

DIFFERENCES IN CONDUIT AND BUFFERING FUNCTION AMONG ARTERIES, VENOUS GRAFTS AND SYNTHETIC PROSTHESIS: IMPLICANCES IN THE DEVELOPMENT OF INTIMAL HYPERPLASIA

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Abstract— Usually vascular surgeons consider the use of vein grafts as the materials of first choice for bypass or reconstruction of small arteries, while synthetic prosthesis (i.e. polytetrafluoroethylene, ePTFE) plays a secondary role in this matter. However, the causes for the superior performance of vein grafts respect ePTFE remain unclear. Our aim was to compare the conduit (CF) and buffering function (BF) of arteries, with those of vein grafts and ePTFE. *In vitro* pressure (Konigsberg) and diameter (Sonomirometry) were measured in ovine arteries and veins, and ePTFE prosthesis, under isobaric and physiological pressures levels. From stress-strain relationship the Kelvin-Voigt time constant was calculated to quantify the BF. The CF was evaluated as $1/Z_C$, where Z_C is the characteristic impedance. Vein graft CF and BF were more similar to native arteries than those of ePTFE prosthesis. Consequently, the higher performance of vein grafts could be related with their superior matching with arteries.

Keywords— vascular prostheses, conduit function, buffering function, intimal hyperplasia.

I. INTRODUCTION

The arteries conduit function (CF) allows the distribution of blood to different tissues, while their buffer function (BF) is responsible for the smoothing of pressure and flow pulsatility determined by the intermittent ventricular ejection (Nichols and O'Rourke, 1998; Bia *et al.*, 2003; 2004; 2005a). Both functions are related and depend on the geometrical and mechanical properties of the arteries, which in turn are determined by passive (elastin and collagen fibers) and active components (smooth muscle cells) of the arterial wall. In this sense, its dynamic behavior depends on the viscoelastic individual contribution of each wall constituent and the structural arrangement among them (Bia *et al.*, 2003; 2004; Nichols and O'Rourke, 1998).

In several circumstances in which an arterial segment is altered, an arterial bypass or reconstruction is performed, in order to restore the functional capability. Looking for improving the arterial substitute patency rate, there have been described several conditions that the ideal graft should fulfill. Related to this, taking in to

account that the mechanical and/or impedance mismatch between the native artery and the alternative conduit may induce graft failure, it is accepted that an ideal arterial substitute must share identical viscoelastic and geometrical properties with the native artery (Tai *et al.*, 1999). Therefore, in theoretical terms, an autologous artery would be the ideal arterial substitute. However, except in certain limited situations, it is difficult to obtain an autologous artery of adequate length and size without the sacrifice of an organ or essential part of the body.

Several synthetic prosthesis have been employed, and nowadays, among the most widely used are expanded polytetrafluoroethylene (ePTFE) prosthesis. However, occlusion rates of ePTFE when used in small or medium size arterial reconstruction are high. For instance, patency rates for 175 ePTFE femoropopliteal bypasses were 62% at 35 months (Veith *et al.*, 1980). Better results have been obtained with venous grafts. So, the use of autologous veins (mainly the human great saphenous vein) is the first choice in bypass or reconstruction of medium and small arteries, while non-biological prosthetic materials (i.e. ePTFE) assume a secondary role in this position (Dardik and Howard, 1999).

The causes of the superior performance of veins respect to the ePTFE, in the medium and small size arteries' reconstruction, remain to be elucidated (Cabrera Fischer *et al.*, 2005; Norberto *et al.*, 1995; Kissin *et al.*, 2000). The main cause of ePTFE graft failure within a short period of time is the development of intimal hyperplasia (IH) in the distal anastomosed artery (Vijayan *et al.*, 2002; Echave *et al.*, 1979), causing outflow stenosis of prosthetic bypass and tissue ischemia. The mechanical mismatch between the ePTFE and the native artery is considered a major cause of intimal hyperplasia and graft failure (Dardik and Howard, 1999).

The pathogenesis of this lesion is described as proliferation of smooth muscle cells of the arterial media and their migration into the intima, in response to injury at the junction of the graft and the native artery. Over the past years, IH in the bypassed or reconstructed arteries has been extensively studied; however the mechanism through which it develops has not been exactly resolved (Dardik and Howard, 1999). It has been de-