The Rationale For Patch-Graft Angioplasty After Carotid Endarterectomy: Early and Long-Term Follow-Up

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SUMMARY  A prospective study was undertaken in March 1980, at the Vascular Surgery Department of the Padua University, Medical School, to establish whether patch graft angioplasty is useful in preventing restenosis after carotid endarterectomy (CE). Seventy-four patients underwent 86 CE (bilateral in 12 cases) for atherosclerotic disease involving the carotid bifurcation. Thirty-eight (51.4%) patients presented TIA’s or nonhemispheric symptoms of cerebrovascular insufficiency; 30 (40.5%) were asymptomatic and 6 (8.1%) had partial nonprogressing or fixed strokes. All operations were performed under general anesthesia, with pharmacologic hypertension and systemic heparinization; in all cases, continuous EEG monitoring and ‘stump pressure’ measurement were employed. The operation was performed without a temporary intraluminal shunt in the patients showing tolerance to carotid clamping. The protection of the shunt was required only in patients with EEG monitoring changes (17). All carotid arteriotomies were extended into the internal carotid artery to overpass the end of the endarterectomy. Overpass was also used in the proximal edge of the arteriotomy, in the common carotid artery. The distal intima was never fixed with stitches and the arteriotomy was routinely closed with a PTFE patch graft angioplasty. Early results of cerebral protection were excellent. No patient presented permanent or transient postoperative neurological problems, no patient died in the postoperative period for causes related to the operation. This is substantiated by results we achieved during the period 1970–1979 in 192 patients, when all carotid endarterectomies were routinely performed without a shunt, with figures of 2.5% of postoperative stroke and 1.5% of mortality.

Long-term follow-up (from 6 to 36 months) was completed in 51 patients (60 operations). All patients were clinically evaluated and tested for patency of the endarterectomized vessel and the contralateral carotid artery by means of c.w. Doppler sonography and, occasionally, by Duplex scanning. Patency of the endarterectomized carotid artery with absence of hemodynamically significant lesions was well detected in all cases. There were 4 late deaths unrelated to cerebrovascular insufficiency. Two patients showed a neurologic deficit. They were investigated with carotidography: both presented intracranial lesions. The absence of carotid restenosis, documented with noninvasive cerebrovascular testing, confirms that the closure with patching effectively delays and prevents this complication by means of a mechanism related to the compensation of the volumetrical increase either of the new atherosclerotic plaque or neointimal hyperplasia. The authors believe that direct closure of the vessel is the primary cause of recurrent stenosis and therefore recommend routine patch graft angioplasty after carotid endarterectomy.

CAROTID ENDARTERECTOMY (CE) is an effective and relatively safe procedure for the treatment of atherosclerotic disease involving carotid bifurcation. In the period immediately after operation during carotid clamping, either technical errors (such as residual intimal flap, suture stenosis or shunt-induced trauma) or the temporary interruption of blood flow, may occasionally cause transient and permanent neurological deficits or fatal strokes.

Although an intraluminal shunt (IS) may be used routinely,1-3 many find it an awkward method to ensure adequate cerebral perfusion during carotid cross clamping. Moreover, this procedure gives a higher percentage of cerebral strokes for purely technical reasons (intimal dissection, embolism, poor blood flow, traumatization and shunt flexion).

Therefore, there is a tendency toward selective placement of an IS in patients who cannot tolerate carotid clamping.4 We have selected patients based on the following: continuous intraoperative EEG monitoring and ‘stump pressure’ measurement. However, restenosis of the endarterectomized segment constitutes a late complication of the CE.5-15 Most, but not all, of these recurrent lesions have no clinical significance. This report describes our surgical experience with the performance of the CE routinely closed with patching to delay and prevent recurrent carotid stenosis.

Material and Methods

From March 1980 to July 1983 at the Department of Vascular Surgery of Padua University, Medical School, 74 patients underwent 86 CE (bilateral in 12 cases) for atherosclerotic disease involving carotid bifurcation with the routine use of continuous intraoperative EEG monitoring and stump pressure measurement.

The decision for surgery in each case was based on the parameters summarized in Table 1, which explains the factors indicating the need for CE. The preoperative symptoms in 74 patients were as follows: TIAs or nonhemispheric symptoms (38 cases); fixed or partial nonprogressing strokes (6) and 30 cases of asymptom-
Indications and Contraindications for Carotid Endarterectomy

A) Indications:
1) Transient ischemic attacks (TIA's) or other symptoms of cerebrovascular insufficiency (stenosis greater than 75% and less than 75%);
2) Asymptomatic patients (stenosis > 75%);
3) Asymptomatic patients (stenosis < 75% with angiographic feature of ulcerated plaque);
4) Asymptomatic patients (stenosis > 50% and complete occlusion of contralateral carotid artery);
5) Asymptomatic patients (bilateral stenoses > 50%).

B) Contraindications:
1) Technical reasons: complete occlusion of internal carotid artery;
2) Acute stroke;
3) Chronic stroke with fixed severe neurological deficit;
4) Biological age;
5) Severe psychic impairment.

Results

In 69 CEs, normal intraoperative EEG monitoring allowed the performance of the operation without protection of an IS. EEG abnormalities during carotid crossclamping appeared in 17 cases. In these patients a temporary IS was inserted selectively. Three of these patients had EEGs consistent with ischemia despite a stump pressure greater than 50 mm Hg (table 2). In 16 patients EEG changes appeared within 20 and 60 seconds after clamping; in 1 patient after 20 minutes.

EEG criteria for placing an IS were as follows: 1)
generalized bilateral slow-wave activity (5 cases); 2) ipsilateral slow-wave activity (9 cases); 3) ipsilateral slow-wave activity with suppression of fast-wave activity (3). Contralateral slow-wave activity never occurred. All 17 patients reverted to baseline after shunt insertion. In 1 patient the EEG abnormalities (bilateral slow-wave activity) appeared after the placement of the needle electrodes and indicated a need to replace the head. EEG changes immediately disappeared.

Lesions of the contralateral internal carotid artery were present in 55 (74%) patients; in the patients bilaterally operated, the first operated side was considered normal. All 17 patients requiring IS protection presented contralateral internal carotid artery lesions (table 3).

None of the 74 patients, whether operated with selective use of the IS (17) or without shunt (57) developed transient or permanent neurological deficits. No patient died in the postoperative period for causes related to the operation.

These data can be compared to case-control patients (192) who had undergone CE (204) performed by the same author from 1970 to 1979. In these patients CE was routinely carried out without IS protection and the arteriotomy was closed with patching,23 with figures of 2.5% of neurological deficits and 1.5% of early mortality.

In the immediate postoperative period, all patients were reassessed with EEG and C.W. Dopplersonography: in all cases the success of the operation was amply tested. Long-term follow-up (from 6 to 36 months) was done in 51 patients (60 CE). All patients of this series were questioned with regard to any postoperative symptoms. Eight patients were unwilling to attend for review, but their general practitioner stated that they did not suffer with any postoperative neuro-

<table>
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<th>Degree of stenosis by arteriography (%)</th>
<th>No. patients</th>
<th>IS use</th>
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<tr>
<td>Thrombosis</td>
<td></td>
<td></td>
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<tr>
<td>&gt; 90</td>
<td>4</td>
<td>1</td>
</tr>
<tr>
<td>75–90</td>
<td>10</td>
<td>2</td>
</tr>
<tr>
<td>50–75</td>
<td>10</td>
<td>0</td>
</tr>
<tr>
<td>10–50</td>
<td>17</td>
<td>5</td>
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<tr>
<td>0</td>
<td>19</td>
<td>0</td>
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*Only in these patients the IS was selectively inserted.
logical symptoms; two patients had moved from the area but also remained well. Since the absence of postoperative symptoms is no guarantee of vessel patency, all patients were also evaluated for patency of the endarterectomized carotid and the contralateral artery by means of C.W. Dopplersonography. 9 M Duplex scanning was occasionally employed.

All patients in whom neurological symptoms appeared or in whom there was evidence of contralateral unoperated carotid artery progressive disease underwent a new carotid angiogram.

There were 4 late deaths. Causes were as follows: myocardial infarction (2); intestinal infarction (1) and cerebral hemorrhage (1). Two years after operation, one patient suffered a transient ischemic episode ipsilateral to endarterectomized carotid. C.W. Dopplersonography tested the complete patency of the vessel operated. A new carotidography confirmed this data but it also revealed a carotid siphon stenosis and the complete occlusion of several intracranial vessels. One year after CE, another patient suffered a minor stroke involving the cerebral hemisphere ipsilateral to the endarterectomized vessel. C.W. Dopplersonography and the control angiogram confirmed the patency of the carotid artery operated. This patient presented a concomitant carotid siphon stenosis with lumen reduction of about 70%. None of remaining patients displayed temporary or prolonged neurological problems and all symptomatic patients showed complete remission of symptoms.

Patency of the endarterectomized carotid artery with absence of hemodynamically significant lesions was well detected in all cases (fig. 2). No pseudoaneurysm developed; and no patch infection occurred.

Discussion

The risk of cerebral stroke following carotid cross-clamping is always present during carotid endarterectomy. Continuous intraoperative EEG monitoring identified those patients who could not tolerate carotid clamping because of an inadequate collateral cerebral perfusion. In this group alone the protection of an indwelling shunt is required. The selective use of this procedure has eliminated the possibility of permanent neurological deficits in a very high percentage of patients undergoing CE, limiting the potential appearance of complications to the small group in which shunt protection is required.

Sundt* showed that four groups of EEG changes can be identified related to different times of ischemia capable of producing irreversible cerebral lesions (4–5 minutes; 8–10 minutes; 15–16 minutes and, finally, 20–22 minutes).

It is evident that not all patients with EEG changes would have suffered cerebral stroke or neurological deficits if IS had not been inserted. In fact, in most

*In discussion at XIIth World Congress of the International Union of Angiology; Rochester, Minnesota, U.S.A. Sept. 1983.
patients the cerebral blood flow following carotid endarterectomy had been restored in sufficient time for preventing cerebral lesions due to ischemia after carotid clamping. The remaining patients with EEG changes consistent with cerebral ischemia, would have presented neurological problems during and after surgery. It would be hypothetical to prognosticate the results of eliminating a shunt, or the duration of the procedure and clamping. The shunt was inserted in all patients with EEG changes, to minimize the possibility of risk of cerebral infarction.

The success of this method is corroborated by comparison with those results achieved during the period from 1970 to 1979, when all CEs were routinely performed without IS protection, relying on CE and rapid patch procedure (12-15 minutes of carotid cross-clamping, in the first series vs. 30-45 minutes in the latter series).21, 22

On the grounds of excellent results achieved in the latter series, confirming the findings of other authors, we can say that this method is effective and draw the following conclusions: 1) On the basis of EEG abnormalities requiring IS protection, carotid cross clamping could cause cerebral stroke or neurological deficits in a percentage of 17 patients; 2) EEG changes normally appear immediately after clamping, but the possibility that this occurs also after 20 minutes must be kept in mind and it is the reason for continuous EEG monitoring; 3) In a very high percentage of cases, EEG changes consistent with cerebral ischemia appear in the presence of occlusion of the opposite carotid artery; 4) Stump pressure alone is not reliable in 100% of cases, as demonstrated by personal observation of three patients with stump pressure greater than 50 mm Hg despite EEG abnormalities suggesting cerebral ischemia; 5) Prejudice against CE in asymptomatic patients is unjustified in view of harmlessness of the operation. Therefore, regardless of very tight carotid artery stenosis, the indication for surgery is also justified in hemodynamically significant lesions. An example is lesions with a degree of lumen stenosis of about 75%. In fact, because the natural anatomical history of carotid lesions is complete occlusion in spite of clinical findings which characterize it (from asymptomatic thrombosis to complete stroke), the indication for surgery is valid since contralateral internal carotid artery is also involved in 80%24, 25 and potentially in 100% of cases. Therefore, during increasing stenosis on one side, the situation will be different depending upon whether the opposite carotid is occluded because of atherosclerotic disease, or patent, due to previous endarterectomy. In the present series, the contralateral unoperated carotid artery showed a progression of the disease in two cases: one asymptomatic complete occlusion and one progression of the stenosis.

The possibility of recurrent carotid stenosis has been documented by several authors,3-19 although the incidence varies from 0.6 to 19 percent, depending upon the method employed for the postoperative follow-up. Restenosis is probably more common than recognized because it may remain undetected until neurological symptoms develop and until prompt clinical investigation.

Technical factors have been suggested by some authors as a major cause of postoperative carotid stenosis. Vascular clamp injury, incomplete removal of plaques, intimal flaps, suturing faults — all may contribute to the formation of stenotic lesions which, in the majority of cases, are localized at the carotid bulb, but which also are located at, or just beyond, the proximal or distal suture line. Further, it is possible that some proximal or distal recurrences are secondary to atherogenesis provoked by intimal damage caused by vascular clamps. In this regard, it is important to emphasize how there is not only one standard technique for CE, but this surgical procedure is performed in many different technical ways, depending upon the details reported in table 4.

No author has attached any importance to the presence or absence of an enlarging patch graft. Stoney and String4 report that in all 29 patients of their series the original arteriotomy had been closed without patching. Cossman and Callow6 noted that of the nine arteries that became restenotic, eight had been closed without patching at the original operation. A pseudoaneurysm along with restenosis developed in a patient whose arteriotomy had been closed with a vein patch graft at the original operation. In the Hertzler’s series,16 of 1252 patients undergoing CE presented a recurrent stenosis; in these patients primary closure had been performed without patching. Cossman and his associ-

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<th>TABLE 4. Carotid Endarterectomy Procedure: the Surgical Technique is Performed in Many Different Ways, Depending upon the Combination of the Following Technical Details</th>
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<tr>
<td>1) Arteriotomy prolonged into the internal carotid artery</td>
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<tr>
<td>2) Proximal overpass</td>
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<tr>
<td>3) Distal overpass</td>
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<tr>
<td>4) Distal intima fixed with stitches</td>
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<tr>
<td>5) Direct closure of arteriotomy</td>
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<tr>
<td>6) Routine patch graft (vein or PTFE) angioplasty</td>
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<td>7) Dissection plane of the plaque</td>
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<td>8) 'Peeling' (removal of medial fragments)</td>
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*Personal surgical technique of carotid endarterectomy.
patch graft angioplasty after carotid endarterectomy

Patch graft angioplasty after carotid endarterectomy/Denius et al

et al present a series of 16 arteries in 14 patients with recurrent stenosis: primary closure of the internal carotid artery had been performed without patch graft angioplasty. In the Terpstra's experience, 11 of 159 endarterectomized carotid arteries presented a late re-stenosis; in this series, patch graft angioplasty never but once had been employed. Clagett and his associates12 have reported there was no significant difference in the incidence of hypertension, diabetes mellitus, coronary artery disease, other vascular operations or family history for atherosclerosis in patients with recurrent stenosis compared to control patients. The report, however, underestimates the importance of the use of the patch graft angioplasty. Zierler et al10 alone, make a superficial mention of the problem of patching, but they conclude that, unfortunately, the number and type of patch angioplasties used is too small to provide any definitive answers. Nevertheless, they maintain that the questions raised by their study indicate the need for further investigation. Since the association between direct closure and recurrence is very strong, these authors are not aware that patching appears to be the most important reason for prevention of recurrent stenosis. For reasons of organization, in the first series of 192 patients surgically treated from 1970 to 1979, a prospective study had not been made. Only in 40 patients was follow-up study possible. None of these patients tested with C.W. Doppler ultrasound presented recurrent stenosis.

Figure 3. This histologic specimen concerns a patient, 62 years old, who underwent a left CE and PTFE patching for a very tight stenosis, in January 1978. After 1 week, the same patient underwent a femoropopliteal bypass for a femoropopliteal complete occlusion with gangrenous at the 1st, 2nd and 3rd toes. He felt very well until February 1, 1982, when he was admitted to the Medicine Clinic of the University of Padua for massive myocardial infarction. On the following day he died. At autopsy, the operated carotid artery was excised. The external surface was dissected to expose the patch. The common carotid artery (c.c.a.) was then opened longitudinally with the incision line running along the posterior wall, opposite to the patch (arrows) and was comprehensive of the internal carotid artery. The external carotid artery was then opened from the ostium. Macroscopic examination showed a very bright surface of contiguous arterial tract corresponding to the endarterectomized tract and to the patch. This segment had not undergone surgical manipulation, except for a small tract of common artery in which traumatism due to autopsy technique remotion was evident (•). The vessel was patent and there was no stenosis.

Figure 4. Low magnification of the histologic specimen. The endarterectomized arterial surface and patch graft (arrow) have been covered by cellular fibrous tissue. This was covered by an intimal "neoendothelium" (Van Gieson for elastic fibers, × 12)
Although our series of 74 patients is not large, in contrast with others, the absence of carotid restenosis, documented with noninvasive cerebrovascular testing,\(^9\)\(^{-20}\) confirms that this procedure effectively delays and prevents this complication. The reliability of C.W. Doppler ultrasound in demonstrating a recurrent stenosis is the same as that concerning studies for assessment of carotid artery disease.\(^9\)\(^{-20}\) In fact, the natural history of patch graft is the same as endarterectomized carotid artery. The patch is covered by sparsely cellular fibrous tissue that may interact with platelets and coagulation factors to induce a proliferative response, especially in the arterial wall; finally the surface is covered by a reendothelial cell proliferation. This is confirmed by the original autopic specimen of the figures 3, 4, and 5 which was microscopically examined.

Therefore, in our opinion, excluding the above-mentioned technical factors, the advantage of patching to prevent the decreasing of lumen is possible by means of two mechanisms: 1) Patch graft enlarges the lumen at the level of possible increase of the myointimal fibroplasia or new atherosclerotic plaque, whether it occurs on the endarterectomized segment or beyond the distal edge of the plaque where intima is intact (overpass) (fig. 6); 2) By enlarging the lumen, the patch may influence the development of intimal lesions;\(^26\)\(^{-27}\) it could serve as a source of cells for a rapid reendothelialization of the endarterectomized segment and so might decrease the incidence of proliferative lesions in the endarterectomized wall.

On the basis of our experience, we believe the direct closure of the endarterectomized carotid artery is the primary cause of recurrent stenosis and we recommend the routine patch graft angioplasty after CE to prevent a possible recurrence.

Acknowledgment

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References


![Figure 5](image1)

**Figure 5.** A higher magnification photo of the intimal 'neoendothelium'. (hematoxylin-eosin, × 160)

**Figure 6.** Diagram shows how patch graft angioplasty following carotid endarterectomy inhibits restenosis.
The rationale for patch-graft angioplasty after carotid endarterectomy: early and long-term follow-up.

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