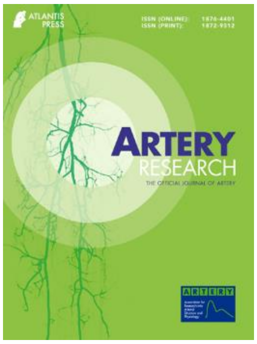


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# 5.1: PULSE PRESSURE AMPLIFICATION AND RENAL FUNCTION IN HYPERTENSION

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### 4.4
### MODULATION OF ARTERIAL TONE INFLUENCES PULSE WAVE VELOCITY IN PROPORTION TO CHANGE IN ARTERIAL DIAMETER

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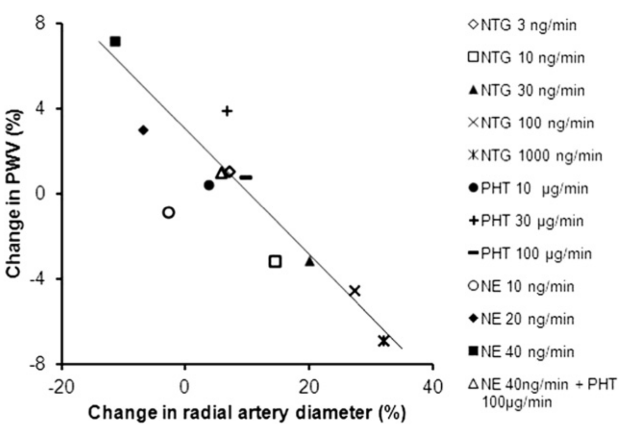
Nitric oxide (NO) is known to influence muscular conduit artery pulse wave velocity (PWV). Whether these changes are mediated exclusively by NO or may be a general consequence of a change in arterial tone is unknown. The aim of this study was to examine changes in local PWV over a range of arterial tone modulated by vasoconstrictor/dilator drugs. Healthy normotensive men (n=7) aged 19-53 years were studied. The right brachial artery was cannulated using a 27 gauge needle. Nitroglycerin (NTG 0.03, 0.1, 0.3, 1 μg/min), phentolamine (PHT 10, 30,100 μg/min) and (NE 0.01, 0.02, 0.04 μg/min) alone and with PHT (100 μg/min) were infused intra-arterially on separate occasions. Radial artery diameter (RAD) was measured by high resolution ultrasound; PWV was measured over the brachial to radial path by simultaneous recording from brachial and radial blood pressure cuffs inflated to 60 mmHg. The path distance was taken from the proximal edge of the upper arm cuff to that of the wrist cuff. NE (0.04 μg/min) reduced RAD by 11.4±4.1% but when co-infused with PHT (100 μg/min) increased RAD by 5.8±3.5%. NTG (1 μg/min) increased RAD by (32.1±5.4%). Over this range of arterial tone, changes in PWV were closely related to those in RAD (figure, R= -0.89; *P*<0.05). This suggests that, under physiological conditions, PWV in muscular arteries is determined by smooth muscle tone rather than being influenced by a specific signalling pathway.




### 4.5
### LOCALISED MICROMECHANICAL REMODELLING OF THE AORTA IN A MODEL OF TYPE 1 DIABETES

R. Akhtar [1], X. Zhao [2], N. Gardiner [2], B. Derby [2], J. K. Cruickshank [3], M. J. Sherratt [2]
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Whilst altered arterial stiffness is a hallmark of diabetes, the causative mechanisms and molecular targets remain poorly defined. Using a streptozotocin (STZ)-induced rat model of type 1 diabetes we have: i) mapped the relative micromechanical changes in the elastic lamellae (EL) and interlamellar regions (ILR) of the aorta and ii) characterised the effects of diabetes on the structure fibrillin microfibrils (long lived elastic fibre components which sequester TGF-β and hence play a key role in tissue homeostasis).
Diabetes was induced in five adult rats by STZ injection. These rats and five age-matched controls were killed after 8 weeks. Aortic cryosections were imaged by scanning acoustic microscopy to map variations in speed of sound (a measure of tissue stiffness). Fibrillin microfibrils were isolated by bacterial collagenase digestion and imaged by atomic force microscopy.
Exposure to a diabetic mileu affected the mechanical properties of aortic EL and ILR. In diabetic animals mean speed of sound was significantly reduced in EL (control, 1766±13 ms$^{-1}$; diabetic, 1722±11 ms$^{-1}$, p<0.05) and in the ILR (control, 1902±8 ms$^{-1}$; diabetic, 1868±11 ms$^{-1}$, p<0.001). Fibrillin microfibril periodicity was also profoundly affected in diabetic animals (control, uni-modal distribution centred at 56nm; diabetic, bi-modal distributions centred at 52 and 78nm).
These observations suggest that profound micro-mechanical changes occur in diabetic blood vessels, in both the EL and collagen-rich ILR, and that pathological changes in the nano-structure of homeostatic components may play a role in driving localised remodelling in these tissues.

### 4.6
### ELEVATION IN CENTRAL BLOOD PRESSURE DURING EXERCISE IS PREDOMINANTLY DRIVEN BY FORWARD-PROPAGATING WAVES: A FIRST IN MAN INVASIVE EXERCISE STUDY

M. G. Schultz [1], J. E. Davies [2], A. Black [3], P. Roberts-Thomson [3], A. D. Hughes [2], J. E. Sharman [1]
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**Introduction:** Exercise hypertension independently predicts cardiovascular mortality, but little is known on exercise central haemodynamics. This study aimed to determine contributions of arterial wave travel and aortic reservoir characteristics to central blood pressure (BP) during exercise. We hypothesised exercise central BP would be principally related to forward wave travel and aortic reservoir function.
**Methods:** Invasive pressure and flow velocity were recorded in the ascending aorta via sensor-tipped intra-arterial wire in 10 participants (age 55±10 years, 70% male) with normal left-ventricular function and free from obstructive coronary artery disease. Measures were recorded at baseline and during supine cycle ergometry. Using wave intensity analysis, dominant wave types throughout the cardiac cycle were identified (forward, backward, compression and decompression), and aortic reservoir and excess pressure were calculated.
**Results:** Central systolic BP increased significantly with exercise (19±12 mmHg, P<0.001). This was associated with significant increases in early systolic forward compression waves (15x10$^{6}$ ± 18x10$^{6}$ W.m$^{-2}$s$^{-2}$, P=0.025) and forward decompression waves in late systole (9x10$^{6}$ ± 5x10$^{6}$ W.m$^{-2}$s$^{-2}$, P<0.001). Despite significant augmentation in BP (+10%, P=0.023), backward (reflected) waves did not increase in magnitude (-1x10$^{6}$±2x10$^{6}$ W.m$^{-2}$s$^{-2}$, P=0.241). Excess pressure rose significantly with exercise (16±9 mmHg, P<0.001), and reservoir pressure integral fell (-5x10$^{5}$ ± 5x10$^{5}$ W.m$^{-2}$s$^{-2}$, P=0.010). The change in reflection coefficient negatively correlated with change in central systolic BP (r=-0.682, P=0.030).
**Conclusion:** Raised exercise central BP is principally driven by increasing aortic forward wave propagation generated by left ventricular ejection, and not wave reflection. These findings have relevance to understanding the pathophysiology of exercise hypertension.

## Oral session 5
## Free Communication Oral Presentations
## In association with the ESH Working Group on Vascular Structure and Function

### 5.1
### PULSE PRESSURE AMPLIFICATION AND RENAL FUNCTION IN HYPERTENSION

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In epidemiological studies including serum creatinine or estimates of glomerular filtration rate (GFR), pulse pressure (PP) emerged as significant predictor of cardiovascular risk and major determinant of age-associated decline in GFR. The finding is mainly observed in subjects with hypertension and/or renal failure but less in atherosclerotic subjects. Blood pressure was measured invasively in the ascending and abdominal aorta (at the level of kidneys) of 101 subjects undergoing coronary angiography. Independently of age, sex, and presence of coronary stenosis, amplification of PP between the ascending and terminal aorta was over 10 mm Hg (P<0.001), whereas mean blood pressure remained unchanged. Irrespective of PP measured in the ascending aorta and at the level of renal arteries, amplification was significantly related to proteinuria. Increased plasma creatinine and aortic pulse wave velocity were independently and positively correlated (P<0.001). The relationship between PP and renal function was mainly present in patients 60 years of age or older. Finally, renal transplant patients and their donors were recruited for evaluation of aortic stiffness and determination of the post-transplant decline trends in GFR. Determinants of filtration rate decline were evaluated at 1 year and at a mean of 9.2 ± 3.5 years after transplantation. The first year decline was related to smoking and acute rejection but the later decline was significantly and exclusively associated with donor age and aortic stiffness. In hypertensive humans, the significant association between PP and GFR indicates a cross-talk between the two parameters with arterial stiffness, and not vascular resistance, as major mediator.

## 5.2
## CUFF AND TONOMETER BASED DEVICE FOR ASSESSMENT OF CAROTID TO FEMORAL PULSE WAVE VELOCITY: VALIDATION ACCORDING TO ARTERY SOCIETY GUIDELINES

M. Butlin [1], E. Bozec [2], E. Millet-Amaury [2], G. Pucci [3], F. Battista [3], A. Qasem [1], G. Schillaci [3], P. Boutouyrie [2], A. Avolio [1]
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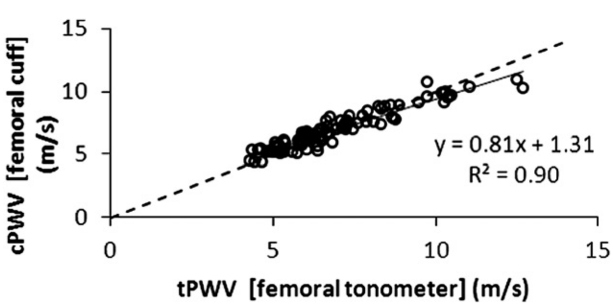
**Background:** There is increasing interest in cuff-based devices for assessment of arterial pulse wave velocity (PWV). A recently developed device utilises a thigh cuff and carotid applanation tonometry for assessment of carotid to femoral PWV (cPWV; SphygmoCor XCEL, AtCor Medical; test device). Our aim was to validate the device against ECG gated tonometric measurement of PWV (tPWV) (SphygmoCor CvMS; control device) according to Artery Society Guidelines [1].
**Methods:** We recruited 94 subjects (48 female, 22-83 years, mean age 45.6 ± 19.4) in 3 centres (Australia, France, Italy). The thigh cuff was inflated automatically to sub-diastolic pressure and the cuff waveform was recorded simultaneously with the tonometric carotid waveform. Control and test devices were used in random order. PWV was determined from wave foot-to-foot delay and distance from suprasternal notch to femoral site or top of cuff, minus distance from suprasternal notch to carotid site. The average was computed of triplicate measurements by two operators.
**Results:** A high correlation was found between devices ($R^2$=0.90; Figure) with a mean difference of -0.02 ± 0.61 (SD) m/s. Mean difference and standard deviation (SD) between cPWV and tPWV was well within the "excellent" category acceptance criteria of the Artery Society guidelines (<0.5 m/sec and <0.80 m/sec, respectively).
**Conclusion:** The femoral cuff technique gives comparable PWV values to those acquired with the accepted standard ECG gated carotid/femoral tonometry PWV measurement technique.




[1] Wilkinson IB *et al.* Artery Society guidelines for validation of non-invasive haemodynamic measurement devices: Part 1, arterial pulse wave velocity. *Artery Research* 2010; 4:34-40

## 5.3
## RELATIONSHIP BETWEEN SHORT-TERM BLOOD PRESSURE VARIABILITY AND LARGE-ARTERY STIFFNESS IN HUMAN HYPERTENSION

G. Parati [1,2], G. Bilo [1], G. Pucci [3,6], S. Laurent [4,7], I. Macquin-Mavier [5], P. Boutouyrie [4,7], F. Battista [3,6], L. Settimi [3], G. Desamericq [5], G. Dolbeau [5,8], A. Faini [1], P. Salvi [1], E. Mannarino [3], G. Schillaci [3,6]
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Short-term blood pressure (BP) variability predicts cardiovascular complications in hypertension, but its association with large-artery stiffness is poorly understood and confounded by methodological issues related to the assessment of BP variations over 24h.
Carotid-femoral pulse wave velocity (cfPWV) assessment and 24-hour ambulatory BP monitoring were performed in 911 untreated, nondiabetic patients with uncomplicated hypertension (learning population) and in 2089 mostly treated hypertensive patients (test population). Short-term systolic BP (SBP) variability was calculated as the following: (1) SD of 24-hour, daytime, or nighttime SBP; (2) weighted SD of 24-hour SBP; and (3) average real variability (ARV), that is, the average of the absolute differences between consecutive SBP measurements over 24 hours. In the learning population, all of the measures of SBP variability showed a direct correlation with cfPWV (SD of 24-hour, daytime, and nighttime SBP, $r$=0.17/0.19/0.13; weighted SD of 24-hour SBP, $r$=0.21; ARV, $r$=0.26; all $P$<0.001). The relationship between cfPWV and ARV was stronger than that with 24-hour, daytime, or nighttime SBP ($P$<0.05) and similar to that with weighted SD of 24-hour SBP. In the test population, ARV and weighted SD of 24-hour SBP had stronger relationships with cfPWV than SD of 24-hour, daytime, or nighttime SBP. In both populations, SBP variability indices independently predicted cfPWV along with age, 24-hour SBP, and other factors.
We conclude that short-term variability of 24-hour SBP shows an independent relation to aortic stiffness in hypertension. This relationship is stronger with measures of BP variability focusing on short-term changes, such as ARV and weighted 24-hour SD.

## 5.4
## HIGHER PULSE PRESSURE IN OLDER PEOPLE IS ASSOCIATED WITH SMALLER AORTIC LUMEN AREA

G. F. Mitchell [1], A. A. Torjesen [1], S. Sigurdsson [2], J. J. M. Westenberg [3], L. J. Launer [4], V. Gudnason [2], T. B. Harris [4]
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High pulse pressure (PP) contributes to the pathogenesis of hypertension and is associated with adverse cardiovascular disease outcomes. There is consensus that aortic wall stiffening contributes to higher PP. However, the role of lumen size in the pathogenesis of elevated PP remains controversial. Prior studies showing an unexpected inverse association between PP and lumen area have been criticized for using echocardiography, which affords limited views of the thoracic aorta. Therefore, we performed cine magnetic resonance imaging (MRI) of the ascending aorta 5 mm above the aortic valve and the proximal and distal descending thoracic aorta in 423 older participants (age 72 to 94, mean 79 years; 57% women) in the Age, Gene/Environment Susceptibility-Reykjavik Study (AGES-Reykjavik). Immediately prior to MRI, supine auscultatory blood pressure (141±19/64±9, PP=77±18 mmHg) and tonometry of brachial, radial, femoral and carotid arteries were performed. Mean aortic lumen area during the cardiac cycle was computed at each level and averaged across the 3 levels to give average lumen area ($LA_M$). Wall area (WA) and elastance ($E = PP \times A_D/(A_S - A_D)$) were similarly averaged. In linear regression models, $LA_M$ was negatively related to PP when considered alone (Model 1) and in a model that adjusted for age, sex, height, weight, heart rate, total and HDL cholesterol, triglycerides, estimated GFR, diabetes, glucose, HbA1c and history of smoking (Model 2). The relation persisted after further adjustment for E and WA (Model 3). In our sample of older people, higher pulse pressure is associated with a smaller lumen area of the thoracic aorta.

**Table** Relations between pulse pressure and average aortic lumen area.

| | Beta | SE | P |
|---|---|---|---|
| Model 1 | 4.5 | 0.9 | <0.0001 |
| Model 2 | 5.7 | 1.1 | <0.0001 |
| Model 3 | 5.9 | 1.2 | <0.0001 |

Effects are expressed as pulse pressure increase per SD decrease in mean lumen area (mm Hg/SD).