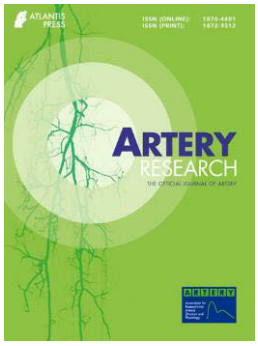


Artery Research

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

06.02: AORTIC AND CAROTID STIFFNESS IN OLDER ADULTS. THE ROTTERDAM STUDY

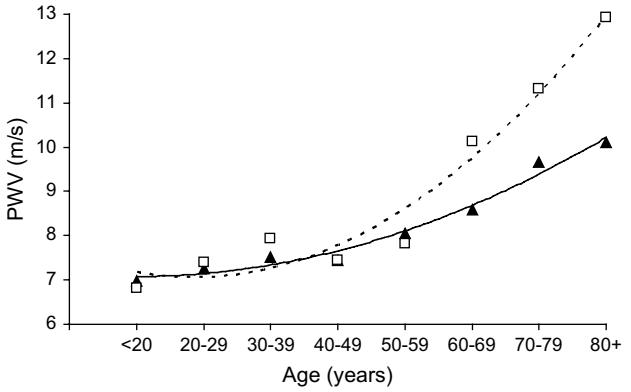
F.U.S. Mattace-Raso, N.M. van Popele, A. Hofman, J.C.M. Witteman

To cite this article: F.U.S. Mattace-Raso, N.M. van Popele, A. Hofman, J.C.M. Witteman (2007) 06.02: AORTIC AND CAROTID STIFFNESS IN OLDER ADULTS. THE ROTTERDAM STUDY, Artery Research 1:2, 50–50, DOI: https://doi.org/10.1016/j.artres.2007.07.047

To link to this article: https://doi.org/10.1016/j.artres.2007.07.047

Published online: 21 December 2019

pressure. In multivariate analyses, hypertension, diabetes, smoking and the presence of cardiovascular disease were independently associated with aortic PWV (R^2=0.65, P<0.001). In conclusion, aortic PWV appears to be a sensitive marker of cardiovascular risk in individuals aged over 50 years.



Age-related changes in aortic PWV in healthy controls (▲) versus diabetics (□)

† Anglo-Cardiff Collaborative Trial.

06.02

AORTIC AND CAROTID STIFFNESS IN OLDER ADULTS. THE ROTTERDAM STUDY

F.U.S. Mattace-Raso [1], N.M. van Popele [2], A. Hofman [2], J.C.M. Witteman [2]. *[1]Department of Internal Medicine, Erasmus University Medical Center, Rotterdam, Netherlands, [2]Department of Epidemiology & Biostatistics, Erasmus University Medical Center, Rotterdam, Netherlands*

The purpose of this study is to describe aortic (aPWV) and carotid stiffness (DC) in older adults according to age, gender and blood pressure. Information on both measures was available for 2766 subjects. The mean value of both aPWV and DC was lower in women than in men. The difference (95% CI) in aPWV was -0.99 (-0.81 to -1.18) (m/s) and the difference in DC was -1.62 (-1.35 to -1.82) (10^{-3}/kPa) for women as compared for men. With aging, both aorta and carotid artery become stiffer but the increase attenuates at high age. Women under 80 years of age had a less stiff aorta and a stiffer common carotid artery as compared to men of the same age. The relations of both measures of arterial stiffness with SBP and PP were non-linear, flattening off at higher levels, whereas the relation with DBP was non-linear and resembled a J-shape. We found a quadratic relationship between DC and aPWV: DC = 27.4 - 1.9 * (aPWV) + 0.04 * $(aPWV)^2$ [p total model ≤ 0.001], (R^2= -0.41, P<0.001). Subjects with increased aortic stiffness had a 30-fold increased risk of also having increased carotid stiffness, OR 31.2 (95% CI 20.9-46.4) (Figure 1).

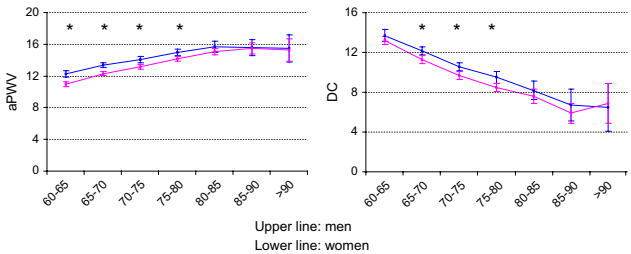


Figure 1. Mean (95% CI) aPWV and DC per 5-years age category in strata of gender. *p < 0.05 for difference between men and women.

06.03

NITRIC OXIDE AND ENDOTHELIUM-DERIVED HYPERPOLARIZING FACTOR REGULATE THE ADAPTATION OF HUMAN CONDUIT ARTERY MECHANICS TO CHANGES IN SHEAR STRESS

J. Bellien, M. Iacob, C. Thuillez, R. Joannides. *Department of Pharmacology - Inserm U644 - Rouen University Hospital, Rouen, France*

The role of NO and endothelium-derived hyperpolarizing factor (EDHF), synthesized by cytochrome epoxygenases and acting through calcium-activated potassium (KCa) channels, in the flow-mediated regulation of human conduit artery mechanics has never been investigated.

In 11 healthy volunteers, whole blood viscosity, arterial pressure, radial artery diameter, wall thickness and flow (echotracking) were measured during hand skin heating in the presence of saline and the NO-synthase inhibitor, L-NMMA, infused alone and combined with the inhibitors of KCa channels, tetraethylammonium, and cytochrome epoxygenases, fluconazole. Wall shear stress, the flow-dependent stimulus, was calculated (Poiseuillean model). Arterial compliance, elastic wall modulus were calculated and fitted as functions of midwall stress (wall loading conditions) to suppress the confounding influence of changes in geometry.

Heating induced in all cases an increase in radial artery flow, diameter, shear stress and midwall stress and a decrease in wall thickness without change in arterial pressure. The increase in diameter with shear stress was reduced by L-NMMA and, in a more extent, by both combinations. Heating induced an upward shift of the compliance-midwall stress curve and a downward shift of the modulus-midwall stress curve under saline demonstrating an associated decrease in smooth muscle tone and wall stiffness with the shear stress increase. The shifts of these curves were decreased by L-NMMA and abolished by both combinations.

These results demonstrate that NO and EDHF regulate the adaptation of conduit artery mechanics to shear stress variations in humans suggesting the major role of the endothelium in maintaining arterial conductance and adjusted cardiac load.

06.04

NON-ALCOHOLIC FATTY LIVER DISEASE IS ASSOCIATED WITH IMPAIRED SECRETION OF FAT PRODUCED HORMONES AND INCREASED CARDIOVASCULAR RISK

K. Baou [1], C. Vlachopoulos [1], E. Manesis [2], N. Ioakeimidis [1], G. Papatheodoridis [2], J. Koskinas [2], A. Archimandritis [2], C. Stefanadis [1]. *[1]1st Department of Cardiology, Athens Medical School, Hippokration Hospital, Athens, Greece, [2]2nd Department of Medicine, Athens Medical School, Hippokration Hospital, Athens, Greece*

Purpose: Adipocytokines may provide a link between metabolic syndrome, inflammation and cardiovascular disorder in non-alcoholic fatty liver disease (NAFLD) patients. We investigated whether NAFLD is associated with fat produced hormones and if this relation can affect the impaired endothelial structure and function.

Methods: We studied 34 patients (age 55 ± 13 years, 20M) with biopsy evidence of NAFLD, and 34 control subjects adjusted for classical risk factors. The changes in the diameter of the brachial artery were measured in response to reactive hyperemia and nitroglycerin. Mean IMT of common carotid arteries and carotid-femoral PWV were determined as markers of atherosclerosis and aortic stiffness respectively. Adipocytokines were measured by ELISA kit.

Results: NAFLD subjects had significantly reduced flow-mediated vasodilation (1.1 ± 1.9% vs 4.3± 3%, p<0.05), and mean value of carotid IMT (0.98 ± 0.3 vs 0.77 ± 0.2 mm, p<0.05) and PWV (8.4 ± 1.6 vs 7.3 ± 1.7 m/s, p<0.01) were increased compared to controls. NAFLD subjects had increased levels of leptin (21.81± ng/ml vs 12.12 ± 10 ng/ml, p<0.01), and resistin (5.174 ± 1.6 ng/ml vs 3.5 ± 1.28 ng/ml, p<0.01) and reduced levels of adiponectin (7.96 ± 5.19 μg/ml vs 13.17 ± 12.4 μg/ml, p<0.05) compared to controls. After adjustment for confounding factors, resistin levels were independently associated with impaired endothelial function (p<0.05, t=7.53, coefficient st=0.883) and leptin levels were independently associated with the increased mean IMT (p<0.01, t=6.92, coefficient st=0.888), and PWV (p< 0.05, t=2.258, coefficient st=0.32) in NAFLD patients.

Conclusion: Although the initiating events that trigger the development of atherosclerosis in NAFLD patients cannot be ascertained, the role of adipocytokines may identify a potential basis.