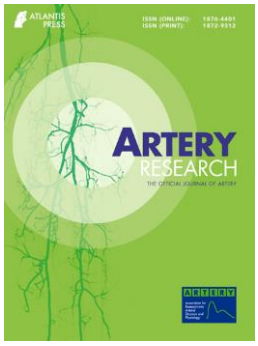

Artery Research

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

07.01: OUTWARD HYPERTROPHIC REMODELING AND INCREASED CAROTID ARTERY WALL STIFFNESS IN PATIENTS WITH RUPTURED INTRACRANIAL ANEURYSMS

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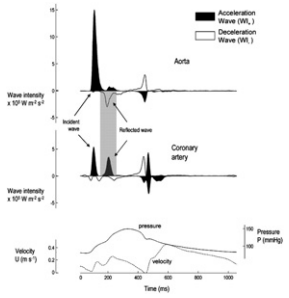
To cite this article: J. Bellien, D. Maltete, M. Iacob, L. Cabrejo, J.F. Proust, B. Mihout, C. Thuillez, E. Guegan-Massardier, R. Joannides (2008) 07.01: OUTWARD HYPERTROPHIC REMODELING AND INCREASED CAROTID ARTERY WALL STIFFNESS IN PATIENTS WITH RUPTURED INTRACRANIAL ANEURYSMS, Artery Research 2:3, 91–91, DOI: https://doi.org/10.1016/j.artres.2008.08.303

To link to this article: https://doi.org/10.1016/j.artres.2008.08.303

Published online: 21 December 2019

due to an increase in aortic stiffening (pulse wave velocity, r= 0.77, p<0.001).
Conclusions: Reflected wave can be followed travelling-back from the proximal aorta into the coronary arteries. These reflected waves augment coronary systolic blood flow. With increasing age the degree of augmentation of systolic coronary blood flow is increased.



doi:10.1016/j.artres.2008.08.302

07.01
OUTWARD HYPERTROPHIC REMODELING AND INCREASED CAROTID ARTERY WALL STIFFNESS IN PATIENTS WITH RUPTURED INTRACRANIAL ANEURYSMS

J. Bellien [1], D. Maltete [2], M. Iacob [1], L. Cabrejo [2], J.F. Proust [2], B. Mihout [1], C. Thuillez [1], E. Guegan-Massardier [1], R. Joannides [1].
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Because an underlying arteriopathy might contribute to the development and rupture of intracranial aneurysms (IAs), we assessed the elastic properties of proximal conduit arteries in 27 patients with antecedent of ruptured IAs (delay: 4.8±0.6 years) and 27 control subjects. Arterial pressure, diameter, intima-media thickness (IMT), circumferential wall stress (CWS) and elastic modulus were determined in the common carotid arteries using applanation tonometry and echotracking. Moreover, carotid augmentation index (AIx) and carotid-to-femoral pulse wave velocity (PWV) were assessed.
Compared to controls, patients with IA exhibit higher carotid systolic (108±2 vs. 122±3 mmHg), diastolic (73±1 vs. 81±1 mmHg) and pulse pressures (35±1 vs. 41±2 mmHg), an increased IMT (0.55±0.01 vs. 0.64±0.01 mm, all P<0.01) without difference in diameter. IMT was correlated with pulse pressure in controls (r=0.539, P<0.001) but not in patients (r=0.152, P=0.2). Despite a similar CWS between groups, patients display an increased elastic modulus (0.21±0.02 vs. 0.37±0.03 kPa.10^3, P<0.001). These increased IMT and modulus were still observed in patients matched with controls for carotid arterial pressures (n=17 in each group). Furthermore, patients with IAs have higher PWV (7.8±0.2 vs. 8.3±0.2 m.s^{-1}, P<0.05) which contributes to the increase in arterial wave reflections (AIx: 15.8±2.1 vs. 21.1±1.6%, P<0.05) and thus in systolic and pulse pressures.
This study demonstrates that patients with IAs display a particular carotid artery phenotype with a partly pressure-independent outward hypertrophic remodeling and altered elastic properties which might contribute together with the fatiguing effect of increased pulsatile stress on the arterial wall, to the pathogenesis of IAs.

doi:10.1016/j.artres.2008.08.303

07.02
TOWARDS NON-INVASIVE ASSESSMENT OF RENAL ARTERY STENOSIS SEVERITY IN THE INDIVIDUAL PATIENT WITH THE AID OF NUMERICAL COMPUTER SIMULATIONS

J. Kips [2], S. Van Bergen [1], B. Drieghe [4], D. Devos [3], K. Van Canneyt [1], B. Van der Smissen [1], B. Trachet [1], P. Verdonck [1], P. Segers [1].
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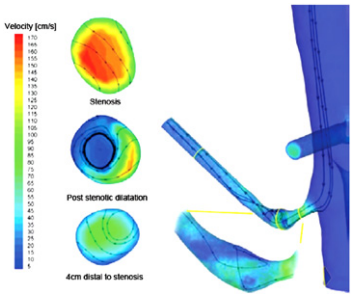
Introduction: Severe renal artery stenosis is responsible for 5% of all hypertension cases. Treatment of the stenosis is often decided upon catheterisation, with a pressure gradient (DP) of 10mmHg used as cut-off, thus requiring invasive measurements. The aim of the present study was to assess the feasibility and accuracy of a non-invasive estimate of DP through numerical simulation in a patient-specific model.
Methods: We constructed a computer model of the abdominal aorta, coeliac trunk, mesenteric superior aorta and two renal arteries from a patient with unilateral renal stenosis (77% area stenosis). Images were obtained from MR angiography scans and segmented to obtain the 3D patient-specific model. Blood flow was simulated assuming an aortic inflow rate of 2.7l/min and prescribed outflow rates at the different arterial outlets. The calculated DP was compared to in vivo measurements.



Results: The numerical calculations yielded a DP of 11.7mmHg, which was in excellent agreement with the value of 10.5mmHg measured in vivo in the same patient (with pressure guide-wires) and with values measured in a silicon hydraulic bench model of the same geometry. A parameter study demonstrated a rapid increase in DP beyond 60% stenosis. In the post-stenotic dilatation zone, secondary flow patterns with recirculation were observed.
Conclusion: These promising results demonstrate the feasibility and utility of patient-specific computer simulations in the diagnosis of individual patients, although further steps will be necessary to include pulsatile blood flow, distensible walls and patient-specific boundary conditions.

doi:10.1016/j.artres.2008.08.304

07.03
FETUIN-A IS INDEPENDENTLY ASSOCIATED WITH PROGRESSIVE AORTIC STIFFNESS IN PATIENTS WITH CHRONIC KIDNEY DISEASE

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Background: There is a disproportionate burden of vascular disease in patients with chronic kidney disease (CKD). Both aortic stiffness, as measured by carotid-femoral pulse wave velocity (C-F PWV), and deficiency in inhibitors of vascular calcification, such as Fetuin-A, have been implicated in the higher rates of cardiovascular mortality observed in this population. We sought to determine whether Fetuin-A concentration was inversely associated with progressive aortic stiffness.
Methods: 54 stable outpatients enrolled in a prospective cohort study of cardiovascular risk in CKD stages 3 and 4 underwent measurement of C-F PWV using Complior under standardized conditions at baseline and 12 months. Baseline plasma Fetuin-A concentration was determined using the Biovendor ELISA kit.
Results: The population was aged 68.0±10.4 years, 80% male, 11% diabetic with a mean eGFR of 32.0±11.5. Baseline Fetuin-A did not correlate with patient age, eGFR, mean arterial blood pressure, albumin, calcium-phosphate product, parathyroid hormone or CRP. Baseline Fetuin-A was inversely correlated with the change in PWV over 1 year (rho=-0.52, p<0.001). After adjustment for change in mean arterial pressure between visits, age, eGFR and presence of diabetes the correlation was maintained (r_p=-0.54, p<0.001). Using stepwise multiple linear regression with a model including age, change in eGFR, parathyroid hormone, CRP and diabetic status, Fetuin-A was the only independent predictor of change in aortic stiffness adjusted for change in MAP (β-coefficient -0.61, p<0.001; R^2 total 0.36).
Conclusion: In a cohort of patients with CKD stages 3 and 4 there is an independent negative association between Fetuin-A and progressive aortic stiffness.

doi:10.1016/j.artres.2008.08.305