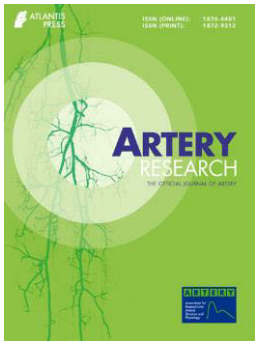

Artery Research

ISSN (Online): 1876-4401 **ISSN (Print): 1872-9312**
Journal Home Page: https://www.atlantis-press.com/journals/artres

P1.24: AORTIC PULSE WAVE VELOCITY IS NOT ASSOCIATED WITH ALL-CAUSE MORTALITY IN YOUNG, LOW RISK, FRENCH POPULATION

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To cite this article: B. Pannier, F. Thomas, L. Guize, K. Bean, B. Jego, A. Benetos (2008) P1.24: AORTIC PULSE WAVE VELOCITY IS NOT ASSOCIATED WITH ALL-CAUSE MORTALITY IN YOUNG, LOW RISK, FRENCH POPULATION, Artery Research 2:3, 97–98, DOI: https://doi.org/10.1016/j.artres.2008.08.331

To link to this article: https://doi.org/10.1016/j.artres.2008.08.331

Published online: 21 December 2019

guideline. The 236 subjects (mean age 51 ± 11 years, range 26-77, 61% men) were analyzed.
Results: The linear regression analysis showed a significant correlation between UACR and baPWV (beta = 9.52; P < 0.0001), that was independent by multiple linear regression model including, as independent variables, age, gender, body mass index, mean arterial pressure, total cholesterol and smoking (beta = 5.66; p = 0.0115). Compared with those in the lowest UACR quartile, subjects in the highest quartile (UACR > 11.7 mg/g) showed higher baPWV (1492 ± 213 vs. 1655 ± 313 cm/sec) with general linear model adjusted for age, gender, body mass index, mean arterial pressure, total cholesterol and smoking (B=98.5; p = 0.0084).
Conclusion: Hypertensive subjects with urinary albumin excretion in the upper normal range were not free from target organ damage. The present study suggests that the current threshold of microalbuminuria should be lowered.

doi:10.1016/j.artres.2008.08.327

P1.21
THE RISK OF HEART FAILURE IS INCREASED IN SUBJECTS WITH RAISED ARTERIAL STIFFNESS: THE ROTTERDAM STUDY

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Objective: The purpose of the present study is to investigate whether subjects with stiff arteries have an increased risk of heart failure.
Methods: The present study is performed within the framework of the Rotterdam study. Blood pressure, aortic pulse wave velocity and carotid distensibility measurements were obtained. Subjects with heart failure at baseline were excluded. We included 4121 subjects with blood pressure measurements, 3290 subjects with information on aortic pulse wave velocity and 2936 subjects with carotid distensibility measurements. Cox proportional hazard models, adjusted for cardiovascular risk factors, were performed to investigate the risk of heart failure associated with blood pressure and arterial stiffness.
Results: The mean age of the subjects was 72 years, 41,5 % was men. After a mean follow- up of 4.1 years 254 subjects had a heart failure. Hazard ratios and corresponding 95% CI of heart failure for systolic, diastolic, pulse and mean arterial pressure were 1.21 (1.08-1.36), 0.94 (0.83-1.06), 1.31 (1.17-1.46) and 1.08 (0.96-1.22), respectively.
After including both systolic and pulse pressure in one model, only the pulse pressure predicted incident heart failure; estimates for systolic and pulse pressure were 0.90(0.69-1.18) and 1.40(1.07-1.85), respectively. Aortic pulse wave velocity increased the risk of heart failure in subjects up to 70 years (HR 1.72;1.23-2.40), whereas the carotid distensibility did not.
Conclusions: The pulsatile components of blood pressure and aortic stiffness are associated with the risk heart failure in the general population.

doi:10.1016/j.artres.2008.08.328

P1.22
POORER LUNG FUNCTION IS ASSOCIATED WITH GREATER PERIPHERAL ARTERIAL STIFFNESS IN YOUNG ADULTS: THE NORTHERN IRELAND YOUNG HEARTS PROJECT (NIYHP)

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Background & Aims: Associations of poorer lung function (LF) with atherosclerosis and/or arterial stiffness (AS) have been suggested as potential mechanisms explaining the increased cardiovascular risk associated to poorer LF (e.g. in COPD patients but also in the general population). We have therefore examined, in a population of young adults, whether: 1) LF was inversely associated with stiffness of central (i.e. aorta) and peripheral (i.e. upper and lower limbs) arterial segments; 2) these associations were similar in smokers and non-smokers; and 3) low-grade inflammation played a mediating role.
Methods: Subjects were 286 young adults (mean age of 23 yrs), participating in the NIYHP. LF [i.e. forced expiratory volume in 1s (FEV1) and forced vital capacity (FVC), expressed in L] was measured by spirometry. AS was assessed by measuring pulse wave velocity (PWV, in m/s) in 3 arterial segments.
Results: After adjustment for sex, age, height, weight, MAP, smoking and asthma status, both FEV1 and FVC were inversely associated with PWV of all 3 arterial segments, but more strongly and significantly so with PWV of the lower limb segment only: [b=-0.23 (95%CI:-0.38; -0.08), p=0.004 and b=-0.22 (-0.41;-0.02), p=0.029, respectively]. No significant interactions with smoking status were observed. Further adjustment for markers of low-grade inflammation (i.e. CRP and fibrinogen) did not attenuate the associations of FEV1 [b=-0.24 (-0.38;-0.08) or FVC [b=-0.22 (-0.42;-0.03)] with PWV of the lower limb.
Conclusions: Young adults with poorer LF have increased peripheral AS. We found no evidence that low-grade inflammation underlies this association, and other mechanisms need to be explored.

doi:10.1016/j.artres.2008.08.329

P1.23
BLOOD PRESSURE AND AUGMENTATION INDEX IN GENERAL POPULATION IN 5 YEARS FOLLOW-UP

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Objectives: The objective of the present study was to assess changes in blood pressure (BP) parameters and AI in general population after 5-years follow-up.
Methods: From the general population we recruited 197 members from random families (99 parent and 98 offspring (age at baseline: 51.4 and 25.5 years) who constituted 110 normotensives and 87 hypertensives). Initially and after 4.8±0.3 years we recorded the radial arterial waveform using the SphygmoCor device. We evaluated peripheral AI (pAI) and central AI (cAI).
Results: In both generations as well as in normo- and hypertensive groups we observed comparable increase in BMI and decrease in heart rate. We found higher increase in aortic SBP with lesser decrease in central DBP in offspring and in normotensives while cPP increase was higher in parent and in participants with initially diagnosed hypertension (p<0.005). We observed greater elevation of brachial SBP with simultaneous lesser reduction in DBP with similar increase in peripheral PP in offspring and in normotensives. Changes in pAI and cAI were more pronounced in younger generation and resulted respectively 4.4 vs 2.9(%); p=0.004 and 5.2 vs 3.7(%); p=0.001. Moreover we observed higher increase in pAI (4.6 vs 4.3(%); p=0.006) and in cAI (4.8 vs 4.6(%); p=0.005) in hypertensives.
Conclusions: Our findings indicate that AI increased in offspring and can be use as effective tool to detect the progressive increase in aortic stiffness in younger individuals. The aortic pulse pressure more effectively indicate age and blood pressure related changes in arterial wall stiffening than brachial pressure.

doi:10.1016/j.artres.2008.08.330

P1.24
AORTIC PULSE WAVE VELOCITY IS NOT ASSOCIATED WITH ALL-CAUSE MORTALITY IN YOUNG, LOW RISK, FRENCH POPULATION

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Objective: To evaluate the association between aortic pulse wave velocity (PWV) and all-cause mortality in a low to moderate risk population.
Methods: 1952 subjects (1319 men, 633 women), who benefited from a standard health check-up at the IPC center (Paris) in 1992/1993, had also an aortic PWV measurement. Mean follow-up was 13.4 ± 1.2 years, and 61 men (4.6%) and 18 women (2.8%) died. The population was divided in two groups of age (<60 and >=60 years). Cox regression model, including age, gender, tobacco, cholesterol, heart rate, blood pressure, glycaemia, assessed the risk of all-cause mortality for an increase of 1 m/sec of PWV (Hazard Ratio (HR), 95% CI).
Results: Age was 45.0±9.3 years in young and 64.5±3.8 years in old subjects. In overall population, PWV was 9.6±2.2 m/sec, and increased with age: 9.2±2.0 m/sec in youngest and 11.0±2.2 m/sec in oldest. After

adjustments, excepted on BP, HR for all-cause mortality risk associated with PWV was 1.12 (1.03-1.22), but after adjustment on all variables, relationship was no longer significant: HR=1.08 (0.98-1.18). Before 60 years, after adjustments, PWV-related risk was 1.09 (0.95-1.24), (NS), but it reached 1.22 (1.08-1.38), $p<0.02$, in patients >60 years.
Conclusion: In a low to moderate risk population, aortic PWV was significantly and independently associated with all-cause mortality only among subjects after 60 years. In such population, the direct impact of BP on aortic stiffness overcomes the intrinsic stiffness alterations which are linked to all-cause mortality, at least in young, low risk, subjects.

doi:10.1016/j.artres.2008.08.331

P1.25
ARTERIAL PROPERTIES IN RELATION TO GENETIC VARIATIONS IN THE ADDUCIN SUBUNITS IN A WHITE POPULATION

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Background: Adducin is a membrane skeleton protein, which consists either of α- and ß- or α- and γ-subunits. We investigated whether arterial characteristics might be related to the genes encoding *ADD1* (*Gly460Trp*), *ADD2* (*C1797T*) and *ADD3* (*A386G*).
Methods: We randomly recruited 1126 Flemish subjects (mean age, 43.8 years; 50.3% women). Using a wall-tracking ultrasound system, we measured the properties of the carotid, femoral and brachial arteries. We studied multivariate-adjusted phenotype-genotype associations, using a population- and family-based approach.
Results: In single-gene analyses, brachial diameter was 0.15 mm ($P=0.0022$) larger, and brachial distensibility and cross-sectional compliance were 1.55 10^{-3}/kPa ($P=0.013$) and 0.017 mm^2/kPa ($P=0.0029$) lower in *ADD3 GG* than *ADD3 AA* homozygotes with an additive effect of the *G* allele. In multiple-gene analyses, the association of brachial diameter and distensibility with the *ADD3 G* allele only occurred in *ADD1 GlyGly* homozygotes. Otherwise, the associations between the arterial phenotypes in the 3 vascular beds and the *ADD1* or *ADD2* polymorphisms were not significant. In family-based analyses, the multivariate-adjusted heritability was 0.52, 0.38 and 0.30 for brachial diameter, distensibility, and cross-sectional compliance, respectively ($P<0.001$). There was no evidence for population stratification ($0.07\leq P\leq 0.96$). Transmission of the mutated *ADD3 G* allele was associated with smaller brachial diameter in 342 informative offspring (-0.12 ± 0.04 mm; $P=0.0085$) and in 209 offspring, who were *ADD1 GlyGly* homozygotes (-0.14 ± 0.06 mm; $P=0.018$).
Conclusions: In *ADD1 GlyGly* homozygotes, the properties of the brachial artery are related to the *ADD3* (*A386G*) polymorphism, but the underlying mechanism needs further clarification.

doi:10.1016/j.artres.2008.08.332

P1.26
THE INFLUENCE OF ENDOTHELIAL NITRIC OXIDE SYNTHASE POLYMORPHISMS AND CURRENT SMOKING ON LARGE ARTERY STIFFNESS

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Background: Nitric oxide belongs to the most important factors influencing structural and functional properties of vessel wall. Both genetic and environmental factors may influence its metabolism. The aim of this study was to explore whether two common polymorphisms of endothelial nitric synthase (eNOS) may, jointly with smoking, influence the stiffness of large arteries, quantified by pulse wave velocity (PWV).
Methods: One hundred ninety four subjects free of manifest atherosclerosis or chronic cardiovascular pharmacotherapy were selected from population-based post-MONICA study. PWVs were measured using Sphygmocor® device between carotic and femoral arteries (aortic PWV) and between femoral and tibialis-posterior arteries (peripheral PWV). Two common eNOS polymorphisms, T786C and G894T, were assessed.
Results: Among current smokers (n=70), homo- or heterozygous carriers of T786C mutation (n=42) showed significantly higher peripheral PWV than normal genotype carriers (14.0 vs 10.7 m/sec, $p<0.002$); the same applied to the carriers of G894T mutation (n=41; 13.9 vs 11.0 m/sec, $p<0.015$). No differences were found in non-smokers, and neither of the eNOS polymorphisms influenced aortic PWV in our setting.
Conclusion: Genetically determined disorder of nitric oxide metabolism was associated with increased stiffness of peripheral muscular-type arteries in generally healthy, untreated subjects, but only in interaction with active smoking.
Supported by the EU Projects InGenious HyperCare and HyperGenes.

doi:10.1016/j.artres.2008.08.333

P1.27
INTERRELATIONSHIPS OF MONOCYTE COUNT WITH CAROTID INTIMA-MEDIA THICKNESS, AORTIC STIFFNESS AND PENILE DOPPLER FINDINGS, IN PATIENTS WITH VASCULOGENIC ERECTILE DYSFUNCTION

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Background: Erectile dysfunction (ED) has been associated with both systemic inflammation and generalized vascular disease. Monocyte count (MNC) represents a sensitive marker of inflammatory activity in atherosclerosis. We examined the possible associations between MNC, penile vascular damage and early atherosclerosis.
Methods: 145 consecutive ED patients were divided into three groups according to pharmacologically stimulated peak systolic velocity (PSV) values of cavernous arteries: Group A (venous occlusive disease), group B (mild arterial insufficiency) and group C (severe arterial insufficiency, PSV<25 cm/s). PSV shows the greatest flow velocity detectable in an artery throughout the systole. Ultrasound-determined intima media thickness (IMT) of carotid arteries and carotid-femoral pulse wave velocity (PWV) as an index of aortic stiffness were used to assess subclinical atherosclerosis.
Results: Patients with severe arterial insufficiency (n=44) compared to subjects in group B (n=41) and A (n=60) had increased IMT (0.96 vs 0.93 vs 0.87 mm, $P<0.05$) and PWV (9.3 vs 8.9 vs 8.5 m/s, $P<0.05$). They also exhibited higher MNC, compared to those of groups A and B (0.47 vs 0.44 vs 0.39 x 10^9/L, $P<0.05$), whereas there were no significant differences between the 3 groups as regards white cell counts. Furthermore, MNC remained significantly different between groups after adjustment for CRP, fibrinogen and risk factors, ($P<0.05$). MNC correlated with IMT ($r=0.23$, $P<0.05$), PWV ($r=0.27$, $P<0.01$) and PSV ($r=-0.26$, $P<0.01$).
Conclusions: Our study shows that there is an augmentation in MNC throughout increasing penile vascular damage and subclinical atherosclerosis. These findings may reflect the potential role of MNC as a marker of early atherosclerosis in ED patients.

doi:10.1016/j.artres.2008.08.334

P1.28
INFLUENCE OF AGE ON CAROTID ENDOTHELIAL FUNCTION AS DETERMINED BY HYPERCAPNIA INDUCED VASODILATATION

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Background: An increase in blood velocity-associated shear stress results in release of endothelial factors, causing endothelium-dependent flow mediated dilatation (FMD). Hypercapnia strongly stimulates cerebral blood flow velocity in the common carotid artery (CCA).
Objective: Test the reliability of hypercapnia induced FMD of the CCA and evaluate the stimulus response relationship (changes in blood velocity and diameter) for different age populations.
Methods: Hypercapnia was induced with inhalation of a gas mixture of 6.8% CO2, 74.5%N2, and 18.6% O2 for a period of 2 minutes in 19 healthy young