



Artery Research

ISSN (Online): 1876-4401

ISSN (Print): 1872-9312

Journal Home Page: <https://www.atlantis-press.com/journals/artres>

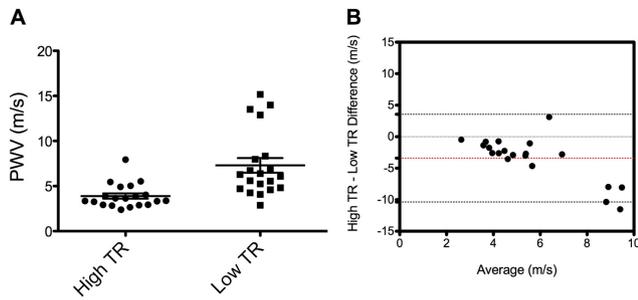
P6.15: HAEMODYNAMIC-MECHANICAL INTERACTION IN TYPE-B AORTIC DISSECTION: EXPERIMENTAL AND COMPUTER MODEL STUDY

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To cite this article: P.A. Rudenick, B.H. Bijmens, P. Segers, D. García-Dorado, A. Evangelista (2013) P6.15: HAEMODYNAMIC-MECHANICAL INTERACTION IN TYPE-B AORTIC DISSECTION: EXPERIMENTAL AND COMPUTER MODEL STUDY, Artery Research 7:3_4, 158–158, DOI: <https://doi.org/10.1016/j.artres.2013.10.196>

To link to this article: <https://doi.org/10.1016/j.artres.2013.10.196>

Published online: 14 December 2019



P6.15 HAEMODYNAMIC-MECHANICAL INTERACTION IN TYPE-B AORTIC DISSECTION: EXPERIMENTAL AND COMPUTER MODEL STUDY

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Background: Type-B aortic dissections (TBADs) show a high morbidity and mortality in the long-term follow up. The role of and interaction between haemodynamics (pressure and flows), mechanics (wall compliance) and morphology (communication between false lumen (FL) and true lumen (TL)) is poorly understood, as well as the potential role of these variables in the outcome of TBADs. **Materials and methods:** We developed both a hydraulic bench (experimental) and an electrical analogue model of a TBAD (Fig. 1). The model was calibrated and validated for eight experimental cases consisting of various permutations of proximal and distal tear sizes (no tear, 4 and 10 mm). **Results:** The computer model was able to reproduce measured intraluminal pressures and velocities across tears with good agreement for all combinations (Fig. 2). Tear size is the major determinant of interaction between FL and TL. Tear sizes of 4 mm are associated with a reduced pressure in the FL, and strong oscillatory flow exchange between TL and FL. A tear size of 10 mm increases FL pressure to the same level or beyond TL pressures, and reduces the oscillatory nature of the flow. **Conclusions:** Both the experimental and computer model reveal a complex dynamic interaction between TL and FL, modulated by the size and combination of the tears.

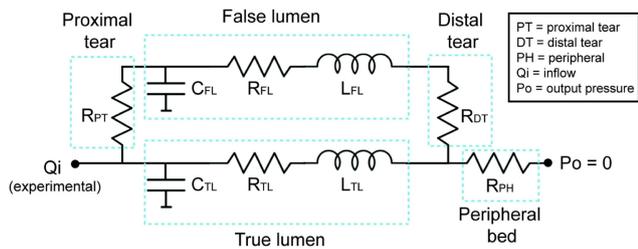


Figure 1 Electrical analogue model of a type-B aortic dissection

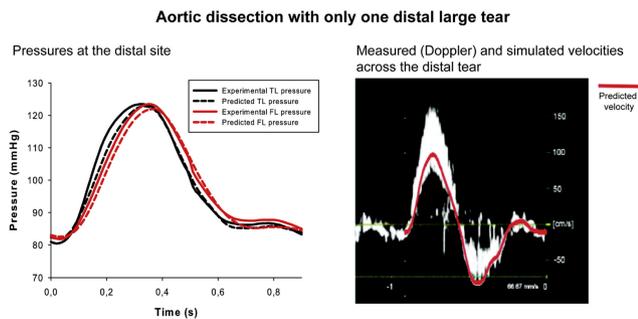


Figure 2 Comparison between predicted and experimental intraluminal pressures and velocities across tear

P6.16 PRONOUNCED INCREASE IN LONGITUDINAL DISPLACEMENT OF THE PORCINE CAROTID ARTERY WALL CAN TAKE PLACE INDEPENDENTLY OF WALL SHEAR RATE

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Objective and methods: We have recently shown that the longitudinal displacements of the arterial wall, i.e. the displacements *along* the artery during the cardiac cycle, undergo profound changes in response to catecholamines. The relations between longitudinal displacements of the arterial wall and wall shear rate (WSR) and wall shear stress (WSS), respectively, from the flowing blood are unexplored. To study this issue the common carotid artery of five anesthetized pigs was noninvasively investigated during different hemodynamic situations using ultrasound. The study protocol included intravenous infusion of epinephrine, as well as intravenous boluses of norepinephrine. Further, effects of β -blockade (metoprolol) were studied. **Results:** During infusion of epinephrine and following boluses of norepinephrine WSR increased. However, when catecholamines were administered during β -blockade, β -blockade effectively counteracted increase in WSR. We found no correlation between longitudinal displacement of the intima-media complex and WSR. This was most obvious when boluses of norepinephrine were administered during β -blockade; β -blockade effectively counteracted increase in WSR, whereas, at the same time, β -blockade was insufficient to counteract a sharp rise in pulse pressure and accompanying pronounced increase in longitudinal displacement of the intima-media complex. **Conclusion:** This study shows that a profound increase in longitudinal displacement of the arterial wall can take place independently of WSR, and thus, using the models of today, independently of WSS from the flowing blood. This strongly suggests that another force, or other forces, than WSS from the blood flow are also working in the direction of the blood flow; i.e. along the arteries.

P6.17 PATTERN BETWEEN ARTERIAL STIFFNESS, BLOOD PRESSURE AND AGE OBSERVED IN INDIVIDUAL PATIENTS AND REFERENCE POPULATION EXPLAINED BY BIOMECHANICS OF WALL REMODELLING

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Background: We recently found in patients that the pressure dependency of arterial stiffness is age-dependent, explaining clinically observed changes in stiffness with blood pressure (BP) lowering. Pulse wave velocity, BP and age found in the Reference Values for Arterial Stiffness Collaboration show a corresponding pattern. We hypothesised that the impact of age on the pressure-dependency of arterial stiffness actually reflects changes in wall constituent properties and adaptation to mechanical stress **Methods and results:** We applied a constitutive model of arterial wall biomechanics (Zulliger, AJP-Heart 2004) to simulate ageing, based on detailed pressure-area curves obtained in our younger (mean age 40 yrs, n=6) and older (65 yrs, n=7) hypertensive patients. Ageing was simulated by adapting the model (initially fitted to the "young" pressure-area curve) to fit the "old" curve (Figure); allowing for elastin degradation, collagen rearrangement and wall thickness adaptation, but not for changes in fractional volumes. The simulation showed a physiologically significant transfer of pressure-load bearing from elastin to collagen, caused by a 40% loss in elastin stiffness and a 5-fold increase in collagen recruitment (Table). Presuming normalisation and homogeneous distribution of wall stress with ageing, the predicted increase in wall thickness was in accordance with intima-media thickness Reference Values (2010 data; 25-yr increment, corresponding risk category).

