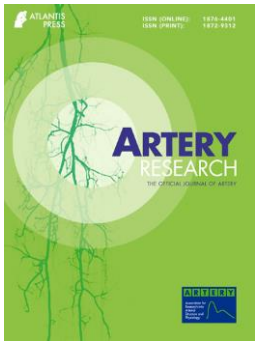

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P5.21: EARLY IABP INFLATION SUPPRESSES LEFT VENTRICULAR FUNCTION AS INDICATED BY WAVE INTENSITY ANALYSIS

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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.

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