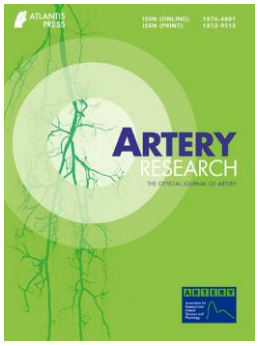

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P6.05: PRESSURE DEPENDENCY OF STIFFNESS DIFFERS WITH AGE: AGREEMENT BETWEEN OBSERVATIONS IN TREATED HYPERTENSIVES AND THE REFERENCE VALUES FOR ARTERIAL STIFFNESS POPULATION

B. Spronck, M.H.G. Heusinkveld, F.H. Vanmolkot, J. Op ’t Roodt, T. Delhaas, A.A. Kroon, K.D. Reesink

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Methods: An idealized common carotid arterial geometry was adopted first, so that comparison could be made between the numerical and analytical solutions. A linear viscoelastic material model was employed where Prony series were used to describe shear and bulk moduli[3]. The flow was assumed to be laminar flow, and physiologically realistic pressure and flow waveforms were imposed at the model inlet and outlet respectively[4]. Two-way coupling of fluid structure interaction was achieved by combining Ansys and Fluent.
Results: It was found that the viscoelastic behaviour of the vessel wall caused a phase lag between the radial wall distension and pressure waveform, as shown in Figure 1. The predicted pressure-diameter hysteresis loop (Figure 2) is in good agreement with the analytical solution. The numerical model presented here is being extended to realistic carotid arteries with and without the influence of arterial diseases (e.g. hypertension and atherosclerosis).

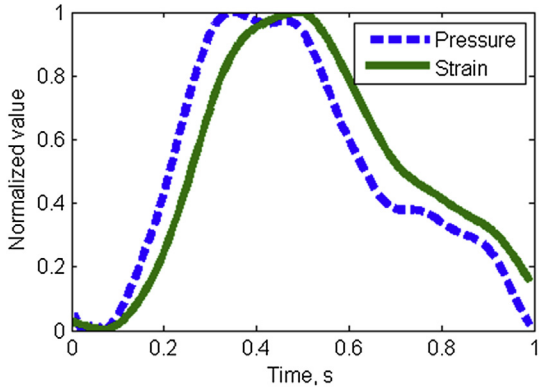

Figure 1

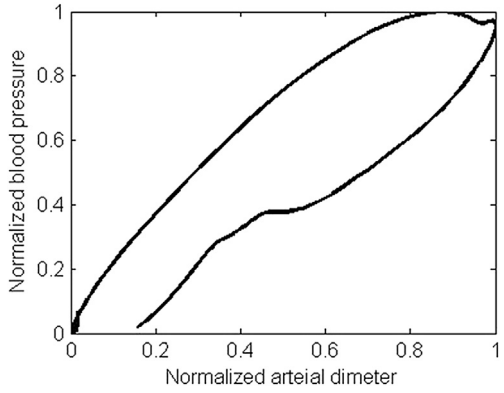

Figure 2

1. Valdez-Jasso et al, *Annals of Biomedical Engineering*, 39:1438-1456, 2011.
2. Hasegawa and Kanai, *Japanese Journal of applied Physics*, 43:3197-3203, 2004.
3. Park and Schapery, International Journal of Solids and Structure, 36:1652-1675, 1999.
4. Warriner et al, *Physiol Meas*, 29:157-79, 2008.

P6.04
PULSE VOLUME HOMEOSTASIS AS A HYPOTHESIZED PRINCIPLE OF ARTERIAL DESIGN

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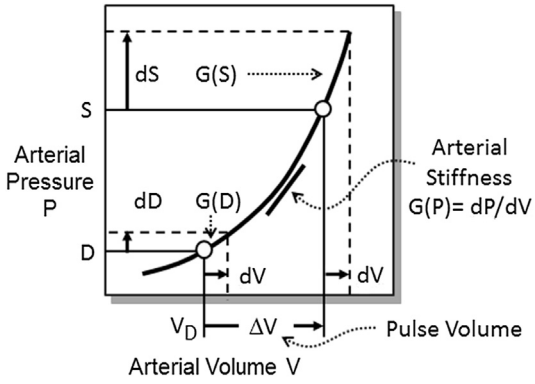
The increase of arterial volume during the systole ('pulse volume') is essential for buffering the pulsatile ventricular output.
Objectives: Deriving some mechanical properties of arteries by assuming pulse volume homeostasis.
Methods: A model that includes a generalized nonlinear relationship between arterial pressure P and volume V (see figure below).
Results: If the pulse volume ΔV is constant, then for a given diastolic volume V_D and pressure D the systolic volume $V_D+\Delta V$ and pressure S are uniquely determined. For this reason, an infinitesimal increase of the diastolic pressure by dD would result in an increase of the systolic pressure by dS with the same volume change dV for both pressures. Since the systolic and diastolic stiffness is defined by $G(S)=dS/dV$ and $G(D)=dD/dV$, respectively, we find that $dS/dD=G(S)/G(D)$ (Eq.1). As D and ΔV determine S uniquely, dS/dD is a function of D and ΔV only. However, if dS/dD is independent of D and ΔV is constant, then $K=dS/dD$ is a constant (Eq.2) equal to the relative increase of arterial stiffness during the systole. The only solution of Eq.2 is the well-documented linear relationship between the systolic and diastolic pressures with slope K and constant A, i.e., $S=A+KD$ (Eq.3). The solution of Eq.3, rewritten as $P(V+\Delta V)=A+KP(V)$, is the observed exponential pressure-volume relationship in arteries that is also expressed by the demonstrated linear dependence of arterial stiffness on pressure.
s: Arterial stiffening at elevated pressures may reflect an arterial design principle that aims at preserving the arterial buffering function via pulse volume homeostasis.



P6.05
PRESSURE DEPENDENCY OF STIFFNESS DIFFERS WITH AGE: AGREEMENT BETWEEN OBSERVATIONS IN TREATED HYPERTENSIVES AND THE REFERENCE VALUES FOR ARTERIAL STIFFNESS POPULATION

B. Spronck, M. H. G. Heusinkveld, F. H. Vanmolkot, J. Op 't Roodt, T. Delhaas, A. A. Kroon, K. D. Reesink
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Background: Arterial stiffness measures have a known dependency on actual blood pressure, requiring due consideration in risk assessment and treatment monitoring. Given the impact of age on arterial wall structural properties, this pressure-dependency might significantly differ between younger and older patients.
Methods and results: Using our previously developed approach combining carotid artery echo-tracking and tonometry waveforms, we obtained individual pressure-area curves in 13 outpatients at baseline (anti-hypertensive medication absent or discontinued) and at 3-month follow-up (medication reinstalled or increased). Using modelled-curve prediction, we found no change in the pressure-area relation after 3 months of treatment; i.e. carotid stiffness (cPWV) changed according to the BP shift along the baseline curve (measured=modelled cPWV; Table A). We discriminated patients aged under and over 50 yrs and averaged the respective groups' pressure-area curves using an established single-exponential model to estimate arterial stiffness for typical hypertensive (160/90) and normotensive (130/70) blood pressure ranges for both age groups (mean ages 40 and 65 yrs; Table B). We compared these values with the aortic stiffness values found in the Reference Values for Arterial Stiffness Collaborative study (2010 data) for corresponding mean arterial pressures (i.e. 118 and 94 mmHg) and mean ages (Table C).

A

		baseline	change at follow-up
anti-HT meds	DDD	0.8 ± 1.2	1.6 ± 0.8*
SBP	mmHg	163 ± 29	-25 ± 10*
DBP	mmHg	94 ± 9	-12 ± 6*
measured cPWV	m/s	**10.7 ± 3.1**	**-0.9 ± 1.1***
modelled cPWV	m/s	10.7 ± 3.1	**-0.9 ± 0.4***
aoPWV	m/s	10.0 ± 1.9	-0.4 ± 1.3

Mean ±SD, *p<0.05 for change (Wilcoxon signed rank test).

B

		age [yrs] 40	65	
SBP/DBP [mmHg]	130/70	7.0	10.7	modelled cPWV [m/s]
	160/90	8.1	12.2	
	Δ	**1.1**	**1.5**	

C

SBP/DBP [mmHg]	130/70	6.9	10.0	Reference Values aoPWV [m/s]
	160/90	8.2	11.7	
	Δ	**1.3**	**1.7**	

Conclusion: Within patients, short-term changes in arterial stiffness of ~1 m/s in the presence of >10 mmHg BP lowering can be deemed entirely pressure dependent. This pressure dependency of arterial stiffness increases with age, in part explaining the well-established pattern between stiffness, BP and age at (reference) population level.

P6.06
AORTIC REFLECTIONS-RELATED TIME AND MAGNITUDE INDICES ESTIMATED FROM THE SUPERIMPOSITION OF CENTRAL PRESSURE AND FLOW WAVEFORMS

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Objectives: While in young subjects, central pressure and flow peaks occur simultaneously and before time to return of reflected pressure (Tr), Tr progressively shifts towards early systole with aging, and eventually occurs before pressure peak. Consequently such parameters along with the corresponding augmentation index (AIx) have been previously shown as relevant indices of arterial aging. However, Tr accurate determination using pressure alone remains challenging, especially in elderly subjects. Accordingly, our aim was to combine central pressure and flow waveforms to provide objective and reproducible reflections-related indices.
Methods: We studied 50 healthy subjects (24 women, age: 46±15years), who underwent ascending aorta velocity-encoded magnetic resonance (1.5T) and carotid applanation tonometry. For each subject, , time delays between Tr and peak flow ($T_{Qmax-Tr}$) and between pressure and flow peaks (T_{Q-Pmax}), which are related to the overlap between incident and reflected pressure waves, were automatically estimated using a custom software that enables superimposition of pressure and flow waveforms. Conventional AIx was also measured from carotid pressure waveform. Forward and backward pressure components obtained using aortic characteristic impedance were used to estimate reflection magnitude (RM) as the ratio between backward and forward pressure magnitudes.
Results: The obtained correlations for comparison against age, AIx and RM were higher for T_{Q-Pmax} than for Tr or $T_{Qmax-Tr}$ (Table). Importantly association with RM, which is known to be a pure index of wave reflection, was superior for the combined index T_{Q-Pmax}.
Conclusions: Superimposition of pressure and flow waveforms helps for an objective and reproducible evaluation of central reflections-related indices.

Table. Comparison of reflection indices against age, augmentation index (AIx) and reflection magnitude (RM).

r (p)	Tr	$T_{Qmax-Tr}$	T_{Q-Pmax}
Age	-0.51 (p=0.0002)	-0.42 (p=0.003)	0.69 (p<0.0001)
AIx	-0.77 (p<0.0001)	-0.60 (p<0.0001)	0.82 (p<0.0001)
RM	-0.46 (p=0.001)	-0.52 (p=0.0001)	0.84 (p<0.0001)

P6.07
A STUDY TO DETERMINE IF THE REFLECTED WAVE TRANSIT TIME FROM BRACHIAL SUPRASYSTOLIC WAVEFORM ANALYSIS IS REPRESENTATIVE OF LARGE ARTERY STIFFNESS

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Background: Aortic stiffness is clinically important, but measurement can be time consuming. The 'reflected wave transit time' (RWTT) is thought to represent aortic stiffness and, since this can be measured quickly by brachial cuff oscillometry, could be a useful new test. This study aimed to compare RWTT with directly measured aortic (as well as brachial) stiffness in non-diabetics and also diabetics where there would be an expectation of increased stiffness (lower RWTT).
Methods: Aortic and brachial stiffness were recorded using tonometric pulse wave velocity (PWV; SphygmoCor) in 68 non diabetic (age 54.9±8.6 years,64.7% male) and 20 patients with type 2 diabetes (T2DM; age 60.5±9.6 years,55% male). RWTT was measured using brachial cuff oscillometry and suprasystolic waveform analysis (Pulscor®) as the time between the first and late systolic waves. Aortic PWV was also calculated from path length/RWTT in a subgroup of 69 patients.
Results: T2DM patients had significantly higher aortic PWV (9.6±2.7 vs 7.7±1.6 m/s, p=0.005), but no difference in brachial PWV (7.6±1.1 vs 8.1±1.4 m/s, p=0.14). RWTT between T2DM and non-diabetics was not significantly different (0.16±0.02 vs 0.17±0.02 s, respectively p=0.12). There were no significant correlations between RWTT and aortic PWV or brachial PWV in either T2DM or non-diabetic groups (r>-0.05, p>0.05 all). Furthermore, calculated aortic PWV was not significantly related to actual aortic PWV (p>0.05).
Conclusions: While brachial artery cuff oscillometric waveform analysis offers potentially useful clinical information, the transit time of pulse waves is not representative of large artery stiffness and, therefore, needs to be measured directly.

P6.08
EVALUATING THE HEMODYNAMIC IMPACT OF ISOLATED NON-DISTENSIBILITY AND RESIDUAL NARROWING AFTER COARCTATION REPAIR USING A COMPUTATIONAL STUDY

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[1]*IBiTech-bioMMeda, Ghent, Belgium*
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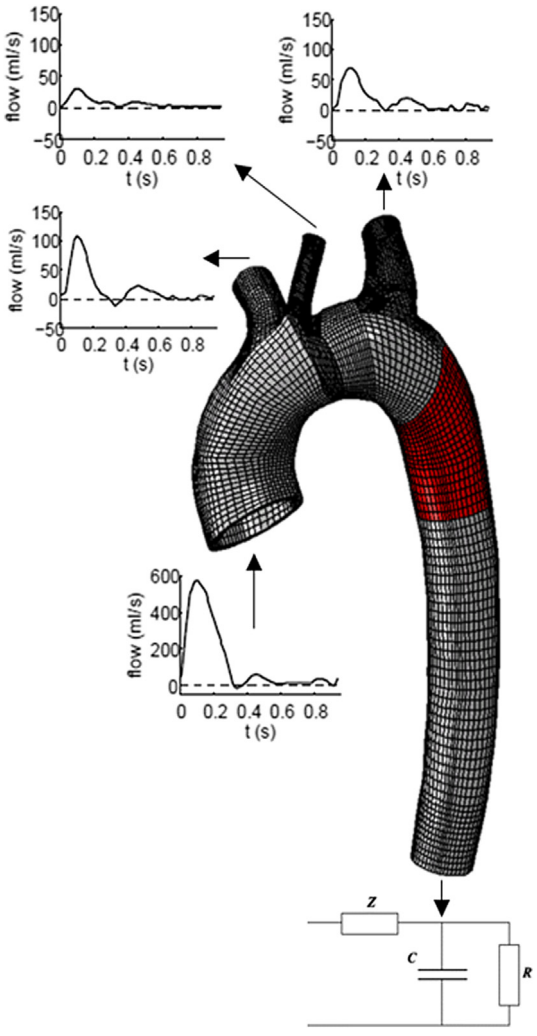

Figure 1 Patient specific model.

Background: Even after successful treatment of aortic coarctation, a high risk of cardiovascular morbidity and mortality remains. Uncertainty exists on the factors contributing to this increased risk among others the presence of (1) a residual narrowing, leading to an additional resistance in the arterial