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P3.01: PHENOTYPIC MODULATION OF VASCULAR SMOOTH MUSCLE CELLS IN RESPONSE TO HYPERTENSION CONFERS A PROTHROMBOTIC STATE WITHIN THE VESSEL WALL

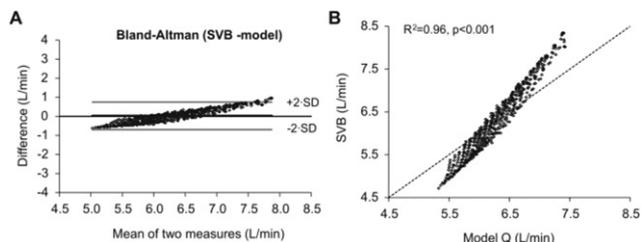
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waves), was 0.042 L/min with 0.341 L/min SD of difference. The limits of agreement were -0.7–0.6 L/min indicating high precision. The intraclass correlation coefficient and the root mean square error between estimated and "real" CO were 0.861 and 0.041 L/min, respectively, indicating very good accuracy.



P2.47 ANALYSIS OF THE RELATIONSHIP BETWEEN THE RADIAL PULSE AND PHOTOPLETHYSMOGRAPHY BASED ON THE SPRING CONSTANT METHOD

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The spring constant method is a newly proposed method to evaluate the arterial stiffness. However, whether the spring constants computed using the radial pulse and photoplethysmography (PPG) show a similar characteristic deserves to be investigated. The experimental group comprised 40 participants (18 men and 22 women), all with diabetes mellitus and ranging between 48 and 75 years of age. All were subjected to the measurements of the radial pulse and PPG pulse. Parameters, the amplitude, the rising slope, the second derivative of the peak and the spring constant of the two types of pulses, were used for analysis. Statistical results showed that only the spring constant parameter revealed the significant relationship (correlation coefficient = 0.78, $p < 0.001$) between the radial pulse and PPG pulse. In other words, regarding to the assessment of arterial stiffness, the radial pulse and PPG pulse showed a similar characteristic. The finding provides more alternatives to evaluate the arterial stiffness in clinic.

P3 – Basic Science

P3.01 PHENOTYPIC MODULATION OF VASCULAR SMOOTH MUSCLE CELLS IN RESPONSE TO HYPERTENSION CONFERS A PROTHROMBOTIC STATE WITHIN THE VESSEL WALL

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The hypothesis that hypertension may confer a hypercoagulable state arises from the main complications associated with hypertension, stroke and myocardial infarction. Our objective was to determine whether spontaneous hypertension confers changes in the coagulation proteins and the thrombin generating capacity in blood and the vascular wall.

We used the model of spontaneously hypertensive rats (SHR) compared with Wistar rats. Thrombin generation was lower in platelet-rich plasma and platelet-free plasma from SHR compared to Wistar. This was related to lower tissue factor (TF) and prothrombin as well as higher TFPI levels in SHR plasma. In contrast, the addition of thoracic aorta rings of SHR to a Wistar plasma pool resulted in a higher increase in thrombin generation compared to the addition of equivalent rings from Wistar. Whereas no difference was observed for endothelial cells, thrombin formation was higher at the surface of cultured SHR aortic SMCs than from Wistar. Exposure of negatively-charged phospholipids was higher on SHR than on Wistar rings as well as on SMCs. TF and TFPI activities were higher in SHR SMCs. These results show opposite thrombin generating capacity of plasma and vessel walls in SHR compared to Wistar. The higher prothrombotic phenotype of the SHR vessel wall was due to the ability of SMCs to support thrombin generation. These findings suggest that the hypertension-induced membrane phospholipid reorganization and synthesis of procoagulant molecules in SMCs provide substrates for increased thrombin formation within the vessel wall.

P3.02 DESCRIBING WAVES IN THE PULMONARY VEINS: APPLICATION OF A RESERVOIR-WAVE MODEL

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Background: The pulmonary venous pressure waveform is typically described by the downstream events in the left atrium and ventricle. These downstream events create waves that contribute to the overall waveform.

Methods: In anesthetised open-chest dogs, measurements of pressure and flow were made in the pulmonary artery and vein. Experiments involved increases to blood volume and the application of 10 cm H₂O positive end-expiratory pressure (PEEP). The reservoir-wave model describes the reservoir pressure, which is subtracted from measured pressure, to result in the excess pressure (P_{excess}). Excess velocity (U_{excess}) is similarly formulated. P_{excess} and U_{excess} are used in wave intensity analysis to calculate wave speed and separate the contributions of waves originating upstream (forward waves) and downstream (backward waves).

Results: Separated waves are shown in the bottom panel of Figure 1. The effect of PEEP resulted in larger decreases to P_{backward} ($p < 0.001$) after the mitral valve opened. As a result, y was lower than x by ~ 2.0 mmHg. With PEEP, the delay between arterial and venous forward waves increased from 155 ± 4 ms to 183 ± 4 ms (mean \pm SE, $p < 0.001$).

Conclusion: The majority of pulmonary venous pressure landmarks can be attributed to the actions of the left atrium and ventricle but the v wave has substantial contributions from waves originating in the pulmonary artery. Diastolic suction has a larger effect with PEEP, presumably from some external constraint applied to the heart and consequently lowered end-systolic left ventricular volume.

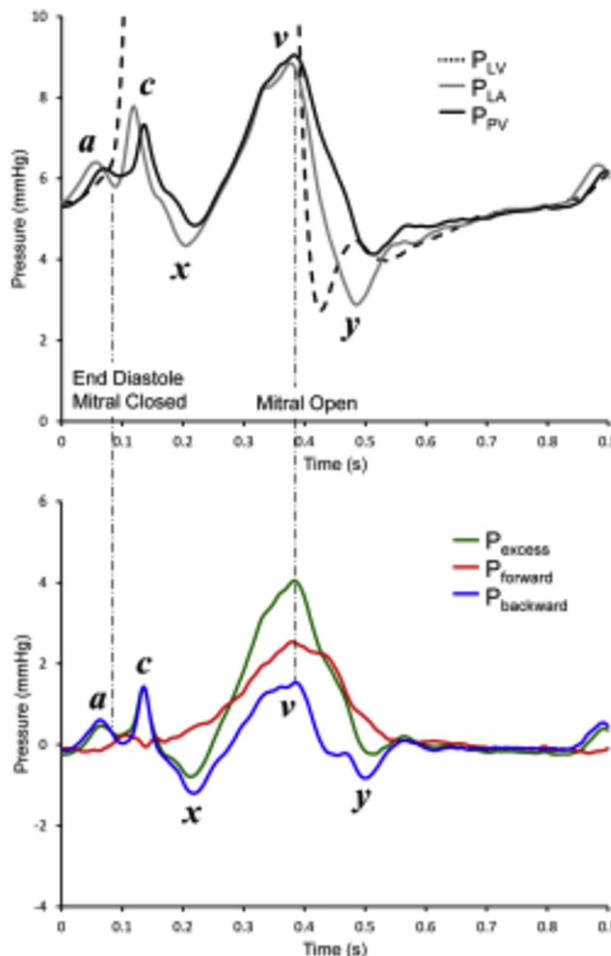


Figure 1 Common venous markers related to measured pressures (top panel) and the separation of P_{excess} into forward and backward components (bottom panel) at control conditions.