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EVALUATION OF MODIFIED OSCILLATORY SHEAR INDEX AND RECIRCULATION ZONE IN A DEPLOYED CORONARY STENT

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ABSTRACT

A major consequence of stent implantation is restenosis which occurs due to neointimal formation. There are several factors affecting restenosis among which wall shear stress plays a significant role. The present computational study of developing pulsatile flow through the entrance region of a deployed Palmaz stent in a coronary artery analyzes the local wall shear distribution and its effect on restenosis. A variation from low positive wall shear stress of around 10 dyn/cm^2 at the upstream of stent strut intersection to a very high positive wall shear stress of 300 dyn/cm^2 at strut intersection and then to a negative wall shear stress of -10 dyn/cm^2 at the downstream of strut intersection was observed. Modified oscillatory shear index was calculated which showed persistent recirculation at the downstream of strut intersection indicating it as a highly prone region to restenosis.

INTRODUCTION

Stent implantation improves the arterial blood flow by redistributing the plaque. Though stents are used to increase the arterial lumen diameter and restore blood flow, restenosis could not be completely eliminated which occurs due to neointimal formation. There are several factors affecting restenosis – geometry and size of vessel, and stent design that affects areas of flow recirculation and flow separation and wall shear stress. Although there have been several studies of coronary flow and physiological consequences of restenosis after stent placement, the effect of stent implantation on local wall shear stress distribution as well as other fluid dynamics parameters and its influence on restenosis are not well established.

METHODS

A pulsatile flow analysis for basal (50 ml/min) to hyperemic condition (200 ml/min) is performed for a freshly deployed stent in a human coronary artery having a three dimensional geometry with an axial length of 10 mm and a diameter of 3 mm. The details of the volume mesh [Figure 1A] and the solver used for computational fluid flow analysis were explained in Holcomb et al. (2003).

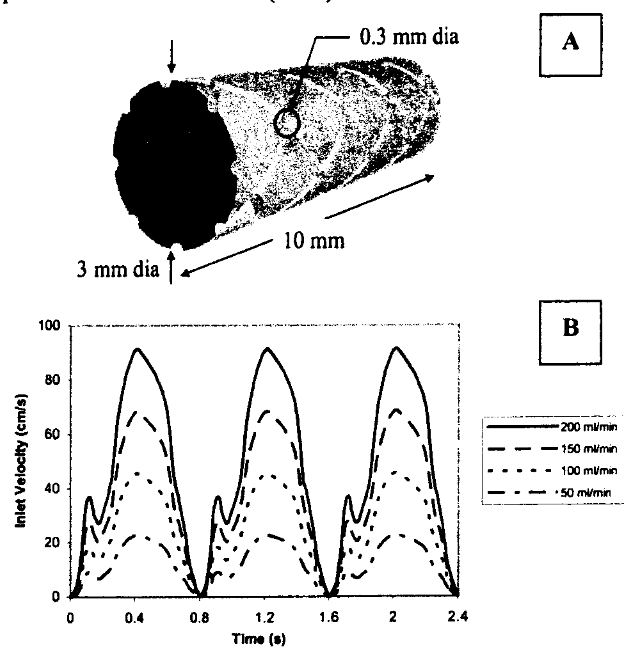

Figure 1: Mesh plot of coronary artery with deployed stent (A). 3 consecutive pulse cycles for basal to hyperemic flows (B).

Time-varying, uniform velocity boundary condition was imposed at inlet with a user-defined subroutine which simulated the worst possible location of the stent in the artery providing an upper bound of desired flow parameters [e.g. shear stress]. This model mimics the stent being placed near the entrance region of a branched coronary artery. The numerical computation was conducted for 3 consecutive pulse cycles [Figure 1B] with a pulse time period of 0.8 s. The results for the 3rd cycle (1.6 s to 2.4 s) are presented here.

RESULTS AND DISCUSSION

Figure 2 shows the axial variation of wall shear stress along the artery wall plotted at various times (acceleration, peak and deceleration) of the pulse cycle for hyperemic flow. Between each strut, in general, the wall shear stress increases in the flow direction from -10 dyn/cm^2 to 100 dyn/cm^2 which is caused by the reattachment of the developing flow. Subsequently, there is a decrease in shear stress to 10 dyn/cm^2 because of flow stagnation at the upstream of strut intersection. Then there is an increase to a very high shear stress of around 300 dyn/cm^2 at the strut intersection with a sharp fall back to a negative shear stress at the immediate downstream of the strut intersection.

Due to the uniform velocity boundary condition at the inlet, the wall shear stress shoots upto a very high value of around 530 dyn/cm^2 near the entrance i.e. at strut intersection 1 and as the flow develops, there is a significant reduction (~55%) of peak positive shear stress to 230 dyn/cm^2 at strut intersection 4.

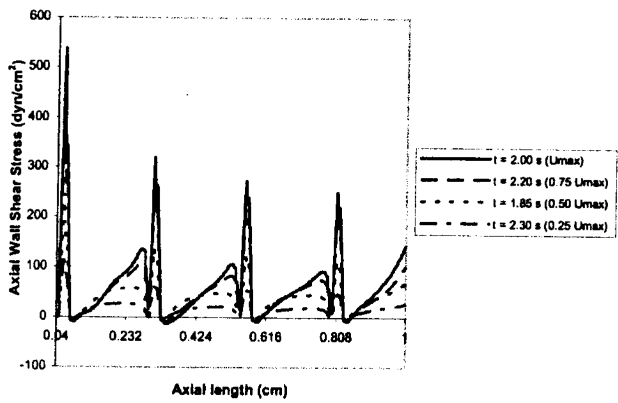

Figure 2: Axial variation of wall shear stress along the artery wall for hyperemic flow

In figure 3B, modified oscillatory shear index (MOSI) is calculated as $\left[\int_0^t \tau_w dt\right] / \left[\int_0^t |\tau_w| dt\right]$ and plotted at vertices 1 to 8 (Fig. 3A) from basal to hyperemic flow rates. MOSI value of 1 implies a positive wall shear stress and -1 implies a negative shear stress during the entire cardiac cycle. From Fig. 3B, it can be inferred that there exists a positive wall shear stress at upstream (vertices 1, 3, 5 & 7) and negative wall shear stress at downstream (vertices 2, 4, 6 & 8) of each strut intersection during the entire cardiac cycle for all flow rates. This implies that there is a persistent recirculation at the downstream of each strut intersection.

Figure 4 shows the variation of recirculation length and height along the struts for basal to hyperemic flows at t = 1.85 s (acceleration). Maximum recirculation of length 0.2 mm and height 0.058 mm occurs near the entrance of the artery i.e. at the downstream of strut intersection 1. As the flow develops from strut intersection 1 to 4, the recirculation length and height decreases.

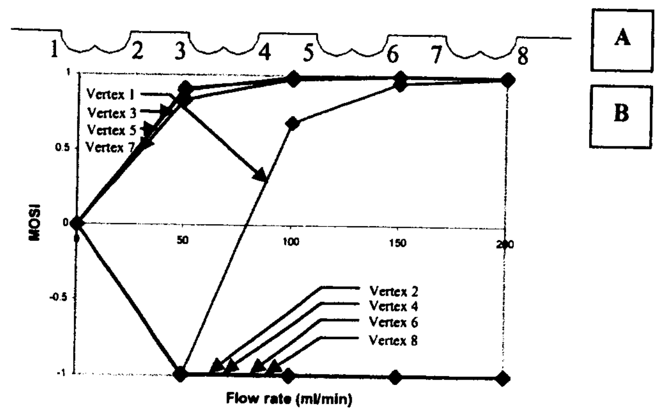

Figure 3: Schematic diagram of the locations of vertices 1 to 8 (Fig. A) and Variation of modified oscillatory shear index at vertices with flow rate (Fig. B)

Figures 2 and 3B show that there is a negative wall shear stress at the downstream, which indicates a predominant region of recirculation zones. Figure 4 shows that significant recirculation zones are formed at the downstream. Essentially, recirculation zones having negative shear values are susceptible to the deposition of macromolecules, e.g., cholesterol, lipoproteins, and other lipid derivatives, thereby may lead to restenosis (Berry et al., 2000; Wentzel et al., 2001).

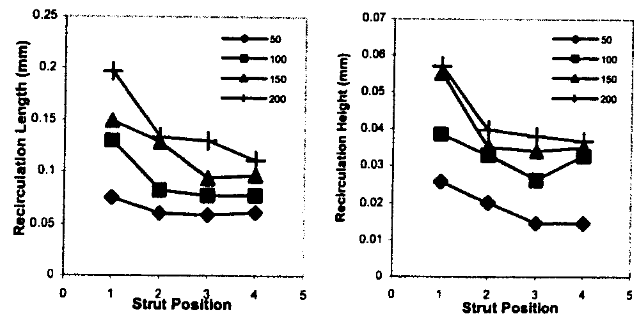

Figure 4: Variation of recirculation length and height near each strut for basal to hyperemic flows at t = 1.85 s

REFERENCES

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