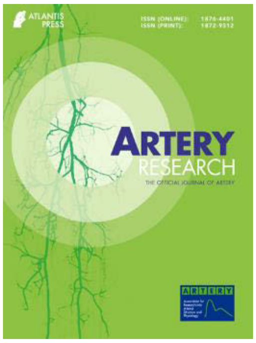

Artery Research

ISSN (Online): 1876-4401 ISSN (Print): 1872-9312
Journal Home Page: https://www.atlantis-press.com/journals/artres

4.4: CAN LASER DOPPLER VIBROMETER DETECT CAROTID STENOSIS FROM SKIN VIBRATIONS? HYDRAULIC BENCH TESTS ON PATIENT-SPECIFIC MODEL

Viviana Mancini, Daniela Tommasin, Yanlu Li, Roel Baets, Stephen Greenwald, Patrick Segers

To cite this article: Viviana Mancini, Daniela Tommasin, Yanlu Li, Roel Baets, Stephen Greenwald, Patrick Segers (2018) 4.4: CAN LASER DOPPLER VIBROMETER DETECT CAROTID STENOSIS FROM SKIN VIBRATIONS? HYDRAULIC BENCH TESTS ON PATIENT-SPECIFIC MODEL, Artery Research 24:C, 76–76, DOI: https://doi.org/10.1016/j.artres.2018.10.043

To link to this article: https://doi.org/10.1016/j.artres.2018.10.043

Published online: 7 December 2019

arterial stiffness = hfPWV 2 and faPWV 2 , respectively; and estimated wall/lumen ratio (W/L) = PWV 2/(central pulse pressure/stroke volume).
Results: In 76 individuals (mean age 55 years, weight 84 kg, BP 138/79 mmHg, resting HR 67; 45% female), WK stiffness was negatively correlated with age($p<0.05$) but not with BP, $hfPWV^2$ or $faPWV^2$. In contrast, $hfPWV^2$and $faPWV^2$ were positively correlated with age ($p<0.0001$ and $p<0.01$, respectively) but neither was correlated with tau or WK stiffness. Using 6 multilinear stepwise backward regression models for WK stiffness, the major contributing factors were: SVR ($p<10^{-6}$), t0 ($p<10^{-6}$), heart rate ($p<10^{-5}$), and W/L ($p=0.01$).
Conclusion: We identified SVR, heart rate, timing of pressure decay, and vessel geometry as correlates of WK stiffness but the lack of relationship between PWV-based arterial stiffness and stiffness derived from the WK model mitigates against a single arterial WK.

4.4

CAN LASER DOPPLER VIBROMETER DETECT CAROTID STENOSIS FROM SKIN VIBRATIONS? HYDRAULIC BENCH TESTS ON PATIENT-SPECIFIC MODEL

Viviana Mancini [1], Daniela Tommasin [1], Yanlu Li [2], Roel Baets [2], Stephen Greenwald [3], Patrick Segers [1]
[1]*Ghent University, bioMMeda, Ghent, Belgium*
[2]*Ghent University, Department of Information Technology, Ghent, Belgium*
[3]*Queen Mary University of London, London, United Kingdom*

Background: Within the H2020 CARDIS project, we explore the use of a Laser Doppler Vibrometer (LDV) [1] to detect asymptomatic carotid stenosis from measurement of skin vibrations on the neck of affected patients. We hypothesise that flow instabilities induced by the stenosis will propagate as mechanical waves through soft tissues of the neck. We here report measurements on an experimental model to assess the ability of LDV to detect stenosis-induced vibrations.
Methods: A compliant carotid bifurcation with Internal Carotid Artery (ICA) 76% area-stenosis model was surrounded by hydrogel and a skin-like layer to mimic neck's skin and soft tissues. Measurements were acquired (20 KHz) at physiological flows (water) through the artery [2, 3], at several distances downstream from the stenosis. Intra-arterial pressure measurements were performed at the same location for reference (Fig. 1A). To assess in which frequency range the Fast Fourier Transform spectra of the signals are most sensitive to changes in flow rate, we constructed a univariate linear model in SPSS for the integral of the normalized spectra (8K, Hann, 50%-overlap, LabChart), where inflow was used as covariate and the frequency range as fixed factor.
Results: The spectrograms (Fig1B) showed that the LDV was able to detect flow-induced instabilities in the 0—500 Hz range. The sensitivity was highest between 50—150 Hz for both LDV and pressure.
Conclusion: The LDV was able to detect stenosis-related flow features with a sensitivity comparable to the intra-arterial manometer, proving the potential of the technique for stenosis diagnosis by detecting neck skin vibrations. In-vivo validation is in progress.

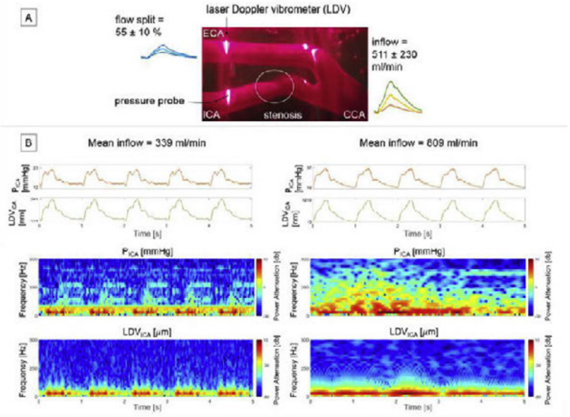

Fig1A: The pressure probe was positioned in the internal carotid artery (ICA) and the laser Doppler vibrometer (LDV) on the surface directly above the pressure probe. Panel B. pressure and LDV signals in the ICA, and their spectrograms (1K, Hann, 93%-overlap, LabChart). The amplitude of the disturbances was greater at a higher mean inflow for both signals.

References

[1] Li Y et al, Optics Express 26(3): 3638-3645, 2018.
[2] Likittanasombut P et al., J Neuroimaging 16: 34-38, 2006.
[3] Groen HC et al., J Biomech 43: 2332-2338, 2010.

4.5

CARDIAC OUTPUT ESTIMATION FROM BEAT-TO-BEAT RADIAL PRESSURE AND PULSE WAVE VELOCITY: A MODEL-BASED STUDY

Vasiliki Bikia [1], Stamatia Pagoulatou [1], Theodore G. Papaioannou [2], Nikolaos Stergiopulos [1]
[1]*Laboratory of Hemodynamics and Cardiovascular Technology, Swiss Federal Institute of Technology (EPFL), Lausanne, Switzerland*
[2]*Biomedical Engineering Unit, 1st Department of Cardiology, "Hippokration" Hospital, Medical School, National and Kapodistrian University of Athens, Athens, Greece*

Background: Cardiac output (CO) monitoring remains a salient challenge. The state-of-the-art is based on generalized transfer functions and parameter estimations from pooled clinical data, which do not necessarily reflect the state of the cardiovascular system in a patient-specific way. Here, we introduce a patient-specific approach to estimate CO from sequential radial pressure measurements and carotid-to-femoral pulse wave velocity (cf-PWV). We do so by effectively tuning a generalized mathematical model of the cardiovascular system (1).
Methods: Initially, the method uses the measured cf-PWV to estimate arterial compliance. We consequently determine aortic flow from beat-to-beat radial pressure measurements based on the assumption of a fairly constant total peripheral resistance (TPR) over several heartbeats (2). Concretely, we developed an algorithm which, starting from an initial flow, employs a gradient-based optimization process (3) to calculate TPR at each beat. This TPR value is subsequently used as input for a new flow approximation. The process is repeated until convergence is reached. To assess the accuracy of the method, we implemented the algorithm on in vivo anonymized data from $n=15$ subjects (4) and compared the method-derived CO to the measured ones.
Results: Our results demonstrated that precise estimates of CO were yielded, with a RMSE of 0.38 L/min (Fig. 1). Small variance in arterial compliance tuning did not show to significantly undermine the accuracy of the CO predictions.
Conclusions: The in vivo validation allows us to conclude that our novel method accurately estimates CO in a patient-specific way. Therefore, the technique may potentially be employed for noninvasive CO monitoring in the clinical setting.

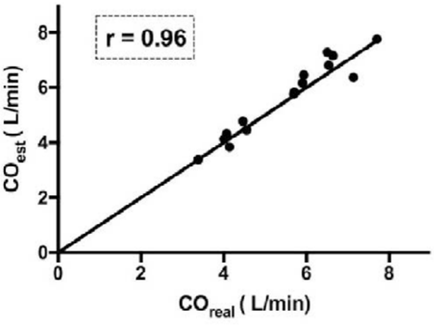

Fig.1. Scattergram of model-derived CO estimates vs. *in vivo* CO data

References

1. Reymond P, Merenda F, Perren F, Rüfenacht D, Stergiopulos N. Validation of a one-dimensional model of the systemic arterial tree. Am J Physiol Heart Circ Physiol. 2009 Jul;297(1):H208-222.
2.Wesseling KH, Jansen JR, Settels JJ, Schreuder JJ. Computation of aortic flow from pressure in humans using a nonlinear, three-element model. Journal of Applied Physiology. 1993 May;74(5):2566—73.
3.Pagoulatou S, Stergiopulos N. Estimating Left Ventricular Elastance from Aortic Flow Waveform, Ventricular Ejection Fraction, and Brachial Pressure: An In Silico Study. ABME-D-17-006793. (Pending for publication)
4. Papaioannou TG, Soulis D, Vardoulis O, Protogerou A, Sfikakis PP, Stergiopulos N, et al. First in vivo application and evaluation of a novel method for non-invasive estimation of cardiac output. Med Eng Phys. 2014 Oct;36(10):1352—7.