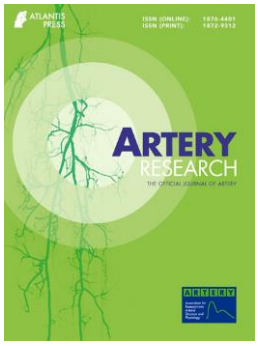

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P3.04: IS MALADAPTIVE CAROTID REMODELING A FEATURE OF TYPE 2 DIABETES?

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P3.04
IS MALADAPTIVE CAROTID REMODELING A FEATURE OF TYPE 2 DIABETES?

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A maladaptive carotid remodeling was described in type 2 diabetes mellitus. However, it is still unknown whether it is a feature of diabetes per se, or rather a consequence of the frequent comorbidity with hypertension. We therefore studied the impact of diabetes, hypertension, and their combination on carotid geometry and remodeling, recruiting to this aim 139 subjects (31 normotensives-NT, 38 hypertensives-HT, 24 diabetic normotensives-DMNT, and 46 diabetic hypertensives-DMHT). Common carotid diameter, intima-media thickness (IMT) and distension were determined using a real-time echo-tracking system "Carotid Studio". Local pulse pressure was obtained by applanation tonometry. Carotid static and pulsatile circumferential wall stress was calculated by Lamé equations. IMT was increasingly higher in the four groups, reaching the highest value in DMHT. As compared to NT, carotid lumen diameter was increased in HT and DMHT, but not in DMNT. HT showed significantly higher circumferential static wall stress than DMHT, while DMNT and NT had similar and significantly lower values. Carotid pulse pressure was similarly increased in DMHT and HT. In a logistic regression model adjusted for confounders, hypertension carried an increased risk (OR 2.33; CI-95% 1.02-5.63) of a widened lumen diameter, whereas diabetes carried an increased risk of IMT above the median value (OR2.49; 1.09-5.68). Hypertension, but not diabetes, was associated to an increased static and pulsatile stress (OR7.74; 2.76-21.72, and 4.86; 1.95-12.10). In conclusion, maladaptive remodeling, previously reported in diabetic patients, is conceivably attributable to the concomitant presence of hypertension.

Parameter	NT	HT	DMNT	DMHT	p (trend)
Lumen diameter (mm)	5.6±1.0	6.3±1.1*	5.9±0.6	6.3±1.1*	0.02
IMT (mm)	0.72±0.09	0.76±0.18	0.78±0.13*	0.81±0.14*	0.04
Static wall stress (kPa)	48.7±12.7	60.4±13.3‡*#	48.8±12.6†	54.6±12.2‡*†	0.001
Pulsatile wall stress (kPa)	27.0±6.2	36.0±12.3‡*	27.6±6.3†	32.5±9.0‡*	0.001
Pulse pressure (mmHg)	43.6±8.9	54.2±15.9‡*	44.7±8.9	53.8±11.8‡*	<0.001

*$p<0.05$ vs NT; † $p<0.05$ vs HT; ‡$p<0.05$ vs DMNT; # $p<0.05$ vs DMHT.

P3.05
DERANGED VASCULAR-VENTRICULAR COUPLING IN HEART FAILURE PATIENTS WITH DEPRESSED LEFT VENTRICULAR CONTRACTILITY: IMPORTANCE OF AORTIC CHARACTERISTIC IMPEDANCE

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Deranged vascular-ventricular coupling (VVC) occurs in heart failure (HF) when arterial elastance (Ea) is increased and/or left ventricular (LV) elastance (Elv) is depressed, leading to increased coupling ratio Ea/Elv. While calculation of Ea is simple (below), it represents the 'lumped' elastance of the arterial tree some of which is attributable to aortic characteristic impedance (Zc) which is more difficult to quantify. We examined the importance of Zc for VVC in 22 patients (7 with HF, 31%; 15 non-HF controls) who underwent echocardiography and analysis of central pressure waveform synthesised from radial artery pressure (SphygmoCor, AtCor Medical, Sydney, Australia). LV outflow tract Doppler recordings were digitised (custom software) and Zc was calculated as the ratio between pressure and flow in early systole before onset of wave reflections. Ea (LV end-systolic pressure / stroke volume (SV)) and Elv (single-beat method) were estimated; compliance (SV / brachial pulse pressure) and total peripheral resistance (TPR: mean arterial pressure / cardiac output) were computed. We found that HF patients had deranged VVC largely due to higher Ea (Table 1). Zc correlated strongly to Ea ($r=0.65$; $p=0.02$) and to Ea/Elv (0.80; $p=0.001$) but neither to Elv ($p=0.81$) nor higher age ($p=0.12$). In conclusion, elevated Zc contributes to the deranged VVC seen in systolic HF.

Variable	HF	Non-HF	p-Value
Age (years)	70 ± 11	54 ± 18	0.045
LVEF (%)	29 ± 15	52 ± 17	0.023
Compliance (mL/mmHg)	0.7 ± 0.3	1.1 ± 0.5	0.07
TPR (mmHg·s/mL)	0.03 ± 0.01	0.04 ± 0.04	0.84
Ea (mmHg/mL)	3.0 ± 1.5	1.7 ± 0.4	0.029
Zc (mmHg·s/mL)	0.39 ± 0.08	0.20 ± 0.06	0.01
Elv (mmHg/mL)	1.7 ± 1.0	2.3 ± 0.3	0.07
VVC ratio: Ea/Elv	0.75 ± 0.17	2.3 ± 1.5	<0.001

P3.06
AORTIC STIFFNESS AND VITAMIN D ARE INDEPENDENT MARKERS OF AORTIC CALCIFICATION IN PATIENTS WITH PERIPHERAL ARTERY DISEASE

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Background: Arterial stiffness is a significant determinant of cardiovascular risk and is related to vascular calcification. Vitamin D may regulate arterial calcification and has been associated with cardiovascular survival benefits. However, data about the relationship between arterial stiffness, aortic calcification, and vitamin D levels in patients with peripheral artery disease (PAD) are limited.
Aim: To examine association between aortic calcification, arterial stiffness, and vitamin D levels in patients with symptomatic PAD and in healthy individuals.
Materials and methods: We studied 78 males with PAD (mean age 63±7 years) and 69 healthy men (mean age 54±7 years). Aortic pulse wave velocity (aPWV) and augmentation index (AIx@75) were determined by applanation tonometry using the Sphygmocor device. Aortic calcification score (ACS) was quantified by computed tomography. Serum 25-hydroxyvitamin D ((25(OH)D) level was measured using a radioimmune assay.
Results: ACS (6.4±5.3 *vs* 0.7±1.7(cm^3); $p<0.001$), aPWV (10.1±2.5 *vs* 7.6±1.6(m/s); $p<0.001$), AIx@75 (28.2±8.1 *vs* 13.7±11.0(%); $p<0.001$), and 25(OH)D (37.7±14.0 *vs* 50.3±16.3(nmol/L); $p<0.001$) were different for the study groups. There was linear relationship between aPWV and ACS for the patients ($p=0.02$) and for the controls ($p=0.049$). 25(OH)D was associated with ACS only in the patient group ($p=0.05$). In multivariate analysis ACS was independently determined by aPWV and 25(OH)D in patients with PAD ($R^2=0.42$; $p<0.0001$).
Conclusion: These results indicate that calcification of the thoracic and abdominal aorta is independently associated with aortic stiffness and serum 25(OH)D level in patients with PAD. Aortic stiffness and abnormal vitamin D level may contribute to vascular calcification in these patients.

P3.07
OXYGEN CONSUMPTION KINETICS IN SUPRA-ANAEROBIC THRESHOLD CONSTANT LOAD EXERCISES ALLOW TO QUANTIFY IN TRAINED AND UNTRAINED SUBJECTS CYTOCHROME C-OXIDASE INHIBITION BY NITRIC OXIDE AND SHOW THIS DIRECT EFFECT AFTER NITRATE

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This work aims to highlight by VO_2 kinetics in constant load supra anaerobic threshold (AT) tests, the nitric oxide (NO) reversible direct inhibitory action on cytochromeC oxidase (cox). This results in decreased ability to oxidise electron flow (EF), by cytochromeC carried, in water, compensated by electron turnover (ET) increment via cox not yet NO bound, thus giving EF continuity along the respiratory chain O_2 ward. When NO production is increased and/or O_2 mitochondrial concentration decreased, as during intense effort, this compensation gradually weakened, owing to cox NO free oxidative capacity end limit, which approaches its saturation, but at the same time allows an O_2 saving and its diffusion deeper in tissues around vessels. O_2 saving effect is quantifiable in two bicycle constant load exercises by VO_2