Review Article

Principles of preparation of vein bypass grafts to maximize patency

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Experience in cardiovascular and peripheral vascular surgery with saphenous vein bypass conduits is reviewed. It is clear that meticulous technique and graft preparation are crucial to short-term and long-term patency. The risk of early thrombosis is related to damage to the graft's native intima, graft flow, and coagulability of the patient's blood. Attention to atraumatic harvesting techniques and perfection of anastomoses are crucial to minimizing intimal damage. Graft inflow and outflow are fundamental principles. The use of vitamin K antagonists and platelet inhibitors may improve graft survival. Subacute occlusion is related to structural alterations in the grafts themselves. These include intimal hyperplasia and medial fibrosis as the grafts become "arterialized," valve fibrosis, aneurysmal dilatation, clamp stenosis, and suture stenosis. Long-term patency is threatened primarily by atherosclerosis in the graft itself. There is some evidence that care in vein harvesting and implantation as well as the use of anticoagulant agents affect the development of this complication.

A technique for graft preparation is presented that is based on the experience of the authors in harvesting grafts for both cerebral and coronary bypass conduits.

KEY WORDS □9 vein graft □9 saphenous vein □9 vascular anastomosis □9 graft patency □9 atherosclerosis □9 surgical technique

SAPHEOUS vein autografting is a mainstay of modern peripheral vascular and cardiac surgery. Recently, there has been interest in applying this technique to the treatment of cerebrovascular disease. In an effort to optimize results in this area, it seems wise to review what has been learned thus far about the saphenous vein as a bypass conduit.

The principles of vascular anastomosis were established at the turn of the century by Carrel and Guthrie. Shortly thereafter, Goyanes used a segment of popliteal vein to repair a popliteal artery after aneurysmectomy. However, it was not until after World War II, with the development of modern surgical materials and angiographic techniques, that the field of vascular surgery would begin its explosive growth. In 1949, Kunlin performed the first saphenous vein bypass graft for femoral artery occlusion and, in 1955, Linton reported his early results for saphenous vein femoropopliteal reconstructions. Application of this technique to the coronary circulation soon followed. In 1974, Sabiston reported an unsuccessful aortocoronary saphenous vein graft performed 12 years previously and, in 1973, Garrett, et al., published a 7-year follow-up angiographic study demonstrating the patency of an aortocoronary graft performed in 1964. Despite these early efforts, credit for the popularization of saphenous vein autografting in the coronary circulation must go to Favaloro, who in 1968 published his results with the first series of such grafts. Since that time, coronary artery bypass grafting has become one of the most common major operations performed in the United States.

With the wide use of the saphenous vein as a bypass conduit, a great deal of clinical and experimental data have been accumulated regarding the factors contrib-
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...utating to the patency of grafts as well as the angiographic and histological changes that they undergo after implantation. Our purpose here is to briefly review some of these data in a fashion that will be useful to the neurosurgeon in implanting the saphenous vein as a bypass conduit to the cerebral vessels. We will review the technical considerations important in the harvesting, preparation, and insertion of the graft, as well as host considerations such as recipient vessel diameter, intraoperative graft flow rates, and the patient's own state of coagulability, all of which seem to play a crucial role in the first hours, days, and weeks after surgery.

During the first year or two after implantation, graft fibrosis and intimal hyperplasia secondary to arterial hemodynamics and tissue reaction to the trauma imparted during harvesting become significant causes of graft failure. After the first 3 years, progression of atherosclerosis in the native vessels as well as the appearance of atherosclerosis in the grafts themselves play a role in determining patency. Each of these phases of occlusion will be discussed in turn with emphasis on factors contributing to each and means of minimizing them.

Normal Venous Histology

Before appreciating the pathological changes observed in vein grafts after implantation, one must review the normal histology. Veins, like arteries, have three layers: tunica intima, tunica media, and tunica adventitia. In general, the boundaries between these layers are much less distinct than in arteries and the elastic tissues are less well developed. There is in fact a great deal of variation in structure among veins. The saphenous vein is uniquely thick-walled and both muscular and elastic by virtue of the hemodynamic stresses to which it is normally subjected. Its intima consists of a flat endothelium with delicate underlying smooth-muscle fibers and interlacing collagen and elastin. The intima is separated by an elastic lamina from the media, which in turn consists of an inner longitudinal and outer circular layer of muscle fibers in bundles separated from one another by collagen and elastic fibers. In the region of the valves the former is more developed, while the latter assumes prominence between valves. Finally, the thick adventitia consists of loose connective tissue fibers, scattered fascicles of smooth muscle, and a rich complex of vasa vasmorum sending penetrating vessels into the outer layers of the media.

Early Graft Thrombosis

Graft occlusion in the first few days or weeks following implantation is most often due to thrombosis. The factors contributing to thrombosis are those that promote fibrin and platelet deposition on the graft wall: these include intimal desquamation with both the loss of its protective fibrinolytic activity and exposure of the underlying thrombogenic collagen fibers, slow graft flow, and the coagulability of the patient's blood.

Loss of Intima

Intimal desquamation may be considered as secondary to two types of trauma: one type is the unavoidable shear placed on the graft as it is transplanted to a high-pressure and high-flow arterial environment. This is largely unalterable, although it should be considered as one fashions the proximal and distal anastomoses to minimize local turbulence. The second contributor to endothelial cell loss is trauma during harvesting.

It is crucial that mechanical trauma to the vein be minimized. Fibrin microthrombi can be demonstrated at the sites of trauma within hours of implantation. For this reason, delicate noncrushing vascular clamps and vascular forceps with rows of fine, interdigitating, nonslipping teeth should always be used. Care must be taken to grasp only the adventitia when possible, and the vein should never be stretched longitudinally.

During harvesting and preparation of the vein, it is common practice to distend the vein under pressure to search for constricting bands and leaks from overlooked tributaries. The optimal solution for distension as well as storage of the vein before insertion is much debated. One study of canine cephalic vein by light microscopy and scanning electron microscopy (SEM) demonstrated more endothelial damage when using normal saline as opposed to whole blood. Another study of canine cephalic vein by light microscopy and transmission electron microscopy (TEM) as well as SEM, however, showed more spasm in veins stored in blood than in plasmolyte. Similarly, a study of human saphenous veins for aortocoronary grafting by Catinella, et al., used TEM and SEM to demonstrate more spasm with endothelial desquamation and fibrin and platelet microaggregate formation when the tissue was stored in blood than in plasmolyte. Venous spasm caused protrusion of endothelial cells into the lumen and the formation of subendothelial extensions of medial smooth-muscle cells which lifted the overlying endothelial cells and exposed the thrombogenic subendothelium. Clinical correlation was provided by postoperative angiography demonstrating improved graft patency at 10 to 14 days from 79.3% to 92.9% when plasmolyte was used rather than blood. The addition of papaverine to the storage solution has been shown to prevent the spasm, but it renders the vein walls more sensitive to injury by distention under pressure. Abbott, et al., found that a balanced salt solution with 10% serum buffered to a pH of 7.0 and held at 4°C best preserved the natural stress/strain characteristics of the vein itself.

The above studies notwithstanding, mechanical trauma is probably of greater significance than the specific storage solution used. An experimental study demonstrated that in situ perfusion with normal saline caused no vein wall alterations, whereas dissection and storage for 90 minutes caused loss of 50% to 60% of the endothelial lining.

Like the mechanical trauma of harvesting, the distention pressure on the vein is probably of greater impor-
tance than the storage solution used. A comparison of heparinized blood and heparinized cardiopulmonary solution used to distend veins at low pressures compared with heparinized saline used at high pressures showed no morphological difference in the low-pressure group and massive endothelial cell disruption in the veins distended at high pressures. Distention under high pressure also causes the post-implantation endothelial losses that result from arterial hemodynamics to occur more rapidly and more extensively. Finally, a study of canine jugular veins demonstrated that distention with as little as 50-torr pressure caused a 40% decrease in fibrinolytic activity. Increases thereafter in distention pressure to 500 torr did not significantly alter fibrinolytic activity, although pressures of 700 torr caused a further 10% drop. Studies of fibrinolytic activity in these grafts after arterial implantation demonstrated that, although all veins lost their activity within the first 12 to 24 hours, activity in the distended veins was lost faster. Histological examination of the grafts demonstrated that endothelial cell flattening began at pressures as low as 50 torr and progressed in a linear fashion to almost total endothelial cell loss at 700 torr. Smooth muscle was exposed to the lumen at pressures exceeding 500 torr.

Graft Flow and Recipient Vessels

In peripheral vascular surgery, it is generally held that "inflow" and "outflow" are crucial determinants of graft patency. The importance of freedom from hemodynamically significant proximal disease affording good inflow with an adequate pressure head would seem obvious, but in practice this principle is not always strictly observed. This key variable is, of course, essentially eliminated in aortocoronary graft surgery where the source of inflow is the ascending aorta. One would expect outflow to be related to the number and diameter of distal vessels as suggested by Poiseuille's law for streamlined flow (F) of viscous fluids through tubes:

\[ F = \frac{\Delta P \pi R^4}{8 L \nu}, \]

where \( P \) is pressure, \( R \) is the radius of the tube, \( L \) is its length, and \( \nu \) is fluid viscosity. Although Darling and Linton did not find femoropopliteal graft occlusion related to the severity of distal disease in their series, DeWeese and Rob, in a 10-year follow-up study, found patency of their grafts to be related not only to the severity of distal atherosclerotic disease and the number of vessels available for runoff, but also to the length of the graft as one might predict from the above equation.

<table>
<thead>
<tr>
<th>Authors &amp; Year</th>
<th>Measured Flow (cc/min)</th>
<th>Follow-Up Period</th>
<th>Patency Rate (%)</th>
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<tr>
<td>Grondin, et al., 1971</td>
<td>&lt; 25</td>
<td>10-21 days</td>
<td>0</td>
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<tr>
<td>Walker, et al., 1972</td>
<td>&lt; 20</td>
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<td>50</td>
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<td></td>
<td>70</td>
</tr>
<tr>
<td></td>
<td>&gt; 40</td>
<td></td>
<td>90</td>
</tr>
<tr>
<td>Marco, et al., 1976</td>
<td>&lt; 40</td>
<td>1 wk</td>
<td>75</td>
</tr>
<tr>
<td></td>
<td>&gt; 40</td>
<td>1 yr</td>
<td>89</td>
</tr>
<tr>
<td></td>
<td>&gt; 40</td>
<td></td>
<td>54</td>
</tr>
<tr>
<td>Björk, et al., 1981</td>
<td>&lt; 20</td>
<td>2 wks</td>
<td>59</td>
</tr>
<tr>
<td></td>
<td>25-60</td>
<td></td>
<td>93.2</td>
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<tr>
<td></td>
<td>&gt; 65</td>
<td></td>
<td>97.3</td>
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<tr>
<td></td>
<td>&lt; 20</td>
<td>1 yr</td>
<td>66.7*</td>
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<tr>
<td></td>
<td>25-60</td>
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<td>78.6*</td>
</tr>
<tr>
<td></td>
<td>&gt; 65</td>
<td></td>
<td>84.6*</td>
</tr>
<tr>
<td>Gohlke, et al., 1981</td>
<td>&lt; 50</td>
<td>8 wks</td>
<td>70-75</td>
</tr>
<tr>
<td></td>
<td>&gt; 90</td>
<td></td>
<td>100</td>
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* Percentage of grafts patent at 2 weeks.

<table>
<thead>
<tr>
<th>Authors &amp; Year</th>
<th>Diameter of Coronary Vessel (mm)</th>
<th>Follow-Up Period</th>
<th>Patency Rate (%)</th>
</tr>
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<tr>
<td>Roth, et al., 1978</td>
<td>≤ 1.5</td>
<td>1 yr</td>
<td>65</td>
</tr>
<tr>
<td>BJörk, et al., 1981</td>
<td>&gt; 1.5</td>
<td></td>
<td>90</td>
</tr>
<tr>
<td></td>
<td>≥ 1.5</td>
<td>2 wks</td>
<td>70</td>
</tr>
<tr>
<td></td>
<td>≥ 1.5</td>
<td>1 yr</td>
<td>96.4</td>
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<tr>
<td></td>
<td>≥ 1.5</td>
<td></td>
<td>84.6*</td>
</tr>
<tr>
<td>Gohlke, et al., 1981</td>
<td>&lt; 1.8</td>
<td>8 wks</td>
<td>83.3-81.3†</td>
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<tr>
<td></td>
<td>&gt; 1.8</td>
<td></td>
<td>91.2-83.8†</td>
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* Percentage of grafts patent at 2 weeks.
† A higher patency rate was associated with postoperative anticoagulation therapy.

Similarly, aortocoronary graft outflow should be related to runoff vessel diameter. Studies of measured flows at surgery and of measured coronary vessel diameter and patency have been performed (Tables 1 and 2). Clearly, flows greater than 40 cc/min and coronary vessel diameters greater than 1.5 mm are associated with higher patency rates. Grondin, et al., found that all of the occluded grafts in their series had flows of less than 45 cc/min. Bourassa, et al., reported that, of the 13% of grafts occluded at 2 weeks in their study, 94% were to vessels 1.5 mm or less in diameter. This study also showed that the significance of recipient vessel diameter as compared with other factors diminishes with time. Of the 32% of grafts occluded at 1 year, only 30% were to vessels 1.5 mm or less in diameter. Similarly, Björk, et al., found that the 1-year patency rates of grafts known to be patent at 1 week were unrelated to coronary vessel diameter or measured graft flow. Marco, et al., echoed this finding in de-
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scribing no significant effect of flows greater or less than 40 cc/min on occlusions after the first week. Finally, it is interesting to note that sequential grafts (those with more than one distal anastomosis) have higher patency rates than have single grafts. This may well be related to improved outflow from the graft.

Host Coagulability

The observation that patients with decreased platelet survival time have a higher risk of aortocoronary graft thrombosis in the first few years postoperatively has led to trials of anticoagulation therapy. Postoperative phenprocoumon, a vitamin K antagonist, has been shown to increase aortocoronary graft patency at 8 weeks from 83.6% to 90.4%, Chesebro, et al., have taken this logic a step farther with aspirin and dipyridamole, the latter actually being started preoperatively, with improvement in patency at 1 month from 90% to 97% and at 6 months from 85% to 96%. The long-term effects of antiplatelet therapy are not yet known. Many believe that the first step in atherogenesis is endothelial injury with platelet adhesion and release of granules containing mitogenic factors causing smooth-muscle proliferation and the formation of a connective tissue matrix with lipid deposition. If this is the case, antiplatelet therapy in the first few weeks after surgery may have far-reaching effects by reducing later occlusion secondary to atherosclerosis. In support of this, therapy with aspirin and dipyridamole has been shown to prevent lipid accumulation in cephalic vein grafts to the femoral arteries in normolipemic monkeys and to significantly reduce it in hyperlipemic animals.

Subacute Graft Occlusion

Reendothelialization of vein grafts in the canine model takes from 6 to 12 weeks. It is probably largely for this reason that between one- and two-thirds of aortocoronary vein graft occlusions occur in the first month. Thereafter, vein grafts begin to undergo structural changes in response to arterial hemodynamics and as a consequence of the trauma they underwent during harvesting. Kern, et al., demonstrated by light microscopy and TEM that aortocoronary grafts universally undergo fibrotic changes (or "arterialization") by the end of the first month, which are most prominent in the subintima and appear similar to those seen in arteriovenous fistulae. Szilagyi, et al., performed an extensive study of the angiographic and histological changes observed in 316 femoropopliteal grafts. They identified on angiography six morphological changes which represented threats to the patency of vein grafts (Fig. 1). These changes were followed by serial angiography and, as cases came to reoperation or postmortem examination, they were studied histologically.

Of these six morphological changes, five occurred with a mean time of onset in the first 1 to 2 years and as such represent subacute changes. Atherosclerotic change appeared with a mean onset time of almost 4 years and as such may be considered a chronic change. This is discussed in the section entitled Late Occlusion. The other structural alterations are discussed in this section.

Intimal Hyperplasia and Medial Fibrosis

The most common morphological alterations are the diffuse changes of "arterialization." Alterations in the media begin as early as the first week after graft implantation, with smooth-muscle necrosis and mural edema followed by hypertrophy of some smooth-muscle cells and fibroblastic transformation of others. These changes appear to be primarily due to medial ischemia caused by the interruption of the vasa vasorum. Elastic-tissue hyperplasia is seen in the adventitia as well, but the greatest threat to graft patency is the intimal thickening often referred to as "intimal hyperplasia." It is identifiable by angiography in at least one-third of femoropopliteal grafts, and even more often in aortocoronary grafts. It is found by microscopic analysis in almost all grafts in place for more than 1 month. Scanning electron microscopy has demonstrated areas of intact endothelium as well as areas of
focal endothelial loss and fibrin layering overlying fibroblast and smooth-muscle proliferation. These cellular elements are separated by a mucopolysaccharide ground substance and collagen fibers.

Sottiurai, et al., examined the distal anastomoses of thrombosed grafts and found intimal hyperplasia most prominent at the floor of the artery and the heel of the graft (Fig. 2). They suggested that the smooth-muscle cells normally present in the subendothelium undergo transformation to one of two observed cell types: 1) a degenerate cell with fatty infiltration, cholesterol droplets, a paucity of myofilaments, and decreased perinuclear organs surrounding pyknotic and hyperchromatic nuclei, or 2) myofibroblasts responsible for laying down the extracellular matrix. Factors thought to contribute to intimal hyperplasia included mismatch of the mechanical properties of the graft and the recipient vessel and local turbulence. Brody, et al., conducted an elegant set of experiments demonstrating that, in the canine model, intimal hyperplasia is related more strongly to arterial hemodynamics than to the trauma of local dissection and consequent ischemia, whereas the converse is true for medial fibrosis. Many argue that intimal damage with local platelet aggregation and release of mitogenic factors is the key step in the development of intimal hyperplasia and, subsequently, of graft atherosclerosis. If this is true, platelet inhibitors should decrease or prevent intimal hyperplasia; dipyridamole and aspirin have been shown to decrease by 50% the degree of luminal narrowing caused by hyperplasia in the canine aortocoronary bypass model.

The natural history of intimal hyperplasia is unclear. Some believe that it is self-limited while others believe that it progresses over years even to graft occlusion in some cases. Some authors argue that the cellular elements are eventually lost as the subintimal layer becomes hyalinized, while others argue that intimal hyperplasia evolves into classical atherosclerosis.

Valve Fibrosis

The third most common change seen was valve fibrosis. Most valves lie flat against the vein wall, posing no threat to graft patency. When they undergo fibrotic change, however, they impinge on the vessel lumen, generating turbulence. It is for this reason that some authors advocate incising the valve leaflets with a valvulotome intending to make it easier for the bicuspid valves to lie flat. Baba, et al., however, demonstrated beneficial effects from valves, with blood flow through an aortocoronary graft in the canine model increasing by 11% when a competent valve is present, perhaps via reduction of systolic backflow. This action would not be significant in the peripheral circulation where reversal of arterial flow as observed in the coronary circulation does not occur.

Aneurysmal Dilatation

Angiographic evidence of aneurysmal dilatation was found associated with some grafts, and when a histological study was possible, microscopic evidence of atherosclerosis was also identified. Stanley, et al., found aneurysmal dilatations in 8% of their aortorenal grafts, but did not identify associated atherosclerosis. The etiology of such dilatations remains unclear. They are most common at the anastomatic site. Their significance to graft patency is unknown.

Clamp Stenosis

Stenotic regions with fibrotic wall thickening are identifiable several centimeters from the distal end of some grafts. These areas probably represent the long-term consequences of trauma to the vein imposed by occlusive clamps. This reinforces the importance of using delicate clamps at these sites.

Suture Stenosis

Finally, some stenoses occur at the site of suture ligation of venous tributaries. They are probably due to ligation too close to the main vessel.

Late Occlusion

Although some authors argue that intimal hyperplasia continues to play a significant role in causing graft occlusion 5 or more years postoperatively, most agree that atherosclerosis is the major cause of late occlusion. Long-term patency of femoropopliteal grafts is probably limited by atherosclerotic narrowing of the native circulation. Szilagyi, et al., found angiographic evidence of atherosclerosis in only 8% of femoropopliteal grafts studied. It was present on histological examination with lipid-infiltrated fibrotic plaques having necrotic centers lying between a collagen base and overlying ulcerated intima with fibrous clot in 80% of grafts in place over 2 years; however, it appeared to take 4 to 6 years to develop to a degree that threatened graft patency. DeWeese and Rob similarly reported angiographic evidence of atherosclerosis in only 6% of the femoropopliteal grafts studied at 5 years. At 10 years postoperatively, atherosclerotic plaque appeared in two of eight grafts. Their conclusion was that ath-
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eerosclerosis develops late in femoropopliteal grafts, if at all.

In contrast, atherosclerotic disease is much more common in aortocoronary grafts, and disease in the grafts themselves appears to be the dominant cause of late graft failure. Bulkley and Hutchins found fibrin thrombus and foam cells in virtually all grafts in place for over 1 month, and fully lipid-laden macrophages in over 50%. Campeau, et al., found angiographic evidence of atheromatous changes in 15.9% of grafts studied in the first 5 years after implantation. At repeat angiography between 6 and 10 years postoperatively, an additional 36.4% developed such changes, suggesting that over time atherosclerotic changes play an increasing role in graft occlusion. Lytle, et al., similarly found that postoperative vein graft attrition increased with time, with 13% of grafts studied in the first 5 years occluded and 26% of the grafts patent at an earlier study occluded at repeat angiography in the second 5 years.

Sziagyi, et al., suggested that the higher prevalence of atherosclerosis in aortocoronary grafts as opposed to femoropopliteal grafts may be due to the greater hemodynamic stresses, more restricted outflow, and less supportive tissue bed present in the former. These may all play a role in intimal damage and delayed repair with consequent platelet and fibrin deposition. Von Rokitansky argued that atheromas were derived from fibrin encrustation on the arterial wall, and many now believe that intimal damage with platelet adhesion and degradation with the release of mitogenic factors affecting subendothelial smooth muscle is the key step in atherogenesis. If this is true, then platelet antagonists may be helpful in preventing the late complication of graft atherosclerosis as well as in reducing early graft thrombosis. There is experimental evidence that aspirin prevents lipid accumulation by vein grafts. We await the long-term follow-up results of the aspirin and persantine trials with aortocoronary grafts.

It also seems likely that attention to serum cholesterol and triglycerides may improve graft patency. Although earlier studies failed to show a significant relationship between the traditionally recognized risk factors for atherosclerosis and femoropopliteal graft occlusion, Campeau, et al., in their long-term follow-up study, found aortocoronary graft occlusion to be correlated with high total cholesterol and high levels of the low-density lipoprotein apoprotein B. Lytle, et al., also found aortocoronary graft loss related to high cholesterol and triglyceride levels, and identified diabetes mellitus as a risk factor; however, neither group found hypertension or tobacco use to be correlated with increased risk of graft occlusion.

Overall Graft Patency

The validity of graft patency rates from various studies depends upon the statistical methods used to calculate those rates in each study. For example, some studies include follow-up angiography in all patients operated on, whereas in others only symptomatic patients underwent repeat study. One can get an idea of the relative rates of graft occlusion by considering the range of rates reported. Reported femoropopliteal graft occlusion rates for the first 1 to 3 years after implantation range between 20% and 37.4%. Five-year rates range from 32% to 42%, and at 10 years about 55% of the grafts are occluded. Aortocoronary grafts fare slightly better, with occlusion rates in patients evaluated in the first 2 weeks to 2 months ranging from 4% to 30%, depending on saphenous graft flow, recipient vessel diameter, use of anticoagulation or antiplatelet agents, and the technique of vein harvesting. Most studies report approximately 10% acute occlusion. At 1 year, occlusion rates range from 10% to 35%, with a yearly attrition rate of 2% to 3% thereafter. There is evidence, however, that graft atherosclerosis is a progressive disease with an incidence increasing with the postoperative interval. Yearly graft occlusion is increasingly common with time. Campeau, et al., similarly reported a yearly attenuation rate of 2.1% in the first 5 postoperative years, and 5.2% in the second 5 years. Lytle, et al., similarly calculated a 13% overall occlusion rate in the first 5 postoperative years, with an additional 26% of the grafts occluded in the second 5 years.

Preparation of the Vein

The above discussion should make it clear that the technical aspects of vein harvesting and preparation are crucial to the long-term success of saphenous vein bypass grafts in any location. On the neurosurgical service at the Mayo Clinic, a segment of the long saphenous vein is usually harvested from the distal part of the leg rather than from the thigh in order to achieve a better match between the diameter of the vein and that of the recipient vessel. The skin incision is made one finger-breadth anterior to the medial malleolus where the vein is readily identifiable (Fig. 3). The incision is then carried proximally as needed, taking care to remain directly over the vein in order to avoid creating any skin flap. Such flaps can slough and become major wound problems.

In our experience the vein, when distended, must measure at least 5 to 8 mm in diameter. We have found that smaller veins have had a high rate of occlusion. Veins larger than 1 cm in diameter are disproportionately large at the distal anastomosis and are theoretically more prone to thrombosis secondary to slower flow, as described by Poiseuille’s law. If the distal vein is too small or is unusable because of varicosities or other structural flaws, the proximal saphenous vein can be identified in the groin and the vein taken distally. The vein can be found near the fossa ovalis two finger-breadths lateral and two finger-breadths inferior from the lateral margin of the pubic tubercle. The incision is made from this part distally, aiming for the medial aspect of the tibial plateau. The vein should always be followed back to the fossa to confirm its identity.

After the skin has been incised, a delicate layer of
FIG. 3. The great saphenous vein is harvested from the leg or thigh. There is considerable variability from patient to patient in the size of this vessel and in the number of branches. A: Small branches are ligated with 5-0 Prolene stick-ties and large branches with 3-0 or 4-0 silk free ties. Sometimes it is preferable to close large tributary vessels with a running 5-0 suture rather than ligate the vessel with a larger suture as the simple ligation with a tie sometimes distorts the lumen of the vein. B: The Garrett orientation line should be placed in the adventitia of the vein before it is harvested as there is frequently 360° to 720° rotation of the vein with distention after it is harvested. The proper orientation at that time cannot be determined. C: The vein is distended to 200 mm Hg with the Shiley distention kit. It is preferable to leave the vein in situ after the tributaries have been ligated and to harvest it only after exposure of the intracranial recipient vessel and the cervical carotid arteries.

FIG. 4. The vein should be harvested by incising the connective tissue surrounding the vessel several millimeters from the wall. This minimizes the handling of the vein itself, and therefore limits both trauma and vasospasm. This technique also provides adequate room for subsequently ligating any small branches that are missed while explanting the vessel.

areolar tissue can be identified overlying the vein. This tissue can be torn away from the vein by using the scissors with a spreading motion. While this is no doubt the quickest way to isolate the vein, one runs the risk of avulsing the tiny branches that lie within this layer. It is preferable to incise this tissue sharply 0.5 cm from the vein wall (Fig. 4). This will leave “stumps” of these small branches which are of sufficient length to be ligated after the vein has been removed from the leg. Large branches are ligated doubly with 3-0 or 4-0 silk free ties. Smaller branches may be similarly tied or closed with 5-0 Prolene stick-ties. Holes made by avulsed branches may be repaired with a 7-0 Prolene mattress stitch. If simple ligation of a particularly large tributary will inordinately distort the lumen, the junction can be oversewn with a running 5-0 or 7-0 Prolene suture.

Prior to removing the vein, a 5-0 Prolene suture is placed in the adventitia of the vessel as an orientation line. We refer to this as a Garrett line after the vascular surgeon from whom this technique was learned. This is an extraordinarily important step in the procedure, as a vein tends to rotate with distention after it is harvested from the leg and unless its proper orientation is identified in situ, there will be a rotation to the vein that cannot be corrected thereafter and which will predispose the vein to twisting and kinking. It is preferable to place the Garrett line before mobilizing and ligating the branches of the vein.

We prefer to mobilize the entire length of vein from one end to the other and then leave the vein in situ until it is ready to be transposed. In this manner, flow is preserved through the vein for as long as possible (the importance of which is indicated above in the discussion of causes for early graft failure). The vein is kept moist with saline packs.

At the time of harvesting, the vein is hydrodistended with a Shiley catheter distention system (Fig. 3). This system has a bulb which distends at 200 mm Hg and thus prevents overdistention and fracture of the vein wall from too much pressure. An early cause for graft occlusion in our series was overdistention of the vein. The vein is worked between the index finger and thumb until the spasm in the vein has been overcome. At this time, the vein can be carefully inspected for any unligated branches.
Preparation of vein bypass grafts

Conclusions
A great deal has been learned about the saphenous vein as a bypass conduit in peripheral vascular and cardiac surgery. Early graft failure is due to thrombosis which in turn is promoted by: 1) loss of the vein’s native intima; 2) slow graft flow; and 3) coagulation characteristics of the patient’s blood. Intimal desquamation can be minimized by fashioning proximal and distal anastomoses in such a way as to minimize turbulence, and by meticulousatraumatic technique while harvesting the vein. Graft flow must be optimized with attention to conduit diameter and the available inflow and outflow. One must select a recipient vessel of adequate diameter; that is, the graft must be anastomosed to a vessel such as the M segment of the middle cerebral artery or P segment of the posterior cerebral artery as more distal vessels will not provide adequate run-off. Anticoagulation with vitamin K antagonist and platelet inhibitors appears to improve graft survival. Subacute occlusions occur as a result of structural alterations in the vein itself, the most significant of which is intimal hyperplasia. This appears related to intimal trauma and desquamation with platelet deposition. It may be reduced by measures to minimize these processes. Graft failure several years after implantation is probably most often caused by the development of atherosclerosis in the veins themselves. The use of aspirin and dipyridamole may have an impact on this. The reduction of serum cholesterol and triglycerides should be pursued in an effort to reduce graft atherosclerosis.

It is difficult to predict the overall patency that can be expected from the saphenous vein as a bypass conduit to the cerebral circulation. It may approximate the 90% rate reported in some coronary studies. Therapy with antiplatelet agents may improve on this. The stable bed in the subcutaneous tissues of the head as compared with the constant movement present in the chest may further increase patency. In any case, meticulous care in harvesting the vein is a controllable variable that is crucial to success.

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