CAROTID ENDARTERECTOMY effectively eliminates a source of emboli or improves blood flow in atherosclerotic stenoses of the internal carotid artery.1-4 Thromboendarterectomy for total occlusion of the internal carotid artery, on the other hand, remains a controversial subject.

Although the earlier literature was less encouraging, more recent studies have shown that patency of the internal carotid artery can frequently be restored by thromboendarterectomy. Most authors report a success rate of approximately 40%, 1, 2, 4, 7-10. Restoration of patency has been reported in as few as 20%, 5 and as many as 64% of operated patients. The earlier the operation is performed after total occlusion is believed to have occurred, the higher is the rate of postoperative patency. 1, 5, 7-10. For example, 100% of occluded internal carotid arteries have been successfully reopened within 6 hours of presumed occlusion, 90% within 48 hours, 1 and 70% within 72 hours. 8

Postoperative mortality 1-5, 7, 8, 10 and morbidity 2, 7 have been low. It is well recognized that mortality resulting from carotid endarterectomy is much higher in patients with recent cerebral infarction, who are neurologically unstable. 2, 8, 12 This has been true for patients with complete occlusion, as well as stenosis, with virtually all operative deaths occurring in drowsy patients with recent cerebral infarction. 8 In series where case selection has excluded such ill patients, the mortality has been low, 19 and in some series there has been no postoperative mortality. 5, 7

The results show that a group of patients can be selected that will have low postoperative mortality and morbidity. The success rate for restoration of blood flow is high, particularly if the operation is performed soon after occlusion. The long-term prognosis in patients in whom patency of the internal carotid artery is restored and maintained appears to be better than in those with persistent occlusion of the carotid artery.

Long-term follow up of patients operated on successfully for total internal carotid artery occlusion indicates that the incidence of recurrent neurological symptoms is low. 1-5, 8, 10 This data must be evaluated by comparison with the natural history of internal carotid artery occlusion in untreated survivors. Immediate mortality 16-18 and morbidity 14 from cerebral infarction following internal carotid artery occlusion are high. Survivors, however, have been shown, by some authors 10 to have relatively low late mortality and morbidity rates from cerebrovascular causes, even when compared with a matched group of patients with carotid stenosis. 14 Patients who reocclude the internal carotid artery after successful thromboendarterectomy have also been shown to have a low rate (4%) of recurrent neurological symptoms. 1 There is dissent regarding the natural history, however, and other authors 14 have found that total occlusion is less benign, with 27% of patients dying from repeated cerebral infarction.

A further problem relating to assessment of the efficacy of thromboendarterectomy for total occlusion of the internal carotid artery is the likelihood of reocclusion after successful operation. Most authors (see above) have not shown results of late follow up angiography. It has been shown that almost 50% of reopened vessels reocclude. 1

With the above problems in mind, we re-examined the results of thromboendarterectomy for total occlusion of the internal carotid artery in our own institution. Applying more recently evolved criteria for operative risk, we wished to determine if better case selection would result in decreased operative mortality and morbidity. We wished to see whether the long-term success rate of maintaining patency,
as proven by late angiography, was acceptable and whether or not patients with maintained patency fared better than those who failed to have patency re-established or maintained.

**Methods**

Forty consecutive patients with angiographically demonstrated total occlusion of at least one internal carotid artery were treated by thromboendarterectomy on the neurosurgical service at Sunnybrook Medical Centre between January 1968 and July 1976. All patients operated on for occlusion during this time period are included in the present series. (All operations were performed by CHT, DWR, or WML).

There were 30 males and 10 females in the group, ranging in age from 42 to 76 years (mean age 58 years). Five patients had bilateral internal carotid artery occlusion. Three of these had unilateral operations and 2 had bilateral operations. Nine patients had contralateral stenosing atherosclerotic plaques and 7 of these had contralateral endarterectomy. In total, 42 thromboendarterectomies were attempted for complete internal carotid artery occlusion.

The presenting clinical symptoms are summarized in table 1. Sixteen patients presented with major cerebral infarction with established neurological deficit which affected activities of daily living. Five patients presented with a minor cerebral infarction producing mild hemiparesis with or without sensory deficit that had been present for more than 24 hours but did not interfere significantly with activities of daily living. Thirteen patients presented with transient ischemic attacks (TIAs) and 3 patients presented with progressing cerebral infarctions. Three patients presented with intellectual impairment or features of brainstem ischemia.

Virtually all of these patients were anticoagulated preoperatively, and anticoagulant therapy was continued postoperatively for 6 weeks to 6 months in patients with successful restoration of flow. Arteriotomy was carried out over a distance of approximately 3 cm, beginning on the distal common carotid artery and extending onto the proximal internal carotid artery. If the proximal internal carotid artery was patent, but backflow was not obtained, a Fogarty balloon catheter was passed distally to the level of the base of the skull or beyond and an attempt was made to extract all distal thrombus. If brisk backflow could not be obtained, the internal carotid artery was ligated and the procedure was abandoned.

Preoperative assessment of risk was carried out retrospectively using a modification of the classification proposed by Sundt, et al. Outpatients were divided into two groups—designated I and II on the basis of neurological, medical and angiographic risk factors. Group I consisted of 14 patients who had none of the neurological, medical or angiographic risk factors described below. All patients with one or more risk factors were placed in Group II (26 patients).

The risk factors were as follows:

**Neurological risk factors.** Major neurological deficit of less than 2 weeks duration, progressing neurological deficit, minor neurological deficit of less than 24 hours duration, deficit secondary to multiple cerebral infarctions or frequent daily TIAs.

**Medical risk factors.** Past history of angina pectoris or myocardial infarction, severe hypertension (B.P. greater than 180/110 mm Hg), congestive heart failure, chronic obstructive pulmonary disease (COPD), chronological age greater than 70 years or severe obesity.

**Angiographic risk factors.** Extensive atherosclerotic lesions in the aortic arch and its branches, the contralateral carotid system, the verteobasilar system or the intracranial arteries. Survivors were followed until the time of this writing. The shortest follow up period was 12 months, and the longest was 99 months. Results were tested for statistical significance using Fisher's exact test for a 2 X 2 contingency table.

**Results**

**Restoration of Flow: Immediate and Long-term Success Rate**

Blood flow was successfully restored in 22 procedures (52%) (in 21 patients). Twelve of these 21 patients had repeat carotid angiography 3 to 6 months postoperatively. The internal carotid artery was patent in only 7 (58%) of these. One patient had an aneurysmal dilation at the operative site but has remained asymptomatic for a further 6 years.

In 6 patients, endarterectomy without thrombectomy was performed because there was no thrombus in the internal carotid artery distally, but only a short segmental occlusion near the carotid bifurcation. The angiograms of all 6 of these patients were critically reviewed and all of them showed total internal carotid artery occlusion. In the experience of 1 of the authors (W.M.L.) the major presenting symptom in such patients has been TIAs. This was true of 2 of the patients in the present series.

Seven procedures were carried out within 72 hours of the presumed time of occlusion (onset of most recent neurological symptoms) and blood flow was restored in 6 of these, (86%). Thirty-five procedures (in 33 patients) were carried out more than 72 hours after the presumed time of occlusion and blood flow restoration was successful in 16 of these procedures (46%). The immediate success rate for restoration of blood flow was 44% in Group I patients and 58% in Group II patients.

**Postoperative Mortality and Morbidity**

Postoperative cerebral and cardiac complications occurring during the hospital admission in which surgery was performed are summarized in table 2.

**Mortality.** None of the patients in Group I died as a result of either myocardial infarction or cerebral complications. In Group II, on the other hand, 2 patients died from myocardial infarction, and 4 patients died as a result of either intracerebral hemorrhage or massive cerebral edema secondary to cerebral infarction.
TABLE 2 Postoperative Mortality and Morbidity, During the Initial Hospital Admission, for Total Occlusion of the Internal Carotid Artery

<table>
<thead>
<tr>
<th>Group</th>
<th>Mortality</th>
<th>Morbidity</th>
<th>Cardiac complication</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Cerebral hemorrhage or infarction</td>
<td>Myocardial infarction</td>
<td>Major</td>
</tr>
<tr>
<td>Group I</td>
<td>0</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>(14 patients)</td>
<td>(15 procedures)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Group II</td>
<td>4</td>
<td>2</td>
<td>5</td>
</tr>
<tr>
<td>(26 patients)</td>
<td>(27 procedures)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total mortality</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Group I</td>
<td>22%</td>
<td></td>
<td>0</td>
</tr>
<tr>
<td>Group II</td>
<td></td>
<td></td>
<td>22%</td>
</tr>
</tbody>
</table>

*(p < 0.05)*

Mortality and morbidity in survivors of thromboendarterectomy occurring after hospital discharge are summarized in tables 3 and 4. Based on preoperative assessment of risk. Fourteen patients remained available for follow up in Group I and 20 patients remained available in Group II (table 3). The overall late mortality in Group I was 21%, as compared with 20% in Group II. This long-term mortality was largely from causes other than cerebral infarction. If one compares the late morbidity from cerebral causes, there was 7% in Group I and 25% in Group II.

Based on patency or nonpatency of the internal carotid artery. Mortality and morbidity in the immediate postoperative period and on follow up, as a function of patency or nonpatency of the internal carotid artery, are summarized in table 4.

There was little postoperative mortality in either group (table 4). Postoperative cardiac morbidity was similar in the two groups. Patients with persistent patency had a 43% incidence of postoperative cerebral morbidity, compared with 17% in patients in whom thromboendarterectomy was unsuccessful. This reflects the inclusion of neurologically unstable patients in the present series. With case selection on the basis of the classification proposed above, these patients would be excluded as candidates for thromboendarterectomy.

There was no long-term mortality or morbidity in patients with persistent patency (table 4). In contrast, patients with persistent occlusion suffered a 17% long-term incidence of mortality and a 25% incidence of long-term morbidity. The long-term incidence of internal carotid artery territory cerebral morbidity in the latter group was 21%. Admittedly, the total number of cases in this group is small.

**Discussion**

The 52% overall success rate for restoration of patency of the totally occluded internal carotid artery in this series is comparable to that achieved in other series.1 9 8 10 Similarly, our success rate of 86% in procedures carried out within 72 hours of the presumed time of occlusion, compares favorably with the experience of other surgeons.1 9 8 10 The 6 cases which showed an angiographic picture of complete obstruction, and were found to have localized occlusion without distal thrombus at operation, are of interest. Unfortunately, only 2 of these patients had late postoperative angiography, but in both, the arteries were patent. Although no firm conclusion can be drawn, such cases may
have a better prognosis for maintenance of patency. It may be possible, as suggested previously, to select these cases preoperatively.

Our experience confirms that many reopened internal carotid arteries reocclude, and we would therefore agree that if one is to attempt to draw any conclusion regarding the benefit of thromboendarterectomy for total internal carotid occlusion, late postoperative angiography is mandatory. One might question the therapeutic implications of performing late angiography in this group of patients.

Use of the Fogarty catheter when spontaneous backflow is not obtained is controversial. Distal embolization and carotid-cavernous sinus fistula are potential complications. Brisk backflow tends to wash out residual distal thrombus, preventing embolization, and if brisk backflow is not obtained the artery is ligated. We have not experienced carotid-cavernous sinus fistula as a complication of thromboendarterectomy. The higher patency rate achieved with the Fogarty catheter, in our view, commends its use.

The overall postoperative mortality and morbidity (table 2) were much higher in Group II than in Group I. Certainly the presence of a few neurologically unstable patients with recent cerebral infarction in Group II, would have been expected to raise the postoperative mortality. The application of more stringent criteria for assessment of operative risk, however, in the two groups in the present series, indicates that one can select those patients with internal carotid occlusion who are likely to suffer higher mortality and morbidity as a result of operation. The three-fold difference in morbidity from cerebral infarction, between the two groups is also important.

The fact that all mortality and morbidity in the intra- and postoperative periods resulted from either cerebral or cardiac complications is not surprising. If one compares the late morbidity and mortality as a function of the preoperative assessment of risk (table 3) it is surprising that the patients in Group II did not fare worse, in terms of mortality, than the patients in Group I. The former was comprised of patients with greater neurological deficits, more severe extracranial arterial occlusive disease, more widespread atherosclerosis, and more severe systemic diseases. Accordingly, Group II patients would have been expected to show a higher late mortality than Group I patients. The fact that they did not, suggests that Group II patients who survive operation may be expected to show a long-term survival comparable to Group I patients. The fact that the long-term mortality was actually lower in Group II is probably explained by the fact that more Group II patients, presumably those who were initially more ill, died during the initial hospitalization. If one examines the overall mortality in the 2 groups, beginning at the time of operation, Group II experienced a 32% mortality compared with 20% in Group I. It should be noted that the cause of death in many of these cases was not cerebral.

The late morbidity, both from cerebral causes and from other causes, was lower in Group I, as would have been expected.

The results of thromboendarterectomy as a function of whether or not the internal carotid artery remained patent three to six months after operation are displayed in table 4. Postoperative cerebral morbidity was actually higher in the patients in whom the internal carotid artery ultimately remained patent. On the other hand, the patients with persistent patency showed no long-term mortality or morbidity. The 17% incidence of late mortality and the 25% incidence of late morbidity in the 24 patients with known persistent internal carotid artery occlusion suggests that this is not a benign condition.

### Table 3: Long-term Morbidity and Mortality in Survivors of Thromboendarterectomy for Total Occlusion of the Internal Carotid Artery, Occurring After Initial Hospital Discharge

<table>
<thead>
<tr>
<th></th>
<th>Died prior to hospital discharge</th>
<th>Survivors available for follow-up</th>
<th>Long-term morbidity</th>
<th>Long-term mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Cerebral infarction</td>
<td>TIA's</td>
<td>Angina pectoris</td>
<td>Intellectual impairments</td>
</tr>
<tr>
<td>Group I (14 patients)</td>
<td>0</td>
<td>14</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Group II (20 patients)</td>
<td>6</td>
<td>20</td>
<td>1</td>
<td>2</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>Died prior to discharge</th>
<th>Survivors available for follow-up</th>
<th>Long-term morbidity</th>
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</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Cerebral infarction</td>
<td>TIA's</td>
<td>Angina pectoris</td>
<td>Intellectual impairments</td>
</tr>
<tr>
<td>Group I (14 patients)</td>
<td>0</td>
<td>14</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Group II (20 patients)</td>
<td>6</td>
<td>20</td>
<td>1</td>
<td>2</td>
</tr>
</tbody>
</table>

- N = 31 because some patients in whom patency of the internal carotid artery was restored did not have late postoperative angiography.
- Comparing total long-term mortality and morbidity in the two above groups p < 0.05.

### Table 4: Postoperative and Long-term Mortality and Morbidity as a Function of Persistent Patency or Persistent Occlusion of the Internal Carotid Artery

<table>
<thead>
<tr>
<th></th>
<th>Mortality</th>
<th>Morbidity</th>
<th>Mortality</th>
<th>Morbidity</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Cerebral</td>
<td>Cardiac</td>
<td>Cerebral</td>
<td>Cardiac</td>
</tr>
<tr>
<td>Persistent Oclusion (24 cases)</td>
<td>0</td>
<td>1</td>
<td>4</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>(Infarction)</td>
<td>(Major Infarctions)</td>
<td>(Infarction)</td>
<td>(2 minor)</td>
</tr>
<tr>
<td>Persistent Patency (7 cases)</td>
<td>0</td>
<td>0</td>
<td>3</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>(2 Major Infarctions, 1 Minor Infarction)</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

- N = 31 because some patients in whom patency of the internal carotid artery was restored did not have late postoperative angiography.
- Comparing total long-term mortality and morbidity in the two above groups p < 0.05.
The present study indicates that by applying the method of preoperative risk assessment outlined above, a group of low risk patients with total internal carotid artery occlusion can be selected for operation. In many of these patients, patency of the internal carotid artery can be restored and maintained, particularly if thromboendarterectomy is carried out soon after occlusion occurs. Patients in whom patency is restored and maintained appear to have a better long-term prognosis, with less mortality and morbidity than patients whose internal carotid artery remain occluded.

The low postoperative mortality and morbidity in the low risk group, the patency rate, and the improved long-term prognosis in patients with patent arteries support a policy of early operation in Group I patients. Most Group II patients, because of the high postoperative mortality and morbidity, should probably not be subjected to thromboendarterectomy. We would emphasize, however, that Group II is not homogeneous. Some patients are undoubtedly at higher risk than others. The present study, therefore, cannot be taken as sufficient support for a policy of universal rejection of Group II patients as candidates for thromboendarterectomy.

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