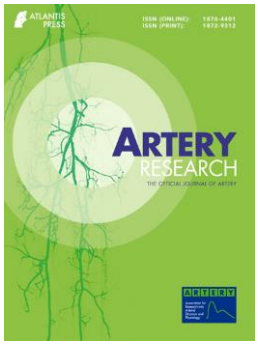

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P6.17: PATTERN BETWEEN ARTERIAL STIFFNESS, BLOOD PRESSURE AND AGE OBSERVED IN INDIVIDUAL PATIENTS AND REFERENCE POPULATION EXPLAINED BY BIOMECHANICS OF WALL REMODELLING

B. Spronck, M.H.G. Heusinkveld, A.A. Kroon, T. Delhaas, K.D. Reesink

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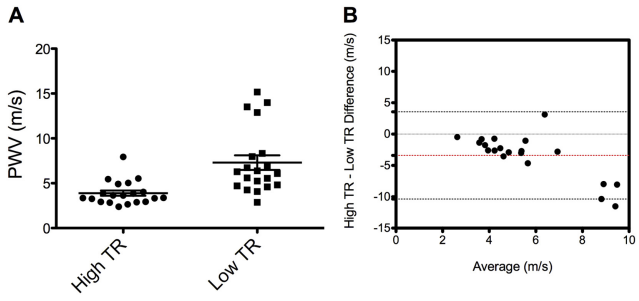

P6.15
HAEMODYNAMIC-MECHANICAL INTERACTION IN TYPE-B AORTIC DISSECTION: EXPERIMENTAL AND COMPUTER MODEL STUDY

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Background: Type-B aortic dissections (TBADs) show a high morbidity and mortality in the long-term follow up. The role of and interaction between haemodynamics (pressure and flows), mechanics (wall compliance) and morphology (communication between false lumen (FL) and true lumen (TL)) is poorly understood, as well as the potential role of these variables in the outcome of TBADs.
Materials and methods: We developed both a hydraulic bench (experimental) and an electrical analogue model of a TBAD (Fig. 1). The model was calibrated and validated for eight experimental cases consisting of various permutations of proximal and distal tear sizes (no tear, 4 and 10 mm).
Results: The computer model was able to reproduce measured intraluminal pressures and velocities across tears with good agreement for all combinations (Fig. 2). Tear size is the major determinant of interaction between FL and TL. Tear sizes of 4 mm are associated with a reduced pressure in the FL, and strong oscillatory flow exchange between TL and FL. A tear size of 10 mm increases FL pressure to the same level or beyond TL pressures, and reduces the oscillatory nature of the flow.
Conclusions: Both the experimental and computer model reveal a complex dynamic interaction between TL and FL, modulated by the size and combination of the tears.

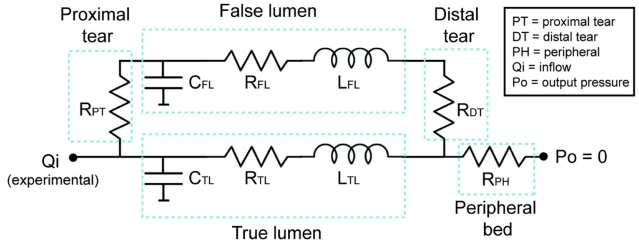

Figure 1 Electrical analogue model of a type-B aortic dissection

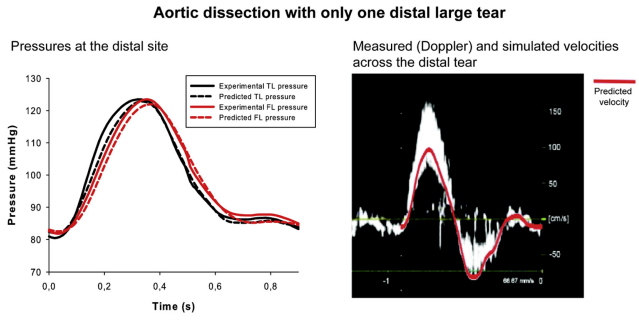

Figure 2 Comparison between predicted and experimental intraluminal pressures and velocities across tear

P6.16
PRONOUNCED INCREASE IN LONGITUDINAL DISPLACEMENT OF THE PORCINE CAROTID ARTERY WALL CAN TAKE PLACE INDEPENDENTLY OF WALL SHEAR RATE

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Objective and methods: We have recently shown that the longitudinal displacements of the arterial wall, i.e. the displacements *along* the artery during the cardiac cycle, undergo profound changes in response to catecholamines. The relations between longitudinal displacements of the arterial wall and wall shear rate (WSR) and wall shear stress (WSS), respectively, from the flowing blood are unexplored. To study this issue the common carotid artery of five anesthetized pigs was noninvasively investigated during different hemodynamic situations using ultrasound. The study protocol included intravenous infusion of epinephrine, as well as intravenous boluses of norepinephrine. Further, effects of β-blockade (metoprolol) were studied.
Results: During infusion of epinephrine and following boluses of norepinephrine WSR increased. However, when catecholamines were administrated during β-blockade, β-blockade effectively counteracted increase in WSR. We found no correlation between longitudinal displacement of the intima-media complex and WSR. This was most obvious when boluses of norepinephrine were administrated during β-blockade; β-blockade effectively counteracted increase in WSR, whereas, at the same time, β-blockade was insufficient to counteract a sharp rise in pulse pressure and accompanying pronounced increase in longitudinal displacement of the intima-media complex.
Conclusion: This study shows that a profound increase in longitudinal displacement of the arterial wall can take place independently of WSR, and thus, using the models of today, independently of WSS from the flowing blood. This strongly suggests that another force, or other forces, than WSS from the blood flow are also working in the direction of the blood flow; i.e. along the arteries.

P6.17
PATTERN BETWEEN ARTERIAL STIFFNESS, BLOOD PRESSURE AND AGE OBSERVED IN INDIVIDUAL PATIENTS AND REFERENCE POPULATION EXPLAINED BY BIOMECHANICS OF WALL REMODELLING

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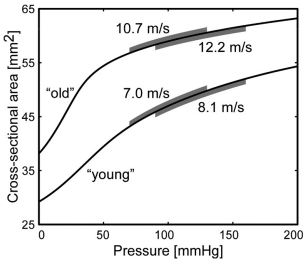
Background: We recently found in patients that the pressure dependency of arterial stiffness is age-dependent, explaining clinically observed changes in stiffness with blood pressure (BP) lowering. Pulse wave velocity, BP and age found in the Reference Values for Arterial Stiffness Collaboration show a corresponding pattern. We hypothesised that the impact of age on the pressure-dependency of arterial stiffness actually reflects changes in wall constituent properties and adaptation to mechanical stress
Methods and results: We applied a constitutive model of arterial wall biomechanics (Zulliger, AJP-Heart 2004) to simulate ageing, based on detailed pressure-area curves obtained in our younger (mean age 40 yrs, n=6) and older (65 yrs, n=7) hypertensive patients. Ageing was simulated by adapting the model (initially fitted to the "young" pressure-area curve) to fit the "old" curve (Figure); allowing for elastin degradation, collagen rearrangement and wall thickness adaptation, but not for changes in fractional volumes. The simulation showed a physiologically significant transfer of pressure-load bearing from elastin to collagen, caused by a 40% loss in elastin stiffness and a 5-fold increase in collagen recruitment (Table). Presuming normalisation and homogeneous distribution of wall stress with ageing, the predicted increase in wall thickness was in accordance with intima-media thickness Reference Values (2010 data; 25-yr increment, corresponding risk category).



		"young"	"old"
age	yrs	40	65
elastin stiffness	kPa	90	55
collagen recruitment at MAP		0.3%	1.6%
load bearing at MAP	elastin	66%	35%
	collagen	9%	47%
	VSM	25%	18%
wall thickness	mm	0.60	0.74

VSM: vascular smooth muscle. MAP: mean arterial pressure.

Conclusion: Ageing influences observed arterial stiffness values at given blood pressures through underlying changes in the properties and mechanical loading of arterial wall constituents.

P6.18
CAROTID-FEMORAL PULSE WAVE VELOCITY ESTIMATED BY AN ULTRASOUND SYSTEM

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To date, regional aortic stiffness can be evaluated by the reference tonometric technique via the pulse wave velocity (PWV) measured in two points: the carotid and the femoral arteries. Based on a similar intersecting tangent algorithm, we have developed a new method for the determination of carotid-femoral PWV using a high-resolution echo tracking ultrasound system. Herein, PWV can be computed from the measurement of the transit time between the foot of the carotid diameter waveform and the foot of the femoral diameter waveform. The study was carried out on 50 consecutive patients at rest (29 men, mean age 30 ± 18 yrs) recruited on the occasion of a vascular screening for atherosclerosis. Carotid-femoral PWV was determined by a trained operator using a tonometric technique, (PWVpp, PulsePen, Italy), and an echotracking ultrasound system, (PWVus, e-tracking Alpha 10, Aloka, Japan). Relationship between PWVpp and PWVus was evaluated by linear regression. A Pearson's correlation coefficient of r=0.95 was found between both variables (95% confidence interval 0.90-0.99; P<0.0001; PWVus= 0,91*PWVpp+0.44). The Bland–Altman plot comparing PWVpp and PWVus showed a systematic offset of -0.07 m.s-1 with a limit of agreement from -1,33 to 1,19 m.s-1. Our results show an excellent and significant correlation between both techniques which confirms that ultrasound system can provide a reliable estimate of the regional aortic stiffness like the tonometric technique does. Additional studies are now needed to show the simplicity of the measurement using ultrasound system while maintaining reliability even in overweight patients.

P6.19
PHYSIOLOGICAL CORRELATES OF AORTIC RESERVOIR AND EXCESS PRESSURE IN MAN

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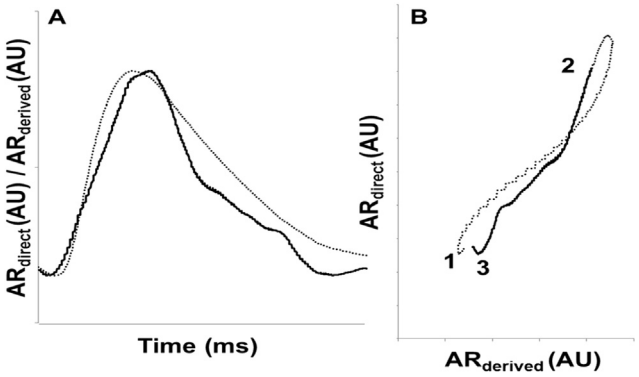
Background: Central (aortic) blood pressure (BP) indices independently predict cardiovascular events and all-cause mortality, but the physiological mechanisms underlying aortic BP waveform morphology are subject to debate. The 'aortic reservoir' and 'excess pressure' are proposed as determinants of aortic BP, but this relationship has only been assessed using a mathematically-derived aortic reservoir-excess pressure model ($AR_{derived}$ and $XP_{derived}$). This study aimed to directly measure the aortic reservoir (AR_{direct}; by cyclic change in aortic volume) and determine the relationship with $AR_{derived}$ and aortic BP.
Methods: Ascending aortic BP and Doppler flow velocity were recorded via intra-arterial wire in 10 males (aged 62±12 years) during coronary artery bypass graft surgery. Simultaneous ascending aortic transesophageal echocardiography was used to measure AR_{direct}. Published mathematical formulae were used to determine $AR_{derived}$ and $XP_{derived}$. A direct excess pressure (XP_{direct}) was calculated by subtracting AR_{direct} from aortic BP.
Results: When normalised to the same scale (Figure A), AR_{direct} (solid line) was strongly and linearly related to $AR_{derived}$ (broken line) during systole (r=0.980, P<0.001, Figure B, point 1-2) and diastole (r=0.987, P<0.001 Figure B, point 2-3). The cyclic relationship between aortic BP and AR_{direct} was qualitatively and quantitatively (P>0.05) similar to the cyclic relationship between aortic BP and $AR_{derived}$. Furthermore, XP_{direct} was linearly related to $XP_{derived}$ during systole (r=0.909, P<0.001) and diastole (r=0.663, P<0.001).
Conclusion: Aortic reservoir and excess pressures are physiological phenomena highly related to mathematically-derived aortic reservoir, excess pressure and aortic BP.



P6.20
EFFECTS OF DIFFERENT MEASUREMENT TECHNIQUES ON CAROTID STIFFNESS EVALUATION

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In recent years, great attention has been placed on local carotid elasticity. Carotid pulse wave velocity (cPWV) can be considered a surrogate marker for carotid stiffness evaluation. Aim of this study was to compare four different techniques for carotid stiffness assessment.
Ten young healthy subjects (34.7±6.9 years, 40% males, BMI 21.6±2.2 kg/m^2) were enrolled. For each volunteer, four different carotid stiffness measurements were obtained: i) ultrasound carotid stiffness (CS) values were estimated from US diameter and tonometric pulse pressure measurements combined by Bramwhell-Hill equation ii) $cPWV_{loop}$ values were calculated from US simultaneous measurements of diameter and flow velocity using the lnD-V loop slope iii) $cPWV_{MRI}$ values were obtained from velocity-encoded MRI images using QA method iv) $cPWV_{Acc}$ values were achieved by means of a new accelerometric system which consists in two percutaneous accelerometers placed 2.4 cm apart on the subject's neck; PWV is calculated dividing the distance between the sensors for the time delay between the signals.
Table 1 shows the results of the comparisons between CS (5.39±0.76 m/s), $cPWV_{MRI}$ (5.81±0.77 m/s), $cPWV_{loop}$ (4.18±0.96 m/s) and $cPWV_{Acc}$ (5.12±1.25 m/s) values. All the comparisons exhibit satisfying correlations. The only non-significative bias is shown by the comparison between CS values and $cPWV_{Acc}$ ones while the comparison between CS measurements and $cPWV_{loop}$ evaluations provides the lowest standard deviation of the difference.
In conclusion, this preliminary study suggests that attention should be placed when using different methods of carotid stiffness assessment, especially in case of comparison between values obtained with different methods.

Table 1

	Mean Difference ± SD of difference (m/s)	R^2
CS vs $cPWV_{loop}$	1.29±0.42	0.81
CS vs $cPWV_{MRI}$	-0.51±0.54	0.55
CS vs $cPWV_{Acc}$	0.27±0.75	0.67
$cPWV_{loop}$ vs $cPWV_{MRI}$	-1.77±0.56	0.71
$cPWV_{loop}$ vs $cPWV_{Acc}$	-1.16±0.57	0.66
$cPWV_{MRI}$ vs $cPWV_{Acc}$	-0.92±0.99	0.39

P6.21
EFFECTS OF PHARMACOLOGICAL DRUGS ON THE AORTIC PRESSURE PULSE: UNDERSTANDING MECHANISMS THROUGH MODELLING

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Aortic pulse pressure and other pulsatile components of the aortic pressure pulse are important predictors of cardiovascular outcomes, however the