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THREE-DIMENSIONAL PULSATILE FLOW THROUGH ASYMMETRICALLY AND SYMMETRICALLY CONSTRICTED VESSELS

Neil W. Bressloff

School of Engineering Sciences
University of Southampton
Highfield, Southampton
SO17 1BJ, UK.

ABSTRACT

Comparisons are made between two- and three-dimensional pulsatile flow through asymmetrically and symmetrically constricted vessels. Most significantly, mean shear stress distributions and levels of oscillation (which have important implications for the prediction of stenosis pathology) are very different, both qualitatively and with respect to peak values. Maximum mean shear stresses are almost 40% higher for the asymmetric constrictions. They are also approximately 30% higher than produced in two-dimensional simulations. The distribution of oscillating wall shear stress suggests ways in which secondary lesions may develop. We also show that the so-called "vortex wave" produced by an asymmetric two-dimensional constriction does not exist in a three-dimensional simulation at the Reynolds and Strouhal numbers of 600 and 0.03 considered here.

INTRODUCTION

Artherosclerotic disease tends to be localized at sites within the vascular system that are subject to particular hemodynamic conditions. There are now strong correlations between regions of low mean shear stress (and high oscillation) and narrowing of the artery lumen [1]. The resulting constriction is clinically referred to as a stenosis. When a stenosis develops, blood flow to vessels downstream is increasingly restricted, and the shear stress through the constriction can increase to such a level as to cause rupture, inducing thrombosis that can totally block blood flow to vital organs, particularly the brain [2].

Computational fluid dynamics (CFD) continues to play an important role in aiding the diagnosis, understanding and treatment of artherosclerosis. In recent years, increases in computing power have made detailed three-dimensional studies possible [3]. Since clinical evidence suggests that stenoses do not necessarily develop in a symmetrical way [4], it is important to understand how the flow and associated wall shear stress distributions differ in symmetrically and asymmetrically constricted vessels. The present article addresses some of these differences in both two- and three-dimensional geometries. In two-dimensions, a symmetrical geometry produces large recirculating regions distal to the stenosis [1], but in asymmetrically constricted vessels, a vortex wave is produced [5] yielding a more complex shear stress distribution. Evidence from the present study shows that the two-dimensional flow patterns are significantly influenced by secondary effects, and that a vortex wave does not occur in the three-dimensional asymmetrical geometry. In both three-dimensional cases, successions of spiraling vortices convect downstream impacting on the walls of the vessel.

Pulsatile flow through constricted vessels is governed by the Reynolds number, $Re=U_{max}h/\nu$, and the Strouhal number, $St=h/(U_{max}T)$, where U_{max}, h, T and ν denote, respectively, the maximum inlet velocity, the height (or diameter) of the vessel, the period of the pulse and the kinematic viscosity (all in SI units). These flows are also characterized by the Wormersley number $\alpha=(\pi ReSt/2)^{-0.5}$.

NUMERICAL METHOD

All results were obtained using a parallel finite volume CFD code developed at the University of Southampton. Simulations were performed on a cluster of Pentium III dual processor (500 MHz) personal computers using the message-passing interface (MPI) for communication between separate processors.

The full Navier-Stokes equations are solved using the pressure implicit splitting of operators method (PISO). Full details of the method including its parallel implementation can be found in reference [6]. A Newtonian fluid is assumed.

The geometry of the constriction is defined by a Beta function with a mean of 0.5 and a variance of 0.02 producing a blockage across 50% of the vessel height (diameter). Numerical experiments suggest an optimum entry length and downstream section of 5 and 37 heights (diameters), respectively. For the two-dimensional geometry 60 cross-stream cells yield grid converged solutions while 2000 cells are similarly employed in the radial plane in the three-dimensional simulations. A forced sinusoidal inflow is imposed on a straight vessel with a minimum net flow rate of zero and with Re=600 and St=0.03. This flow specification produces a Wormersley number, $\alpha=5.32$, that is within the range of physiological interest. Using a time

step size of 1.786×10^{-5} (deemed appropriate to avoid solution dependence on the time step) the three-dimensional simulations require approximately 128 hours of run-time.

RESULTS AND DISCUSSION

There now exist strong correlations between regions of low mean wall shear stress and artherogenesis and between high shear stress and thromobogenesis. Thus, since the hemodynamic effect on arterial walls is of primary clinical interest in the current context, results are presented for the indicator functions of time-averaged nondimenionalized mean wall shear stress (WSS) and the oscillatory shear index (OSI). WSS and OSI are defined in [7]. Figures 1 and 2 show the main (axial) component of WSS and OSI for the three-dimensional asymmetrical and the symmetrical constrictions, respectively. The contour maps (varying between -0.1 and 0.3 for WSS and between 0.0 and 0.5 for OSI) are unrolled semi-perimeters with end-points on the centerline through the constriction. In the symmetrical configuration, the perimeter distances are scaled so that the constriction fits appropriately in the axes of the plot.

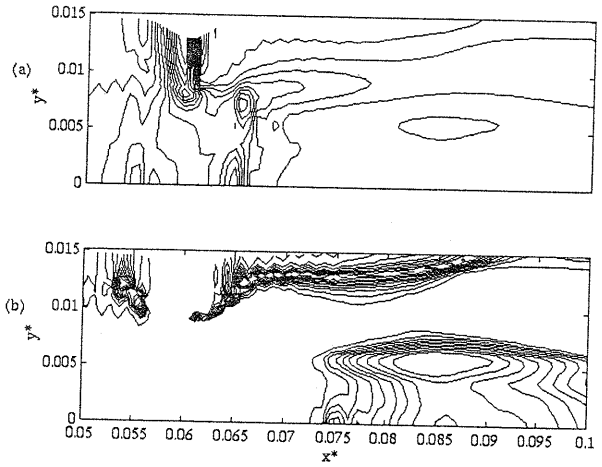

Figure 1 Asymmetrical constriction (a) contours of mean wall shear stress, (b) contours of OSI. x* and y* are non-dimensional distances (x100) from the inlet and around the semi-perimeter, respectively.

In both Figures 1a and 2a, maximum WSS, corresponding to peak absolute shear stress, is located at the points of minimum cross sectional area, as expected. These values are approximately 30% higher than those observed for the equivalent two-dimensional simulations. With respect to three-dimensional effects, it is also interesting to note that the circumferential mean shear stress component (not shown here) is approximately 40% the axial component.

While regions of high WSS are easy to detect due to their accompanying large gradients, areas of low WSS are widely spread. Thus, the OSI becomes a useful indicator function since it reveals regions in the field of low WSS that have relatively high OSI. These sites may be associated with secondary stenoses as observed in clinical practice [7]. Therefore, an interesting question concerns the effect of constriction asymmetry on stenosis development. X-ray angiograms of diseased carotid bifurcations [4] depict significant asymmetry with a second, smaller, asymmetric stenosis on the same wall region as the largest part of the primary occlusion. If artherogenesis does correlate with high OSI, the asymmetry shown in Figure 1b supports the prediction that an asymmetric secondary stenosis forms downstream of an asymmetric primary stenosis. In contrast, Figure 2b suggests that symmetrical arterial wall thickening yields symmetrical secondary thickening.

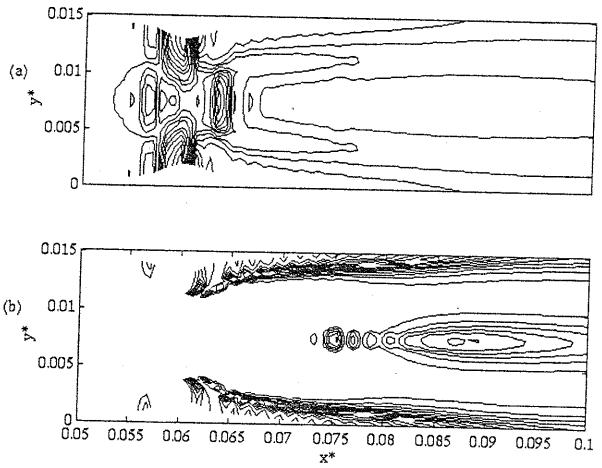

Figure 2 Symmetrical constriction (a) contours of mean wall shear stress, (b) contours of OSI.

The limits of space preclude the inclusion of plots comparing two- and three-dimensional results. As mentioned above, peak values of WSS are approximately 30% lower in the two-dimensional simulations. The other striking comparison concerns the apparent non-existence in the three-dimensional flow of the vortex wave produced by the two-dimensional geometry. Cross-stream slices through the vessel show a complex pattern of spiraling vortices repeatedly impinging on the vessel walls. The cross-stream mixing generated by these vortices, which probably prevent the vortex wave from forming, produces a significant circumferential (mean) shear stress

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