PHYSIOLOGICAL FLOW IN A MODEL OF ARTERIAL STENOSIS

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The term arterial stenosis refers to narrowing of an artery where the cross-sectional area of the blood vessel is significantly reduced. The most common cause is atherosclerosis where cholesterol and other lipids are deposited beneath the intima of the arterial wall. As the amount of this fatty material increases there is an accompanying proliferation of connective tissue with the whole forming a thickened region called plaque. This plaque impedes blood flow, which may as a result transient to turbulent at the post-stenotic region, where a high level of flow recirculation is achieved, which could cause thrombosis (blood clotting) in the post stenotic region as well as abnormal vessel wall shear stresses. The study of the transitional flow through stenoses is of clinical interest as outlined above, however, accurate modelling of such flow can be challenging. In the present paper we propose a Large Eddy Simulation (LES) technique to study physiological pulsatile transition to turbulent flow in a 3D model of arterial stenosis. In LES, a Gaussian type spatial *grid*-filter is applied to the Navier-stokes equations of motion for separating the large scale flows from the small scale or sub-grid scale (SGS). The large scale flows are then resolved fully while the unresolved SGS motions are modelled using the Germano-Lilly dynamic model [Lilly, 1992].

The computational domain chosen in our study is a simple channel with a biological type stenosis formed eccentrically on the top wall. Physiological pulsation was generated at the inlet using the first harmonic of the Fourier series of pressure pulse [Womersley, 1955]. Some interesting features of the flow physics are achieved and these will be presented in details in the full scale paper and their importance to pathological issues are also explained physically. However, in this abstract some samples of LES result are shown below.

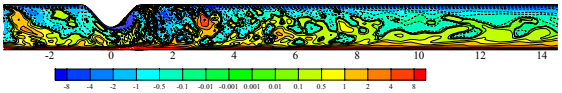

Figure 1: Spanwise-averaged vorticity

Figure 1 shows the spanwise-averaged vorticity for the Reynolds number of 2000. The increasing velocity creates a shear layer from the centre of the stenosis, which then separates and begins to roll up into the final form of shear layer as an anti-clockwise (dashed lines) rotating vortex, where an adverse pressure gradient occurred. On the other hand, the secondary shear layer which separates from the bottom wall and induces a clockwise vortex (solid lines).

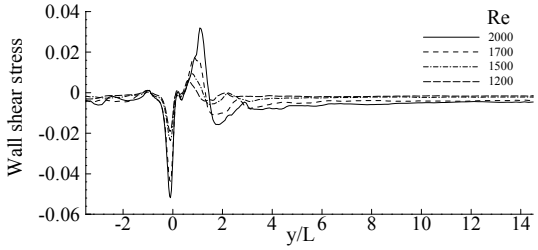

Figure 2: Normalised time-mean wall shear stress.

The normalised time-averaged wall shear stress is depicted in Figure 2 for the different Reynolds numbers. At the site of upper wall stenosis, there is a stress drop at the centre of the stenosis due to the maximum adverse pressure gradient. At the post lip of the stenosis the peak wall shear stress is achieved. This extreme stress is the main focus of concern as regards potential for pathological disruption of the endothelial intimal arterial lining.

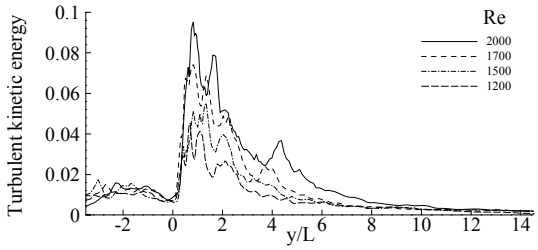

Figure 3: Normalised turbulent kinetic energy

Figure 3 illustrates the normalised turbulent kinetic energy (TKE) for the different Reynolds numbers. Before the stenosis the TKE is very small, which is expected, as the pulsatile flow is laminar. However, beyond the centre of stenosis at $0<y/L<5$ the level of TKE is so high that haemolysis (red blood cell disruption) may potentially occur. Furthermore, apprximatley 31% of energy is dissipated through SGS to the flow for Re=2000.

The application of LES in the field of bio-engineering is novel. LES has the capability to model time accurate physiological flow, as demonstrated here and therefore, we believe that LES will play a significant future role in bio-engineering.

D. K Lilly, Phys. Fluids A., 4(3):633-635, 1992.
J. R. Womersley, J Physiol, 127:553-563, 1955.