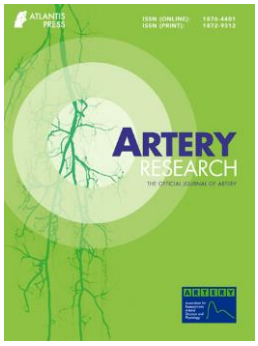

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P9.08: THE ASSOCIATION BETWEEN METABOLIC SYNDROME AND AORTIC STIFFNESS IN GENERAL POPULATION

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(129.3±74.0) and without prevalent CVD(120.2±68.6). Participants with a high waist (4th quartile, gender specific) had higher IL18 levels than those with a low waist(1th quartile)(125.9±77.9 versus 117.2±61.0). The increase in IL18 was accompanied by an increase in subclinical atherosclerosis, as reflected by a lower ABI (1.09 versus 1.11), a thicker IMT(0.86 versus 0.83 mm) and increased arterial stiffness, as reflected by an increased PWV(10.3 versus 9.6 m/s) and an increase in all derived central pressure parameters.
Conclusion: In our population-based cohort obesity, as reflected by an increased waist circumference, was accompanied by increased IL-18 levels and an increase in non-invasively determined subclinical atherosclerosis. Our data support the hypothesis that the increased CVD risk in obesity might be caused by increased inflammation, although prospective studies are needed to conclude on causality of this relation.

P9.06
SERUM URIC ACID LEVELS AND ARTERIAL STIFFNESS AND CARDIAC AND CAROTID ARTERY STRUCTURE IN A GENERAL POPULATION IN NORTHERN ITALY

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Background: The relationship between serum uric acid (UA) levels and CV disease has been described since the late 19th century.The role of UA as an independent risk factor for CV events and its association with TOD is however less clear. Aim of the study was to assess the relationship between UA and TOD in a general population (Vobarno Study).
Methods: 385 subjects (age 56±9yrs, 44%males, 64% hypertensives, 32% treated) underwent laboratory examinations and clinic and 24 hours BP measurement.Left ventricular and carotid artery structure were assessed by ultrasound and carotid-femoral PWV was measured using Complior.
Results: Subjects with increased UA (>6 mg/dl in ♀ and >7 mg/dl in ♂) were older, had greater BMI, higher BP, glucose, cholesterol and triglycerides levels and lower HDL cholesterol and e-GFR.Subjects with increased UA had also increased PWV (11.1±4.1 vs 13.3±3.7m/sec,$p<0.0001$), and a slight increase in left ventricular mass index (LVMI) (38.7±10.6 vs 43.0±11gr/$m^{2.7}$, $p<0.05$) and IMT (Meanmax 1.1±0.28 vs 1.2±0.29 mm, $p<0.05$).After adjusting for confounders, including e-GFR, in a multivariable model, PWV was significantly greater in subjects with increased UA (11.1±2.41 vs 13.4±3.7 m/sec, $p<0.001$), while no significant difference in LVMI and IMT was observed. A significant correlation between UA levels and PWV($r=0.279$, $p<0.001$),LVMI($r=0.157$,$p<0.001$),meanmax IMT($r=0.159$,$p<0.001$) was observed. After adjusting for confounders, serum UA levels were independently correlated to PWV, but not to LVMI and IMT.
Conclusions: Subjects with increased UA have increased arterial stiffness, but comparable left ventricular anatomy and carotid artery structure. The increase in arterial stiffness might contribute to the higher CV risk in these subjects.

P9.07
RELATION OF CENTRAL AND BRACHIAL BLOOD PRESSURE TO LEFT VENTRICULAR HYPERTROPHY.
THE CZECH POST-MONICA STUDY

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Objective: Recently central aortic blood pressures were shown to be better predictors of target organ damage, cardiovascular events and mortality when compared with conventional brachial blood pressure. Whether central blood pressure is a better predictor of left ventricular hypertrophy (LVH) determined by electrocardiographic criteria is not know.
Methods: Radial applanation tonometry and ECG were performed in 563 individuals from the Czech post-MONICA study (a randomly selected 1% representative population sample, mean age 46±11 years, 44% of men). LVH was determined using electrocardiographic criteria. Brachial blood pressure was measured using mercury sphygmomanometer according to standardized protocol; central systolic blood pressure was derived from radial pulse wave using generalised transfer function.
Results: Of 563 subjects 39 (7%) had ECG signs of LVH. In the univariate analysis patients with LVH were older (50.4±11.3 vs. 46.6±11.3, $p=0.04$), had higher central systolic (129.7±31.8 vs. 116.7±16.8, $p< 0.0001$), diastolic (83.3±10.3 vs. 79.2±9.7, $p=0.04$), pulse (46.5±13.1 vs. 37.5±12, $p< 0.0001$) and mean pressure (103.5±14.8 vs.96±11.8, $p< 0.01$), higher brachial systolic (136.6±19.4 vs. 122.8±14.8, $p< 0.0001$),pulse (67.2±19.3 vs. 52.6±16.8, $p< 0.0001$) and mean (91.8±10.3 vs. 87.2±8.9,$p=0.02$) pressure and aortic pulse wave velocity (8.3±2.1 vs. 7.5±1.8, $p=0.02$) then subjects without LVH. However, in the binary logistic regression only central systolic pressure (OR 2.2, 95% CI 1.4-3.4, $p=0.001$) and male sex (OR 4.8, 95% CI 1.3-17.6, $p=0.02$) were predictors of LVH.
Conclusion: Noninvasively determined central systolic blood pressure is more strongly related to LVH determined by electrocardiographic criteria then brachial systolic and pulse pressure. This is a further support of closer association of central blood pressure with target organ damage. Prospective studies with central blood pressure as a treatment target will be needed in the future.

P9.08
THE ASSOCIATION BETWEEN METABOLIC SYNDROME AND AORTIC STIFFNESS IN GENERAL POPULATION

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Background: Despite being a cluster of conventional risk factors, metabolic syndrome (MetSy) has been recognized as independent predictor of cardiovascular diseases. We aimed to establish the association between metabolic syndrome and aortic pulse wave velocity (aPWV) as a measure of arterial stiffness in Czech general population.
Methods: 576 subjects (mean age 48.03y (SD 14.8)), 41.5% males), a Pilsen sub-sample of postMONICA survey, were included into cross-sectional study. APWV was estimated using Sphygmocor device, subjects with MetSy were identified using common NCEP-ATPIII definition.
Results: Subjects with MetSy showed signifficantly higher aPWV (9.02 vs. 7.42 m/sec, $p<0.001$), also if diabetic (8.75 vs. 7.18, $p<0.001$) or diabetic and hypertensive patients (7.96 vs. 6.84, $p<0.001$) were excluded from the analysis (p value adjusted for age).
The significance of association between MetSy and aPWV remained significant after adjustment for age, gender, current smoking, mean arterial pressure, serum glucose and other risk factors as potential confounders ($b=-0,088$, $p=0.023$).
Conclusion: In our sample of general population, we found that MetSy represents an additive risk factor of increased aortic stiffness independent of age, blood pressure, glucose status and other conventional factors.

P9.09
DIFFERENCE IN AGE-RELATED PATTERNS OF ARTERIAL STIFFNESS AND WAVE REFLECTIONS AMONG PATIENTS WITH KIDNEY DISEASE: RESULTS OF THE UK RESEARCH ALLIANCE INTO KIDNEY DISEASE AND ARTERIAL STIFFNESS (UREKA) COLLABORATION

L. A. Tomlinson [1], B. Caplin [2], S. J. Carr [3], C. Delles [4], C. J. Ferro [5], J. Goddard [6], S. G. Holt [7], P. A. Kalra [8], P. S. Lacy [3], C. M. McEniery [1], C. W. McIntyre [9], E. P. McQuarrie [4], C. Rajkumar [7], T. Savage [10], M. W. Taal [9], C. R. Tompson [10], J. N. Townend [5], D. J. Webb [6], D. C. Wheeler [2], I. B. Wilkinson [1]
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Background: Patients with CKD may have higher aortic PWV (aPWV) and augmentation index (AIx) but studies are limited by size and lack of control population and differences may be due to joint risk factors. We examined whether aPWV and AIx are increased in CKD patients with no vascular comorbidities compared to controls and how change with age varies between populations.