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EDITORIAL

Effects of Aortic Graft Implantation on Heart and Downstream Vessels: An Artery is not a Rigid Pipe

In this issue of the *European Journal of Vascular and Endovascular* Surgery (EJVES), Rong *et al.*[1] investigated the immediate impact of ascending aortic prosthetic graft replacement on the descending aorta, using intra-operative transoesophageal echocardiography. They showed that prosthetic graft replacement induced increases in circumferential deformation and paralleling distensibility in the native descending aorta but without significant modification of systemic haemodynamics and left ventricular (LV) stroke work, suggesting that replacing native aorta with a graft increases pulse pressure and energy propagation downstream to the graft. This is of clinical importance as such findings could partly explain mechanisms leading to early or late graft failure, or to disease progression in other aortic segments.

The inability of synthetic grafts to mimic the elastomechanical characteristics of the native arterial tissue and the consequent lack of adequate compliance (currently available vascular grafts are four times less compliant than native arteries) can lead to a cascade of haemodynamic and biological alterations deeply affecting cardiovascular homeostasis.[2] The notion of "compliance mismatch" between the graft and the native aorta has therefore been introduced to explain what emerged as a silent but worrying concept regarding the effects that the presence of a prosthetic graft can induce in native cardiovascular structures, either proximal or distal to the graft, and consequently on the long term outcomes of such prosthetic grafts and their impact on the patient's prognosis.[3,4]

Deleterious effects can occur proximal to the graft and are related to the haemodynamic wave reflection of the compliance mismatch on the LV as well as on the aortic valve. Indeed, in physiological conditions, the LV and its afterload are a matched system. It has been demonstrated that optimal ventricular–arterial coupling is reached when a maximum energy, or stroke work, is transferred to the vascular tree.[4,5] The aorta and some of the proximal large vessels store about 50% of the LV stroke volume during systole, acting as an elastic buffering chamber behind the heart. In diastole, the elastic forces of the aortic wall forward this volume to the peripheral circulation, thereby creating a nearly continuous peripheral blood flow. This systolic–diastolic interplay represents the Windkessel function, which has an influence on the peripheral circulation and also on the heart, resulting in a reduction of LV afterload, and improvement in coronary blood flow and LV relaxation.[4]

The replacement of the highly elastic native aorta with a non-compliant graft will affect characteristic impedance and pulse wave reflection but above all, the loss of the Windkessel effect and alteration of the pulse wave propagation translate into additional workload for the LV eventually inducing adaptive hypertrophy.[3,5] The prosthetic graft thus generates a significant change in impedance at the interface with the native elastic artery inducing a reduction in ventricular pumping efficiency.[3] Moreover, the loss of diastolic systemic blood pressure augmentation can reduce organ perfusion and particularly coronary flow leading to myocardial ischaemia despite the absence of coronary artery stenosis.[6] Further, increased inflammation, oxidative stress, and myocardial and vascular fibrosis can be related to impaired ventricular-arterial coupling. The prosthetic graft also influences the aorta–valve complex, inducing aortic valve malfunctioning, since the haemodynamic load exerted on the prosthetic graft might induce progressive annular dilation eventually leading to aortic incompetence.

Deleterious effects can also occur distal to the graft. The compliance mismatch between the graft and the native aorta reduces blood accumulation in the ascending aorta, resulting in increased propagation of systolic flow to the descending aorta as compared with pre-operative conditions. This is therefore related to increased energy propagation to the distal aorta due to the presence of a non-compliant graft which cannot absorb forward stroke volume.[7] Accordingly, the replacement of the proximal aorta leads to pulse pressure increase, primarily caused by forward wave increase.[8] On another hand, the unfavourable elastomechanical properties and the compliance mismatch between the graft and the native aorta are thought to exert excessive stress at the suture lines, resulting in the development of pseudo-aneurysms.[3] This might be attributed to the continuous traction on the suture lines of non-compliant grafts.[2,6] The compliance mismatch can also lead to long term ascending aortic prosthetic graft size change. It has been demonstrated that the diameter of grafts used in the ascending aorta increases by around 26% compared with the package size immediately after implantation, and that such grafts dilate gradually at 3.2% per year in diameter after implantation.[8] The compliance mismatch and loss of elasticity may be responsible for alterations in the graft yarn architecture and be further responsible for the dilation profile shown by these grafts.[8]

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Deleterious effects, either proximal or distal to the graft, might also be exacerbated, considering that surgery is usually performed in elderly patients whose aortas are already further along in the degenerative process and might have reduced compliance due to calcification and stiffness. Introduction of non-compliant grafts in such patients might lead to even more haemodynamic changes. These patients should however be differentiated from young patients treated with prosthetic grafts for coarctation or aneurysm, or young patients treated with endografts for aortic transection. Considering the young age of these patients and their long life expectancy, the surgeon should be aware of the possible risk of re-intervention. In such patients, the haemodynamic load exerted on a non-compliant graft may be considered as a risk factor for late graft failure. This might also be more pronounced in patients with Marfan syndrome, in whom congenital abnormal fibrillin metabolism can render residual aortic tissues even weaker and more prone to dilation.

In conclusion, despite the overall successful performance of aortic grafts in current use, the compliance mismatch between the native aorta and prosthetic grafts should trigger future research. The editors of EJVES are looking forward to the submission of high quality translational research on alternative conduits mimicking native elasto-mechanical vascular properties.[9]

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