Occlusion of the Carotid Artery: Prognosis (Natural History) and the Possibilities of Surgical Revascularization

BY PETER GRILLO, M.D., AND RUSSEL H. PATTERSON, JR., M.D.

Abstract:
A retrospective study was made of a group of 44 patients with arteriosclerotic occlusion of the internal carotid artery trying to identify a group that might benefit from extracranial-intracranial arterial bypass. Eight of the 44 patients died of cerebral infarction. Seven of these deaths were due to the cerebral infarction that prompted the arteriogram and the only fatal infarct during the period of follow-up occurred in the opposite cerebral hemisphere. Four nonfatal strokes occurred during the follow-up period, and these also were in the cerebral hemisphere contralateral to the carotid occlusion. We conclude that the role of extracranial-intracranial arterial bypass is limited to the occasional patient who has recurrent symptoms due to vascular insufficiency in the cerebral hemisphere distal to an occluded carotid or middle cerebral artery. The main role of surgery appears to lie in the opening of the occluded carotid artery in selected patients with acute stroke and in the prophylactic repair of contralateral carotid stenosis.

Introduction
Carotid endarterectomy is an effective operation in which ulcerating or obstructing arteriosclerotic plaques near the carotid bifurcation are removed in order to eliminate a source of cerebral emboli or to improve cerebral blood flow.1-4 However, angiography may disclose that the internal carotid artery of a symptomatic patient is occluded, in which case the chances of restoring flow by means of an operation are slim unless the occlusion is quite recent.5 We wondered if the prognosis in these patients was grave enough to justify some surgical measure to improve cerebral blood flow such as an extracranial-intracranial arterial anastomosis. Hoping to answer this question, we undertook a retrospective study of 44 patients seen at The New York Hospital with occlusion by the arteriosclerotic process of one or both internal carotid arteries.

Methods
The angiographical findings were reviewed in all patients submitted to arch aortography or cerebral angiography at The New York Hospital-Cornell Medical Center between 1967 and July 1972. We included in the present series all patients having occlusions of one or both internal carotid arteries except those in whom the occlusion was secondary to causes other than arteriosclerosis such as trauma, collagen vascular disease, and surgical ligation. Patients were also excluded if the occluded artery was opened surgically. In this retrospective analysis we paid particular attention to the longevity of patients after the diagnosis of internal carotid artery occlusion; the state of health if the patient survived; the cause of death if the patient did not survive; and the frequency and pathogenesis of any further episodes of cerebral ischemia. During the period of follow-up the patients were managed in a variety of ways. Some underwent endarterectomy of contralateral carotid stenosis, others were anticoagulated, and still others received no specific treatment. Of the 48 patients selected for the study, complete follow-up was obtained in 44, and this is the group on which our report is based. The group was composed of 31 men and 13 women ranging in age from 45 to 84 years. The mean age was 64 and the mean time of follow-up of surviving patients was three years. In 23 cases the right internal carotid artery was occluded and in 19, the left. Both arteries were occluded in two cases.

Results

SYMPTOMS
Fifteen patients had transient ischemic attacks (TIA), including three patients with amaurosis fugax. Twenty-three patients had completed strokes, three patients had strokes-in-evolution, and three had no complaints suggesting cerebrovascular insufficiency (table 1).

CLINICAL COURSE
Seventeen of the patients have died since the time of
diagnosis by angiography (fig. 1). Seven deaths were due directly to the cerebral infarct that prompted the angiogram, one was due to recurrent cerebral infarction, seven were secondary to arteriosclerotic heart disease, one was caused by renal failure, and one patient died of pneumonia having been debilitated by the cerebral infarct. All but one of the deaths due to cerebral infarction occurred within two weeks of the diagnosis of carotid occlusion. The seven who succumbed to myocardial infarction did so between six months and four years after the diagnosis of carotid occlusion was made (fig. 1). The cause of death seemed unrelated to the age of the patient, being an average of 67 years in both the group that died of cerebral infarction and the group that died of heart-related events.

**TABLE 1**

| Presenting Symptoms in 44 Patients With an Arteriosclerotic Occlusion of a Carotid Artery |
|---|---|
| Symptom                  | %  |
| TIA                      | 34 |
| Completed stroke         | 52 |
| Stroke-in-evolution       | 7  |
| Nonspecific complaints    | 7  |

**Transitory Ischemic Attack**

**LATERALIZATION**

The TIA which were occurring at the time the carotid occlusion was diagnosed involved the cerebral hemisphere distal to the occluded carotid artery in seven cases and the hemisphere distal to the patent carotid artery in three cases. Two patients had symptoms referable to the vertebrobasilar system, and in three others the symptoms could not be localized accurately. Both internal carotid arteries were occluded in one of these patients.

**CLINICAL COURSE**

Ten of the 15 patients who suffered from TIA at the time carotid occlusion was diagnosed are alive and asymptomatic. Four patients sustained a cerebral infarction during the period of follow-up that resulted in a permanent neurological deficit in three of them and was fatal in the fourth. Each of these four cerebral infarctions occurred in the cerebral hemisphere opposite the occluded carotid artery. Two patients died of myocardial infarction, including one of the four patients who also had a stroke.

Treatment of these patients was as follows. Of the ten patients who remained asymptomatic, three underwent endarterectomy of the opposite carotid artery, one was anticoagulated, and two received no specific treatment (table 2).
TABLE 2
Follow-Up Over a Mean of Three Years in 15 Patients With TIA Associated With an Arteriosclerotic Occlusion of a Carotid Artery

<table>
<thead>
<tr>
<th></th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Asymptomatic</td>
<td>67</td>
</tr>
<tr>
<td>Cardiac-related death</td>
<td>13</td>
</tr>
<tr>
<td>Subsequent completed stroke in other hemisphere</td>
<td>27</td>
</tr>
<tr>
<td>Subsequent TIA</td>
<td>0</td>
</tr>
</tbody>
</table>

Completed Stroke

LATERALIZATION

Fifteen of the 23 patients presented with neurological signs related to the cerebral hemisphere supplied by the occluded carotid artery. Five had symptoms related to the opposite side, and in four of them a carotid endarterectomy was performed on an associated stenotic carotid artery. In three cases it was difficult to associate neurological signs with one hemisphere. In one of these cases both carotid arteries were occluded.

CLINICAL COURSE

Twenty-three patients had a completed stroke before angiography. Five of them died from the effects of the cerebral infarction within two weeks of the onset of symptoms (table 3). Five patients survived the presenting stroke but subsequently died because of myocardial infarction. Another patient survived the stroke but had pronounced neurological deficit predisposing him to a fatal case of pneumonia one year later. Only 12 of the 23 patients are living. Nine of them have a mild to moderate neurological deficit and have had no new symptoms. Another patient had a single episode of amaurosis fugax. Two patients had recurrent strokes, and in both the stroke occurred in the cerebral hemisphere opposite the occluded carotid artery.

Eleven of the 15 patients who survived the first stroke underwent carotid endarterectomy because of disease of the opposite carotid artery, and the other four received no specific treatment (table 3).

TABLE 3
Follow-Up Over a Mean of Three Years in 23 Patients With Completed Stroke and Arteriosclerotic Occlusion of a Carotid Artery

<table>
<thead>
<tr>
<th></th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Asymptomatic</td>
<td>52</td>
</tr>
<tr>
<td>Died of presenting stroke</td>
<td>22</td>
</tr>
<tr>
<td>Cardiac-related death</td>
<td>22</td>
</tr>
<tr>
<td>Died of pneumonia</td>
<td>4</td>
</tr>
<tr>
<td>Subsequent completed stroke in contralateral hemisphere</td>
<td>9</td>
</tr>
</tbody>
</table>

Stroke-in-Evolution

CLINICAL COURSE

Three patients had strokes-in-evolution at the time of angiography. Two had progression of neurological signs and died soon thereafter. One patient improved and was discharged from the hospital but had severe generalized arteriosclerosis, including an abdominal aortic aneurysm, that accounted for his death in renal failure 11 months later. All patients with strokes-in-evolution had symptoms related to the cerebral hemisphere supplied by the occluded internal carotid artery.

Nonspecific Symptoms

Three patients had nonspecific symptoms such as headache and blurred vision. These symptoms cleared, and the patients are all living and well.

Discussion

ANALYSIS OF DEATHS

Most of the deaths in this series fall into two categories, neurological or cardiac. Of the neurological deaths, seven out of eight were due to the effects of the presenting stroke, the patients being devastated and dying within two weeks. This group of patients deteriorated so rapidly that the opportunity for surgical intervention was brief, if it was present at all. On the other hand, if a patient survived occlusion of a carotid artery the chances of another neurological insult in the same cerebral hemisphere were slight compared to the risk of dying of cardiac disease. Our results are compatible with those obtained by Dyken et al. They compared a group of patients with occlusion of at least one internal or common carotid artery with another group with less than 60% occlusion of any single artery and found that the outlook in the two groups were similar provided the patient survived the effects of the carotid occlusion.

McDowell et al. reported a series of 57 patients with a higher incidence of postocclusion transient ischemic attacks (13 of 43 survivors) but a relatively low incidence of subsequent stroke (7%). They also noted a high incidence of cardiac disease among survivors. Hardy et al. collected a series of 153 patients that differs from ours in the relatively high incidence of death from subsequent stroke (27%) but does not indicate that heart disease posed a serious threat to the survivors.

PATHOPHYSIOLOGY

Establishing with certainty when the carotid artery became occluded is difficult. The possibility existed in some of our patients that emboli from a stenotic contralateral carotid artery may have accounted for symptoms. However, the observation that most of the completed strokes were referable to the hemisphere supplied by the occluded internal carotid artery suggests cause and effect. The fact that among our
patients further strokes did not occur in the hemisphere ipsilateral to the occluded carotid artery also lends support to the hypothesis that if a cerebral infarction and ipsilateral carotid occlusion coexist, the occlusion is fresh. Our data cannot settle this matter entirely since 14 of our patients were subjected to end-arterectomy of a contralateral stenosis and others received anticoagulant drugs, which might also account for the protected and favored status of the cerebral hemisphere distal to the carotid occlusion.

The causes of TIA in our group of patients are speculative. In most instances the TIAs were referable to the ipsilateral hemisphere. These episodes could be due to ischemia secondary to the inadequate carotid blood flow, possibly associated with a cardiac arrhythmia. Another possibility is that the TIAs were due to emboli from an arteriosclerotic plaque that ultimately resulted in carotid occlusion. This theory gains support from the observation that no patient had recurrent TIA in the ipsilateral cerebral hemisphere after the carotid occlusion was diagnosed. Presumably the source of emboli was eliminated when the occlusion occurred.

SURGICAL POSSIBILITIES

We had wondered if this study would identify a group of patients that might benefit from an extracranial to intracranial arterial anastomosis around an occluded carotid artery. We found no such group. Our patients were either unable to tolerate the occlusion and soon died or, if they survived, they were more at risk from myocardial infarction than repeated neurological insults. In fact, all subsequent strokes in our group of patients with an occluded internal carotid artery occurred in the opposite hemisphere. Therefore, our data cannot be used to justify extracranial-intracranial anastomosis as a routine, prophylactic procedure in patients who recover from the effects of occlusion of a carotid artery. Among our patients, the operation would have had to be done at the time of the presenting stroke to be effective. But, as we have speculated earlier, the carotid occlusion is probably fresh in acute stroke, and a good chance exists that cerebral blood flow could be restored by a surgical approach to the carotid artery itself. Even so, most of our patients who died had such severe neurological deficits when first seen that restoring carotid blood flow probably would not have improved their prognosis. If extracranial-intracranial arterial bypass has a role, it might be in the occasional patient who has recurrent symptoms due to vascular insufficiency in the cerebral hemisphere distal to an occluded carotid or middle cerebral artery. The main role of surgery appears to lie in the opening of the occluded carotid artery in selected patients with acute stroke and in the prophylactic repair of an ulcerated or stenotic lesion at the contralateral carotid bifurcation.

References

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