Correlation of Postoperative and Two-year Follow-up Angiography with Neurological Function in 99 Carotid Endarterectomies in 86 Consecutive Patients

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Eighty-six consecutive patients in 1982 underwent 99 endarterectomies and routine postoperative digital subtraction angiography. Ten vessels were closed primarily and 89 with a patch graft. Minor morbidity was 2%, major morbidity 0%, and mortality 1%, but these varied according to the patient's preoperative medical and neurological function and angiographic findings. Postoperative patency for the common carotid artery (CCA) and internal carotid artery (ICA) was 100% and for the external carotid artery (ECA) 97%. Seventy-nine vessels were evaluated by a DSA 2 years after surgery. There was one asymptomatic occlusion in follow-up and one symptomatic re-stenosis in a patient with a proven heparin induced hypercoagulability state. The three postoperative ECA occlusions were associated with a lethal postoperative stroke, the only ICA occlusion in follow-up, and a 50% stenosis of the CCA in follow-up at the site of ECA occlusion. Vein patch grafting protected the ICA but not the CCA from recurrent stenosis. The carotid slim sign on preoperative angiograms is judged to indicate a patient at high risk of stroke morbidity.

RECENTLY, there has been an appropriate increase in interest concerning the recurrence of carotid stenosis following endarterectomy.[1-5] Some of these reports have been directed toward the frequency of symptomatic recurrence, while others have been based on evidence of re-stenosis from noninvasive testing. Two reports of large series have suggested that symptomatic recurrence is in the order of 1 to 2%.[6,7] However, an actuarial analysis of 151 consecutive patients undergoing carotid endarterectomy for focal cerebral symptoms by Whisnant et al.[8] revealed a 2% stroke rate per year, with two-thirds occurring ipsilateral to the side of endarterectomy; and DeWeese,[9] after studying carefully major reports of long-term follow-up,[10-16] concluded that 10% of patients develop recurrent symptoms if followed for a sufficient period of time. Furthermore, noninvasive testing has suggested that recurrent carotid stenosis greater than 50% or occlusion occurs in 10–50% of patients undergoing a carotid endarterectomy.[3-5,17-20] In most of the studies to date, cases were not consecutive and either the vessel was closed primarily or no information was given regarding the type of arterial closure.

There have been no reports in which a well-defined series of consecutive patients has been routinely re-examined angiographically in follow-up to determine the vascular durability of the operation. This is the subject of our report.

Methods and Case Material

Design of Study

From January 1982 through December of 1982, all patients undergoing a carotid endarterectomy on one of our neurovascular surgical services underwent postoperative digital subtraction angiography (DSA). When possible, these 86 patients (73 unilateral endarterectomies and 13 bilateral endarterectomies) were re-examined by DSA in 1984 at a point approximating two calendar years from the date of surgery and the angiograms compared and correlated with the patient's neurological function. Two patients were lost and six died in follow-up. The rate of vessel follow-up by DSA in living patients was 86% and the rate of clinical follow-up was 98%.

Preoperative Evaluation

All patients had detailed neurological examinations prior to surgery and were categorized according to criteria previously established for the identification or assessment of risk factors for patients with primary (*i.e.*, nonrecurrent) atherosclerotic stenosis.[21] Four risk groups are used:

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Submitted for publication: August 6, 1985.

Group I are those patients under age 70 physiologically with no medical or neurologically determined risks; Group II are those patients who are found to have an angiographically determined risk factor such as a high carotid bifurcation, long plaque, coexisting siphon stenosis, carotid slim sign, or opposite carotid artery occlusion; Group III are individuals in whom the overriding consideration is the coexistence of a major medical risk such as severe coronary artery disease or chronic obstructive pulmonary disease, regardless of angiographic findings; and Group IV are those patients with and without medical risks who are considered neurologically unstable because of crescendo transient ischemic attacks, symptoms of both focal and general cerebral ischemia, or a recent infarct from which the patient has recovered with new superimposed ischemic events.

Recurrent stenosis is not included in this grading system since the technical aspects of the surgical procedure are quite different from those for primary stenosis as is the underlying pathology. Two patients underwent surgery for recurrent stenosis, one of whom had undergone initial surgery elsewhere and the other here 7 years previously.

In the 97 operative procedures for primary stenosis the patients were classified as follows: Grade I (39), Grade II (23), Grade III(14), and Grade IV (21). There were 27 operations for severe asymptomatic stenosis, among which seven had an opposite carotid occlusion and eight followed a contralateral endarterectomy. Seventy-two operations were performed for symptomatic stenosis including: amaurosis fugax (24), TIAs (17), small infarct (11), progressing stroke or crescendo TIA's (6), and generalized (nonfocal) cerebral ischemia (14).

Prior to surgery, all patients underwent conventional retrograde femoral angiography with visualization of both the intra- and extracranial circulation.

Surgical Technique

The CCAs and ICAs were routinely reconstructed with a saphenous vein patch graft unless the vessel appeared larger than average or the plaque was essentially limited to the carotid bulb with minimal extension beyond that level into the internal carotid artery. Ten arteries had primary closure, 88 had a saphenous vein patch graft, and one had a fabric patch graft.

The plaque from the ECA was extracted by telescopically everting the distal portion of the vessel through the orifice of the artery and circumferentially working the plaque away from the vessel until it broke cleanly from the distal vessel wall. If a palpable superficial temporal artery pulse was not present following restoration of flow, the ECA was temporarily occluded proximally and distally and a separate arteriotomy created in the vessel. Ninety ECAs were reconstructed by everting the vessel and extracting the plaque in a standard fashion. A separate arteriotomy was made in the ECA in nine cases in order to excise the distal end of the plaque; eight of these arteriotomies were closed primarily and one with a patch graft.

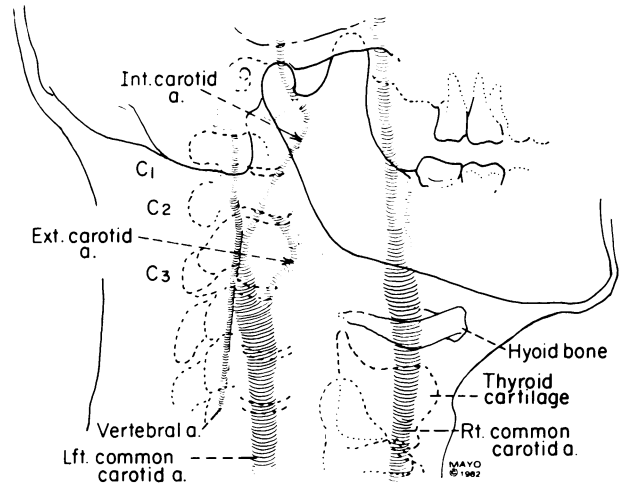

FIG. 1. Position of patient's head and neck for DSA examination of right carotid artery.

All operative procedures were monitored with continuous electroencephalograms (EEGs) and cerebral blood flow measurements. These techniques have been described previously.[22] Forty-one cases required intraoperative shunting.

Postoperative Evaluation

Following surgery, all patients had a detailed neurological examination in the recovery room, and this examination was repeated at least once daily in addition to less complete, more frequent examinations until the date of discharge. Oculoplethysmometric readings (OPGs) were recorded the day after surgery. An abnormal OPG on our service is considered to be an indication for immediate angiography, but the OPG was normal or improved on the operated side in all of these cases. Routinely, 1 week following surgery the patients underwent a DSA. The carotid arteries were examined in two planes (Figs. 1 and 2).

Follow-up Evaluation

The patients were contacted intermittently during the period of follow-up and scheduled for a return visit at a date approximating 2 years from the date of the operation. At this time, a detailed neurological examination was completed and a history recorded concerning neurological function during the period of follow-up. DSAs were obtained at the time of follow-up examination. In those patients unable to return for a postoperative visit, contact

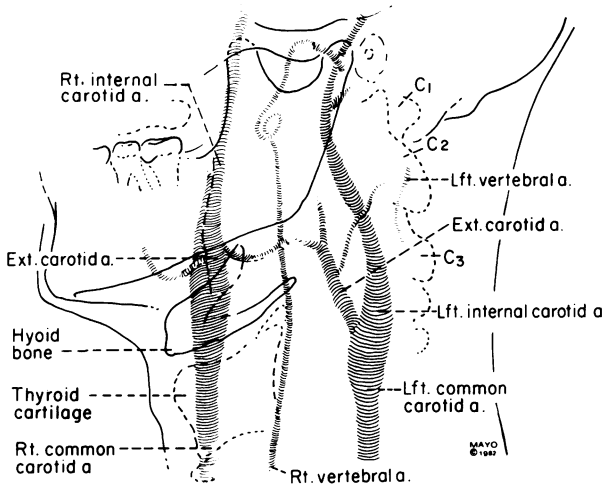

FIG. 2. Position of patient's head and neck for DSA examination of patient's left carotid artery.

regarding neurological function was assessed through the local physician, and arrangements were made for a DSA to be completed in the local setting. Copies of these angiograms were then forwarded to us for the patient's permanent file.

Terminology

(1) Transient neurologic dysfunction. A transient change in neurological function of less than 24 hours duration was considered as an episode of transient neurological dysfunction. This could include a typical transient ischemic attack (TIA), seizure, migraine variant, or episode of postural hypotension with global ischemia.

(2) Reversible ischemic neurologic deficit. Neurological dysfunction that persisted for longer than 24 hours but was reversible within 3 weeks was characterized as a reversible ischemic neurological deficit (RIND).

(3) Minor stroke. Any cerebral infarction leading to minor neurological dysfunction such as decreased hand dexterity that did not prevent that patient functioning in his or her previous capacity was considered a minor stroke.

(4) Major stroke. Any neurological deficit preventing the patient from resuming his or her previous level of functional activity was considered a major deficit.

(5) Carotid slim sign. Angiographic appearance of a very contracted ICA distal to the point of a very high grade stenosis. It results from an exceptionally low intraluminal perfusion pressure that is so low that the artery is not fully distended.[23] These arteries can be considered to be physiologically occluded as they are contributing very little to effective cerebral blood flow.

Angiographic Readings

All preoperative retrograde femoral angiograms, postoperative DSAs and follow-up DSAs were evaluated by a neuroradiologist (O.W.H.) without knowledge of the patient's history or follow-up neurological examination. The operated and nonoperated sides were compared and notations made regarding the status of the right and left vertebral arteries. DSAs were, in general, inadequate for analysis of the siphon areas but these areas were included in the preoperative analysis of the angiograms. The ipsilateral carotid siphon was severely stenotic in 14 cases and mildly stenotic in 29. The opposite ICA was occluded in 14 cases and 16 cases had an ipsilateral carotid slim sign.

The angiographic appearance of the ECA, ICA, and the CCA were recorded on a standard format. Information regarding each of these three vessels included notations regarding various degrees of stenosis, irregularity, ulceration, loops, kinks, intimal flaps, aneurysmal dilatations, cuffs, occlusions, and stumps.

Data Analysis

Pertinent data concerning the patient's clinical condition prior to and following surgery, the operation, preoperative and postoperative angiographic findings, followup neurological function, and follow-up angiography were all recorded systematically and then entered in a computer bank for analysis and retrieval.

Results

There were 97 vessels operated in 84 patients (13 patients had bilateral endarterectomies) for primary atherosclerosis and two vessels in two patients for recurrent disease. For the purpose of angiographic analysis, the patients with bilateral endarterectomies are considered as two separate cases so the denominator for this data is 99 rather than 86.

Neurological Complications

(1) Transient neurological dysfunction. One patient with a long history of migraine had a typical migrainous event with an aura of positive visual hallucinations the night following surgery. The entire event lasted approximately 30 minutes and was followed by ispilateral headache. Another patient had a visual hallucination of wavy lines followed by ispilateral headache and contralateral tingling in the hand of several minutes duration the night following surgery. Both of these patients had crescendo transient ischemic events prior to surgery and were operated as semiemergencies. A carotid slim sign was present on the preoperative angiogram in one of these cases. In-

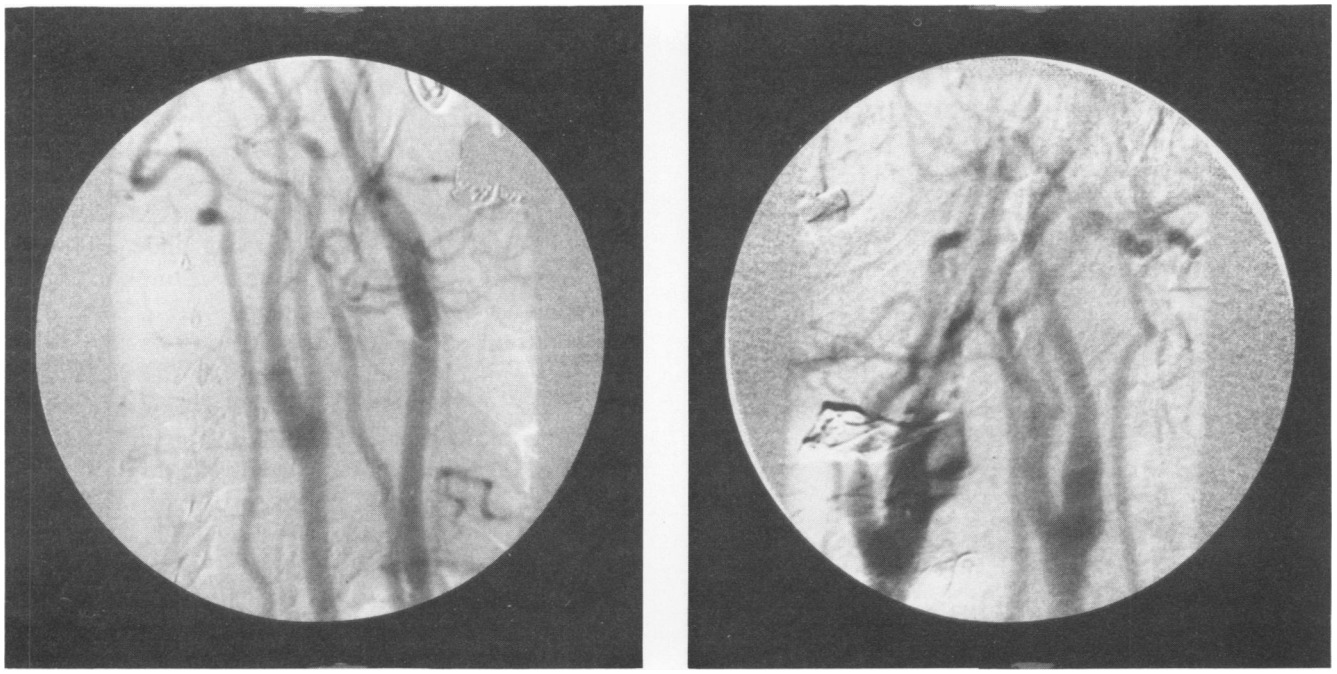

FIG. 3. *Left.* Typical normal postoperative DSA of right carotid. *Right.* Typical postoperative DSA of left carotid artery.

tracerebral blood flow measurements tripled in the first and doubled in the second of these two patients following endarterectomy. Neither of these patients had any further transient neurological dysfunction, and each was considered to have had a form of migraine variant related to cerebral hyperperfusion.

One 80-year-old patient who had a preoperative hemiparesis and had undergone emergency endarterectomy was found to have focal lateralized epileptiform discharges during routine postoperative electroencephalography. The patient had no symptoms related to these epileptiform discharges. She ultimately recovered good neurological function. This individual also had a doubling of cerebral blood flow after endarterectomy and the epileptiform activity was considered to be a manifestation of hyperperfusion in an area of preoperative ischemia.

One patient with coexisting severe siphon stenosis had a 40-minute period of contralateral paresis 12 hours after surgery. This cleared without residual deficit.

(2) RIND. No patients in the group had a RIND following surgery.

(3) Minor stroke. Two patients developed a minor persistent deficit that was not present prior to surgery. Each of these patients had crescendo TIAs prior to surgery. In one patient, the deficit ultimately resulted in only a minor reduction in the alternate motion rate in the fingers of one hand, but the other patient sustained a permanent apraxia in the hand. The second of these two patients ultimately was proven to have a heparin-induced coagulopathy with a circulating heparin antibody and is the only patient in the series who had a symptomatic recurrence in follow-up (see below). This second patient had an angiographic slim sign on the preoperative angiogram.

(4) Major stroke. One patient operated for crescendo TIAs with an angiographic slim sign had a major postoperative stroke in the recovery room. Angiography in this patient revealed widely patent ICAs and CCAs but the ECA was occluded with a stump (see below). There was evidence on angiography of intracranial embolization, and it was assumed that the emboli arose from the stump of the ECA or from thrombi coexisting with the slim sign. This patient ultimately succumbed to pulmonary embolism but the underlying cause for the death was ischemic stroke.

In summary, combined morbidity (permanent deficit) and mortality for the series according to the number of operative procedures was 3%. In this small series, there was no morbidity or mortality in patients classified as Grade I through III but the combined morbidity-mortality in Grade IV patients was 15%. Neither of the two patients with recurrent stenosis had an operative complication.

Postoperative Angiography

All patients underwent a DSA following surgery. There was 100% patency of the CCAs and ICAs. Entirely normal studies are illustrated in Figure 3. Specific variants from the normal were described for the ICAs, CCAs, and ECAs.

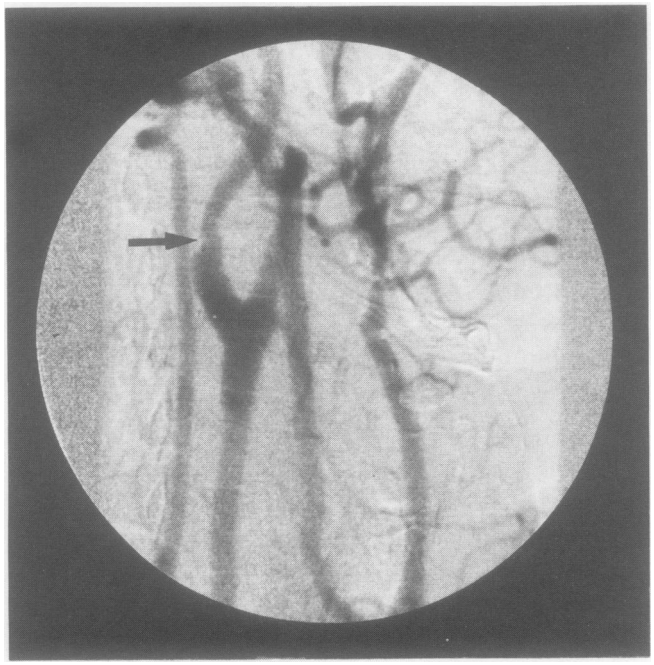

FIG. 4. Postoperative DSA showing demarcation or notch at distal point of vein graft. This type of defect resolved routinely on follow-up angiography. Demarcation point is identified by arrow.

(1) Internal carotid artery. The operated lumen of the ICA was judged to be normal in 49 cases, wider than usual in 47, aneurysmal in one, and minimally stenotic in two. The stenosis was less than 20% in each of the two cases in which this was recorded as a finding. One of these two cases was closed primarily and one with a patch graft. No identifiable changes were present in the ICA distal to the endarterectomy that could be attributed to the use of a shunt. One patient with a preoperative physiological occlusion and carotid slim sign had an ICA dissection distal to the site of endarterectomy that was thought to be related to the variation in elasticity between the media and intima in this chronically contracted vessel. The patient remained asymptomatic, and follow-up angiography 6 months later revealed resolution of the dissection.

There was no ledge or cuff at the distal site of endarterectomy in 82 cases, but a small defect was identified distally in 17 cases (Fig. 4). In each of these patients, the vessel had been closed with a patch graft, and the point of notching was nonstenotic and represented the terminal point of the patch graft. Fourteen of these 17 cases had follow-up angiography, and in these the defect had resolved in 12 and remained unchanged in two.

In two patients, there was a small defect in one wall of the proximal ICA of undetermined origin, and each of these patients had been closed with a patch graft. The operative note on one of these two patients indicated that the ICA was much thinner than average, and this defect probably represented a transverse crease or kink on one side of graft. Follow-up angiograms were normal in these two cases.

(2) External carotid artery. The ECA was found to be patent with a normal lumen in 62 cases, patent but with a cuff at the demarcation point of endarterectomy in 30 patients (Fig. 5), patent but with a flap at the distal point of endarterectomy in three cases, and undetermined in one. The ECA was occluded without a stump in two patients and occluded with a stump in one. The patient who had occlusion of the ECA with a stump was the patient who had a major postoperative stroke, leading to death. One of the two patients who had occlusion of the ECA without a stump remained asymptomatic but is the only patient in the series who had occlusion of the CCAs and ICAs on follow-up angiography (see below). The other developed a 50% stenosis of the distal CCA in follow-up at the site of the ECA occlusion (Fig. 6).

(3) Common carotid artery. The lumen of the CCA was found to be normal in 56 cases and wider than average in 43. The walls of the CCA appeared to be entirely normal in 72 cases. However, a ledge or step-off was identified proximally in 22 cases, which was related to diffuse atherosclerotic thickening of the CCA proximal to the site of endarterectomy. A transverse crease or undulation in one wall of the CCA distal to the proximal point of end-

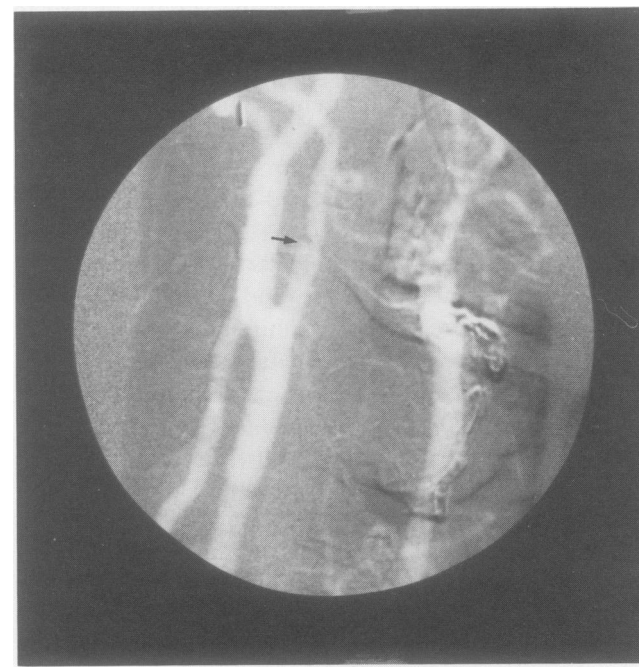

FIG. 5. Postoperative DSA showing demarcation point (arrow) of endarterectomy in ECA. In patients in whom a separate arteriotomy was made in the ECA during endarterectomy because of a poor temporal artery pulse following restoration of flow, an intimal flap was usually identified. It is assumed that this defect in the ECA represents an intimal flap.

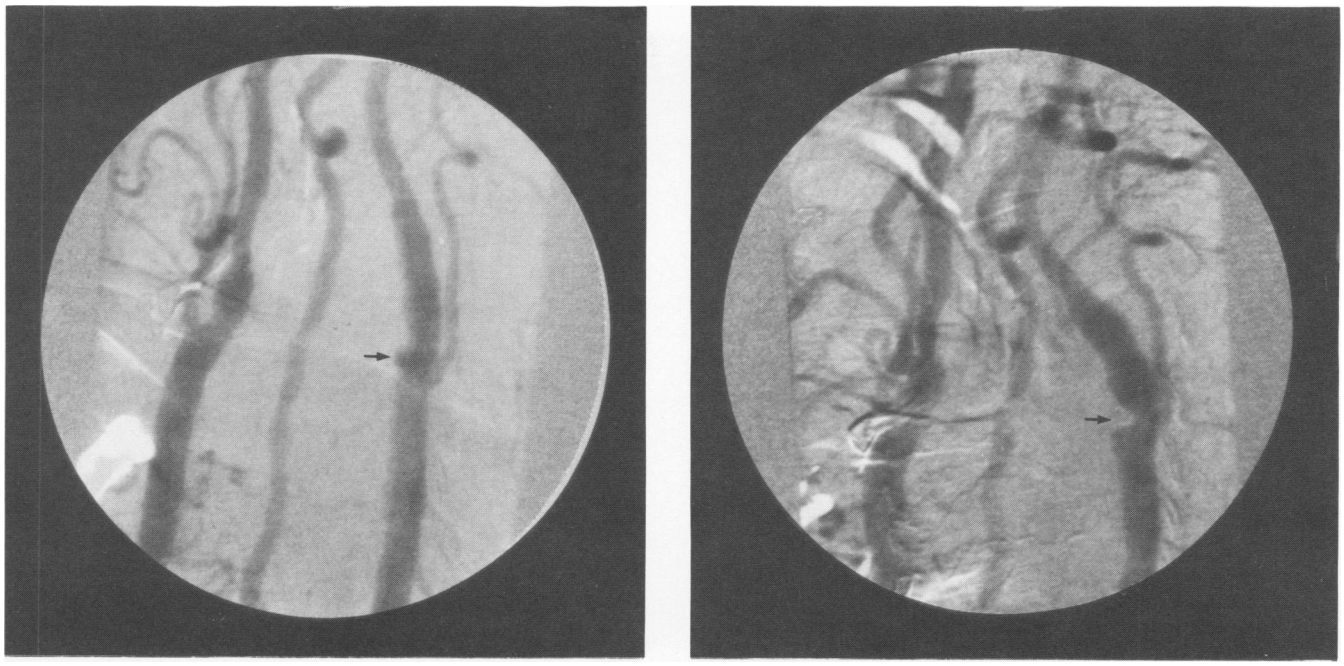

FIG. 6. *Left.* Postoperative DSA showing occlusion of ECA. Vertebral artery should not be mistaken for a patent ECA. Occlusion point noted with arrow. *Right.* Same vessel 2 years later now showing 50% stenosis of CCA at the point of origin of ECA (arrow).

arterectomy was identified in five cases (Fig. 7). All five cases had been closed with a patch graft. These were nonstenotic lesions, which resolved by the time of follow-up angiography except in the patient closed with a fabric patch graft in which the undulating defect was unchanged.

Follow-up Neurological Evaluation

Follow-up evaluation of neurological function was obtained in 84 patients (97 procedures): 60 by personal interview and examination and 24 by phone or letter. There were six deaths in follow-up, two related to myocardial infarction, two from cancer, and two from unknown causes (one patient was 82 and the other was age 61). Two patients with primary atherosclerosis had a single TIA in follow-up (one of whom was the only patient in the series to have an immediate postoperative TIA), and one of the two patients operated for recurrent stenosis had several ill-defined transient ischemic events in follow-up. This patient had a mild nonulcerative re-stenosis distal to the site of endarterectomy (see below). There were two strokes in follow-up. One of these was definitely related to a lacunar infarct, in turn the result of hypertensive arteriolar sclerosis. This individual returned to see us and underwent conventional four vessel angiography. The operated vessel was normal. Computerized tomography confirmed the presence of a small lacunar infarct that was consistent with the patient's symptoms (this occurred 20 months after operation).

The other patient with a stroke in follow-up developed recurrent stenosis with a soft clot at the site of endarterectomy and was one of the two patients in the series to

FIG. 7. Transverse crease or undulation in wall of CCA distal to the origin of internal carotid artery is marked by arrow. This is considered to be the result of a unilateral crease or kink in the wall of the CCA. Defects such as this cleared or were not apparent on follow-up DSA 2 years later. They were not associated with a neurologic complication.

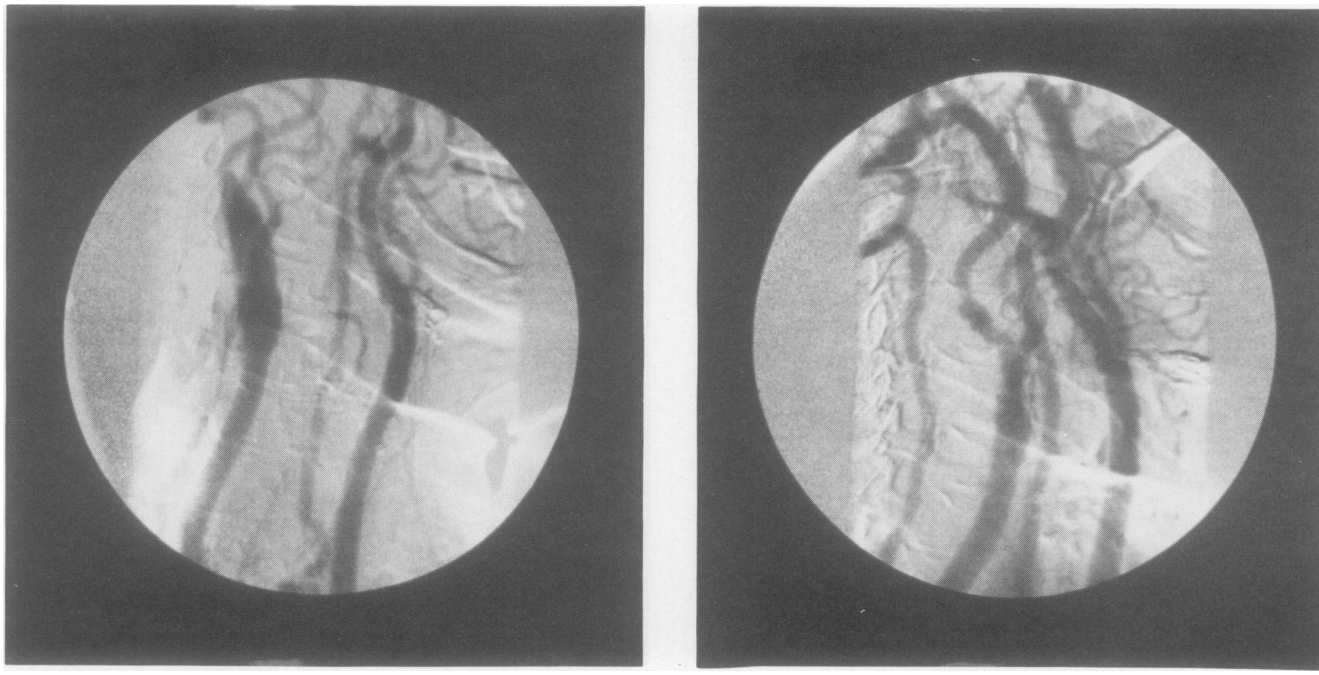

FIG. 8. *Left.* Postoperative study of patient with hypercoagulability state reveals site of endarterectomy to be widely patent with no apparent thrombi. *Right.* Same patient 2 years later now has areas of stenoses and possible soft thrombi within areas of endarterectomy in the internal carotid artery. Patient underwent surgery for recurrent symptoms. A soft thrombus was found under which the intima appeared to be normal. There was no evidence of intimal hyperplasia.

have a minor immediate postoperative stroke. This developed 32 months following the initial surgery, and at the time of reoperation a soft jelly clot was identified overlying apparently normal endothelium in the distal internal carotid artery (Fig. 8). This patient was subsequently proven to have a heparin-induced hypercoagulability state (confirmed with heparin antibodies in the serum), which in retrospect was probably the cause for her initial postoperative complications. She had complications from her second surgery as well.

Follow-up Angiography

Seventy-nine vessels were evaluated by a DSA follow-up. Eight of these vessels had been closed primarily and 71 with patch grafting.

(1) Internal carotid artery. The caliber of the ICA was found to be normal in 55 cases, wider than usual in 17, diffusely dilated or aneurysmal in one, stenotic in five, and occluded in one. The occlusion occurred in a vessel closed with a patch graft that had occlusion of the ECA on postoperative angiography. Two of the patients with re-stenosis had been closed primarily, the re-stenosis was less than 20% in one of these but greater than 50% in the other. Three of the arteries with recurrent stenosis were closed with a patch graft. Of these, one was less than 20% and not significant, one developed 32 months following the initial surgery and was associated with the heparin-induced coagulopathy discussed above, and one occurred in one of the two patients operated for recurrent disease.

Mild undulation or insignificant irregularity in the wall of the ICA was identified distally in three cases. Each of these had been closed with a patch graft.

(2) External carotid artery. The ECA was patent in 67 vessels with a normal lumen and patent in eight with a small cuff or flap. The ECA was occluded with a stump in one patient and occluded without a stump in two patients. Two of these had been known to be occluded from the postoperative studies but the third occluded during the period of follow-up. In this third case, the postoperative angiogram had shown a cuff or ledge at the distal site of endarterectomy in the ECA. In one patient, the ECA could not be evaluated.

(3) Common carotid artery. The lumen of the CCA was normal in 58 arteries, wider than usual in 16, and occluded in one (same case that had occlusion of ICA). There was stenosis present in four vessels at the proximal end of the endarterectomy at the junction point between the endarterectomized and the nonendarterectomized artery. All of these had been closed with a patch graft. This degree of stenosis was less than 20% in one and essentially insignificant. However, the stenosis exceeded 70% in three cases and extended somewhat proximally into the nonendarterectomized artery in all three cases (Fig. 9). A 50% restenosis of the distal CCA occurred at the site of the

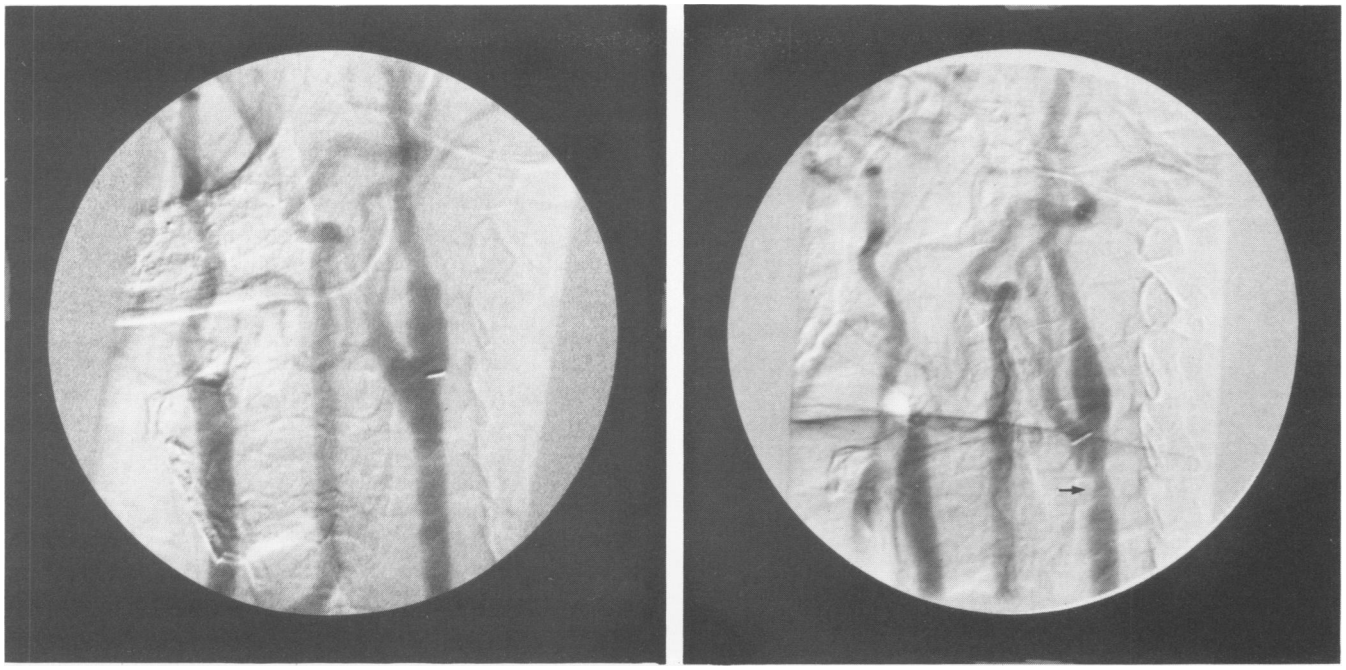

FIG. 9. *Left.* Normal postoperative angiogram following endarterectomy with saphenous vein patch graft reconstruction reveals good transition point between endarterectomized and nonendarterectomized segments of the CCA. *Right.* Recurrent stenosis at proximal end of endarterectomy 2 years later (arrow). Stenosis extends into the diffusely sclerotic proximal CCA. This patient was asymptomatic and thus not re-explored; thus, cause of restenosis is undetermined.

postoperative ECA occlusion in one additional patient (Fig. 6 right).

A proximal ledge or nonstenotic cuff identifying the proximal point of the endarterectomy was identified in seven cases. All of these had been closed with a patch graft.

Non-operated Carotid Artery

The opposite ICA was occluded before operation in 13 cases, and, of these, 11 patients returned for angiography and the vessel had remained occluded in all. A nonoperated patent carotid bifurcation was normal or unchanged in 38 cases. There was slight progression of bifurcation atherosclerosis in 10 cases and moderate progression in six. The comparison of the carotid bifurcations was not possible in the remaining cases because of artifacts from motion or calcium-like densities in the vessel wall.

Discussion

Reliability of Digital Subtraction Angiography

In our experience, digital subtraction angiography performed by an intravenous injection of contrast material is inadequate for the preoperative evaluation of patients with transient ischemic attacks from suspected carotid artery atherosclerosis.[24] These limitations are based on the inadequacy of visualization of the cerebral circulation and amount of calcium in and thickness of the vessel wall at the bifurcation, which prevents good visualization of the lumen. The first of these two limitations is not relevant in the postoperative evaluation of the carotid bifurcation unless the patient has sustained a neurological complication from the surgery and the second is eliminated by the operation itself, which removes the thickened atherosclerotic plaque from the vessel wall. We have found that digital subtraction angiography in the postoperative patient gives excellent visualization of the carotid bifurcation if the patient is properly positioned as indicated in Figures 1 and 2.

Review of the Literature

A comparison of the various reports in the literature concerning the frequency of recurrent carotid stenosis is difficult because of differences in the manner in which patients were collected (consecutive or nonconsecutive), studied (contrast *vs.* noninvasive) and the completeness of follow-up. Furthermore, it is necessary to distinguish between symptomatic recurrence and asymptomatic recurrence as stressed by others. Table 1 summarizes some of the follow-up studies based on noninvasive testing.[2-5,17-19]

Based on the above reports, approximately 10 to 30% of the endarterectomized vessels closed primarily were restenosed, with approximately one-half of these occurring

TABLE 1. *Significant Re-stenosis in Follow-up—Noninvasive Testing**

Author	Year Reported	±2 Years	>2 Years	Comments
Cossman	1978	3.6%	—	2-year follow-up
Kremen	1979	3%	10%	Based on OPG 45-month mean follow-up
Turnipseed	1980	3%	6%	3-year follow-up
Bodily	1980	—	49%	Duplex scan, 3-month to 96-month follow-up
Cantelmo	1981	8%	4%	OPG-CPA
Norvving	1982	—	35%	6-year mean follow-up
Zierler	1983	20%	—	Duplex scan and spectral analysis
Baker	1983	10%	—	23-month mean follow-up; OPG and Doppler

* Defined here as approximately 50% or more.
CPA = Carotid phonoangiography.

within the first 24 months after surgery. However, it should be noted that only the study by Norrving was based on consecutive patients, with approximately 80% of the series having postoperative angiograms and 64% undergoing follow-up studies. Therefore, the denominator in most instances is undetermined.

Neurologic Function

(1) Operative complications. One patient died from an ischemic stroke, and two patients developed a minor permanent neurological deficit that was not present prior to surgery. One of these patients had a heparin-induced coagulopathy and two of the three patients had a carotid slim sign. The carotid slim sign indicates a very low perfusion pressure within the vessel and a very slow flow; thus, it is possible that the complications in these patients were related to emboli distal to the site of the endarterectomy. All three complications occurred in the recovery room. Interpretation of the intraoperative EEG was difficult in each of these cases as all three were operated as semiemergencies because of an evolving neurological deficit and the baseline EEG was abnormal. Postoperative angiography revealed patency of the ICA and the CCA in all three cases but the ECA was occluded in two. In the patient who sustained a major deficit, there was a stump at the origin of the ECA.

Two patients had transient migraine equivalents probably related to cerebral hyperperfusion. The relationship of this type of event to cerebral hyperperfusion following endarterectomy has previously been documented. We consider it to be related to vascular reactivity and hyperperfusion.

In this small series, we did not have any intracerebral hemorrhages or myocardial infarctions leading to death. However, in our overall group of approximately 2100 endarterectomies to date monitored with cerebral blood flow measurements and EEG's, there is a 0.5% incidence of fatal myocardial infarction and intracerebral hemorrhage. We believe that the absence of these complications in the current group was fortuitous.

(2) Neurologic function in follow-up. There were two strokes in follow-up. One of these was a lacunar stroke related to arteriolar sclerosis and was proven to be unrelated to the carotid endarterectomy. The other occurred in one of the three patients who had had a postoperative stroke and had a heparin-induced coagulopathy. The recurrence in this patient was atypical in that a soft clot was identified at surgery overlying smooth endothelium. The specimen was not consistent with myointimal hyperplasia or with recurrent atherosclerosis. It was, in fact, composed solely of soft thrombotic material.

Comparison of Postoperative and Follow-up DSAs

In general terms, minor deficits or defects that were present on the postoperative angiogram resolved by the time the follow-up study was performed. Transverse creases or undulations and notching at the distal point of patch grafting proved to be insignificant. However, occlusion of the ECA was found to be highly significant. There were three ECAs occluded on postoperative angiography; one of these was associated with the major stroke referenced above, one with the only occlusion in the series of the CCA and ICA in follow-up, and one with a 50% stenosis of the CCA at the site of the origin in the ECA in follow-up.

In patients operated on for primary carotid stenosis, the ICA protected by patch grafting appeared to be essentially free of recurrent disease during the period of follow-up in this series. However, three cases or 4% of those closed with a patch graft had significant yet asymptomatic recurrent stenosis of the CCA at the proximal end of endarterectomy in follow-up angiography. Each of these patients had had diffuse atherosclerosis prior to surgery. The recurrent stenosis appeared to involve the junction point between the endarterectomized and nonendarterectomized segments of the vessel primarily but extended proximally into the CCA in each case. One ICA occluded asymptomatically in follow-up. This patient had had occlusion of the ECA on postoperative angiography.

One ECA occluded in follow-up that had been patent on postoperative angiography. This patient had a cuff or demarcation point at the distal end of the endarterectomy on the postoperative study, and it is concluded that this was the cause for occlusion in follow-up. Fortunately, this was an asymptomatic ECA occlusion.

Primary Closure versus *Closure With Patch Graft*

A comparison in our series of cases closed primarily and those with a patch graft is not meaningful, as primary

closures were used for the shorter plaque and less complicated cases. Table 2 summarizes data from reported studies of intraoperative and postoperative angiograms.[18,25–30] Little et al. compared primary closure and closure with a saphenous vein patch graft and found a highly significant difference in postoperative patency between the group closed primarily and that with a patch graft (6% occlusion rate of ICA without patch grafting *versus* 0% with patch grafting—p value = 0.004).[30] Imparato's group[31] and Deriu[32] have emphasized improved patency with patch grafting *versus* a primary closure. We prefer the use of a patch graft but primary closure is still preferred by most surgeons.

TABLE 2. *Results of Intraoperative or 1 Week Postoperative Angiography*

Author	Year Reported	↓ Patency ECA	↓ Patency ICA	Significant ICA or CCA Stenosis	Type of Closure	Type of Study
Blaisdell	1967	—	95	20	Primary	Intraoperative angiogram
Rosental	1973	96	99	8%	Primary	Intraoperative angiogram
Anderson	1978	98	100	3%	Primary	Intraoperative angiogram
Hertzer	1982	95	98	1%	Primary	DSA
Norvving	1982	?	93	4%	Primary	Conventional angiogram
Diaz	1982	80	96	0%	Primary	Conventional angiogram
Little	1984	100%	94%	6%	Primary	DSA
Little	1984	100%	100%	0%	Saphenous vein patch graft	DSA
Sundt	1985	97%	100%	0%	Saphenous vein patch graft	DSA

Causes for Re-stenosis

Re-stenosis within 2 years from the time of surgery is generally attributed to myointimal hyperplasia and re-stenosis thereafter to atherosclerosis.[1] In this small series, no angiographic changes typical of myointimal hyperplasia were identified, but we have had patients return with symptomatic re-stenosis from myointimal hyperplasia and consider it to be a proven entity. The cause for re-stenosis at the proximal end of the carotid endarterectomy is undetermined in this series, as the patients were asymptomatic and not re-explored.

The significance of ECA occlusion in this series was striking. One case developed a definite re-stenosis at the site of ECA occlusion and another had occlusion of the ICA and CCA in follow-up—the only occlusion to occur in the series. One cannot help postulating that stenosis or occlusion in these cases was related to propagation and gradual development of an occluding thrombus at the site of the ECA occlusion. In some cases, re-stenosis is probably related to thrombus formation, which subsequently undergoes degenerative changes not unlike those seen in a complicated primary atherosclerotic plaque.[33,34]

Contralateral Stenosis or Occlusion

The risk of recurrent stenosis is significant enough to justify caution in recommending surgery for asymptomatic lesions.[10,35] This must be balanced against the natural history of the plaque.[36–38] In the group reported here, there were no strokes in follow-up related to contralateral stenosis or occlusion, but patients with complicated or high-grade stenoses underwent prophylactic endarterectomy following surgery for the symptomatic stenosis. There was significant progression in this 2-year follow-up of a non-stenotic plaque in 29% of the cases. Contralateral occlusion (14 cases) was not associated with an increase in the risk of surgery in this group.

Carotid Slim Sign

A carotid slim sign was present on the preoperative angiogram in the patient who had a major postoperative stroke, in one of the two patients with a minor postoperative stroke, one of the two patients who had a migraine variant, and the only patient with a dissection of ICA distal to the site of surgery. This is not surprising, since it is indicative of a very low perfusion pressure and often is found in patients who are neurologically unstable. It is an ominous finding and should alert the surgeon to the increased risks associated with surgery in these patients. These risks include postoperative cerebral hyperperfusion, possible embolization from thrombotic material on the wall of the vessel distal to the site of endarterectomy, and dissection of the ICA related to variation in the distensibility of chronically contracted intima and media. In our limited experience, a postoperative dissection should be managed like a spontaneous dissection, *i.e.*, conservatively.

Although patients with this finding are often neurologically unstable and are high risks for surgery, results can be dramatic,[39] and the risks without surgery are, in our experience, even higher. Thus, we recommend surgery unless a major fixed deficit is present.

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