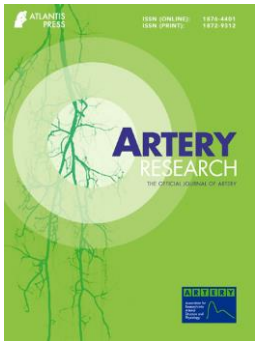

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3.3: TOTAL ARTERIAL ELASTANCE IS MORE STRONGLY ASSOCIATED WITH CARDIOVASCULAR DISEASE THAN CAROTID-FEMORAL PULSE WAVE VELOCITY

C.M. Park, T. Tillin, K. March, N. Chaturvedi, A.D. Hughes

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hour. Therefore, a fee for service to measure central BP of approximately $30 could be both economically and financially justifiable.
Conclusions. Management of hypertension using central BP has cost-savings relating to decreased medication and may be regarded as cost-neutral when factoring in a fee for central BP measurement.

Oral Session 3

Young Investigator Oral Presentations

In association with the European Society of Hypertension Working Group on Vascular Structure and Function

3.1
OUTCOME-DRIVEN THRESHOLDS FOR AMBULATORY PULSE PRESSURE IN 9938 PEOPLE RECRUITED FROM 11 POPULATIONS

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Background. Evidence-based thresholds for risk stratification based on pulse pressure (PP) are currently unavailable.
Methods. To derive outcome-driven thresholds for the 24-h ambulatory PP, we analyzed 9938 people randomly recruited from 11 populations (47.3% women). After age stratification (<60 *vs.* ≥60 years) and using average risk as reference, we computed multivariable-adjusted hazard ratios (HRs) to assess risk by tenths of the PP distribution or risk associated with stepwise increasing (+ 1 mm Hg) PP levels.
Results. Among 6028 younger participants (68,853 person-years), the risk of cardiovascular (HR, 1.58; $P = 0.011$) or cardiac (HR, 1.52; $P = 0.056$) events increased only in the top PP tenth (mean, 60.6 mm Hg). Using stepwise increasing PP levels, the lower boundary of the 95% confidence interval of the successive thresholds did not cross unity. Among 3910 older participants (39,923 person-years), risk increased ($p \leq 0.028$) in the top PP tenth (mean, 76.1 mm Hg). HRs were 1.30 and 1.62 for total and cardiovascular mortality, and 1.52, 1.69 for all cardiovascular, cardiac events. The lower boundary of the 95% confidence interval of the HRs associated with stepwise increasing PP levels crossed unity at 64 mm Hg. While accounting for all covariables, the top tenth of PP contributed less than 0.3% (generalized R^2 statistic) to the overall risk among elderly.
Conclusions. In randomly recruited people, ambulatory PP does not add to risk stratification below age 60; in the elderly, PP is a weak risk factor with levels below 64 mm Hg probably being innocuous.

3.2
LOCAL ARTERIAL STIFFNESS ASSESSED BY ECHO TRACKING IS NOT ASSOCIATED WITH AN INCREASED PULSE WAVE VELOCITY IN HYPERCHOLESTEROLEMIC RABBIT

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Arterial stiffness (AS) is generally measured by regional arterial pulse wave velocity (rPWV) or locally by arterial distensibility. Although often used interchangeably, some studies show a weak correlation between these well accepted AS index depending on population. Thus, we aim to investigate in an experimental model of hypercholesterolemic rabbit the comparison between arterial pulse wave velocity and local arterial distensibility. Male New Zealand rabbits (8 week-old) received diet with 0.3% cholesterol for 17 weeks. Under anaesthesia, rPWV was measured with catheters between ascending aorta and illiac artery by the foot-to-foot method. Local arterial stiffness was assessed by echotracking with local PWV using the Moens-Korteweg equation $= (1.050xD)^{-1/2}$, from distensibility as $D = A/AxP$ at different sites: carotid (cPWV), aorta (aPWV and femoral (fPWV). Vessel lesions were analysed by fat red staining.
Table 1 presents the results. As expected, local PWV is comparable in elastic arteries (carotid and aorta) and higher in muscular arteries (femoral artery). Surprisingly, we observe no modification of rPWV and cPWV between the two groups. However, local aPWV and fPWV are increased in hypercholesterolemic rabbits. Only a correlation between the rPWV and local aPWV was observed in control rabbits ($r^2=0.76$ $P = 0.02$). Vessel plaques are more important in aorta and femoral artery than in carotid artery. In conclusion, in a rabbit model of atherosclerosis, we observe an increase in local arterial stiffness in aorta and femoral artery associated with plaques. However, this local stiffening seems to have a weak impact on regional PWV.

Table 1

	rPWV (m/s)	cPWV (m/s)	aPWV (m/s)	fPWV (m/s)
Control	5.0 ± 0.2	6.6 ± 0.4	5.9 ± 0.2	18.8 ± 1.2
Hyperchol	4.9 ± 0.2	6.7 ± 0.4	6.9 ± 0.3 *	25.9 ± 1.8 *

3.3
TOTAL ARTERIAL ELASTANCE IS MORE STRONGLY ASSOCIATED WITH CARDIOVASCULAR DISEASE THAN CAROTID-FEMORAL PULSE WAVE VELOCITY

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Background. Arterial stiffness is associated with an increased risk of cardiovascular disease (CVD) and carotid-femoral pulse wave velocity (cfPWV) is considered as the 'gold standard' measurement method. However cfPWV does not assess the stiffness of the proximal aorta and its sensitivity at higher levels of arterial stiffness, such as in the elderly, has been questioned. We compared associations between CVD and cfPWV and total arterial elastance (TAE) in a cohort of elderly individuals.
Methods. 961 individuals (69±6yrs; 76% male) underwent echocardiography (Philips iE33), cerebral MRI, cfPWV (Pulse Trace) and central blood pressure (Pulsecor) measurements. Central pulse pressure and stroke volume were used to calculate TAE. Coronary heart disease (CHD) was defined as a coronary event or revascularisation identified by medical record review, and adjudicated by an independent committee. Cerebrovascular disease was defined as those with infarcts (2+) on MRI or adjudicated history of stroke. Associations are presented as odds ratios (OR) [95% confidence intervals] by logistic regression.
Results. A modest correlation was observed between cfPWV and TAE (spearman's rho = 0.14). Associations with CVD were stronger for TAE than cfPWV and subdividing participants based on cfPWV had little effect on associations (Table).

Table. Association of cfPWV and TAE with CHD and cerebrovascular disease (adjusted for age, sex and ethnicity). Individuals were also subdivided into two groups based on their cfPWV.

	All participants $n=961$		PWV < 12 ms^{-1} $n = 643$		PWV ≥ 12 ms^{-1} $n = 318$	
	OR [95%CI]	*P*	OR [95%CI]	*P*	OR [95%CI]	*P*
CHD						
cfPWV	1.05 [1.01,1.09]	0.03	1.06 [0.93,1.21]	0.4	1.05 [0.97,1.10]	0.2
TAE	2.15 [1.34—3.45]	0.002	2.11 [1.14, 3.91]	0.02	2.00 [0.93,4.30]	0.07
Cerebro-vascular disease						
cfPWV	1.04 [0.99,1.08]	0.1	0.99 [0.88,1.12]	0.9	1.01 [0.93,1.10]	0.8
TAE	1.40 [0.86-2.26]	0.2	1.54 [0.85,2.80]	0.2	1.12 [0.49,2.59]	0.8

Conclusions. cfPWV is less strongly associated with CHD and cerebrovascular disease than TAE and may have more limited prognostic value in elderly individuals.

3.4
VASCULAR AGING MAY CONTRIBUTE TO TELOMERE LENGTH IN PATIENTS WITH T2DM

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It is known that telomere length (TL) shortening is a marker of cell aging, which accelerated and progresses in type 2 diabetes mellitus (T2DM), despite the patient's age, leading to the vascular aging.
The aim of the study was to compare the vascular and cellular aging in patients with and without T2DM.
Methods. TL was assessed by quantitative polymerase chain reaction (PCR) in 35 patients with T2DM (mean age 61±2,6 years) and in 43 healthy patients in mean age of 51±1,8 years. IMT and PP were determined by ultrasonography in both left and right carotid arteries. AS was appreciated by aortic pulse wave velocity (PWV) measuring by SphygmoCor (AtCor Medical).
Results. All patients were divided into 2 groups by TL — "long" and "short" telomeres. Comparison of vascular aging parameters was carried out in groups with and without T2DM. Results are summarized in Tables 1 and 2.

Table 1 Comparison in patients with "long" TL.

	T2DM+ (n = 15)	T2DM− (n = 23)	p
TL	10.2 ± 0.05	10.4 ± 0.1	0.06
PWV (m/s)	10.58 ± 0.1	10.5 ± 0.5	0.913
IMT (mm)	0.904 ± 0.09	0.77 ± 0.03	0.1227
PP (number)	0.886 ± 0.4	0.782 ± 0.2	0.979

Table 2 Comparison in patients with "short" TL.

	T2DM+ (n = 20)	T2DM− (n = 20)	p
TL	9.24 ± 0.1	9.28 ± 0.06	0.735
PWV (m/s)	15.08 ± 1.3	10.7 ± 0.5	**0.0151**
IMT (mm)	0.87 ± 0,1	0.78 ± 0.1	0.1814
PP (number)	1.125 ± 0.29	0.789 ± 0.22	**0.04**

Conclusion: in patients with short TL and T2DM the severity of vascular disorders is higher than in healthy people. In contrast, in patients with long TL with T2DM there are no significant differences in the vascular structure as compared with healthy individuals.

3.5
CHANGES IN BLOOD PRESSURE AND ARTERIAL MECHANICAL PROPERTIES AFTER ANTIANGIOGENIC DRUGS: ASSOCIATION WITH CANCER PROGRESSION AND MORTALITY

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Objective. Hypertension is a frequent side effect of antiangiogenic drugs (AAD). Targeting VEGF pathway may also affect large and small artery properties, along with or independently of blood pressure changes. We hypothesized that large and small artery property changes in response to AAD reflect their effect on the microcirculation at the site of the tumor, and thus may be related to cancer progression and mortality.
Design and Method. We included 60 patients [age 58 (15) years, mean SBP 127(21) mmHg] in whom treatment with AAD was indicated for various metastatic solid tumors. Noninvasive arterial investigation was performed before AAD (V0), 1 week later (V1) and then every two weeks for two months (V1 to V4): carotid-femoral pulse wave velocity (cfPWV), central SBP and augmentation index (cAIx) by applanation tonometry (SphygmoCor®), and carotid stiffness (CStiff) and internal diameter (CiD) by high resolution echotracking (Artlab®). Cancer progression and mortality were assessed at 6 months.
Results and Conclusion. 28(47%) patients developed hypertension during follow-up. bSBP significantly increased during follow-up (V0-V1: +9.3 ± 15.2mmHg, $P < 0.001$; V0-V4: +6.0 ± 17.8mmHg, $P = 0.03$), as well as PWV, CStiff, and CiD. Baseline cAIx predicted cancer progression (RR=0.73 per 10%) and mortality (RR=0.73 per 10%, $P < 0.001$) while SBP did not. The V0-V1 increase in CStiff predicted cancer progression (RR=1.37 per 1 m/s, $P = 0.02$), independently of age and MBP. In conclusion, increased AIx and arterial stiffness, but not brachial or central SBP, were related with the effects of AAD on cancer progression and mortality.

Oral Session 4
Young Investigator Oral Presentations

4.1
ACUTE, SYMPATHETIC-INDEPENDENT INCREASES IN HEART RATE BY WAY OF CARDIAC PACING RAISES AORTIC AND BRACHIAL BLOOD PRESSURE WITH INCREASED CARDIAC OUTPUT AND ARTERIAL STIFFNESS

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Objective. Whilst the effects of heart rate (HR) on the cardiovascular system have been studied cross-sectionally or during exercise, the effect of acute, sympathetic-independent changes in HR on arterial stiffness, cardiac output (CO), mean pressure (MAP) and total peripheral resistance (TPR) has not previously been studied.
Methods. Sixteen subjects (aged 70 to 79 years, 3 female) with *in situ* permanent cardiac pacemakers or implantable cardioverter defibrillators were studied. Each subject was paced in a random order at 60 to 100 beats per minute (bpm) in 10 bpm increments. At each heart rate, TPR and CO were derived from measured finger arterial pressure waveform (Finometer®). Brachial (b) and central aortic (c) systolic (SBP), diastolic (DBP), MAP, and aortic augmentation index (AIx) were determined by brachial cuff-based pulse wave analysis, and carotid-femoral pulse wave velocity (PWV) measured using a thigh cuff and carotid tonometry (SphygmoCor® XCEL). Aortic to brachial pulse pressure amplification (PPA) was calculated.
Results. All parameters except for TPR and cSBP changed significantly with HR (Table, data presented as mean ± sem). This indicated that HR-driven changes in MAP were due to increased CO, not changes in TPR. PWV showed an increase with increasing HR. However, this was not significant once corrected for changes in MAP (PWV_c, Table).
Conclusion. Acute, sympathetic-independent increases in HR through cardiac pacing raises CO, which in turn increases MAP and results in increased arterial stiffness.

	60 bpm	80 bpm	100 bpm	P
bSBP (mmHg)	127 ± 5	131 ± 4	133 ± 5	0.01
bDBP (mmHg)	69 ± 2	76 ± 2	83 ± 3	<0.001
cSBP (mmHg)	116 ± 4	118 ± 4	120 ± 4	0.51
cDBP (mmHg)	70 ± 2	76 ± 2	86±3	<0 .001
MAP (mmHg)	87 ± 2	94 ± 3	102 ± 4	<0.001
TPR (dyn.s/cm^5)	1749 ± 276	1656 ± 227	1482 ± 137	0.14
CO (L/min)	5.0 ± 0.4	5.4 ± 0.4	5.8 ± 0.4	<0.001
PPA	1.26 ± 0.01	1.30 ± 0.02	1.50 ± 0.03	<0.001
AIx (%)	33 ± 3	31 ± 3	22 ± 3	<0.001
PWV (m/s)	9.5 ± 0.5	10.3 ± 0.5	11.1 ± 0.4	<0.001
PWV_c (m/s)	10.3 ± 0.4	10.4 ± 0.4	10.4 ± 0.4	0.75

Data for 70 bpm and 90 bpm were measured and analysed but not shown

4.2
DO LEVEL AND VARIABILITY OF SYSTOLIC BLOOD PRESSURE PREDICT ARTERIAL PROPERTIES OR VICE VERSA?

Yan-Ping Liu [1], Yu-Mei Gu [1], Lutgarde Thijs [1], Kei Asayama [1,4], Yu Jin [1], Lotte Jacobs [1], Tatiana Kuznetsova [1], Peter Verhamme [1], Luc Van Bortel [2], Harry A. J. Struijker-Boudier [3], Jan A. Staessen [1]
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