



Artery Research

ISSN (Online): 1876-4401

ISSN (Print): 1872-9312

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

2.6: FEASIBILITY OF AORTIC WAVE INTENSITY ANALYSIS FROM SEQUENTIALLY ACQUIRED CARDIAC MRI AND NON-INVASIVE CENTRAL BLOOD PRESSURE

Anish Bhuva, Niro Nadarajan, Andrew D'Silva, Camilla Torlasco, Redha Boubertakh, Siana Jones, Paul Scully, Rachel Bastiaenen, Guy Lloyd, Sanjay Sharma, James Moon, Kim Parker, Charlotte Manisty, Alun Hughes

To cite this article: Anish Bhuva, Niro Nadarajan, Andrew D'Silva, Camilla Torlasco, Redha Boubertakh, Siana Jones, Paul Scully, Rachel Bastiaenen, Guy Lloyd, Sanjay Sharma, James Moon, Kim Parker, Charlotte Manisty, Alun Hughes (2018) 2.6: FEASIBILITY OF AORTIC WAVE INTENSITY ANALYSIS FROM SEQUENTIALLY ACQUIRED CARDIAC MRI AND NON-INVASIVE CENTRAL BLOOD PRESSURE, Artery Research 24:C, 71–71, DOI: <https://doi.org/10.1016/j.artres.2018.10.029>

To link to this article: <https://doi.org/10.1016/j.artres.2018.10.029>

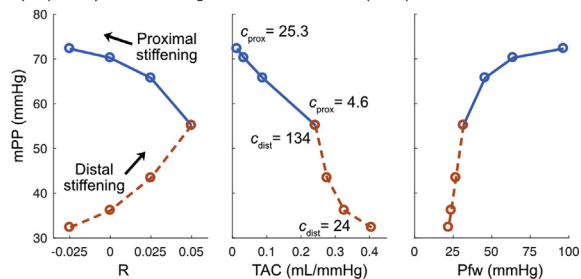
Published online: 7 December 2019

keeping average wave speed in all terminal vessels constant ("proximal-stiffening", see Figure). An elastance heart model was applied at the inlet and simulations were performed with a one-dimensional flow solver (2).

Results: Proximal-stiffening and distal-stiffening had opposing effects on R but the same effects on mPP, whereas mPP increased monotonically with decreasing TAC and increasing Pfw in both settings (Figure).

Conclusion: Wave reflection per se does not provide protection from high mPP since greater reflection also entails greater transmitted pressure. Although a decreased R may accompany proximal arterial stiffening, the likely mechanism of increased mPP with aging is decreased TAC and greater Pfw.

Figure. Reflection coefficient, R (at every junction), TAC and Pfw vs mPP with proximal and distal stiffening. Arrows indicate effect of stiffening. c_{prox} and c_{dist} are wave speeds (m/s) in the proximal inlet segment and distal terminals (mean).



References

- Mitchell GF. Effects of central arterial aging on the structure and function of the peripheral vasculature: implications for end-organ damage. *J Appl Physiol.* 2008;105(5):1652-60.
- Mynard JP, Smolich JJ. One-Dimensional Haemodynamic Modeling and Wave Dynamics in the Entire Adult Circulation. *Ann Biomed Eng.* 2015;43(6):1443-60.

2.6

FEASIBILITY OF AORTIC WAVE INTENSITY ANALYSIS FROM SEQUENTIALLY ACQUIRED CARDIAC MRI AND NON-INVASIVE CENTRAL BLOOD PRESSURE

Anish Bhuvu^{1,2}, Niro Nadarajan³, Andrew D'Silva⁴, Camilla Torlasco⁵, Redha Boubertakh², Siana Jones³, Paul Scully^{3,2}, Rachel Bastiaenen⁴, Guy Lloyd², Sanjay Sharma⁴, James Moon^{3,2}, Kim Parker⁶, Charlotte Manisty^{3,2}, Alun Hughes^{3,7}

¹University College London, UK

²Barts Heart Centre, London, UK

³Institute of Cardiovascular Science, University College London, UK

⁴Cardiovascular Sciences Research Centre, St. George's, University of London, London, United Kingdom

⁵IRCCS, Istituto Auxologico Italiano, Milan, Italy

⁶Department of Bioengineering, Imperial College London, UK

⁷MRC Unit for Lifelong Health and Ageing at UCL, London, UK

Background: Wave intensity analysis (WIA) in the aorta offers important clinical and mechanistic insights but is difficult non-invasively. We performed WIA by combining high temporal resolution cardiovascular magnetic resonance (CMR) flow velocity and non-invasive central blood pressure (BP) waveform data.

Method: 206 healthy volunteers (36 ± 11 years, 47% male) underwent sequential phase contrast CMR (Siemens Aera 1.5T, $1.97 \times 1.77 \text{ mm}^2$, $\sim 9 \text{ ms}$ temporal resolution) and supra-systolic oscillometric central BP (Uscom Ltd BP+) measurement. Velocity (U) and central pressure (P) waveforms (200 Hz) were aligned using the wave foot, and local wave speed was calculated both from the P-U slope during early systole (c) and the sum of squares method (cSS) (Figure 1), and compared with CMR aortic arch pulse wave velocity (PWV) by transit time.

Results: The peak intensity of the initial compression wave (di+1), backward compression wave (di-) and protodiastolic decompression wave (di+2) were 69.5 ± 28 , -6.6 ± 4.2 and $6.2 \pm 2.5 \text{ W/m}^2$ respectively. PWV correlated with c or cSS ($r = 0.60$, and 0.68 respectively; bias -1.3 [limits of agreement: -3.8 to 1.2 m/s], and bias -0.64 [limits of agreement: -3.0 to 1.7 m/s] respectively), Figure 1.

Conclusion: Wave intensity patterns and values are similar to those measured using invasive methods. Local wave speed showed good agreement with PWV. CMR and central blood pressure provides a novel non-invasive technique for performing wave intensity analysis and is feasible for large scale studies.

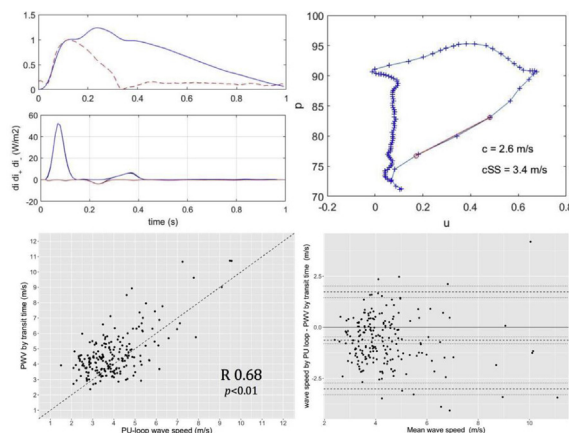


Figure 1. Calculation of wave speed from a pressure – velocity (P-U) loop and comparison with pulse wave velocity by transit time. Top left: alignment of scaled pressure (blue) and velocity (red) waveforms and example of wave intensity analysis showing initial compression (di+1), backward compression (di-) and protodiastolic decompression (di+2) waves. Top right: P-U loop showing wave speed measurement in early systole (c); and using sum of squares (cSS). Bottom left and Bottom right: Correlation and Bland-Altman analysis of cSS and PWV from phase-contrast MRI showing good correlation and slight underestimation.

2.7

FITNESS MODIFIES THE ASSOCIATION BETWEEN EXERCISE BLOOD PRESSURE AND LEFT-VENTRICULAR MASS IN ADOLESCENCE

Zhengzheng Huang¹, Ricardo Fonseca², James Sharman², Nish Chaturvedi³, George Smith⁴, Deborah Lawlor⁴, Laura Howe⁴, Chloe Park³, Alun Hughes³, Martin Schultz², Martin Schultz²

¹Menzies Institute for Medical Research, Hobart, Australia

²Menzies Institute for Medical Research, University of Tasmania, Hobart, Australia

³Institute of Cardiovascular Science, University College London, London, UK

⁴MRC Integrative Epidemiology Unit, University of Bristol, Bristol, UK

Objective: Exaggerated exercise blood pressure (BP) is associated with higher left-ventricular mass index (LVMI). Paradoxically, exercise BP and LVMI may be higher with greater fitness, but underlying factors are poorly understood. This study aimed to determine the influence of fitness on exercise BP and its relationship with LVMI in adolescents.

Methods: 4835 adolescents from the Avon Longitudinal Study of Parents and Children, aged 15.4(0.3) years, 49% male completed a submaximal cycle test. Exercise BP was measured immediately on test cessation and fitness calculated as physical work capacity 170 adjusted for lean body-mass. LVMI ($n = 1589$), cardiac output (CO, $n = 1628$) and total peripheral resistance (TPR, $n = 1628$) were measured by echocardiography 2.4 (0.4) years later.

Results: Each unit of fitness was associated with a 6.46 mmHg increase (95% CI: 5.83, 7.09) in exercise systolic BP. Exercise systolic BP increased step-wise by third of fitness (difference 6.06 mmHg, 95% CI: 4.99, 7.13 first vs. middle; 11.13 mmHg, 10.05, 12.20 middle vs. highest). Each 5 mmHg increase in exercise systolic BP was associated with 0.25 g/m^2 . 7 (0.16–0.35) greater LVMI, attenuated with adjustment for fitness. There was evidence of an interaction between fitness and exercise BP on LVMI, more-marked in the middle fitness third (difference -0.27 g/m^2 , $-0.51, 0.04$ vs. first third), but similar in lowest and highest fitness thirds. CO increased (difference 0.06 L/min, $-0.05, 0.17$; 0.23 L/min, 0.12, 0.34), TPR decreased (difference -0.13 AU , $-0.84, 0.59$; -1.08 AU , $-0.1, 0.35$ with fitness).

Conclusion: Fitness may modify associations between exercise BP and LVMI in adolescence. Higher CO, but lower TPR suggests a physiological exercise BP-LVMI relationship with higher fitness, rather than pathological elevations in exercise BP and LVMI.

2.8

RELATIONSHIPS BETWEEN ADIPOSITY AND LEFT VENTRICULAR FUNCTION IN ADOLESCENTS: MEDIATION BY BLOOD PRESSURE AND OTHER CARDIOVASCULAR MEASURES

Hannah Taylor¹, Alun D. Hughes^{2,3}, Abigail Fraser⁴, Laura Howe⁴, George Davey Smith⁴, Debbie Lawlor⁴, Nishi Chaturvedi^{3,2}, Chloe Park²

¹University College London, London, UK

²Department of Population Science & Experimental Medicine, Institute of Cardiovascular Science, University College London, UK