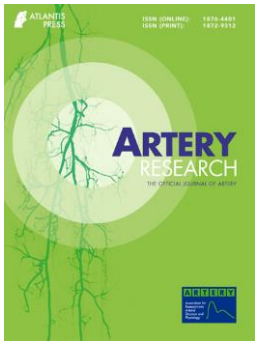

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P5.5: COMPENSATORY EFFECT BETWEEN AORTIC STIFFENING AND REMODELLING DURING AGEING

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corresponding bifurcation. An extension to further segments can then be achieved by multiplication of the associated functions. This leads to an analytical transfer function which incorporates all model parameters and enables a patient-specific choice of these parameters reflecting arterial properties (eg. arterial lengths, radii, stiffness).
The transfer function based on an arterial tree model and wave propagation theory can be personalised by optimising model parameters to fit a specific patient. This may provide more accurate estimates of central pressure curves and thus could enhance the precision of pulse wave analysis and its clinical value.

P5.3
CHARACTERIZATION OF THE BIOMECHANICS OF THE RAT XENOGRAFT MODEL OF ABDOMINAL AORTIC ANEURYSM BY RING TEST

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Setting up new strategies to prevent the growth and fatal rupture of aneurysms remains a challenge. The rat xenograft model of abdominal aortic aneurysm (AAA) has been used to develop gene and cell therapies with the aim of countering the expansion of the diseased arterial wall. It is of primary importance to characterize the mechanical behavior of this model before studying the effects of those therapies.
Ring tests were performed on 34 samples extracted at several locations along rat AAAs and 8 samples from native healthy rat aortas (NA). The circumferential ultimate strength and extensibility were measured. Material parameters from an exponential isotropic hyperelastic strain energy density function were identified by a finite-element inverse approach. The changes of mechanical properties along the axial direction of AAA were analyzed through the correlation between parameters and external radius.
AAAs display significantly higher radius and thickness than NAs and appear weaker, with circumferential ultimate strength and extensibility significantly lower. Best-fit values for material parameters show significant differences between both groups. We reveal strong correlations between ultimate strength and external radius ($r=-0.58$, $p<0.001$). As well as between the material parameter accounting for non-collagenous matrix response and external radius ($r=-0.79$, $p<0.001$). These associations are explained by the degradation and loss elastin, which are more important as we get closer to the zone of maximal dilatation.
These results indicate that the most dilated parts are the most prone to rupture. Further studies should evaluate stress distributions within the arterial wall in order to better predict rupture risk.

P5.4
CENTRAL PRESSURE APPRAISAL: CLINICAL VALIDATION OF A SUBJECT-SPECIFIC MATHEMATICAL MODEL

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Increased blood pressure represents a major cardiovascular risk factor for western populations. Usually blood pressure is measured peripherally, but current evidence suggested that central blood pressure better predicts cardiovascular events. However, central blood pressure measurement is not feasible in daily clinical practice. New instruments can estimate non-invasively central blood pressure from applanation tonometry at peripheral sites and transfer function. Accuracy of this evaluation has been questioned. Remarkable development in medical imaging and computation techniques granted the opportunity to explore mathematical models describing the cardiovascular functioning.
Aim of the present study is the clinical validation of a mathematical model for appraisal of central blood pressure from subject-specific non-invasive measurements (i.e. brachial pressure, age, height, weight, ESV, EDV, etc.).
A total of 52 healthy young male were selected for the present study. Central pressures were estimated with subject-specific model and compared with a common non-invasive technique (Sphygmocor).
Model estimated systolic and diastolic blood pressure resulted to be significantly related to Sphygmocor central systolic (r 0.65 $p<0.0001$) and diastolic (r 0.84 $p<0.0001$) blood pressure. The model showed a significant over-estimation of systolic (+7.8 [-2.2;14] mmHg, $p=0.0003$) and under-estimation of diastolic (-3.2 [-7.5;1.6], $p=0.004$) values.
In conclusion, the proposed mathematical model allows non-invasive prediction of central aortic pressure with good accuracy in more than one half of this population. Both the systematic over-estimation of aortic systolic pressure and the under-estimation of diastolic values compare well with the error reported by large meta-analysis when Sphygmocor is used with non-invasive calibration.

P5.5
COMPENSATORY EFFECT BETWEEN AORTIC STIFFENING AND REMODELLING DURING AGEING

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The arterial tree exhibits a complex spatio-temporal wave pattern, whose healthy behaviour depends on a balance between mechanical and geometrical properties. Several clinical studies demonstrated that this balance progressively breaks down during ageing, when the aorta stiffens and remodels. This dual mechanism is investigated by a validated multi-scale model aiming to elucidate how aortic stiffening and remodelling quantitatively impact the waves pattern in the aorta.
The detrimental increase of maximum pressure and left-ventricular work during ageing is here shown to results from the impairment of fluid dynamic balance between generation-propagation of forward waves and reflections- damping of backward waves. Our quantitative outcomes confirm several clinical studies results. Our results shed light on how ageing-induced aortic stiffening and remodelling affect this balance: the former enhances first pressure pulse at ventricular-aortic interface during ejection, while the latter damps it; although stiffening tends to decrease reflection coefficients at bifurcations, their remodelling-induced growth prevails; aortic remodelling undermines the protective wave-trapping mechanism while stiffening enhances it; aortic stiffening reduces pulse pressure amplification while remodelling augments it; by contrast, both stiffening and remodelling contribute to limit the growth of left-ventricle work with age.
These results suggest that an excessive imbalance between aortic stiffness and geometric remodelling during clinical treatment of elderly subjects should be avoided. Our results show how a dramatic reduction of the pulse wave velocity in a dilated aorta can lead to an increase of pulse pressure amplification, with potential detrimental effects on organs such as the kidneys.

P5.6
CORONARY FLUID MECHANICS IN AN AGEING CARDIOVASCULAR SYSTEM

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Coronary artery diseases are the most common type of cardiovascular diseases and have an increasing prevalence with age. Epidemiological studies demonstrated that coronary failure is associated with several pathologies (as atherosclerosis, hypertension, diabetes, endothelial dysfunction, renal insufficiency). Since the majority of these pathologies has an increasing prevalence with age, little is known about the effect of ageing alone on the coronary circulation.
In this context, the aim of the present work is to isolate the impact of ageing on the coronary flow. A fluid mechanics-based approach is adopted, based on the coupling of the mathematical model of the systemic circulation we proposed and validated with a model of the coronary circulation proposed and validated by Mynard et al (Am J Physiol, 2014). The resulting evolution of coronary pressure/flow waveforms and their coupling with the left-ventricular condition are analysed using impedance analysis and wave intensity analysis.
Confirming results from several clinical studies, we highlight a complex spatiotemporal coronary wave pattern, where intense waves originate from the aorta and from the deep myocardium, during both isovolumic compression and diastolic phase. The subendocardial viability ratio decreases with age, total coronary flow is slightly reduced, and left-ventricular work increases. Consequently, the left-ventricular work per unit of blood flow increases, thus limiting cell oxygen availability abundance, therefore increasing the risk of myocardial infraction.
Our results highlight a physiological age-induced supply/demand unbalance in the coronary arteries, which can augment the risk of myocardial ischemia and can contribute to pave the way to other coronary pathologies.