MRI GUIDED CFD SIMULATIONS OF LDL TRANSPORT IN SUBJECT SPECIFIC CAROTID ARTERIES: AN INITIAL METHODOLOGY

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INTRODUCTION

In recent years MRI has been successfully employed as a means of providing high quality data for anatomical and velocity input into CFD simulations of patient specific hemodynamics. Of particular interest has been its use in studying diseases such as atherosclerosis where the role of regional flow and wall fluid shear is fundamental to disease genesis and progress. The work presented here takes this approach one step further by using MRI and CFD to predict the transport of blood borne chemicals to the arterial vessel wall by also solving the species transport equations. This can be used for fundamental investigation into the interactions between convection, diffusion and reaction in the transport of species and their relative roles in diseases such as atherosclerosis and thrombosis. Potentially, individual, patient specific models can be used. This abstract will present our initial approach to this problem and present results from a single volunteer's carotid artery simulation.

METHODS

For this study the transport of LDL to the endothelium in the normal carotid bifurcation was investigated. A 3D TOF MRI sequence (80 x 80 x 60 mm FOV and 256 x 256 x 40 matrix) was used to obtain the carotid anatomy in a normal volunteer on a Philips NT scanner using a 47 mm surface coil. A 3-component phase velocity encoded slice (80 x 80 mm FOV, 256 x 256 matrix, 3 mm slice thickness and 31 ms heart phase interval) was placed co-incident with the foot end of the TOF block to provide input common carotid velocity information. The commercial software package Analyze (3.1, BIR) was used to segment the common, internal and external carotid arteries from the TOF data as a triangulated surface mesh. Only the internal and external carotid arteries were included meaning that arterial branches were artificially removed. The surface was extracted from the MRI data block by first converting the lumen of the vessel of interest into splines and then by growing a surface onto these splines. This surface was adjusted, smoothed, truncated and fixed, using Magics (v5.4, Materialise) software and exported as a CAD file into the CFD front-end software package Gambit (v2.0.4, Fluent Inc). Truncation was necessary as the surface-fitting algorithm produced rounded ends unsuitable for CFD boundaries. To account for the lost data due to truncation, extra slices were added to the end of the MRI TOF data block before importing into Analyze. In GAMBIT, the mesh was regenerated for simulation with the finite difference CFD solver FLUENT (v6.0.12 Fluent inc). In particular a boundary layer was added to the vessel wall. Blood LDL was modelled as a blood borne species which reacted with the vessel wall via a first order reaction mechanism. The fluid viscosity was fixed at 0.0035 Pa.s and LDL diffusivity in blood 5×10^{-8} m^2/s. The mass, momentum and species transport equations were solved simultaneously using the pulsatile MRI velocity data as the flow input boundary condition imposed by a purpose written user defined function. Second and first order upwinding schemes were used for the mass-momentum and species solvers respectively. These solutions were uncoupled. Two solutions were computed with first order wall reaction mechanism with reaction rates of 2×10^{-3} and 2×10^{-5} kgm^2s^{-1}.

The solutions were run for multiple cardiac cycles until no significant difference between cycles was detected.

RESULTS AND DISCUSSION

Figure 1 shows the axial velocity contours at (t/t_c)= (a) 0.16 (b) 0.2 (c) 0.24 (d) 0.32 (e). The carotid bulb is on the right and indicates an expected low /reversed velocity region which increases in size over the cardiac cycle. High flows further downstream are due to a narrowing of the vessel but also due to the absence of further bifurcations. The complex axial inlet velocity profile derived from MRI images is also seen. It is clear, however, that this complexity is not as apparent further donwstream but still within the main carotid artery. This may indicate that detailed MRI inlet velocity is not required provided the inlet is sufficiently far from the region of interest. Figure 2 shows the simulated transport of LDL to the wall, with $k=2\times10^{-3}$ at t/t_c = (a) 0.04 (b) 0.2 (c) 0.32 (d) 0.52 (e)0.78. There is clearly a large regional dependence on the degree of LDL transport with significant

differences in transport at the bifurcation compared to the common, internal and external carotid arteries.

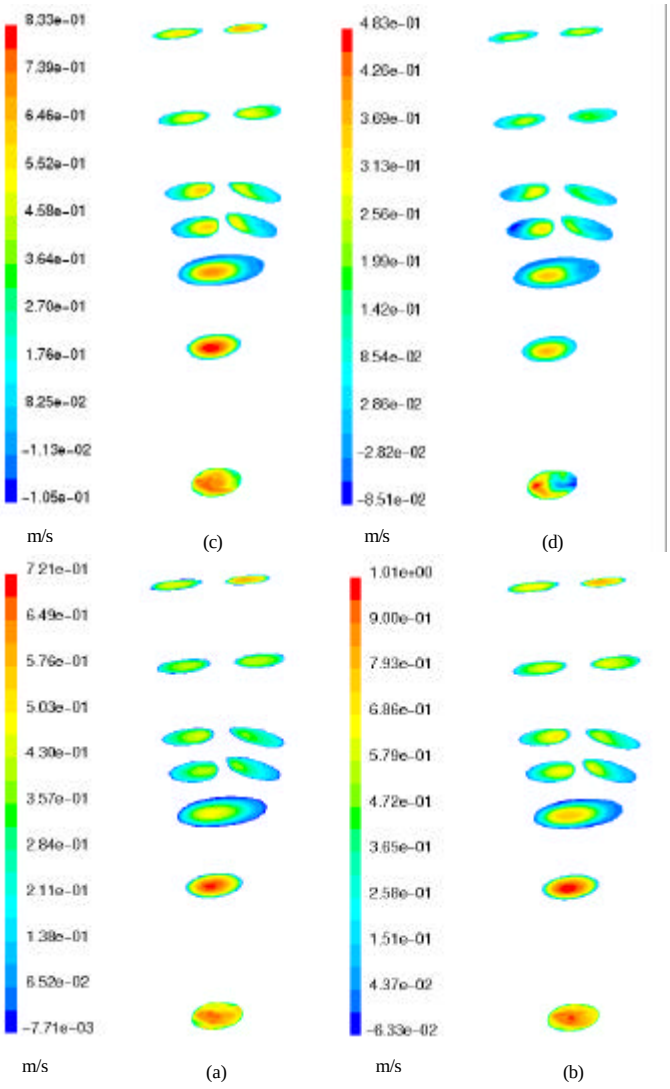

Figure 1. Axial velocity profiles.

The initial high rate of transport at the inlet is due to a constant inlet mass fraction being specified here and consequently an zero thickness species boundary layer. As a constant (i.e. non-regional) wall reaction mechanism was used, these regional variations are due to flow phenomena (convection / diffusion of LDL, separation regions and stagnation points) which in turn are a result of the inlet boundary condition and the bifurcation geometry. Clearly, both of these are a result of the MRI data, although from the argument above it would seem that the geometry information is particularly crucial and therefore the manner in which the anatomy is extracted from the MRI data is also very important. Convergence of the species solution was found to depend upon location, time in the cardiac cycle and wall reaction rate magnitude. The higher wall reaction rate simulation converged in 6 cycles compared to 9 for the lower rate. We also found that mesh independence was difficult to attain at all times and locations unless a very dense and hence uneconomic mesh was used through out. Mesh independence could be obtained but often not over the entire cardiac cycle as the position of mesh dependence changed with time. In future for an efficient solution process, an adaptive mesh should be used.

CONCLUSIONS

MRI in combination with CFD has been shown to be a valuable tool in predicting blood biochemical transport. As such it may play a major role in the fundamental understanding of wall / blood interactions and consequently conditions such as atherosclerosis and thrombosis. Some problems relating to mesh density need to be improved, however, if an efficient solution is to be obtained.

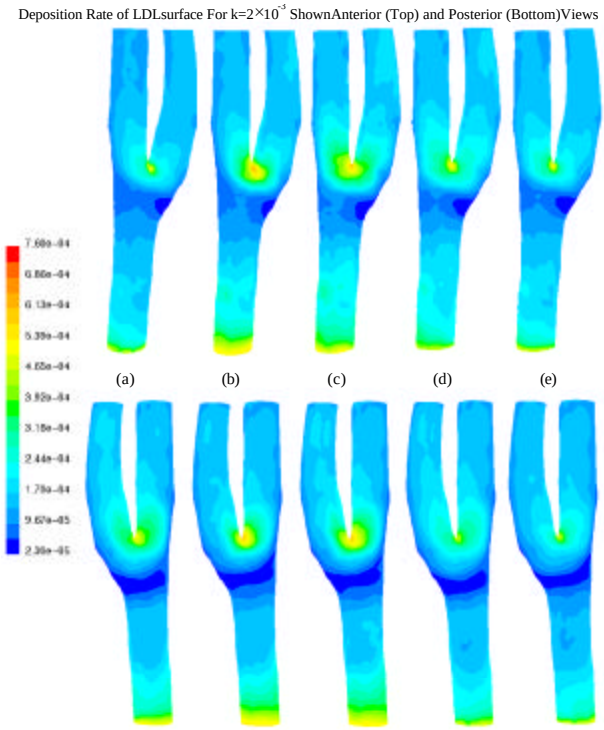

Figure 2. LDL transport to the wall

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