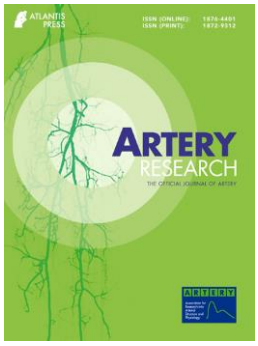

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15.9: MODELLING ARTERIAL PULSE PRESSURE FROM HEART RATE DURING SYMPATHETIC ACTIVATION

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the shape and wave details of the flow wave, where all features are reproduced in a rather faithful manner.
Conclusions: The extended model yields realistic pressure and flow waveforms in arteries of the hand and the foot. After full validation, this extended model will be used to assess the performance of diagnostic and screening devices relying on peripheral hemodynamics signals, such as the pOpmètre®.
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15.9
MODELLING ARTERIAL PULSE PRESSURE FROM HEART RATE DURING SYMPATHETIC ACTIVATION

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Background: The duration of the time segment between the systolic (SP) and post-dicrotic notch peak pressures (PDP) of the arterial pressure wave in humans has been proposed to be related to arterial pulse pressure (PP).[1,2] We considered an effect of RR-interval length on the diastolic pressure run-off and tested the hypothesis that heart rate (HR) affects the timing of systolic and post-dicrotic notch peak pressures.
Methods: We modelled the effects of sympathetic stimulation by progressive central hypovolemia on PP changes based on morphological features of a peripherally measured arterial blood pressure wave shape and HR, making use of linear mixed effect (LME) models. Changes of the arterial pulse wave were tracked from rest towards central hypovolemia in 44 subjects by exposing them to continuous −50 mmHg lower body negative pressure (LBNP). SP and PDP, and HR were extracted from arterial finger pressure and used as model input to predict PP.
Results: From rest to sympathetic stimulation, HR (30%) and thoracic impedance (15%) increased and systolic (SAP) fell by 10%. Model errors of PP (median, and 1st and 3rd quartiles) were 5.2 [3.3 8.9], 4.9 [3.8 7.7], and 4.9 [3.7 8.6] for LME models of, respectively, SP-PDP, HR and their combination.
Conclusion: Our study highlights that during sympathetic stimulation by progressive central hypervolaemia, HR affects arterial pressure wave characteristics and that linear models from both HR and SP-PDP duration allow for estimating PP.
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15.10
FORM FACTOR OF THE FEMORAL ARTERY: AN INVASIVE STUDY

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Background: There is a growing interest in precisely estimating mean blood pressure (MBP) in large arteries. The form factor (FF) is the fraction of pulse pressure that must be added to diastolic pressure to estimate the actual MBP, i.e., the pressure integrated (averaged) over the whole cycle. It is admitted that FF of the radial artery is 0.33, while FF of the aorta and carotid and brachial arteries is in the 0.40-0.45 range. The FF of the femoral artery remains to be determined.
Methods: Sixty-five hemodynamically stable intensive care unit patients equipped with an indwelling femoral catheter were prospectively studied (mean age+/-SD = 64+/-14 years). FF of the femoral artery was calculated as the time-averaged MBP minus diastolic blood pressure difference divided by pulse pressure (FF = (MBP − DBP) / PP).
Results: Form factor of the femoral artery was 0.35+/-0.04 (n=65 range 0.22-0.47). FF was similar in female (n=23) and male (n=42) patients (0.36+/-0.05 vs 0.34+/-0.04, respectively) and in patients receiving vasopressors (n=43) or not (n=22) (0.34+/-0.05 vs 0.35+/-0.03, respectively). FF of the femoral artery was positively related to MBP (r^2=0.11) and DBP (r^2=0.07) (each $P < 0.05$) while it was not related to patient's age, body height, body weight, heart rate, systolic pressure and PP.
Conclusions: The mean form factor of the femoral artery was 0.35, a value closer to the FF of radial artery than to the FF of central and brachial arteries. The implications for pressure wave transmission to the lower limbs remain to be studied.

15.11
TOWARDS NONINVASIVE CARDIAC CATHETERISATION

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Background: Doppler echocardiographic measures of diastolic function, such as E/e' are correlates of left ventricular (LV) end-diastolic pressure (p_{ed}) and diastolic compliance (C_d) [1]. We developed a noninvasive computational approach to obtain these essential markers of LV diastolic abnormalities and tested it against the invasive gold standard.
Methods: In patients undergoing coronary angiography (n=8, age 60+/-13yrs, with no atrial fibrillation or other dysrhythmia), we obtained mitral and aortic valve Doppler tracings, LV wall thickness and cavity volumes, brachial systolic and diastolic blood pressure (BP) and, for validation purposes, LV pressure and volume invasively by conductance catheter. Repeated echocardiography and BP measurements were performed at baseline conditions and averaged. Catheter measurements were performed during baseline and Valsalva manoeuvre. The latter causes a change in LV preload, enabling a robust estimation of C_d. We fitted a computational model describing the cardiovascular circulation (CircAdapt,www.circadapt.org) to the noninvasively measured data. Catheter measurements served as a reference to validate model-predicted p_{ed} and C_d.
Results: Catheter-measured p_{ed} was found to be 21+/-6mmHg (mean+/-SD, n=8) and C_d was 3.1+/-3.0ml/mmHg (n=6). The bias and limits of agreement between the model-estimated and catheter-measured p_{ed} and $C_d$were -0.9+/-7.5mmHg and 1.1+/-2.6ml/mmHg, respectively.
Conclusions: We found reasonable agreement between our noninvasive modelling-based method of estimating p_{ed} and C_d and catheter measurements. Due to its noninvasiveness, our method could be useful for detection of LV diastolic abnormalities in more patients and settings. Next, we will investigate how measurement errors propagate into the uncertainty of model predictions of p_{ed} and C_d.
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