P1.8: DEVELOPMENT OF A TECHNIQUE FOR DETERMINATION OF PULMONARY ARTERY PULSE WAVE VELOCITY IN HORSES

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HEMODYNAMICS OF PULMONARY HYPERTENSION: APPLICATION OF THE RESERVOIR-WAVE APPROACH

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RESERVOIR-WAVE APPROACH

HEMODYNAMICS OF PULMONARY HYPERTENSION: APPLICATION OF THE

Use the reservoir-wave approach, previously we characterized pulmonary vascular mechanics with multiple interventions in a canine model. In the present study, we measured high-fidelity pulmonary arterial (PA) pressure, Doppler flow velocity, and pulmonary capillary wedge pressure in 11 patients referred for evaluation of exertional dyspnea. The analysis was performed using the reservoir-wave approach; wave intensity analysis was subsequently utilized to characterize the PA wave pattern. Our objective was to identify specific abnormalities associated with pulmonary hypertension. Seven patients with varying PA pressures had reduced pulmonary vascular conductance (i.e., the amount of flow that the lungs can accept per pressure gradient), suggesting that these patients might benefit from pulmonary vasodilator therapy, some even in the absence of markedly elevated PA pressure.

Right ventricular (RV) performance was assessed by examining the work done by the wave component of systolic PA pressure. Work done, the non-recoverable energy expended by the RV to eject blood, varied directly with mean PA pressure. Wave pressure was partitioned into two components: forward-travelling and reflected backward-travelling waves. Among patients with lower PA pressures, we found pressure-decreasing backward waves that aided the RV during ejection, as previously reported in normal experimental animals. Among patients with higher PA pressures, we detected pressure-increasing backward waves that impede RV ejection. We conclude that it is important to measure pulmonary vascular conductance to properly assess the pulmonary vasculature. The reservoir-wave approach and wave intensity analysis may prove to be valuable tools to evaluate RV performance and may facilitate development of therapeutic strategies.

AGE AND HYPERTENSION STRONGLY REDUCE AORTIC VISCO-ELASTIC PROPERTIES IN RATS AT BASAL AND MATCHED BLOOD PRESSURE LEVELS

George Lindesay 1, Christophe Ragouen, Stefano Chimenti,

AGE AND HYPERTENSION strongly reduce aortic visco-elastic properties in rats at basal and matched blood pressure levels.

Age and hypertension are major causes of large artery stiffening, a cardiovascular risk factor for heart and kidney damage. Long term hypertension induces vascular remodeling, accelerating vascular aging. The aged Spontaneously Hypertensive Rat (SHR) model is recognized for human cardiovascular pathology but discrepancies are apparent in studies of arterial stiffness. We performed experiments using a robust aortic visco-elasticity analysis via echoechocardiog

PLATELET-LOCALIZED FXI PROMOTES A GLYCOPROTEIN Iβs DEPENDENT FEEDBACK LOOP IN ARTERIAL HYPERTENSION AND VASCULAR INFLAMMATION

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Background: Interactions of platelets, leukocytes and the vessel wall play pivotal roles in activating coagulation and precipitating thrombosis. High levels of angiotensin II (ATII) cause arterial hypertension by a complex inflammatory pathway requiring leukocyte recruitment and reactive oxygen species production within the vessel wall.

Objective: The aim of this work was to explore the role of platelet glycoprotein Iβs dependent thrombin-FXII feedback loop in arterial hypertension.

Methods: FXI-II-, FXI-/-, and hIL-4R/1βs mice and 5/6 nephrectomized rats were used for this study. Mice where treated with ATII (1mg/kg/d-1 for 7