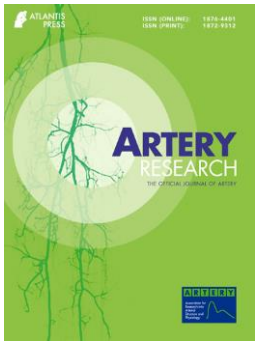

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P9.03: WAVE PRESSURE IS HIGHER IN ATENOLOL-TREATED INDIVIDUALS AND INDEPENDENTLY PREDICTS CARDIOVASCULAR EVENTS IN THE CAFE SUBSTUDY OF ASCOT

J.E. Davies, A. Malaweera, P.S. Lacy, K. Cruickshank, A. Stanton, D. Collier, H. Thurston, B. Williams, K.H. Parker, S.A.M. Thom, A.D. Hughes

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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