Carotid endarterectomy in diabetic patients

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Objective: **The purpose of the current study was to identify the possible short- and long-term effects of diabetes on the outcome of carotid endarterectomy.**

Methods: **Medical records were reviewed for 781 carotid endarterectomies (in 734 patients) performed by the same vascular surgeon in a university medical center between January 1994 and December 1998. Patients were divided two groups: those with diabetes (n = 181 patients; 193 operations) and those without diabetes (n = 553; 588 operations). The two groups were similar with respect to mean age, male-female ratio, and contralateral lesions. The only significant differences were a higher prevalence of peripheral vascular disease and dyslipidemia in the diabetic group and a higher prevalence of hemispheric transient ischemic attacks among the nondiabetic patients. Carotid color duplex ultrasound scan had been performed in all patients, and in 56 patients from the diabetic group and 56 patients from the nondiabetic group (matched for age, sex, and contralateral lesions), the distal extension of the lesion from the carotid bifurcation had also been defined. Both of these subgroups were fully representative of their respective groups of origin. Carotid endarterectomy was performed after the induction of general anesthesia; electroencephalographic monitoring was continuous.**

Results: **Except for the significantly higher prevalence of calcified plaques in the diabetic patients ($P < .0001$), the characteristics of the carotid disease in the two groups were similar. In the 56-member subgroups, 73.2% of the diabetic and 35.7% of the nondiabetic patients ($P < .0001$) had lesions extending more than 2 cm beyond the carotid bifurcation. Mean length of plaque beyond the bifurcation was 2.3 ± 0.09 cm for the diabetic and 1.7 ± 0.08 cm for the nondiabetic patients ($P < .0001$). Diabetes was the only factor significantly correlated with plaque length. In the diabetic subgroup, surgery was characterized by significantly longer carotid arteriotomies ($P = .03$) and clamp times ($P < .003$). Operative mortality was 1.5% in the diabetic group (2 myocardial infarctions + 1 stroke) and 0.5% in the nondiabetic group (1 myocardial infarction + 2 strokes; *P* value not significant); stroke rates were 1.5% (3 major strokes) and 0.5% (2 major strokes + 1 minor stroke), respectively (*P* = not significant). Long-term survival (5 years) was not significantly lower among the diabetic patients.**

Conclusions: **Diabetes mellitus does not seem to significantly increase the surgical risk for carotid endarterectomy. The presence of more extensive plaques has no significant effect on the results of surgery. (J Vasc Surg 2001;33:148-54.)**

Carotid endarterectomy (CEA) is justified only when the complication rate remains below 6% in symptomatic patients and is less than 3% for those without symptoms.[1-3] The main causes of operative mortality in these cases are myocardial infarction and stroke. Neurologic complications can be the result of technical errors during surgery, clamping ischemia, or hemorrhage. The identification of patients at risk for these complications is important because this information might influence indications and, in some cases, the surgical and anesthesiology techniques to be used.

A number of studies have demonstrated that diabetes mellitus is an independent risk factor for both stroke and myocardial infarction.[4-7] Alterations in the concentration and composition of lipoproteins, hyperinsulinemia, and hyperglycemia itself all contribute to the development of atherosclerosis, which appears earlier and is more widespread in diabetic than in nondiabetic patients.[8] The Framingham Study revealed that the risk of stroke in subjects with non–insulin-dependent diabetes is 2.6 times higher for men and 3.8 times higher for women than the risk in nondiabetic patients.[4] A Finnish study based on follow-up (mean, 3.5 years) of 1298 subjects between the ages of 65 and 74 years confirmed the increased risk of stroke among diabetic patients, particularly those who were more elderly and those who were female.[5] In a more recent study from Finland, in which 8077 men and 8572 women were followed up for a mean of 16.4 years, it was confirmed that diabetes is an important and independent risk factor for stroke. Once again, the risk was found to be especially high among women: 33% of all women who die of strokes are diabetics, as opposed to only 16% of men.[6]

The role of diabetes as a risk factor for carotid surgery is more controversial. Some groups maintain that the disease exerts a negative effect on the results of CEA,[8,9] but others insist that the outcomes are no different from those for nondiabetic patients.[10] The purpose of the current study was to identify the possible short- and long-term effects of diabetes mellitus on the outcome of CEA.

METHODS

Medical records were reviewed for 781 consecutive CEAs performed in 734 patients by the same vascular sur-

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Table I. Characteristics and histories of diabetic (group A) and nondiabetic (group B) patients undergoing CEA

	Group A (diabetic): n = 181	*Group B (nondiabetic): n = 553*	P *value*
Mean age; range (y)	67.3; 56-80	68.1; 48-85	NS
Male	143 pts (79.0%)	416 pts (75.2%)	NS
Female	38 pts (21.0%)	137 pts (24.8%)	NS
Atherosclerotic risk factors			
Smoking	129 pts (71.3%)	404 pts (73.1%)	NS
Hypertension	111 pts (61.3%)	356 pts (64.4%)	NS
Dyslipidemia	28 pts (15.5%)	54 pts (9.8%)	.03
Associated vascular disease			
Coronary	48 pts (26.5%)	126 pts (22.8%)	NS
Peripheral	62 pts (34.2%)	108 pts (19.5%)	<.0001
Aortic aneurysms	7 pts (3.9%)	39 pts (7.0%)	NS
Neurologic symptoms			
Asymptomatic	69 pts (38.1%)	177 pts (32.0%)	NS
Hemispheric TIA	69 pts (38.1%)	268 pts (48.5%)	.01
Vertebrobasilar TIA	28 pts (15.5%)	58 pts (10.5%)	NS
Stroke	15 pts (8.3%)	50 pts (9.0%)	NS

pts, Patients.

geon (G.R.P.) between January 1994 and December 1998. The patients were divided into two groups, the principal characteristics of which are shown in Table I. Group A (24.7% of the total study population) was composed of 181 diabetic patients, including 63 (34.8%) who were insulin dependent and 118 who were taking oral hypoglycemics; group B (75.3%) was composed of 553 nondiabetic subjects.

For the purpose of risk-factor analysis, each patient was classified as a smoker if he or she smoked at least one cigarette per day at the time of admission or had stopped smoking less than 10 years before admission. Hypertension was defined as a diastolic pressure greater than 90 mm Hg and/or a systolic pressure of more than 160 mm Hg (as measured on admission) and/or current use of antihypertensive drugs. Patients with elevated cholesterol and/or triglycerides levels on admission (measured in all cases) and those being treated with oral hypolipemics were classified as having dyslipidemia. Coronary artery disease was defined on the basis of histories and admission findings. All patients had undergone cardiologic workups with electrocardiograms and echocardiograms on admission. Those with findings and/or histories indicative of coronary artery disease had also had Holter monitoring, stress tests, myocardial scintigraphy, and, in some cases, cardiac catheterization. The presence of peripheral vascular disease was documented by means of ultrasonographic studies of the lower extremities followed by angiography, if indicated.

Color duplex ultrasound scan had been performed in all patients to define plaque morphology and degree of stenosis (based on measurements of peak systolic and end-diastolic velocities).[3] One hundred sixty (88.4%) of the diabetic and 467 (84.4%) of the nondiabetic patients had also undergone carotid angiography, in which stenosis was defined according to the North American Symptomatic Carotid Endarterectomy Trial criteria[1] on the basis of the ratio of the smallest diameter of the vessel at the level of stenosis to that of the distal segment of the unaffected internal carotid artery. Comparable findings were obtained with the two imaging techniques, which confirms the observations of various authors[11-13] on the accuracy of duplex scans. In our analysis, carotid disease was defined on the basis of ultrasound scan findings.

In patients operated on after September 1996 (85 diabetics, 298 nondiabetics), distal extension of the lesion from the carotid bifurcation had also been recorded during duplex scanning. To evaluate possible correlations between plaque size and preoperative risk factors, operative technique, and/or postoperative morbidity and mortality, we created two homogeneous subgroups from this subset of patients: subgroup A (diabetic patients) and subgroup B (nondiabetic patients). The subgroups (each containing 56 patients) were matched for age, sex, and the presence of bilateral lesions, and both subgroups were fully representative of their groups of origin (Table II). Every patient in the two subgroups underwent single CEA.

In 118 (65.2%) of the patients in group A and 389 (70.3%) of those in group B, cerebral vasomotor reactivity had been assessed preoperatively by means of transcranial Doppler (TCD) ultrasound scan through use of the breath-holding test.[14] Cerebrovascular autoregulation was defined as *sufficient* when the mean increase in middle cerebral artery flow velocity exceeded 30%, as *diminished* when the mean increase was between 15% and 30%, and as *exhausted* when the increase was less than 15%.

Twelve patients (6.6%) from group A and 35 patients (6.3%) from group B underwent both right and left CEAs on separate occasions; thus, a total of 193 diabetic carotids and 588 nondiabetic arteries were operated on. Each CEA was performed by the same surgeon with the patient under general anesthesia; electroencephalographic (EEG) monitoring was continuous. The anesthesiologic risk was classified according to the parameters of the American Society of Anesthesiologists.[15]

Table II. Characteristics and histories of subgroups A (diabetics) and B (nondiabetics): comparisons with groups of origin

	Subgroup A (diabetic): n = 56	P *value (vs group A)*	*Subgroup B (nondiabetic): n = 56*	P *value (vs group B)*
Mean age; range (y)	66.7 (58-80)	NS	66.9 (43-80)	NS
Male	48 pts (85.7%)	NS	46 pts (82.1%)	NS
Female	8 pts (14.3%)	NS	10 pts (17.9%)	NS
Atherosclerotic risk factors				
Smoking	41 pts (73.2%)	NS	44 pts (78.6%)	NS
Hypertension	29 pts (51.8%)	NS	36 pts (64.3%)	NS
Dyslipidemia	4 pts (7.1%)	NS	6 pts (10.7%)	NS
Associated vascular disease				
Coronary	16 pts (28.6%)	NS	16 pts (28.6%)	NS
Peripheral	24 pts (42.9%)	NS	11 pts (19.6%)	NS
Aortic aneurysms	—	NS	1 pt (1.8%)	NS
Neurologic symptoms				
Asymptomatic	21 pts (37.5%)	NS	17 pts (30.4%)	NS
Hemispheric TIA	22 pts (39.3%)	NS	28 pts (50.0%)	NS
Vertebrobasilar TIA	8 pts (14.3%)	NS	7 pts (12.5%)	NS
Stroke	5 pts (8.9%)	NS	4 pts (7.1%)	NS

pts, Patients.

To evaluate the possible influence of diabetes on the surgical procedure itself, we considered the following variables: length of arterial incision (2 cm or less past the carotid bifurcation vs more than 2 cm past the carotid bifurcation), use of tacking sutures, Dacron patch (Intervascular, La Ciotat, France) closure, and clamp time (in minutes).

Carotid artery patency was verified by means of duplex Doppler scans in each patient on the day of surgery. In those who experienced neurologic complications, brain computed tomography (CT) was performed. Follow-up evaluations, including color duplex ultrasound scan, were performed 1, 3, 6, and 12 months after surgery and at least once a year thereafter. The mean follow-up was 30 months (range, 3 months to 5 years).

The statistical significance of differences between the two groups was evaluated through use of the χ^2 test. Differences in clamp time, degree of stenosis, and plaque extension were assessed by means of 1-way analysis of variance. Logistic regression analysis was then performed to identify variables correlated with neurologic and/or cardiac morbidity/mortality during the postoperative phase. Variables considered were sex, age (> 65 years), diabetes, smoking, hypertension, dyslipidemia, coronary artery disease (cardiac complications only), degree of stenosis, presence of contralateral lesions, ischemic brain lesions on CT, and intraoperative shunting (neurologic complications only). Long-term mortality was evaluated through use of Kaplan-Meier survival analysis. Statistical significance was accepted when *P* was less than .05.

RESULTS

Table I shows the characteristics of the diabetic and nondiabetic groups. The only significant differences were the higher frequencies of peripheral vascular disease ($P < .0001$) and dyslipidemia ($P = .03$) among the diabetic patients and a higher prevalence of hemispheric transient ischemic attacks (TIAs) among the nondiabetic patients ($P = .01$). On the basis of histories and admission findings, coronary artery disease was found to be slightly more common among the diabetic patients, although this difference was not significant. As shown in Table III, the clinical characteristics of coronary artery disease in these patients was not significantly related to diabetes.

Table IV shows the characteristics of the carotid disease in groups A and B based on the results of (1) color duplex ultrasound scan (in most cases) or angiography and (2) CT. On average, stenosis was slightly more severe in arteries from the diabetic group, although CT-documented ischemic brain lesions were somewhat less common in these patients. However, the only significant difference between the two groups was the prevalence of calcified plaques identified by ultrasound scan studies, which was significantly higher in Group A (168/193 [87.0%] of the diabetic carotids operated on vs 249/588 [42.3%; $P < .0001$] of the arteries treated in Group B).

TCD findings in 118 diabetic and 389 nondiabetic patients showed that cerebral vasomotor reactivity was sufficient in 69 diabetic (58.5%) vs 244 nondiabetic patients (62.7%; *P* value not significant [NS]), diminished in 37 diabetic (31.4%) vs 101 nondiabetic patients (26.0%; *P* value NS), and exhausted in 12 diabetic (10.2%) vs 44 nondiabetic patients (11.3%; *P* value NS).

Ultrasound scan or angiographic characteristics of the carotid disease in subgroups A and B are shown in Table V. The two subgroups differed significantly only in mean plaque length beyond the bifurcation and number of lesions extending for more than 2 cm beyond the bifurcation; both of these were significantly higher for the diabetic subgroup. Multivariate analysis showed that plaque length was not correlated with smoking, hypertension, dyslipidemia, sex, or age, but there was a highly significant correlation with diabetes ($P = .0001$).

Table III. Characteristics of diabetic and nondiabetic patients with coronary artery disease who underwent CEA

Characteristic	*From group A (diabetic): 48 pts**	*From group B (nondiabetic): 126 pts†*	P *value*
Myocardial infarction	32 pts (66.7%)	64 pts (50.8%)	NS
Congestive heart failure	7 pts (14.6%)	23 pts (18.2%)	NS
Angina	5 pts (10.4%)	21 pts (16.7%)	NS
Coronary revascularization	4 pts (8.3%)	18 pts (14.3%)	NS

*Consisting of 26.5% of group A.
†Consisting of 22.8% of group B.
pts, Patients.

Table IV. Characteristics of carotid disease* in diabetic and nondiabetic patients who underwent CEA

Characteristic	*Group A (diabetic): n = 181*	*Group B (nondiabetic): n = 553*	P *value*
Unilateral carotid stenosis	114 pts (63.0%)	366 pts (66.2%)	NS
Bilateral carotid stenosis	43 pts (23.8%)	116 pts (21.0%)	NS
Contralateral carotid occlusion	24 pts (13.2%)	71 pts (12.8%)	NS
Percent stenosis (mean ± SEM)	84.1% ± 0.97%	82.8% ± 0.61%	NS
Ischemic brain lesions on CT	56 pts (30.9%)	190 pts (34.4%)	NS

*Findings are based on duplex Doppler scan data.
pts, Patients.

The operative risk was classified as American Society of Anesthesiologists group III-IV in 34.2% of the diabetic patients (n = 62); this compared with 26.6% of the nondiabetic patients (n = 147), but the difference was not statistically significant.

Cross-clamping was associated with EEG changes requiring temporary shunting in a slightly lower percentage of the operations performed on diabetic patients (23/193 [11.9%] vs 78/588 [13.3%] for nondiabetic patients; *P* value NS). A more distal dissection and a longer arteriotomy were required in a higher percentage of the operations performed on diabetic patients, and the mean clamp time for the CEAs in subgroup A was also significantly longer. Analysis of surgical variables in subgroups A and B (Table VI) revealed that longer arteriotomies (> 2 cm past the bulb) were significantly more common in the diabetic subgroup, which reflects the presence of longer plaques seen on preoperative ultrasound scan studies. As a result, clamp time was also significantly prolonged in subgroup A. Tacking sutures and Dacron patches were used with similar frequencies in the two subgroups.

Short-term (ie, 30-day) mortality/morbidity figures are shown in Table VII. The overall rate of complications was quite good, and with respect to mortality rates the two groups were not significantly different. Strokes were actually more common among the nondiabetic patients, although this difference was not significant. The only significant difference was a higher rate of nonfatal complications in group A ($P = .02$). Multivariate analysis revealed no significant correlation between neurologic or cardiac complications and any of the variables assessed (including diabetes).

Postoperative duplex scans confirmed the patency of the carotid arteries that had been operated on in all patients, including those who had experienced neurologic complications. In all of the latter patients, neurologic deficits were observed as soon as the patients regained consciousness. Brain CTs showed no hemorrhages. In one patient from group B, a review of the intraoperative EEG tracing revealed clamp ischemia that had not been reported by the neurologist during surgery. All of the remaining neurologic complications were, in our opinion, caused by embolic phenomena.

Of the 728 patients who survived the first 30 days after surgery, 70 were lost to follow-up; long-term data were thus available for 166 (91.7%) of the group A patients and 492 (89%) of the group B patients. The overall mortality rate for the population analyzed, with a mean follow-up of 30 months (range, 3 months to 5 years), was 11.8% (78 patients): 17.5% (29 patients) for group A and 10% (49 patients) for group B (*P* value NS). The single most frequent cause of long-term mortality was coronary artery disease (39 patients [50%]: 14 [48.3%] from group A and 25 [51%] from group B [*P* value NS]). Cancer was the second most common cause of death (18 patients [23.1%]: 7 [24.1%] from group A and 11 [22.4%] from group B [*P* value NS]) and stroke the third (3 patients [3.8%]: none from group A and 3 [6.1%] from group B [*P* value NS]). Nonfatal strokes occurred in three patients (0.5%): 1 (0.7%) from group A and 2 (0.4%) from group B (*P* value NS).

DISCUSSION

Data from several randomized multicenter trials confirm that surgical treatment is superior to medical management in the prevention of stroke in symptomatic and asymptomatic patients with severe carotid stenosis.[1-3] These studies have also shown, however, that the benefits

Table V. Characteristics of carotid disease in subgroups A (diabetic) and B (nondiabetic) in which plaque length was measured ultrasonographically

	Group A (diabetic): n = 181	*Group B (nondiabetic): n = 56*	P *value*
Unilateral carotid stenosis	35 pts (62.5%)	35 pts (62.5%)	NS
Bilateral carotid stenosis	13 pts (23.2%)	13 pts (23.2%)	NS
Contralateral carotid occlusion	8 pts (14.3%)	8 pts (14.3%)	NS
Percent stenosis (mean ± SEM)	83.2% ± 1.55%	82.9% ± 1.54%	NS
Mean length of plaque (cm)	2.3 ± 0.09	1.7 ± 0.08	<.0001
Lesion size > 2 cm	41 pts (73.2%)	20 pts (35.7%)	<.0001
Ischemic brain lesions on CT	16 pts (28.5%)	19 pts (33.9%)	NS

pts, Patients.

Table VI. Analysis of surgical variables in subgroups A (diabetic) and B (nondiabetic)

Surgical variable	*Subgroup A (diabetic): n = 56*	*Subgroup B (nondiabetic): n = 56*	P *value*
Longer arteriotomies (> 2 cm)	12 pts (21.4%)	4 pts (7.1%)	.03
Clamp time (min)	24.1 ± 0.73	21.5 ± 0.44	<.003
Dacron patches	12 pts (21.4%)	9 pts (16.1%)	NS
Tacking sutures	7 pts (12.5%)	4 pts (7.1%)	NS

pts, Patients.

of surgery, in terms of stroke risk reduction, are inversely proportional to the rate of perioperative complications. Maintaining cumulative mortality/morbidity rates within acceptable limits depends on the criteria used for patient selection, the surgical and anesthesiologic techniques used, and the prevention of intraoperative and postoperative complications. Identification of high-risk patients is particularly important, inasmuch as the presence of certain conditions may influence treatment options and indications, surgical strategies, and/or the approach to anesthesia.

Risk assessment in a candidate for CEA is based on a number of factors, including neurologic symptoms, plaque characteristics, unilateral vs bilateral involvement, vasomotor reactivity, and the presence of ischemic brain lesions on CT.[16-19] A complete understanding of the patient's general condition can play an important role in reducing perioperative morbidity and mortality and improving the long-term prognosis. The single most important factor to consider in the timing of carotid surgery is the cardiologic risk,[20] and arterial hypertension, which promotes hemorrhage and myocardial infarction, is unanimously regarded as a major risk factor.[21-25]

The significance of diabetes mellitus is somewhat less clear, and not all investigators agree that the disease is an independent risk factor for cardiac and neurologic complications after CEA. In 1990, Salenius et al[8] affirmed that diabetes was indeed a predictor of post-CEA stroke, which occurred in 20% of diabetic patients but only 8.2% of those without diabetes. This conclusion was based on the authors' retrospective analysis of a series of patients operated on between 1965 and 1984, and its value is limited by an overall frequency of neurologic complications that is clearly unacceptable by today's standards. In a more recent study of 732 patients, 284 of whom were diabetics, Akbari et al[10] found that neurologic morbidity and mortality were similar in the two groups. However, cardiac complications were decidedly more common among the diabetic patients; this was due, in all probability, to the significantly higher prevalence of preexisting heart disease in these patients. In contrast, the study by Ombrellaro et al[20] of 266 patients with CEA, including 62 diabetic patients, revealed no significant difference with respect to the rate of cardiac complications between patients with and patients without diabetes (14% and 16%, respectively).[20]

In the current series, the diabetic and nondiabetic groups were not significantly different as far as the preoperative prevalence of heart disease is concerned. People with diabetes are known to have a higher-than-normal incidence of silent myocardial disease,[26-29] however, and the true rates of coronary artery disease in our study groups is unknown, inasmuch as stress tests, Holter monitoring, myocardial scintigraphy, and cardiac catheterization were not routinely included in the preoperative cardiac workup. Nonetheless, the surgical results achieved in the diabetic patients were good. Multivariate analysis showed that diabetes mellitus is not an independent risk factor for post-CEA morbidity or mortality.

From a clinical point of view, the significantly higher prevalence in the diabetic group of peripheral vascular disease ($P < .0001$) reflects the more extensive atherosclerotic involvement in these subjects. Recent ultrasound scan studies have shown that intima-media thickening occurs earlier and atherosclerotic plaques are more common in diabetic patients without any cerebrovascular symptoms than in nondiabetic patients.[30-32] Hyperinsulinemia seems to play a central role in this process by stimulating the pro-

Table VII. Short-term (30-day) mortality and morbidity in diabetic (group A) and nondiabetic (group B) patients undergoing CEA

	Group A: diabetics	*Group B: nondiabetics*	*Total*	P *value*
No. of operations	193	588	781	—
Fatal cardiac events	2 (1.0%)	1 (0.2%)	3 (0.4%)	NS
Fatal neurologic events	1 (0.5%)	2 (0.3%)	3 (0.4%)	NS
Mortality (all causes)	3 (1.5%)	3 (0.5%)	6 (0.8%)	NS
Nonfatal cardiac events	2 (1.0%)	1 (0.2%)	3 (0.4%)	NS
Nonfatal neurologic events	4 (2.1%)	4 (0.7%)	8 (1.0%)	NS
Morbidity (all causes)	6 (3.1%)	5 (0.8%)	11 (1.4%)	.02

liferation and migration of smooth muscle cells, increasing lipid stores in muscle cells and fibroblasts, reducing the elimination of cholesterol from foam cells, and stimulating the release of growth factors.[33]

The influence of diabetes on neurologic morbidity/mortality is also the subject of some debate. In the current study, fatal and nonfatal neurologic events were slightly, though not significantly, more common in the diabetic group despite the fact that this group contained higher percentages of asymptomatic patients and patients without ischemic lesions before surgery. Calcified plaques were significantly more common in group A, which also presented a higher mean percentage of stenosis. The relative stability of calcified plaques and the fact that ultrasound scan screening studies are performed more frequently in diabetics may explain the higher number of asymptomatic subjects in this group, which is consistent with the findings of other investigators,[30,34] as well as the lower rate of ischemic lesions on preoperative CT.

Skydell et al[9] found that diabetes was the only variable associated with post-CEA hypertensive crises leading to neurologic complications and hypothesized that the role of the disease was based primarily on the negative effects that it exerts on cerebrovascular autoregulation. However, this conclusion is in contrast with our previous observations[35,36] and the results of TCD ultrasound scan in the current study. The percentage of patients with exhausted cerebral vasomotor reactivity in group A was not any higher than that in group B, and clamp ischemia, which is a reflection of the inefficiency of collateral circulation and vasomotor autoregulation, was actually less common in the diabetic group (11.9% of the operations) than in the nondiabetic group (13.1%).

Riles et al[37] analyzed the causes of stroke after CEA and concluded that 65% of these events can be attributed to surgical errors. In their study of 2365 patients (3062 CEAs), the overall incidence of stroke was 2.2%, as opposed to 3.6% for the subgroup with diabetes. Even in the latter cases, however, technical errors appeared to play a central role, and the authors concluded that the brains of diabetic patients seem to be more susceptible to ischemic insults. This hypothesis is supported by data from studies on experimentally induced diabetes,[38] in which the incidence of brain damage after ischemic insults was found to be higher in the presence of altered glucose metabolism. Prompted by these observations, we attempted to determine how plaque characteristics in the diabetic affect surgical technique. This analysis was limited to subgroups of the diabetic and nondiabetic patients because plaque dimensions had not been recorded on duplex Doppler scans performed in the earlier cases. As shown in Tables II and V, however, these subgroups were fully representative of their groups of origin. Ultrasound scan data on plaque length could not be confirmed by intraoperative measurements, which were unavailable in most of these patients' charts, but our experience (unpublished data), as well as that of other investigators,[39] indicates that this noninvasive method provides highly accurate data on plaque morphology and dimensions even in the presence of heavy calcification. Our findings show that diabetes is a major independent predictor of plaque length in patients undergoing CEA, and this factor was associated with longer arteriotomies and longer clamp times in the diabetic subgroup. However, although almost all of the neurologic complications observed in the current series appear to be related to intraoperative embolization, these events occurred with similar frequencies in the diabetic and nondiabetic groups. Moreover, diabetes did not seem to be a negative prognostic factor as far as long-term survival after CEA is concerned.

Although the overall long-term mortality rate was higher among the diabetic patients whom we studied than among the nondiabetic patients (17.5% vs 10%), this difference was not statistically significant. Moreover, in both groups, more than half of the deaths that occurred after the first 30 days were related to myocardial infarction, but these events were actually somewhat more frequent among nondiabetic patients. Strokes, both fatal and nonfatal, were also slightly more common in group B. Our findings thus contrast with those of Hallett et al,[40] who studied a series of 254 cases of CEA performed at the Mayo Clinic (Rochester, Minn) between 1970 and 1995. In that investigation, there was a drastically reduced 8-year survival rate among the 56 diabetic patients of the study population—ie, 27% vs 67% in controls ($P = .002$). The significance of this discrepancy is difficult to explain, but it might simply reflect the longer follow-up of the Mayo Clinic study (8 years vs 5 years in the current study).

CONCLUSIONS

Our experience indicates that diabetes is associated with (1) atherosclerotic disease that is more diffuse than that seen

in nondiabetic patients and (2) more extensive plaques in the internal carotid arteries. However, diabetes does not appear to be an independent risk factor either for cardiac or neurologic events (fatal or nonfatal) after CEA or for the long-term rates of mortality and morbidity. Therefore, in our opinion, CEA remains the most effective means for prevention of stroke, even for this group of patients.

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