Improved Results of Carotid Endarterectomy in Patients with Symptomatic Coronary Disease: An Analysis of 1,546 Consecutive Carotid Operations

COYNESS L. ENNIS, JR., M.D., GERALD M. LAWRIE, M.D., GEORGE C. MORRIS, JR., M.D., E. STANLEY CRAWFORD, M.D., JIMMY F. HOWELL, M.D., MICHAEL J. REARDON, M.D., and STEPHEN C. WEATHERFORD, M.D.

SUMMARY The significant risk of fatal myocardial infarction after carotid endarterectomy in patients with coronary disease long has been recognized. In 1,546 consecutive carotid endarterectomies performed in 1,238 patients over the last 10 years, angina pectoris was present in 17% (212/1,238) of patients; a further 32% (396/1,238) of patients were asymptomatic, but had a history of myocardial infarction. The perioperative mortality (30 day) in the 1,206 consecutive endarterectomies in 1,026 patients without symptomatic coronary artery disease was 1.5% (15/1,026 patients). Of the 212 patients with symptoms, 85 carotid endarterectomies were performed in 77 patients without prior coronary bypass operation with an operative mortality of 18.2% (14/77 patients). The remaining 135 patients had 155 carotid endarterectomies but were treated by either prior coronary artery bypass (84 patients) or simultaneous carotid endarterectomy and coronary artery bypass (51 patients) with an operative mortality of 3% (4/135 patients). The greatly improved survival in those patients with symptomatic coronary disease who had a coronary artery bypass prior to or at the same time as carotid endarterectomy, and the absence of permanent neurological deficit in the 51 of those 135 patients who had simultaneous carotid endarterectomy and coronary artery bypass suggests that significantly improved survival can be achieved after carotid endarterectomy in these high risk patients by the use of simultaneous coronary artery bypass surgery.

ISOLATED CAROTID endarterectomy performed in the presence of severe coronary artery occlusive disease has been reported to be associated with an operative mortality of up to 20% and a delayed mortality rate of 25-50% from myocardial infarction.1,2 In recent years, since the coronary bypass operation has emerged as an effective therapy, we have been interested to determine whether the application of this technique could alter favorably the recognized high mortality from heart disease associated with carotid reconstruction. This study was undertaken to ascertain the appropriate management of the candidate for carotid reconstruction who also has symptomatic coronary artery disease.

Materials and Methods

The fate of 1,238 consecutive patients who underwent 1,546 carotid endarterectomies between 1967 and 1977 was reviewed. There were 776 (63%) males and 462 (37%) females. The ages ranged from 32-92 (mean 64 years) with the greatest incidence in the sixth and seventh decades of life. Staged bilateral operations were performed in 308 patients (25%). Associated risk factors for atherosclerosis were common (table 1). Patients with blood pressures greater than 160 milligrams per 100 cc of plasma or a cholesterol level greater than 260 milligrams per 100 cc of plasma were considered hyperlipidemic. Patients with an elevated serum triglyceride level greater than 160 milligrams per 100 cc of plasma or cholesterol level greater than 260 milligrams per 100 cc of plasma were considered hyperlipidemic. Patients with drug treatment for diabetes mellitus. Patients with drug treatment for diabetes mellitus.

In order to determine the value of coronary artery bypass in these patients, the overall series was divided into three groups (table 2). Group I consisted of 1,026 patients who had no symptoms of coronary artery disease (i.e. angina pectoris, congestive heart failure or serious ventricular arrhythmias) at the time of carotid reconstruction. Group II was composed of 77 patients who had symptomatic coronary artery disease at the time of carotid reconstruction, but did not undergo coronary artery bypass at that time. In group III, there were 135 patients who had symptomatic coronary artery disease, and who had undergone coronary artery bypass surgery prior to (84 patients) or at the time of (51 patients) their carotid reconstruction. The clinical syndromes of cerebrovascular insufficiency were similar in each group, except that carotid reconstruction for an asymptomatic bruit was more common in those patients who were also candidates for myocardial revascularization (table 3). There were 330 patients with asymptomatic bruits only. Cerebrovascular symptoms consisted of transient ischemic attacks in 678 patients (55%), evolving strokes in 51 patients (4%), and 179 patients (14%) presented with evidence of a prior completed stroke.

Cardiac symptoms consisted of stable angina in 157 patients (12.7%) and unstable angina in 55 patients (4.4%). In the overall group of 1,238 patients, a previous myocardial infarction had occurred in 293 patients (23.7%). Of these 293 patients, 184 had no cardiac symptoms (i.e. angina pectoris, congestive
RESULTS, 1,546 CAROTID OPERATIONS/Ennix et al.

TABLE 1  Risk Factors (1,238 Patients)

<table>
<thead>
<tr>
<th>Risk Factor</th>
<th>Percentage (Number)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypertension</td>
<td>56.0% (693)</td>
</tr>
<tr>
<td>Smoking</td>
<td>41.8% (517)</td>
</tr>
<tr>
<td>Obesity</td>
<td>20.3% (251)</td>
</tr>
<tr>
<td>Diabetes</td>
<td>13.6% (168)</td>
</tr>
<tr>
<td>Hyperlipidemia</td>
<td>12.6% (156)</td>
</tr>
</tbody>
</table>

TABLE 2  Classification According to Symptoms of Coronary Disease at Time of Carotid Surgery

<table>
<thead>
<tr>
<th>Group</th>
<th>Classification</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group I (1026 patients)</td>
<td>No symptoms of coronary disease</td>
</tr>
<tr>
<td>Group II (77 patients)</td>
<td>Symptomatic coronary disease</td>
</tr>
<tr>
<td>Group III (135 patients)</td>
<td>Symptomatic coronary disease treated by prior or simultaneous coronary bypass</td>
</tr>
</tbody>
</table>

heart failure or serious ventricular arrhythmias) and therefore were placed in Group I.

Carotid angiography was performed under general anesthesia with bilateral percutaneous injections of 10 ml of 50% Hypaque. When indicated, vertebral arteriograms were also obtained. Recently, some studies have been performed by a retrograde transfemoral approach. A lesion was considered significant when the diameter of the carotid lumen was reduced by 50% or it was ulcerated. Significant bilateral disease which required staged bilateral operations was present in 308 patients (25%) (table 4). Cerebrovascular symptoms referable to the second side consisted of transient ischemic attacks in 191 patients, evolving strokes in 5 patients, a completed stroke in 45 patients and asymptomatic bruits in 67 patients.

Coronary angiograms were performed in 60 patients in Group II and all 135 patients in Group III. The angiographic findings are summarized in table 4.

Results

In the overall group of 1,546 carotid procedures (table 5) the major stroke rate was 1.1% (17/1,546), a major stroke being defined as a new neurological deficit manifested as a permanent paralysis or dysphasia. In another 1.3% (10/1,546) of operations, minor strokes occurred, a minor stroke being defined as a new neurological deficit manifested as a permanent mild weakness or clumsiness of a limb. The overall myocardial infarction rate was 1.6% (25/1,546) and patient mortality 2.7% (33/1,238).

In the 1,026 patients who had no symptoms of coronary artery disease at the time of their carotid reconstruction (Group I), the perioperative mortality was 1.5% (15/1,026). The perioperative myocardial infarction rate was 1.0% (10/1,026). It was interesting to note that the 184 patients with a previous history of myocardial infarction, but no current symptoms, had an outcome similar to the remaining patients in Group I who had no history of myocardial infarction. The incidence of major stroke after opera-
noscence was 1.0% (1/102). However, none of the patients in this group had a history of MI.

In Group II, 8 of the 102 patients (8.0%) developed MI following surgery. Of these, 7 were related to the carotid reconstruction. There was 1 death (0.1%) related to a perioperative myocardial infarction. The other 7 MI's were nonfatal.

In Group III, 8 of the 102 patients (7.9%) developed MI following surgery. Of these, 6 were related to the carotid reconstruction. One patient died of cardiac arrest following surgery. The other 5 MI's were nonfatal.

Of the 15 patients comprising Group III, 51 had simultaneous carotid endarterectomy and coronary artery bypass surgery. The perioperative mortality was 5.9% (3/51). This relatively low mortality was achieved despite the fact that these patients were 10 years older and at higher risk than our overall patient population undergoing coronary artery bypass surgery alone. Of the 15 deaths, 7 were related to perioperative myocardial infarction. A fatal pulmonary embolus occurred in another patient while renal failure occurred in a third patient. There were 2 (3.9%) nonfatal myocardial infarctions. There was one TIA (2.0%), but no other serious neurological complications among these patients who underwent simultaneous operations.

The causes of perioperative mortality in each group were divided into 3 categories: cardiac, cerebral, and others (Table 6). In Group I, 66.6% (10/15) of deaths were related to cerebral causes while only 13.3% (2/15) of the deaths were cardiac in origin. Two other patients died of other causes. In Group II, 21.4% (3/14) of deaths were cardiac in origin, while 78.6% (11/14) of deaths were cerebral in origin. Two other patients died of other causes. In Group III, 78.6% (11/14) of deaths were cardiac in origin, while 21.4% (3/14) of patients died of cerebral related factors. In the Group III patients (who had myocardial revascularization prior to or at the time of their carotid reconstruction) there were only 4 deaths. One death was related to a perioperative myocardial infarction in a 62-year-old female who had unstable angina. A second death resulted from a massive postoperative stroke despite carotid reconstruction of the appropriate side. Another patient died of renal failure complications in the postoperative period. The fourth patient died on the tenth postoperative day from a massive pulmonary embolus.

In summary, patients who had symptomatic coronary artery disease at the time of carotid reconstruction, the mortality decreased to 3% (4/135) compared to the 18.2% of Group II. Only 4 patients (2.6%) in Group III had postoperative myocardial infarction. There were 2 patients who had postoperative minor strokes. No patient in this group suffered a major postoperative stroke.

Although the good ventricle/poor ventricle distribution in Groups II and III would tend to favor Group III, the incidence of left main coronary artery stenosis and triple vessel disease (severe forms of coronary disease) was higher in Group III than Group II (Table 4). Of the 135 patients comprising Group III, 51 had simultaneous carotid endarterectomy and coronary artery bypass surgery. The perioperative mortality was 5.9% (3/51). This relatively low mortality was achieved despite the fact that these patients were 10 years older and at higher risk than our overall patient population undergoing coronary artery bypass surgery alone. Of the 15 deaths, 7 were related to perioperative myocardial infarction. A fatal pulmonary embolus occurred in another patient while renal failure occurred in a third patient. There were 2 (3.9%) nonfatal myocardial infarctions. There was one TIA (2.0%), but no other serious neurological complications among these patients who underwent simultaneous operations.

The causes of perioperative mortality in each group were divided into 3 categories: cardiac, cerebral, and others (Table 6). In Group I, 66.6% (10/15) of deaths were related to cerebral causes while only 13.3% (2/15) of the deaths were cardiac in origin. Two other patients died of other causes. In Group II, 21.4% (3/14) of deaths were cardiac in origin, while 78.6% (11/14) of deaths were cerebral in origin. Two other patients died of other causes. In Group III, 78.6% (11/14) of deaths were cardiac in origin, while 21.4% (3/14) of patients died of cerebral related factors. In the Group III patients (who had myocardial revascularization prior to or at the time of their carotid reconstruction) there were only 4 deaths. One death was related to a perioperative myocardial infarction in a 62-year-old female who had unstable angina. A second death resulted from a massive postoperative stroke despite carotid reconstruction of the appropriate side. Another patient died of renal failure complications in the postoperative period. The fourth patient died on the tenth postoperative day from a massive pulmonary embolus.

In summary, patients who had symptomatic coronary artery disease at the time of carotid reconstruction, the mortality decreased to 3% (4/135) compared to the 18.2% of Group II. Only 4 patients (2.6%) in Group III had postoperative myocardial infarction. There were 2 patients who had postoperative minor strokes. No patient in this group suffered a major postoperative stroke.

Although the good ventricle/poor ventricle distribution in Groups II and III would tend to favor Group III, the incidence of left main coronary artery stenosis and triple vessel disease (severe forms of coronary disease) was higher in Group III than Group II (Table 4).
was due to cardiac related causes. On the other hand, in the group of patients in which symptomatic coronary artery disease was present and surgically treated (Group III), the mortality decreased to 3% despite the inherent high risk nature of this group and was related equally to stroke, myocardial infarction, and general complications of operation.

Discussion

Perioperative myocardial infarction long has been recognized as a leading cause of early and late death following carotid reconstruction for cerebrovascular insufficiency. The natural histories of carotid and coronary artery occlusive disease are certainly intertwined. Operative mortality in patients having surgery for cerebrovascular insufficiency has been shown to be directly related to the incidence of coronary disease. Cerebral complications have been reported to occur in 2–12% of patients after coronary artery bypass. These patients can accordingly be considered to be at high risk for neurological deficits at the time of cardiopulmonary bypass and in the postoperative period. It is clear, therefore, that the isolated presentation of atherosclerotic disease in the coronary or cerebrovascular system demands a systematic evaluation of both.

Simultaneous carotid endarterectomy and coronary artery bypass has been recommended as a routine procedure because of the inherent danger of performing these procedures separately. Because of the low mortality and the significant incidence of myocardial infarction following carotid artery surgery in patients with symptomatic coronary artery disease, we believe consideration should be given to a concomitant coronary bypass procedure in all cases. In our experience, combining these operative techniques has not increased mortality significantly. To stage these procedures, operating on the more severely diseased system first, is associated with the distinct chance of converting a stable clinical state into an unstable one, resulting in cerebral or myocardial infarction.

Because of the greatly improved survival in our patients with carotid artery disease and cardiac symptoms who had coronary artery bypass at the same time or prior to carotid endarterectomy, we believe that the candidate for carotid reconstruction who also has symptoms of coronary artery disease is best protected by simultaneous corrective operations as this seems to be an effective means of consistently avoiding cerebral injury and myocardial damage in these very high risk patients.

References

Improved results of carotid endarterectomy in patients with symptomatic coronary disease: an analysis of 1,546 consecutive carotid operations.
C L Ennix, Jr, G M Lawrie, G C Morris, Jr, E S Crawford, J F Howell, M J Reardon and S C Weatherford

Stroke. 1979;10:122-125
doi: 10.1161/01.STR.10.2.122

Stroke is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1979 American Heart Association, Inc. All rights reserved.
Print ISSN: 0039-2499. Online ISSN: 1524-4628

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://stroke.ahajournals.org/content/10/2/122

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Stroke can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Stroke is online at:
http://stroke.ahajournals.org//subscriptions/