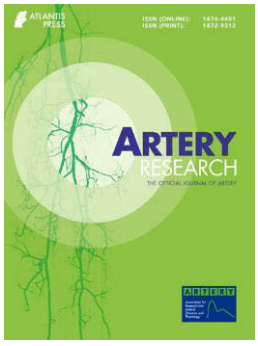

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4.7: THE EFFECT OF TRANSCATHETER AORTIC VALVE IMPLANTATION ON AORTIC STIFFNESS AND HEMODYNAMICS

Vasiliki Gardikioti, D. Terentes-Printzios, C. Vlachopoulos, K. Toutouzas, M. Xanthopoulou, G. Benetos, G. Latsios, V. Penesopoulou, V. Tsigkou, G. Siasos, E. Vavuranakis, D. Tousoulis

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4.6
INFLAMMATION AND AORTIC STIFFNESS. A MULTICENTRE LONGITUDINAL STUDY IN PATIENTS WITH INFLAMMATORY BOWEL DISEASE

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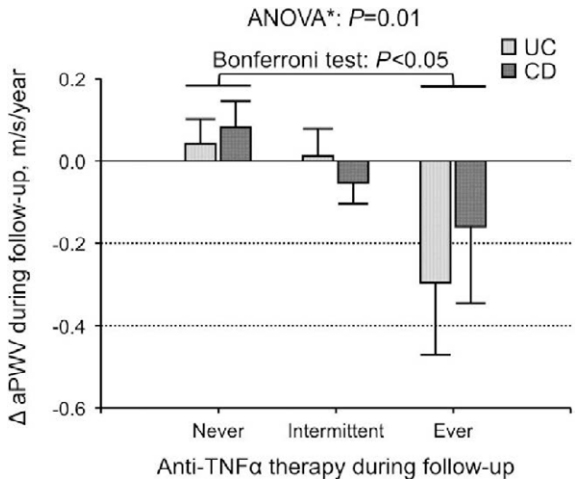
Background: Inflammatory Bowel Disease (IBD) is characterized by a low prevalence of traditional risk factors, an increased aortic pulse-wave velocity (aPWV) [1] and an excess of cardiovascular events. We have previously hypothesized that the difference between expected and observed cardiovascular risk could be explained by chronic inflammation [2]. In this multicentre longitudinal study, we tested the hypothesis that increased aPWV is reversible with anti-tumor necrosis factor-alpha (anti-TNFα) therapy.
Methods: We enrolled 334 patients (82 patients with ulcerative colitis [UC], 85 patients with Crohn's disease [CD] and 167 healthy control subjects matched for age, sex and mean blood pressure) from 3 Centres in Europe and followed up them for 4 years (range 2.5—5.7 years).
Results: At baseline, IBD patients had higher aPWV than controls. IBD patients in remission and those treated with anti-TNFα during follow-up experienced an aortic destiffening whereas aPWV increased in those with active disease and those treated with salicylates (Figure 1, P = 0.01). Disease duration (P = 0.02) and, in UC patients, the increase in CRP during follow-up (P = 0.02) were associated with aortic stiffening. All these results were confirmed after adjustment for major confounders. Finally, the duration of anti-TNFα therapy was not associated with the magnitude of the reduction in aPWV at the end of follow-up (P = 0.85). This finding could suggest that anti-TNFα therapy has a beneficial effect on functional arterial stiffening.
Conclusions: Long-term anti-TNFα therapy reduced aPWV, an established surrogate measure of cardiovascular risk, in patients with IBD. This suggests that effective control of inflammation may reduce cardiovascular risk in these patients.



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4.7
THE EFFECT OF TRANSCATHETER AORTIC VALVE IMPLANTATION ON AORTIC STIFFNESS AND HEMODYNAMICS

Vasiliki Gardikioti [1,2], D. Terentes-Printzios [2], C. Vlachopoulos [2], K. Toutouzas [2], M. Xanthopoulou [2], G. Benetos [2], G. Latsios [2], V. Penesopoulou [2], V. Tsigkou [2], G. Siasos [2], E. Vavuranakis [2], D. Tousoulis [2]
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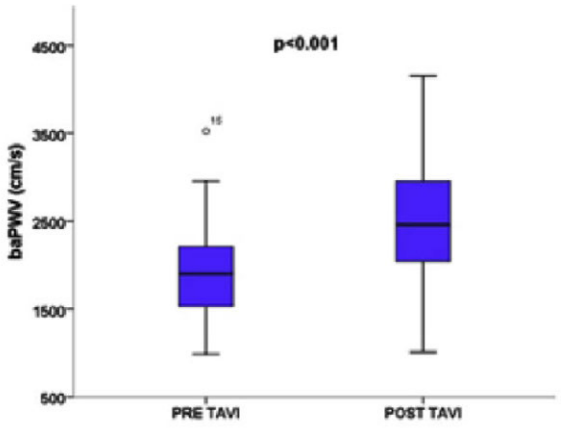
Purpose/Background/Objectives: Aortic stiffness and central hemodynamics are established vascular biomarkers. Transcatheter aortic valve implantation (TAVI) is a promising new technique for the treatment of aortic valve stenosis in elderly patients. We examined the effect of TAVI on the elastic properties of the aorta and on central hemodynamics.
Methods: We included fifty patients (mean age 80.7 ± 8.3 years, 27 male) with symptomatic aortic stenosis treated by TAVI. In measurements prior and acutely after the procedure, carotid-femoral pulse wave velocity (cfPWV) and brachial-ankle pulse wave velocity (baPWV) were used as indicators of arterial stiffness. Aortic pressures and aortic augmentation index corrected for heart rate [AIx@75] were used to assess aortic hemodynamics.
Results: There was a statistically significant increase in measurements of arterial stiffness (7.7 ± 1.5 vs 8.3 ± 1.9 m/s for cfPWV and 1931 ± 577 vs. 2469 ± 682 cm/s with p = 0.006 and p0.05). Peripheral pulse pressure (p = 0.047) increased significantly and peripheral DBP (p = 0.05) decreased significantly.
Conclusions: Our study led to the observation that patients undergoing TAVI present with an increase in arterial stiffness in the acute phase after the procedure, accompanied by an improvement of wave reflections. At the same time, a dissociation between aortic and peripheral BP after TAVI was observed, which may indicate important clinical value.



4.8
PLACENTAL NA/K-ATPASE INHIBITOR MARINOBUFAGENIN INDUCES ARTERIAL WALL FIBROSIS IN PREECLAMPSIA

Olga Fedorova [1], Natalia Aglakova [2], Yulia Grigorova [3], Vitalily Reznik [4], Valintina Zernetkina [3], Wen Wei [3], Edward Lakatta [3], Alexei Bagrov [5]
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Background: Previous studies implicated cardiotonic steroids, including Na/K-ATPase inhibitor marinobufagenin (MBG), in the pathogenesis of preeclampsia (PE). Immunoneutralization of heightened MBG by Digibind, a digoxin antibody, reduces blood pressure (BP) in patients with PE, and anti-MBG monoclonal antibody lessens BP in a rat model of PE. Recently, we demonstrated that MBG induces fibrosis in cardiovascular tissues via mechanism involving inhibition of Fli1, a nuclear transcription factor and a negative regulator of collagen-1 synthesis.
Objectives and Methods: We hypothesized that in PE, elevated placental MBG levels is associated with development of fibrosis of umbilical arteries. Thirty patients with PE (mean BP = 118 ± 4 mmHg; 29 ± 2 years; 35 weeks gest. age) and 26 gestational age-matched normal pregnant subjects (mean BP = 92 ± 2 mmHg; controls) were enrolled in the clinical study.