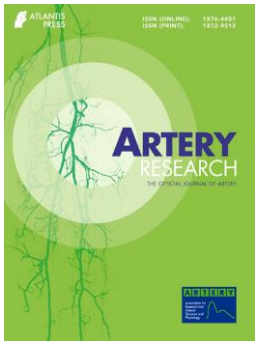


Artery Research

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

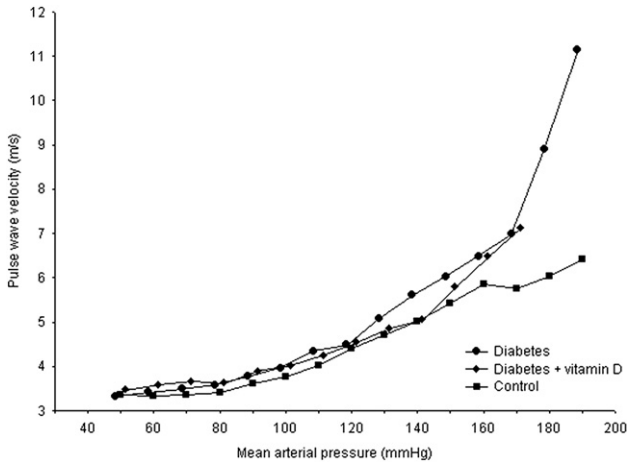
P4.08: BIOMECHANICAL STUDY OF ANEURYSM RUPTURE

J. Kim, A. Romo, P. Badel, A. Duprey, J.N. Albertini, J.P. Favre, S. Avril

To cite this article: J. Kim, A. Romo, P. Badel, A. Duprey, J.N. Albertini, J.P. Favre, S. Avril (2011) P4.08: BIOMECHANICAL STUDY OF ANEURYSM RUPTURE, Artery Research 5:4, 161–161, DOI: https://doi.org/10.1016/j.artres.2011.10.053

To link to this article: https://doi.org/10.1016/j.artres.2011.10.053

Published online: 14 December 2019



P4.06
MECHANICAL PROPERTIES AND STRESSES IN CAROTID ARTERIES QUANTIFIED USING CLINICAL DATA FROM NORMOTENSIVE AND HYPERTENSIVE HUMANS

I. Masson [1], H. Beaussier [2], P. Boutouyrie [2], S. Laurent [2], J. D. Humphrey [3], M. Zidi [1]
[1]*Université Paris-Est Créteil Val de Marne (UPEC), CNRS EAC 4396, Créteil, France*
[2]*Service de Pharmacologie, Université René Descartes, Hôpital Européen Georges Pompidou, INSERM U970, Paris, France*
[3]*Department of Biomedical Engineering, Malone Engineering Center, Yale University, New Haven, United States of America*

Objectives: To model the in vivo nonlinear mechanical behavior of human common carotid arteries (CCAs), to compute wall stresses and to deduce changes in wall micro-constituents (elastin-dominated matrix, collagen fibers, vascular smooth muscle cells (VSMC)) in normotensive subjects (NT) and hypertensive patients (HT).
Methods: Clinical data were obtained non-invasively from CCAs in 16 NT (21-64 years old) and 25 treated HT (44-69 years old). Medial diameter, intimal-medial thickness and blood pressure (BP) were measured during several cardiac cycles by high-resolution echotracking (Art.Lab®) and applanation tonometry (SphygmoCor®) systems, respectively. For the theoretical mechanical modeling, the CCAs were assumed to be hyperelastic, anisotropic, active-passive, and residually- stressed. We semi-analytically solved the boundary value problem to compute the intraluminal pressure from carotid distension, while accounting for perivascular tissue. Best-fit values of model parameters were adjusted by minimizing the difference between computed and measured inner BP over the cardiac cycle.
Results: In NT, age was positively correlated ($p<0.05$) with residual stresses and fibrillar collagen (stiffness and orientation). Despite treatment, HT had increased VSMC tone ($p=0.003$, +17.3%), a stiffer elastin-dominated matrix ($p=0.01$, +20.5%), and higher levels of stresses.
Conclusions: We were able to estimate wall stress fields and to quantify changes in mechanical characteristics of wall micro-constituents with aging and hypertension from non-invasive clinical data, though mechanical modeling of the wall behavior. Our results are consistent with prior reports on effects of age and hypertension, but provide increased insight into evolving contributions of cell and matrix mechanics to arterial behavior in vivo.

P4.07
ON THE RELEVANCE OF A PPG BASED TWO PULSE SYNTHESIS MODEL FOR SCREENING AGAINST CORONARY ARTERY DISEASES

D. Goswami [1], B. P. Chatterjee [2], S. Ray [2], K. Chaudhuri [1], J. Mukherjee [1]
[1]*Indian Institute of Technology, Kharagpur, India*
[2]*AMRI Hospital, Kolkata, India*

Abstract: Arterial stiffness is an independent predictor for coronary artery diseases (CAD). Various methods to predict coronary artery involvement are known but most of them have limited applicability in large-scale population screening due to cost barrier [1].
Photoplethysmography (PPG) to capture digital volume pulse signal is a simple, low-cost and noninvasive method for rapid assessment of arterial health conditions. An important application of PPG signal analysis has been towards early detection of altered arterial stiffness leading to increased risk of CAD. A Two Pulse Synthesis (TPS) model has been suggested recently for measurement of vascular parameters based on PPG signal and the usefulness of the model has been established for hypertensive and diabetic subjects [2].
This communication reports the outcome of a study based on the use of TPS model on finger-tip PPG of 40 suspected CAD subjects. The TPS model-based parameters considered in the study are Rise Time, Reflection Index, Foot-to-Foot Delay, Differential-Pulse-Spread and the Spread-Delay Ratio. Angiography has been subsequently carried out on these subjects and the findings have been compiled to find out sensitivity, specificity, positive and negative predictive values.
The study shows that the Positive Predictive Value of the TPS model is respectable (69%) while the Negative Predictive Value (93%) is high. It appears that this mathematical model may be applied to predict or rule out CAD conditions fairly successfully. Gradual development of functional embarrassments even in the absence of clinical manifestations may be possible with PPG analysis through periodic application of TPS model.
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P4.08
BIOMECHANICAL STUDY OF ANEURYSM RUPTURE

J. Kim [1], A. Romo [1], P. Badel [1], A. Duprey [2], J. N. Albertini [2], J. P. Favre [2], S. Avril [1]
[1]*Ecole Nationale Supérieure des Mines de Saint-Etienne, Center for Health Engineering, CNRS UMR5146, INSERM IFRESIS, Saint Etienne, France*
[2]*CHU Nord University Hospital Center, Department of Vascular Surgery, Saint Etienne, France*

The rupture of aortic aneurysms is a catastrophic event that represents a major public health issue. It has received a large interest from the scientific community. However, only limited research has provided quantitative values of mechanical stresses that may assess the risks of rupture of aneurysms [1].
In this study we have applied an imaging approach for measuring the deformations of the aneurysmal tissue tested in a biaxial inflation test [2]. The tissues have been taken from the thoracic ascending aorta of 6 diseased patients operated for aneurysm treatment by conventional surgery at the University Hospital of Saint-Etienne, France.
Quantitative values of ultimate stresses are reported in Tab. 1. Rupture is anisotropic, but primarily induced by axial stresses. Moreover, it is observed that rupture in aneurysms is preceded by a local weakening of the mechanical properties of the tissue, especially in the intima and media layers which are more fragile, and that these effects announcing a pending rupture can be detected by advanced imaging techniques. Our investigations continue in that sense for proposing novel diagnosis methodologies based on these observations.
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Tab. 1. Quantitative values of ultimate stress measured at rupture

Patient gender	M	M	M	M	M	M
Age	81	81	68	69	76	76
Type	adventitia	media	media	media	media	adventitia
Stress at rupture (MPa)	0.6257	0.3719	0.3686	0.4107	0.3483	1.0522

P4.10
THE SEMICARBAZIDE-SENSITIVE AMINE OXIDASE (SSAO): A NEW ACTOR IN ATHEROSCLEROSIS IN THE APOE MOUSE MODEL?

N. Mercier [1], S. Taleb [2], B. Fève [3], S. Jalkanen [4], Z. Mallat [2], P. Lacolley [1]
[1]*Inserm U961, Faculté de Médecine de Nancy Brabois, Vandœuvre-lès-Nancy, France*
[2]*Inserm U970, Centre de recherche cardiovasculaire, Hôpital Européen Georges Pompidou, Paris, France*
[3]*UMR S 938, Faculté de Médecine de St Antoine, Paris, France*
[4]*MediCity laboratory, University of Turku, Turku, Finland*

The expression of « semicarbazide sensitive amine oxidase » (SSAO), an enzyme which transforms primary amines into aldehydes, ammonia and hydrogen peroxide, increases during smooth muscle cell differentiation (VSMC) and is widely expressed in the media layer from the arterial wall. The SSAO has been implicated in LDL oxidation and in inflammation. We hypothesized that the absence of SSAO should prevent the development of atherosclerosis. The progression of the disease and the implicated mechanisms were studied in double ApoE/SSAO knock out mice (ApoE-/-SSAO-/-) established in the laboratory.
Surprisingly 25 week-old ApoE-/-SSAO-/- mice presented a significant 50 % increase in plaque surface associated with an 80% decrease in a-actin expression in the media of aortic sinus from ApoE-/-SSAO-/- mice compared to ApoE-/- mice. We noticed a small T-cell infiltration in the media from ApoE-/-SSAO-/- mice whereas no T-cell infiltration was observed in the media from ApoE-/- mice. No difference was detected in monocytes/macrophages infiltration in the plaque in aortic sinus from APOE-/-SSAO-/- mice and ApoE-/-. The pro- (TNFa and INFg) and anti-inflammatory (IL10 and TGFb) cytokine expressions were similar in the spleen from ApoE-/- and ApoE-/-SSAO-/- measured at 25 and 15 week old.
In conclusion, the absence of the SSAO increases the atherosclerosis in ApoE-/- mice. This result could be explained either by a modification of VSMC phenotype or to an increase in VSMC apoptosis in inflammatory situation. Thus, the SSAO could be a new potential actor implicated in the inhibition of atherosclerosis development.

P4.11
GALECTIN-3 IS A POTENTIAL MEDIATOR OF ALDOSTERONE EFFECTS IN VASCULAR REMODELING

L. Calvier [1], P. Reboul [4], B. Martin-Fernandez [2], V. Lahera [2], F. Zannad [1,3], V. Cachofeiro [2], P. Lacolley [1], P. Rossignol [1,3], N. Lopez-Andres [1]
[1]*UMR 961 INSERM-UHP, Vandoeuvre-Lès-Nancy, France*
[2]*Universidad complutense de Madrid, Madrid, Spain*
[3]*Inserm clinical investigation center, CIC 9501, Vandoeuvre-Lès-Nancy, France*
[4]*UMR 7561 CNRS-UHP, Vandoeuvre-Lès-Nancy, France*

Background. Aldosterone (Aldo) plays a major role in extracellular matrix (ECM) remodeling leading to heart failure (HF), but the mechanisms remained unclear. Galectin-3 (Gal-3), a β-galactosidase-binding lectin, plays an important role in inflammation and HF. We investigated whether Gal-3 mediates Aldo-induced ECM remodeling in vascular smooth muscle cells (VSMCs) *in vitro* and *in vivo*.
Methods. *In vitro*, primary cultured VSMCs were stimulated with Aldo (10^{-8}M) for 24h, with or without mineralocorticoid receptor (MR) antagonist. Gal-3 was over-expressed (transfection) and knocked-down (siRNA). Gal-3 expression and ECM production were evaluated by RT-PCR, Western blot and immunohistochemistry. *In vivo*, Wistar rats were treated by Aldo (1mg/kg/day)+salt or Aldo+salt+spironolactone (200mg/kg/day) for 3 weeks. Gal-3 and ECM proteins (collagen type I and III, fibronectin and elastin) were quantified by Western Blot and immunohistochemistry, and metalloproteinase (MMP) activities by zymography.
Results. *In vitro*, VSMCs spontaneously expressed Gal-3. Gal-3 over-expression enhanced ECM synthesis. Aldo up-regulated Gal-3 and ECM protein expression via MR. The Gal-3 silencing blocked Aldo-induced ECM production by VSMCs. In Aldo-salt hypertensive rats, Gal-3 aortic expression, ECM proteins and MMP activities were enhanced. Spironolactone treatment reversed all the above effects. Aortic Gal-3 expression was positively correlated with collagen type I, elastin, MMP-2 and MMP-13 activity.
Conclusions. Gal-3 is spontaneously expressed by VSMCs and induces ECM synthesis. It mediates Aldo-induced ECM remodeling via MR activation *in vitro* and *in vivo*. Our data suggest a key role for Gal-3 in Aldo-induced vascular alterations.

P4.12
MODEL BASED ESTIMATION OF AORTIC PULSE WAVE VELOCITY

S. Wassertheurer [1], B. Hametner [1], T. Weber [2]
[1]*Austrian Institute of Technology, Vienna, Austria*
[2]*Klinikum Wels-Grieskirchen, Wels, Austria*

The proposed model is inspired by the theoretical frameworks of (1) Moens-Korteweg, (2) Bramwell & Hill, (3) Waterhammer equation used in ARCSolver. All of the mentioned equations allow the determination of velocity in an elastic tube from a single point measurement. Whereby Moens-Korteweg consider the tension of the wall and the radius of the vessel as well as the viscosity of blood which can be assumed constant and near one in the human circulation for simplicity. It's now not really surprising that with increasing pressure both wall tension and wall radius will elevate. In younger age both parameters likely to the same extent and with only minor effects on PWV. With increasing age distensibility of the arterial wall degenerates. Subsequently an increase in pressure will not be compensated by a diameter change, even more tension within the wall will increase and PWV as well. All changes affect PWV non linear. Equation 2 rewrites equation 1 to consider observable variables like pressure and volume flow. Simply spoken, PWV is a result of pressure changes and volume displacement. In complex transmission line theory using Fourier analysis, the relation between arterial flow and blood pressure is described by the so called characteristic impedance (Zc) illustrated in the Waterhammer Equation (3). ARCSolver calculates (Zc) using an adopted Windkessel model. Determinants of ARCSolver-PWV are wall tension (impedance), aortic blood pressure and age.

P4.13
RELATION OF AORTIC AUGMENTATION INDEX TO ARTERIAL AND VENTRICULAR PROPERTIES

B. Jiang [1], P. Chowienczyk [1], D. Hou [2], X. Zhou [2]
[1]*St Thomas' Hospital, London, United Kingdom*
[2]*Capital Institute of Pediatrics, Beijing, China*

Aortic augmentation index, the amount by which aortic pressure is "augmented" above the first systolic shoulder of the aortic pulse wave, expressed as a ratio of aortic pulse pressure, is thought to relate to arterial properties. In particular it has been cited as an index of "arterial wave reflection" related to arterial stiffness. However, it could also be influenced by the characteristics of ventricular ejection. The objective of the present study was to examine the relation of AIx to arterial stiffness and ventricular contraction-relaxation. We studied 74 asymptomatic subjects, aged 24-89 years, 7-15 subjects per decade) using an Aloka α10 ultrasound system with 3MHz cardiac probe to perform conventional echocardiography, obtain tissue Doppler ventricular velocities and mitral flow velocities. Using the same system with a 10MHz linear vascular transducer, ECG-referenced echo-tracking of the carotid and femoral arteries was used to obtain carotid augmentation index (AIx) and carotid-femoral pulse wave velocity (PWV). The relation of AIx to PWV and ventricular tissue velocities was examined using univariable and multivariable regression analysis. On multivariable analysis, entering age, mean arterial blood pressure and all measures correlated with AIx on univariable analysis (including PWV), AIx was independently negatively correlated only with heart rate and the ratio of diastolic to atrial mitral valve flow velocities (E/A). Together these variables explained 39% of the variability in AIx. These results suggest that AIx is more closely related to ventricular contraction-relaxation than to arterial properties.

P5 — Clinical Science 1

P5.01
PRE-PREGNANCY TO EARLY PREGNANCY CHANGES IN MATERNAL CARDIOVASCULAR PHYSIOLOGY

A. A. Mahendru [1], C. C. Lees [1], T. R. Everett [1], I. B. Wilkinson [2], C. M. McEniery [2]
[1]*Fetal Medicine Department, Addenbrooke's Hospital, Cambridge, United Kingdom*
[2]*Clinical Pharmacology Department, Addenbrooke's Hospital, Cambridge, United Kingdom*

Maternal heart rate, blood pressure and cardiac output change as early as 5-6weeks in pregnancy. However, most of the longitudinal studies assessing maternal haemodynamic adaptation have used late first trimester