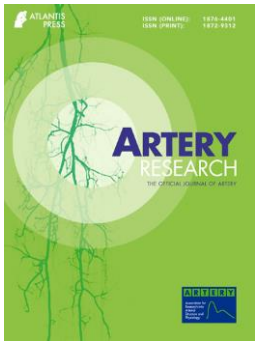

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P10.02: INSTABILITY PHENOMENA IN THE MECHANICAL BEHAVIOR OF THE ANEURYSM ARTERY

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of this receptor in the maternal uteroplacental circulation during pregnancy. To investigate this question, mice were injected with an anti-VEGFR-1 antibody (35 mg/kg i.p.) every other day beginning on day 8 (n= 9) or 12 (n=11) of gestation; vehicle-only injected mice were used as controls (n=12). All animals were killed late in pregnancy (day 19), prior to onset of parturition for determination of average pup number, resorption rate, and fetal and placental weights. Gestational vascular remodeling was evaluated by measuring the unstressed diameter and length of the main uterine artery and vein, as well as segmental artery diameter and length. Day 8 Ab injection resulted in a reduction in the average number of viable pups from 10 $\pm$ 1.2 to 3 $\pm$ 1.0 ($p<0.01$) and a high rate of fetal resorption (75 $\pm$ 7% vs. <5% in controls; $p<0.05$). Reproductive performance was also compromised in the day 12 group, although to a lesser extent. Placental and pup weights were similar throughout. Main and segmental uterine artery diameters were unchanged in either Ab group, although the diameter of the main uterine vein was reduced by 38 and 33% in both 8- and 12-day Ab-injected mice, respectively ($p<0.05$), Main uterine and segmental artery lengths were also significantly reduced. These results indicate that VEGFR-1 inhibition significantly compromises both reproductive performance and uterine vascular remodeling during murine pregnancy.

P10.02
INSTABILITY PHENOMENA IN THE MECHANICAL BEHAVIOR OF THE ANEURYSM ARTERY

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This study proposes a mathematical model to investigate stability of arteries. The artery is considered as a prestressed thick-walled tube subjected to dynamical pressure and made of a hyperelastic and composite material [1]. To model the mechanical contributions of the different arterial components, the here considered constitutive law of the wall takes into account the isotropic part due to the elastin-dominated matrix and the anisotropic due to the collagen fibers [2]. In this context, the purpose of this work focuses on the initial formation of aneurysms in human arteries which may be modelled as instability phenomena. For that, a perturbation technique is used on the equations of motion to highlight possible instabilities of the artery. This instability interpretation provides a theoretical approach under which different biological mechanisms leading to the risk of aneurysm formation can be assessed.
The proposed approach shows the influence of disturbances on the time variation of the radial deformation at the inner surface of the arterial wall. This means that the stress distributions are very sensitive to disturbances and may explain the aneurysm formation and its growth.
[1] E. Diouf, M. Zidi, *Finite azimuthal shear motions of a transversely isotropic compressible elastic and prestressed tube*, International Journal of Engineering Science 43, 262-274, 2005.
[2] I. Masson, C. Fassot, M. Zidi, *Finite dynamic deformations of a hyperelastic, anisotropic, incompressible and prestressed tube. Applications to in vivo arteries*, European Journal of Mechanics - A/Solids 29, 523-529, 2010.

P10.03
EVALUATION OF ARTERIAL STIFFNESS IN ATHEROSCLEROTIC RABBITS IN VIVO VIA ECHOTRACKING

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Objectives: We have shown that large artery stiffening, a major risk factor in cardiovascular diseases, can be evaluated in hypertensive rats by echotracking, analysing arterial compliance and also the arterial pulsatile diameter distension. We aimed to analyze similarly arterial stiffness in a model of atherosclerosis.
Methods: Male 9 week-old rabbits were fed 0.3 % cholesterol diet (ATH) or standard diet (CON) during 28 weeks. Then, under anaesthesia, blood pressure was recorded by catheterization and diameter via an ArtLab device, in a motion mode to detect pulsatile displacement of aortic walls (distension).
Results: Compliance, distension and distension/pressure loop were greatly decreased in ATH aorta versus CON, without mean diameter or blood pressure alteration. Basal femoral artery parameters were lower than aortic parameters. In ATH femoral artery, compliance, distension and the distension/pressure loop were reduced when recorded at a plaque level but increased at the upstream adjacent site; mean diameter was increased at both sites versus CON. Aortic endothelial function, assessed by ACh relaxation ex vivo was abolished in aorta and reduced in femoral artery; the lesions area in aorta (55 %) was 4x that observed in femoral artery.
Conclusions: This study analysed for the first time the in vivo dynamic arterial compliance in atherosclerotic rabbit. The data indicate a reduced arterial compliance and pulsatile distension and also show that the upstream adjacent site of a plaque is submitted to a higher stress and increased distension, in agreement with human data, which may participate to the plaque progression.

P10.04
ELASTIN AND COLLAGEN DEGRADATION REDUCES THE MECHANICAL STABILITY OF ARTERIES

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Arteries with elastin deficiency demonstrate tortuosity in human and animals, but the underlying mechanism has not been clearly elucidated. Our previous studies suggested that mechanical instability is a mechanism that leads to vessel tortuosity [1]. The objective of this study was to determine the role of extracellular matrix proteins in maintaining the mechanical stability of arteries. To this end, two groups of porcine carotid arteries were treated with elastase (8U/ml) and collagenase (2000 U/ml) respectively and tested before and after the treatments. The arteries were tested for pressurized inflation and the data were fitted with a Fung strain energy function to determine their stress-strain relationship. The critical pressures, at which the arteries became unstable and started to bend, were determined by a buckling test. The specimens were then processed for elastin staining and collagen staining and microscopy examinations. Our results demonstrated that elastase and collagenase treatment led to significant decreases in wall stiffness and critical buckling pressure of arteries. For example, the pre- and post- elastase treatment critical pressures of arteries are 19.9$\pm$5.3 kPa and 9.1$\pm$3.6 kPa, respectively, at *in vivo* length (n=6. $p<0.05$, see Figure 1). These results suggested that elastin and collagen degradation reduced the stability of arteries making them more susceptible to buckling and that mechanical buckling could initiate vessel tortuosity.
Acknowledgment: Supported by the NSF CAREER award 644646 and NHLBI grant HL095258 and NO1-HV-00244.
Reference: 1. Han HC: Blood vessel buckling within soft surrounding tissue generates tortuosity. J Biomech 2009;42:2797-2801.

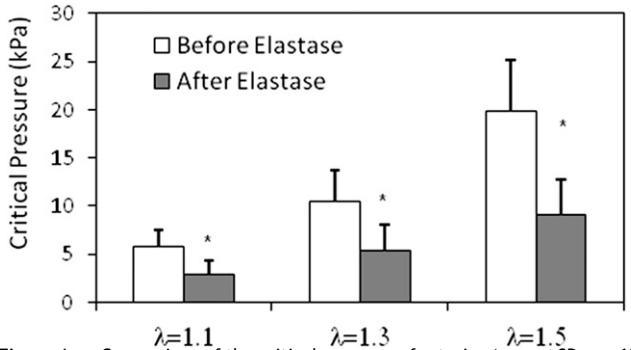

Figure 1 Comparison of the critical pressure of arteries (mean$\pm$SD, n=6) measured before and after elastase treatment. * $p < 0.05$.

P10.05
DOES THE AORTIC VALVES CORRESPOND TO A STABLE ANATOMICAL LANDMARK?

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Purpose: In order to determine if the height of a subject could be a reliable surrogate variable to determine the pulse wave travelling distance within the aorta, we investigated the anatomical distance between the aortic valve nidus and the hyoid bone.
Methods: Using 28 patient's chest CT-scans. From MPR reconstructed oblique plans we measured 1) the length of the aortic arch from the aortic valve (AV) to the intercept of an horizontal line passing through the aortic valves