Prevention of early restenosis and thrombosis-occlusion after carotid endarterectomy by saphenous vein patch angioplasty

JP Archie, Jr

The online version of this article, along with updated information and services, is
located on the World Wide Web at:

http://stroke.ahajournals.org

raphy scans in acute cerebral infarction. Neuroradiology **135**: 655–662

12. Gawkrodger DJ, Penny ME: Cerebral infarction mimicking arteriovenous malformation on contrast computerised tomography. Brit J Clin Prac **37**: 273–275, 1983

13. Henrikson GC, Dushyant VP: Enhancing cerebral infarction simulating arteriovenous malformation on computed tomography. J Comput Assist Tomogr **9**: 502–506, 1985

14. Vonofakos D, Artmann H: CT findings in hemorrhagic cerebral infarct. Computerized Radiol **7**: 75–83, 1983

Prevention of Early Restenosis and Thrombosis-Occlusion After Carotid Endarterectomy by Saphenous Vein Patch Angioplasty

JOSEPH P. ARCHIE, JR., PH.D., M.D.

SUMMARY The hypothesis that saphenous vein patch angioplasty protects against early postoperative restenosis and thrombosis-occlusion was tested by comparing the clinical outcome and carotid artery status of 100 carotid endarterectomies with and 100 without saphenous vein patch angioplasty performed by a single surgeon over a 30-month period. The patient population, selection, perioperative management, and the technical aspects of the operation, except for the vein patch, were essentially identical in both groups. Carotid artery status was assessed by direct continuous wave Doppler and Gee OPG at three to six months and again at one year postoperatively. There were two hospital deaths, both in the nonpatched group, one cardiac and the other neurologic due to internal carotid thrombosis. Two reversible neurological deficits due to thrombosis and one due to restenosis occurred in the non-patched group. Asymptomatic > 50% diameter restenosis occurred in four and asymptomatic occlusion in one non-patched carotids. There were no restenosis, no occlusions and no neurologic symptoms in the patched group. Morbidity, mortality, restenosis or thrombosis-occlusion occurred in 10/100 (10%) non-patched and 0/100 (0%) patched arteries ($p < 0.01$ by Chi Square). Restenosis or thrombosis-occlusion occurred in 9/100 (9%) of non-patched and 0/100 (0%) patched arteries ($p < 0.01$). These results support the use of saphenous vein patch angioplasty reconstruction of carotid endarterectomy to protect against early restenosis and thrombosis-occlusion.

Stroke Vol 17, No 5, 1986

POSTOPERATIVE RESTENOSIS or thrombosis-occlusion occurs in 3% to 19% of endarterectomized carotid arteries,[1–9] and many of these occur early. Fortunately only 20 to 25% of restenosed carotid arteries identified after discharge from the hospital become symptomatic,[3, 5] but this is still a major cause of late post-carotid endarterectomy morbidity and mortality. Similarly early postoperative thrombosis-occlusion plays a major role in neurologic morbidity and mortality. Refinement of perioperative care and the technical aspects of carotid endarterectomy have reduced the hospital morbidity and mortality of this operation to 1 to 3% for many surgeons and institutions, but the need for prevention of early postoperative thrombosis and restenosis is evident. The complexity and variability of extracranial carotid artery atherosclerosis, including long tongues of plaque extending up the internal carotid, small diameter arteries and atheroma in poststenotic dilations make the technical difficulty of carotid endarterectomy unpredictable prior to operation in many patients. Patch angioplasty with synthetic materials[10, 11] and vein[12–16] have been recommended to over-

come these problems and in some series used routinely. However, there is little data to support vein patching after carotid endarterectomy. The purpose of this study is to test the hypothesis that saphenous vein angioplasty reconstruction after carotid endarterectomy protects against early thrombosis-occlusion and restenosis when compared to a similar group of non-patched carotid endarterectomies.

Methods

Over a 30-month period, 209 carotid endarterectomies were performed on 188 patients by the author. Of these 200 were primary endarterectomies, 8 were operations for restenosis in symptomatic patients and one was an emergency procedure with attempted thrombectomy of an acute internal carotid occlusion. The data base for this study is the 200 primary carotid endarterectomies performed on 181 patients. The selection of patients for vein patch angioplasty was not randomized. Of the first 105 operations in this series only eight arteries had vein patch angioplasty, and of the last 95 only three arteries were not patched. The operations were performed at two community hospitals with essentially identical angiographic, operative and intensive care unit facilities and personnel. All procedures were performed in the entirety by the author. There were no differences in surgical technique during this time frame with the exception of vein patch

angioplasty. The indications for operation for the patched and non-patched groups are listed in table 1. All asymptomatic carotid stenoses were greater than 80% diameter. Standard two view arch four vessel arteriograms were obtained in all patients except four who had high quality venous digital subtraction angiography. Selective biplane extracranial and intracranial views were obtained in 75% of the cases.

General endotracheal anesthesia was used in all but two operations, both of these being done with local. There was no attempt to rigidly control arterial carbon dioxide levels by the anesthesiologist but in general mild hypocarbia was employed. Operative technique included careful dissection with minimal handling of the carotid bulb, use of a very soft bulldog vascular clamp on the internal carotid artery above the diseased segment, elastic vessel loops on the external carotid artery branches, and a Fogerty hydrogrip clamp or soft elastic loop on the common carotid artery. The decision for selective shunting was based upon the collateral cerebral perfusion pressure method with a shunt being used when the pressure was less than 18 mm Hg.[17] Five or six thousand units of intravenous heparin depending on the patient's weight was given. The arteriotomy was carried up the internal carotid to the level of the end point when no patch was used. When a vein patch was placed, the arteriotomy extended 4 to 5 mm distal to the preserved endothelium of the internal carotid artery, creating a completely endotheolized distal tapering of the new bulb and a widened partially endothelized endarterectomized arterial segment with patch placement. An attempt was made to obtain a good quality distal end point with complete removal of the tongue of the plaque. End point tacking sutures were used when needed. Careful flushing of the vessels was performed prior to completion of closure, and the reconstructions were performed with a running 6-0 Proline suture technique. Optical 2½ power magnification was used for all procedures. Five to six cm of greater saphenous vein was harvested just above the ankle and prepared for patch angioplasty by longitudinal opening. In one patient the vein was removed from the thigh because of a previous vein stripping. During and after the operative procedure systemic blood pressure was controlled at preoperative normal levels for each patient using intravenous nitroglycerin or sodium nitroprusside (Nipride) for patients with elevated pressure and phenylephrine (Neosynephrine) for elevation of low pressure. At the completion of the reconstruction and restoration of flow, continuous wave Doppler velocity signals were obtained with a sterile probe interrogating all three vessels to assure satisfactory hemodynamics.[18, 19] All patients were placed in an intensive care unit for at least 20 hours, and approximately 30% required continuous intravenous pharmacologic control of systemic blood pressure during the initial 1 to 18 hours after operation. Table 2 contains the data on patient age, sex, collateral cerebral perfusion pressure, use of indwelling shunts, carotid occlusion time, use of end point tacking sutures, and the necessity for carotid re-exploration in the operating room based on direct Doppler velocity analysis.

Patients were followed in the office with noninvasive evaluation of their carotid status at two to six and twelve to twenty-six weeks after operation in all patients. A one-year follow up was obtained in most patients. Direct carotid artery interrogation with a continuous wave Doppler was done in all patients and Gee oculoplethysmography was possible in 94% of the patients. The presence of a cervical bruit was documented by stethoscope, phonoangiography or both.

Results

There were no operating room or intensive care unit deaths. The hospital postoperative mortality, neurologic morbidity and asymptomatic restenosis and occlusion results are given in table 3 for the patched and non-patched groups. The single cardiac death occurred acutely 36 hours after operation from a presumed arrhythmia after an uneventful early postoperative course. At autopsy the endarterectomized carotid artery was widely patent without evidence of thrombosis or other abnormality. The four non-patched patients with asymptomatic restenosis were identified two to six months after operation. Two were re-operated upon for evidence of high grade disease confirmed by arteriogram and both had myointimal hyperplasia in the endarterectomized segment and underwent patch an-

TABLE 1 *Indications for Carotid Endarterectomy for 100 Non-patched and 100 Patched Arteries*

	Non-patched	Patched
Transient ischemic attack (TIA)	31	29
Completed stroke/RIND	14	13
Amaurosis fugax	16	18
Nonlateralizing TIA	12	11
Asymptomatic single side	11	8
Asymptomatic second side	7	12
Asymptomatic major operation*	9	9
Totals	100	100
	(93 patients)	(88 patients)

*With or prior to coronary artery bypass or abdominal aortic surgery.

TABLE 2 *Population and Operative Technical Data on Non-patched and Patched Groups*

	Non-patched		Patched
Age (years, mean ± 1SD)	63.9 ± 10.1		65.4 ± 9.04
Sex (M/F)	54/39		51/37
Carotid occlusion time (min, mean ± 1SD)	20.0 ± 4.24	*	28.2 ± 4.63
Shunt used	8/100		8/100
End point tack sutures	12/100	*	4/100
Re-exploration based on doppler	1/100		0/100
Collateral cerebral perfusion pressure (mm Hg, mean ± 1SD)	39.2 ± 15.3		36.1 ± 15.7

*$p < 0.05$, p by Chi Square or unpaired t test.

TABLE 3 *Morbidity, Mortality, Restenosis and Occlusion in Non-patched and Patched Groups*

	Non-patched (n = 100)	Patched (n = 100)
Mortality		
neurologic (thrombosis)	1	0
cardiac (arrhythmia)	1	0
Morbidity		
TIA/stroke (< 24 hr post op)	0	0
TIA/stroke (> 24 hr post op)	3*	0
Asymptomatic occlusion (< 26 weeks)	1	0
Asymptomatic restenosis		
< 50% diameter (< 26 weeks)	4	0

*Two occlusions, one restenosis, one permanent neurological deficit.

gioplasty (the second operations are not included in this series). The other two patients had evidence of moderate, estimated 50 to 60% diameter, restenosis and are currently being followed. Their oculoplethysmographic pressure differences between the eyes were six and eight millimeters of mercury respectively and the internal carotid Doppler frequency increases are moderate. No restenosis or occlusion not previously identified was found at one year in the 94 patched and 93 non-patched arteries available for follow up at one year. Three patients died of cardiac disease and two of cancer in the first year. There were no known late strokes.

The single neurologic death resulted from carotid thrombosis in the recovery room in a diabetic patient operated on for a TIA with an ulcerated plaque. He initially awakened neurologically intact but developed progressive hemiparesis over two hours. Reexploration revealed thrombosis of the endarterectomized internal carotid artery and thrombectomy and patch angioplasty with Dacron was performed. The patient developed thrombosis of the intracranial vessels and died three weeks later from recurrent progressive stroke. There were no transient ischemic attacks or other new central neurologic deficits in either group during the first twenty-four hours after operation. A single patient with vein patch angioplasty developed ipsilateral transient ischemic symptoms six months after surgery and an arteriogram demonstrated an intact nonaneurysmal nonstenotic saphenous vein patch angioplasty and severe siphon ulcerative and stenotic disease not present on the initial arteriogram. An extracranial-intercranial bypass was performed.

In the non-patched group 10 of 100 operations (10%) resulted in either mortality, neurologic morbidity, restenosis or thrombosis-occlusion (table 3, $p <$ 0.01 by Chi Square analysis when compared to 0/100 in the patched group). Restenosis or thrombosis-occlusion occurred in 9/100 (9%) of the non-patched and 0/100 (0%) of the patched arteries (table 3, $p < 0.01$). The non-patched group had a postoperative cervical bruit on the operated side by stethoscope or phonoangiography in 53/98 (54%), whereas only 27/100 (27%)

of the patched group had a bruit ($p < 0.05$ by Chi Square).

Discussion

These results support the hypothesis that saphenous vein patch angioplasty is advantageous in prevention of thrombosis-occlusion and restenosis in the early postoperative period. However, it remains unclear what the long term advantages or disadvantages of vein patching may be and in which subgroup of patients undergoing carotid endarterectomy this technique may be beneficial. While the temporary and permanent neurologic deficit and neurologic death rates are clearly not statistically different between the non-patched and patched groups in this small series (four versus zero) because of Type II statistical error, the combination of these events with asymptomatic restenosis and thrombosis-occlusion does yield statistical significance and indicates that saphenous vein patch angioplasty may be advantageous. The differences in results between nonpatched and patched groups are evident in this series in part because of the absence of new neurologic events in the first few hours after operation, probably because of adherence to well established methods of endarterectomy including confirmation of an acceptable hemodynamic result.

These results are subject to the criticism of any series of consecutive cases and must be interpreted in this light.[20] Further, these are the results of a single surgeon, and while on the one hand this assures that the surgical technique was probably similar for each operation, it does not mean that it is necessarily reproducible. Also there was no attempt to randomize the use of a patch and the majority of the non-patched endarterectomies were done prior to those that were patched. This is clearly subject to criticism since the operating surgeon's technique may have improved with time and experience, even though he had performed over 200 carotid endarterectomy prior to this series. In support of these results, the patients come from the same population pool, were operated upon similarly, and had essentially identical indications for surgery.

Patch angioplasty reconstruction after primary carotid endarterectomy has been used since the early reports of the results of this operation.[10] It is, however, difficult to determine if results with synthetic patching are superior to non-patching because of the variability in both patients and surgery within and between early series. Recent results of carotid endarterectomy patching with modern synthetic materials in a small series suggest an advantage to patch use.[11]

There are clear advantages to using a saphenous vein patch over synthetic materials. First, a fresh vein has a preserved endothelial surface which is non-thrombogenic if care is used in preparation. Second, vein is easier to handle and third, infection is theoretically less likely to occur using vein. The results of Sundt and colleagues[13-15] using greater saphenous vein harvested from just above the ankle as a patch are comparable to ours. In a very large series, prior to patching they had an early post operative carotid occlusion rate of 3%

which decreased to less than 0.05% when saphenous vein patch reconstruction was routinely used. A recent report of post carotid endarterectomy occlusion confirmed by angiography showed 6 of 70 non-patched and 0 of 50 patched carotids occluded ($p < 0.04$).[16]

Of clear concern is the natural history of carotid vein patch angioplasty over a longer period of follow-up. It has been suggested that saphenous vein patch angioplasty in the carotid position may become aneurysmal with time,[21] but information regarding aneurysm formation is hard to obtain.

The advent of accurate noninvasive vascular laboratory techniques has clearly established that recurrent stenosis and occlusion is common after carotid endarterectomy.[1-9] While it is difficult to determine the etiologies of restenosis and occlusion at various periods after operations, it is likely that many early postoperative thrombosis-occlusions or restenosis are due to technical problems. Asymptomatic abnormalities may not be picked up until the noninvasive test are done. Post hospital causes are myointimal hyperplasis which may regress with time,[1, 22] and late atherosclerosis, which is probably a continuum. The restenosis and occlusion rates of this series of non-patched carotid arteries is consistent with most reports using similar diagnostic techniques.[7, 8] Digital subtraction arteriography gives a lower incience of post endarterectomy restenosis.[22, 23]

The incidence of restenosis in symptomatic patients when confirmed by angiography is less than 4%.[24-26] Further, a low incidence of symptomatic restenosis has been reported in large series when patching was not employed.[27] In order to achieve the acceptable 1 to 3% overall morbidity and mortality generally acceptable for this operation[28] selective vein patching may be advisable to prevent early problems.

There are theoretical advantages of vein patch angioplasty in certain situations. A tongue of plaque or stenosis extending above the bulb of the internal carotid artery may require closure of a long arteriotomy in a small endarterectomized vessel. Similarly a small artery may be more advantageously patched than primarily closed even when optical magnification, microsurgical techniques and fine suture material are used. The availability of a saphenous vein patch may make the surgeon more aggressive in obtaining a complete endarterectomy with a longer arteriotomy and removal of the tongue of plaque. This is supported by the significantly lower incidence of having to tack the intima in the patched arteries in this series (table 2). The geometry of the patch moves the carotid bulb up the internal carotid artery. This significantly widens the endarterectomized segment and provides a partial endothelial surface. Peak and mean blood flow velocities and hence the wall shear stresses in the internal carotid are accordingly lower in a patched segment than in a nonpatched one and this may aid to preventing thrombosis and restenosis.

The technical aspects of vein patch angioplasty increase carotid occlusion time as indicated in table 2, but the absence of early new postoperative neurologic

deficits thought to be associated with prolonged occlusion time suggest this is not a problem. Interoperative carotid Doppler interrogation, blood flow measurement or angiography should lower the rate of unrecognized operative technical problems. In an earlier study we found that 4% of our carotid endarterectomies without patching required reconstruction based on intraoperative electromagnetic flow measurements.[19] However, in the present series of nonpatched arteries only one artery required re-exploration.

The interesting finding of a higher incidence of postoperative cervical bruits in the non-patched group is presumptive evidence that turbulence and disturbed flow patterns are more frequent in non-patched than in vein patched carotids. This suggests that the flow characteristics of patched internal carotid arteries may be superior to those of the unpatched ones in terms of generation of early thrombosis and hyperplasia.

Finally, the use of saphenous vein patch angioplasty will probably remain controversial until the subgroup in whom it may be beneficial is identified. It may be the method of choice when long endarterctomy of the internal carotid artery is necessary and for small arteries.

References

1. Zierler RE, Bandyk DR, Thiele BL, Strandness DE: Carotid artery stenosis following endarterectomy. Arch Surg 117: 1408–1415, 1982
2. Thomas M, Otis SM, Rush M, Zyroff J, Dilley RB, Bernstein EF: Recurrent carotid artery stenosis following endarterectomy. Am Surg 200: 74–79, 1984
3. Colgan MP, Kingston V, Shanik G: Stenosis following carotid endarterectomy. Arch Surg 119: 1033–1035, 1984
4. Gonzales LL, Partusch L, Wirth P: Noninvasive carotid artery evaluation following endarterectomy. J Vasc Surg 1: 403–408, 1984
5. D'Donnell TF, Scott G, Callow AD, Mackey WC, Heggerick P, Shepard A: Ultrasound characteristics of recurrent carotid disease: Hypothesis explaining the low incidence of symptomatic recurrence. Vasc Surg
6. Baker WH, Hayes ACA, Mahler D, Littooy FN: Durability of carotid endarterectomy. Surgery 94: 112–115, 1983
7. Salvian A, Baker JD, Machleder HI: Cause and noninvasive detection of restenosis after carotid endarterectomy. Am J Surg 1446: 29–34, 1983
9. Norrving B, Nilsson B, Olsson JE: Progression of carotid disease after endarterectomy. Ann Neurol 12: 548–522, 1982
10. Ott DA, Cooley DA, Chapa L, Coelho A: Carotid endarterectomy without temporary intraluminal shunt. Ann Surg 191: 708–714, 1980
11. Deriu GP, Bullotta E, Bonavina L, Grego F, Alvino S, Franceschi L, et al: The rational for patchgraph angioplasty after carotid endarterectomy: Early and long-term follow up. Stroke 15: 972–979, 1984
12. Imparato Am. Discussion. Surgery 80: 709, 1976
13. Sundt TM, Sandok BA, Whisnant JP: Carotid endarterectomy. Complications and preoperative assessment of risk. Mayo Clinic Proc 50: 301–306, 1975
14. Whisnant JP, Sandok BA, Sundt TM: Carotid endarterectomy for unilateral carotid system tarnsient cerebral ischemia. Mayo Clinic Proc 58: 171–175, 1983
15. Sundt TF: The ischemic tolerance of neural tissue and the need for monitoring and selective shunting during carotid endarterectomy. Stroke 14: 93–98, 1983
16. Little JR, Bryerton BS, Furlan AF: Saphenous vein patch grafts in carotid endarterectomy. J Neurosurg 61: 743–747, 1984
17. Archie JP, Feldtman RW: Determinants of cerebral perfusion pres-

sure during carotid endarterectomy. Arch Surg 117: 319–322, 1982

18. Barnes RS, Garrett WV: Intraoperative assessment of arterial reconstruction by Doppler ultrasound. Surg Gyn Obst 26: 255–261, 1979

19. Archie JP, Feldtman RW: Intraoperative assessment of carotid endarterectomy by electromagnetic blood flow measurements. Surg Gyn Obst 158: 457–460, 1984

20. Moses LE. The series of consecutive cases as a device for assessing outcomes of interrention. NEJM 311: 705–710, 1984

21. Rosenman J, Edwards WS, Robillard D, Geary G: Carotid arterial bifurcation advancement. Surg Gyn Obst 159: 260–264, 1984

22. Pierce GE, Iliopoulos JI, Holcomb MA, Rieder CF, Hermreck AS, Thomas JH: Incidence of recurrent stenosis after carotid endarterectomy determined by digital subtraction angiography. Am J Surg 148: 848–853, 1984

23. Raithel D, Schweiger H, Gentsch HH, Seyferth W, Zeitler E: Digital transvenous angiography in follow-up examinations after carotid reconstruction. J Cardiovasc Surg 25: 400–403, 1984

24. Stoney RJ, String ST: Recurrent carotid stenosis. Surgery 80: 705–710, 1976

25. Thompson JE, Talkington CM: Carotid endarterectomy. Ann Surg 184: 1–15, 1976

26. Hertzer NR, Martinez BD, Benjamin SP, Bevem EG: Recurrent stenosis after carotid endarterectomy. Surg Gyn Obst 149: 360–364, 1979

27. Thompson JE: Carotid endarterectomy, 1982 — the state of the art. Br J Surg 70: 371–376, 1983

28. Kistler JP, Ropper AH, Heros RC: Therapy of ischemic cerebral vascular disease due to atherobrombosis. NEJM 311: 100–105, 1984

Transcranial Doppler in Cerebrovascular Disease

LAWRENCE R. WECHSLER, M.D.,* ALLAN H. ROPPER, M.D.,†‡ AND J. PHILIP KISTLER, M.D.†

SUMMARY   Doppler analysis of flow in intracranial arteries is now possible using a 2 MHz probe allowing sufficient penetration of bone to obtain signals noninvasively. Thirty-two normal subjects, and 11 patients with cerebrovascular diseases including vasospasm following subarachnoid hemorrhage, middle cerebral artery stenosis, and extracranial internal carotid artery stenosis were studied by transcranial Doppler. Increased peak velocity and spectral broadening of the reflected signal corresponded to clinical and angiographic evidence of middle cerebral artery vasospasm or stenosis. Decreased peak velocity and blunted waveforms occurred in the middle cerebral artery ipsilateral to severe extracranial internal carotid stenosis with poor crossfilling from the contralateral carotid artery. Abnormalities resolved following carotid endarterectomy. Transcranial Doppler identifies vasospasm or stenosis of the middle cerebral artery and may allow noninvasive evaluation of collateral flow across the anterior circle of Willis in patients with extracranial carotid artery stenosis.

DOPPLER EXAMINATION of the extracranial internal carotid artery permits noninvasive detection of extracranial stenosis with considerable accuracy.[1] Studying intracranial vessels is now possible using a 2 MHz probe that allows sufficient ultrasound to penetrate bone.[2] This report describes preliminary results of transcranial Doppler (TCD) studies, performed in a systematic and uniform fashion, in patients with vasospasm of the middle cerebral artery (MCA) due to subarachnoid hemorrhage, MCA stenosis, and hemodynamically significant extracranial internal carotid artery (ICA) stenosis. Each group includes only a few patients, however, characteristic abnormalities are identified, in most cases correlating with clinical and angiographic evidence of vascular disease. These results provide an indication of the clinical utility of TCD for studying patients with cerebrovascular disease.

Methods

Instrumentation

A 2 MHz pulsed Doppler instrument with an external probe diameter of 22 mm (TC2-64, EME, Überlingen, W. Germany) was used for transcranial Doppler examinations. Focal depth of the Doppler signal varied in 5 mm increments from 25 to 150 mm. Pulse repetition frequencies included 5, 8 or 10 KHz depending on depth. Bidirectional signals were recorded with a 10 KHz low-pass filter and a 150 Hz high-pass filter. Spectral analysis was accomplished with fast fourier transformation and 64 point resolution. Spectral information was displayed either as KHz frequency shift or velocity. Calculation of velocity from Doppler shift frequency assumed an angle between probe and blood column of 0 degrees.[2]

Technique

Doppler signals from the middle cerebral artery were obtained by placing the probe over the temple and adjusting its position for a maximal reflected signal at a depth of 45 to 55 mm. Depth of focus was then increased until bidirectional flow appeared from the terminal bifurcation of the carotid artery, confirming that the original signal was from the MCA. Recordings were obtained from all depths at which an MCA signal could be detected. Depth of focus was then further