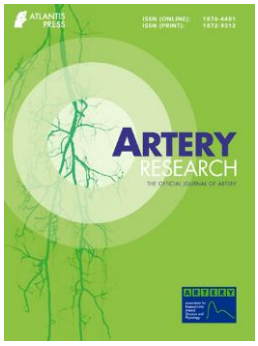

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P5.19: STIFF ARTERIES, STIFF HEARTS?

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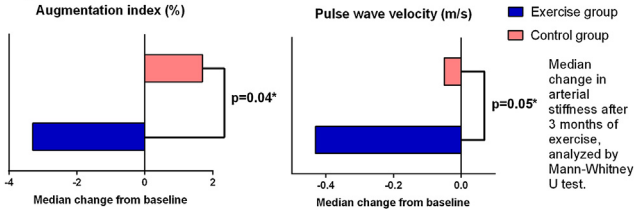
wave velocity (PWV) were assessed at baseline and after intervention. Statistical analyses (SPSS 20) were performed using Mann-Whitney U test to compare median change (from baseline to 3 months) of the parameters between EG and CG. Analyses were performed pr protocol.
Results: 28 AS patients were recruited, 24 patients fulfilled the study, 10 in the EG and 14 in the CG. There were some differences in demographics (EG vs. CG): age, years [median (min-max)] 43 (30-67) vs. 50 (26-68), male gender: 20% vs. 71%. After the study period, arterial stiffness was reduced in the EG compared CG, both significant for AIx (%) median (min-max) -3.3 (-24.5-2.5) vs. 1.7 (-13.5-10.3), p=0.04 and for PWV (m/s) median (min-max) -0.4 (-1.9-0.1) vs. -0.1 (-1.5-0.1), p=0.05 (figure).
Conclusion: Intervention with high intensity aerobic exercise in AS patients reduced arterial stiffness after 3 months compared to controls.

Figure



P5.15
SEX DIFFERENCES IN CENTRAL ARTERIAL STIFFNESS AND PRESSURES BEFORE AND FOLLOWING MAXIMAL EXERCISE

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Objective: There are sex differences in resting central pulse wave velocity (cPWV) but it is unclear if there are sex differences in the response to exercise. We evaluated potential sex differences of two measures of arterial stiffness and central pressures before and following maximal exercise.
Methods: We compared aortic and carotid systolic blood pressure (SBP), carotid β-stiffness and cPWV at rest, 15 minutes and 30 minutes following peak aerobic exercise in 73 participants (age=24 yrs; male n=34, female n=39).
Results: Women had lower aortic SBP, carotid SBP and CPWV, but similar carotid β-stiffness as men. Aortic SBP did not change, but carotid SBP and carotid β-stiffness increased 15 min post exercise ($p<0.05$) and returned to baseline at 30 min post exercise in both men and women. cPWV was unchanged with exercise in women, but decreased 30 min post exercise in men ($p<0.05$). These sex differences were unchanged when the data were corrected for differences in resting BP.
Conclusions: Resting cPWV and the cPWV response to exercise differ between men and women, without any sex differences in carotid stiffness. This suggests that sex may affect arterial stiffness differently in different arterial segments. Furthermore, the central BP response to exercise differs between the aorta and carotid arteries, in both men and women, suggesting the BP response to exercise is dependent on the arterial segment where it is measured.

P5.16 Withdrawn by author

P5.17
NOCTURNAL CHANGES OF AUGMENTATION INDEX MAY BE RELATED TO DIPPING STATUS

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Aim: Important prognostic significance of day-night brachial blood pressure (BP) decline for cardiovascular morbidity and mortality has been established. It is little known about diurnal variations of arterial stiffness parameters — augmentation index (AI@75) and aortic pulse wave velocity (PWV) as the non-invasive techniques for their 24-h ABPM has been just recently developed and validated. The aim of the study was to evaluate diurnal variations of AI and PWV in hypertensive subjects.
Methods: ABPM was done in untreated hypertensive subjects using BPLab VASOTENS ("OOO Petr Telegin", Nizhniy Novgorod, Russia) brachial oscillometric device which allows to derive aortic AI and PWV. Analysis included the successful ABPM results of 87 (47 male, age 61 years) subjects. Diurnal BP pattern was evaluated by diurnal index of systolic BP=(daytime SBP-nighttime SBP)/day-time SBP. Subjects were classified to dippers, non-dippers, night-peakers and over- dippers using usual cut-offs for brachial systolic BP.
Results: In dippers (n=29) nocturnal decline was 12,8±2,5% for brachial and 16,3±3,6% for aortic SBP, night- and day-time values of PWV and AI@75 were similar: 10,8±1,1 and 10,2±1,3 m/s, and 25,9±15,0 and 27,3±14,5%, respectively. In non-dippers nocturnal decline was 5,9±2,6% for brachial and 4,3±2,9 for aortic SBP, day- and night-time PWV values were similar (10,8±0,9 and 10,3±0,9 m/s), AI@75 tended to be higher during night then daytime (32,7±16,8 vs 27,9±14,6%). In night-peakers (n=13) night-time nocturnal decline was -3,9+3,75% for brachial and -4,8±3,9% for aortic SBP, day- and night-time PWV values were similar (10,6±1,0 and 10,5±1,3 m/s) and AI@75 tended to be higher during night then daytime (27,6±16,0 vs 36,8±32,6%). In over-dippers (n=4) night-time nocturnal decline was 20,7±0,9 for brachial and 20,2±1,0 for aortic SBP, day- and night-time PWV values were similar (10,3±0,9 and 9,5±0,8 m/s) and AI@75 tended to be higher during day then night-time (20,2±13,1 vs 14,2±18,7%).
Conclusion: The results suggest that PWV is relatively constant during 24-h, but nocturnal changes of AI@75 may vary across different SBP diurnal patterns

P5.18
CENTRAL HEMODYNAMIC' S ARE ASSOCIATED WITH DIABETIC COMPLICATIONS IN TYPE 1 DIABETES

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Objectives: We investigate associations between central hemodynamics and complications in type 1 diabetes.
Methods: Cross-sectional study, 676 type 1 diabetes patients, mean±SD age 55±13, 375(56%) male. Central hemodynamics measured by pulse wave analyses (PWA) (SphygmoCor (Atcor Medical, Australia) as central aortic systolic pressure (CASP), central pulse pressure (CPP), central diastolic pressure (CADP) and subendocardial viability ratio (SEVR) (index of myocardial oxygen supply and demand). Standardized values of hemodynamic measures were used in adjusted analyses. Complications were presence of albuminuria (≥30mg/24-hour), cardiovascular disease (CVD), retinopathy or autonomic dysfunction (heart rate variability <11 beats/minute).
Results: PWAs were available in 636 patients. Mean±SD CASP: 118±17 mmHg, CADP: 75±10 mmHg, CPP: 43±14 mmHg and SEVR: 150±32.
CVD (n=120) and autonomic dysfunction (n=349) was associated with: CASP (per +1 standard deviation (SD)): odds ratios (OR)=3.6(2.0-6.5) and 4.8(2.6-8.8); CPP (per +1SD): OR=2.0(1.5-2.7) and 2.2(1.6-3.1); CADP (per -1SD): OR=2.9(1.7-5.0) and 2.9(1.7-5.1); and SEVR (per -1SD): OR=1.7(1.1-2.6) and 2.4(1.6-3.5) (adjusted for gender, diabetes duration, mean arterial pressure, heart rate, height, urinary albumin excretion rate (UAER), eGFR, HbA_{1c}, cholesterol, antihypertensive medication and smoking). None of the hemodynamic variables were associated with albuminuria (n=335) or retinopathy (n=469) ($p\geq0.14$). However, if analysing UAER as a continuous variable, all hemodynamic variables were independently associated with level of UAER ($p\leq0.001$).
Conclusions: In patients with type 1 diabetes, central hemodynamics are independently associated with CVD, autonomic dysfunction and level of UAER, but not with albuminuria grade or retinopathy. Future studies are needed to determine if targeting central hemodynamics improve outcome.

P5.19
STIFF ARTERIES, STIFF HEARTS?

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Objectives: To examine the relationship between arterial stiffness and diastolic function in healthy normotensive subjects.
Methods: For this study, 43 male (40±10 years) and 64 female (40±9 years) subjects were recruited. All were lifelong non-smokers, normolipidaemic, normoglycaemic and had normal 24-hour blood pressure responses (SBP\DBP <140\90). For each subject, metabolic profile and anthropometric measurements were recorded. Carotid-femoral pulse wave velocity (PWV) was measured to assess arterial stiffness. Early/late mitral valve filling velocity (MV E/A) and isovolumetric relaxation time (IVRT) was used to assess diastolic function.

Results: The indices of diastolic function, LV IVRT & MV E/A ratio, and 24-hour ambulatory systolic & diastolic blood pressure were significantly correlated with PWV. In addition, age, anthropometric and metabolic values, such as waist/height ratio, fasting glucose (Glucose $_{Fast}$), glycosylated haemoglobin (HbA_{1c}) were also significantly correlated with PWV (Table 1). In a multiple regression analysis, age, 24-hour DBP, and MV E/A ratio remained independent predictors of PWV.

Table 1 Spearman's univariate correlation with PWV.

	Univariate Correlation with PWV	
n=107	r	*P*
Age	0.45	<0.0001
BMI	0.18	0.06
Waist/Height	0.22	<0.05
$Glucose_{Fast}$	0.22	<0.05
HbA_{1c}	0.26	<0.01
24-hour SBP	0.28	<0.01
24-hour DBP	0.32	<0.001
LV IVRT	0.28	<0.01
MV E/A	-0.44	<0.0001

Conclusion: Left ventricular diastolic function is associated with arterial stiffness even in "healthy" normotensive subjects, suggesting, that changes in arterial and left ventricular walls occur in parallel.

P5.20
RELATIVE CONTRIBUTIONS OF FORWARD AND BACKWARD COMPRESSION WAVES TO PULSATILE COMPONENTS OF BLOOD PRESSURE IN HYPERTENSION

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To what degree elevated pulsatile components of blood pressure arise from a primary interaction of ventricular contraction with the impedance of the arterial tree or from more complex phenomena involving backward wave travel in the arterial tree is unknown. We used wave intensity analysis to explore the haemodynamic basis for elevated pulsatile components of blood pressure in 20 hypertensive subjects (47.4± 13.4 years, 158.5±27.7/98.7±14.2 mmHg, means ± SD) compared to 20 normotensive controls (52.2±12.3 years, 108.7±12.2/71.8±7.7 mmHg, means ± SD). Secondly we used dobutamine and norepinephrine as inodilators and vasoconstrictors in normotensive subjects to examine the contributions of ventricular contractility and peripheral vasoconstriction to waves disproportionately elevated in hypertensive compared to normotensive subjects. An elevated central pulse pressure in hypertensive subjects was accounted primarily by the forward wave component (50.4±3.4 vs 35.2±1.8 mmHg, $P < 0.001$) but the backward wave also contributed significantly (8.9±1.7 vs 1.6±0.4 mmHg, $P < 0.002$) and was a particularly important component of augmentation pressure (13.5±3.6 vs 0.3±0.6 mmHg, $P < 0.01$). The forward component arose from the primary forward compression wave (FCW) and the backward component from a backward compression wave (BCW). The BCW/FCW intensity was greater in hypertensive compared to normotensive subjects and in normotensive subjects could be increased by norepinephrine but not by dobutamine. Increased pulse pressure in hypertension results primarily from the FCW but the BCW provides a significant contribution to pulse pressure components particularly augmentation pressure.

P5.21
EARLY IABP INFLATION SUPPRESSES LEFT VENTRICULAR FUNCTION AS INDICATED BY WAVE INTENSITY ANALYSIS

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Background: Timing errors during Intra-Aortic Balloon Pump (IABP) support can be detrimental when interfering with cardiac function. In vivo hemodynamics and left ventricular (LV) function were monitored during premature IAB inflation, and the associated mechanisms investigated with Wave Intensity Analysis.

Methods: Six healthy, anaesthetised, open-chest sheep received IABP support with frequency 1:3 (n=3) and 1:1 (n=3). Aortic (Qao) and coronary (Qcor) flow, and aortic (Pao) and left ventricular (PLV) pressure were recorded simultaneously with and without IABP. Early inflation (EI) was triggered -91±9ms (1:3) and -105±36ms (1:1) before the incisura. Integrating systolic Qao and subsequent negative Qao yielded stroke volume (SV) and backflow, respectively. Forward expansion (FEW) and forward compression (FCW) wave energies, generated by the slowing of LV contraction preceding inflation and by early LV ejection following deflation, respectively, were obtained. Results are mean±standard deviation.
Results: EI increased diastolic (d) Qcor in 1:3 but not in 1:1. EI appears to be detrimental for LV function, as indicated by substantial FEW reduction in both frequencies, concurring with increased backflow. Systolic (s) Pao and PLV did not corroborate suppressed LV function, but SV tended to decrease at 1:3 in the systole preceding EI. FCW reduction was also observed.

	IABP *on*		**IABP *off***	
	1:3	*1:1*	*1:3*	*1:1*
FEW (J/m^2)	0.026±0.015*	0.031±0.017*	0.059±0.026	0.073±0.026
FCW (J/m^2)	0.117±0.044*	0.086±0.051*	0.147±0.048	0.087±0.060
SV (*ml*)	67±16	78±28	71±17	77±32
Backflow (*ml*)	3.0±0.3*	4.410.9*	1.8±0.3	3.1±0.4
dQcor (*ml/min*)	182±17*	148±6*	150±17	149±36
sPLV (*mmHg*)	67±10	69±5*	67±13	76±7
sPao (*mmHg*)	71±11	73±8*	71±13	81±8

*P<0.05 comparing EI to *off*, #P<0.05 comparing 1:1 to 1:3.

Conclusion: With the aortic valve still open during EI, the IABP may displace blood directly into the LV and interrupt late LV ejection, as demonstrated by reduced FEW energy. Coronary perfusion is not affected by this mechanism. Concurrent reduction in FCW energy, albeit not exclusively caused by EI, further indicates compromised LV function.

P5.22
RELATIONSHIP BETWEEN ARTERIAL STIFFNESS, LEFT VENTRICULAR GEOMETRY AND THE SEVERITY OF ERECTILE DYSFUNCTION: A VENTRICULOARTERIAL INTERACTION IN ERECTILE DYSFUNCTION PATIENTS

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Objectives: Erectile dysfunction (ED) represents an early marker of vascular damage and a potent factor for future cardiovascular events. Our study aims to investigate interrelationships between ED, arterial stiffness and left ventricular geometry.
Methods: We analyzed data from 85 ED patients (mean age: 56±7 y/o). Diagnosis and grading of the erectile disorder were performed according to the International Index of Erectile Function (IIEF-score). Parameters estimating the additional hemodynamic afterload, were carotid-femoral pulse wave velocity (PWVc-f), augmentation index (AIx) and augmented pressure (AP). Consequently for LVgeometry, LV mass index and relative wall thickness (RWT) were measured.
Results: IIEF-score was negatively correlated with age (r=-0.285, P<0.01), systolic pressure (r=-0.258, P<0.01) and pulse pressure (r=-0.335, P<0.001). Patients with severe ED (IIEF<10) had significantly higher age and BP- adjusted PWVc-f (9.1±1.5 vs 7.8±1.5 m/s, P=0.005), AIx (24±7 vs 19±8%, P<0.05) and AP (10±4 vs 7±4 mmHg, P<0.05) compared to subjects with a higher IIEF score. Furthermore, after adjusting for age and BP level, patients with severe ED had higher LV mass index and RWT compared to subjects with a higher IIEF score (82±11 vs 76±9 g/m2 and 0.42 ± 0.06 vs.0.39±0.04, respectively, all P<0.05).
Conclusions: ED severity is significantly associated with arterial stiffness indeces and alteration of LV geometry. Our data confirm the extent of cardiovascular damage in ED population with severe penile functional disorder, and allow identification of those in an altered cardiovascular status, who