P1.15: CONDITIONAL INACTIVATION OF INTEGRIN AV SUBUNIT IN VASCULAR SMOOTH MUSCLE CELLS REGULATES FIBROSIS IN VESSELS

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P1.14 ANALYSIS OF LEFT VENTRICULAR FILLING DYNAMICS

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Diastolic filling of the left ventricle (LV) occurs in two phases, early and late filling. Early filling, manifest as the “E-wave”, is thought to be substantially due to diastolic suction (DS), a phenomenon where the LV aspirates blood and fills itself, independent of atrial activity. Late filling, resulting in the mitral flow “A-wave” is a result of left atrial contraction. Adequate filling of the LV is necessary to maintain normal heart function at rest and under stress. DS is thought to be an important mechanism in the efficiency of filling. To study DS, we have invasively measured pressure and used cardiac MRI to evaluate cavityary volume and flow in an animal model to quantify different measures of DS under varied experimental conditions.

The amount of filling due to DS (VDS), determined by the change in volume between mitral valve opening and LV pressure minimum of the pressure-volume loop (Katz 1930), is related to the measured end systolic volume (ESV). As ESV decreases the VDS increases. The smaller the ESV, the larger the released energy of the LV as it relaxes towards resting volume. This contributes increased energy for the suction of blood into the ventricle in early filling.

Wave intensity analysis (the separation of forward and backwards waves and wave type) and intraventricular pressure gradients will also be considered in order to determine which best describes DS and whether they can be used together to better understand changes in filling dynamics under varied loading conditions.

P1.15 CONDITIONAL INACTIVATION OF INTEGRIN AV SUBUNIT IN VASCULAR SMOOTH MUSCLE CELLS REGULATES FIBROSIS IN VESSELS

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Integrin avb3 is expressed at high density in vascular smooth muscle cells (VSMCs). It functions as a receptor for adhesion proteins in VSMCs with phenotypic modulation plays a pivotal role in arteriosclerosis and atherosclerosis. The aim was to study the role of integrin avb3 in angiotensin II (Ang II)-induced arterial fibrosis in mice and in human samples of atherosclerotic arteries in situ. Transgenic mice conditionally inactivated for integrin av subunit in VSMCs (avSMKO) were treated with Ang II (1.5 mg/kg/day) for 4 weeks. Immunohistochemical analysis of atherosclerotic plaques at different stages of development and primary cultures of human aortic VSMCs were used. At baseline, blood pressure was lower in avSMKO compared to control (WT) mice. Isobaric carotid distensibility was increased and remained higher in avSMKO in response to Ang II. The increase in collagen content in response to Ang II was lower in avSMKO than in WT (15 vs 36%) for similar increase in avSMKO in response to Ang II. The increase in collagen content in response to Ang II was lower in avSMKO than in WT (15 vs 36%) for similar increase in collagen content in response to Ang II. It functions as a receptor for adhesion proteins in VSMCs which phenotypic modulation plays a pivotal role in arteriosclerosis and atherosclerosis.

P1.17 THE VENTRICLE'S PROMINENT ROLE IN PRESSURE AMPLIFICATION; AN INCREMENTAL EXPERIMENTAL STUDY

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Despite central pressure’s predictive power of cardiovascular risk, brachial pressure is the clinical standard. However, amplified brachial systolic pressure varies significantly with age, and during therapy. Our aim was to modulate individual arterial and ventricular parameters in an experimental model of the cardiovascular system, to quantify each parameter’s contribution to arterial pressure and its amplification.

A piston driven ventricle provided computer-controlled flow waveforms into various silicone arterial trees. Silicone tubes diameters (20, 15, 10mm), wall thicknesses (0.5, 0.7, 1.0, 1.5mm), lengths (30-400cm), taper (20mm inlet to 20, 15, 10 and 5mm outlets), were each applied with various ventricular stroke profiles (sawtooth to sinewave). Intravascular pressure-tip wires and ultrasonic flow probes measured pressure and flow. MAP, Flow and HR were maintained between tests for comparison.

Ventricular stroke profile independently augmented pressure amplification from 16% to 82% between sinewave and sawtooth ejections profiles. As expected for any arterial model, the transfer function from central to distal pressure measurement sites remained constant. Decreasing taper, wall thickness, and length, and increasing diameter each increased amplification by shifting the peak of the amplifying transfer function towards the more prominent lower frequencies, (1-3Hz). However, the amplification variation between all vascular parameters was <30%. Despite the arterial tree dictating how the ventricular pulse will propagate, the ventricle provides the wave packet of frequencies with which it is amplified. These findings correlate well with observations of decreasing amplification with age as the native inotropy decreases, and increasing amplification associated with decreased LV mass during hypertensive drug therapy.

P2.1 AGING AND STRUCTURAL ALTERATIONS OF SUBCUTANEOUS SMALL RESISTANCE ARTERIES IN HYPERTENSIVE PATIENTS

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Background: It was proposed that early vascular aging may be an important mechanism of vascular damage in large conductance arteries. However it is not known whether aging may also affect small resistance artery morphology.

Patients and methods: For this reason, we investigated 100 patients with essential hypertension. Secondary forms of hypertension were excluded according to standard clinical evaluations and biochemical or instrumental assessments. In all patients, an evaluation of small resistance arteries morphology was performed by wire micromography. A small amount of subcutaneous tissue was obtained by local biopsy or during election surgery and histomorphometric assessment. In all patients, an evaluation of small resistance arteries morphology was performed by wire micromography. A small amount of subcutaneous tissue was obtained by local biopsy or during election surgery and histomorphometric assessment.

Conclusion: Our data suggest that age may affect microvascular structure in hypertensive patients. It is also possible that hypertension may anticipate the effects of physiological aging, and this should be explored in a relatively large population of normotensive subjects.

P2.2 RESISTANT HYPERTENSION AND STRUCTURAL ALTERATIONS OF SUBCUTANEOUS SMALL RESISTANCE ARTERIES

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