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Three-Dimensional Flow Simulations of End-to-Side Vascular Anastomoses : Flow Dynamic Aspect on Preferential Development of Intimal Hyperplasia or Thrombosis

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=Abstract=

Three-dimensional steady and pulsatile flows in an end-to-side anastomosis were investigated using a finite difference method in order to understand the flow dynamics in the preferential development of distal anastomotic intimal hyperplasia or thrombosis. Steady flow results revealed that a double helical vortex was formed in the host artery and flow recirculations near toe and heel regions were restricted due to the secondary flow. Oscillating wall shear stress with significant secondary flow might be flow dynamic reason of developing intimal hyperplasia or thrombosis near the anastomotic region.

Key words : Anastomosis, Intimal hyperplasia, Thrombosis, Double helical vortex, Wall shear stress.

INTRODUCTION

Intimal hyperplasia or thrombosis have been found primarily at the distal anastomosis as a major source of failure in by-pass graft replacements. Important interactions between fluid dynamics and the chronic development of intimal hyperplasia have been suggested. Many investigators[1~4] have correlated intimal thickening or plaque development in the region of low and oscillating shear stress with flow separation. Clinical results[5, 6] show that a preferred location for the development of hyperplasia is near the toe of the anastomosis and along the outer wall where the inlet stream strikes the wall.

Simple two-dimensional steady flow simulations showed low shear stress distributions near the heel and toe region. It was also reported that a smaller anastomotic junction angle is recommended in the distal end-to-side anastomosis in the hemodynamic point of view[7]. The secondary flow motion as well as the double helical vortex are very important in three-dimensional flow near an end-to-side anastomosis, but are not well described yet. Therefore, the aim of the present study is to investigate steady and pulsatile flows near the region of an end-to-side anastomosis using a finite difference method. This will provide valuable quantitative information relating abnormal flow dynamics with regions of anastomotic intimal hyperplasia or plaque development.

METHODS

A computational fluid dynamics software, FLUENT (FLUENT Inc., New Hampshire, U.S.A.), was used to investigate the three-dimensional flow characteristics near the anastomosis. A graft of the same diameter of 6 mm as

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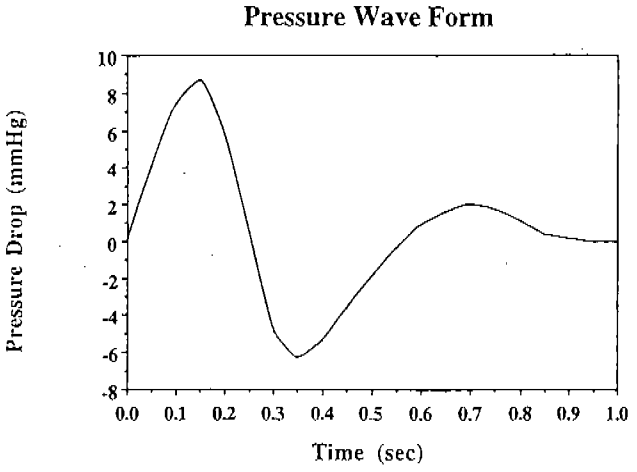

Fig. 1. Pressure gradient wave form for pulsatile flow simulation.

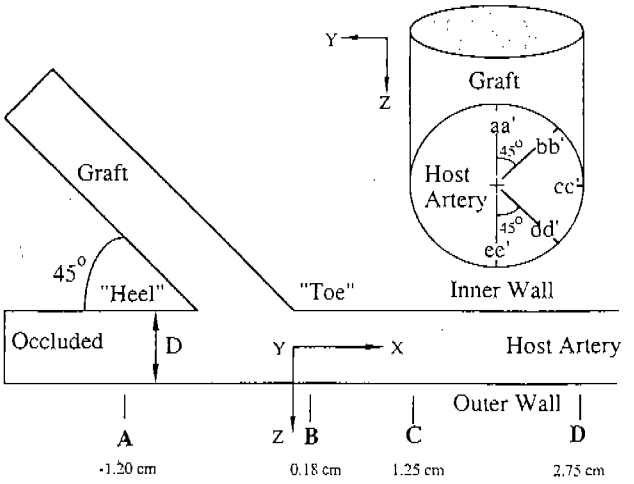

Fig. 2. Specified locations in the end-to-side anastomosis.

the host artery was anastomosed with a junction angle of 45°, and the proximal end of the artery was assumed to be occluded. A non-uniform boundary-fitted structural grid distribution of 41 × 10 × 31 was used for the study.

For incompressible Newtonian fluid, the governing equation for three-dimensional unsteady laminar flow can be expressed as:

$$\nabla \cdot u = 0 \tag{1}$$

$$\frac{\partial u}{\partial t} + u \cdot \nabla u = -\nabla p + \frac{1}{\mathrm{Re}} \nabla^2 u \tag{2}$$

where u are the velocities, p is the pressure and Re is the Reynolds number based on the vessel diameter. In order to solve the above partial differential equations, computational domain was discretized into a collection of control volumes and the differential equations were approximated by a set of algebraic equations. Patankar's SIMPLE procedure was introduced to treat the coupling of velocities and pressure. Detailed procedures for numerical simulations can be referred to Kim et al[3].

The mean Reynolds number was chosen to be 510 for the steady flow simulation, representative of the peak values for flow in the femoral artery. Figure 1 shows the physiological pressure gradient wave form over a cardiac cycle, which was used for the boundary condition in pulsatile flow simulation.

RESULTS

To help the understanding of the results, the following specified locations on the host artery are introduced, as shown in Figure 2: aa' is the inner wall, bb' is a point rotated 45° in the clockwise direction from the inner wall, cc' is the side wall, dd' is a point rotated 45° in the counter-clockwise direction from the outer wall and ee' is the outer wall. A, B, C and D represent locations -1.2 cm, 0.18 cm, 1.25 cm and 2.75 cm respectively, apart from the toe along the host artery in the x-direction.

Steady Flow

At the stagnation point along the outer wall (ee'), the flow stream split into two parts with one stream moving toward the outlet branch and the other stream moving toward the occlusion. Flow recirculations distal to the toe region along the near wall were restricted due to the secondary flow. Figure 3 shows axial velocity profiles and secondary flow velocity vectors on the cross-sections of the specified locations in the host artery. The axial velocity profile at the distal locations of the host artery became skewed towards the outer wall, as shown in cross-sections at C and D in Figure 3. Secondary vortices in the distal host artery were very significant and their circumferential motion became maximal at the side wall (cc') and minimal

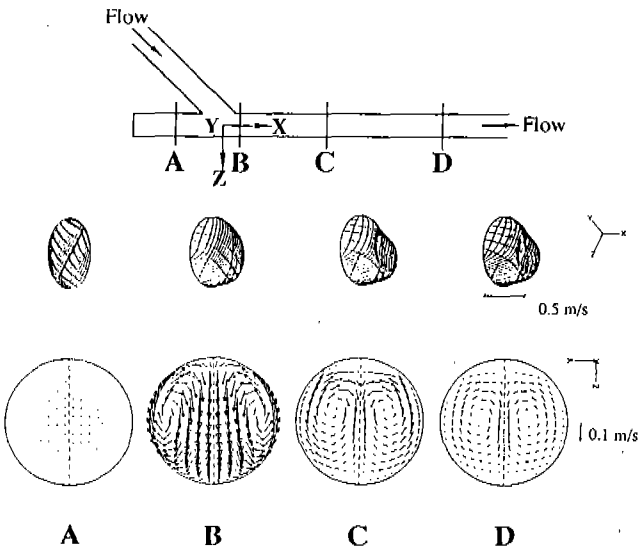

Fig. 3. Axial velocity profiles and secondary flow velocity vectors in steady flow (Re = 510).

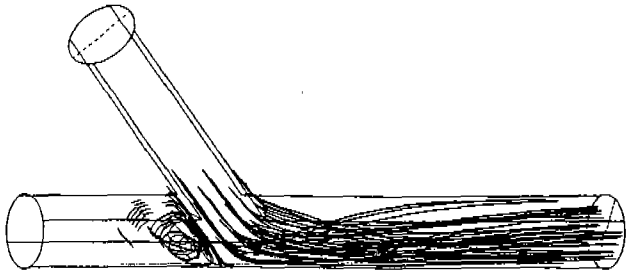

Fig. 4. Streaklines illustrating the double helical vortex.

at the outer wall (ee'). Fluid close to the wall in the anastomotic junction quickly turned around with these helical vortices, while fluid near the center line of the host artery accelerated. Figure 4 shows the streaklines illustrating the double helical vortex in the host artery.

Pulsatile Flow

As shown in Figure 1, the whole cardiac cycle time, T, is 1.0 sec. Figure 5 (a) -5 (f) describe velocity vector fields in the plane of symmetry at the following periods: systolic acceleration t/T = 0.1, peak systole t/T = 0.15, early reversed pressure gradient t/T = 0.3, late reversed pressure gradient t/T = 0.4, mid-diastole t/T = 0.7 and late diastole t/T = 0.9 respectively. At t/T = 0.1, a small vortex was present below the heel region near the outer wall (ee') and axial velocity profiles became skewed toward the outer wall (ee') in the downstream host artery. During peak systole t/T = 0.15, axial velocity profiles became skewed more toward the outer wall and the vortex near the outer wall became even stronger. At this time, axial velocity components at C and D significantly increased due to the secondary flow motion. At t/T = 0.3, only a small amount of forward flow was observed and the vortex near the heel region moved downstream and toward the graft. A widespread flow recirculation was also shown distal to the toe region. At t/T = 0.4, due to the negative pressure gradient, reversed flow occurred from the host artery to the graft and the stagnation point on the outer wall disappeared. Flow close to the outer wall near the anastomosis remained relatively stagnant. Velocity profiles in the graft became also skewed toward the near wall and velocities near the anastomotic unction were relatively small. At t/T = 0.7, only a small amount of flow was observed and some forward flow was found close to inner and outer walls. Flow in the graft turned slowly into the proximal occlusion along the outer wall (ee').

Axial and circumferential wall shear stress distributions along the side wall (cc') are shown in Figure 6. During systole, high axial wall shear occurred just distal host artery with the maximum value of about 12 Pa. Strong secondary flow influenced wall shear stress near the anastomotic region and the circumferential maximum wall shear of 5.5 Pa was found at 0.2 cm from the anastomotic site. At further downstream host artery, the circumferential wall shear decreased dramatically due to the helical motion of flow.

DISCUSSION

To date, the exact etiology of the anastomotic neointimal hyperplasia and thrombosis is unknown. The greatest amount of intimal thickening in a distal graft anastomosis occurred at localized area of low or highly oscillating wall shear stress such as the toe, heel and near the stagnation point. Fluid particles reside in the recirculation zone for a relatively long time, which might result in a higher potential for the mass transfer across the intimal layer to create intimal hyperplasia. Stresses in the high wall shear regions

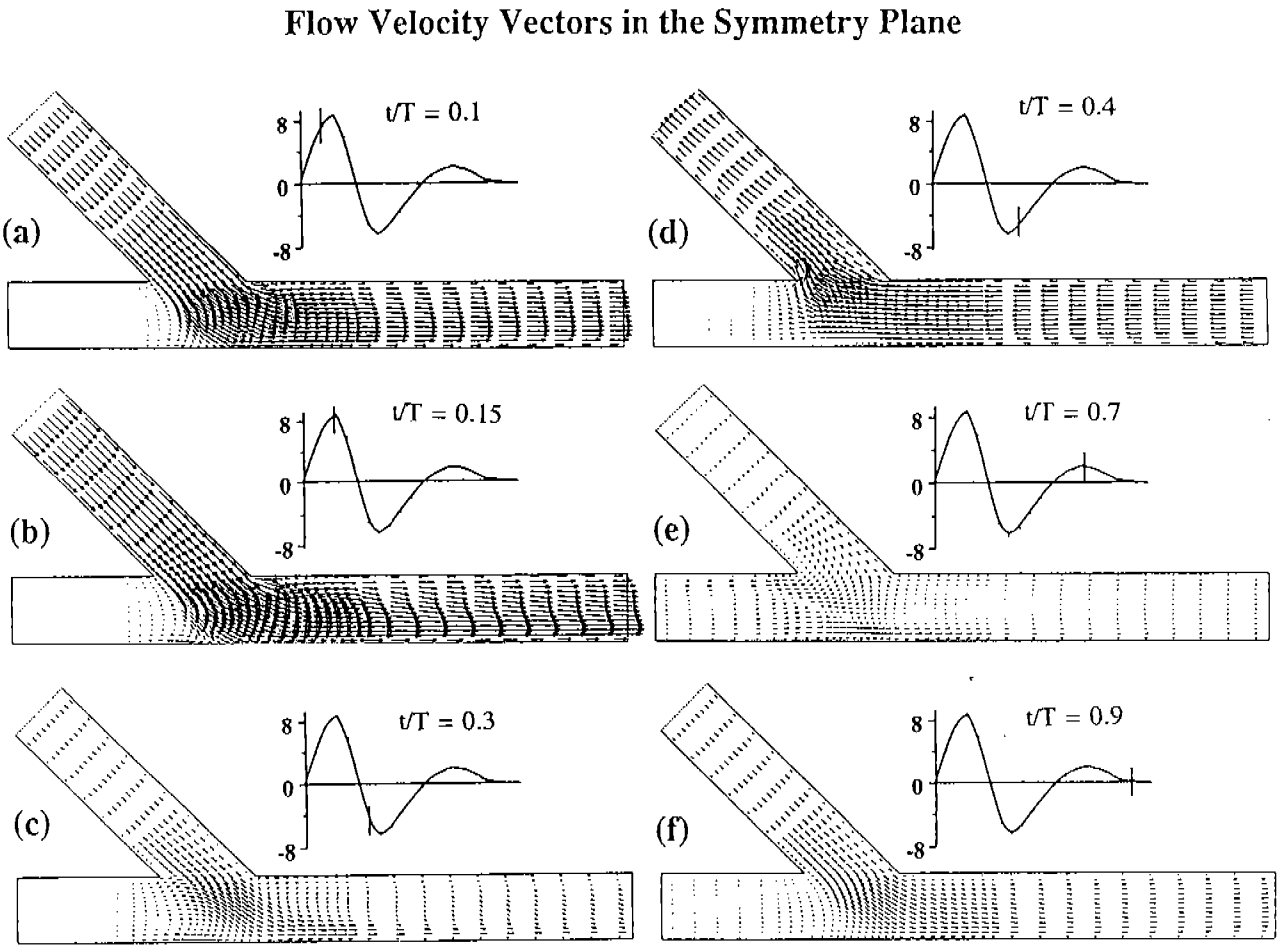

Fig. 5. Pulsatile velocity vector fields in the plane of symmetry.
(a) t/T = 0.1 **(b)** t/T = 0.15 **(c)** t/T = 0.3
(d) t/T = 0.4 **(e)** t/T = 0.7 **(f)** t/T = 0.9

may also induce primary intimal hyperplasia in the low shear regions, by causing endothelial cells in the high wall shear regions to secrete growth factors that are transported and to accumulate in the low flow regions. Fry[8] suggested that deformation, swelling, and eventual erosion of the endothelium might occur at sites where the local shear stress is relatively high. Giddens et al.[9] recently reported that the determining factor of the intimal hyperplasia may be the maximum wall shear experienced during the cycle, regardless of directions and not simply the mean shear value. Secondary flow in the anastomotic region is very important and large circumferential wall shear stresses occurred along the arterial wall. These high circumferential wall shear stresses might deform fibrous tissue on the vessel wall and in turn damage the intimal layer by acting with axial wall shear stresses.

CONCLUSION

Double helical vortex and the resulting secondary motions were found to be very important in flow across the end-to-side anastomosis. Flow recirculations near the toe and heel regions were restricted due to the secondary flow. Pulsatile flow results revealed that oscillating wall shear stresses were observed at regions such as heel, toe and the stagnation point where intimal hyperplasia or thrombosis

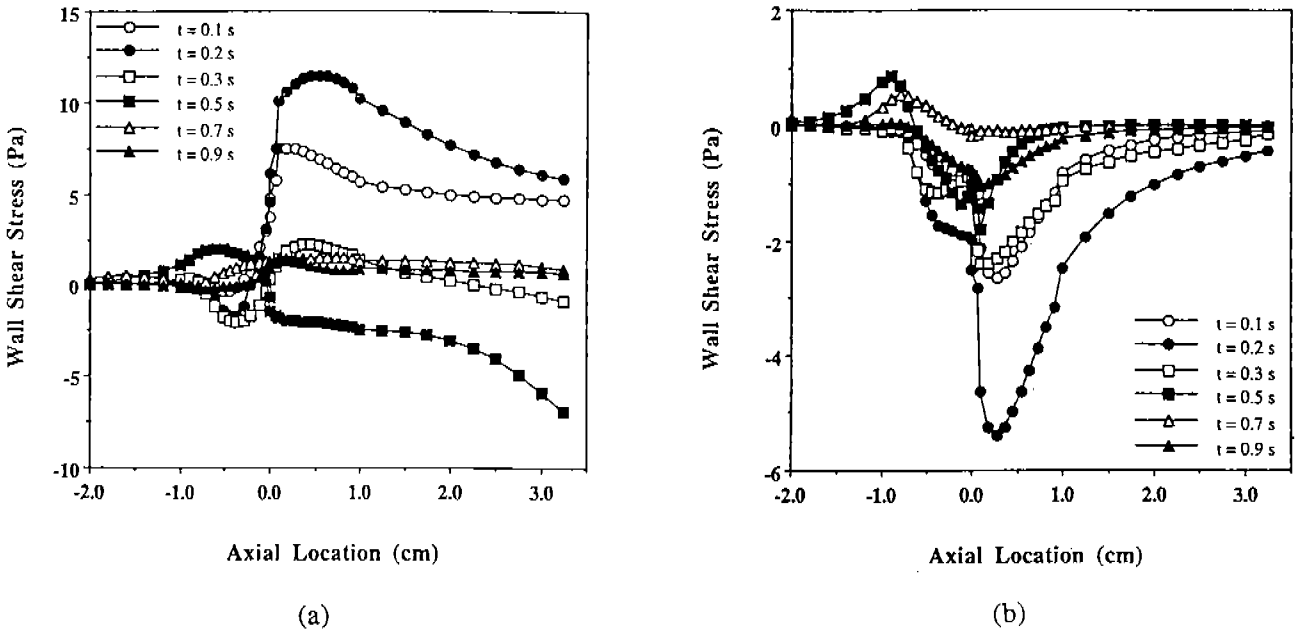

Fig. 6. Wall shear stress distributions along the side wall (cc') in the pulsatile flow.
(a) Axial wall shear stress **(b)** Circumferential wall shear stress

is popular. Large circumferential wall shear stress near the anastomosis might also be one of the hemodynamic sources of graft implantation failure.

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