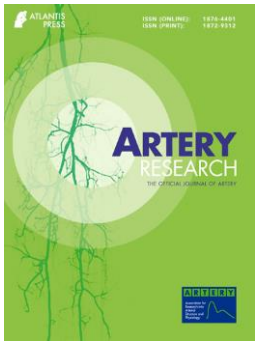

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P9.05: IMPACT OF INTERLEUKIN-18 AND OBESITY ON SUBCLINICAL ATHEROSCLEROSIS IN THE GENERAL POPULATION

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P9.02

TELEVISION TIME IS ADVERSELY ASSOCIATED WITH ARTERIAL STIFFNESS IN YOUNG ADULTS: THE AMSTERDAM GROWTH AND HEALTH LONGITUDINAL STUDY

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Purpose: To investigate 1) the association between television (TV) viewing and arterial stiffness (AS); and 2) whether any such association is independent of other lifestyle risk factors (RFs), most notably vigorous habitual physical activity (HPA), and/or is explained by TV-time-related adverse associations with biological RFs.
Methods: We investigated repeated measures (at ages 32 and 36) of TV time and other RFs among 373 subjects in whom stiffness of 3 large arteries was assessed with ultrasonography at age 36. Generalized estimating equations were used to determine the average differences in TV time between subjects across sex-specific tertiles (T1-3) of the inversed carotid, brachial and femoral distensibility (DC) and compliance (CC) coefficients, and the carotid Young's elastic modulus (E_{inc}).
Results: Compared with subjects in T1 (less stiff), those in T3 (stiffer arteries) of the carotid DC, CC and E_{inc} spent more time (*in min/day*) [19.9 (95%CI:5.9;33.9), 16.7 (2.3;31.1) and 17.6 (3.7;31.5), respectively] on TV viewing during the 4 preceding years (Table, model 1). These differences were independent of vigorous HPA and other lifestyle RFs (model 2-3), and only in part (up to 31%) explained by TV-time-related associations with biological RFs (model 4). Qualitatively similar results were found for femoral, but not brachial, stiffness estimates.
Discussion: TV time is associated with higher levels of carotid and femoral stiffness in young adults, independently of HPA and other RFs. Promotion of more physical activity *but also* less sedentarism (two distinct behaviours) should, therefore, be encouraged to prevent AS and related *sequelae*.

Model: adjustments	Carotid DC (T3 vs T1)		Carotid CC (T3 vs T1)		Carotid E_{inc} (T3 vs T1)	
	β	95%CI	β	95%CI	β	95%CI
1. Crude + sex, time and height	19.9	5.9;33.9	16.7	2.3;31.1	17.6	3.7;31.5
2. 1+ vigorous HPA	20.0	6.1;33.9	16.4	2.1;30.8	17.3	3.4;31.1
3. 2+ smoking/alcohol statuses and energy intake	22.4	8.7;36.1	18.4	4.2;32.5	19.7	6.0;33.4
4. 3+ MAP, sum of skinfolds, VO_{2max},total-to-HDL cholesterol, triglycerides, heart rate	15.4	1.2;29.6	14.1	-0.3;28.4	14.7	0.7;18.6

P9.03

WAVE PRESSURE IS HIGHER IN ATENOLOL-TREATED INDIVIDUALS AND INDEPENDENTLY PREDICTS CARDIOVASCULAR EVENTS IN THE CAFE SUBSTUDY OF ASCOT

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Background: Wave reflection accounts for augmentation of aortic pressure, but most studies have failed to show an independent relationship between augmentation index (AIx) and cardiovascular (CV) events. We hypothesise this is because conventional pulse wave analysis does not distinguish wave pressure from that attributable to charging and recoil of the aorta.
Aim: To identify if the wave pressure integral (WPI) is differentially affected by atenolol- vs. amlodipine-based therapy and is an independent predictor of CV events in the CAFE sub-study of the ASCOT trial.
Methods: Radial pressure waveforms were acquired using Sphygmocor in 2070 subjects (63±8 yrs; 1675 male) and WPI was calculated. A total of 134 CV events accrued over a median 3.4 years of follow up.
Results: WPI was higher in the atenolol arm (786±284 vs 744±253 mmHg.s, $p<0.001$), despite similar brachial systolic BP (134±16 vs 134±14 mmHg, $p=0.78$). WPI predicted total CV events (Hazard ratio =2.5 [1.51-4.14], $p<0.001$), and remained significant after adjustment for age, sex, treatment, number of CV risk factors, brachial BP, central systolic pressure, central pulse pressure, AIx and heart rate. Central systolic pressure and AIx did not predict CV events.
Conclusions: Wave pressure was higher in the atenolol arm and independently predicted CV events in the CAFE study. WPI is easily calculated from the BP waveform and may have potential for optimization of therapy and risk evaluation.

P9.04

AORTIC STIFFNESS IS REDUCED BEYOND BLOOD PRESSURE LOWERING BY SHORT AND LONG-TERM ANTIHYPERTENSIVE TREATMENT: A META-ANALYSIS OF INDIVIDUAL DATA IN 294 PATIENTS

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Background: Arterial stiffness is an independent predictor of cardiovascular events and mortality in hypertensive patients. The influence of different antihypertensive drug classes on improving arterial stiffness beyond blood pressure reduction was not well demonstrated because of lack of power studies related to this subject. We aim to determine if the artery stiffness can be improved due to antihypertensive treatment independently of blood pressure lowering.
Methods: We conducted a meta-analysis of individual data from 15 randomized, controlled, double-blind, parallel group trials performed in our laboratory between 1987 and 1994. The primary endpoint was the changes in carotid-femoral pulse wave velocity (PWV) after treatment in 294 patients with mild to moderate essential hypertension untreated. Treatments tested were placebo (n = 88), angiotensin converting enzyme inhibitors (ACEI) (n = 75), calcium antagonists (CA) (n = 75), beta-blocker (n = 30), and diuretic (n = 26).
Results: In the short and long-term trials, PWV decreased significantly by -0.75 m/s and -1.3 m/s in the active treatment group compared to by +0.17 m/s and -0.44 m/s in the placebo group respectively. Active treatment was independently related to the changes in PWV and explained 5% and 4% of the variance in the short and long-term trials respectively. In the short-term trials, ACEI was more effective than CA and placebo on improving arterial stiffness. In the long-term trials, ACEI, CA, beta-blocker, and diuretic reduced significantly PWV compared to placebo.
Conclusion: Our study shows that antihypertensive treatment improves the arterial stiffness beyond its effect on blood pressure.

P9.05

IMPACT OF INTERLEUKIN-18 AND OBESITY ON SUBCLINICAL ATHEROSCLEROSIS IN THE GENERAL POPULATION

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Objective: The prevalence of obesity and consequently the risk for cardiovascular diseases(CVD) increases dramatically worldwide. It has been suggested that increased serum levels of interleukin-18(IL18), reflecting systemic inflammation, could be the missing link between obesity and increased cardiovascular risk. We hypothesized that obese individuals show increased IL18 levels and that this would be accompanied by increased subclinical atherosclerosis (non-invasively determined) in our population-based cohort.
Methods & Results: We measured IL18, waist, and subclinical atherosclerosis with a panel of non-invasive measurements of atherosclerosis(NIMA) in 1517 participants of the Nijmegen community. We determined intima-media thickness(IMT), pulse wave velocity(PWV), pulse wave analysis and ankle-brachial index(ABI). IL18(pg/ml) was higher in men(128.7±69.2) compared to women (114.2±68.7) but IL18 was not different between participants with

(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.