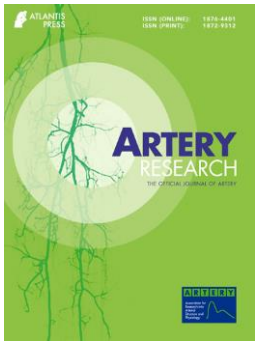

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# 3.4: VASCULAR AGING MAY CONTRIBUTE TO TELOMERE LENGTH IN PATIENTS WITH T2DM

E.N. Dudinskaya, N.V. Brailova, I.D. Strazhesko, O.Y. Isaikina, M.S. Pokrovskaya, O.N. Tkacheva, S.A. Boytsov, M.V. Shestakova

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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?

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