Carotid Endarterectomy for Cerebrovascular Insufficiency:

Long-Term Results in 592 Patients Followed up to Thirteen Years

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DURING the past 16 years since the first successful carotid artery reconstructions for stroke were reported,[6, 9] carotid endarterectomy has become accepted therapy for selected patients with cerebrovascular insufficiency. Recent data from the Joint Study project[12] indicate that 74% of patients with ischemic stroke syndromes have at least one significant stenotic lesion in the extracranial vasculature at a surgically accessible site. In most cases the intracranial vessels are surprisingly free of demonstrable disease. It has thus become feasible to restore cerebral blood flow by surgical means with an acceptable mortality and morbidity. Several large series of cases have now been reported,[1, 2, 4, 5, 7, 8, 11, 15, 16, 19, 21] and indications for surgical therapy in the various clinical categories are becoming more precise. There are still unsolved problems in certain areas, however, which require resolution based on statistical comparison of surgical results with non-surgical control series.[2]

This report is a continuation of previous clinical investigations into the use of carotid endarterectomy for the management of patients with cerebrovascular insufficiency.[16, 17, 18, 19] It is based on an analysis of results in 592 patients subjected to carotid endarterectomy during a 13-year period. Since carotid lesions are those most commonly encountered the report is limited to a discussion of carotid occlusive disease.

Material and Methods

Seven hundred and forty-eight carotid artery operations were performed on 592 private patients during a 13-year period beginning April 16, 1957. Three hundred and seventy-three (63%) were men and 219 (37%) were women. Ages of the men ranged from 37 to 87, with an average of 63.1 years, while the range among women was from 33 to 84 with an average of 66.5 years. One hundred and fifty-six patients had bilateral operations (26.4%). All patients had significant cervical carotid occlusive lesions demonstrated by arteriography. A lesion was considered significant when the diameter of the internal carotid lumen was reduced 50% or more or when its appearance was suggestive of deposition of platelet thrombi or debris that by dislodgment could become cerebral emboli.

Arteriography was performed under general anesthesia using 50% sodium diatrizoate (Hypaque). Several technics have been employed. In the early years of the study bilateral cervical percutaneous injections were made and both antero-posterior

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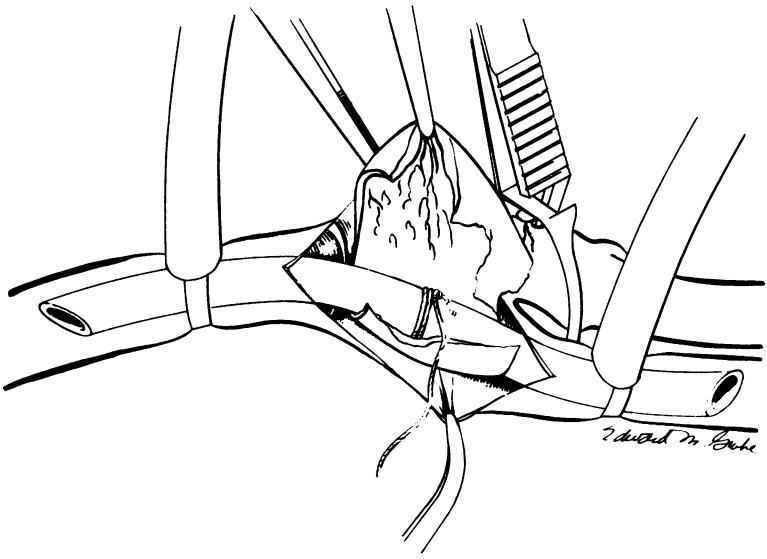

FIG. 1. The carotid artery has been opened through the full extent of the plaque in the internal carotid and a #10 French plastic catheter is in place as a temporary inlying bypass shunt. The plaque, which is being removed by endarterectomy, usually feathers off into thin intima in the distal artery. If it does not, it is stitched down with three or four interrupted sutures.

and lateral films of the head and neck were taken serially and simultaneously with the biplane Schönander x-ray unit. When indicated, vertebral arteriograms were also obtained utilizing a percutaneous retrograde brachial technic. More recently excellent three-vessel studies of both carotids and the right vertebral have been obtained using left percutaneous carotid and right retrograde brachial punctures. In some instances four-vessel studies have been done employing a retrograde transfemoral approach, injecting selectively the vessels arising from the aortic arch.

All patients had carotid endarterectomy performed. The operation at present is done under general anesthesia employing halothane. Technical considerations are important and no unnecessary risks should be taken. We routinely employ a temporary inlying bypass shunt for all partially occlusive lesions (Fig. 1). An important technical point to emphasize is that utmost gentleness must be used in manipuation of the artery lest debris be dislodged and pass into the brain as cerebral emboli producing neurological deficits.

A vertical arteriotomy is made extending beyond the plaque in the internal carotid artery. The end of the plaque is thus under direct vision and can be tailored or stitched down so that distal intimal dissection leading to postoperative thrombosis does not occur. The arteriotomy is closed with 6-0 dacron sutures. A patch graft is not used in reconstructing the arterial lumen except in rare instances when the artery is small. Blood pressure is maintained at normal levels for the particular patient using 500 to 1,000 ml. of lactated Ringer's solution or small amounts of vasopressors if necessary.

Operative complications following carotid endarterectomy have been few. Three patients were re-operated upon becaurse of progressing neurological signs and were found at operation to have thrombosis of the endarterectomized segment secondary to intimal flap dissection. Thrombectomy was carried out, the distal intima was sewn down, and flow was successfully restored in all three. Eight patients developed wound hematomas requiring operative evacuation. Six patients with dacron patch grafts applied early in the series, sewn in place with silk sutures, developed false aneurysms because of disruption of the silk suture line. All were suc-

cessfully regrafted without incident using either tubular dacron grafts and dacron sutures or vein grafts. No patient has developed an aneurysm in an endarterectomized artery closed without a patch graft. There have been no wound infections in the entire series of 748 operations. Bilateral operations, when indicated, are done at separate stages at least a week apart.

Classification of Patients

The clinical syndromes of cerebrovascular insufficiency vary from a few minor symptoms to the catastrophic stroke with paralysis and coma. It is important to classify these patients into specific clinical categories. Only in this way can the proper selection of patients for operation be made and the results of different methods of therapy within the same category be compared. For purposes of surgical considerations we have classified our patients into four groups: (1) frank stroke, (2) transient cerebral ischemia, (3) chronic cerebral ischemia, and (4) asymptomatic bruit.

Frank Stroke. This group includes all patients with a neurological deficit at the time of operation, whether improving, progressively worsening, or stable. All degrees of severity may be present, ranging from mild residual deficits to profound strokes with hemiplegia, aphasia, and coma. The so-called reversible ischemic neurological deficit (RIND) is included in this group and is not classified as a transient ischemic episode because recovery requires more than 24 hours. A few patients with old stable strokes also are included.

Transient Cerebral Ischemia. This group includes patients with focal attacks of neurological dysfunction and transient symptoms of generalized cerebral ischemia lasting minutes or hours but without residual neurological deficit at 24 hours. Focal attacks include ocular, speech, sensory and motor disturbances. Features of generalized ischemia include dizziness, blackout spells, headaches and other nonlocalizing symptoms.

Chronic Cerebral Ischemia. Patients in this category exhibit obvious cerebrovascular insufficiency with loss of memory, impaired mentation or overt motor and mental deterioration. They are not numerous, are difficult to classify, but logically cannot be placed in any of the other groups.

Asymptomatic Bruit. In this group are patients without neurological symptoms who are found to have carotid bruits during routine auscultation of the neck and and who upon subsequent arteriography show significant occlusive carotid plaques.

TABLE 1. *Operative Mortality Following Carotid Endarterectomy*
Total Experience—Thirteen Years

Clinical Category	No. of Patients	No. of Operations	No. of Deaths	Patient Mortality	Procedure Mortality
Frank stroke	217 (36.7%)	262	16	7.4%	6.1%
Transient cerebral ischemia	293 (49.5%)	378	4	1.4%	1.1%
Chronic cerebral ischemia	17 (2.9%)	21	0	0	0
Asymptomatic bruit	65 (10.9%)	87	0	0	0
Total	592 (100%)	748	20	3.4%	2.7%

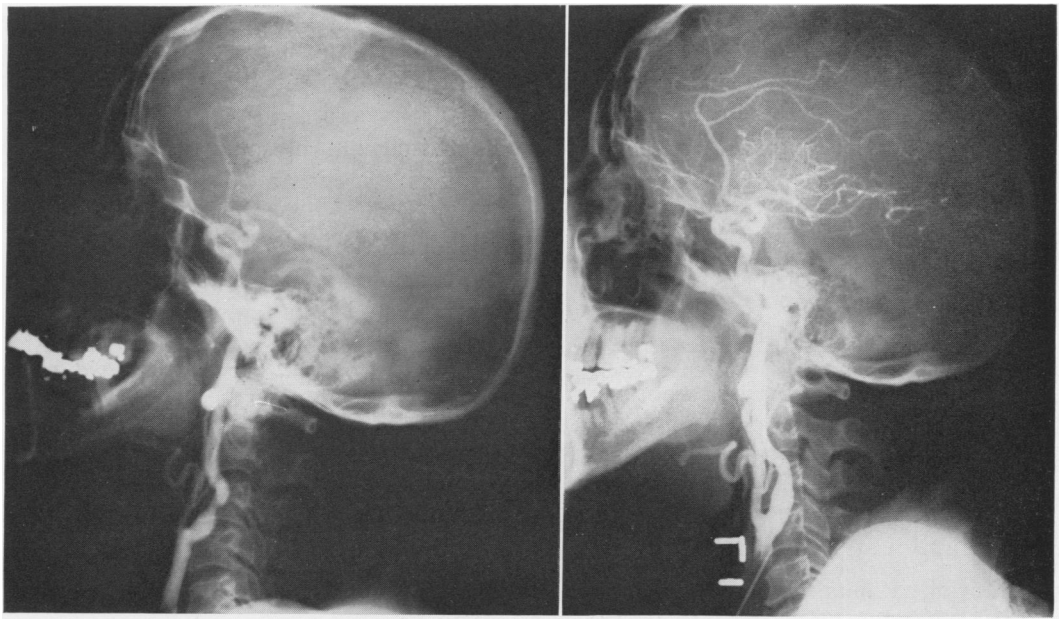

FIG. 2. Preoperative carotid arteriogram (left) of a patient with transient ischemia showing an atherosclerotic plaque with stenosis in the left internal and external carotid arteries, and postoperative arteriogram (right) of same patient 4 years following carotid endarterectomy showing normal caliber of arterial lumen after simple closure of arteriotomy without a patch graft.

These cases may be considered prophylactic.

Table 1 lists the number of patients in each of the four categories in this series.

Operative Results

Anatomic Restoration of Blood Flow

Among 630 partially occluded carotid arteries in 480 patients cerebral blood flow was restored by operation in all instances. Five patients were found to be occluded in the immediate postoperative period.

TABLE 2. *Causes of Operative Deaths*

Cause of Death	Frank Stroke	Transient Cerebral Ischemia	Total
Cerebral	12	2	14
Cardiac	3	2	5
Pulmonary embolism	1	0	1
Total	16	4	20

Three of these were re-operated upon and found to have thrombosis on the basis of intimal flap dissection; flow was successfully restored in all. Thus successful restoration of blood flow was accomplished in more than 98% of patients with partially occluded arteries. Postoperative arteriography has not been done routinely but no other occlusions were suspected on clinical grounds. Over the years a number of postoperative arteriograms have been carried out for various reasons. Our studies and those of others have demonstrated that arteries reconstructed by endarterectomy remain patent for many years without evidence of constriction [3, 10] (Fig. 2). One patient has required re-operation for recurrent carotid stenosis at the site of previous endarterectomy without a patch graft, 9 years following his original operation.

The highly successful restoration of cerebral blood flow of partially occluded arteries is not the case, however, when the

TABLE 3. *Neurological Deficits Related to Operation*

Category	No. Patients	No. Operations	Transient Deficits	Permanent Deficits	
				Mild	Severe
Frank stroke	217	262	0	7 (2.7%)	4 (1.5%)
Transient cerebral ischemia	293	378	3 (0.8%)	4 (1.06%)	4 (1.06%)
Asymptomatic bruit	65	87	1 (1.1%)	1 (1.1%)	0
Chronic cerebral ischemia	17	21	0	0	0
Total	592	748	4 (0.5%)	12 (1.6%)	8 (1.1%)

internal carotid is totally occluded. Among 118 totally occluded arteries in 112 patients flow was restored in 48 arteries (40.7%). Flow could not be restored in 70 arteries (59.3%) since the distal internal carotid was occluded and no retrograde flow was obtained. This degree of success in opening totally occluded arteries has been consistent over the years in our experience.[18]

Operative Mortality

The operative mortality in our total series following carotid endarterectomy is shown in Table 1. Mortality in patients with frank strokes is highest at 6.1%, while in patients with transient cerebral ischemia it is 1.1%. There have been no operative deaths in patients with chronic ischemia and asymptomatic bruits. Most of the deaths occurred in the frank stroke group (16 of 20) and were largely from cerebral causes. Of the 20 deaths 14 were due to cerebral causes, five to myocardial infarction, and one to pulmonary embolism (Table 2).

The significance of these observations in the selection of patients for arteriography and operation and the proper timing of the operative procedures will be discussed below. As experience has increased mortality has progressively decreased. Thus, in the past 6 years the overall operative mortality for all categories has been 1.47%, or only seven deaths in 476 consecutive carotid endarterectomies in 376 patients. There has been no appreciable difference in operative mortality among 436 patients having unilateral operation when compared with 156 patients having bilateral operation in stages (312 procedures).

Strokes Related to Operation

The chief hazard involved in carotid endarterectomy is the production of neurological deficits not present preoperatively or the aggravation of previously existing

TABLE 4. *Length of Follow-up on Patients Operated Upon (Hospital Survivors)*

Time Followed (Months)	No. of Long-term Survivors	No. of Long-term Deaths	Total No. of Patients
6–12	63	52	115
13–24	54	31	85
25–36	41	22	63
37–48	53	23	76
49–60	46	10	56
61–72	36	12	48
73–84	32	5	37
85–96	38	3	41
97–108	16	9	25
109–120	13	2	15
121–132	8	3	11
	400	172	572

TABLE 5. *Causes of Long-Term Deaths*

Cause of Death	Frank Stroke	Transient Cerebral Ischemia	Chronic Cerebral Ischemia	Asymptomatic Bruit	Total No.	Total Per Cent
Cardiac	31	48	3	8	90	52.3%
Cerebral	15	8			23	13.4%
Generalized atherosclerosis	9	2	4		15	8.7%
Malignancy	6	9		1	16	9.3%
Uremia	1	3			4	2.3%
Pulmonary	5	1			6	3.5%
Hypertension	1				1	0.6%
Pulmonary embolism		1			1	0.6%
Intestinal obstruction		1			1	0.6%
Ruptured abdominal aneurysm		2			2	1.2%
Infections		1			1	0.6%
Burns		1			1	0.6%
Following other surgery	1	2		1	4	2.3%
Undetermined	6		1		7	4.1%
	75	79	8	10	172	100%

ones. Because of the consequences involved no undue risks should be taken during carotid surgery, especially in patients with transient cerebral ischemia and asymptomatic bruits.

Table 3 shows the incidence of neurological deficits related to operation in the various categories. These deficits were caused by failure to use a shunt in a few cases early in the series, intimal flap dissection postoperatively resulting in thrombosis of the artery, hypotension postoperatively, hypertension postoperatively causing intracranial hemorrhage, and cerebral embolization of debris during dissection. There were 24 deficits related to operation (3.2%), four of which were transient (0.5%) and 20 permanent (2.7%), 12 mild and eight severe.

Follow-up Studies

Long-term follow-up studies have been carried out on all patients operated upon between April 16, 1957 and September 15,

TABLE 6. *Long-term Functional Results Prior to Death in Patients Dying during Follow-Up Period*

Clinical Category	No. of Patients	Long Term Neurological Status Prior to Death: Normal	Improved	Same	Worse	Normal or Improved Then Worsened	Total Improved
Frank stroke	75	15 (20%)	33 (44%)	17 (22.7%)	5 (6.7%)	5 (6.7%)	48 (64%)
Transient cerebral ischemia	79	53 (67.1%)	15 (19%)	2 (2.5%)	7 (8.9%)	2 (2.5%)	68 (86.1%)
Chronic cerebral ischemia	8	0	4 (50%)	3 (37.5%)	0	1 (12.5%)	4 (50%)
Asymptomatic bruit	10	10 (100%)	0	0	0	0	10 (100%)
Total	172	78 (45.3%)	52 (30.2%)	22 (12.8%)	12 (7%)	8 (4.7%)	130 (75.5%)

1969. Thus all patients were checked from 6 months to 13 years postoperatively. Long-term studies are 100% complete, no patient having been lost to follow-up. The number of patients in each of the time-interval groups is shown in Table 4. Of 592 patients, 20 died in the hositpal following operation. During the follow-up period 172 of the 572 patients surviving operation died. This represents 30% of the hospital survivors.

The causes of long-term deaths are listed in Table 5. Over half (52.3%) of the 172 deaths were due to cardiac disease. Only 23 deaths (13.4%) were due to strokes. These 23 deaths represent 3.9% of the entire series. There were no long-term deaths from stroke in the chronic cerebral ischemia or asymptomatic bruit categories. Fifteen of 75 long-term frank stroke deaths (20%) and eight of 79 transient ischemia deaths (10.9%) were due to cerebral causes.

Table 6 shows the long-term postoperative neurological status prior to death of 172 patients dying during the 13-year period. Eight patients who were either normal or improved following operation worsened neurologically as time went on. This phenomenon is not unexpected in this group of elderly atherosclerotic patients followed for this length of time because of the progressive nature of atherosclerotic disease.

Functional Results

The long-term functional results after carotid endarterectomy in 400 surviving patients are given in Table 7. The goals of therapy should be kept in mind in every patient with cerebrovascular insufficiency considered for operation, since they may differ in the various clinical categories. In contrast to many diseases, with cerebrovascular insufficiency one is concerned almost as much with morbidity as with mortality; that is to say, the quality of survival

TABLE 7. *Long-term Functional Results after Carotid Endarterectomy in Surviving Patients*

Clinical Category	No. of Patients	No. of Deaths		No. of Survivors	Long Term Neurological Status					
		Opera-tive	Long Term		Normal	Improved	Same	Worse	Normal or Improved Then Worsened	Total Improved
Frank stroke	217	16	75	126	38 (30.2%)	74 (58.7%)	6 (4.7%)	3 (2.4%)	5 (4.0%)	112 (88.9%)
Transient cerebral ischemia	293	4	79	210	170 (81%)	33 (15.7%)	0	4 (1.9%)	3 (1.4%)	203 (96.7%)
Chronic cerebral ischemia	17	0	8	9	3 (33.3%)	2 (22.2%)	1 (11.1%)	0	3 (33.3%)	5 (55.5%)
Asymptomatic bruit	65	0	10	55	51 (92.7%)	0	1 (1.8%)	2 (3.6%)	1 (1.8%)	51 (92.7%)
Total	592	20	172	400	262 (65.5%)	109 (27.3%)	8 (2%)	9 (2.2%)	12 (3%)	371 (92.8%)

is important. The disability in a patient who cannot talk, write, walk, or care for himself is entirely different from that seen in patients with severe intermittent claudication or heart disease, for example.

The functional results of any method of treatment for frank strokes are difficult to assess since the clinical course is so variable and the natural history of improvement so well known.[19] A patient who survives an initial stroke usually improves, at least for a time and to some degree. Likewise as the years pass, patients with cerebrovascular insufficiency who were previously normal or improved following operation, in some instances may deteriorate and worsen functionally.

Frank Stroke. Of 126 frank stroke survivors, 88.9% are normal or improved. Eight patients are worse: three as a result of operation-induced deficits, one from a stroke due to severe intracranial occlusive disease, and four from progressive cerebral deterioration.

Transient Cerebral Ischemia. Of 210 survivors in the transient ischemia group, 81% have had no further attacks, while an additional 15.7% have had fewer attacks of less severity. Seven patients are worse. Two patients had operation-induced deficits. Two patients had strokes from total occlusion of an unoperated carotid artery. Three others have had slowly progressive cerebral changes.

Asymptomatic Bruit. Among 55 survivors 51, or 92.7%, are normal. One patient developed a meningioma later and is not normal but has had no stroke. One patient sustained a severe stroke one year postoperative from total occlusion of a previously operated artery on the basis of narrowing in the siphon. Another patient had a mild stroke from intracranial disease, with near-total recovery. A fourth patient has had progressive cerebral deterioration.

Chronic Cerebral Ischemia. Of nine survivors, three are normal. In general, however, mental improvement has been limited. No patient has had a stroke during the follow-up period but cerebral deterioration has been common. The principal result of operation appears to be the prevention of strokes by removal of occlusive carotid plaques. Indications for operation in this category are obviously narrow.

It is difficult to judge the efficacy of carotid surgery for frank strokes because of the lack of adequate control studies. Several reports bear on this matter, however.[2, 7, 8, 19] In a series of 73 medically treated patients with old and recent completed strokes followed for 42 months, Bauer *et al.*[1] reported that 37% were improved, 28.8% were unchanged, 9.6% worsened, and 24.6% died. In a study of functional results following operation for frank strokes in patients with totally occluded carotid arteries, we concluded that patients in whom blood flow was restored improved more rapidly and to a greater degree than occurred in the course of the natural history of recovery from a stroke.[18]

A group of frank stroke patients in the present series operated upon early in the course of their disease was analyzed in an effort to determine which patients exhibited marked improvement following operation. It was found that those patients with mild preoperative deficits, such as monoparesis or dysphasia, who were improving were those most likely to return to a normal or near-normal status. Only rarely did a patient with a profound frank stroke with flaccid hemiplegia, severe mental obtundity, aphasia, or coma make any dramatic improvement when subjected to operation in the acute phase, and many did not survive operation.

In summary, it appears that in patients with *frank strokes* endarterectomy has decreased the mortality from subsequent stroke, has lowered the incidence of recur-

rent strokes, and probably has been responsible for improvement in neurological status beyond that expected from the natural course of the disease.

Patients with transient cerebral ischemia are ideal candidates for surgical therapy since disabling symptoms can be relieved and frank strokes can be prevented. Our data indicate that these goals have been accomplished. A number of reports [2, 5, 7, 8, 11, 16, 21] testify to the beneficial effects of carotid endarterectomy in reducing the recurrence of transient ischemic attacks, lowering significantly the incidence of subsequent strokes, and increasing long-term survival rates. At the same time operative mortality and morbidity have become acceptable for this elderly group of atherosclerotic patients.

Total Carotid Occlusions and Acute Strokes

When atherosclerotic plaques are partially occlusive and the distal internal carotid artery is patent on the arteriogram, restoration of cerebral blood flow by surgical reconstruction is almost uniformly successful. This is not the case, however, if the internal carotid is totally occluded. In the present series 118 totally occluded internal carotid arteries were explored in 112 patients (Table 8). There were seven operative deaths, an operative mortality of 6.2%. Cerebral blood flow was restored by operation in 48 arteries (40.7%). Flow could not be restored or postoperative arteriograms revealed an occluded vessel in 70 arteries (59.3%).

These cases have been analyzed carefully in an effort to determine the factors responsible for successful restoration of blood flow. Probably most important is the time interval from onset of occlusion to operation. The earlier patients are operated upon the greater likelihood of success. Four patients operated upon within 6 hours of onset all had flow restored. Surprising is the finding that even after several weeks of occlusion it is still possible to restore flow in a few instances.

TABLE 8. *Data on Totally Occluded Carotid Arteries Treated by Endarterectomy*

No. of patients operated upon		112
No. of operations for total occlusion		118
No. of operations for partial occlusion		31
Total number of operations		149
No. of operative deaths		7
Patient mortality		6.2%
Operative procedure mortality		4.7%
No. of totally occluded arteries with flow restored	48	40.7%
No. of totally occluded arteries with flow *not* restored	70	59.3%

At times the arteriograms are helpful in predicting a successful outcome. Films may show filling of the ophthalmic artery, the siphon, or distal internal carotid on the occluded side via collaterals from the ipsilateral external carotid or vertebral or from contralateral vessels through the circle of Willis. Patients taking anticoagulants at the time total occlusion occurs appear to have a better chance for restoration of flow than those not on the drugs. The Fogarty balloon catheter has been helpful in removing fresh thrombi from the distal internal carotid in a few instances. Other unknown factors must exist to explain successful restoration of flow in some cases. We have noted in occasional patients a vessel, probably the ascending pharyngeal, leaving the carotid bifurcation more distal than usual on the internal carotid side. It is possible that such an abnormally situated vessel distal to the total block has been responsible for keeping the internal carotid patent since there are no other branches leaving this artery in the neck.

The nature of the stroke itself in the individual patient is probably the most important factor determining surgical intervention in these patients.[18] Discussion of management of totally occluded carotid arteries cannot be divorced from the subject of acute frank strokes, since a large propor-

tion of the latter are caused by the former. It is now generally agreed that it is inadvisable to operate early upon patients with acute frank strokes because of the prohibitive mortality and morbidity.

In the present series there were 54 patients with acute profound strokes who were operated upon. There were 11 operative deaths, a mortality of 20.4%. Nine of the deaths occurred in patients operated upon within 26 hours of onset, one after 2 days, and one after 3 days.

Mortality rates up to 60% have been reported following operation for acute strokes in the early course of the disease, whereas if arteriography and operation are delayed at least 2 weeks or more following onset of the stroke, mortality and morbidity rates are significantly less.[4, 5, 8, 19, 20] Pathologic studies have shown that it requires more than 8 days for an area of cerebral infarction or softening to become sufficiently stabilized so that intracranial hemorrhage will not occur when an increased flow of blood under pressure is restored to the area by carotid endarterectomy.

It is our present policy not to operate as an emergency on patients with acute profound strokes, rapidly progressing strokes or rapidly improving strokes, but allow them to stabilize for at least 2 weeks and then consider them for studies and possible operation. Rarely, if a patient is in the hospital when an acute stroke occurs, and operation can be done within 6 hours of onset, emergency endarterectomy may be justifiable.

On the basis of these considerations the patient with a totally occluded carotid artery should not be routinely operated upon nor should he be categorically rejected for operation. Judicious consideration of the following factors will aid in the selection of patients for operation and the appropriate timing of the procedures. If seen within 6 to 12 hours of onset of occlusion the patient may be operated upon with a reasonable expectation of restoration of cerebral blood flow. Thereafter the success rate progressively declines so that after a week or two chances of restoring flow drop to 20%. Clinical considerations of the stroke itself are the most important factors determining operability. Patients with acute profound strokes of 12 hours' duration or more should not be operated upon as an emergency but be allowed to stabilize for at least 2 weeks. Surviving patients with total occlusions having flow restored by endarterectomy show a more rapid and greater degree of long-term improvement than those who fail to have flow restored.[18]

By avoiding operation on acute strokes and by using a shunt routinely for all stenotic lesions, one reduces sharply the operative mortality following carotid endarterectomy. Table 9 summarizes our experience during the last 6 years since this policy was adopted. Mortality for frank strokes has been 3.7% while that for transient ischemia has been 0.77%, or an overall procedure mortality of 1.47%. Of the seven deaths, four were cerebral and three were cardiac in origin.

Asymptomatic Carotid Bruits

Considerable controversy exists concerning the advisability of performing arteriography and surgery on patients with asymptomatic carotid bruits. We have made a study of this problem in an attempt to determine whether such a course of action is justifiable and worthwhile. During 13 years we have performed 87 elective carotid endarterectomies upon 65 patients with asymptomatic carotid bruits (Table 1). There have been no operative deaths. One patient had a mild transient deficit postoperatively, with full recovery, while a second patient has a mild permanent deficit related to operation, probably due to embolization of debris during manipulation of the artery. During the fol-

TABLE 9. *Operative Mortality Following Carotid Endarterectomy Recent Experience—Six Years*

Clinical Category	No. of Patients	No. of Operations	No. of Deaths	Patient Mortality	Procedure Mortality
Frank stroke	112	136	5	4.5%	3.7%
Transient cerebral ischemia	206	261	2	0.97%	0.77%
Chronic cerebral ischemia	7	9	0	0	0
Asymptomatic bruit	51	70	0	0	0
Total	376	476	7	1.86%	1.47%

low-up period one patient has had a mild stroke with near-complete recovery, while a second patient had a severe stroke 1 year postoperatively.

In an analysis of bruits correlated with arteriographic findings we found that 90% of audible midcarotid bruits were due to internal carotid lesions, representing a stenosis of 50% or more.

In an effort to obtain a control series for comparison with our elective surgical series we have followed 37 patients with bruits who were not operated upon primarily. There were various reasons for not operating upon these patients at the time they were first seen. In the early days of the study we were unsure as to whether they should be operated upon at all. In some patients the bruit was unilateral and very soft. In some cases the patient did not wish to have arteriography or surgery considered, while in others the patient's referring physician did not wish to have anything done. Occasionally the treatment of other diseases took precedence over management of the bruit. In a few cases the patients did not return for regular follow-up after being instructed to do so.

These individuals have been followed up to 8 years after detection of the bruit, with an average follow-up of about 24 months. During this time 13 of the 37 patients have remained asymptomatic (35%). Fourteen patients developed episodes of transient cerebral ischemia and were operated upon. In this group four were found to have total occlusion of the artery with complete disappearance of the bruit prior to operation. Ten patients have had frank strokes without episodes of transient ischemic attacks at intervals varying from 2 days to 27 months following detection of the bruit. In these 10 patients total occlusions were found by arteriography in nine, in all of whom the bruit had disappeared. Six of these were operated upon. Thus, 24 of the 37 patients, or 65%, developed either symptoms of transient ischemia without strokes or frank strokes without episodes of transient ischemia during the follow-up period.

With a bruit audible over one carotid but none over the other, one frequently finds on arteriograms a stenotic lesion on one side and a totally occluded carotid on the other, even in the absence of symptoms. Disappearance of a bruit ordinarily means that the artery has become totally occluded. The importance of this observation lies in the fact that when stenotic arteries become totally occluded there is a significant incidence of acute frank stroke without an episode of transient cerebral ischemia. We have observed this in retrospect in our series of total carotids, and in nine of the 10 patients in this group of followed bruits.

The clinical consequences which we have observed in patients with carotid bruits during long-term follow-up may be listed thusly: (1) nothing may happen if a lesion is present in only one artery and adequate collateral circulation develops, (2) transient cerebral ischemic attacks

may supervene, (3) frank strokes may occur without intervening episodes of transient ischemia, (4) total occlusion of the artery may occur without any symptoms, and (6) total occlusion may occur with the production of either transient ischemia or a frank stroke.

As arteriography has become increasingly safer[12] we believe it should be recommended fairly liberally for studying patients with asymptomatic bruits. If a lesion is found and no contraindications to operation exist, endarterectomy may be cautiously considered. This is particularly true if bilateral stenoses are found, if stenosis is found on one side and total occlusion on the other, if the patient has major occlusive lesions elsewhere in the peripheral vasculature, if the patient is contemplating major surgery of another sort where a hypotensive episode might well result in a stroke, if a patient with bilateral bruits is found to have disappearance of a bruit even without symptoms, or the patient is in the younger age group (40's or 50's). It is important to consider the age and general status of the patient before deciding on operation. Thus an elderly poor-risk patient with generalized cerebrovascular, atherosclerotic, or other serious disease who presents with carotid bruits should not be considered for studies or operation because of his limited outlook.

The recent report of Javid *et al.*[14] on the natural history of carotid atheromas has important implications with regard to the management of carotid bruits. On serial arteriograms these authors noted no change in size of the atheromas in 38% of the lesions studied. There was a significant increase in 62% of the atheromas. An increase greater than 25% per year was noted in 34% of lesions, an increase of less than 25% per year in 20%, and recurrent stenosis or thrombosis in 7.4%. These studies when combined with our clinical observations reported above lend further credence to the thesis that an asymptomatic carotid bruit may not be an innocent lesion but may indeed represent a potential stroke hazard in certain individuals. If carotid endarterectomy is to be seriously considered for these patients, however, it is obligatory that the necessary precautions be taken to ensure that operative mortality and operation-related neurological deficits be at an absolute minimum, that is, in the neighborhood of 1% or below.

Cerebral Protection during Endarterectomy

Although the majority of patients requiring carotid surgery can tolerate temporary clamping of the artery without deleterious effects, the rest require some form of cerebral protection if strokes are to be prevented and aggravation of neurological deficits is to be avoided. That cerebral protection in the latter group is necessary and beneficial has been amply demonstrated.[13, 15, 17] Patients with severe vascular disease and multiple large-vessel occlusions are least tolerant of carotid clamping.

Several methods to determine the adequacy of cerebral blood flow during carotid clamping have been described. These include temporary occlusion under local anesthesia while checking the neurological status; observation of the adequacy of retrograde blood flow from the opened internal carotid; determination of blood pressure in the occluded distal internal carotid; monitoring of blood gases, especially jugular venous oxygen saturation; and electroencephalography.[19]

A number of technics have been devised to render cerebral protection during carotid surgery. These include general anesthesia, hypothermia, induced hypertension, controlled hypercarbia, administration of Diamox, intraluminal bypass shunts, and various combinations of these.[19]

When general anesthesia is regularly used, two principal methods of cerebral

protection are currently available: (1) hypercarbia, with or without induced hypertension, and (2) a temporary inlying bypass shunt. Recent discussion has centered around the necessity for routine use of a shunt. Several articles have appeared advocating the performance of carotid endarterectomy without a shunt while employing other adjuncts.[5, 13, 21]

The effectiveness of any method of cerebral monitoring or cerebral protection during carotid endarterectomy must be demonstrated in patients with transient ischemia and/or asymptomatic bruits since these are patients without neurological deficits prior to operation. The relative effectiveness of various methods of cerebral support may be judged from the results in patients with transient ischemia listed in several recent publications compared with results in the present series. The parameters used for comparison include operative mortality, the incidence of transient neurological deficits, and the incidence of permanent neurological deficits, both mild and severe.

De Weese *et al.*[8] used no special cerebral protection other than general anesthesia and maintenance of normal or slightly hypertensive blood pressure. They reported operative mortality of 3%, an incidence of transient weakness of 13%, and an incidence of permanent deficits of 7%.

Bloodwell *et al.*[5] employed hypercarbic general anesthesia without a shunt and reported operative mortality of 4.3% and an incidence of permanent neurological worsening of 2.6%.

Young *et al.*[21] using hypercarbic general anesthesia without a shunt, listed an operative mortality of 4.4% and an incidence of operation-related stroke of 2.9%.

Fields *et al.*[11] reporting for the Joint Study of Extracranial Arterial Occlusion, in which various methods of cerebral support were used, gave an operative mortality of 3.5%, an incidence of transient deficits of 1.8%, and an incidence of permanent deficits of 7.7%.

TABLE 10. *Operation—Related Neurological Deficits in Patients with Transient Cerebral Ischemia and Asymptomatic Bruits Using a Temporary Inlying Shunt*

Number of Patients—358 Number of Operations—465			
Severity of deficit	No.	Patient Incidence %	Procedure Incidence %
Permanent, severe; not related to operative technic	3	0.84	0.65
Permanent, severe; related to operative technic	1	0.3	0.2
Permanent, mild	5	1.4	1.1
Transient, mild	4	1.1	0.86

Table 10 shows the data in the authors' series, in which a shunt has been used in nearly all cases, and routinely since 1962, together with general anesthesia, without other adjuncts than adequate hydration and satisfactory levels of blood pressure. The operative mortality has been 1% (Tables 1, 9). The incidence of procedure-related transient deficits has been 0.9%, and that of permanent deficits, both mild and severe, has been 2%.

An additional and most strenuous test for any method of cerebral protection is its efficacy in the patient with ipsilateral carotid stenosis and contralateral total carotid occlusion, when the former is being operated upon. In a series of 92 such patients, Bloodwell *et al.*,[5] using general anesthesia and hypercarbia, reported an operative mortality due to stroke of 7.6%, and neurological worsening of 5.4%. Fields *et al.*,[11] summarizing the Joint Study data on 30 patients operated upon, reported no operative deaths while permanent neurological deficits related to operation occurred in three patients (10%).

There were 64 patients in the present series with unilateral carotid stenosis and

TABLE 11. *Timing of Carotid Endarterectomy for Cerebrovascular Insufficiency*

A. Elective operation
1. Stable stroke, recent or old
2. Transient cerebral ischemia
3. Asymptomatic bruit
4. Chronic cerebral ischemia
B. Delayed operation (days to weeks)
1. Acute stroke
2. Fluctuating stroke
C. Emergency operation (rare)
1. Frank stroke
a. Disappearance of bruit
b. Slowly worsening
c. Fluctuating
2. Transient cerebral ischemia
a. Severe stenosis, especially if bilateral
b. Disappearance of bruit

contralateral occlusion. Eighty-six operations were performed, 64 for partial and 22 for total occlusions. A shunt was used for all partially occlusive lesions. There was no operative death and no instance of production or aggravation of neurological deficits. This is further evidence for the efficacy of an internal shunt.

Granted that a shunt may not be necessary in every case, but in the absence of precise knowledge as to which patient needs it, we have elected to use it routinely. It adds very little to the operating time but does add a great deal of security. Combined with general anesthesia and gentleness during dissection it is the simplest and most reliable method of temporary cerebral support for keeping the complications of carotid surgery at a minimal and acceptable level. When one becomes familiar with its use, complications are few and it may be employed in any operating room without the necessity of other complicated apparatus. A number of surgeons share this view.[7, 14, 15]

Indications for and Timing of Operation

Clinical considerations in each of the four categories of patients determine the indications and contraindications for carotid endarterectomy. The procedure may be carried out at a time of election, delayed from several days to several weeks, or done as an emergency. Although delayed operation is an important principle in stroke surgery, emergency operation is not often necessary. Table 11 summarizes the appropriate timing of operation for the various clinical categories.

Occasionally one finds patients in whom occlusive lesions are located in the carotid artery on the ipsilateral or paradoxical side relative to the neurological signs and symptoms. A number of patients in this series have had such lesions removed surgically, and they have responded as expected had the lesions been located on the appropriate side. This observation is in keeping with the concept of total cerebral blood flow on the basis of collateral circulation, which gives one a sound basis for recommending endarterectomy of inappropriate lesions.

Operation is contraindicated for cervical occlusions in the presence of severe intracranial disease. Mild intracranial disease, however, is an indication rather than a contraindication for operation. Age itself is not necessarily a contraindication to operation when the patient's general condition otherwise does not pose any undue hazard.

Summary

Seven hundred forty-eight carotid endarterectomies were performed on 592 patients with cerebrovascular insufficiency during a 13-year period.

Overall operative procedure mortality was 2.7%. In the last 6 years, using a shunt routinely and avoiding operation on acute strokes, mortality was 1.47%. In frank strokes it was 3.7%; in transient ischemia, 0.77%; and zero for chronic ischemia and asymptomatic bruits. Incidence of operation-related deficits among transient ischemia and asymptomatic bruit patients

was 0.9% for transient weakness and 2% for permanent deficits.

Of 172 long-term deaths, 23 were due to cerebral causes, or 3.9% of the entire series. Among frank stroke survivors, 30.2% are normal and 58.7% improved. In transient ischemia survivors 81% are normal and 15.7% improved.

In 65 asymptomatic bruit patients operated upon electively, two had strokes during follow-up, one mild and one severe. Among 37 asymptomatic bruit control patients, 24 or 65% developed symptoms of transient ischemia or frank strokes.

Of 118 totally occluded carotid arteries explored, flow was restored in 48 (40.7%) but could not be restored in 70 (59.3%).

For cerebral protection during carotid endarterectomy the routine use of a temporary inlying bypass shunt with general anesthesia is advocated for all partial occlusions.

Endarterectomy is most useful for transient ischemia and selected patients with mild frank strokes and asymptomatic bruits. Acute profound and rapidly progressing strokes should not be operated upon as an emergency, but allowed to stabilize for several weeks and then be considered for possible operation.

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Discussion

Dr. Edward A. Stemmer (Long Beach): To date, we have performed 109 carotid endarterectomies, considerably fewer than Dr. Thompson. Like the authors, we have performed a sizable number (15% of the total) on patients with asymptomatic bruits associated with significant arterial stenosis. Usually, this operation was performed as a preventive measure prior to carrying out major vascular procedures in the same patient.

As the authors have stated, nonoperative treatment has little to offer these patients and we believe they are best treated with endarterectomy.

[Slide] This shows our results classified according to the technic of operation. The most significant observation was that the use of vasopressors to artificially elevate the blood pressure was distinctly detrimental to the outcome of operation. It resulted in high mortality and a high incidence of postoperative stroke.

We regularly employ hypercarbia which is carefully monitored to maintain a P_{CO_2} between 60 and 80 mm. Hg which is monitored during operation. We are convinced that hypercarbia is beneficial and it did improve our results.

Although it is probably true that most patients can be operated upon safely without a shunt, we agree with the authors that use of an internal shunt adds to the safety of the procedure in a significant number of patients. The advantages of a shunt certainly outweigh its disadvantages as our data indicates when we use CO_2 with the shunt. The 1.4% mortality represents a single patient who died of myocardial infarction 10 days postoperatively. We had no neurologic deficits.

With long-term follow-up, 72% of our patients with transient episodes were either cured or markedly improved. The two patients indicated in parentheses died of unrelated causes long after the carotid endarterectomy. By contrast, only 30% of those with fixed neurological deficits preoperatively were significantly improved. No patient who had undergone a so-called prophylactic endarterectomy suffered any adverse effects from operation. Most of our problems have occurred in patients with multiple sites of cerebrovascular disease. Sixty-six per cent of our patients had more than one vessel involved.

[Slide] This summarizes our usual management of patients undergoing carotid endarterectomy. We employ carefully monitored hypercarbia in the operating room with general anesthesia (preferably with penthrane to eliminate arrhythmias that can occur during hypercarbia). We use an internal shunt when the internal carotid artery is patent. We use systemic heparinization while the shunt is in place and we do not use vasopressors.

Finally, I would like to ask Dr. Thompson if he has long-term follow-up arteriography on those patients in whom a completely occluded internal carotid was reopened. We have also attempted to do this and have been able to open up about a fourth of the internal carotid arteries, but we have a serious question about how long they really stay open.

Dr. J. Alex Haller (Baltimore): I wish to ask one question of Dr. Thompson.

The most difficult group to evaluate are those with the asymptomatic carotid bruits. If I understood your figures correctly, Dr. Thompson, there were 37 patients with bruits alone who subsequently developed difficulty and of this group, ten of the 37 developed frank strokes.

If I remember correctly, patients who develop strokes fit in the categories of approximately one-fourth having carotid disease and three-fourths having intracranial cerebral disease.

I this is true also in your group, this would mean that perhaps two or three of the patients who developed frank strokes had this on the basis of carotid occlusion. What I would like to ask is whether the autopsies on these patients confirm the same incidence of intracranial disease as in the general population? One of the problems in this type of palliative operation is whether there is such a thing as "preventive palliation." Therefore, it would be of considerable consequence to know exactly where the occlusion occurred in those patients with asymptomatic bruits whom you have carefully followed and who subsequently developed fatal strokes.

Dr. Charles G. Rob (Rochester): I would wish to raise the question again of the asymptomatic bruit. Essentially, operations on these patients are "entirely prophylactic" and one must remember that the bruit comes and goes. It is heard one day and then perhaps not on the next day.