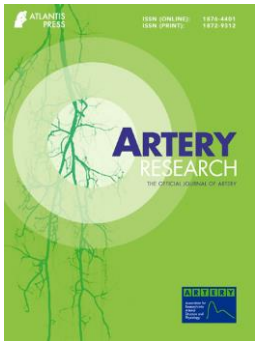

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P6.03: A FSI MODEL OF CAROTID ARTERIES WITH VISCOELASTIC WALL BEHAVIOUR

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Conclusions: In the present study reverse dipping status is associated with lower levels of HRQOL.

P6 Methods and Modelling

P6.01
IMPLANTABLE PULSE WAVE VELOCITY SENSOR

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In case of vascular weakness or thrombosis stents are implanted and brace vessels from the inside to assure the proper blood flow. A widespread method to monitor the state of blood vessels in terms of their stiffness and diameter is to measure the pulse wave velocity (PWV). The stiffer or the narrower a vessel is the higher is the pulse wave velocity. At present no diagnosis method exists to determine the state of the implanted stent directly and non-invasive. In case of a reasonable suspicion catheterization for precise diagnosis is required. The new approach developed at Fraunhofer IPA allows measuring the PWV locally, so it can be applied in shorter intervals. This approach is based on inductive coupling. It consists of two passive units integrated into the stent and an extra-corporal detection unit. The passive units consist of a capacitive pressure sensor and an air-coil (Fig.1). They form an oscillating circuit, the resonance frequency of which functionally depends on the local blood pressure. The extra-corporal detection unit consists of an excitation coil which generates an alternating magnetic field and a circuit for signal detection. The spreading pulse wave changes the resonance frequency of the passive oscillating circuits inside the vessel in such a way, that it crosses the frequency of the externally applied field while the pulse wave passes by (Fig. 2). The short resonance leads to a shift in the impedance measured at the excitation coil. As the distance between the two sensors is known the PWV can be determined.

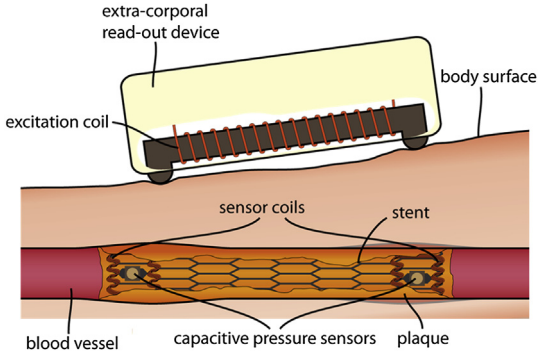

Figure 1 Scheme of functional principle

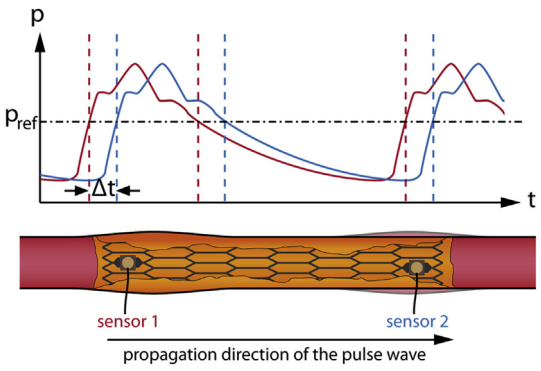

Figure 2 Schematic pulse wave propagation

P6.02
TOTAL ARTERIAL COMPLIANCE ESTIMATED BY A NOVEL METHOD AND ALL-CAUSE MORTALITY IN THE ELDERLY: THE PROTEGER STUDY

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Aortic stiffness assessed by carotid-to-femoral pulse wave velocity (PWV) often fails to predict cardiovascular (CV) risk and mortality in the very elderly. This may be due to the non-linear association between PWV and compliance, or to blood pressure decrease in the frailest subjects. Moreover, total arterial compliance (C_T) is the most relevant arterial property regarding cardiac function and ventriculo-arterial coupling. A new method for C_T estimation, based on PWV, was recently proposed. We aimed to investigate the value of this method to predict all-cause mortality at the elderly.
Methods: PWV was estimated (Complior) in 279 elderly subjects (85.5±7.0 years) who were followed-up for a mean period of 1 year. C_T was estimated by the formula $C_T = k \times PWV^{-2}$; coefficient *k* is body-size dependent based on previous *in silico* simulations. In this study, *k* was adjusted for body mass index (BMI) with a 10% change in BMI corresponding to almost 11% change in *k*. For a reference BMI = 26.2 kg/m^2, k = 37.
Results: Survivors (n = 185) and non-survivors (n = 94) had similar PWV (14.2±3.6 versus 14.9±3.8 m/s, respectively; p = 0.139). In contrast, non-survivors had significantly lower C_T than survivors (0.221±0.1 versus 0.198±0.128 ml/mmHg; p = 0.018). Cox-regression analysis showed that C_T was a significant predictor of mortality (p = 0.022, odds ratio = 0.326), while PWV was not (p = 0.202). Interestingly, age was an independent determinant of C_T (p = 0.016), but not of PWV.
Conclusions: It was demonstrated that C_T, estimated by a novel method, can predict all-cause mortality in the elderly. C_T could be a more sensitive arterial biomarker than PWV regarding CV risk assessment.

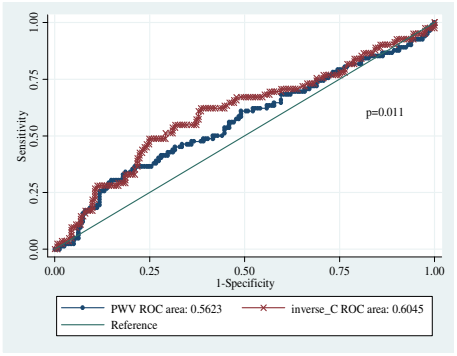

Figure. Receiver-operator-curve analysis of carotid-to-femoral pulse wave velocity (PWV) and total arterial compliance (inversed values) for the prediction of

Figure Receiver-opertor-curve analysis of carotid-to-femoral pulse wave velocity (PWV) and total arterial compliance (inversed values) for the prediction of

P6.03
A FSI MODEL OF CAROTID ARTERIES WITH VISCOELASTIC WALL BEHAVIOUR

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Objectives: Human carotid arteries exhibit viscoelastic behaviour characterised by hysteresis of the pressure-diameter relation and longitudinal wall motion[1]. Ultrasound techniques have been used to measure vessel wall displacements, as well as pressure and diameter waveforms, from which viscoelastic properties can be derived[2]. The aim of this study is to develop a fully-coupled numerical model for pulsatile flow in human arteries with viscoelastic wall behaviour.

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).

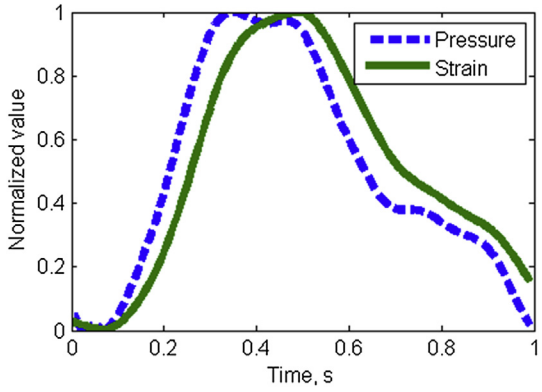

Figure 1

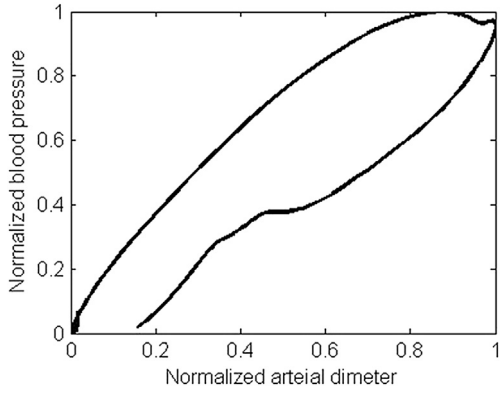

Figure 2

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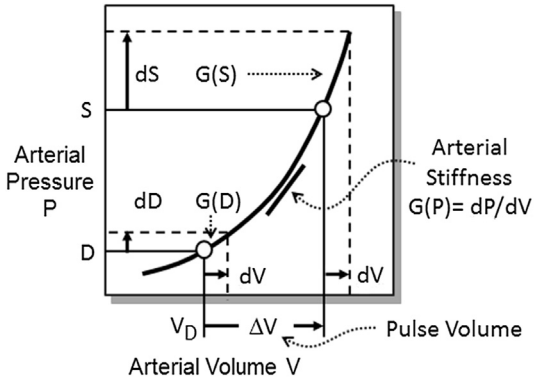
The increase of arterial volume during the systole ('pulse volume') is essential for buffering the pulsatile ventricular output.
Objectives: Deriving some mechanical properties of arteries by assuming pulse volume homeostasis.
Methods: A model that includes a generalized nonlinear relationship between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