Numerical Investigation of Variations in Wall Shear Stress Distributions at the Diseased Human Carotid Artery Bifurcation

M. NavidBakhsh[i], M.Z. Bidhandi [ii]

ABSTRACT

From clinical practice, it is known that the non-divider side of carotid sinus is very sensitive for the development of atherosclerotic lesions. Because of drastic consequences of a severely narrowed carotid artery bifurcation, there is a need for detecting mild stenosis, less than 25% area reduction, in a very early stage of the atherosclerotic disease. The computerized simulation of the homodynamic behaviour of blood flow in the 3-D model of 25% and 35% stenosed carotid artery with different varying angles and varying mass flow rates at the entrance of CCA has been studied. The fluid flow in the carotid bifurcation is investigated by using finite volume method with a non-Newtonian constitutive model, in which the shear thinning behaviour of blood is described by the Kuang-Luo (K-L) equations. The results show that at the apex of bifurcation junction, because of noticeable changes in blood flow pattern as well as presence of flow separation, recirculation regions, the probability of deposition of atherosclerotic plaque would increase especially on the posterior wall of carotid sinus, due to low wall shear stress. The presence of stenosis will produce two definite regions in ICA, especially near the non- divider side. The first region locates in sinus region which incorporates high WSS and the second region is out of the sinus with low WSS. These considerable variations in WSS and turbulence in downstream of stenosis region can cause a series of non-physiologic phenomena which may ultimately result in brain stroke.

KEYWORDS

carotid artery - stenosis - wall shear stress

1. INTRODUCTION

An enormous number of studies have been carried out to understand the detailed characteristics of blood flow in arteries, particularly at arterial bifurcations [1-3]. Arterial branching is of great interest with regard to atherosclerosis, because of their frequent involvement with deposits. A thorough understanding of hemodynamic associated with flow at vessel ramifications could be useful to unravel some of the mysteries of atherogenesis. Due to the noticeable mortality and morbidity associated with carotid artery disease, it is very important to investigate the detailed fluid dynamics in a carotid artery bifurcation. In the atherosclerosis of carotid artery, the localized deposits and accumulation of cholesterol and lipid substances as well as profilteration of connective tissues, cause a partial reduction mainly in sinus cross-sectional area (stenosis) and the wall stiffness will considerably increase subsequently [2]. As a mild stenosis is developed, the resulting flow disorder plays an important role in further development of the disease. Therefore, many investigators have already performed various experimental measurements and theoretical analysis on the flow through carotid stenoses [4,5]. Experimental techniques including flow visualization, velocity measurements and shear stress measurements have played an important role in obtaining both qualitative and quantitative information about the flow field of interest. Palmen et al. were probably the first who performed Hydrogen Bubble Visualization experiments to analyze the flow in stenosed carotid artery bifurcation models [5]. Flow visualization provides the best overall but the least quantitative picture. Three-dimensional *in*

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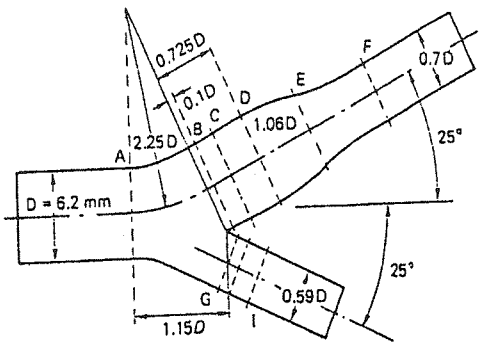
vivo measurements have now become possible through technological developments in the fields of Doppler ultrasound and magnetic resonance imaging (*MRI*). However, measurements of shear stress have proved to be difficult. Although approaches are available, such as direct measurement using a hot - film probe [6] or an electrochemical technique [7], and indirect estimation using an extrapolation method [8], they do not provide accurate information for in vivo conditions. Although experimental studies have provided some useful information to the understanding of blood flow dynamics in a stenosed artery, they are usually invasive, expensive and difficult to carry out or repeat under similar conditions. Hence, researchers have turned to mathematical models to provide solutions to problems relating to blood flow dynamics in arteries [9,10]. Besides these analytical methods, in recent years the advent of high technological digital computers and the availability of computational fluid dynamics (CFD), computer codes have provided researchers with another powerful tool to carry out investigations in this field [11-15]. This study employs a powerful design package so-called Gambit to construct and mesh the carotid model and a commercial CFD code (Fluent 6.5.12) [16] for analysis of blood flow, especially wall shear stress variations in human carotid artery with varying degrees of apex angle and stenosis. Reynolds number, ranging from 257 to 870 has been considered for steady state flow in the carotid artery [17]. Although blood flow in the arteries is time dependent, the current investigation first simplifies it to a steady flow condition, the knowledge of steady flow patterns will provide a basis for understanding the much more complicated flow field expected in pulsatile flow.



a

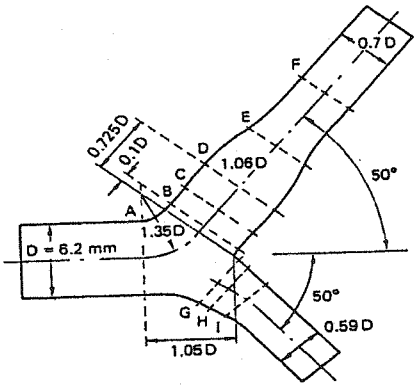


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Fig.1 –Two human carotid artery models. a, CARA1;b,CARA2.

2. STENOSIS MODEL

The carotid artery bifurcation consists of a main branch, namely the common carotid artery (*CCA*), which asymmetrically divides in to two branches, the internal and external carotid arteries. The internal carotid artery (*ICA*) is characterized by a widening in its most proximal part, the sinus or the bulb. The geometrical models considered in this study are based on the model used by the Ku et al. and by Reneman et al. (Fig.1)[18]. The diameter of *CCA*, ($D=6.2mm$), of the *ICA* ($D_i=0.7D$) and of the *ECA* ($D_{ex}=0.59\ D$) as well as sinus diameter ($D_s=1.06D$). The angle between common carotid axis and internal carotid axis are 50° (*CARA1*), 100° (*CARA2*). Two assumptions were made in this model: (a) the arteries and the carotid sinus are circular in cross - section; (b) the parent and the daughter vessels are in the same plane at the bifurcation. Based on these assumptions and geometrical data, first the 3-D numerical grid for a normal carotid bifurcation was generated and then to simulate the stenosis, the study performed by Palmen et al. (1994) was used [5]. According to their study, the main categories of carotid stenosis which frequently locates at the non-divider wall of the carotid bulb can be distinguished: sharp-edged stenoses which are directed either (i) downstream or (ii) upstream and (iii) smooth symmetrical stenoses. The smooth symmetrical stenosis was found to be most frequently presented (44%) followed by the sharp edged downstream directed one (39%). Because of geometrical simplicity, the sharp edged stenosis that was directed downstream was chosen in their study. The geometry chosen for the stenosed outer wall is shown in Fig. 2. In the plane of symmetry, the stenosed wall is formed by two intersecting straight lines. In this model, the top of stenosis is reached at $L_1=5D/4$ and the end of the stenosis is positioned at $L_2=11D/8$, where D represents the diameter of the common carotid artery. For the area reduction, for example 25% stenosis means that $A_2/(A_1+A_2)=0.25$ .The cross section perpendicular to the plane of symmetry is bounded by two arcs, Fig. 2c.

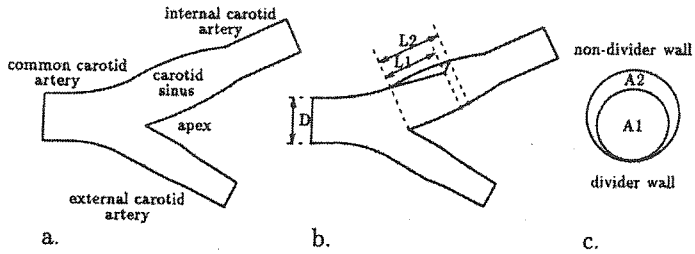

Fig. 2 – Geometries of the non-stenosed (a) and 25%stenosed (b) bifurcation in the plane of symmetry and a cross section of the stenosed carotid sinus(c).

In this stage, Gambit (Fluent Inc.) was used to not only model the stenosis but also to generate the grid on the flow domain. The grid was refined several times to acquire the most appropriate degree of refinement in which the results are mesh independent and further refinement of the grid will not have a significant effect on the characteristics of the flow domain (Fig.3).

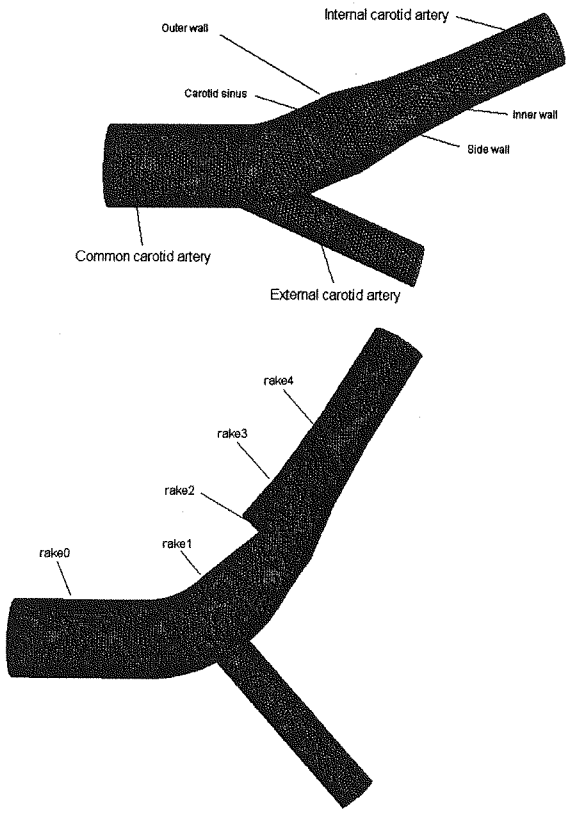

Fig. 3 –Two constructed models of normal and stenosed carotid artery, CARA1, CARC2.

The summarized list of the models studied in this paper, are shown in Table 1.

Table 1- List of the studied models.

Group	model	Apex angle	Pct. of stenosis	No.& Type of cells	No. of Nodes
B	CARB2	100	25%	306161 Tetrahedral	57703
C	CARC2	100	35%	306161 Tetrahedral	57703

3. EQUATIONS

The governing equations are the three-dimensional incompressible Navier-Stokes equations and are given as:

$$\rho\left(\frac{\partial \boldsymbol{u}}{\partial t}+\boldsymbol{u}.\nabla \boldsymbol{u}\right)=-\nabla p+\nabla.\boldsymbol{T} \qquad (1)$$

where $\boldsymbol{u}$ is the fluid velocity vector, ρ the density and p ,the pressure. $\boldsymbol{T}$ is the stress tensor and linearly dependent on the rate of deformation tensor D with a relation of $\boldsymbol{T}=2\eta(\dot{\gamma})D$, where $D=\frac{1}{2}\left(\nabla \boldsymbol{u}+\nabla \boldsymbol{u}^{T}\right)$, η represents the viscosity of the blood, and $\dot{\gamma}$ is the shear rate. For a Newtonian fluid η is a constant and independent of the shear rate $\dot{\gamma}$. The divergence-free condition is imposed on the velocity $\boldsymbol{u}$:

$$\nabla.\boldsymbol{u}=0 \qquad (2)$$

Hence, we consider the blood as a non-Newtonian fluid. As well known, the shear thinning and viscoelasticity of blood are closely relevant to its microscopic structures, e.g., aggregation, deformation and alignment of the red blood cells determine mainly the rheological behavior of blood [19]. Investigations on non-Newtonian steady flow in a carotid bifurcation model indicates that the shear thinning is the dominant non-Newtonian property of the blood and the viscoelasticity may be ignored for the prediction of the velocity distribution (Gijsen et al., 1999a, b)[19,20]. The importance of the viscoelasticity of the blood analogue fluids on the flow phenomena has been presented by Liepsch and Moravec, Ku and Liepsch [19].

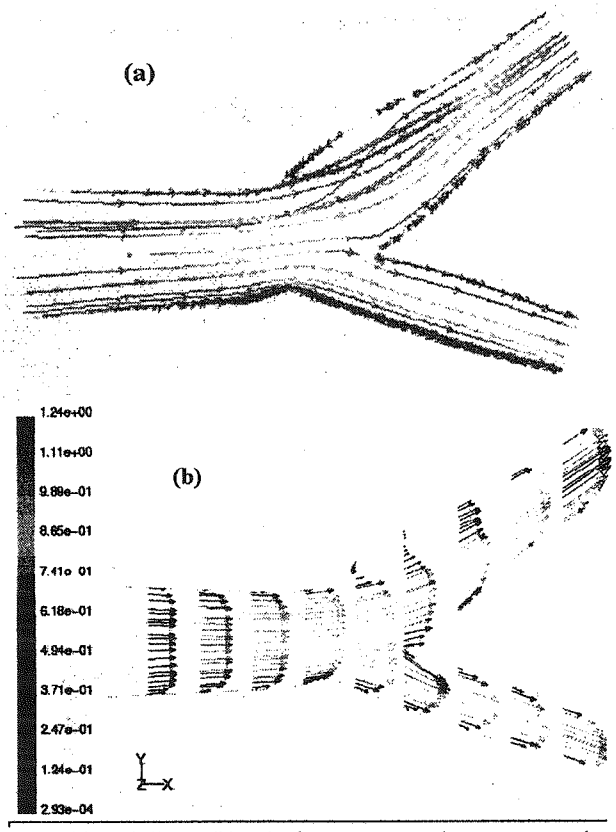

Fig.4 –(a) Path lines -(b) velocity vectors on the symmetry plar of the model by the apex angle of 50° and V_m=0.5 m/s.

In this study, shear thinning is accounted for by using th Kuang-Luo model (Luo, Kuang; 1992) for the viscosit [20]. According to Kuang-Luo studies, the non-Newtonia behavior of blood can be best described by the followin equation;

$$\tau = \tau_y + \eta_2 \dot{\gamma}^{1/2} + \eta_1 \dot{\gamma}, \ \dot{\gamma} \le \dot{\gamma}_c, \tau \ge \tau_y$$
$$\dot{\gamma} = 0, \qquad \tau \prec \tau_y \qquad (3)$$

Where the three parameters τ_y, η_1 *and* η_2 are functions of hematocrit, plasma viscosity and other chemical variables. $\dot{\gamma}_c$ is the critical shear rate beyond which the flow becomes turbulent.

The first two terms of equation (3) are responsible for shear thinning effect of blood, and the third term is the Newtonian contribution.

The apparent viscosity μ_a defined by equation(3) is:

$$\mu_a = \tau / \dot{\gamma} = \eta_1 + \eta_2 \dot{\gamma}^{-1/2} + \tau_y \dot{\gamma}^{-1} \qquad (4)$$

The parameters in the above equation are employed from the experimental data based on $T = 37^{\circ}C, H = 47.6\%$ i.e. $\tau_y = 4.968 mPa$, $\eta_1 = 4.076 mPas$, $\eta_2 = 16.066\, mPas^{\frac{1}{2}}$

4. RESULTS

In Fig. 4(a) the path lines on the symmetry plane of the model of carotid artery bifurcation with the apex angle 50° and is shown. It is observed that at the apex of carotid, the magnitude of velocity vectors is of greater value relative to the sinusoidal region in *ICA*. Fig. 4(b) shows the velocity vectors of normal carotid artery. It is seen that by traveling from the center line of the *CCA* toward the exit of *ICA* and *ECA* the velocity has a growth which is more tangible at the *ICA*.

As well known, the wall shear stress is the tangential force caused by the flow of blood along the surface of the endothelium. It is believed that the atherosclerotic plaques form in the regions of low wall shear stress and disturbed flow. Using both *in vivo* and *in vitro* models, it has been demonstrated that vessels, which are prone to plaque formation (e.g., carotid artery), have the flow pattern characterized by low wall shear stress, flow separation and stasis, and oscillations of flow [22]. The appearance of low wall shear stress appears to coincide with early intimal thickening in the carotid artery.

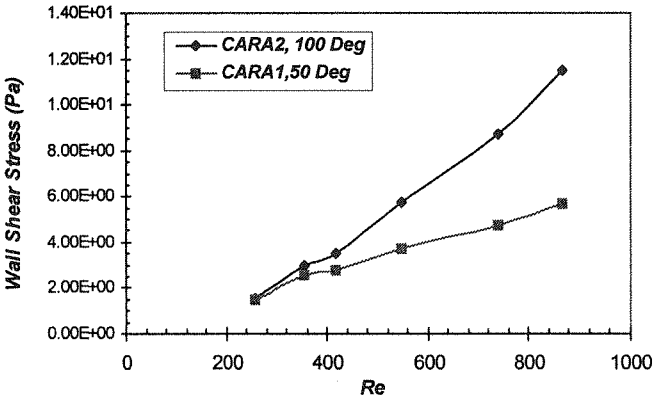

Fig. 5 –Wall shear stress variations on the apex in different velocities for two models CARA1, CARA2.

According to the calculated results, the velocity is skewed toward the flow divider and the low wall shear stress is found at the non-divider walls of carotid sinus. These behaviors are consistent with the previous findings (Caro et al., 1978; Pedley, 1980). Generally the configuration and branch angle of the internal carotid sinus produce an area of low wall shear stress and alter hemodynamics along the outer wall of the vessel, where atherosclerotic plaques tend to be formed. For instance, Fig. 5 demonstrates the effect of the branch angle on wall shear stress variations in the apex. With the increase of bifurcation angle from *CARA1* to *CARA2* and constant blood velocity, wall shear stress would increase and also enhanced flow recirculation and increased reversed flow would occur. From the clinical point of view, it is known that large angle bifurcations have unfavorable characteristics in the development of atherosclerosis. To investigate the effect of stenosis on wall shear stress distributions, the spatial variations of wall shear stress,

Fig. 6 has been plotted for different walls (i.e., downstream and upstream of the stenosis and also on the stenosis wall) and different inlet velocities.

The presence of stenosis will produce two different regions near the outer wall of internal carotid artery. The first region locates in carotid sinus which incorporates high wall shear stress and the second one would be in the outside of sinus region which includes low wall shear stress, the most significant effect of stenosis. Fig. 6b shows the flow recirculation downstream of stenosis and a very low wall shear stress on the wall just after the recirculation zone. Fig. 6c indicates the fact that wall shear stress has a high magnitude on the tip of the stenosis. On one hand, recirculation downstream of the stenosis alters the shear distributions on the endothelium in post stenotic regions and on the other hand, stress fields induced by the flow can also cause fracture of the plaque, which could further result in embolization and occlusion of arteries and finally arterial collapse. The occurance of flow separation can be observed in Fig. 6d.

Fig. 7 shows the effect of percent of stenosis. Generally, it is observed that in a definite inlet velocity of the blood to *CCA*, the variations in wall shear stress on the *CCA* wall (rake 0) and the distal wall of *ICA* (rake 4) would have an approximately uniform variation. Meanwhile, the increase in stenosis area would cause the wall shear stress reduction in the first surface of the stenosis and on the contrary, wall shear stress increase on the second surface of the throat area (stenosis).

Thus, it is very important that both bifurcation angle and percent of stenosis should be considered as a whole when interpreting the carotid angiograms, because bifurcation angle augments the increased resistance and wall shear stress due to stenotic lesions.

It is also established that even for mild stenosis when occurring at high curved sections, it can produce worse effects than for higher percentage stenosis occurring on a straight tube.[22]

5. CONCLUSIONS

This study concentrates on three-dimensional hemodynamics and wall shear stress for human carotid artery bifurcation under average conditions. It is observed that at the apex of bifurcation junction, due to changes in blood flow pattern, flow separation, recirculation regions, the probability of deposition of atherosclerotic plaque especially the posterior wall of carotid sinus in *ICA*, will increase as result of low shear stress. The results show that the pressure at the stenosis area is lower.

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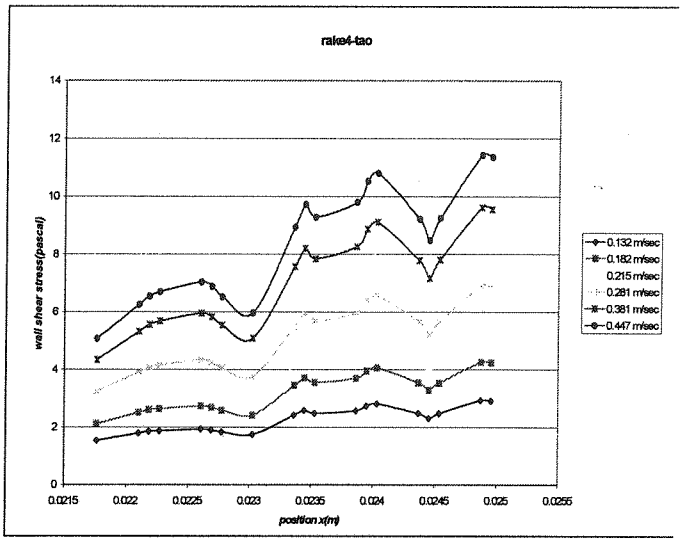

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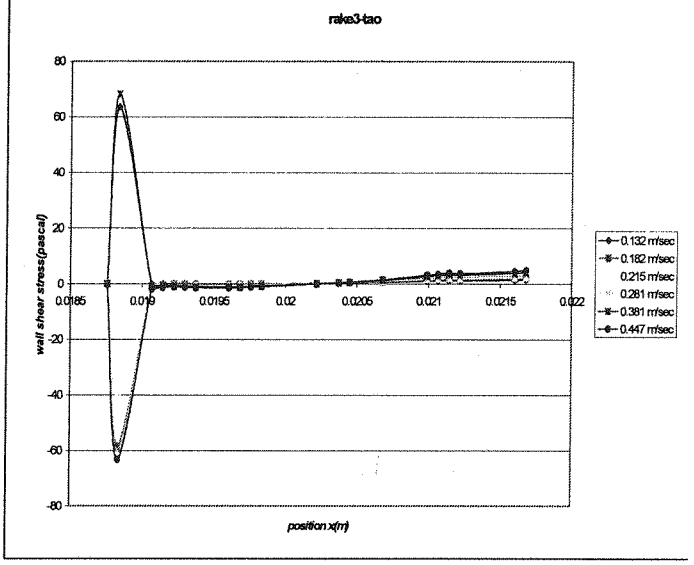

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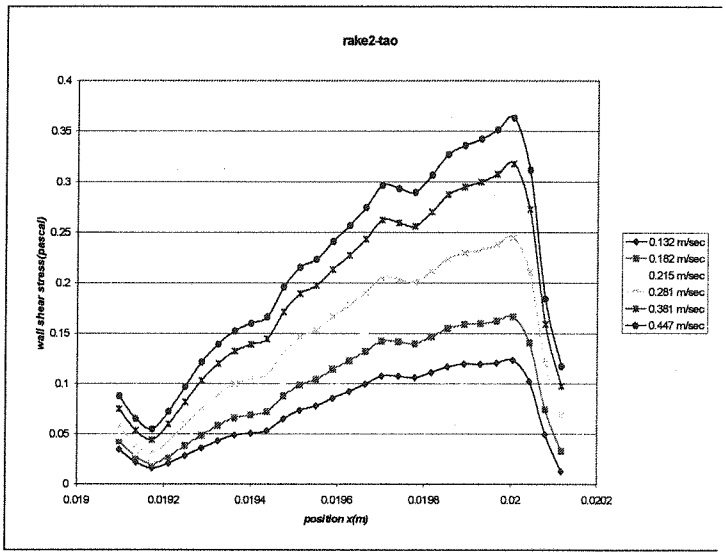

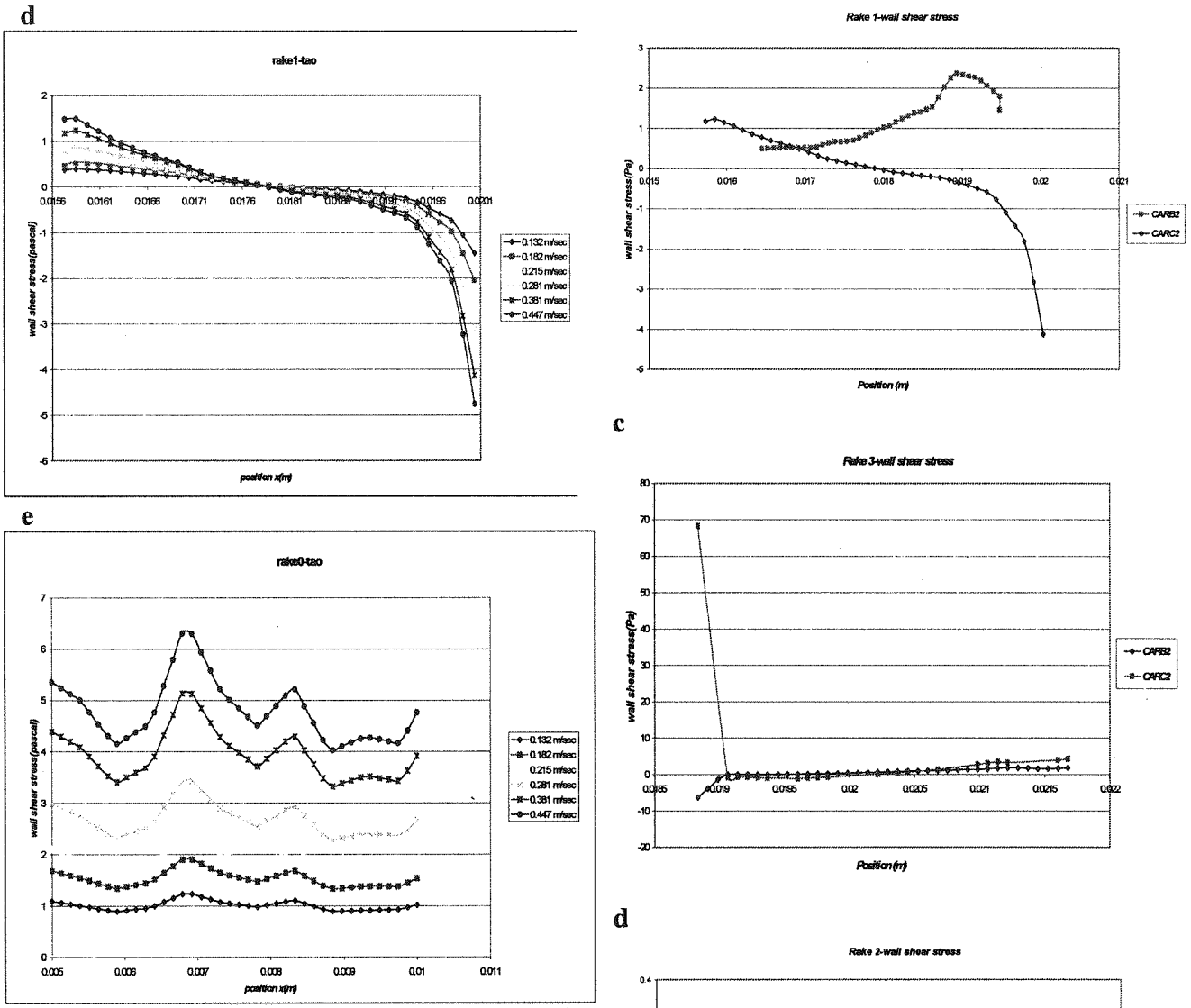

Fig. 6 – Spatial variations of wall shear stress in different walls of stenosed carotid artery (CARC2); a to e.

e

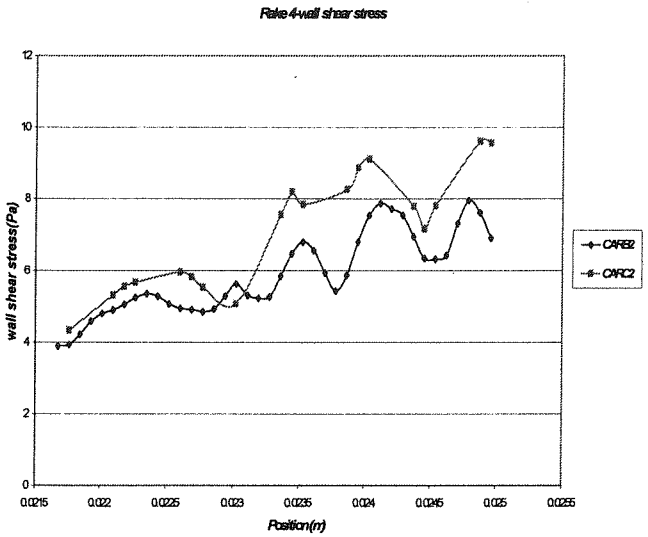

Fig. 7 – Spatial variations of wall shear stress in different walls of carotid artery with different degree of stenosis CARB2, CARC2, a to e.

Although the effect of percent stenosis on pressure drop is more significant, the curvature and stenosis should be considered as a whole when interpreting carotid angiograms. The presence of stenosis will produce two definite regions in IC. The first region locates in sinus area incorporating high WSS, while the second region is out of sinus region including low WSS. The most significant effect of stenoses with high percentage is the increase of WSS in sinus area and creating regions of low WSS in downstream of stenosis. The variability in shear stress can also result in a predilection towards atherosclerotic.

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