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P121: IDENTIFYING HAEMODYNAMIC DETERMINANTS OF PULSE PRESSURE: AN INTEGRATED NUMERICAL AND PHYSIOLOGICAL APPROACH

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hypertension, with investigators, however, not reaching a consensus on the relative importance of each wave component (1,2).

Objective: The aim of the current investigation was to examine the wave profile over time after developing an age-adapted, mathematical, one-dimensional model of the cardiovascular system.

Methods: Our state-of-the-art 1-D model (3,4) was extended to include turbulence and inertial effects of the flow exiting the left ventricle. Literature data on the age-associated changes in arterial stiffness, peripheral resistance and cardiac contractility were gathered and used as an input for the simulation.

Results: The predicted evolution of pressure and augmentation index with age followed accurately the curves obtained in a number of large-scale clinical studies. Analysis of the relative contribution of the forward and backward wave components showed that the forward wave becomes the major determinant of the increase in central and peripheral SBP and PP with advancing age.

Conclusions: The 1-D model of the ageing tree and heart captures faithfully and with great accuracy the central pressure evolution with ageing. The stiffening of the proximal aorta and the resulting augmentation of the forward pressure wave is the major contributor of the systolic pressure augmentation with age.

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IDENTIFYING HAEMODYNAMIC DETERMINANTS OF PULSE PRESSURE: AN INTEGRATED NUMERICAL AND PHYSIOLOGICAL APPROACH

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Purpose: Hypertension, the single biggest killer worldwide¹, arises mainly as a result of an increase in central pulse pressure (PP)², yet haemodynamic basis of that increase is still disputed. We examined the ability of a simple "reduced" model comprising a proximal characteristic impedance linked to a Windkessel element to accurately predict PP from aortic blood flow and applied the model to examine PP dependence on cardiac and vascular properties.

Method: PP obtained from the model was compared with theoretical values obtained in silico and in vivo. Theoretical values were obtained using a distributed multi-segment model in a population of "virtual" subjects (n = 3,095) in which cardiovascular properties were varied over the pathophysiological range. In vivo measurements were in normotensive subject (n = 13) during modulation of physiology with vasoactive drugs with divergent actions on cardiac and vascular properties and in hypertensive subjects (n = 156).

Results: PP derived from the model agreed with theoretical values (mean difference SD, -0.09 ± 1.96 mmHg) and with measured values (-1.95 ± 3.74 and -1.18 ± 3.67 mmHg for normotensive and hypertensive subjects respectively). Parameters extracted from the model agreed closely with theoretical and measured physical properties. PP was seen to be determined mainly by total arterial compliance (inversely associated with arterial stiffness) and ventricular dynamics: the volume of blood ejected up to time of pulse pressure and the rate of ventricular ejection up to this point.

Conclusion: Increased flow and/or volume accounted for 20.1 mmHg (52%) of the 39.0 mmHg difference in pulse pressure between the upper and lower tertiles of the hypertensive subjects.

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CALCULATING RESERVOIR PRESSURE WITH OR WITHOUT FLOW INFORMATION: SIMILARITY AND ALGORITHMIC SENSITIVITY AT RADIAL ARTERY

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Background: Reservoir pressure is typically estimated from the pressure waveform information only. Comparability with estimates made using pressure and flow depend on assumptions, e.g. a proportional relationship between excess pressure and flow [1]. In this study, we compared (i) results using flow and pressure versus pressure-only at the radial artery, and (ii) two different algorithms used in the literature for pressure-only analysis.

Methods: Reservoir pressure separations were performed on 95 hypertensive individuals where radial pressure and flow velocity waveform measurements were available [2]. Algorithm (F) used flow and pressure information [3]. Algorithms (P1) and (P2) refer to the two different pressure-only implementations as used in [4, 5], and [1, 6], respectively. Reservoir curves characterized by physiologically implausible parameters, i.e. a rate constant $b < 0$ or an asymptotic pressure $P_{\infty} < 0$, were discarded, leaving 63 subjects with valid reservoir pressure data.

Results: Estimated reservoir parameters are shown in Table 1. Algorithm (F) showed statistically significant differences in most of the parameters compared to (P1) and (P2), although, except time constant τ and asymptotic pressure P_{∞} , there was a strong correlation between methods. Significant differences were observed in reservoir pulse pressure and area estimates between (P1) and (P2) despite their, in general, high correlation.

Table 1. Quantification of reservoir pressures p_{res} obtained by methods (F), (P1) and (P2) at radial artery in the format of mean \pm standard deviation based on 63 subjects whereby PP denotes the reservoir pulse pressure, A_p the area of reservoir pressure above diastolic blood pressure, τ the time constant describing the diastolic pressure decay, P_{∞} the asymptotic blood pressure and $a, b = 1/\tau$ the rate constants. Peripheral (area) resistance and compliance, i.e. R and C, were estimated from the rate constants a and b for (P1) and (P2) using flow information. The correlation coefficient r was computed between relevant methods. The statistical significance of the differences between methods was based on a paired t-test with * indicating $p < 0.05$.

Radial artery	p_{res} (F)	p_{res} (P1)	p_{res} (P2)	r(F,P1)	r(F,P2)	r(P1,P2)
PP [mmHg]	41.5 \pm 10.0	36.3 \pm 7.2	35.7 \pm 7.0	0.82*	0.82*	0.96*
A_p [mmHg s]	17.5 \pm 4.3	15.6 \pm 3.7	15.5 \pm 3.7	0.94*	0.94*	1.00*
τ [S]	0.3 \pm 0.1	0.6 \pm 0.4	0.6 \pm 0.3	0.36*	0.42*	0.88
P_{∞} [mmHg]	65.7 \pm 10.3	63.9 \pm 15.2	64.8 \pm 12.6	0.45	0.53	0.79
a [1/s]	–	8.1 \pm 5.2	7.4 \pm 2.7	–	–	0.93
b [1/s]	–	2.2 \pm 1.1	2.1 \pm 0.8	–	–	0.84
R [mmHg s/m]	419.0 \pm 188.8	453.7 \pm 348.2	436.7 \pm 302.6	0.68	0.75	0.92
C [mm/mmHg]	0.8 \pm 0.3	1.7 \pm 1.0	1.7 \pm 1.0	0.70*	0.70*	1.00