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

A. Angelis, N. Ioakimidis, A. Agelakas, K. Aznaouridis, C. Chrysohoou, A. Samentzas, K. Ageli, E. Tsiamis, C. Vlachopoulos, C. Stefanadis

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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.

n=107	Univariate Correlation with PWV	
	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 (Q_{ao}) and coronary (Q_{cor}) flow, and aortic (P_{ao}) 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 Q_{ao} and subsequent negative Q_{ao} 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) Q_{cor} 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) P_{ao} 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
dQ _{cor} (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 LV geometry, 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 indexes 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

may warrant more intensive strategies in preventing further deterioration of their physiology.

P5.23

A MEDICAL CONFERENCE DINNER'S IMPACT ON CENTRAL BLOOD PRESSURE AND VASCULAR AGE

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Central blood pressure (BP) is recognised as a predictor of cardiovascular disease (CVD). Moderate alcohol consumption has been shown to have a beneficial effect on CVD. However, consumption of a lipid-rich meal may exert the opposite effect. Thus, the aim of the present study was to examine whether the immediate effect of a medical conference dinner was associated with reduced central BP and hence improved vascular age.

We examined attendees at a medical conference before and after the conference dinner which included a three course meal and wine menu. Participants had brachial and central BP measured. Central BP was measured in duplicate over the right radial artery using the Sphygmocor device (Atcor Medical, Sydney, Australia).

The cohort consisted of 60 attendees (43% women) with a median age of 40 years (IQR 35 – 54) and a mean follow-up period of 4 ± 1 hour. Only one attendee smoked, whereas six took antihypertensive medication. While heart rate increased, all measurements of brachial and central BP were reduced after the dinner (Table 1). Multiple regression showed that central systolic BP and augmentation index (Alx) was reduced after the dinner independently of age, gender, height, and baseline heart rate ($p = 0.008$ and $p = 0.01$). Furthermore, calculations of the slope of the regression lines between $Alx@HR75$ and age before and after the dinner revealed a reduction of 5.5 years in the vascular age.

In conclusion, central BP was reduced and vascular age improved by 5.5 years after intake of a medical conference dinner.

P5.24

EFFECTS OF IVABRADINE AND ATENOLOL ON CENTRAL AORTIC PRESSURE IN HYPERTENSIVE PATIENTS WITH STABLE ANGINA

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The aim of the study was to assess the effects of ivabradine (I) and atenolol (A) on central aortic pressure in patients with stable angina and arterial hypertension (AH).

Methods: The study was conducted on 31 hypertensive patients (57.1% male), mean age 61.9 ± 8.4 years, with angina pectoris of II-III functional class and without history of myocardial infarction and chronic heart failure. Two weeks before randomization all patients received nifedipine SR (30 mg o.d.) additionally to antihypertensive treatment. Then patients were randomly assigned to I ($n=15$) and A ($n=16$) dose up-titration for 2 weeks. Next 4 weeks doses of I and A were consistent. Mean doses were 14,4 mg for I and 137,5 mg for A. Heart rate, peripheral systolic (SBP) and diastolic blood pressure (DBP), central aortic systolic (CSP) and diastolic pressure (CDP), aortic pulse pressure (PP) were measured at baseline and at the end of the study.

Results: Heart rate (HR) decreased from 74.0 to 54.0 bpm with I and from 74.5 to 54.5 bpm with A (both $p=0.001$). SBP decreased from 132.0 to 129.5 mm Hg with I ($p=0.55$) and from 132.0 to 122.0 with A ($p=0.01$). DBP decreased from 80.0 to 79.5 mm Hg with I ($p=0.96$) and from 80.0 to 76.0 mm Hg with A ($p=0.001$). CSP decreased by 6.9 mm Hg with I ($p=0.01$) and 8.0 mm Hg with A ($p=0.002$). DSP decreased by 3.0 mm Hg with I ($p=0.01$) and 4.0 mm Hg with A ($p=0.004$). PP decreased by 4.0 mm Hg after I ($p=0.64$), while PP increased by 5.0 mm Hg after A ($p=0.76$). **Conclusion:** After achieving of target HR ivabradine without influence on peripheral BP decreased CSP compared with atenolol.

P5.25

DIURNAL VARIATION OF CARDIO-ANKLE VASCULAR INDEX IN INDIVIDUALS WITH AND WITHOUT HEART DISEASE

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Background: Clinical studies revealed age and pathological-related arterial stiffening. Arterial stiffening is associated with a higher risk of cardiovascular disease. Cardio-ankle vascular index (CAVI), which reflects both central elastic and peripheral muscular arterial stiffness, has been applied as a simple noninvasive method to evaluate the risk for cardiovascular events. However, whether it is necessary to standardize the time of the day when performing this measurement is unknown. We aim to examine the effect of daytime on CAVI in individuals with and without heart disease.

Methods: We investigated the daytime variation of CAVI using Vasera VS-1500N (Fukuda Denshi; Japan) in 23 healthy young individuals (28.3 ± 4.7 yr, HY), 22 healthy elderly individuals (61.1 ± 9.0 yr, HE) and 25 patients with heart disease (63.9 ± 11.5 yr, HD).

Results: The effect of time on CAVI was shown to be significant in both univariate and multivariate analysis. Age was found as a significant determinant of CAVI ($p < 0.001$). After adjustment for age, sex and MAP, CAVI was shown to be 4% (09:00 versus 13:00, $p=0.022$) and 5% higher (09:00 versus 17:00, $p=0.002$) in the morning than the following time points. Furthermore, the patterns of variation over the day showed no significant differences among groups in CAVI.

Conclusion: CAVI showed a significantly higher value in the morning, which provides further support to standardize the time for measurements of arterial stiffness using CAVI in routine clinical practice and longitudinal studies.

P5.26

24 HOURS PULSATILE HEMODYNAMICS IN BORDERLINE VERSUS RESISTANT HYPERTENSIVES

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Background: Diurnal variations of brachial blood pressure have important prognostic implications. The aim of this study was to investigate differences in day- and night-time values of brachial and central hemodynamic parameters in two groups of borderline and resistant hypertensives.

Methods: We performed 24 hour pulse wave analysis, using a brachial cuff and validated ARCSolver algorithms. Central pressures were derived with a generalized transfer function using measured mean and diastolic pressure for calibration. 50 borderline hypertensives (BH; mean age: 52 years, mean 24h brachial blood pressure (bBP): 123/81 mmHg) and 25 resistant hypertensives (RH; mean age: 58 years, mean 24h bBP: 138/85 mmHg) were included in the study. Day-time was specified as 09:00-21:00 and night-time as 01:00-06:00.

		BH	RH
Brachial systolic BP (mmHg)	Day	127*	139 ⁵
	Night	113	135 ⁵
Central systolic BP (mmHg)	Day	128	144 ⁵
	Night	123	147 ⁵
Brachial pulse pressure (mmHg)	Day	41	51 ⁵
	Night	40	54 ⁵
Central pulse pressure (mmHg)	Day	41*	54 ⁵
	Night	49	64 ⁵
Heart rate (bpm)	Day	72*	69*
	Night	59	60
Alx	Day	23*	29* ⁵
	Night	29	38 ⁵
Alx@75	Day	22	25*
	Night	20	30 ⁵
Amplitude backward wave (mmHg)	Day	16*	22* ⁵
	Night	21	28 ⁵
Reflection magnitude	Day	61*	64*
	Night	70	71

* $p < 0.05$ day vs night; ⁵ $p < 0.05$ BH vs RH.