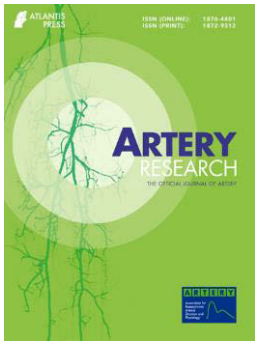

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P2.27: MECHANICAL MODELING OF IN VIVO HUMAN CAROTID ARTERIES FROM NON-INVASIVE CLINICAL DATA

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Experiments were performed on a Bruker 4T NMR system using a custom-developed setup adapted to small animal investigations.
Tissue perfusion quantification in *gastrocnemius* of mice was performed with a NMR sequence combining fast imaging with ASL[1]. It uses magnetically-labeled blood water as an endogenous tracer to quantify perfusion.
NMR method to determine BP is based on the sphygmomanometric principle: the caudal artery of the mouse was subjected to an external pressure from a tail air cuff. Arterial inflow signal was collected by single-slice dynamic NMR angiography and analyzed by reference to air pressure in the tail cuff.
These hemodynamics parameters were measured during reactive hyperemia after arterial occlusion in 10-week-old male db/db diabetic mice (n=17) and controls (n=14).
Using our dynamic NMR approach, we found both decreases in mean BP (68±9 vs 91±15 mmHg; $p<0.05$) and maximal perfusion (82±19 vs 105±38 $ml.min^{-1}.100g^{-1}$ $p<0.05$) in young db/db mice. However, we found that their maximal vascular conductance was not altered (1.22±0.3 vs 1.27±0.7 $ml.min^{-1}.100g^{-1}.mmHg^{-1}$), hence the relevance of our integrated approach.
1-Raynaud MRM 2001

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P2.27
MECHANICAL MODELING OF IN VIVO HUMAN CAROTID ARTERIES FROM NON-INVASIVE CLINICAL DATA

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Background: For mechanical modeling, in vivo data are relatively incomplete in comparison to in vitro results. However, identification of mechanical properties from human clinical data to compute wall stress fields can play an important role in understanding better pathological evolutions.
Aim: Demonstrate the feasibility of material identification and stress computation from clinical data.
Methods: In vivo human common carotid arteries (CCAs) were explored non-invasively. During several cardiac cycles, medial diameter, intimal-medial thickness and blood pressure were measured by a high-resolution echotracking (Art.Lab®) and applanation tonometry (SphygmoCor®), respectively. To study the wall mechanical behavior, the CCA was assumed to be a thick-walled, three-dimensional cylinder subjected to dynamical intraluminal pressure and perivascular constraints. We also assumed a nonlinear, hyperelastic, fiber-reinforced, incompressible material with smooth muscle activity and residual stresses. We included wall mechanical contributions by micro-constituents: an elastin-dominated matrix, collagen fibers, and vascular smooth muscle. We solved the in vivo boundary value problem semi-analytically to compute the intraluminal pressure during a cardiac cycle. Minimizing the difference between computed and measured inner pressures over the cardiac cycle provided the identification of optimal model parameters employing a nonlinear regression. Illustrative data were from two healthy subjects.
Results: The fit-to-data gave very good results. The predicted radial, circumferential, and axial stretches and stresses within the wall during the cardiac cycle were sensible.
Conclusion: We were able to identify experimentally unknown geometric and material parameters directly from in vivo human data. We can extend the proposed approach to pathological cases such as hypertension.

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P2.28
DEVELOPMENT AND VALIDATION IN FLOW-PHANTOMS OF A SIMPLE ULTRASOUND-BASED METHOD FOR ESTIMATION OF WALL SHEAR STRESS IN-VIVO

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Background: Wall shear stress (WSS) is an important measure of endothelial function however there are few clinical studies due to lack of a widely available measurement technique.
Aim: To develop and validate a simplified technique for estimation of WSS.
Methods: The Womersley equations were used; these describe pulsatile blood flow. With input of the vessel diameter (from B-mode ultrasound) and the centre-line blood velocity waveform (from Doppler ultrasound), the equations provided velocity profile as a function of time. Wall shear rate was estimated from the velocity gradient at the vessel wall. WSS was estimated from WSR*(viscosity), with an assumed viscosity of 4mPas. The technique was validated in a pulsatile flow phantom for vessels of physiological depth, diameter and flow-waveform.
Results: Estimated mean WSS was in error by 9±1% for brachial, 7±1% for carotid, 22±4% for femoral and 17±10% for fetal aorta.
Discussion: The errors are comparable with those obtained using dedicated WSS measuring systems. The method assumes that the vessel is rigid, straight and that flow is fully developed. There are several arteries where these flow conditions might hold, in health and in early disease where lumen diameter is preserved through outward remodelling. In-vivo validation is needed, possibly against a 'gold-standard' of MRI and computational fluid dynamics.
Conclusion: A simple method for estimation of WSS has been developed which is suitable for clinical studies.
Acknowledgement: The work has been published (Ultrasound Med Biol 2008, 34, 760-774) and previously presented (British Medical Ultrasound Society meeting, UK, December 2007)

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P2.29
AN IN SILICO MODEL OF THE ROLE OF ELASTIN ON GEOMETRY AND BIOMECHANICAL PROPERTIES OF ARTERY IN WILLIAMS BEUREN SYNDROME

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Arteriopathy in Williams-Beuren syndrome (WBS) patients with elastin gene deletion represents the most important mortality and mobidity causes and seems directly correlated with elastin quantity. There is a need for a comprehensive model that accounts for the quantity of elastin and its role in the geometry and biomechanical properties of artery. Such model might improve our understanding of the pathophysiology and improve innovation in drug development for WBS. We present an *in silico* model for the adaptation of human carotid artery in response to elastin deficiency. The model is based on the hypothesis that elastin deficiency-induced growth and remodelling occurs via the excessive production of vascular smooth muscle cells (VSMCs), change in collagen engagement, and increased laminar units to ensure that the average stress of lamina unit in the homeostatic state is unchanged under normal condition. Using an elastin-stress driven model and a constituents-based model, which considers the contributions of elastin, collagen, and VSMCs in an explicit form, we illustrate capabilities of the model in predicting the arterial thickness and biomechanical properties with varying elastin quantity before and after elastin restoration. Alternatively, the model has the potential to estimate, indirectly, the fraction of remaining elastin using the values of arterial thickness and mechanical properties. Our model provides a new approach for mathematically assessing the arterial growth and remodelling in human vascular disease with insight into the importance of constituent distributions.

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P2.30
SERUM FATTY ACID COMPOSITION AS MEDIATORS OF AORTIC PULSE WAVE VELOCITY'S IMPACT ON MORTALITY

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Aims/Hypothesis: Aortic stiffness predicts all-cause and cardiovascular mortality in essential hypertension, diabetes and the community. Potential intermediaries in this relationship include serum fatty acids (FA). We examined whether serum FA influenced aortic stiffness, measured as pulse wave velocity (aPWV), and its impact on mortality.
Methods: After randomly sampling population registers, 174 nondiabetic participants had fasting blood samples and a 75g glucose challenge (GTT), measures of doppler-derived aortic PWV and then serum FA composition determined by HPLC. Mortality data over 18 years' follow-up were obtained via the national registry and principal component (PC) analysis used for statistical modelling.
Results: Docosahexaenoic acid (DHA; $\rho = -0.22$; $p = 0.02$) and Arachidonic acid (AA; $\rho = -0.25$; $p<0.001$) were inversely related to PWV. PC analyses, including 10 measured serum FAs, ethnicity, age and sex, identified five