Arterial Revascularization of the Heart

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General Surgery

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ABSTRACT

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Expanded use of arterial grafts for myocardial revascularization is based on accumulating data of superior late patency of arterial grafts compared with venous conduits.The primary consideration that has led to the gradual transition of the use of internal mammary artery as conduits is relative freedom from atherosclerosis with follow up to 20 years.

Key words: Arterial - Revascularization - Heart

Contents

List of Abbreviations

CABG :Coronary artery bypass graft.

OPCAB :OFF Pump Coronary artery bypass.

LIMA :Left internal mammary artery.

LITA : Left internal thoracic artery.

RIMA :Right internal mammary artery.

RITA :Right internal thoracic artery.

RA :Radial artery.

GEA :gastroepiploic artery.

IEA :inferior epigastric artery.

UA :ulnar artery.

BIMA :Bilateral internal mammary artery.

SVG :Saphenous vein graft.

LAD :Left anterior descending.

RCA :Right coronary artery.

OM :Obtuse marginal.

PDA :Poster descending artery.

PL :Posterolateral.

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INTRODUCTION:

HISTORICAL BACKGROUND AND AIM OF THE WORK

INTORDUCTION

Coronary artery disease remains the major cause of mortality worldwide. Despite aggressive application of percutaneous coronary interventions, the surgical approach, namely coronary artery bypass grafting (CABG), remains a major approach for the treatment of coronary artery disease. Usage of arterial grafting has been well accepted as the main approach to increasing the long-term patency of the graft and long-term survival .
[Guo-Wei He(2004)]

Expanded use of arterial grafts for myocardial revascularization is based on accumulating data of superior late patency of arterial grafts compared with venous conduits**(Lytle BW et al (1985).**The primary consideration that has led to the gradual transition of the use of internal mammary artery as conduits is relative freedom from atherosclerosis with follow up to 20 years. During the last decade the frequency of coronary artery revascularization procedures increased considerably in patients with diseased or absent great saphenous vein, alternatives to this vein conduit have been sought for as, the right mammary and radial artery [**Loop FD et al (1986)**

HISTORICAL BACKGROUND

Vineberg postulated that anastomoses could develop between the coronary arteries and the transplanted internal thoracic artery (ITA) as early as 1941 at McGill University. He mobilized the left ITA, leaving the side branches, and ligated the vessel distally. The ITA was subsequently implanted into a tunnel in the left ventricular muscle alongside the left anterior descending coronary artery (LAD) .

It may have been**[Longmire (1958)]** who first used the ITA to bypass lesion in the right coronary artery.

In 1968, the ITA became more widely applied as new surgical techniques evolved. Bailey and Hirose attached the right ITA to the right coronary artery.

In 1971, Carpentier used for the first time the radial artery (RA) to bypass the coronary arteries .

AIM OF THE WORK

The aim of this work is to review literature concerning the advantages and disadvantages of using arterial grafts in the domain of long term patency and long term survival.

The Pathophysiology of Chronic CAD

Lesion Formation:

Previously considered a cholesterol storage disease, we curently understand atherogenesis as a complex interaction of risk factors including cells of the artery wall and the blood and molecular messages that they exchange. A useful organizing theme, which emerged first from laboratory studies and has now gained currency in the clinic, accords inflammation a major role in all stages of atherogenesis. Inflammation also participates in the local, myocardial, and systemic complications of atherosclerosis**. (Libby P 2002**)

When the arterial endothelium encounters certain bacterial products or risk factors as diverse as dyslipidemia, vasocontrictor hormones inculpated in hypertension, the products of glycoxidation associated with hyperglycemia, or proinflammatory cytokines derived from excess adipose tissue, these cells augment the expression of adhesion molecules that promote the sticking of blood leukocytes to the inner surface of the arterial wall. Transmigration of the adherent leukocytes depends in large part on the expression of chemoattractant cytokines regulated by signals associated with traditional and emerging risk factors for atherosclerosis. Once resident in the arterial intima,the blood leukocytes—mainly mononuclear phagocytes and T lymphocytes —communicate with endothelium of the arterial wall. Major messages exchanged among the cell types involved in atherogenesis depend on mediators of inflammation and immunity, including small molecules that

include lipid mediators such as prostanoids and other derivatives of arachidonic acid, eg, the leukotrienes. Other autacoids, such as histamine,

classically regulate vascular tone and increase vascular permeability. Recently, much attention has focused on protein mediators of inflammation and immunity, including the cytokines and complement components. Virtually unknown by cardiologists a mere decade ago, the cytokines have joined the mainstream of our specialty. As a major consequence of the inflammatory ferment underway in the early atheroma, SMCs migrate from the tunica media into the intima. These cells proliferate and elaborate a rich and complex extracellular matrix. In concert with endothelial cells and monocytes, they secrete matrix metalloproteinases (MMPs) in response to various oxidative, hemodynamic, inflammatory, and autoimmune signals.

MMPs, in balance with their endogenous tissue inhibitors, modulate numerous functions of vascular cells, including activation, proliferation, migration, and cell death, as well as new vessel formation, geometric remodeling, healing, or destruction of extracellular matrix of arteries and the myocardium. **(Libby P 2000).**

Certain constituents of the extracellular matrix (notably proteoglycans) bind lipoproteins, prolong their residence in the intima, and render them more susceptible to oxidative modification and glycation (nonenzymatic conjugation with sugars). These products of lipoprotein modification, including oxidized phospholipids and advanced glycation end products, sustain and propagate the inflammatory response**.(Tabas 1999).**

As the lesion progresses, calcification may then occur through mechanisms similar to those in bone formation. In addition to proliferation, cell death (including apoptosis) commonly occurs in the established atherosclerotic lesion. The death of lipid-laden macrophages can lead to extracellular

deposition of tissue factor (TF), some in particulate form. The extracellular lipid that accumulates in the intima can coalesce and form the classic, lipid-rich“necrotic”core of the atherosclerotic plaque. **(Bogdanov VY et al 2003).**

ROLE OF VENOUS GRAFTS IN ARTERIAL GRAFTING

Use of SVG in Combination with Arterial Grafts:

Nowadays, it is rare that the SVG is used alone. It is almost always used in combination with arterial grafting; preferably, it is used for those territories that are not grafted with arterial grafts. There is large variation among surgeons as to the target vessel for the vein graft. However, it is almost unanimously accepted that the LIMA is grafted onto the LAD. Therefore, the SVG is grafted onto either the diagonal, obtuse marginal (or ramus marginalis), or RCA, depending on the use of the second arterial graft . **[Tatoulis et al(2004)].**

Methods to Improve SVG Patency:

The major causes for the lower patency of the SVG compared to IMA may involve a few factors. First, as a free graft, the disruption of the vasa vasorum of the venous wall causes ischemia of the vein. This is an unavoidable cause. Second, the SVG almost inevitably goes into vasospasm when taken from the leg due to surgical stimulation.

The spastic vein requires distension as the normal procedure to overcome vasospasm and to check leaking from side branches. The distension pressure, which is not normally monitored, may easily go up to 500 mmHg without the surgeon being aware of it **[He GW et al (1993)],** and this high pressure distention has been demonstrated as being damaging to both the endothelium and the smooth muscle of the vein. The damage of the vein causes late occlusion of the graft. This factor becomes more important during minimally invasive harvesting of the saphenous vein because more

surgical manipulation may be involved. In undistended saphenous vein segments isolated from patients under going minimally invasive surgical and open techniques of harvesting, there was no acetylcholine-mediated endothelium-dependent relaxation in the minimally invasive surgery group but it exists in the vein taken by the open technique.

Therefore harvesting of the saphenous vein through multiple small incisions might result in endothelial dysfunction, possibly caused by traction injury **[Cook RC et al (2004)].**

In order to improve SVG patency, it is essential to perform CABG using the SVG in such a way that the vein structure can be better preserved. In particular, the distension procedure should be improved. This is feasible by gentle manipulation of the vein when it is taken in order to minimize the surgical damage to the vein. Further, the use of proper vasodilators on the vein may improve the preservation of the vein. For example, a mixed VG (verapamil-nitroglycerin) solution used for arterial grafts can also be used to prevent venous spasm, reducing the distension pressure and therefore preserving the endothelium and smooth muscle of the vein .

[Souza et al (2002)] compared the patency of the SVG that was harvested by three different methods.

With the conventional method, the vein was stripped, distended, and stored in saline; with the intermediate group, the vein was stripped, local application of papaverine was used instead of distention, and the vessel was then stored in heparinized blood. In the no-touch group, the vein was harvested with the surrounding tissue, not distended, and stored in heparinized blood. The vein graft patency at an average of 18 months was 88.9%, 86.2%, and 95.4%, respectively, and they concluded that preservation of the surrounding tissue of the saphenous vein using this no-

touch technique abolishes venospasm intra operatively and plays an important role in maintaining vein graft function and patency.

Another import factor in the SVG occlusion is anastomosis technique. A perfect anastomosis may improve the patency.

Other technical points are also worth taking into account. For example, in diffuse coronary artery disease, the arteriotomy can be treated by extending over the plaques, with graft patency rates comparable to those of bypass grafts onto less diseased segments **[Doss M, et al (2001)].**

There are new technical innovations including the use of the St. Jude Medical connector. The angiographic patency ranges from 86% to 100%.

[Semrad M, et al (2003)]. However, further long-term studies are required to confirm the safety of the St. Jude Medical connector with regard to endothelial function and restenosis.

In addition, fibrin glue effectively prevents over distension and preserves some distensibility in the high pressure range in both the upper and lower leg saphenous vein. This might provide a basis for clinical application of perivenous support**[Stooker W et al (2003)].**

Finally, the major reason for the difference in patency between the SVG and IMA may be the structure differences between arteries and veins.