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The fate of large-diameter Dacron® vascular grafts in surgical practice: Are we really satisfied?

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Taking from the relative lack of systematic review in the field of large vessel prosthetic replacement with synthetic grafts, we analyzed the literature on the long-term performance and complications of Dacron® grafts for large vessel reconstruction in the abdominal and thoracic districts, with particular attention to the late local and systemic consequences associated to these procedures. Aortic replacement with Dacron® prostheses is widely performed with acceptable outcomes and a relatively low rate of graft-related and post-implantation complications, such as rupture or infection. Despite the advances in the manufacturing techniques, progressive graft dilation and mechanical failure are the most relevant issues especially in the long-term follow-up, posing troublesome questions on the optimal clinical management of both abdominal and ascending aorta pathologies.

However, what really emerged from the analysis of the long-term surgical experience with Dacron® grafts is the increasing evidence that the mismatch in biomechanical properties between prosthetic material and native aorta is at the root of daunting issues in the long-term follow-up of these patients. Indeed, a careful analysis of the literature revealed that the loss of compliance associated to aorta replacement with prosthetic material is emerging as a real "worrisome enemy" in the long-term outcome of these operations, leading not only to prosthesis-related complications, but also to detrimental effects on cardiac and even systemic arterial system. Introduction of non-compliant grafts determines a significant increase in the systemic arterial characteristic impedance, resulting in augmented systolic and pulse pressure. Dacron® is stiffer than the native aorta (ca. 24 times stiffer than healthy aortic arch tissue, according to Tremblay et al. [1]). The ability to convert the hydraulic pulsatile energy imparted by the ventricle into a steady constant distal flow, known as *Windkessel* function, relies on the elastic reservoir properties of the aorta and replacement with a non-compliant artificial material determines the loss of its "hydraulic damper" function, eventually leading to local alterations at the peri-anastomotic site and to significant changes in arterial hemodynamics and left ventricular load. Indeed, compliance mismatch between the prosthetic graft and the native aorta has been demonstrated to impart excessive stresses at the suture lines, resulting in intimal hyperplasia, suture fatigue, or anastomotic aneurysm. When the graft interposition occurs in the ascending aorta site, stiffening of the "neovasculature" and increased aortic impedance translate into altered transmission of the pulse wave with retrograde detrimental effects on aortic valve and on cardiac function and perfusion, eventually leading to left ventricle hypertrophy [2]. A careful analysis of the echocardiographic follow-up data of patients undergone aortic root replacement throughout different studies indicates a cumulative percentage of more than 40% of patients showing a mild-to-moderate aortic valve insufficiency [3]. Both *in vitro* and *in vivo* studies with phase-contrast magnetic resonance demonstrated that the subversion of aortic compliance and elastomechanical properties associated to these procedures diminished aorta distensibility and pulsatility due to increased wall stiffness, and that the consequent loss of elastic recoil during cyclic loading might induce a redistribution of forces causing both annulus dilation and cusp dysfunction with valve incompetence [4]. Additionally, data show that loss of dynamic compliance of the vascular tree results in left ventricle overload due to the progressive increase in wall stress, mandating for an increased energy demand and an augmented generation of pressure load for the ventricle in order to maintain cardiac output through an unchanging peripheral resistance [5]. Chronic *in vivo*

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reflex of this situation is the adaptive left ventricular hypertrophy demonstrated in clinical reports on ascending and/or abdominal aorta bypass for thoracoabdominal aneurysm using woven Dacron® grafts [6]. Moreover, vasculature stiffening might induce cardiac dysfunction and ischemia for coronary occlusion and increased oxygen demand [7]. Interestingly, most of the studies analyzed in this review date back to the early nineties, suggesting that—at the time—a problematic scenario was already perceived. However, the majority of current literature keeps underpinning the satisfactory clinical outcome of aortic replacement, and concentrating—in some cases—on graft-related complications as rupture, pseudo-aneurysm formation, or infection, while ignoring potential reflexes on the systemic cardiovascular function. It remains unclear whether this reported scarce attention to systemic effects has to be attributed to a poor correlation between experimental models and the real clinical scenario, or to clinical studies which are underpowered, and their follow-up not adequately prolonged to detect other systemic complications.

However, while the conclusions that can be drawn from this literature survey remark the overall clinical success of aortic replacements by polyester grafts, in the authors' opinion, a warning on potential long-term complications should be taken into account when performing surgical work-up and operative strategy decision. The current liberal use of non-compliant grafts in endovascular or surgical reconstruction of the thoracic aorta might be reconsidered, and a renovated attention to the development of vascular conduits able to mimic or match the native artery compliance is desirable in this context.

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Left ventricular fibrosis in adults after coarctation repair, does it play a role?

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Hypertension is one of the most important contributors to this increased cardiovascular risk in aortic coarctation [1,2]. Organ damage involving left ventricular hypertrophy (LVH) and an increased LV mass are therefore often found in CoA. However, extensive LVH has also been described in neonates after early repair and in normotensive CoA, which suggests that the LV abnormalities might have a more developmental origin, which has been previously suggested [3–5]. In general two different types of cardiomyopathies can be distinguished. In patients with hypertrophic cardiomyopathy LVH has a genetic origin, whereas in patients with aortic valve stenosis (AS) or hypertension, LVH is caused by pressure overload. Discrimination between cardiomyopathies can readily be made with cardiac MRI (CMR) as cardiomyopathies often show distinct patterns of fibrosis [6,7]. The aim of this study is to investigate the presence and pattern of LV fibrosis in CoA which may

Table 1
Baseline characteristics of 27 adult post-coarctectomy patients.

	N = 27	
Age (yrs)	34	(11–57)
Male	16	59%
BSA (M^2)	1.90 ± 0.23	
Age repair (yrs)	7.0 ± 8.6	(0.34)
Time after surgical repair (yrs)	26.8	(0–51)
Hypertension	10	37%
History of recoarctation	3	11%
Bicuspid aortic valve	15	56%
LV mass (g/m^2) ± SD	230.2 ± 75.4	
Increased LV mass (g/m^2)	16	59%
EF (%)	55 ± 14.3	
Aortic valve stenosis		
≤20 mm Hg	19	70%
≥20 mm Hg	8	30%
Aortic insufficiency		
None/mild	25	93%
Moderate/service	2	7%

BSA = body surface area; LV = left ventricular; EF = ejection fraction.

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