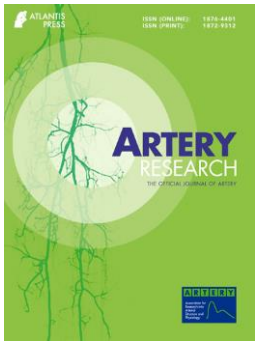

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P6.14: THE EFFECT OF TEMPORAL RESOLUTION ON MR ASSESSMENT OF PULSE WAVE VELOCITY

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The method used for pulse transit time (PTT) estimation, affects critically the accuracy of pulse wave velocity (PWV) measurements. The existing methods for PTT estimation yield often substantially different PWV values. Since there is no analytical way to determine PTT in vivo, these methods cannot be validated except by using in silico or in vitro models of known PWV and PTT. We aimed to validate and compare the most commonly used "foot-to-foot" methods: "diastole-minimum", "tangential", "maximum 1st derivative" and "maximum 2nd derivative". Also, we propose a new "diastole-patching" algorithm aiming to increase the accuracy and precision in PWV measurement. Methods: We simulated 2000 cases under a range of different hemodynamic conditions using a validated, distributed 1-D arterial model. The new algorithm "matches" a specific region of the pressure-wave foot between the proximal (i.e. carotid) and distal (i.e. femoral) waveforms. Intraclass correlation coefficient (ICC), mean difference (bias) and standard deviation of differences (SDD) were used to assess accuracy and precision. Results: The " diastole-minimum" and the " diastole-patching" methods showed an excellent agreement compared to the "real" PWV values of the model, as indicated by high values of ICC(>0.86).
The "diastole patching" method resulted in low bias (0.26m/s). In contrast, PWV estimated by 1st or 2nd derivatives and the "tangential" method presented a low to moderate agreement and poor accuracy (ICC<0.79, bias>0.9 m/s). The "diastole-patching" method yielded PWV measurements with the highest agreement, accuracy, precision and the lowest variability and its validity remains to be further examined in vivo.

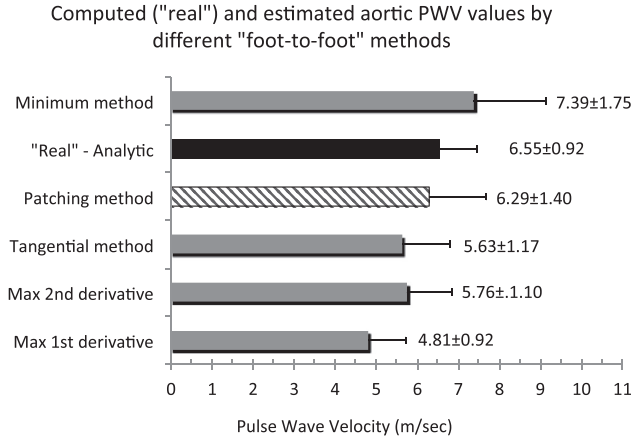

Figure 1 Mean and standard deviation for the aortic PWV estimations of the 5 validated algorithms. Bar in black represents the "real" PWV of the model.

P6.12
WAVE INTENSITY ANALYSIS OF REFLECTIONS IN THE BRACHIAL ARTERY FOLLOWING CUFF OCCLUSION AND HAND WARMING

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Background: Wave intensity analysis (WIA) is a mathematical tool used to study wave reflections in the arteries. Reflections are believed to contribute to BP augmentation and are also independent predictors of cardiovascular risk. Until now, the use of this technique has been largely confined to the aorta and carotid arteries.
Methods: 8 healthy subjects (age 30 ± 7.1) underwent wrist occlusion using a cuff inflated to >50mmHg suprasystolic pressure for 5min and hand warming at 55°C for 12min. Brachial artery diameter and blood flow velocity were measured using wall tracking and doppler ultrasound with an ALOKA SSD-5550 equipped with a 7.5 MHz probe. Wave intensity was calculated and reflections were quantified as the energy of the reflected wave/energy incident wave (WRI, %). Central aortic pressure following hand warming was also estimated using applanation tonometry (Sphygmocor) in separate studies.
Results: Cuff inflation resulted in a significant increment in WRI from 12.4 ± 4.15% to 26.8 ± 8.34% ($p=0.001$) whereas a marked reduction from 16.3 ± 6.60% to 4.09 ± 1.62% followed hand warming ($p=0.0017$). Cuff release was immediately associated with a significant attenuation in WRI ($p=0.01$). Hand-warming had no significant effect on the contralateral brachial or aortic SBP or DBP compared to baseline ($p=0.3$, 0.08 respectively).
Conclusion: Radial artery occlusion and hand warming respectively led to an augmentation and reduction in the reflected wave in the brachial artery. Hand warming was not associated with a significant change in peripheral or central BP.

P6.13
ADULT GUIDE-LINES ARE NOT APPLICABLE TO MEASURE PWV PATH LENGTH IN PAEDIATRICS

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Aortic pulse wave velocity (PWV) is a sensitive marker of arterial stiffness in children. In our previous study we have presented reference tables for PWV normal values in children. A recent consensus document provides arguments for the use of 80% of the direct carotid femoral distance as the most accurate distance estimate in adults. In the present work we aimed to assess if a transposition of the adult PWV measurement method is valid in childhood. Data of children participating to our previous work establishing age and height specific PWV normal values were re-evaluated. A total of 1008 healthy children (mean age:15.2 years, 495 males) were included in the study. We have recalculated PWV values using the subtractive method path length (L(SM)) and 80% of direct path length (L(0.8)). We have constructed Bland-Altman (BA) plots to assess the difference between PWV(SM) and PWV(0.8), and the distances L(SM) and L(0.8) in different age groups. The concordance between PWV(SM) and PWV(0.8) is excellent in children below 14 years (BA, ΔPWV mean:0.19 m/s, SD:0.40).However, in children >14 years, the difference increases (BA, ΔPWV mean:0.57 m/s, SD:0.36), and there is a proportional error between PWV(SM) and PWV(0.8) (BA, r:0.18; $p<0.001$), and in parallel there is also a proportional error between L(SM) and L(0.8) (BA, r:-0.24;$p<0.001$). The path length measurement suggested for adults may not be transponible to children throughout all age groups without reservation. Thus we propose to keep the current tables and values, unless the validity of a particular measurement is proved.
(Grant: OTKA100909)

P6.14
THE EFFECT OF TEMPORAL RESOLUTION ON MR ASSESSMENT OF PULSE WAVE VELOCITY

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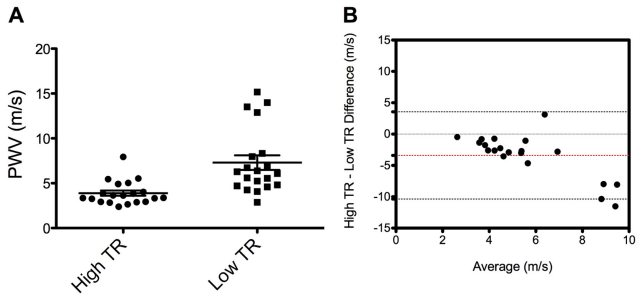
Objectives: PWV can be measured by velocity-encoded phase-contrast magnetic resonance imaging (PC-MR) in a single location. One method utilises the change in flow (ΔQ) divided by the change in area (ΔA) at the beginning of systole, when it is assumed that only forward running waves are present. However, the duration of a reflection free period is short (~30ms) and therefore a high sampling frequency is required to interrogate this period. Most PC-MR is performed with a low TR of approximately 30-40ms. In this study, we compared PWV calculated using high TR (10ms) and simulated low TR (30ms).
Methods: High TR (10ms) PC-MR was performed in 20 volunteers in the ascending aorta. TR reduction to 30ms was simulated by filtering the flow and area waveforms using a zero-phase, low-pass, high-order Butterworth filter with normalized cut-off frequency of 0.33 in Matlab. PWV was calculated from the gradient of the flow-area line at the onset of ejection, corresponding to the first 3 points of the foot of the area curve.
Results: There was a significant difference ($p<0.0004$) between PWV calculated using high TR mean 3.89m/s (SD 1.31) compared with simulated low TR, mean 7.30m/s (SD 3.64), Figure A. The mean bias between methods was 3.4m/s with wide limits of agreement (Figure B).
Conclusion: PWV calculated using a single location method is significantly inflated when data is acquired at low TR, as simulated using low-pass filtering. This suggests that conventional PC-MR may produce erroneous results in clinical studies.



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.

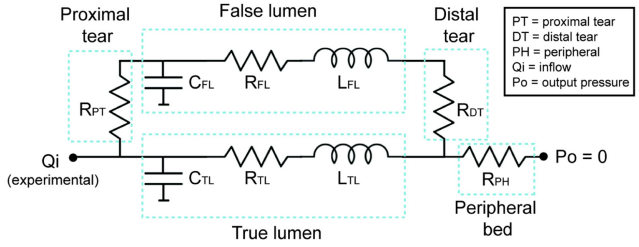

Figure 1 Electrical analogue model of a type-B aortic dissection

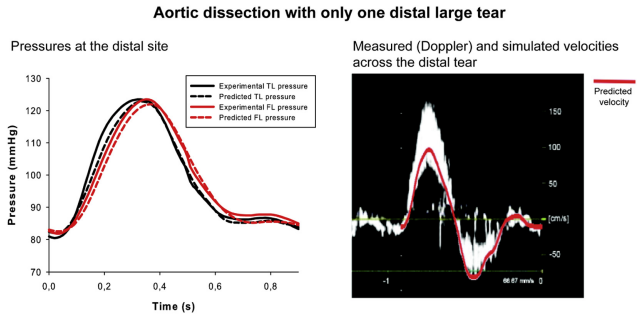

Figure 2 Comparison between predicted and experimental intraluminal pressures and velocities across tear

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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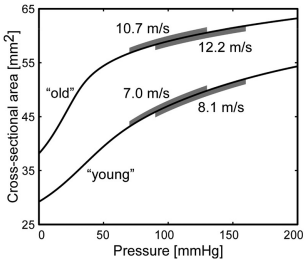
Background: We recently found in patients that the pressure dependency of arterial stiffness is age-dependent, explaining clinically observed changes in stiffness with blood pressure (BP) lowering. Pulse wave velocity, BP and age found in the Reference Values for Arterial Stiffness Collaboration show 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.