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**Alteration of Reynolds number and flow partition modify wall shear stresses at arterial branches in a way that can explain age- and species-dependent patterns of arterial disease**

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Atherosclerotic lesions have a highly non-uniform distribution within the arterial system, suggesting that haemodynamic stresses, particularly wall shear stress (*WSS*), control their development. Around the origins of aortic side branches, lesion patterns vary with age and species. We investigated for the first time whether different patterns of *WSS* resulting from different Reynolds numbers (*Re*) and branch flow rates ($Q_{branch}$) can account for this.

The emergence of an intercostal artery from the aorta, which has a 10-fold larger diameter, was modelled as a cylindrical tube emerging perpendicularly from a flat plate; the idealised geometry simplifies interpretation of effects of *Re* and $Q_{branch}$. High-order steady flow computations were performed with spectral/*hp* element methods, assuming a parabolic inlet velocity.

Aortic blood flow and *WSS* patterns around the branch ostium changed markedly with *Re* and $Q_{branch}$. At *Re* 100, appropriate for the mouse, *WSS* was almost uniform. With increasing *Re*, blood entered the branch from regions nearer the aortic wall, affecting *WSS* more. *WSS* was reduced at the sides of the ostium and increased upstream and downstream of it. Increasing $Q_{branch}$ caused *WSS* to decrease downstream, where a stagnation region formed, and blood increasingly entered the branch from more distant areas. Combined increases in *Re* and $Q_{branch}$ caused competing effects on *WSS*, reducing it at the sides and downstream.

Lesions develop at the sides of intercostal ostia in adult human and rabbit aortas, but more uniformly around intercostal ostia in mice. These patterns correlated well with patterns of *WSS* obtained here by altering *Re* or $Q_{branch}$. Hence, despite the simplicity of the present model, this study suggests that alteration of simple blood flow parameters such as *Re* and $Q_{branch}$ results in changes to the pattern of *WSS* that in turn can explain at least some of the age- and species-dependent lesion patterns.

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**Quantitative analysis of ct-scanned images of coronary vasculatures of porcine hearts**

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Coronary heart diseases are one of the major causes of deaths in the United States as well as in other Westernized countries. The hemodynamic parameters leading to these diseases are mostly unknown due to inherent difficulties to conduct experiments in the beating heart especially in the inner layers. In order to understand coronary artery disease, a complete analysis of coronary circulation is needed. Virtual/computational models depicting structure-function relation of the heart are imperative in this endeavor. Such models can serve both as training objects for students as well as for cardiologists allowing them to study coronary circulation. Even further, once the scanning technology has advanced so that real-time scanning of life patients is possible at reasonably high resolution, the proposed technique can be used for in-vivo analysis of the coronary arteries.

The temporal and spatial distribution of coronary blood flow, pressure, and volume are determined by branching pattern and three-dimensional geometry of the coronary vasculature, and by the mechanics of heart wall and vascular tone. Consequently, a realistic modeling of the coronary blood flow requires, as a first step, an accurate representation of the coronary vasculature in a three-dimensional model of the beating heart. As a basis for such a geometric model, statistical databases for vessel diameter, vessel lengths, as well as bifurcation angles for vessels of different size and order can be used. Then, an entire generic model of the coronary vasculature can be generated that includes vessels from the large proximal coronary arteries down to the capillary level (3 orders of magnitude difference in diameter).

In order to extract the necessary information for such a database from a series of CT scanned porcine hearts, the vessels are extracted from the scanned images. By determining the center lines of the extracted vessels, the vessel radius, vessel length, and bifurcation angles can be identified for vessels of different orders. The extraction algorithm is based on a topological analysis of a vector field generated by the normal vectors of the extracted vessel walls. With this approach, special focus on achieving the highest accuracy of the measured values possible is ensured.

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**Effects of implementing a viscoelastic arterial wall model for solving the one-dimensional equations of blood flow**

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One-dimensional blood flow models are powerful tools for modeling flow and pressure waves in large networks of blood vessels for applications such as vascular surgical planning to assess disease and predict outcomes with low computational cost. Arteries exhibit viscoelastic properties such as hysteresis and damping – so, an arterial model with viscoelasticity, rather than just elasticity, is necessary to reproduce pulse wave propagation characteristics in flow simulations [1,2]. The purpose of the work presented is to incorporate a viscoelastic constitutive model [2] for the arterial wall in an existing one-dimensional, finite-element blood flow simulation environment and assess its effect on flow and pressure waves [3]. It is able to provide more comprehensive understanding of the effects of viscoelasticity than is available, especially in large arterial network models.

In a carotid artery model, with physiologic flow as the inlet boundary condition (BC) and impedance BC at the outlet, pulse pressure was 17% lower with viscoelasticity compared to the elastic case. Energy loss, calculated as the area of the hysteresis loop as a fraction of the total stored energy during a cardiac cycle, was 2.4% on average over the length of the vessel (similar to experiments in [2]). The same model with high frequency oscillations in the inlet flow showed considerable damping in flow and pressure waves as seen from their Fourier transforms. In an abdominal bifurcation model, difference in pulse pressure was higher than for the carotid model (30%) and the average energy loss was about the same (2.7%). Similar comparisons for much larger network models such as the aorta with all its main branches are in progress. In the longer term we aim to include viscoelasticity into 3D deformable-wall blood flow simulations.

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**Effect of cardiac motion on haemodynamics in the superior mesenteric artery (SMA)**

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The SMA is very unusual in that it appears largely to be spared from the process of atherosclerosis [1]. This is in stark contrast to other large vessels arising close to the SMA, and to the aorta itself.

Because location of atherosclerotic disease is often associated with unfavourable wall shear stress (WSS) distribution at arterial junctions, we hypothesise that this sparing might be attributed to more favourable haemodynamic characteristics in the SMA. The purpose of this paper is to describe a methodology for the haemodynamic characterisation to support subsequent comparative analysis with adjacent aortic branches.

During the study it became apparent that the SMA is highly mobile, and thus that an analysis based on rigid geometry would be inappropriate. A fully transient computational fluid dynamic (CFD) model is constructed, based on dynamic magnetic resonance imaging (MRI) data, using a novel mesh-morphing algorithm operating on a parametric mesh constructed from simple geometric primitives. The mesh generation routine is efficient in the production of transient nodal co-ordinate data and has the merit that it is easy to control the element size distribution mapped onto the original geometric primitives. It is robust in operation, and is ideally suited to the generation of dynamic CFD meshes of arterial systems that are free from major pathology. Flow boundary conditions are determined based on phase contrast MRI velocity measurements. Haemodynamic characteristics of the aorta/SMA branch are evaluated and contrasted for rigid and mobile geometries.

Results for the rigid and moving-walled analyses showed no substantial difference in terms of time-varying WSS distributions, despite significant gross motion of the SMA. However, in both cases the models predicted a low propensity for the development of atherosclerosis in the SMA compared to the abdominal aorta.

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