The Geometry and Mechanics of Saphenous Vein Patch Angioplasty after Carotid Endarterectomy

Joseph P. Archie, Jr., M.D., Ph.D.

To elucidate some of the mechanisms that may account for the potential advantages and disadvantages of saphenous vein patch angioplasty (VPA) after endarterectomy of the carotid artery, the author evaluated 50 VPAs with respect to geometry, blood-flow velocities, and wall mechanics. The mean internal carotid artery (ICA) diameter cephalad to the vein patch was 4.5 mm, whereas the mean diameter of the patched segment was 7.4 mm. The mean ratio between the cross-sectional area of the patched ICA and the cross-sectional area calculated as if the artery were nonpatched was 2.9. In the presence of restenosis sufficient to occlude the nonpatched artery, this ratio was decreased to 1.9 when the artery alone was stenosed and to 0.49 when both the artery and the vein patch were stenosed. The mean ratio between the peak blood-flow velocity distal to the patch and the blood-flow velocity in the patched segment was 3.4. These area and velocity ratios indicate that the wall shear stress in the patched segment was three to five times lower than that in the distal ICA.The normal stress in the circumferential vein patch wall was two to three times greater than that in veins used as bypass grafts at the same intraluminal pressure. In comparison to primary arteriotomy closure after carotid endarterectomy, VPA provides a partially endothelialized flow surface, a significantly greater cross-sectional area, and a relatively mild wall shear stress; all three of these factors may protect the artery against both hemodynamically significant restenosis and early postoperative thrombosis. ***(Texas Heart Institute Journal 1987; 14:395-400)***

Key words: Endarterectomy; internal carotid artery; saphenous vein patch angioplasty; restenosis; thrombosis; peripheral vascular disease

AFTER ENDARTERECTOMY of the internal carotid artery (ICA), restenosis and early postoperative thrombosis are a common source of mortality and morbidity. This complication has been estimated to occur after 5% to 19% of carotid endarterectomies, but only 20% to 30% of the patients become symptomatic.[1-8] Because of advances in operative techniques and perioperative care, the incidence of postoperative neurologic deficits tends to be low; nevertheless, restenosis and early postoperative thrombosis remains a major problem. Patch angioplasty reconstruction with synthetic materials[9,10] and saphenous vein segments,[11,12] as well as carotid bifurcation advancement,[13] has been advocated as a means of decreasing the risk of this complication. Recent evidence has revealed that the incidence of early postoperative thrombosis and restenosis after endarterectomy is significantly lower when reconstruction is achieved with a saphenous vein patch angioplasty (VPA) than when a primary arteriotomy closure is used.[14]

Whereas some of the mechanisms that account for the possible advantages of VPA

From Wake Medical Center, Raleigh, North Carolina.

Address for reprints: Joseph P. Archie, Jr., M.D., 3417 Williamsborough Court, Raleigh, NC 27609.

are obvious (such as a partially endothelialized flow surface and an increased cross-sectional area in the endarterectomized segment), others are not. Of particular importance is the reconstruction of small ICAs, as well as of long arteriotomy incisions and endarterectomies that extend cephalad to the bulbous segment of the ICA. To elucidate some of the mechanisms that may account for the potential advantages and disadvantages of VPA, the author evaluated 50 VPAs with respect to geometric characteristics, blood-flow velocities, and wall mechanics.

METHODS

This study was based on 50 carotid endarterectomies in which the arteriotomy and endarterectomy extended cephalad to the bulbous segment of the ICA. In each operation, the endarterectomy was completed, without tacking sutures, and the endarterectomy end point extended distal to what was judged to be the terminal point of the ICA bulb. The arteriotomy incision extended 4 to 6 mm distal to the end point of the endarterectomy; therefore, placement of the vein patch resulted in a tapered, completely endothelialized end point. In all cases, a segment of the greater saphenous vein was obtained from just above the patient's ankle. The segment was tailored to provide a 5- to 8-mm-wide patch that did not balloon out and that was not excessively large. Figure 1 shows the sites of the geometric measurements, which were made with a sterile caliper in the operating room after the vein had been reconstructed and the blood flow reinstituted. These measurements included the diameter of the ICA cephalad to the endartarectomy and the vein patch, the diameter of the patched segment 1 cm from its cephalad end, and the major and minor axes of the elliptical cross-section of the carotid bulb. The diameter of the patched segment of the common carotid artery below the bulb was also measured.

Cross-sectional areas were calculated according to the formula $A = \pi d^2/4$, in which d = diameter. Figure 2 is a schematic view that shows the basis for calculating the surface area occupied by the vein patch and the cross-sectional areas, if restenosis is postulated to be sufficient to occlude the nonpatched artery. Restenosis of the endarterectomized artery alone, as well as of both the artery and the vein patch, is considered. The relationship between the cord of the vein patch, the patched ICA diameter (d) shown in Figure 1, and the angle (θ) of Figure 2 was: cord $= d \sin \theta/2$. This equation allowed calculation of the angle θ in order to determine the percentage of surface area occupied by the vein patch.

Intraoperative continuous-wave Doppler velocity wave forms were recorded with a sterile probe, held at an approximately 45° angle to the blood-flow axis over the midsegment of the patched ICA and over the portion of the artery distal to the vein patch. Normalization of mean and peak blood-flow velocities for comparison between patients was accomplished by using the ratio between the flow velocity in the ICA cephalad to the vein patch and the flow velocity in the patched ICA segment. Estimates of wall shear stress are based on the formula $T = \mu dV/dr$, in which μ = viscosity, V = velocity, and r = the radial coordinate.[15] The velocity gradient at the wall can be approximated with the formula $dV/dr \simeq \Delta d/\Delta r$, in which ΔV = the mean velocity gradient from the wall (zero velocity) to mean main-stream velocity V_{mean}, and Δr = the distance from the wall to mean main-stream velocity. Thus, the ratio of wall shear stress in the patched ICA segment to the wall shear stress in the cephalad ICA is $\Delta V_{patch}/\Delta V_{distal}$, when $\Delta r_{patch} = \Delta r_{distal}$; this is a reasonable assumption based on known velocity profiles of pulsatile flow in round conduits.[16] Since $\Delta V = V_{mean}$, the wall shear stress ratio of two segments can be estimated from the ratio of the mean velocity ratios, or $T_{patch}/T_{distal} = V_{patch}/V_{distal}$. Maximum values occur at peak systolic flow. A second way of estimating the shear stress ratio in these two arterial segments can be derived from simple principles of fluid mechanics: For incompressible fluids, such as blood, flowing in a conduit with a circular cross-section of area A and diameter D, the continuity equation[15] would be $Q = VA$ for each segment where Q = flow rate and V = mean velocity. Because flow in the patched ICA segment $Q_{patch} = Q_{distal}$, then $(VA)_{patch} = (VA)_{distal}$, or $V_{patch}/V_{distal} = A_{distal}/A_{patch} = (D_{distal}/D_{patch})^2$, since $A = \pi D^2/4$. Thus, the ratio between the wall shear stress in the patched segment and the wall shear stress in the distal segment can be estimated from the reciprocal of the diameter ratios squared, or $T_{patch}/T_{distal} \simeq (D_{distal}/D_{patch})^2$.

Normal stresses affecting the vein patch wall were calculated according to the standard formulas for estimating circumferential (Tc) and longitudinal (Tl) stress in a thin-walled cylinder[17]:

$$Tc = Pr/t \text{ and } Tl = Pr/2t$$

where P = hydrostatic transmural pressure, r = radius, and t = wall thickness. Calculation of the maximum wall circumferential normal stress in the common carotid bulb was based on the maximum radius of curvature of an ellipse, $r = A^2/2B$, where A = the major axis diameter and B = the minor axis diameter. Wall stress calculations for the vein used as a tube graft were made according to the standard geometric formulas for determining the relationship between the measured cord and the circumference of the vein. In calculating the areas, wall thickness was not considered; therefore, the outside and inside diameters were considered identical (a minor source of error).

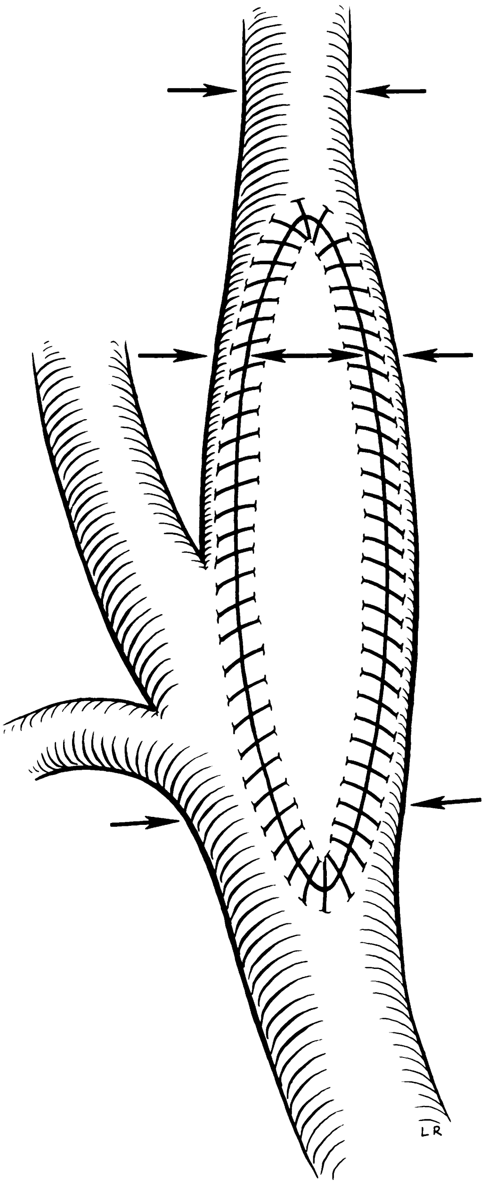

Fig. 1 Schematic diagram of saphenous vein patch reconstruction of an endarterectomized carotid artery. The arrows indicate the sites where the three arterial diameters and the vein patch cord diameter were measured. The diameters of the major and minor axes of the elliptical cross-section of the common carotid bulb were also evaluated.

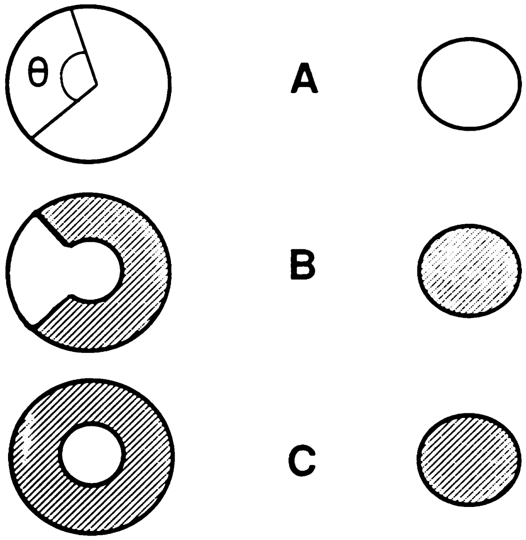

Fig. 2 Cross-sectional schematic views of an endarterectomized internal carotid artery after reconstruction with a vein patch segment (left) and after primary closure (right): (A) at completion of reconstruction; (B) after restenosis of the endarterectomized segment sufficient to occlude the nonpatched artery; and (C) after restenosis of the endarterectomized and vein patch segments sufficient to occlude the nonpatched artery.

RESULTS

Table I presents the geometric measurements associated with VPA, as well as the area calculations, velocity ratios, and wall stresses (both shear and normal). In comparison to primary arterial closure above the ICA bulb, VPA yielded a significantly greater cross-sectional area in the distal patched ICA segment. The values for the residual luminal cross-sectional areas presented in Table I are shown in Figure 2. The similarity between the mean blood-flow velocity ratios and the cross-sectional area ratios is consistent with the principle of the conservation of mass.[15] The longitudinal wall stress equalled only half of the circumferential stress; accordingly, only the latter variable was of clinical importance. The maximum circumferential stress occurred in the elliptical segment of the common carotid bulb, where the radius of curvature is maximum.

Postoperative follow-up consisted of clinical and noninvasive laboratory testing with Gee OPG and direct continuous-wave Doppler techniques. Over the 6- to 18-month follow-up period, no vein patch rupture or aneurysm formation was encountered, and there was no instance of ICA thrombosis or hemodynamically significant restenosis.

DISCUSSION

The use of saphenous VPA to reconstruct carotid endarterectomies that extend distal to the ICA bulb may be advantageous to primary closure of the distal segment, which may lead to early thrombosis or significant restenosis. Because VPA results in a threefold increase in the artery's cross-sectional area, this technique may provide a margin of safety against the development of restenosis in the endarterectomized segment. Even if both

TABLE I. Geometry, Calculated Areas, Velocity Ratios, and Stresses Associated with Saphenous Vein Patch Angioplasty after Endarterectomy of the Internal Carotid Artery (ICA)

Variable	Mean ± 1 SD (n = 50)
Diameter of the ICA cephalad to the vein patch	4.5 ± 0.98 mm
Diameter of the patched ICA segment 1 cm from its cephalad end	7.4 ± 1.03 mm
Diameter of the common carotid bulb (circular cross-section)	7.8 ± 1.11 mm
Diameter of the common carotid bulb (elliptical cross-section):	
Major axis, A	9.7 ± 1.57 mm
Minor axis, B	7.9 ± 1.21 mm
Percentage of the ICA surface composed of vein	37.6 ± 6.05
Ratio of the cross-sectional area of the patched segment to the cross-sectional area of the distal ICA (see Fig. 2A)	2.9 ± 0.86
Ratio of the cross-sectional area of the patched segment to the cross-sectional area of the distal ICA with restenosis sufficient to occlude the nonpatched artery (restenosis of the artery only) (see Fig. 2B)	1.9 ± 0.86
Same as above, but with restenosis of both the artery and the vein patch (see Fig. 2C)	0.49 ± 0.245
Ratio of the mean blood-flow velocity in the distal ICA to that in the patched segment	3.4 ± 1.31
Ratio of the wall shear stress in the patched segment to that in the distal ICA:	
From velocity ratio	0.19 ± 0.056
From diameter ratio	0.34 ± 0.119
Ratio of the wall circumferential stress in a patch at the carotid bulb to that in a vein used as a tube bypass:	
Circular cross-section	2.1 ± 0.33
Elliptical cross-section	3.1 ± 0.36

the artery and the vein were to develop myointimal hyperplasia sufficient to occlude the nonpatched artery, there would be only a 50% area stenosis in the patched segment.

Blood-flow characteristics are believed to play a major role in the development of atherosclerosis, myointimal proliferation, and platelet activation. Because of the increase in cross-sectional flow area provided by VPA, this technique has the theoretical advantage of reducing wall shear stress as much as fivefold compared to primary arteriotomy closure. Thus, the maintenance of predominantly laminar flow through the patched segment, including the boundary layer, is more likely after VPA. Boundary layer separation, with local turbulence, has been shown to occur at high flow velocities, particularly on rough surfaces such as those present in nonendothelialized endarterectomized carotid arteries.[15] Boundary layer separation has also been implicated as a stimulator of platelet agglutination and fibroblast activity.[18] Although these issues are not yet well understood and the calculated shear stresses reported herein are only simplified estimates, their importance makes them worthy of discussion.

Fry[19] demonstrated that severe wall shear stress, at high flows, can cause endothelial damage, but this finding has subsequently been disputed.[16] Such stress, however, could be an etiologic factor in both early postoperative thrombosis and restenosis. Because the ICA has a relatively high normal blood-flow rate, primary closure of a small ICA or an endarterectomized ICA above the bulb may result in high wall shear stress. Furthermore, high shear stresses may cause platelet activation, which in theory could lead to atherosclerosis and, more important in this setting, thrombosis. Interestingly, approximately 50% of the endarterectomies subjected to primary closure show early evidence of significant flow disturbance, consistent with boundary layer separation and local turbulence, on Doppler ultrasound examination.[20] In contrast, low wall shear stress in the carotid bulb has also been shown to produce boundary layer separation and perhaps to play a role in atherogenesis at this site.[21]

The author's method of estimating the magnitude of wall shear stress on the basis of continuous-wave Doppler velocity tracings is subject to criticism. The peak flow velocity during the cardiac cycle is not directly known, but, with the Doppler probe held at an approximately 45° angle to the flow axis over both the distal ICA and the patched segment, the ratio of the two maximum velocities should represent a reasonably accurate generalized estimate of the wall shear stress ratio. The assumption that the velocity profile at peak flow is blunted or "slug flow," thereby allowing an estimate of wall shear stress, is based on known in vivo velocity profile measurements of pulsatile blood flow.[15,19,21] The role of wall shear stress in the development of hyperplasia and atherosclerosis is complex, poorly understood, and probably dependent on the analysis of local boundary layer characteristics. The simplistic approach used in this study suggests that VPA is associated with a three- to fivefold reduction in maximum global ICA wall shear stress.

An interesting correlation can be made between the geometric findings in this study and the geometric characteristics of a normal ICA bulb. On the basis of radiographic measurements of an average carotid bulb diameter of 7.2 mm and an ICA diameter of 4.3 mm, the ratio between the cross-sectional area of the normal carotid bulb and that of the ICA is calculated to be 2.8. This value is similar to the mean ratio between the area of the patched segment and that of the ICA, which was determined to be 2.9. The blood-flow velocity ratios are identical. Because only 25% to 30% of the common carotid artery flow normally enters the external carotid artery, the blood-flow velocity must be significantly lower in the normal ICA bulb than in the more distal ICA. Saphenous VPA, as described herein, extends the normal-sized carotid bulb up the ICA above the endarterectomized segment, thus converting the hemodynamics of this segment into those of the normal bulb rather than those of the smaller distal segment.

The calculated circumferential wall normal stress in a saphenous vein segment used as a carotid patch is approximately twice what it would be in the same segment if it were used for a bypass graft at the same arterial pressure. The circumferential wall stress is maximal in the bulb segment at the caudad end of the patch, where the radius of curvature is greatest. This site of maximal stress is probably just caudad to the external carotid orifice, where the cross-section is slightly elliptical. Compared to the circumferential wall stress in a segment of greater saphenous vein used as a tube graft, the stress in a carotid vein patch may be three times as high. There are, however, other surgical procedures in which a vein graft may have a segment with a comparable or larger radius of curvature and therefore be subjected to a similar high circumferential wall stress. For example, the hood of a vein bypass placed on the ascending aorta or on a large common femoral artery has a large radius of curvature and hence incurs severe wall stress (probably more severe than that in a carotid patch). The elevated normal wall stress in the vein patch, however, may be a problem. Aneurysm formation has been reported after saphenous VPA in the carotid position,[13] but the incidence of this complication is unknown and is probably quite low. Patch rupture has also been reported; because of enhanced wall stress, such rupture may be a potential problem, particularly in thin or damaged veins.

REFERENCES

1. Zierler RE, Bandyk D, Thiele BL, Strandness DE. Carotid artery stenosis following endarterectomy. Arch Surg 1982; 117:1408-1415.
2. Thomas M, Otis SM, Rush M, Zyroff J, Dilley RB, Bernstein E. Recurrent carotid artery stenosis following endarterectomy. Am Surg 1984; 200:74-79.
3. Colgan MP, Kingston V, Shanik G. Stenosis following carotid endarterectomy. Arch Surg 1984; 119:1033-1035.
4. Gonzalez LL, Partusch L, Wirth P. Noninvasive carotid artery evaluation following endarterectomy. J Vasc Surg 1984; 1:403-408.
5. O'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. J Vasc Surg 1985; 2:26-41.
6. Baker WH, Hayes AC, Mahler D, Littooy FN. Durability of carotid endarterectomy. Surgery 1983; 94:112-115.
7. Salvian A, Baker JD, Machleder HI. Cause and noninvasive detection of restenosis after carotid endarterectomy. Am J Surg 1983; 146:29-34.
8. Norrving B, Nilsson B, Olsson JE. Progression of carotid disease after endarterectomy. Ann Neurol 1982; 12:548-552.
9. Ott DA, Cooley DA, Chapa L, Coelho A. Carotid endarterectomy without temporary intraluminal shunt. Ann Surg 1980; 191:708-714.
10. Deriu GP, Ballotta E, Banavina L, Grego F, Alvino S, Franceschi L, Meneghetti G, Saia A. The rationale for patch-graft angioplasty after carotid endarterectomy: Early and long-term follow-up. Stroke 1984; 15:972-979.
11. Little JR, Bryerton BS, Furlan AJ. Saphenous vein patch in carotid endarterectomy. J Neurosurg 1984; 61:743-747.
12. Sundt TM, Houser OW, Whisnant JP, Fode NC. Correlation of postoperative and two-year follow-up angiography with neurological function in 99 carotid endarterectomies in 85 consecutive patients. Ann Surg 1986; 203:90-100.
13. Rosenman J, Edwards WS, Robillard D, Geary G. Carotid arterial bifurcation advancement. Surg Gynecol Obstet 1948; 159:260-264.
14. Archie JP. Prevention of early restenosis and thrombosis-occlusion after carotid endarterectomy by saphenous vein patch angioplasty. Stroke 1986; 17:901-905.
15. Schlichting H. *Boundary Layer Theory.* New York, McGraw-Hill, 1960.
16. McDonald DA. *Blood Flow in Arteries*. Baltimore, Williams and Wilkins, 1974.
17. Douglas RA. *Introduction to Solid Mechanics*. Belmont CA, Wadsworth, 1963.
18. Brown CH, Leverett LB, Lewis CW, Alfrey CP, Hellums JD. Morphological, biochemical and functional changes in human platelets subjected to shear stress. J Lab Clin Med 1975; 86:462-471.
19. Fry DL. Acute vascular endothelial changes associated with increased blood velocity gradients. Circ Res 1968; 22:165-167.
20. Bodily KC, Zierler RE, Marinelli MR, Thiele BL, Green PM, Strandness DE. Flow disturbances following carotid endarterectomy. Surg Gynecol Obstet 1980; 151:77-80.
21. LoGerfo FW, Crawshaw HM, Nowak M, Serrallach E, Quist WC, Vareri R. Effect of flow split on separation and stagnation in a model vascular bifurcation. Stroke 1981; 12:660-665.