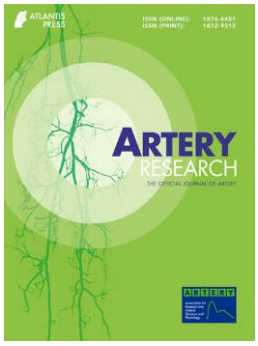

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P6.04: PULSE VOLUME HOMEOSTASIS AS A HYPOTHESIZED PRINCIPLE OF ARTERIAL DESIGN

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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).

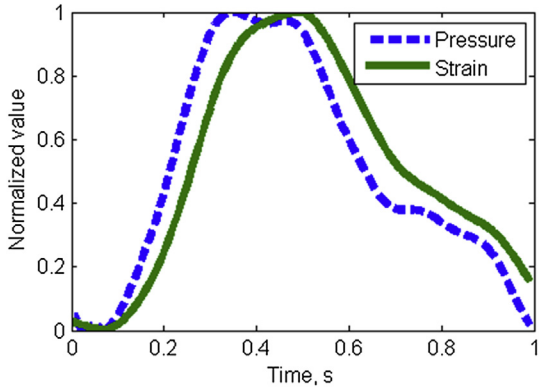

Figure 1

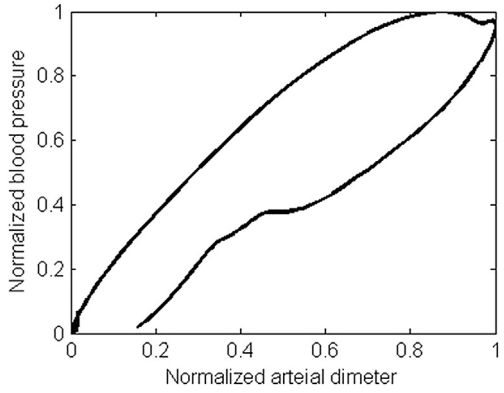

Figure 2

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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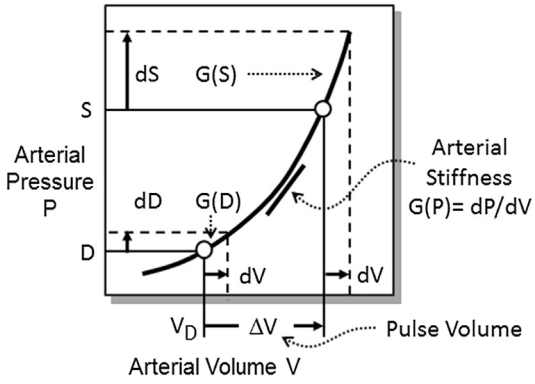
The increase of arterial volume during the systole ('pulse volume') is essential for buffering the pulsatile ventricular output.
Objectives: Deriving some mechanical properties of arteries by assuming pulse volume homeostasis.
Methods: A model that includes a generalized nonlinear relationship between 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

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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**	