Eur J Cardio-thorac Surg (1996) 10:129–140

ORIGINAL ARTICLE

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Arterial and venous conduits for coronary artery bypass

A current review

Received: 20 March 1995
Accepted: 23 May 1995

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Abstract Poor long-term patency of saphenous vein grafts limits the long-term success of the coronary artery bypass operation. If this is to be improved, either measures that increase the patency of saphenous vein grafts or alternative conduits are required. The benefits of using the left internal mammary artery as a pedicled graft to the left anterior descending coronary artery have prompted increasing use of arterial grafts to further improve outcome. Concurrently advances in the understanding of the pathological processes underlying saphenous vein graft occlusion raise the possibility of improving vein graft patency. In this paper we review the problem of vein graft occlusion and possible solutions, the theoretical benefits of arterial grafts and the clinical results associated with their use.
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Key words Myocardial revascularisation · Saphenous vein grafts · Arterial grafts

Introduction

The use of arterial conduits for myocardial revascularisation is not new; Vineberg used the left internal mammary artery for his eponymous procedure in the 1950s [186] and reports of the left internal mammary artery as a direct coronary artery graft appeared in the late 1960s [78, 102]. Following Favoloro's publication, in 1968, of a series of patients in whom saphenous vein was used as a conduit [51], veins eclipsed arteries as conduits for coronary artery bypass. However, long-term benefit following myocardial revascularisation depends to a large extent upon the continued patency of bypass grafts. It has become increasingly apparent that the long-term patency of vein graft is poor, with consequent recurrent angina and impairment of ventricular function [29, 37, 55, 57]. These disappointing long-term results mean that coronary reoperations now constitute a large part of the workload of some instituions [32, 120]. Reoperation is, however, associated with significantly higher mortality and morbidity than first time surgery [90, 130] and relief of angina and improvement in functional status are less certain [48]. If the long-term results of myocardial revascularisation are to be improved either measures that increase the patency of saphenous vein grafts or alternative conduits are required.

The benefits of a left internal mammary artery graft to the left anterior descending coronary artery were established in 1986 [111] but early attempts to increase the scope of arterial grafting with use of the radial and splenic arteries met with disappointment [54, 59]. Interest in multiple arterial grafting with the right internal mammary, gastroepiploic, inferior epigastric and radial arteries is, however, growing again. Concurrently, advances in the understanding of the pathological processes underlying saphenous vein graft occlusion raise the possibility of improving vein graft patency. In this paper we review the problem of vein graft occlusion and possible solutions, the theoretical benefits of arterial grafts and the clinical results associated with their use.

Saphenous vein graft occlusion

Natural history of aorto-coronary saphenous vein grafts

The natural history of aorto-coronary saphenous vein grafts is reasonably established. In the first post-operative month 13%–14% of grafts occlude due to thrombosis [147, 182]. As grafts within the same patient act in a dependent way with respect to thrombotic occlusion [84], occluded grafts tend to cluster within certain patients. Thus, although overall only around 14% of grafts thrombose, in any one patient with graft thrombosis between 21% and 38% of the grafts will be occluded [71, 123].

In the first post-operative year there is little further increase in graft occlusion [57, 80]. However, during this time smooth muscle cells begin to proliferate within the intima of the vein wall [12, 61] and by the end of the 1st year this intimal hyperplasia is well developed in most grafts [31, 37, 180]. Although the development of intimal hyperplasia does not necessarily produce graft occlusion initially, it does reduce graft diameter, as assessed by angiography, by 25%–30% [18]. In the following years, as intimal hyperplasia progresses, grafts occlude at a rate of 2% per year [20].

Beyond 5 years the changes in vein grafts become those of complicated atherosclerosis with the development of necrosis, haemorrhage, calcification and thrombosis [24, 122]. Between 5 and 7 years, 25–35% of the grafts have occluded [18, 30] and by 10 years only 50% remain patent [18, 57, 79] and half of these have severe atherosclerosis [19].

Aetiology of saphenous vein graft occlusion

Thrombosis. Although technical factors such as improper anastomosis and kinking of the graft may contribute to graft thrombosis, the influence of the grafted vessel, systemic factors and intra-operative graft injury have been more extensively studied. Poor graft "run off" is an important determinant of early graft patency [105], the risk of occlusion being highest in coronary arteries with a diameter of less than 1.5 mm [17, 75] and when initial graft flow is less than 50 ml/min [29, 69, 79]. Although endarterectomy is performed to increase the "run off" of a diffusely diseased coronary artery, this may be associated with reduced graft patency [140], probably related to native vessel wall damage [190].

The hypothesis that some patients are "hypercoaguable" and therefore more susceptible to graft thrombosis receives some support from the finding of low levels of antithrombin III, high thrombin-generation index, Factor VIII or increased platelet adhesiveness in a small group of patients with graft thrombosis [194]. Patients with enhanced platelet reactivity (elevated plasma beta thromboglobulin concentration) also seem to be at increased risk of graft thrombosis [68]. Further work is needed before the relative importance of these factors can be established, however.

Much has been written on the intra-operative damage to vein grafts caused by handling, storage in various media prior to use and distension to overcome spasm and identify leaking branches. Certainly these manipulations can produce severe endothelial [14] and medial injury [3]. Loss of the protective endothelial barrier encourages thrombosis [87] and in an experimental model uncontrolled distension promotes early vein graft occlusion [5]. Although techniques have been described that allow adequate preparation of the vein with preservation of endothelial and medial function [6] these have not been tested in a clinical trial and preparative methods which cause endothelial damage are still used by over 70% of surgeons in the UK [4]. However much care is taken in preparation of the vein, once it is implanted into the arterial circulation it is exposed to a much higher transmural pressure than it is accustomed to and this itself may reduce endothelial function [125].

Intimal hyperplasia. This process, which is characteristic of vein grafts placed into the arterial circulation, initially involves proliferation of smooth muscle cells within the media. These proliferating cells then migrate into the intima of the vein where they secrete a connective tissue matrix [144]. The process leads to progressive intimal thickening with reduction in the lumen of the graft. The aetiology of these changes is only poorly understood. Surgical preparation, which favours thrombotic occlusion [5], also provokes intimal hyperplasia [162] and changes in wall stress are implicated, too [197]. These stimuli probably act by promoting the scretion of platelet-derived growth factor [72, 73] and basic fibroblast growth factor [109, 110] although their precise relationship and the importance of other influences, such as immune response [143], remain to be clarified.

Atherosclerosis. Intimal hyperplasia is the substrate for the development of atherosclerosis and the features typically associated with this are found in vein grafts [24]. The development of atherosclerosis involves lipid infiltration in areas of myointimal hyperplasia and is associated with elevated total serum cholesterol and low density lipoprotein as well as a raised low density to high density lipoprotein ratio [31, 161].

Improving the patency of aorto-coronary vein grafts

Early thrombotic occlusion

Graft thrombosis represents an interaction between blood and the luminal surface of the vein and, although most research concentrates on modifying the blood component of

this equation, the possibility of reducing the thrombogenicity of the vein wall has recently come under investigation [177, 179].

That the antiplatelet agents aspirin and dipyridamole improve the short-term patency of vein grafts is well established [36, 94]. Anticoagulants such as warfarin confer no additional advantage but are associated with a higher incidence of complications and their use is not recommended [140]. Despite the proven value of antiplatelet agents, there is still little agreement concerning the optimum dose, the time of starting and duration of treatment. The lack of clear answers despite around 23 trials in the last 10 years reflects the difficulty in interpreting the results. This is principally due to differing inclusion and exclusion criteria and the difficulty in controlling for factors other than antiplatelet treatment which affect graft patency [185]. Despite these limitations some recommendations can be made.

A single daily dose of apsirin of 300 mg is as effective as a higher dose of 1000 mg [74] and a combination of aspirin and dipyridamole [21, 74]. One trial has suggested that a dose of 100 mg is sufficient but methodological flaws rather weaken this conclusion [112]. The efficacy of antiplatelet treatment is greater the sooner it is started [85]. Mural thrombi probably form within a few hours of surgery [33] and ideally the first dose of aspirin should be given before this occurs, that is pre-operatively. This, however, increases post-operative bleeding and blood transfusion requirements [150]. Giving the first dose 6 h after surgery seems to be as effective and as safe as any other regime. The appropriate duration of treatment is unclear. A single daily dose of 300 mg improves patency at 1 year and prevents the occurrence of new thrombotic lesions in grafts at high risk of occlusion [69]. However, aspirin has no effect on the development of intimal hyperplasia [65] and is therefore unlikely to have a beneficial effect on patency rates beyond 1 year. Patients still have coronary artery atherosclerosis, however, and as aspirin reduces their risk of myocardial infarction life-long treatment may be beneficial [178]. The most appropriate antiplatelet regime is, therefore, aspirin in a single daily dose of 300 mg, started 6 h after surgery and continued for at least 1 year [176].

Even with effective antiplatelet treatment, 7.4% of grafts will still thrombose in the 1st post-operative week [76] and, in high risk grafts, this proportion may reach 40% [137]. To further reduce early vein graft thrombosis, other avenues need to be explored. One approach that has given encouraging experimental results is the use of topically applied agents that provide intense, local anticoagulation, without systemic effects [177, 179].

Intimal hyperplasia

To date, no treatment has been shown to be clinically effective in inhibiting the smooth muscle cell proliferative response and its consequence, intimal hyperplasia. Although increasing intra-cellular levels of cyclic 3′5′ adenosine monophosphate or cyclic 3′5′ guanosine monophosphate substantially reduce intimal thickening in *cultures* of human saphenous vein [162], it remains to be seen if this approach is effective in vein *grafts*. Heparin also prevents smooth muscle cell proliferation in-vitro but fails to do so in experimental vein grafts [22] or after angioplasty [49]. Many other drugs, including calcium antagonists, steroids and angiotensin converting enzyme inhibitors, have antiproliferative effects on smooth musle cells, but none has been found effective in clinical studies although most of these have been carried out after angioplasty [148].

Other strategies to prevent the development of intimal hyperplasia continue to be explored and include the use of external vessel supports [13, 196], and photodynamic therapy [135] although the later may be difficult to apply in the clinical situation. Recent advances in molecular biology have provided further avenues which may, in the future, result in improvements of vein graft patency by inhibiting the smooth-muscle cell responses which occur in the vessel wall. These have focused on inhibition of growth factors, thought to be important in the generation of intimal hyperplasia using specific antibodies [52, 110], or the prevention of growth-factor messenger RNA translation using specific antisense oligonucleotides [152]. The majority of this work has to date only been shown to be effective in-vitro and in animal models. The idea that gene therapy may be a useful method of preventing intimal hyperplasia has also recently received an enormous amount of interest [129] although, once again, research in this area is in its infancy.

Atherosclerosis

Patients with raised low density lipoprotein and total cholesterol concentrations are more likely to develop graft atherosclerosis [40, 81]. Lipid-lowering treatment slows the development of atherosclerosis in vein grafts and native coronary arteries following coronary artery bypass surgery [16]. Smoking also promotes graft atherosclerosis [56] and late thrombotic occlusion [160]. Rehabilitation programmes may lower lipid levels, reduce smoking and improve vein graft patency [50].

The theoretical basis for using arterial grafts

These failings of saphenous vein grafts and the limitations of current strategies for increasing their patency have led to the increasing use of arterial grafts. There are sound theoretical reasons for believing that arterial grafts will perform better than vein grafts. Their structure and endothelial function make them inherently more suitable as cor-

onary artery bypass grafts, conferring favourable flow characteristics, the ability to adapt to changes in myocardial oxygen demand and resistance to atherosclerosis.

Structure

The internal mammary artery has a media that is principally elastic with few smooth muscle cells and a dense internal elastic lamina with few fenestrations [183]. This may be an important barrier to smooth muscle cell migration and, therefore, the development of intimal hyperplasia [155]. Although gastroepiploic and inferior epigastric arteries are principally muscular with fenestrated internal elastic laminae [183], these fenestrations are fewer than in sapehnous vein, suggesting that, although they may be more susceptible to the development of intimal hyperplasia than internal mammary arteries, they are still more protected than saphenous vein [155].

Endothelial function

Endothelium resists thrombosis and is an important modulator of vascular reactivity [184]. Two important endothelial products, endothelial derived relaxing factor or nitric oxide and prostaglandin I_2 are involved in both of these functions. Internal mammary arteries release more prostaglandin I_2 than saphenous vein [34, 134, 164] and show greater nitric oxide mediated endothelium dependent relaxation [113, 138]. Gastroepiploic [134] and inferior epigastric [146] arteries also produce high levels of prostaglandin I_2. These vessels are therefore better equipped than saphenous vein to resist thrombosis. The role of these endothelial products in the prevention of atherosclerosis is less clear but high levels of cyclic 3′5′ guanosine monophosphate, which inhibits smooth muscle proliferation [9, 96], are found in internal mammary arteries [171].

The endothelium also beneficially modulates vasoconstrictor stimuli in the internal mammary artery. Aggregating platelets contract saphenous vein but evoke substantial endothelium dependent relaxation in the internal mammary artery [192] and activated neutrophils also produce greater contraction in saphenous vein than internal mammary artery [2]. Gastroepiploic arteries contract in response to aggregating platelets [108], however, they are more sensitive to the vasodilating effect of vasoactive gastrointestinal peptides [114, 115], although the precise role of these peptides in regulating vascular tone is unknown. That the endothelial function of arterial grafts is important in determining their flow characteristics is supported by the observation that the recovery of regional wall motion in myocardium revascularised by internal mammary artery grafts is impaired when the endothelium is removed [62].

Flow

Flow through internal mammary artery grafts is laminar [47, 63] with a large diastolic component characteristic of native coronary artery flow [10, 43]. Saphenous vein grafts, acting simply as passive conduits, show diastolic flow throughout their length [10]. Internal mammary artery grafts, however, actually modulate flow from principally systolic proximally, representing systemic input, to predominantly diastolic distally to match coronary vascular resistance [10]. This flow pattern is lost in failed grafts [121]. The rise in diastolic flow velocity in internal mammary artery grafts is greater and more sustained than in saphenous vein grafts [64] and wall shear stress is higher [10]. High wall shear stresses beneficially modulate endothelial responses [44, 145, 151], resist neutrophil adhesion [107] and inhibit smooth muscle cell proliferation [163]. Reduction in wall shear stress decreases arterial diameter [106], and it has been propsoed that the development of intimal hyperplasia and atherosclerosis represents an attempt to increase wall shear stress by vascular remodelling [70].

In contrast to vein grafts, there is little relationship between flow volume and occlusion of internal mammary artery grafts [38]. Flow volume through internal mammary artery grafts is often less than that through vein grafts early post-operatively [58, 82], due to the resistance imposed by the internal mammary artery [47], and low internal mammary artery graft flow may produce early post-operative haemodynamic collapse [93, 174, 181, 187]. Although the incidence of clinically manifest inadequate flow is low, there is increasing evidence that, compared to saphenous vein grafts, internal mammary artery grafts are associated with subtle reductions in left ventricular function early post-operatively [153] which are more extensive when bilateral mammary artery grafts are used [60]. One month post-operatively this reduction persists and is manifested as exercise induced wall motion abnormalities [97] or a fall in ejection fraction in response to exercise [98]. Although the clinical implications of these findings are unknown, they may explain the increased incidence of ventricular arrhythmias in patients with bilateral internal mammary artery grafts [8], and the experimental finding that progressive reduction of internal mammary artery graft flow produces regional and then gobal left ventricular dysfunction [83] leaves some cause for concern.

Little data has been published for right gastroepiploic and inferior epigastric artery grafts. As these conduits are not usually used to graft the left anterior descending coronary artery, concerns about inadequate flow may be less marked although limited clinical experience with the right gastroepiploic artery as a left anterior descending graft is good [170]. However, grafts originating from the descending aorta show less diastolic flow than those originating from the ascending aorta [175, 189] and flow through in-situ right gastroepiploic grafts may therefore be further

compromised by their origin from the coeliac axis, although they still show diastolic flow predominance [132].

The capacity of right gastroepiploic artery grafts to increase their flow velocity in response to exercise is similar to that of internal mammary artery grafts [104, 172] and they have the unique property of showing a postprandial increase in flow [172, 173]. Areas of the myocardium revascularised by gastroepiploic arteries show an increase in myocardial blood flow [169], although changes in regional ejection fraction have not been reported as they have for internal mammary artery grafts. Radial [35] and gastroepiploic artery [127, 165] grafts seem to be more susceptible to spasm, especially free gastroepiploic artery grafts [126]. This may reflect their muscular structure [183] and contraction to the products of platelet activation [108].

Adaptability

Internal mammary artery grafts retain an ability to dilate in response to increased myocardial blood flow demand [157, 172] and vasoactive drugs [42, 156]. Five years postoperatively 12% of internal mammary artery grafts have increased in diameter as a result of vein graft occlusion, progression of native vessel atherosclerosis or myocardial hypertrophy [15]. By 11 years, with the increased attrition of vein grafts, 31% have increased in diameter [89]. In children internal mammary artery grafts grow in proportion with the growth of the child [100, 101] and in-situ right gastroepiploic grafts may share this growth potential [88]. This adaptability may also result in a reduction in diameter in a few grafts which seems to be a response to competitive flow through the native coronary artery [15]. The degree of proximal coronary artery stenosis determines internal mammary artery graft diameter [149] and flow [91, 92]. Thus when grafted to vessels with a high native flow, internal mammary artery grafts may lose angiographic patency [7] but seem to retain the potential to re-open if the stenosis progresses [45, 166]. The long-term adaptability of other arterial grafts is unknown.

Resistance to atherosclerosis

Atherosclerosis is uncommon in the internal mammary artery [99, 124, 158] even in the presence of coronary artery atherosclerosis [154, 158, 167, 183] and after long-term implantation as a bypass graft [95]. Gastroepiploic [167, 183] and inferior epigastric arteries [183] are also remarkably disease free in patients with coronary artery atherosclerosis although medial calcification is found in up to 25% of inferior epigastric arteries [188].

Clinical results with arterial grafts

Single internal mammary artery grafts

The use of a pedicled left internal mammary artery to the left anterior descending coronary artery is the only combination of aterial graft and recipient coronary artery that has consistently been shown to improve the results of coronary artery bypass surgery when compared to saphenous vein alone. Although the only prospective randomised trial comparing these two strategies failed to show a significant survival advantage at 9 years [195], only 80 patients were randomised and larger, retrospective comparisons have consistently shown the left internal mammary artery to left anterior descending coronary artery combination to confer a survival advantage of about 10% at 10 years [27, 28, 39, 111]. This survival advantage becomes apparent after about 5 years [28] and is maintained in patients with two or three, but not single, vessel disease [39], left main stem stenosis [28] and impaired ventricular function [28, 39] regardless of age [28] or sex [28]. In none of these studies were patients completely matched for other variables that influence survival following coronary artery surgery, however when these factors are taken into account use of the internal mammary artery remains an independent predictor of survival although not as important as age [28, 39], impaired ventricular function [28, 39], incomplete revascularisation [27, 28], female sex [27, 28], left main stem stenosis [27], extent of coronary artery disease [27, 28] and emergency surgery [28].

The influence of the left internal mammary artery graft on other post-operative events is less clear. It reduces the risk of late myocardial infarction [28, 111] other non-fatal cardiac events [111] and re-operation [27, 111] but its impact on recurrent angina is less certain [27, 28, 111].

The improved results associated with use of the left internal mammary artery are traditionally attributed to its improved patency compared to saphenous vein. Saphenous vein grafts to the left anterior descending coronary artery have greater patency than to other coronary arteries [116], so care has to be taken when comparing crude patency rates. When comparisons are restricted to vein grafts to the left anterior descending coronary artery, although at 1 year there is no difference [182], by 10 years patency of the internal mammary artery is between 69% and 94% [118, 133, 195] whilst that of the saphenous vein is only 45%–76% [118, 133, 195]. This difference becomes significant after 5 years [118], which accords with the survival advantage associated with the internal mammary artery.

Bilateral internal mammary artery grafts

The improved results associated with the use of a single internal mammary artery graft have prompted the use of

bilateral internal mammary artery grafts in an attempt to further enhance the results of coronary artery grafting. Although this increases the complexity of the operation, with experience there is no detrimental effect on operative morbidity and mortality [41]. Use of both internal mammary arteries does, however, confer a substantial risk of sternal wound complications in diabetics [41, 103] the elderly [41] and obese [103].

Despite the safety of the procedure and good long-term results [117] the expected benefits over single internal mammary artery grafts have yet to be established conclusively. A prospective, though not randomised, study comparing over 1000 patients with single and bilateral internal mammary artery grafts has failed to show an advantage for two internal mammary artery grafts at 4 years, though it may do so with longer follow-up [128]. A retrospective comparison of patients with single and bilateral internal mammary artery grafts initially suggested that at 15 years, bilateral internal mammary artery grafts increased survival [53]. With improved patient follow-up and better statistical analysis it became apparent that there was no survival advantage [131] but, despite this, the proportion of patients with recurrent angina was reduced from 36% to 27% [53]. This apparant failure of bilateral internal mammary artery grafts to confer a survival advantage may be due to the large number of graft-coronary artery combinations available with bilateral internal mammary artery grafts and, therefore, difficulty in elucidating a winning combination. Alternatively, use of both internal mammary arteries may not confer a survival advantage. This would be true if right internal mammary artery grafts have only a similar patency to vein grafts or if occlusion of a graft to a vessel other than the left anterior descending increases the risk of recurrent angina but not death.

The data on patency of right internal mammary artery grafts is conflicting and difficult to interpret because crude comparisons fail to take into account the confounding variables that influence graft patency. The short-term patency of right internal mammary arteries to the left anterior descending or circumflex coronary arteries is better than to the right coronary artery [46]. Long-term, however, some authors report a similar patency for right and left internal mammary artery grafts [66, 142] but others, comparing grafts placed to the same vessels, report that the patency of right internal mammary artery grafts is no better than that of saphenous vein [86]. As a conclusive survival advantage for bilateral internal mammary artery grafts has yet to be demonstrated, this suggests that, if such an advantage exists, it is probably small.

Right gastroepiploic, inferior epigastric and radial artery grafts

The search for additional, autogenous arterial conduits has continued, however, as even when both internal mammary arteries are used, although complete arterial revascularisation is possible [174], many surgeons prefer a less complex strategy with more than one source of arterial inflow [11]. Use of the right gastroeploic artery, either as a free or pedicled graft, and free inferior epigastric or radial artery grafts enable this to be achieved in most cases. The use of these arterial conduits is a recent development and only short-term results are available.

A right gastroepiploic artery graft does not seem to increase operative mortality [168] or jeopardise gastric mucosal blood flow [168] though diaphragmatic herniation may develop as a postoperative complication [25, 136] and, more seriously, gastric perforation has been reported [191]. Short-term patency of free and pedicled right gastroepiploic artery grafts is similar to [119], or only marginally worse than, that of the left internal mammary artery [67, 77, 141]. The short-term patency of the inferior epigastric artery is less satisfactory [23, 42, 139]. Although initial experience with radial artery grafts was disappointing, with improved harvesting techniques and use of peri- and postoperative diltiazem, patency of 94% is reported at a mean of 9 months [1, 26]. This is rather worse than internal mammary artery grafts but comparable with the inferior epigastric artery. Larger series with longer follow-up need to be reported before any firm conclusions can be made about these other arterial conduits.

Conclusion

The current success of the coronary artery bypass operation is built upon the use of saphenous vein grafts [193]. Although using the left internal mammary artery as a pedicled graft to the anterior descending coronary artery improves the outcome, the benefits of extending the use of arterial grafts with coth mammary arteries have yet to be demonstrated conclusively. The advantages of using additional, autogenous arterial conduits to achieve "total arterial revascularisation" are largely theoretical and it will be many years before sufficient data are available to allow firm conclusions to be drawn. In the United Kingdom in 1993–1994, of patients having between two and five distal anastomoses, an internal mammary artery was used for one anastomosis in 71%, two anastomosed in 11% and three or more in 1% of cases [159]. Vein grafts alone were used in 17% [159]. Even when a positive attitude is taken to using multiple internal mammary artery grafts, this is not possible in almost 30% of patients [128].

Vein grafts still play an important role in contemporary cardiac surgical practice and are likely to continue to do so for the immediate future. If the theoretical advantages of extended arterial grafting are realised, their role will diminish. However, if the experimental advances in further reducing vein graft thrombosis and intimal hyperplasia are translated into clinical practice, they may undergo a resurgence.

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