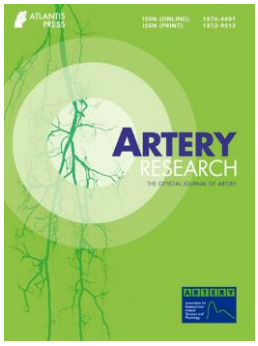

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# 4.2: DO LEVEL AND VARIABILITY OF SYSTOLIC BLOOD PRESSURE PREDICT ARTERIAL PROPERTIES OR VICE VERSA?

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

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**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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**Background**. No longitudinal study addressed whether systolic blood pressure level (SBPL) or variability (SBPV) predict arterial properties or vice versa.
**Methods and Results**. In families randomly recruited from a Flemish population, we determined SBPL and SBPV from 5 consecutive blood pressure readings. The indexes of SBPv were variability independent of mean (VIM), the difference between maximum and minimum SBPL (MMD), and average real variability (ARV). We measured carotid intima-media thickness (cIMT) and distensibility (cD) by ultrasound and carotid-femoral pulse wave velocity (cfPWV) by tonometry (SphygmoCor, version 8.2). Effect sizes were computed for 1 SD increments in the predictors, while accounting for covariables and family clusters. Among 1171 participants (51.0% women; mean age, 39.8 years), followed up for 2.48 years (median), higher SBPL predicted ($P \leq 0.036$) higher cIMT (+14 μm), lower cD (−1.57 10-3/kPa) and faster cfPWV (+0.298 m/s) at follow-up, whereas none of the SBPV indexes predicted the arterial traits at follow-up ($P \geq 0.11$). In a subset of 749 participants, followed up for another 3.07 years, lower cD predicted ($P \leq 0.026$) higher SBPL (+1.69 mm Hg), VIM (+0.304 units), MMD (+1.05 mm Hg) and ARV (+0.389). Higher cfPWV predicted a 1.06 mm Hg increase SBPL ($P = 0.027$).
**Conclusions**. Temporality and effect size suggest that SBPL but not SBPV cause arterial stiffening and cIMT thickening. Carotid stiffening, independent of SBPL, predicts SBPV, possibly because baroreflexes originating from a stiffer carotid artery wall are impaired. Finally, stiffening of the aorta contributes to the age-related SBPL possibly because faster returning reflected waves augments SBPL.

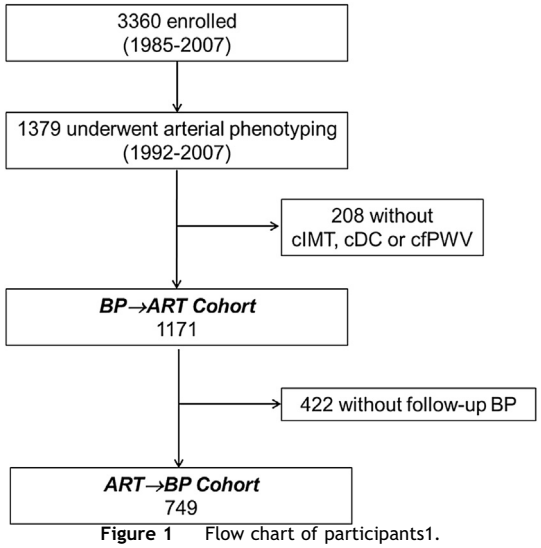

**Figure 1** Flow chart of participants1.

### 4.3
### CAROTID HEMODYNAMICS DURING SYMPATHETIC NERVOUS SYSTEM STIMULATION VIA HANDGRIP AND COLD PRESSURE TESTING IN YOUNG HEALTHY SUBJECTS

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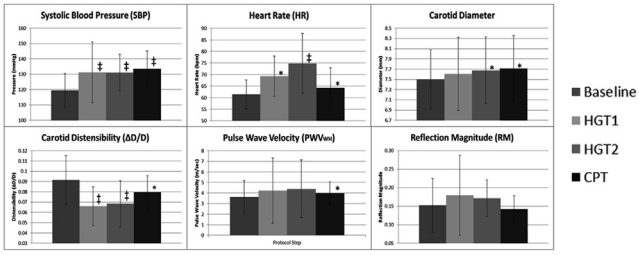
**Introduction**. Assessing arterial properties and hemodynamics in response to a physiological perturbation might provide additional information on an individual's "vascular health". The aim of this study was to assess the feasibility of measuring changes in carotid stiffness and hemodynamics in response to sympathetic nervous system stressors (Hand Grip Test (HGT) or Cold Pressor Test (CPT)).
**Methods**. A non-invasive protocol, consisting of two HGT (30% (HGT1) and 40% (HGT2) of maximal voluntary contraction) and CPT, was performed in 12 young healthy volunteers (6 males/6 females). Measurements included continuous finger blood pressure recordings (NexFin; non-dominant hand) and ultrasound measurement of common carotid diameter distension and flow velocity at discrete moments in time during the protocol (GE Vivid 7 system). Carotid distensibility (ÄD/D), local wave speed using the waterhammer equation ($PWV_{WH}$) and reflection magnitude (RM; ratio of backward to forward diameter wave) were derived from the data.
**Results**. Consistent with the overall increase in blood pressure, carotid diameter increased while ÄD/D decreased. $PWV_{WH}$ and RM showed an increase during both HGT and a decrease during CPT (see Figure).
**Conclusion**. It is feasible to monitor the carotid hemodynamic response to a sympathetic nervous system stimulus. In this young, healthy population, the net result of the increased diameter and decreased distensibility on local $PWV_{WH}$ was similar for all tests (increase) and on the magnitude of reflections was different for HGT (increase) and CPT (decrease).



The difference from baseline is indicated as significant * ($p<0.05$) or highly significant ‡ ($p<0.001$).

### 4.4
### ASSOCIATIONS BETWEEN OBJECTIVELY MEASURED PHYSICAL ACTIVITY ENERGY EXPENDITURE AND CENTRAL HAEMODYNAMICS. THE ADDITION-PRO STUDY

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**Objective**. Physical activity (PA) has been associated with reduced cardiovascular disease (CVD). However, improvements in conventional risk factors do not explain the full benefit of PA on CVD risk. Therefore, we examined the association between PA and central haemodynamics to provide new insight into the link between PA and CVD.
**Methods**. We performed cross-sectional analyses of data from a health examination of 1,607 Danish adults at low to high diabetes risk. PA energy expenditure (PAEE) was measured by combined accelerometry and heart rate monitoring (ActiHeart®) expressed as kJ/kg/day. Aortic stiffness was assessed by applanation tonometry (SphygmoCor®), as aortic pulse wave velocity (aPWV), and central blood pressure was estimated from wave forms recorded at the radial artery. Associations between PAEE and central haemodynamics were examined by linear regression successively adjusted for sex, age, waist circumference, and smoking. Additionally, aPWV was adjusted for mean blood pressure. aPWV was logarithmically transformed. Individuals with previously myocardial or cerebral infarction were excluded ($n = 183$).
**Results**. Median age was 66 years (IQR: 61;71), 52% was men, median PAEE was 28 kJ/kg/day (IQR: 20;39), and median aPWV 8.0 m/s (IQR: 6.9;9.4). A higher PAEE of 10 kJ/kg/day was associated with a 1.0 % lower aPWV (CI: -0.17;-0.03). Associations with systolic blood pressure and pulse pressure were not statistically significant (-0.5 mmHg (CI: -0.11;0.01) and -0.02 mmHg (CI: -0.06;0.03)).