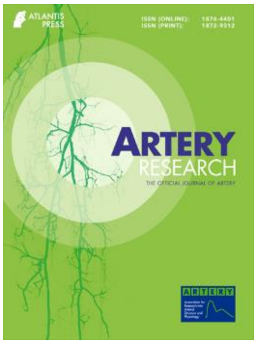

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P2.46: THE “SYSTOLIC VOLUME BALANCE” METHOD FOR THE NON-INVASIVE ESTIMATION OF CARDIAC OUTPUT BASED ON PRESSURE WAVE ANALYSIS

O. Vardoulis, T.G. Papaioannou, N. Stergiopulos

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CAVI is decreased with alfa1 selective blocker, doxazosin. Therefore, CAVI is supposed to be composed of organic stiffness and also of contracture of smooth muscle cells. Moreover, the relationships between CAVI and cardiac functions were reported.
Thus, CAVI may be not only a surrogate maker of arteriosclerosis and vascular age, but, also an indicator of contracture of smooth muscle cells. CAVI might develop a new field of vascular function.

P2.43
DEVELOPMENT OF A NEW MODEL FOR CALCULATING VENOUS COMPLIANCE IN THE LIMBS OF HUMANS

J. Skoog [1], M. Ekman [1], H. Zachrisson [1], M. Lindenberger [1,2], L. Ewerman [1], T. Länne [1]
[1]*Department of Medicine and Health Sciences, Division of Cardiovascular medicine, Linköping University, Sweden*
[2]*Division of Cardiology, Jönköping, Sweden*

Venous compliance (Vc) reflects the elastic properties of the venous wall and altered Vc affects venous return as well as hemodynamic stability. Venous occlusion plethysmography (VOP) is used to measure Vc in the limbs. However, capillary filtration from blood to tissue could be a potential confounder. We conducted a series of studies to validate VOP in lower limb. A method was developed to identify fluid filtration and to evaluate whether this is a significant confounder in the study of Vc.
Strain-gauge technique was used to study calf volume changes in 15 healthy females (22.9±3.2 years). A thigh cuff was inflated to 60 mmHg for 8 min with a subsequent linear decrease of 1 mmHg/s in cuff pressure (P). Intravenous pressure (IVP) was simultaneously measured in a foot vein. Vc was determined using the first derivate of a quadratic regression equation describing the volume-pressure relationship [Compliance $=\beta 1+2\beta 2(P)$]. The capillary filtration was subtracted from the volume curve to correct for the potential effect on Vc.
The increase in IVP showed 100% transmission and steady state was reached within 3-4 min. The following decrease of 1 mmHg/s in cuff pressure correlated well with IVP reduction ($r=0.99$, $P<0.001$). The volume increase during VOP was augmented further by approximately 60% due to capillary filtration. Without correction of capillary filtration Vc was underestimated with the most marked differences $\geq$30 mmHg ($P<0.01$).
Capillary filtration is a confounder in the study of limb Vc. The new model seems to be a valuable tool in the future studies of venous wall function as well as hemodynamic regulation.

P2.44
A COMPARISON OF THE CAROTID AND POPLITEAL ARTERIES IN YOUNG AND OLDER CAUCASIAN MEN

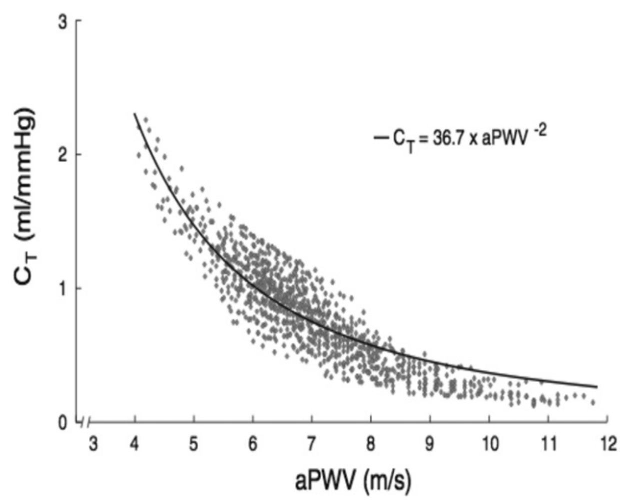
J. M. van Rooyen [1,2], L. J. Kotzee [2], C. M. C. Mels [2], R. Kruger [1,2], A. Burger [2]
[1]*North-West University, Potchefstroom, South Africa*
[2]*HART, Potchefstroom, South Africa*

According to Debasso *et al.*, the popliteal artery depicts a muscular artery with wall properties similar to that of the central conduit arteries. Some literature show differences in arterial composition between popliteal arteries and other fellow peripheral muscular arteries and may comprise both central and peripheral properties. The objective was to investigate whether the popliteal artery resemble the carotid artery in structure and function in young and older Caucasian men. Forty one participants were divided in a young-, aged 20-30 years ($n=21$) and an older group aged 40-60 years ($n=20$). Cardiovascular and anthropometric measurements were executed which included blood pressure, carotid femoral PWV (Complior SP Acquisition system) as well as popliteal and carotid IMT (Vivid E9, GE). An inverse association ($r=-0.78$; $p<0.001$) between popliteal IMT and c-fPWV in young men were encountered after adjusted for age, BMI and smoking. Carotid IMT and popliteal IMT differed significantly ($p<0.001$) in both age groups. The mean CSWA of carotid and popliteal arteries also differed significantly when young and older men were compared. In the young men the mean CSWA of the carotid artery differed significantly from the popliteal CSWA (1.83 cm vs. 1.60 cm; $p=0.013$) but not in the older men. To conclude the popliteal and carotid arteries in young and older Caucasian men do not exhibit similar structural or functional properties.

P2.45
ESTIMATING TOTAL ARTERIAL COMPLIANCE FROM AORTIC PULSE WAVE VELOCITY

O. Vardoulis, T. G. Papaioannou, N. Stergiopulos
École Polytechnique Fédérale de Lausanne (EPFL), Lausanne, Switzerland

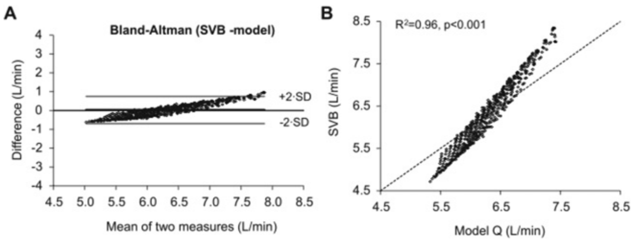
Total arterial compliance (CT) is a main determinant of cardiac afterload, left ventricular function and arterio-ventricular coupling. *CT* is physiologically more relevant than regional aortic stiffness. However, direct, *in vivo*, non-invasive, measurement of *CT* is not feasible. Several methods for indirect *CT* estimation require simultaneous recording of aortic flow and pressure waves, limiting *CT* assessment in clinical practice. In contrast, aortic pulse wave velocity (aPWV) measurement, which is considered as the "gold standard" method to assess arterial stiffness, is noninvasive and relatively easy. Our aim was to establish the relation between aPWV and *CT*. Totally, 1000 different hemodynamic cases were simulated, by altering heart rate, compliance, resistance and geometry using an accurate, distributed, nonlinear, one- dimensional model of the arterial tree. Based on Bramwell-Hill theory, the formula $C_T=k\cdot aPWV^{-2}$ was found to accurately estimate *CT* from aPWV. Coefficient *k* was determined both analytically and by fitting *CT* vs aPWV data (Fig. 1). *CT* estimation may provide an additional tool for cardiovascular risk (CV) assessment and better management of CV diseases. *CT* could have greater impact in assessing elderly population or subjects with elevated arterial stiffness, where aPWV seem to have limited prognostic value. Further clinical studies should be performed to validate the formula *in vivo*.



P2.46
THE "SYSTOLIC VOLUME BALANCE" METHOD FOR THE NON-INVASIVE ESTIMATION OF CARDIAC OUTPUT BASED ON PRESSURE WAVE ANALYSIS

O. Vardoulis, T. G. Papaioannou, N. Stergiopulos
École Polytechnique Fédérale de Lausanne, Lausanne, Switzerland

Cardiac output (CO) monitoring is essential for the optimal management of critically ill patients. Several methods have been proposed for CO estimation based on pressure waveform analysis. Most of them depend on invasive blood pressure recording and calibrations, while they suffer from decreased accuracy under specific conditions. A new Systolic Volume Balance (SVB) method was derived from a volume balance approach on the conservation of mass ejected into and flowed out of the arterial system during systole. The formula was validated by a one-dimensional model of the systemic arterial tree. Comparisons of CO estimates between the proposed and previous methods were performed in terms of agreement and accuracy by using the "real" CO values of the model as a reference. 507 different hemodynamic cases were simulated by altering cardiac period (T), arterial compliance (C) and resistance. CO could be accurately estimated by the SVB method: $CO=C\cdot PP_{ao}/(T\text{-}P_{sm}\cdot T_s/P_m)$ where PP_{ao} aortic pulse pressure, T_s systolic duration, P_{sm} mean systolic pressure and P_m mean pressure. SVB applied on aortic pressure waves did not require calibration or empirical correction for CO estimation. An empirical coefficient (k) was necessary for brachial pressure wave analysis. The difference of SVB-derived from model CO, (for brachial 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

C. C. Wei, W. C. Tsai
Chaoyang University of Technology, Taichung, Taiwan, ROC

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

K. Ait Aissa [1,2], J. Lagrange [1,2], J. P. Max [1,2], P. Challande [3], P. Lacolley [1,2], V. Regnault [1,2]
[1]*Inserm U961, Nancy, France*
[2]*Université de Lorraine, Nancy, France*
[3]*Université Paris 6, Paris, France*

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

J. C. Bouwmeester, N. G. Shrive, J. V. Tyberg
University of Calgary, Calgary, Canada

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 H_2O 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 ± 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.

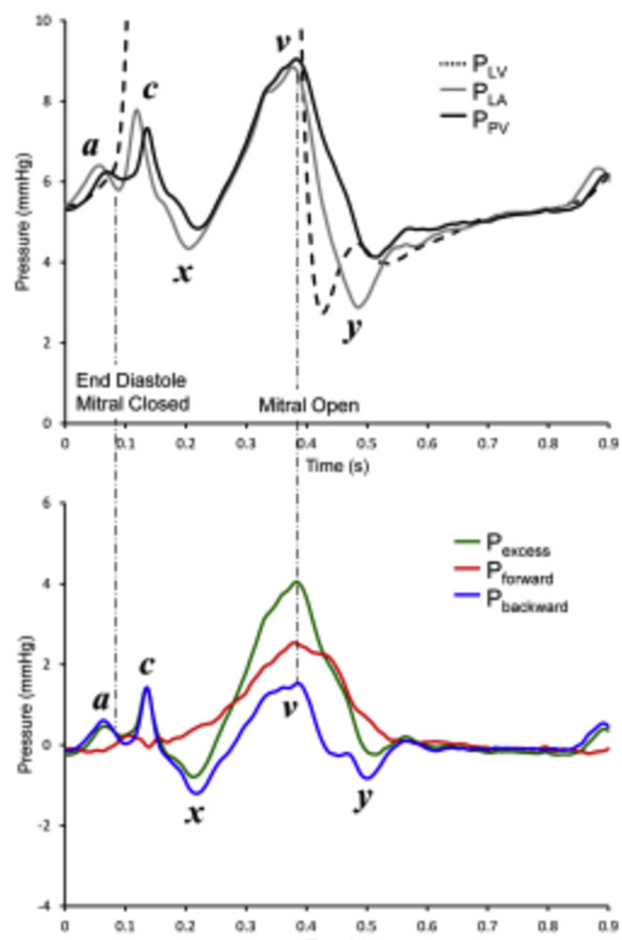


Figure 1 Common venous markers related to measured pressures (top panel) and the separation of P_{excess} into forward and backward components (bottom panel) at control conditions.