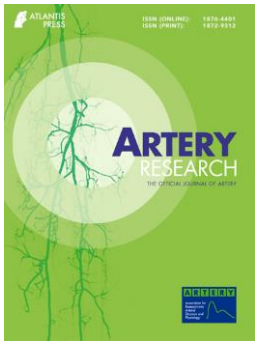

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P4.02: INFLUENCE OF PLAQUE GEOMETRY ON PEAK CAP STRESS

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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)

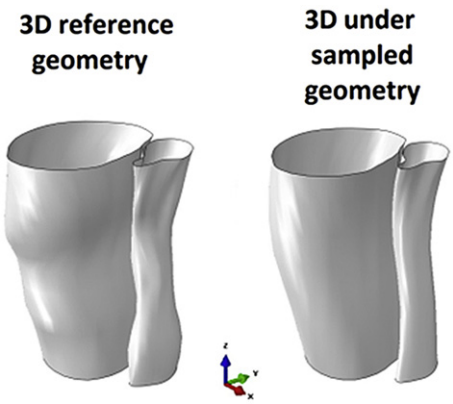

Figure 1 3D geometries (lumen and lipid surfaces shown)

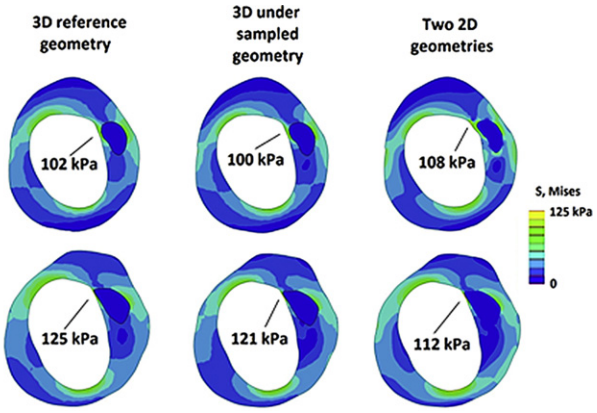

Figure 2 Von Mises stress distributions.

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.

References

[1] Speelman, 2009,JofBiomechanics, 42(11),pp.1713
[2] Akyildiz, 2011,BioMedEngOnline,10:25

Table: Peak cap stress in kPa (mean±st. dev.)

	Median Value	Peak cap stress in low group (kPa)	Peak cap stress in high group (kPa)	p-value
Min cap thickness	146 μm	170 ± 110	85 ± 40	<0.05
Max necrotic core th.	537 μm	172 ± 113	83 ± 30	<0.05
Necrotic core angle	58°	148 ± 103	109 ± 81	>0.05
Min intima thickness	196 μm	142 ± 130	116 ± 38	>0.05
Min media thickness	78 μm	138 ± 100	119 ± 90	>0.05
Min adventitia th.	47 μm	124 ± 82	134 ± 108	>0.05

P4.03

THE RESERVOIR-WAVE PARADIGM INTRODUCES ARTEFACT INTO WAVE INTENSITY ANALYSIS: A COMPUTER MODELLING AND IN VIVO STUDY

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Background: Wave intensity (WI) has traditionally been calculated from measured pressure and velocity waveforms. In the recently proposed reservoir-wave approach, wave intensity is calculated using excess pressure (WI_{RW}), i.e. the difference between measured and reservoir pressures. Although it has been suggested that WI_{RW} is more accurate than WI [1], this has not yet been validated in a system with known wave reflection properties.
Methods. This study assessed WI and WI_{RW} in two one-dimensional models, first, a simple network containing three bifurcations (two producing positive reflection and one producing negative reflection) and second, a full model of the systemic arterial tree. In both, a forward component of pressure was prescribed at the (non-reflecting) inlet, thus forward waves were known *a priori*. 3-element windkessels formed the model outlets. Modelling results were compared with measurements in the ascending aorta of five adult sheep.
Results. WI accurately predicted the three reflection sites in the simple network, whereas WI_{RW} did not detect the first or third (positive) sites and overestimated the second (negative) site. In both models, an artefactual mid-systolic forward expansion wave appeared in WI_{RW} but not WI. In the systemic arterial model, WI_{RW} predicted predominant negative reflection during systole, whereas WI correctly predicted positive reflection; in vivo results were qualitatively similar.
Conclusion: WI_{RW} introduces artefactual expansion waves and attenuates or eliminates compression waves. This may limit the utility of WI_{RW} in the assessment of both forward-running waves and wave reflection in arterial networks.
[1] Tyberg J. et al, Med Biol Eng Comput, 47(2):221-232, 2009

P4.04

CARDIOVASCULAR PARAMETERS OTHER THAN MEAN ARTERIAL PRESSURE ARE PREDICTIVE OF DYNAMIC CHANGES IN AORTIC STIFFNESS IN THE RAT

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It has been observed that phenylephrine induced mean arterial pressure (MAP) changes in rats cause different values of aortic stiffness, measured by pulse wave velocity (PWV), at the same MAP during increasing, compared to decreasing pressure. The reason for this hysteresis in the MAP-PWV relationship is not understood. This study evaluates the influence of a variety of cardiovascular factors on PWV. Five, 12 week old Wistar-Kyoto rats were anaesthetised and two pressure sensors introduced into the thoracic and abdominal aorta. Pressure was increased with intravascular phenylephrine infusion (30μg/kg/min), and allowed to return to baseline following cessation of infusion. This was repeated following intravascular hexamethonium bolus (20mg/kg), blocking autonomic activity to quantify the role of the sympathetic system. PWV, thoracic MAP, pulse pressure, form factor, maximum slope of pressure (dP/dt, surrogate measure of cardiac contractility), and R to R interval from the electrocardiogram were calculated for each individual pulse. These variables along with hexamethonium presence, and dynamic MAP direction were entered into a stepwise linear regression model (dependent variable: PWV). MAP, R to R interval, sympathetic activity, pulse pressure, dynamic MAP direction and dP/dt were significant predictors (Table). The results indicate that cardiovascular parameters other than MAP, especially pulse pressure, and notably sympathetic activity and R to R interval, are predictive of PWV. MAP direction remained in the model, indicating that other factors are at play, or that the aorta displays a short term viscoelastic memory. Further studies are required to isolate each of these factors and their effect on PWV.

	standardised coefficient	p	r^2 change
MAP	0.242	<0.001	0.476
R to R interval	0.275	<0.001	0.105
sympathetic activity	-0.193	<0.001	0.034
pulse pressure	0.453	<0.001	0.005
MAP direction	0.129	<0.001	0.014
maximum dP/dt	-0.168	<0.001	0.011

P4.05

CHARACTERISTICS OF AORTIC STIFFNESS IN DIABETIC RATS TREATED WITH VITAMIN D

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Background: Diabetes mellitus is associated with macrovascular complications characterized by increased aortic stiffness. Mechanisms of diabetes-induced vascular impairment include persistent activation of the inflammatory system that may be improved by the immunomodulatory effects of orally administrated vitamin D. This study aimed to examine the effects of diabetes on the elastic properties of the aorta and the protective effects of vitamin D treatment.
Methods: Diabetes was induced by injection of streptozotocin in male Wistar rats (age 4 months), followed by oral administration of cholecalciferol (500 IU/kg) for 10 weeks. Aortic pulse wave velocity (PWV) and other hemodynamic parameters were recorded over a mean arterial pressure (MAP) range of 50 to 200 mmHg using a dual pressure sensor catheter. Serum 25-hydroxyvitamin D (25[OH]D) level was measured using a radioimmune assay.
Results: In diabetic rats, PWV was significantly elevated across MAP range between 120 and 200 mmHg. PWV across lower MAP range did not reveal any significant differences between all groups. Administration of vitamin D did not improve aortic stiffness, despite high levels of serum 25(OH)D in the treatment group, compared to the placebo group (513±132 nmol/L *vs* 108±38 nmol/L, respectively). Other hemodynamic parameters (heart rate, systolic and diastolic blood pressure, and pulse pressure) were not different between all groups at any given level of MAP.
Conclusion: PWV profile determined under isobaric conditions demonstrated increased aortic stiffness in diabetic rats that was not improved by vitamin D supplementation.