A maladaptive carotid remodeling was described in type 2 diabetes mellitus. However, it is still unknown whether it is a feature of diabetes per se, or rather a consequence of the frequent comorbidity with hypertension. We therefore studied the impact of diabetes, hypertension, and their combination on carotid geometry and remodeling, recruiting to this aim 139 subjects (31 normotensives-N, 38 hypertensives-HT, 24 diabetic normotensives-DMNT, and 46 diabetic hypertensives-DMHT). Common carotid diameter, intima-media thickness (IMT) and distension were determined using a real-time echotracking system "Carotid Studio". Local pulse pressure was obtained by applanation tonometry. Carotid static and pulsatile circumferential wall stress was calculated by Lamé equations. IMT was increasingly higher in the four groups, reaching the highest value in DMHT. As compared to NT, carotid lumen diameter was increased in HF and DMHT, but not in DMHT. HF showed significantly higher circumferential static wall stress than DMHT, while DMNT and NT had similar and significantly lower values. Carotid pulse pressure was similarly increased in DMHT and HT. In a logistic regression model adjusted for confounders, hypertension carried an increased risk (OR 2.33; CI-95% 1.02-5.63) of a widened lumen diameter, whereas diabetes carried an increased risk of IMT above the median value (OR2.49; 1.09-5.68). Hypertension, but not diabetes, was associated to an increased static and pulsatile stress (ORT7.42; 2.76-21.72, and 4.86; 1.95-12.10). In conclusion, maladaptive remodeling, previously reported in diabetic patients, is conceivably attributable to the concomitant presence of hypertension.

### Table 1: Comparison of carotid parameters among the study groups

<table>
<thead>
<tr>
<th>Parameter</th>
<th>NT</th>
<th>HT</th>
<th>DMNT</th>
<th>DMHT</th>
<th>p (trend)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lumen diameter (mm)</td>
<td>5.6±1.0</td>
<td>6.3±1.1*</td>
<td>5.9±0.6</td>
<td>6.3±1.1*</td>
<td>0.02</td>
</tr>
<tr>
<td>IMT (mm)</td>
<td>0.72±0.09</td>
<td>0.76±0.18</td>
<td>0.78±0.13*</td>
<td>0.81±0.14*</td>
<td>0.04</td>
</tr>
<tr>
<td>Static wall stress (kPa)</td>
<td>48.8±12.7</td>
<td>60.0±13.3*</td>
<td>48.8±12.6*</td>
<td>54.6±12.2**</td>
<td>0.001</td>
</tr>
<tr>
<td>Pulsatile wall stress (kPa)</td>
<td>27.6±6.0</td>
<td>36.0±12.3*</td>
<td>27.6±6.3*</td>
<td>32.5±9.0*</td>
<td>0.001</td>
</tr>
<tr>
<td>Pulse pressure (mmHg)</td>
<td>43.6±8.9</td>
<td>54.2±15.9*</td>
<td>44.7±8.9</td>
<td>53.8±11.8*</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

*P<0.05 vs NT; †P<0.05 vs HT; ‡P<0.05 vs DMNT; #P<0.05 vs DMHT.

**P3.06**

AORTIC STIFFNESS AND VITAMIN D ARE INDEPENDENT MARKERS OF AORTIC CALCIFICATION IN PATIENTS WITH PERIPHERAL ARTERY DISEASE

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**Background:** Arterial stiffness is a significant determinant of cardiovascular risk and is related to vascular calcification. Vitamin D may regulate arterial calcification and has been associated with cardiovascular survival benefits. However, data about the relationship between arterial stiffness, aortic calcification, and vitamin D levels in patients with peripheral artery disease (PAD) are limited.

**Aim:** To examine association between aortic calcification, arterial stiffness, and vitamin D levels in patients with symptomatic PAD and in healthy individuals.

**Materials and methods:** We studied 78 males with PAD (mean age 63.7 years) and 69 healthy men (mean age 54.7 years). Aortic pulse wave velocity (apPWV) and augmentation index (AbaX75) were determined by applanation tonometry using the Sphygmocor device. Aortic calcification score (ACS) was quantified by computed tomography. Serum 25-hydroxyvitamin D (25(OH)D) level was measured using a radioimmuno assay.

**Results:** ACS (6.4±5.3 vs 0.7±1.7 cm²); p<0.001, apPWV (10.1±2.5 vs 7.6±1.6 m/s); p<0.001, AbaX75 (28.2±8.1 vs 13.7±11.0); p<0.001, and 25(OH)D (37.7±14.0 vs 50.3±16.3 nmol/L); p<0.001 were different for the study groups. There was linear relationship between apPWV and ACS for the patients (p=0.02) and for the controls (p=0.049). 25(OH)D was associated with ACS only in the patient group (p=0.05). In multivariate analysis ACS was independently determined by apPWV and 25(OH)D in patients with PAD (R²=0.42; p<0.0001).

**Conclusion:** These results indicate that calcification of the thoracic and abdominal aorta is independently associated with aortic stiffness and serum 25(OH)D level in patients with PAD. Aortic stiffness and abnormal vitamin D level may contribute to vascular calcification in these patients.

**P3.07**

OXYGEN CONSUMPTION KINETICS IN SUPRA-ANAEROBIC THRESHOLD CONSTANT LOAD EXERCISES ALLOW TO QUANTIFY IN TRAINED AND UNTRAINED SUBJECTS CYTOCHROME C-OXIDASE INHIBITION BY NITRIC OXIDE AND SHOW THIS DIRECT EFFECT AFTER NITRATE

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2Sports Medicine Institute of Bologna, Bologna, Italy

This work aims to highlight by VO2 kinetics in constant load supra anaerobic threshold (AT) tests, the nitric oxide (NO) reversible direct inhibitory action on cytochromeC oxidase (cox). This results in decreased ability to oxidise electron flow (EF), by cytochromeC carried, in water, compensated by electron turnover (ET) increment via cox not only NO bound, thus giving EF conti-

nuity along the respiratory chain O2 ward. When NO production is increased