inadequate to reliably identify only those patients who truly require shunting to avoid infarction. Elevation of the blood pressure during carotid artery clamping has been used to promote collateral flow to the ischemic hemisphere. This has the capacity to prevent or reverse changes on the EEG, and presumably can avert ischemic injury. Induced hypertension, particularly in patients with severe atherosclerosis, prior cerebral infarction, or coronary artery disease, carries its own liabilities. Even the low risks of intracerebral hemorrhage or perioperative myocardial infarction may negate the benefits of this type of cerebral protection. The concept of cerebral protection, without the risks associated with shunt insertion or induced hypertension, is appealing. At this point in time, the only established and widely validated agents for pharmacological protection against ischemic brain damage are the barbiturates. In an effort to provide maximum cerebral protection with minimum coincident risk, we have administered barbiturates prior to and during operative carotid occlusion and have avoided shunting.

Barbiturates

Barbiturates reduce the metabolic requirements of neural tissue. This may be responsible for extending the tolerance of the brain for the reduction in substrate supply that occurs during ischemia. While barbiturate administration has been shown to reduce cerebral blood flow, this effect is most dramatic in nonischemic regions. Shapiro has postulated that barbiturate-related increases in vascular resistance in nondiseased areas of the brain may shunt the flow into regions of critically reduced cerebral blood flow in focal cerebrovascular disease. Other neurochemical mechanisms may also play contributory roles in barbiturate-induced cerebral protection. Regardless of the mechanism, barbiturates (specifically, thiopental and pentobarbital) clearly have the capacity to modify or prevent cerebral injury due to focal ischemia. Barbiturate therapy is most effective if the agent is administered prior to a period of temporary focal ischemia. Our laboratory experience with a primate model has established that preemptive administration of barbiturates provides dramatic cerebral protection even during 6 hours of middle cerebral artery occlusion. Michenfelder and Mihle and Nehls, et al. (in preparation), found that the degree of protection with barbiturates far surpasses that provided by other general anesthetic agents. Barbiturates provide less protection...
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after the onset of ischemia and appear to convey no protection (and probably are deleterious) if given in the setting of permanent vascular occlusion. The planned temporary ischemic period during carotid endarterectomy would appear to be an ideal application for barbiturate therapy.

Gross and associates reported the use of barbiturate cerebral protection in carotid endarterectomy; however, they did not administer barbiturates routinely. Thiopental was given only if a trial of carotid artery clamping produced a significant EEG change that was not reversed by the pharmacological induction of hypertension. Of their 41 patients who underwent endarterectomy, seven required barbiturates; none of the seven developed an ischemic deficit. A subsequently treated patient in whom barbiturates were required (described in an addendum to their paper) awoke from anesthesia with a hemiparesis.

We have administered barbiturates prior to carotid artery cross-clamping in all cases. Thiopental was given until burst suppression was achieved. This has been determined to be the appropriate endpoint for effective barbiturate cerebral protection. Arterial blood pressure was maintained at a normal level during the period of occlusion. Routine use of barbiturate protection permits the performance of a precise unhurried endarterectomy. This technique also provides the time that may be needed for complicated arterial reconstructions, such as vein grafting for recurrent stenosis, segmental arterial resection for redundancy or kinking, or interposition vein grafting. By eliminating the pressure that is related to occlusion time, the surgeon can proceed in a meticulous manner. We agree that a "rush" endarterectomy is dangerous because of the disastrous consequences of even minor technical errors.

The administration of barbiturates to all patients who underwent endarterectomy proved to be feasible and safe. No complications were recognized as being caused by barbiturate therapy. The drawbacks of barbiturate administration are limited to intraoperative hypotension and a prolonged wake-up time after surgery. The judicious use of vasopressors and volume expansion reversed the modest hypotension that accompanied barbiturate infusion in a small number of cases. Many of the patients required continued intubation in the recovery room, but seldom for more than 1 hour after the completion of surgery. Neurological evaluation was possible, however, as brain-stem reflexes were preserved and the patients would invariably move in response to painful stimulation within minutes of arriving in the recovery room. Most importantly, in our experience barbiturate therapy appeared to be effective in protecting the brain during surgery. Only one patient awoke from surgery with a new neurological deficit. This complication appeared to be due to embolization rather than to the effects of hypoperfusion during cross-clamping.

Barbiturate therapy may have provided unexpected benefits. Symptomatic myocardial ischemia as a peroperative complication was not encountered in our patients. Barbiturate therapy specifically may have played a role in the avoidance of cardiac complications. The use of barbiturates obviated the necessity for induced hypertension during carotid artery clamping and may have blunted the spontaneous hypertensive response that often accompanies carotid artery clamping and cerebral hypoperfusion. Barbiturates also decrease cardiac output, and through these effects may reduce the myocardial stress that is associated with surgery. It is also possible that barbiturates exert an as yet undefined direct protective effect.

Postoperative Embolization and Thrombosis

The risks of embolization or hemodynamic insufficiency during surgery for carotid atherosclerosis have been emphasized. Embolization or thrombosis that originates in the postoperative endarterectomized segment of the vessel may, however, be responsible for a greater percentage of the neurological complications associated with this operation. Deficits from these causes may occur from minutes to days after completion of the procedure. Many of these events are directly related to postendarterectomy structural irregularities of the vessel wall.

Operating Microscope. Residual fragments of atherosclerotic plaque, intimal flaps, and vessel stenosis all cause blood flow abnormalities that can predispose to intravascular thrombus formation, embolization, and occlusion. Such major technical defects have been identified by postendarterectomy intraoperative angiography in 5% to 25% of cases. All of these structural defects can be avoided by meticulous unhurried attention to technical detail during performance of the procedure.

In order to refine the technical performance of carotid endarterectomy we have used the operating microscope, which is employed just after removal of the atherosclerotic plaque. The magnification and illumination of the operating microscope make possible a more precise separation and more complete removal of the small fragments of plaque and diseased intima that remain adherent to the vessel wall after endarterectomy. The most critical region of the vessel, the distal end of the ICA plaque, can be visualized easily. A defect at this point can be identified and managed by excision of the elevated intimal edge or by the accurate placement of fine intimal tacking sutures. With the use of the microscope, a precise suture of the arteriotomy can be made with small closely spaced bites of the vessel wall. Each stitch can be seen to include the appropriate amount of adventitia and media. The infolding of fragments of adventitia into the lumen is avoided. A careful closure is particularly important at the distal end of the arteriotomy, in which the use of large bites and widely spaced stitches can easily cause significant stenosis of the ICA. The results of postoperative angiography, performed as a routine 2 to 4 days after
surgery, have borne out our opinion that the advantage of the magnification and illumination of the operating microscope provides improved technical results. Postoperative mural irregularities and ICA stenosis have been very rarely demonstrated angiographically in our cases (Fig. 3).

Antithrombotic Therapy. As noted above, the process of thrombosis at the site of endarterectomy appears to be responsible for many postoperative ischemic complications. The exposed media in the operated segment is highly thrombogenic. In experimental studies, endarterectomy and postendarterectomy intraoperative angiograms have shown significant mural thrombus formation within minutes of vessel deocclusion.4.7,16 The thrombogenic process is frequent enough to result in ICA occlusion in as many as 5% of cases.7,50,58 Both platelet aggregation and activation of the intrinsic coagulation mechanism are involved in the formation of thrombus in the operated vascular segment.50,25 The removal of the atherosclerotic plaque. With resumption of flow, platelets adhere to the collagen and eventually form a pseudoendothelial monolayer that is thought to be nonthrombogenic.5,25,55 Coincident with adhesion, the platelets release several active compounds, including adenosine diphosphate and thromboxane A2, which are potent platelet aggregators.44 However, platelet adherence to collagen, which is critical for the formation of the nonthrombogenic platelet monolayer, is not aborted by aspirin.38 Aspirin administration, in a dose equivalent to that which we use, has been demonstrated to be effective in preventing arterial thrombosis after canine carotid endarterectomy.26 Further, aspirin therapy has been shown to improve patency rates in coronary artery bypass grafts.14,15

The use of aspirin has been avoided by some surgeons in the belief that platelet inhibition may predispose to postoperative hemorrhagic complications, particularly intracerebral bleeding.74 Intracerebral hemorrhage has been a very rare complication in our experience, and we believe that its risk does not outweigh the benefits of aspirin therapy. Absolute wound hemostasis may be somewhat more difficult to achieve, but delayed heparin reversal and the use of the Shaw scalpel have solved this problem.

Only two patients had postoperative carotid artery thrombosis in this series. In one case this was explained by the coexistence of a severely stenotic ipsilateral siphon lesion, and in the other by an elevated intimal flap which was corrected uneventfully.

Postoperative Intracerebral Hemorrhage

Intracerebral hemorrhage is a particularly devastating, although uncommon, complication of carotid endarterectomy.6,12,19,74 The incidence of this complication has varied from series to series, and its occurrence has been related to several patient profile characteristics. Postoperative intracerebral hemorrhage occurs more frequently in patients with a history of completed (and particularly recent) cerebral infarction, in hypertensive patients, and in patients receiving anticoagulant medications.6,19,21,52,74,79

Sundt, et al.,74 identified a small group of patients who appear to have an unusually high risk of hemorrhage. These patients fell into the Sundt Grade IV category, had high-grade carotid artery stenosis, and showed evidence of marked cerebral hyperperfusion after endarterectomy. The only patient who had a fatal complication in our series fell into this group. This hypertensive patient developed an intracerebral hemorrhage 4 days after removal of a preocclusive carotid plaque. Sundt's group and others5,74 have speculated that these patients have developed paralysis of autoregulation in the chronically under-perfused hemisphere. The removal of the obstructive carotid lesion causes focal hyperperfusion that manifests as migrainous headache, focal seizures, and intracerebral hemorrhage. These events may be analogous to the normal perfusion pressure breakthrough phenomenon that can accompany the removal of large cerebral arteriovenous malformations.65,67,70 In both cases, chronically hypoperfused brain is suddenly exposed to a dramatic increase in perfusion.

Postoperative neurological complications have a com-
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pelling relationship to postoperative hypertension. This applies particularly to the complication of intracerebral hemorrhage. The most important factor in reducing the incidence of postendarterectomy hemorrhage is to strictly control the postoperative blood pressure. Blood pressure control is particularly critical in patients who are categorized into the high-risk group for hemorrhage: those with prior infarction or those with particularly tight carotid artery stenosis. The occurrence of the warning signs of migraineous headaches and focal seizures should prompt judicious blood-pressure reduction and discontinuation of anticoagulant or platelet antiaggregant drugs. Platelet antiaggregant drugs alone do not appear to cause a significant increase in the risk of intracerebral hemorrhage. The incidence of hemorrhage in our series (0.5%) is not significantly different from that in other series in which the patients did not receive routine perioperative aspirin.

Hypertension in the immediate postoperative period was rather common in our patients. The most effective method of controlling blood pressure was to administer a constant infusion of sodium nitroprusside. In patients with preoperative hypertension and in those in whom the blood pressure began to rise at the end of the procedure, we often began the infusion in the operating room. Postoperative hypotension has been reported as a frequent complication in these patients, and is probably related to hypovolemia. With adequate intraoperative fluid therapy, none of our patients had significant postoperative hypotension.

Conclusions

No single study can definitively identify the specific elements of management that will reduce the perioperative complication rate for a procedure to its absolute minimum. The theoretical advantages of our management protocol and the documented safety of this protocol in practice has been presented. When carotid endarterectomy can be performed with the low incidence of serious complications associated with this protocol, the procedure can be expected to have a significant positive impact on stroke incidence in appropriately selected patients.

Addendum

Since the acceptance of our manuscript, we have changed our approach slightly with respect to shunting selected patients undergoing carotid endarterectomy. Rather than categorically not shunting any patient, we have elected to insert a shunt in those patients in whom a major EEG change is observed at the time the vessels are occluded (despite barbiturate protection to the point of EEG burst suppression). In the last 75 procedures, two patients had shunt placement. This change in our philosophy followed an experience with a patient who underwent an otherwise uncomplicated carotid endarterectomy, but showed a significant EEG change despite barbiturate burst suppression; postoperatively, this patient had an ipsilateral hemispheric infarct.

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