known to stabilize the vascular wall highlighting the important role pericytes play in vascular homeostasis. In addition we show for the first time that pericytes do not only react to stretch but are sensitive to direct shear-stress.

Effect of Intensified Decellularization of Equine Carotid Arteries on Scaffold Biomechanics and Cytotoxicity

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Introduction: Decellularized equine carotid arteries (dEAC) are suggested to represent an alternative for alloplastic vascular grafts in haemodialysis patients to achieve vascular access. Recently it was shown that intensified detergent treatment completely removed cellular components from dEAC and thereby significantly reduced matrix immunogenicity. However, detergents may also affect matrix composition and stability and render scaffolds cytotoxic.
Methods: Intensively decellularized carotids (int-dEAC) were evaluated for their biomechanical characteristics (suture retention strength, burst pressure and circumferential compliance at arterial and venous systolic and diastolic pressure), matrix components (collagen and glycosaminoglycan content) and indirect cytotoxicity (WST-8 assay) and compared with native (n-EAC) and conventionally decellularized carotids (con-dEAC).
Results: Both decellularization protocols led to comparable reduction of matrix compliance (venous: 32.2% and 27.4% of n-EAC; $p < 0.01$ and arterial: 26.8% and 23.7% of n-EAC, $p < 0.01$) but had no effect on suture retention strength and burst pressure. Matrix characterization revealed unchanged collagen contents but a 39.0% (con-dEAC) and 26.4% (int-dEAC, $p < 0.01$) reduction of glycosaminoglycans, respectively. Elastine fibres were scattered and less wavy in both dEAC. Cytotoxicity was not observed in either dEAC matrix.
Conclusion: Thus, even intensified decellularization generates matrix scaffolds highly suitable for vascular tissue engineering purposes, e.g. the generation of haemodialysis shunts.

Impact of Thoracic Endovascular Aortic Repair on Pulsatile Aortic Changes

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Introduction: The thoracic aorta experiences high hemodynamic forces and shows significant aortic pulsatile changes during the cardiac cycle. Thoracic endovascular aortic repair (TEVAR) may cause modifications to this dynamic condition, which may vary based on the aortic disease. Analysis before and after TEVAR, may help to evaluate the impact of stent-grafts on aortic pulsatile changes and its implications on post-TEVAR thoracic aortic behaviour.
Methods: Custom developed software and dynamic CT imaging were used to quantify aortic radial expansion and elongation during the cardiac cycle, together defined as aortic pulsatile changes. Through the assessment of the aortic vessel centreline, diameter and area changes were measured at the level of the sinotubular junction (level A), 1 cm proximal to the brachiocephalic trunk (level B), left subclavian artery (LSA, level C), 10 cm distal to LSA (level D), 20 cm distal to LSA (level E), and celiac bifurcation (level F) (Figure 1). Two patients treated with TEVAR for type B aortic dissection, of whom one affected by Marfan syndrome, one with thoracic aortic aneurysm and one healthy control subject were analysed. Aortic elongation changes, including length ascending aorta (L1), length aortic arch (L2), length descending aorta (L3), and total length (L) (Figure 1), were measured pre- and post TEVAR.
Results: In all patients two stent-grafts were implanted. Pre- and post-operative radial expansion and elongation changes were visualised during the cardiac cycle. Pre-operative aortic pulsatile changes were more evident in the patient with Marfan syndrome, in particular in the ascending aorta. After stent-graft placement, a trend of overall increased aortic pulsatile changes was observed, with aortic elongation marked proximally to the stent-graft (Figure 2).
Conclusion: The aortic pulsatile changes were overall increased after TEVAR, in particular aortic elongation proximally to the stent-graft. This study represents an initial investigation for understanding the impact of TEVAR on both aortic elongation and radial expansion. Further studies are warranted to understand if these observations may have implications for patient selection, stent-graft sizing, design, durability, and prevention of TEVAR related complications.

CCL5-dependent Mediation of Transplant-induced Atherosclerotic Lesion Formation in the Aorta

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Introduction: The CCL5 chemokine plays an active role in the initiation of inflammation, and by recruiting monocytes to sites of atherosclerotic lesion growth is critically involved in atherosclerosis. A function of CCL5 in circulating cells versus resident vessel wall cells in atherosclerosis within transplanted aortic segments has not yet been examined.
Methods: We have orthotopically transplanted infrarenal abdominal CCL5−/− Apoe−/− aortic segments into Apoe−/− mice and Apoe−/− aortas in CCL5−/− Apoe−/− mice ($n = 4$-6 mice) (anastomosis time 22 min). After 4 weeks, the intimal plaque size in the region of the transplanted aorta was analyzed in serial sections, and the plaque macrophage assessed by immunohistochemical staining.
Results: Deficiency of CCL5 in vascular cells of the transplanted segment (transplantation of CCL5 Apoe−/− aortas into Apoe−/− mice) entailed a reduction in the formation of atherosclerotic plaques and the accumulation of macrophages, compared to the deficiency of CCL5 in circulating