The role of carotid screening before coronary artery bypass

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Five hundred thirty-nine patients with no symptoms of cerebral ischemia undergoing coronary artery bypass were preoperatively evaluated for presence of cartoid stenosis by noninvasive methods (duplex scanning and ocular pneumoplethysmography-Gee). Overall prevalence of carotid stenosis > 75% was higher (8.7%) than that generally reported. Age > 60 years was significantly related to presence of carotid stenosis > 75% (11.3% vs 3.8%, $p = 0.003$). Risk factors such as hypercholesterolemia, hypertension, diabetes mellitus, and smoking were not predictive for carotid stenosis, postoperative stroke, or death. Carotid stenosis > 75% (odds ratio 9.87, $p < 0.005$) and coronary artery bypass redo (odds ratio 5.26, $p < 0.05$) were both independent predictors of stroke risk. Patients were divided into four groups: group 1, minimal or mild degree of carotid stenosis (<50%), not submitted to prophylactic carotid endarterectomy (432 patients, 80.1%); group 2, moderate degree of stenosis (50% to 75%), no prophylactic carotid endarterectomy (60 patients, 11.2%); group 3, severe carotid stenosis; (>75%), submitted to prophylactic carotid endarterectomy (19 patients, 3.5%), group 4, severe carotid stenosis (>75%) no prophylactic carotid endarterectomy (28 patients, 5.2%). Patients in group 4 had significantly higher stroke rate (14.3%) compared to the other three groups (1.1%) ($p = 0.0019$). The finding of carotid stenosis greater than 75% in patients over 60 years of age was associated with occurrence of stroke in 15% of cases. Carotid screening is helpful to determine patients at increased risk of stroke and should be performed in patients >60 years. Although mechanism of stroke is unknown in patients with carotid stenosis, prophylactic carotid endarterectomy may reduce its occurrence (J VASC SURG 1990;12:724-31.)

In an effort to reduce the stroke rate of patients after coronary artery bypass (CAB), many authors have studied the prevalence of the extracranial carotid disease and its role in determining neurologic morbidity and mortality rates. Carotid stenosis (CS) is usually reported as infrequent in these patients. Studies on large series of patients documented presence of carotid lesions greater than 50% in only 2% to 4% of cases.[1-3] Although the presence of CS increases the stroke risk after CAB, the role of prophylactic endarterectomy remains controversial.

We have tried to assess the prevalence of CS in patients undergoing heart revascularization procedures by retrospectively reviewing 539 patients treated in an institution where noninvasive carotid examination (duplex scanning and ocular pneumoplethysmography [OPG]) is routinely performed and to correlate it with the perioperative stroke and mortality rate. Hypotheses on the mechanism of stroke after CAB are also discussed.

MATERIAL AND METHODS

Data from all patients undergoing CAB from Jan. 1, 1989, through Sept. 30, 1989, were reviewed ($N = 539$); gender, age, and preoperative risk factors were noted. No patient had experienced neurologic symptoms within 12 months of CAB, although nine patients had a remote history of neurologic events (four transient ischemic attack [TIA], five stroke).

All the patients were preoperatively evaluated by duplex scanning (Acuson 128; Acuson Inc., Mountain View, Calif.) and OPG-Gee. Carotid lesions were classified in three groups (<50%, 50% to 75%, >75%), and OPG was considered abnormal when pressure difference between the eyes was >5 or the eye/arm ratio was <0.69. When a stenosis > 75% was identified, decision about prophylactic carotid endarterectomy (PCE) was made by the primary phy-

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Table I. Association between risk factors and carotid stenosis >75% (unilateral or bilateral)

	No. patients	*Percent of patients*	*Risk of stenosis >75% (odds ratio)*
Gender, female	163	30.2	1.27
Hypercholesterolemia	358	66.4	1.34
Hypertension	252	46.8	1.56
Diabetes mellitus	106	19.7	1.35
Smoking	316	58.6	0.75

Table II. Association between risk factors and postoperative mortality and stroke rates (odds ratio)

	No. patients	*Percent of patients*	*Odds ratio*	
			Mortality	*Stroke*
Gender, female	163	30.2	0.50	0.81
Hypercholesterolemia	358	66.4	0.43	0.24
Hypertension	252	46.8	0.72	0.87
Diabetes mellitus	106	19.7	1.34	0.36
Smoking	316	58.6	0.90	0.86
CS >75%	47	8.7	0.68	9.87*
PPVS	13	2.4	23.57†	—
CAB redo	40	7.4	3.8‡	5.26§

PPVS, Previous peripheral vascular surgery.
*$p < 0.005$.
†$p < 0.001$.
‡$p < 0.07$.
§$p < 0.05$.

sician/surgeon. Prophylactic carotid endarterectomy before CAB was performed with the patient under cervical block without electroencephalographic monitoring.

The patients were divided into four groups according to the status of their carotid disease and the treatment received: group 1, minimal or mild degree of stenosis (<50%), no PCE; group 2, moderate degree of stenosis (50% to 75%), no PCE; group 3, patients with an asymptomatic severe stenosis (>75%) submitted to PCE; group 4, patients with an asymptomatic severe stenosis (>75%) not submitted to PCE.

All patients with a potential postoperative neurologic deficit were evaluated by a neurologist, and most had a CT scan.

Attempts were made to correlate both the severity of CS and incidence of postoperative neurologic events to one or more of the risk factors. The incidence of postoperative neurologic deficit was related to the severity of CS and performance of PCE.

Statistical analysis was by chi-square analysis and Fisher's exact test when appropriate. Odds ratios (OR) for risk factors were calculated with the logistic regression model simultaneously considering the following variables: gender, hypercholesterolemia (>200 mg/dl), hypertension, diabetes mellitus, smoking, CS > 75% either unilateral or bilateral, previous peripheral vascular surgery, and CAB redo. This analysis indicates the ratio of the odds for end point occurrence where the risk factor is present to the odds for end point occurrence if the risk factor is absent.[4]

RESULTS

The average age of our patient group was 63.45 ± 9.23 years. A demographic description of the patient population with its risk factors is presented in Tables I and II.

The population was predominantly male; smoking, hypertension, and elevated cholesterol were common. The distribution of extracranial carotid disease in these patients is presented in Table III. Severe stenosis was unilateral in 31 cases and bilateral in 16. Eleven patients had carotid occlusions; six contralateral to stenosis > 75% and five contralateral to stenosis ≤ 50%. Prophylactic carotid endarterectomy was performed in 19 patients. In most (17) it was undertaken several days before CAB, whereas in two cases both operations were performed under the

Table III. Frequency of postoperative neurologic deficit and mortality rate by group

Group	*No.*	*TIA*	*Stroke*	*Mortality rate*
1	432 (80.1%)	4 (0.9%)	5 (1.2%)	8 (1.8%)
2	60 (11.2%)	1 (1.7%)	0	2 (3.3%)
3	19 (3.5%)	0	0	0
4	28 (5.2%)	1 (3.5%)	4 (14.3%)*	2 (7.1%)
Total	539 (100%)	6 (1.1%)	9 (1.7%)	12 (2.2%)

*$p = 0.0019$ compared to group 1+2+3.

same anesthetic. Prophylactic carotid endarterectomy was not performed in 28 patients for a variety of reasons. Six of these patients had unilateral asymptomatic carotid occlusion. We could demonstrate no difference between the patients in groups 3 and 4 other than the increased incidence of asymptomatic occlusion in group 4.

Operative mortality and stroke rates were independent of gender and the coexistence of diabetes, hypertension, hypercholesterolemia, and smoking. Overall postoperative mortality rate was 2.2% (12 patients). Death occurred from 0 to 28 days after surgery and was caused by cardiac complications, (i.e., myocardial infarction, heart failure) in nine patients. Causes of death in the three remaining patients were respiratory failure, pulmonary embolism, and the multiple organ failure, respectively. No patients died as the result of stroke. No deaths occurred in patients with PCE and CAB (group 3).

Postoperative death rate was higher in patients in group 4 (7.1%) compared to the other three groups (group 1, 1.8%; group 2, 3.3%; group 3, 0%), but this difference did not achieve statistical significance ($p > 0.1$). Increase in death rate was observed in patients submitted to CAB redo (OR, 3.88; $p = 0.072$) and previous peripheral vascular surgery (OR, 23.57; $p < 0.001$; Table I).

Neurologic complications were observed in 15 cases (2.8%) and consisted of nine strokes and six TIAs. None of the nine patients with a remote history of stroke or TIA had a postoperative neurologic deficit in this series. None of the patients with TIAs experienced a subsequent stroke. The occurrence of TIAs was randomly distributed through all patient groups (p = NS). The single patient with >75% stenosis who experienced a TIA had an arm hemiparesis contralateral to the side of his CS on the ninth postoperative day. Another patient with stenosis <50% had a right hemispheric TIA together with an episode of peripheral embolization.

Postoperative stroke occurred in nine patients and was more common in patients with CS > 75% and no PCE (group 4) ($p = 0.0019$) (Table III). Although the numbers of patients in groups 3 and 4 are small, no patients who underwent PCE in this series had a postoperative neurologic deficit. Significantly all strokes in group 4 were in the hemisphere ipsilateral to the CS. In these four patients CS > 75% was bilateral in two patients. Although there were six patients in group 4 with carotid occlusion, none of them had a postoperative stroke. Postoperative stroke incidence was significantly higher in patients with CS > 75% (OR, 9.87; $p < 0.005$). Coronary artery bypass redo was also an independent predictor of stroke (OR, 5.26; $p < 0.05$, Table II).

Since CS > 75% was associated with an increased risk of postoperative stroke, we evaluated risk factors that might predict the presence of severe carotid disease. Of all the risk factors evaluated, only age was related with increased prevalence of significant carotid bifurcation stenosis. Each decade of age increased the odds of CS > 75% by 2.03 ($p < 0.001$). The other risk factors evaluated were not related significantly to the presence of a stenosis > 75% (Table I). We could not demonstrate any correlation between the number of risk factors and the complication or mortality rate.

Carotid stenosis was infrequent in patients less than 60 years (3.8%) but was seen in 11.3% of those greater than 60 years of age ($p = 0.003$). Although the incidence of postoperative stroke increased with age, this increase was seen almost exclusively in older patients with CS > 75%. In patients older than 60 years with CS > 75% the stroke rate was 15% compared to a rate of 0.6% in patients of similar age without evidence of severe extracranial cartoid disease.

Stroke seemed to occur in two distinct clinical patterns. Patients with CS < 75% manifested their neurologic deficit on recovery from anesthesia in two cases and in three cases within the first postoperative week. In contrast, the four patients with >75% CS all awoke without neurologic deficit and experienced their ischemic event 2 to 9 days after surgery. In all

Table IV. Incidence of CS in patients undergoing CAB reported from the literature

Author	*Year*	*No. patients*	*Percent of patients*	*Degree of stenosis*
Turnipseed[6]	1980	170	11.8	>50%
Brener[1]	1984	1910	2.5	>50%
Cosgrove[2]	1985	NS	1.7	NS
Barnes[5]	1985	449*	18.9	>50%
Brener[3]	1987	4047	3.8	>50%
Minami[9]	1988	1471	3.2	>75%/ulcerated
Hertzer[15]	1989	9714	2.8	>70%
Our data	1990	539	8.7	>75%

NS, Not specified.
*CAB and peripheral vascular disease considered together.

Table V. Progression of the risk of stroke in relationship to age according to the literature

	CAB	*CAB*	*CAB + CE*
Age	*Gardner[24]* %	*(our data)* %	*Hertzer[14]* %
<45	0.2	—	—
51-60	1.3	1.5	8.3
61-70	3.0	0.8	9.0
71-75	6.3	3.2	
>75	8.0	6.25	14.0 (>70)

CE, Carotid endarterectomy.

cases with CS > 75% the event was in the hemisphere ipsilateral to the stenosis.

All but one of the patients with strokes survived; the only patient who eventually died had a bilateral >75% CS and manifested a left hemispheric stroke on this sixth postoperative day. Death occurred from multiple organ failure after 4 weeks.

DISCUSSION

Most large studies report a low incidence of CS in patients undergoing CAB (Table IV), with stenosis > 50% found in only 2.3% to 3.2% of patients. Only a few reports suggest higher incidence of CS in these patients.[5,6] The diagnostic methods and criteria for identifying significant stenosis were different in each report. Carotid bruits,[6-8] continuous wave Doppler,[3,6,9,10] OPG,[1,3,5,11,12] angiography,[2,9,13-15] and duplex scanning[1,3] have all been used to assess the presence of CS and to determine its severity.

The most reliable estimate of CS can be obtained either with angiography or duplex scanning.[16-20] The latter, because it is not invasive, can be safely used in the screening of a population at risk for stroke. However, the policy of carotid screening of all the patients undergoing CAB remains controversial. The low incidence of CS reported, cost of the techniques, and equivocal benefit obtained with PCE are all arguments made against routine carotid screening in asymptomatic patients.[21,22]

On the other hand, several authors have stressed the importance of accurate noninvasive carotid assessment before CAB.[3,5,9,12] Studies have shown a significant higher neurologic complication rate after CAB in patients with CS. Brener et al.[1,3] in two consecutive papers showed significant increase of stroke in patients with CS > 50% undergoing CAB. Hertzer et al.[14] reported an incidence of neurologic deficits as high as 20% in patients with CS > 50% and contralateral occlusion or 90% stenosis documented by angiography.

Death after CAB also seems to increase in the presence of CS. Several studies documented a higher mortality rate (up to 14.3%) in patients with CS > 50%.[1,3,10]

Therefore irrespective of the issue of PCE, carotid evaluation before CAB can provide important information about the patients risk of stroke and/or death after CAB. The role of PCE in these patients remains unclear. Brener et al.[3] and Barnes et al.[5] suggest that simultaneous PCE will not reduce the morbidity and mortality that follows CAB in patients with severe CS. Others have performed PCE with acceptable re-

sults in selected patients,[22,23] and Hertzer et al.[13-15] believe that simultaneous PCE reduced morbidity and mortality in their experience. There is evidence to suggest that PCE performed before CAB will lower postoperative stroke rate, and our experience, although small, tends to support this.

The carotid screening in our group of patients with duplex scanning showed an unexpectedly high frequency of CS, three times greater than that usually reported: almost 20% of patients had a diameter reduction >50%, and 8.7% had a severe stenosis, either unilateral or bilateral. The severity of CS increased with age. In particular, patients older than 60 years had an incidence of CS > 75 % significantly greater than the rest of the population undergoing CAB.

The postoperative risk of stroke was higher in patients with >75% stenosis and significantly increased over that of patients with normal carotid artery, CS < 75%, or CS >75% previously treated with PCE. The risk of stroke was particularly high in patients older than 60 years with CS > 75% (15%). This finding has been previously noted by other authors. Gardner et al.[24] described progression of stroke risk with advancing age and Hertzer et al.[14] reported the same trend in a group of patients submitted to concomitant carotid and coronary procedures (Table V).

We support carotid screening before CAB in patients over 60 years of age. This policy follows from our observation of an increased prevalence of CS > 75% in this age group and an increased postoperative stroke rate in older patients with severe bifurcation disease. Although the role of PCE in these patients remains unresolved, carotid screening can identify a high-risk group who should be the subject of further clinical trials in attempts to reduce these complications.

Mechanism of stroke during CAB procedures is not clearly understood. Different causes proposed include embolization from the heart,[7] from atherosclerotic plaques in the aorta or in the extracranial vessels,[6,7,24] low flow during cardiopulmonary bypass,[1] and sudden blood pressure changes in the postoperative period.[6,25]

During CAB procedure the risk of stroke has been related to the presence of aortic arch disease and duration of cardiopulmonary bypass.[24] Jones et al.[7] reported data that supported cardiac embolism as a major cause of stroke, rather than low-flow states. The study from Kartchner and McRae,[11] which reported that five of seven perioperative strokes in a series of 234 patients occurred from 3 to 5 days after surgery, and from Turnipseed et al.,[6] where postoperative hemispheric events usually did not occur during surgery, suggest that intraoperative events may be less important than those occurring in the postoperative period.

Our data suggest the presence of two different groups of patients in which stroke occur with different mechanisms. The first group includes patients with CS < 75%. In this group five strokes occurred, two during surgery and three during the first postoperative week. Pathogenesis of stroke in these patients is presumably related to a variety of causes, for example, embolization from the heart and from atherosclerotic plaques in the aortic arch, coagulability problems, and blood pressure changes. The second group is composed of patients with CS > 75%. Some authors postulated that low flow states during surgery are the major cause of cerebral ischemia in such cases.[1] The four patients with CS > 75% who had a postoperative stroke in our series all developed neurologic symptoms after the second postoperative day. In addition, none of the six patients with unilateral carotid occlusion in group 4 had a neurologic deficit after surgery. This supports the observations of other authors[6,11,25] and suggests that postoperative changes are more important than intraoperative events in these patients.

The nature of these postoperative alterations is unknown but might include changes in blood viscosity, platelet adhesion, or hemodynamics. Our study is retrospective and the numbers too small to be conclusive; a prospective, larger study in this issue is needed to understand pathogenesis of stroke during CAB in patients with CS. The therapeutic implications are obvious.

In conclusion, we observed a high incidence (20%) of severe (8.7%) and moderate (11.2%) CS in patients undergoing CAB. Presence of CS > 75% was increased in patients over age 60 years, and led to an unacceptable risk of stroke. Although the mechanism of cerebral ischemia during CAB is not completely clear in patients with CS, our data suggest that its occurrence, as well as mortality rate, may be reduced by PCE.

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DISCUSSION

Dr. David E. Eisenbud (Newark, N.J.). The current report addresses several aspects of the problem of detecting carotid disease with coronary disease that deserve further attention. The first issue relates to frequency of CS in patients undergoing CAB.

Several years ago we undetook a prospective study involving the noninvasive screening of all cardiac surgical patients, and most of the severe stenoses that were detected were confirmed angiographically. We found that of the more than 2000 patients screened, 2.5% had a significant stenosis and another 0.6% had already had their carotid arteries operated on. A higher incidence, about 8% or 9% of significant disease found in today's study may reflect a trend in coronary surgery toward operating on older patients with significant associated problems. It is not stated in this report how many of those were confirmed angiographically.

The rate of perioperative TIA or stroke was markedly increased in the group with significant carotid disease, 15% versus 2% in the nondiseased carotid arteries. The figures from today's study are remarkably similar.

Of course, the more controversial aspect of the present study is the proposed ability of carotid endarterectomy to prevent perioperative stroke during CAB. Part of the problem is that cerebral ischemia and neurologic dysfunction after cardiac surgery have several possible causes. The relative contribution of these causes to perioperative strokes is not known. Some are due to carotid disease, and many are not. Many studies have found a poor correlation be-

tween the anatomic location of a stroke and the presence of a significant carotid lesion, suggesting the involvement of these latter factors.

We examined the results of combined coronary and carotid operations in a follow-up study of nearly 4000 patients. The stroke rate for patients without carotid disease was about 2%. When the patients were stratified according to the configuration of their carotid disease, either lateral stenosis or bilateral occlusion plus stenosis in our reptrospective data, we could not show any difference when the carotid artery was fixed along with the coronary artery or not. These were retrospective and nonrandomized patients.

The approach we have adopted is to do separate carotid and coronary operations unless the patient has significant on going symptoms from both lesions. In general, whichever system is more symptomatic or has the more dangerous lesion is treated first. If the patient has a neurologic problem after surgery, it is certainly much easier to sort out and treat when the operations are staged.

The present study lends support to the concept of PCE, but it is clear that this was conducted in a highly selected group of patients. When the group with significant CS and PCE is compared with the diseased group without PCE there is a barely significant advantage for PCE. But they had no TIAs, strokes, or deaths in that group, and it is clear that would not hold up with a larger patient experience.

I have several questions for the authors. On what grounds were patients selected for or against the PCE? How many of these patients who had PCE were symptomatic from their carotid lesions before surgery? Can the authors provide any guidelines for us in deciding when to stage the two operations and when to combine them?

Dr. Thomas O'Donnell (Boston, Mass.). This paper presents some data that weren't evident to us before, particularly about the timing of stroke after a CAB. Timing is important, because as the authors point out, it lessens the role of flow-related phenomena.

I presume these were all in asymptomatic patients. Dr. Eisenbud asked if these were symptomatic. By definition, prophylactic implies none of the patients had neurologic symptoms. This is different than the type of patient we usually see. The fact that you are able to operate on a carotid artery not with the CAB but preparatory to it must imply that the cardiac disease is of a lesser degree. What are the cardiac criteria?

You never mentioned the role of OPG, a transcranial Doppler. Does that enter into selection process on the carotid end as far as looking at flow relationship? And in that light, what is the role of contralateral occlusion to a high-grade stenosis? Does that bias you toward a higher rate of surgery?

I would view with caution interpreting that this report proves that carotid endarterectomy is beneficial in patients with asymptomatic stenosis at the time of coronary artery bypass graft.

Dr. Brian L. Thiele (Hershey, Pa.). I think the data the authors present help us a little but still leave us in a quandry. In virtually all the series that attempt to address this issue it is difficult to sort out the differences between isolated carotid artery stenosis and patients with carotid artery disease. I think most of us accept the premise that the more disease you have, the more likely you are to have problems. It is difficult on the basis of this presentation, as Dr. O'Donnell has already alluded to, to define the relative outcome of patients who have bilateral severe, greater than 75% stenosis, people who have contralateral occlusion, and the like.

You note in your presentation that all of the patients who had neurologic events had the neurologic events ipsilateral to the severe stenosis. I presume therefore that the contralateral side in those people was in fact not involved but it is unclear.

Clearly, I think we are not going to get the answer to this complex problem until we can somehow stratify the data to define not only the presence of disease but how extensive the disease is in an individual patient. Was there any attempt to stratify the patients in this series based on disease distribution as well as severity?

Second, did you do follow-up duplex scans on these patients who had neurologic events to ascertain whether the ipsilateral carotid artery was still patent or whether it had occluded?

Dr. Richard M. Green (Rochester, N.Y.). I would like to ask Dr. Faggioli if he could comment on his title, the role of carotid screening before CAB. It seems to me the authors have not tried to make a case for carotid endarterectomy or not but have tried to make a case for getting data before CAB with regard to stroke risk, and I think this is important.

Do you think that all patients over 60 years of age should have a duplex scan before CAB.

Dr. Jack L. Cronewett (Hanover, N.H.). In your presentation did you indicate that previous vascular surgery was also a significant predictor of stroke? If that was the case, is this an independent predictor or does it just mean that patients with previous vascular surgery are more likely to have severe carotid disease?

Dr. Brooke Roberts (Fort Washington, Pa.). In a number of series when strokes have occurred after coronary operations they have occurred 3 or 4 days later, and some of that timing occurs here. I would like to ask the authors why they think this occurs. It certainly does not sound as if it is intraoperative. It is delayed, and there is something that we do not know going on.

Dr. Faggioli. Dr. Eisenbud asked about the method of selection of the patients in our study; because our study was retrospective it obviously was not homogeneous, and since the patients were treated by different surgeons, the criteria varied. We were not able to classify the patients according to strict criteria. You asked also about the symptoms these patients had. There were some patients in this group who underwent PCE, who actually had some remote

(>6 months) symptoms that resolved. They were TIAs, which resolved previously. Obviously no patient with stroke or current neurologic events were operated on.

Dr. O'Donnell, we did not look at the cardiac criteria. Our paper focused on the problems of CS in this group of patients, therefore we did not stratify the patients according to their cardiac status. We did use OPG-Gee. We did not use transcranial Doppler. The criteria for OPG/Gee permitted us to achieve a sensitivity of 99.4%, and the specificity was 100%. For the duplex scan the sensitivity in our group of patients was 96% and the specificity again was 100%. This was assessed according to the angiography, which we were able to do in 26 patients of our series.

Dr. O'Donnell asked about the role of occlusion as an indication for surgery. We had six patients with occlusion and moderately severe stenosis on the contralateral side. Five of the six patients underwent surgery. Only one patient did not have surgery actually, and this patient did not experience any neurologic event after the cardiac procedure.

I think this will also answer the question from Dr. Thiele about the contralateral occlusion. Again, only one patient was not operated on with a contralateral occlusion, and he did not have a neurologic event. We did not do any follow up study with duplex scanning in patients with neurologic symptoms. Again, our study was on the role of carotid screening before so we did not follow up these patients.

Dr. Green, the role of carotid screening in our study, as you mentioned, suggests that all patients over 60 years should undergo carotid evaluation before the cardiac operation.

Dr. Cronewett, the slide I showed on the previous peripheral vascular surgery was not related to the stroke incidence after the cardiac procedure but only to the mortality rate, and this was highly significant. Past peripheral vascular surgery did correlate with an increase in mortality rate after CAB. We looked at these data, and this was an independent cause of death.

Dr. Roberts asked about the mechanisms of stroke, we can obviously just speculate about them. We are planning to do a prospective study on this issue. What we can say is that there are a variety of problems that can arise in these patients like a change in blood flow pressure, change in coagulability, or again some cardiac problem with embolization to the brain that can cause these events.

Dr. John Ricotta. I want to emphasize what Dr. Faggioli said and what Dr. Green mentioned. We are talking about carotid screening here and not the efficacy of PCE. We had only 19 patients and we cannot say anything about PCE. The reasons that we did this study was that when I got to Buffalo, the cardiac surgeons were screening everybody, which meant that I was screening 1200 patients a year. I wanted to know if this was worthwhile, so I decided to review our data, and I was surprised to find out that there was a higher incidence of carotid disease in these patients than I expected.

I was also surprised to find that the strokes did not occur at the time of surgery, and I think this is probably the most interesting finding from my perspective. Why this is, I do not know. It suggests that something is going on in the perioperative period. Whether it is hypercoagulability or platelet activation or hemodynamic instability I do not know. I think that has been the most exciting finding for us.

We try to do these endarterectomies before surgery whenever we can. Whether or not we are right is hard to know, but I would suggest that unless we start to screen these patients we are never going to answer this question. So I would suggest that in the patients over 60 years they probably should be screened so that we can try to study the question further.