

Technical Factors In Enhanced Patency Rates In Aorto Coronary

R.L. Patel, B.T. Williams*

Coronary artery bypass grafting with autologous saphenous vein has a well established therapeutic role for both relief of symptoms and prolongation of life¹.

Graft occlusion occurs postoperatively and 15% of all grafts suffer thrombotic occlusions during the first 12 months². Subsequent failures occur at a rate of 2% per year owing to accelerated myointimal hyperplasia with or without superimposed artheroma^{2,3}. Patency rates quoted are from operations carried out during early days of coronary artery bypass surgery. These may not be applicable today since our understanding of various factors which promote graft occlusion are better.

A proportion of grafts occlude due to progression of the native coronary arterial disease but there are well established and avoidable causes of graft failure. Improving the long term patency rates of saphenous vein grafts is therefore an important and worthwhile goal. These will be discussed under the following headings:

1. Harvesting.
2. Preparation.
3. Anatomical construction of vein grafts.

Autologous materials used with proven good results include the long saphenous vein and the internal mammary artery. Both patency rates and life expectancy have been shown to be improved using internal mammary artery compared with saphenous vein grafts^{4,8}. Arm veins make a less satisfactory conduit than do saphenous veins¹⁶. Several substitutes for inadequate or missing saphenous veins and mammary

artery include saphenous allografts, dacron prosthesis, expanded polytetrafluoroethylene grafts, glutaraldehyde treated umbelical vein, gastro epiploic artery and radial artery¹⁷⁻²⁰. All of these are much less satisfactory than conventional materials.

1. Harvesting.

A series of well defined pathological and degenerative changes occur in saphenous vein grafts during and after implantation^{14,23}. Endothelial separation and partial desquamation with resulting exposure of basement membrane occur during surgical preparation^{11,13}. The resulting exposure of collagen promotes platelet activation and fibrin attachment^{14,15}. Endothelial damage also reduces production of prostacycline³, a powerful inhibitor of platelet aggregation, and this further promotes platelet activation. Damage of vein graft media is associated with leukocyte infiltration. Platelet activation may precipitate early thrombosis or lead to secretion of mitogens responsible for the rapid medial and neointimal proliferation that occurs in vein grafts during the first few months^{9,15}.

It is reasonable to suggest that methods of harvesting that produce vein with undamaged endothelium and media would enhance patency rates.

2. Preparation.

Various fluids have been used to prepare harvested vein. Heparinised blood is the most physiological. Use of saline (pH7.0) should be avoided. Crystalloid cardioplegic solutions injected into the vein grafts once the distal anastomosis has been constructed will cause endothelial loss. However addition of blood or

* St. Thomas' Hospital, London, U.K.

albumin solution to raise the oncotic pressure reduces endothelial damage²⁶. The use of metal bulldogs on grafts have also been shown to cause endothelial damage and for the same reasons passage of probes to dilate internal mammary artery should be avoided. Surgical trauma by axial and overdistention must be avoided to prevent endothelial loss^{10,13}.

Studies have identified distention of vein as a principal cause of medial damage^{13,21,22,24,25}. Overdistention results in acute morphological changes in both vein graft media and endothelium and to exacerbate later neointimal hyperplasia. Accumulation of cholesterol and apolipoprotein markers associated with vein graft arterhoma, has been shown to be greater in grafts distended to above normal arterial pressures²⁸.

To avoid overdistention an in line measurement of distending pressure by means of two-way tap attached to manometer line is used as shown in the following figure:

3. Operative factors

The anatomy of a saphenous vein bypass graft should be designed to minimise turbulence. The effects of turbulence is shown in the figure below:

Ideally, the site selected for arteriotomy should be on or near the epicardial surface, straight, not near to major branches, devoid of artheroma and greater than 1.5mm in diameter²⁷. Retracting or occluding suturs and bulldogs around artery should be avoided to prevent intimal fracture.

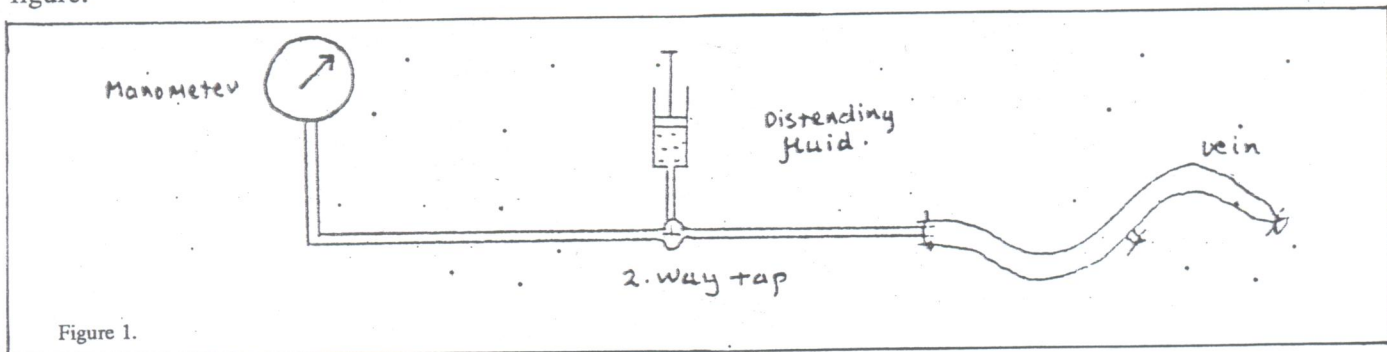


Figure 1.

Distending pressure should be below 102mm HG.

Extreme care must be taken to prevent localised areas of stenosis at the ligature sites or leave blind stumps to initiate clot formation.

Saphenous vein grafts are passive conduits having no elastic recoil and as the myocardial blood delivery

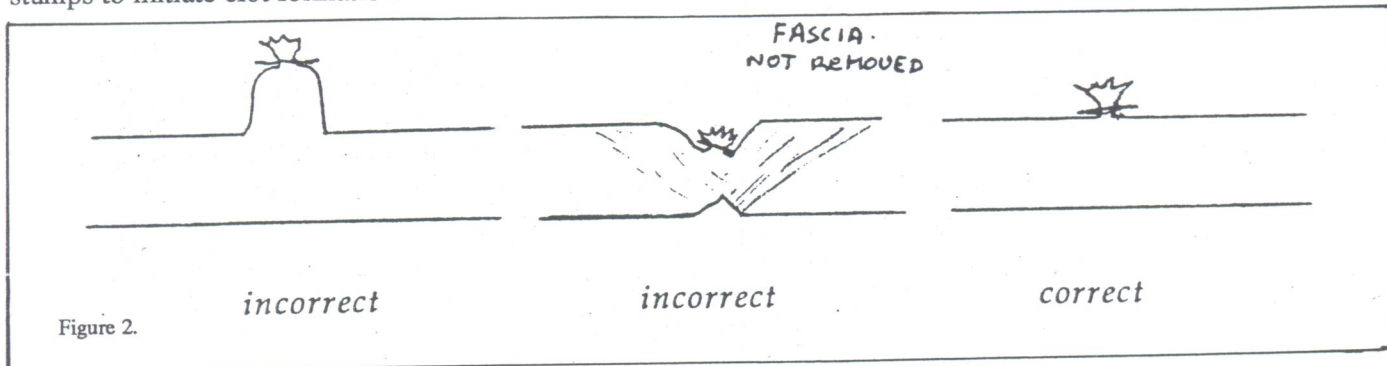
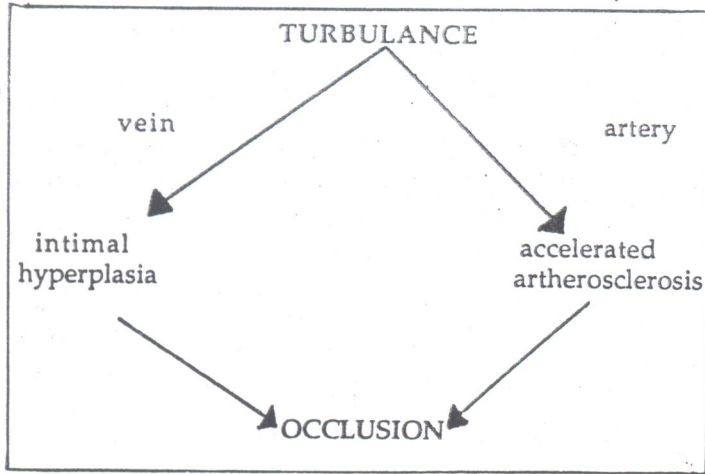


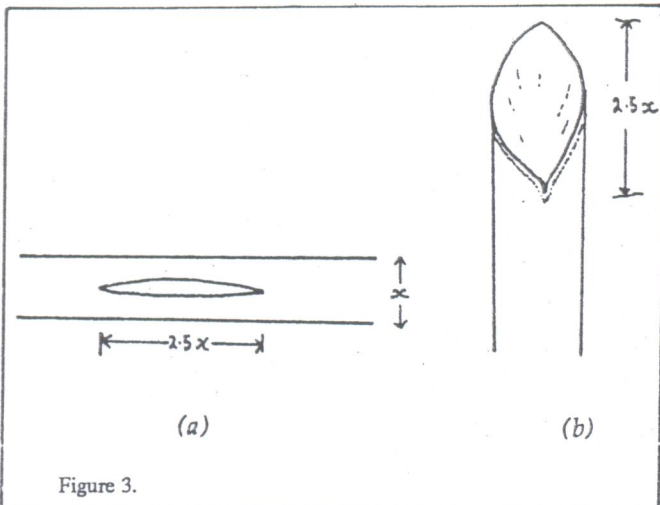
Figure 2.



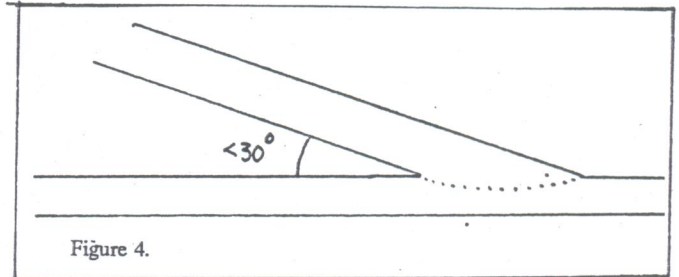
is in diastole it is important that the size of the graft is adequate to contain blood for the delivery during this phase of cycle. Hydraulic studies indicate it should be 2.5X the size of the coronary artery. This size also facilitates construction of anastomosis and handling of the graft.

The aims of constructing an anastomosis are to achieve minimal blood turbulence in the conduit and coronary artery without production of stenosis, to avoid undue tendency to localised thrombosis, and attain a blood-tight seal between graft and artery.

The length of the arteriotomy should be judged in relation to the size of the artery and vein. In order to promote turbulent free flow through the anastomoses it should be 2.5X the diameter of the artery. The vein is then beveled to accommodate the size of the arteriotomy.



If the vein is then anastomosed end to side, the angle between the coronary artery and the vein graft will be less than 30 degrees. This has been shown to produce least turbulence in the region of separated flows and distributes wall stress over a wider area, commencing opposite the entrance to the anastomotic site.



For the same reasons where an artery is located several millimeters below the epicardial surface, particularly where this is due to presence of fat, the artery should be made more accessible by incising into the fat over the vessel for a centimeter or two, in order to allow the graft to approach the artery more gradually, avoiding a near perpendicular approach of the graft to the artery.

Inadvertent production of a degree of stenosis in the grafted coronary artery is much easier with small calibre vessels, and usually takes the form of a stenosis at either extremity of the anastomosis, caused by sutures constricting the artery in this area. It is therefore important to see with precision just exactly where sutures are placed in these vulnerable areas. Both prograde and retrograde flow is necessary and as much care is required at the proximal end of the anastomosis as at the distal. It is essential to take small "bites" of both artery and vein.

Various suture techniques are advocated for achieving satisfactory anastomosis. Undoubtedly two suture techniques using double armed 6-0 or 7-0 prolene and commencing at separate ends will achieve the following:

- 1) Vulnerable points of anastomosis, i.e., "heel and toe" will be anastomosed under direct vision.
- 2) Precise eversion will be achieved of both graft and artery wall.

- 3) Size disparity will be avoided achieving a blood tight seal between graft and artery.
- 4) Lifting off minor plaques and fragmenting the wall of the artery when sutures are passed from inside to outside the artery will be avoided.
- 5) Using prolene takes advantage of the property of minimal tissue drag and allows ease with which the sutures will pull through.

The choice of size of the needle is more important than the size of the suture using the everting technique of anastomosis. Everting technique also voids exposure of collagen of graft and coronary artery wall which promote platelet activation and fibrin attachment^{14,15}.

Precise attention to the proximal anastomosis is equally important. Creation of a hood simulates a sinus, where like in the aortic sinus of valsalva turbulence is maximum but least as the blood is directed down the graft.

maximum. Due to the angle of impact the flow is converted to turbulent flow adding further stress to the graft wall down stream, thus promoting intimal proliferation and graft atherosclerosis. Flow pattern as shown in (b) is preferable.

Creation of a "sinus" demands precise geometric orientation of the take-off of the graft from the aorta to avoid kinks which not only stenose the graft but increases turbulence resulting in early graft disease.

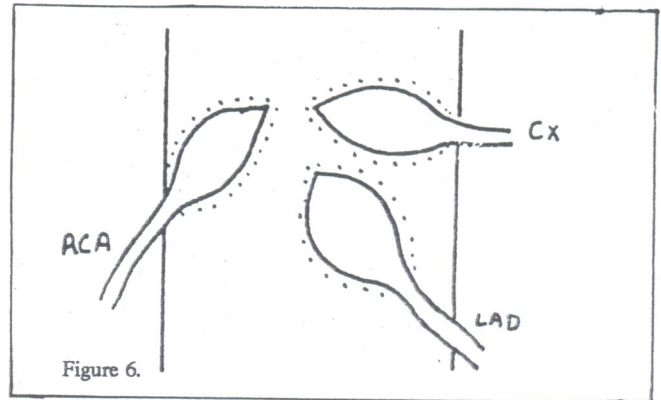
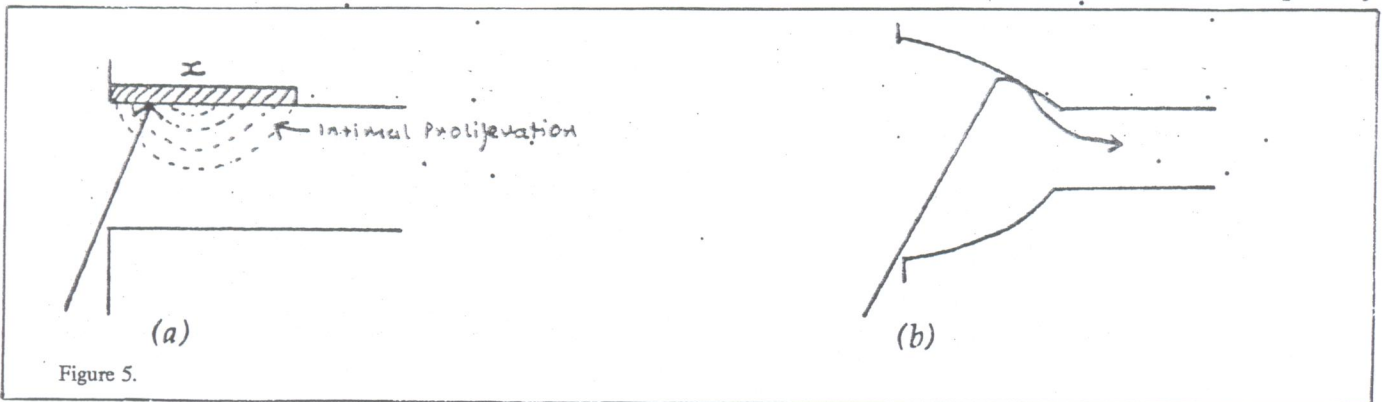


Figure 6. Accurate alignment of graft is important. Redundant length will undoubtedly create kinks especially



As shown (a) the point of impact of the blood stream is in area marked X. It is at this point (up to 1 centimeter from the origin) that intimal proliferation is

when the chest is closed. This may completely stop flow resulting in graft thrombosis or lead to accelerated myointimal proliferation. Too short a graft will

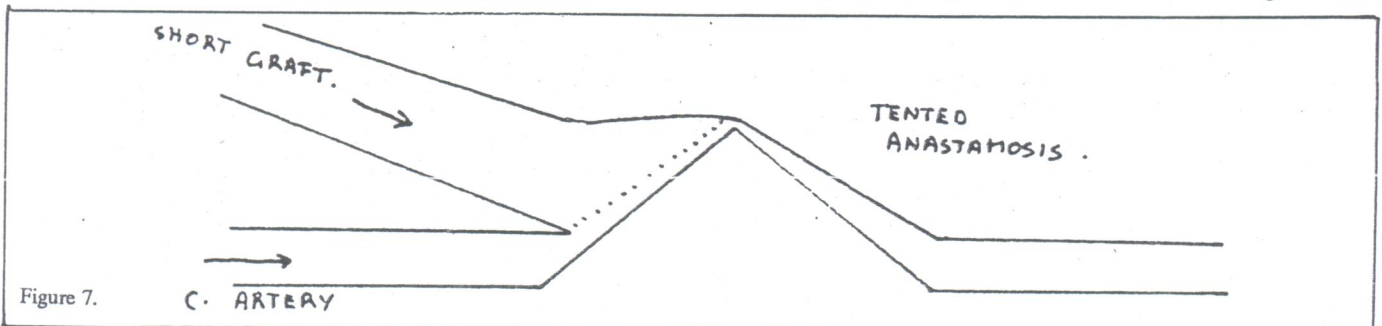


Figure 7.

tent the distal anastomosis and apart from disruption of the suture line, it may angulate the coronary artery and lead to poor run off.

Closure of pericardium will highlight minor mis-judgments and therefore not recommended.

The following figure in summary shows the creation of an anatomically ideal saphenous vein graft:

Though a proportion of graft failure is attributable to poor run off due to progression of native coronary arterial disease, attention to the above factors will undoubtedly prolong graft patency by preventing both early and late graft occlusion by thrombosis or accelerated intimal proliferation complicated by arteroma. These factors are avoidable and careful detail at each step is necessary. This may well reduce the need for redo-coronary artery bypass grafting.

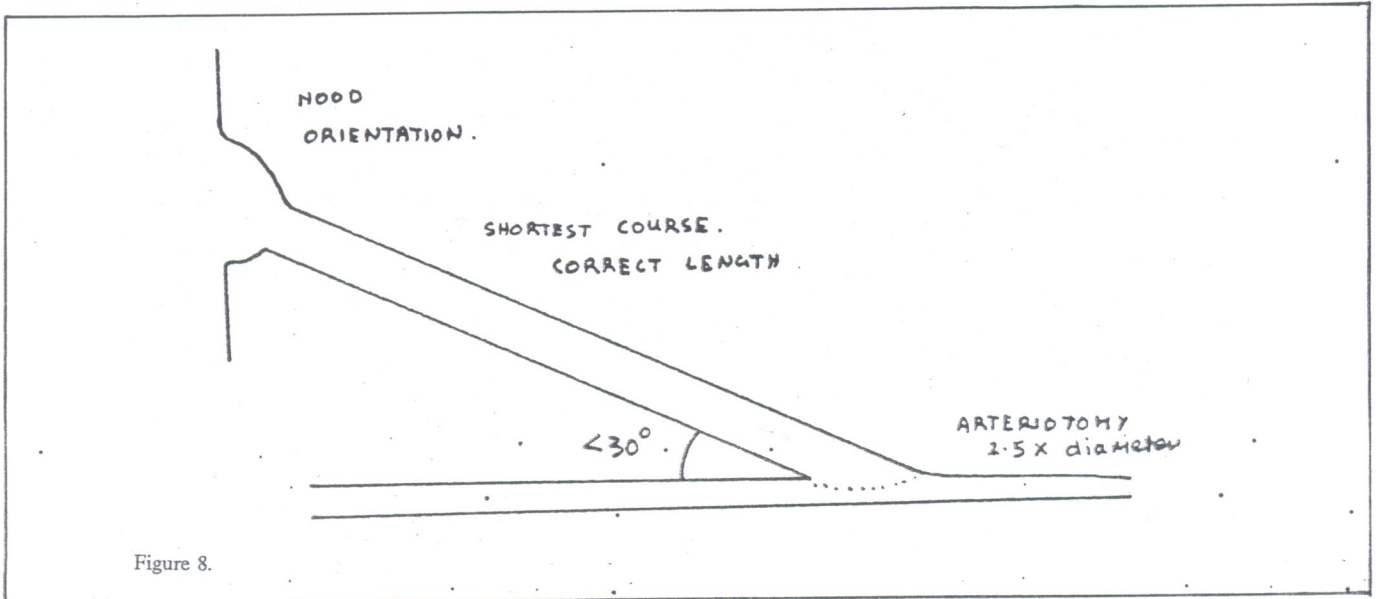


Figure 8.

REFERENCES:

1. Julian DG. The practical implications of the coronary artery surgery trials. *Br Heart J* 1985; 54: 343-50.
2. Grondin CM. Late results of coronary artery grafting. Is there a flag on the field? *J Thorac Cardiovasc Surg* 1984; 87: 161-6.
3. Angelini GD, Brekenridge IM, Psaila JV, Williams HM, Henderson AH, Newby AC. Preparation of human saphenous vein for coronary artery bypass grafting impairs its capacity to produce prostacyclin. *Cardiovasc Res* 1987; 21: 28-33.
4. Acinapya AJ, Rose DM, Jacobvowitz IJ, Kramer MD, Cunningham JN, et al. Internal Mammary artery bypass grafting: Influence on recurrent angina and survival in 2,100 patients. *Ann Thorac Surg* 1989; 48: 186-91.
5. Barner HB, Swartz MT, Mudd JG, Tyras DH. Late patency of the internal mammary artery as a coronary bypass conduit. *Ann Thorac Surg* 1982; 34: 408-12.
6. Tector AJ, Schmahl TM, Janson B, Kallies JR, Johnson G. The internal mammary artery graft: its longevity after coronary bypass. *JAMA* 1981; 246: 2181-3.
7. Grondin CM, Campeau L, Lesperance J, Enjalbert M, Bourassa MG. Comparison of late changes in internal mammary artery and saphenous vein grafts in two consecutive series of patients 10 years after operation. *Circulation* 1984; 70 (Pt 2): 1208-12.
8. Lytle BW, Loop FD, Cosgrove DM, Ratliff NB, Easley K, Taylor PC. Long-term (5 to 12 years) serial studies of internal mammary artery and saphenous vein coronary bypass grafts. *J Thorac Cardiovasc Surg* 1985; 89: 248-58.
9. Fuster V, Chesebro JJ. Aortocoronary artery vein graft disease: experimental and clinical approach for the understanding of the role of platelets and platelet inhibitors. *Circulation* 1985; 72 (pt 2): V65-70.
10. Gundry SR, Jones M, Ishihara T, Ferrans VJ. Optimal preparation techniques for human saphenous vein grafts. *Surgery* 1980; 88: 785-94.

11. Hasse J, Graedel E, Hofer H, Guggenheim R, Amsler B, Mihatsch MJ. Morphological studies in saphenous vein grafts for aortocoronary bypass surgery. *Thorac Cardiovasc Surg* 1981; 29: 38-40.
12. Abbot WH, Wieland S, Austen WG. Structural changes during preparation of Autogenous venous grafts. *Surgery* 1974; 76: 1031-40.
13. Storm FK, Gierson ED, Sparks FC, Barker WF. Autogenous vein bypass grafts: Biological effects of mechanical dilatation and adventitial stripping in dogs. *Surgery* 1975; 77: 261-7.
14. Unni KK, Kottke BA, Titus JC, Frye RL, Wallace RB, Brown AI. Pathological changes in aortocoronary saphenous vein grafts. *Am J Cardiol* 1974; 34: 526-32.
15. Ross R. The pathogenesis of atherosclerosis: an update. *N Engl J Med*. 1986; 314: 488-500.
16. Stipa S. The cephalic and basilic veins in peripheral arterial reconstruction. *Ann Surg* 1972; 175: 581-6.
17. Tice DA, Zerbino VR, Isom OW, et al. Coronary artery bypass with freeze preserved saphenous vein allografts. *J Thorac Cardiovasc Surg* 1976; 71: 378-82.
18. Silver GM, Katske GE, Stutzman FL, Wood NE. Umbelical vein for aortocoronary bypass. *Angiology* 1982; 33: 450-6.
19. Sapsford RN, Oakley GD, Talbot S. Early and late patency of expanded PTFE. vascular grafts in aortocoronary bypass. *J Thorac Cardiovasc Surg* 1981; 81: 861-8.
20. Parsonnet V, Gilbert L, Gielchinsky I, et al. Choosing a vessel for aortocoronary bypass. *Vasc Surg* 1976; 10: 275-8.
21. Angelini GD, Brekenridge IM, Butchart EG, et al. Metabolic damage to human saphenous vein during preparation for coronary artery bypass grafting. *Cardiovasc Res* 1985; 19: 326-34.
22. Logerfo FW, Quist WC, Crenshaw HM, Haunderschild CC. An improved technique for preservation of endothelial morphology in vein grafts. *Surgery* 1981; 90: 1015-24.
23. Fuchs JCA, Mitcherol JS, Hagen PO. Postoperative changes in autologous vein grafts. *Ann Surg* 1978; 188: 1-15.
24. Ramos JR, Bergen K, Mansfield PB, Savage LR. Histological fate and endothelial changes of distended and non-distended vein grafts. *Ann Surg* 1976; 183: 205-28.
25. Malone JM, Gervin AS, Kisher CW, Venous fibrinolytic activity and histological features with distention. *Surg Forum* 1978; 29: 479-80.
26. Angelini GD, Brekenridge IM, Williams HM, Newby AC. A surgical preparative technique for coronary bypass grafts of human saphenous vein which preserves medial and endothelial functional integrity. *J Thorac Cardiovasc Surg* 1987; 94: 393-8.
27. Favaloro RG. Direct myocardial revascularization by saphenous vein graft: Present operative technique and indications. *Ann Thorac Surg* 1970; 10: 97-105.
28. Boerhoom LE, Olinger GN, Bonchek LI, et al. The relative influence of arterial pressure versus intraoperative distention on lipid accumulation in primate bypass grafts. *J Thorac Cardiovasc Surg* 1985; 90: 756-64.

Correspondence to:

Mr. Bryn Williams, St. Thomas' Hospital, London, SE1 7EH. U.K.