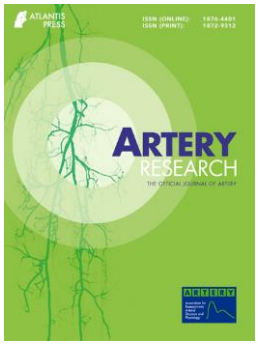

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# P1.02: INCREASED CAROTID IMT IN PATIENTS WITH TYPE 2 DIABETES FREE OF CARDIOVASCULAR COMPLICATIONS APPEARS TO BE AN ADAPTIVE MECHANISM TO AN INCREASED WALL STRESS MORE THAN ATHEROMASIC DEGENERATION

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# Poster Presentation Abstracts

## P1 Clinical Science 1

### P1.01
### MARKER OF ARTERIAL STIFFNESS IN CHRONIC KIDNEY DISEASE – A PROSPECTIVE COHORT STUDY

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**Objectives:** Parameters associated with elevated arterial stiffness and structural vascular remodeling, like aortic pulse wave velocity, have been identified as important contributors to predict cardiovascular outcome in end stage renal disease but less is known about their value in early stages of chronic kidney disease (CKD).
**Methods:** In order to investigate the progression of arterial stiffness markers in CKD we prospectively studied a cohort of 135 (41% female) patients with CKD stages 2/3/4 (N=20/75/40), and 89 (49% female) controls by cross sectional analysis in a first step. Subsequently we were able to follow up 129 patients for an average period of 42 months to evaluate the predictive value of arterial stiffness by means of central hemodynamics, pulse pressure amplification (PPA) and aortic pulse wave velocity (aPWV). Renal endpoints were halving of renal function and/or start of renal replacement therapy.
**Results:** With respect to arterial function in cross sectional analysis augmentation index (AIx) and aortic pulse wave velocity (PWV) were significantly increased in CKD patients, whereas pulse pressure amplification (PPA) was reduced as compared to control subjects. In longitudinal analysis stratification according to CKD stage 2-4 PPA predicted renal endpoints whereas PWV was associated with mortality.
**Conclusion:** PPA and PWV have been evidenced as predictor for cardiovascular morbidity and mortality. Within this cohort PPA was associated with the progression of CKD and PWV with mortality.

### P1.02
### INCREASED CAROTID IMT IN PATIENTS WITH TYPE 2 DIABETES FREE OF CARDIOVASCULAR COMPLICATIONS APPEARS TO BE AN ADAPTIVE MECHANISM TO AN INCREASED WALL STRESS MORE THAN ATHEROMASIC DEGENERATION

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Type 2 diabetes (DM2) and poor glycemic control adversely affect common carotid intima media thickness (IMT), considered marker of preclinical atherosclerosis. However, studies evaluating the effect of DM2 and glucose levels on IMT did not consider carotid diameter, known to affect IMT. A certain IMT increase could reflect a mutual adjustment between diameter and wall thickness aimed to maintain constant wall tensile stress (WTS).
**Aim:** To compare carotid IMT, luminal diameter, WTS and local wave speed (WS) between patients with uncomplicated DM2 and healthy controls.
**Methods:** Eighty-four patients with well controlled DM2 (HbA1c <7.8%) and 84 controls matched for sex, age and BMI. were studied by radiofrequency-based carotid ultrasound (QIMT® and QAS®, Esaote).
**Results:** DM2 against controls had higher ($p<0.0001$) IMT (720±131 vs. 620±76 μm), luminal diameter (6.6±0.6 vs. 6.0±0.7 mm), WS (8.3.6±1.7 vs. 6.5±1.2 m/s) and pulse pressure (58±13 vs. 47±8 mmHg), but comparable WTS (49±8 vs. 50±14 kPa; $p=0.82$). In the entire population, fasting glucose was not independently related to IMT, but was related to carotid diameter (together with male sex and waist), pulse pressure and local WS (together with age and antihypertensive treatment). In DM2, HbA1c was independently related to carotid diameter, pulse pressure and WS.
**Conclusions:** Chronically increased plasma glucose levels may induce intrinsic stiffening of large artery and widening of pulse pressure. Increased pulsatile load in stiff arteries causes luminal dilatation and increases WTS, triggering an increase in arterial wall thickness. Hyperglycaemia affects arterial wall, but through a "sclerotic" more than "atherogenic" mechanism.

### P1.03
### LEFT VENTRICULAR REMODELING: IMPACT OF GLOBAL, REGIONAL AND LOCAL AORTIC STIFFNESS

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**Objectives:** Left ventricular (LV) concentric remodeling predicts all cause and CV mortality in various subsets of patients. Large arteries stiffness determines cardiac afterload and therefore LV remodeling. Since elastic properties of the arterial wall vary along the aortic pathway, we hypothesized that stiffness of consecutive aortic segments could have different impact on LV remodeling.
**Methods:** Global aortic stiffness was obtained from carotid-femoral pulse wave velocity (cfPWV, m/s) aplanation tonometry (Pulse Pen), and regional stiffness from aortic arch PWV phase contrast MRI. Local stiffness (m/s) was calculated in ascending (aaPWV) and descending aorta (daPWV) using cine MRI acquisition and surface change estimation (Bramwell-Hill equation). LV remodeling, MRI estimated, was expressed as LV mass over end diastolic volume ratio (M/V).
**Results:** We evaluated 146 patients (41±15 years old; 43.8% women) free of overt CV disease (hypertensives: 9.6%; smokers: 8.9%; diabetics: 0.6%; BMI: 23.8±3.5). In multivariate regression analysis cfPWV and aaPWV strongly correlated to M/V (partial $R^2=0.07$, $p=0.0011$; and partial $R^2=0.10$, $p=0.0001$, respectively), after adjustment for age, sex, BMI and brachial MBP. Challenged together, both cfPWV and aaPWV independently determined M/V, with 5% and 8% of explained variance, respectively. To a lower extent, daPWV (Partial $R^2=0.04$, $p=0.0115$) was also independently related to M/V. Arch PWV was not independently associated with M/V.
**Conclusions:** In this cross-sectional study, stiffness of diverse segments of the aorta had different influence on LV remodeling, except arch PWV. Stiffness of ascending aorta and the carotid-femoral pathway were complementary and also the strongest explanatory variables for LV remodeling.