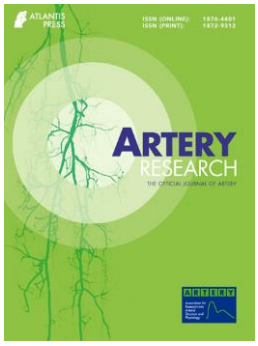

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P3.10: EFFECTS OF NITROGLYCERIN TO REDUCE AORTIC PRESSURE AUGMENTATION MAY NOT BE MEDIATED BY DILATION OF MUSCULAR ARTERIES

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20.4±23.2 to 5.9±5.9 and 3.9±3.20 mg/dl, whereas the atheromatic index, defined as total cholesterol/high density cholesterol, remained unchanged (from 3.4±1.1 to 3.1±0.8 and 3.0±0.6).
Conclusions: Short-term treatment with tocilizumab reversed endothelial dysfunction and improved arterial elasticity in a pilot study of RA patients, possibly via decreases of the systemic inflammatory burden.

P3.10
EFFECTS OF NITROGLYCERIN TO REDUCE AORTIC PRESSURE AUGMENTATION MAY NOT BE MEDIATED BY DILATION OF MUSCULAR ARTERIES

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Nitroglycerin (NTG) reduces aortic pressure augmentation (AP) and index (AIx), by a mechanism thought to involve a reduction in pressure wave reflection by selective dilation of muscular arteries. We examined this assumption by comparing effects of NTG with those of phentolamine (PHT) a vasodilator with little action on muscular arteries. Seven healthy subjects aged 35-62 were studied receiving PHT (1.12, 2.25 and 4.5 mg bolus/infusion i.v.) and NTG (90, 300, 900 μg i.v. infusion over 30 min) on separate occasions in a cross-over study. Central pressures (from radial tonometry) and carotid-femoral pulse wave velocity (cfPWV) were assessed using the SphygmoCor system. Brachial, carotid and abdominal aortic diameter were recorded by ultrasound. NTG (90 μg) reduced AIx from 22.2±5.5 to 13.7±5.7% ($P<0.05$) and PHT (4.5 mg) produced a similar reduction from 24.6±5.1 to 15.6±5.7% ($P<0.05$). These changes in AIx were observed in the absence of any significant fall in mean arterial blood pressure (MAP) or cfPWV. NTG (90 μg μg/min) produced a greater dilation of the brachial artery than of the carotid or aorta and vasodilation of the brachial artery by this dose of NTG was significantly greater than that by PHT (4.5 mg): 11±0.19 vs. 2.6±0.13% ($P<0.01$). These results suggest that, although actions of NTG are associated with dilation of muscular arteries, this is not necessarily the action leading to a reduction in AIx. Actions on other parts of the vascular tree and/or on ventricular ejection may be responsible for the reduction in AIx by NTG and PHT.

P4 — Basic science and modelling 1

P4.01
STRESS CALCULATIONS IN 3D RECONSTRUCTIONS OF ARTERIES: THE INFLUENCE OF AXIAL IMAGE RESOLUTION

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Computational modeling of the stress distribution in vulnerable atherosclerotic plaques facilitates identification of high stress locations which can be related to plaque rupture. The first step in doing 3D biomechanical stress simulations is to accurately re-create the artery geometry from histology or in-vivo imaging. This research investigated the influence of the axial sampling resolution of histology on the stress distribution in plaques.
A 3D reference geometry of a diseased human coronary artery was constructed based on 7 histological images with an axial spacing of 0.5 mm. Three under sampled models were generated: a 3D model based on four slices (1 mm spacing) and two 2D models based on one slice only (Figure 1). The stress distribution was calculated using the Finite Element Method (FEM).
The under sampled 3D model underestimates the peak stress by approximately 3% (Figure 2). The peak stress in the 2D models is 6% higher in one case and 12% lower in the other case. It can be concluded that a lower axial sampling resolution leads to a lower stress estimation due to smoothing of the geometry. Performing 2D simulations results in a more unpredictable stress distribution in that slice. However, approximate stress values and the location of peak stresses can be predicted well with a 3D under sampled geometry, indicating that 1 mm axial sampling might be sufficient for clinical FEM studies.
This research was supported by the Center for Translational Molecular Medicine and the Netherlands Heart Foundation (PARISk)

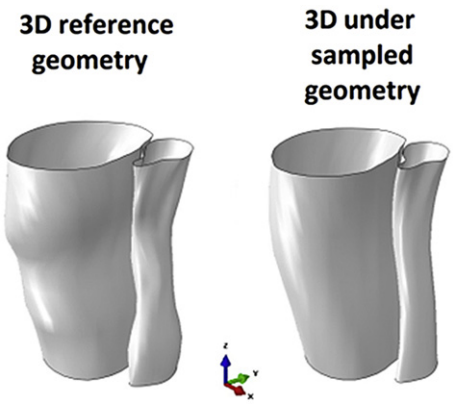

Figure 1 3D geometries (lumen and lipid surfaces shown)

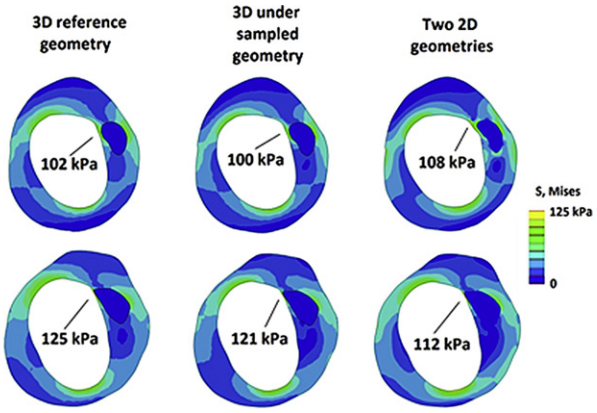

Figure 2 Von Mises stress distributions.

P4.02
INFLUENCE OF PLAQUE GEOMETRY ON PEAK CAP STRESS

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Introduction: Cap of an atherosclerotic plaque ruptures when mechanical stresses in the cap exceeds local strength. In this study, we investigated influence of plaque geometry on cap stresses.
Methods: Histology images of 30 cross-sections from 10 atherosclerotic human coronary vessels (perfusion fixed at 100 mmHg) were segmented. Mechanical stresses at 140 mmHg blood pressure were computed using finite element analysis after initial stresses were obtained using Backward Incremental Method [1]. Relation between cap stress and six geometric parameters (see Table) were evaluated. For each geometric parameter, two groups were created: the high group containing cross-sections with a value higher than the median value ($n=15$) and the low group ($n=15$). Mean values were compared for the two groups (Student's t-test).
Results: Thin cap cross-sections showed higher stresses than thick cap cross-sections (Table). Plaques with thinner necrotic core (NC) had larger stresses than the plaques with thick NC. Other geometric parameters were statistically insignificant.
Discussion: Higher peak cap stresses for thinner cap cross-sections confirm previous studies [2]. However, higher peak cap stresses for plaques with thin NC contradict an earlier study [2] with idealized geometries. Sharp corners at NC edges in realistic geometries might affect stress distribution in cap. Moreover, not only thickness but shapes of plaque components might influence peak cap stresses. In the near future, the analysis will be extended by incorporating other geometric parameters describing plaque component shapes and multivariate analysis to assess cross correlations between geometric parameters.

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[2] Akyildiz, 2011,BioMedEngOnline,10:25