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### **P3.19: ALTERED WSS IN THE HUMAN AORTA WITH AGE – IMPLICATIONS FOR WALL REMODELLING AND LESIONS?**

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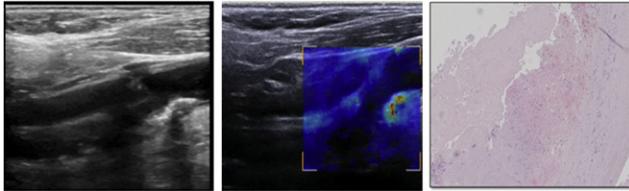
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**Results:** All patient presented with >80% stenosis in either of the internal carotid arteries. Mean YM was 108 (52-195) kPa. All five plaques varied in echogenicity classification. On histology, 2 plaques were 'probably or definitely' unstable and 2 were probably stable (Fig.1). There was better correlation between YM and histology finding compared to echogenicity.

**Conclusion:** This present insight into the role of novel ultrasonic based technique in providing further information in characterising carotid plaque properties which may be of value in identifying vulnerable plaques.



**Figure 1** Patient with echogenic plaque on B-mode, YM of 66kpa and histology classification of definitely unstable.

### P3.19 ALTERED WSS IN THE HUMAN AORTA WITH AGE – IMPLICATIONS FOR WALL REMODELLING AND LESIONS?

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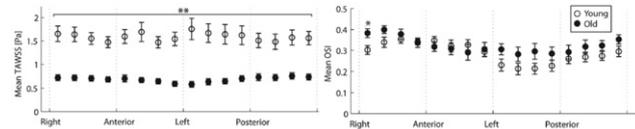
Arteries are continuously affected by pulsating blood flow resulting in a wall shear stress (WSS) load. WSS has been related both to the remodeling process in the arterial walls as well as atherosclerosis. These processes are both related to ageing.

10 young (23.5±1.4 years, BMI 21.8±1.4) and 8 old (58.0±2.8 years, BMI 24.0±2.3) subjects were studied. Computational fluid dynamics (CFD) together with magnetic resonance imaging (MRI) measurements were used to investigate the WSS in the healthy thoracic aorta (TA). MRI images (geometry and flow) were obtained and segmented to fit the CFD simulations. Time averaged WSS (TAWSS) and oscillating shear index (OSI) were extracted. In young and old subjects, the diameter of the descending TA was 26±2.2 mm vs. 36±3.7 mm ( $p < 0.001$ ), and cardiac output (CO) 6.75±1.04 L/min vs. 4.17±0.90 L/min ( $p < 0.001$ ). In the whole TA, the TAWSS in the old was lower than in the young, while OSI only showed small differences. Fig 1a and b show reduced TAWSS with age in a cross-section of TA, and to some extent also in OSI.

**Conclusions:** WSS show distinct age-related changes with a major reduction in TAWSS, while OSI show only minor changes. This is associated with an increase in aortic diameter as well as decrease in CO. The distinct WSS-alterations could be a contributing factor both to the remodeling and atherosclerosis process over age.



**Figure 1a** cross-section location.



**Figure 1b** TAWSS and OSI distribution between the young and old subjects at a cross-section of the descending aorta (±SEM). Significance is indicated by \* and \*\* ( $p < 0.05$  and  $p < 0.01$ , respectively).

### P3.20 ESTROGENS REGULATE ENDOTHELIAL NITRIC OXIDE PRODUCTION IN RATS ARTERIES DURING EARLY POSTNATAL PERIOD

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During maturation the vascular system undergoes structural and functional changes. Earlier we showed that endothelium of 1-2-week-old rats exerts an anticontractile effect due to tonic nitric oxide (NO) production which correlates with a higher eNOS expression level compared to adults. Estrogens are powerful regulators of eNOS expression and activity in arterial endothelium. This study tested the hypothesis that anticontractile effect of endothelium in young rats is associated with the influence of endogenous estrogens.

From the 2<sup>nd</sup> postnatal day male Wistar rats were daily treated with estrogen receptor blocker ICI182,780 (1 mg/kg/day, s.c.), age-matched control pups were treated with vehicle. On 10-12 postnatal days saphenous artery was isolated for measuring the levels of eNOS and aromatase mRNA expression (qRT-PCR) and contractions to methoxamine (alpha<sub>1</sub>-adrenoceptor agonist) before and after eNOS inhibition (wire myograph, DMT A/S, Denmark). Serum estradiol concentration (ELISA) in young rats was 20% higher than in adults. mRNA expression levels of eNOS and aromatase, the key enzyme of estradiol synthesis, in pup arteries were much higher than in adults. Importantly, chronic ICI182,780 treatment reduced the impact of endothelium on methoxamine-induced contraction: eNOS inhibitor L-NNA had small effect in "ICI" group, but strongly potentiated the contraction in control. mRNA expression level of eNOS in ICI-treated group tended to be lower compared to control.

Our results suggest that estrogens (both blood-delivered and locally produced in the arterial wall) are important determinants of endothelial secretion in postnatal rats. Effects of estrogens lead to tonic NO production which weakens arteries responses to contractile influences.

### P3.21 CONDUCTANCE AND CAPACITANCE EFFECTS OF ACUTE, ELECTRICAL, CAROTID BAROREFLEX STIMULATION

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**Introduction:** Chronic baroreflex activation is a therapy for resistant hypertension and has potential as a therapy in heart failure.

We hypothesized that acute baroreflex activation therapy (CVRx, Inc.) would increase both the capacity of the abdominal venous circulation (lowering "preload") and aortic conductance (reducing "afterload").

**Methods:** Six 20-kg mongrel dogs were anaesthetized and ventilated. Arterial blood pressure (BP) and diaphragmatic aortic and caval flow (ultrasonic) were measured. Venous capacity changes were evaluated using a modified Brooksby-Donald technique\*. A CVRx electrode was affixed to the right carotid sinus.

BP and flow data were collected under control conditions and during device activation and drug infusions. Angiotensin II (ANG II) was infused to raise BP to hypertensive levels; the current from the device was then increased.

**Results:** Device activation decreased mean aortic BP 22.5±1.3 mmHg, decreased heart rate 14.7±3.4% and cardiac output 10.8±3.9%, increased aortic conductance 16.2±4.9%, and increased abdominal blood volume at a rate of 2.2±0.6 mL/kg/min (5-minute activations). ANG II infusion increased BP 40.3±3.4 mmHg and reduced venous capacitance at a rate of 1.1±0.5 mL/kg/min. Subsequent electrical stimulation restored BP to baseline while aortic conductance only returned to 82.3±3.3% of control. Venous capacitance increased at a rate of 3.4±0.7 mL/kg/min, reversing the ANG II effects.

**Conclusions:** Acute electrical activation of the carotid baroreflex increases arterial conductance, decreases BP, and increases venous capacitance.