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A COMPARISON OF 2D PULSATILE HEMODYNAMIC ANALYSES WITHIN A STENOSED CAROTID ARTERIAL BIFURCATION AND ITS NORMAL COUNTERPART

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ABSTRACT

A 2D pulsatile hemodynamic analysis based on the finite element technique was performed on a minimally stenosed carotid artery and its "normal" counterpart to isolate the contribution of the stenosis to complex flow. The interpretations were based on velocity profile, wall shear stress, and streamline data provided by the hemodynamic analysis. This information may be helpful in imaging modalities such as magnetic resonance.

INTRODUCTION

The present analysis is based on the x-ray depiction of a minimally stenosed carotid angiogram and its normal counterpart. Narrowings in the artery are referred to as stenoses. The goal is to isolate the effect of the stenosis on the flow pattern through a comparison of the velocity profile, shear stress, and streamline data for the normal and stenotic cases. To date, hemodynamic analyses on the carotid artery have been based mostly on geometrical simplifications rather than on the exact shape of the artery[1-6]. In fact, the effect of small stenoses in bifurcations has not been extensively studied in the literature[6]. Stenoses and bifurcations usually occur in close proximity[7,8] and are both associated with secondary flows and zones of flow recirculation[9]. It is important to determine the stenotic contribution to complex in-vivo flows. A comparison of the flow distributions within the stenotic artery and its normal counterpart, will enable us to isolate and understand the effects of stenosis.

The 2D pulsatile flow analysis is based on the finite element technique. The generic finite-element code FIDAP is used for the simulations. The x-ray depiction of the artery constitutes the basis for the rigid arterial geometry. The Doppler ultrasound spectral velocity recording of the patient is used for the pulsatile velocity waveform specification. Blood is modelled as a non-Newtonian fluid with the viscosity determined by the Carreau model[10,11]. The velocity profile, shear stress, and streamline data acquired from the hemodynamic analysis provide information on the flow distribution. The results are validated through different numerical convergence criteria and through shear stress comparisons.

The hemodynamics of the carotid artery have been of great interest to the physician as well as to the fluid analyst, partly due to the propensity of plaque build-up in the artery and partly due to the severe ramifications of having a clot in this artery. The studies so far have focused mostly on the cause of plaque development.The experimental studies of Zarins et al[12] have associated plaque development with low wall shear stress. The studies of Ku et al[2] and Balasubramanian et al[13] have attributed the cause of plaque development to low and oscillating shear stress. Our goal is not to detemine the cause of stenosis, but rather to isolate its effect on the flow pattern, to determine how it may interfere with the imaging of the carotid artery, in modalities such as magnetic resonance which are known to be sensitive to velocity gradients. The two-dimensioanal pulsatile flow studies of Van de Vosse et al[6] and Nazemi et al[8] have found that recirculating zones increase during periods of deceleration. Flow reversal regions separate from the wall on the non-divider side and additional flow reversal zones develop on the side of the flow divider. A three-dimensional steady flow study of Rindt et al[5] has shown that the carotid geometry produces significant secondary flows. In neither of these studies however, the distinction between the contribution of stenosis and bifurcation is made. Since two-dimensional time-dependent analyses sufficiently provide the the cut-away view of the 3D arterial flow along the centerline, we have conducted our study as two-dimensional pulsatile flow

substantially saving on computer storage space and CPU time[14]. Our comparison of the stenotic and normal geometry helps isolate the contributon of stenosis.

METHODS

The time-dependent flow distribution of an incompressible non-Newtonian fluid within a specified geometry is described by the equations of conservation of mass and momentum[11]:

$$u_{j,j} = 0 \tag{1}$$

$$\rho\left[\frac{\partial u_i}{\partial t} + u_j u_{i,j}\right] = - P_{,i} + \left[\eta \cdot (u_{i,j}+u_{j,i})\right]_{,j} + \rho f_i \tag{2}$$

where u_i denotes the ith component of the velocity vector, P refers to the pressure, and η is the viscosity. The viscosity is determined by the non-Newtonian Carreau model[10],

$$\eta = \eta_\infty + (\eta_0 - \eta_\infty)\frac{1}{1 + (\lambda\dot{\gamma})^m} \tag{3}$$

where the characteristic time l is 3.313 s, the power-law index n is 0.3568, the zero-shear-rate viscosity η_0 is 0.56 poise, the infinite shear-rate-viscosity η_∞ is 0.0345 poise, and $\dot{\gamma}$ refers to the local shear rate. The flow distribution is determined by solving the equations for the flow domain defined by the geometry of the artery. The digital subtraction angiographic (DSA) representation forms the basis for the arterial geometry. Figure 1a is the x-ray depiction of the stenotic portion of the carotid angiogram. Figure 1b is the corresponding computer mesh representation. The stenosis corresponds to a 15% reduction in the cross section of the conduit immediately prior to the bifurcation. Figure 1c is the mesh representation for the presumed normal counterpart of the artery, acquired by redefining the vessel boundary at the stenotic site. For both arteries, the mesh sizes along the walls and prior to the bifurcation are made smaller, to minimize numerical error due to large velocity gradients.

FIG. 1a

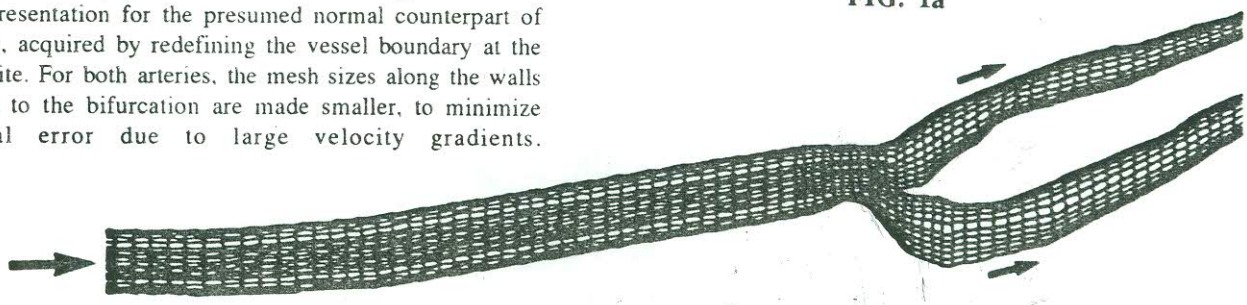

FIG. 1b

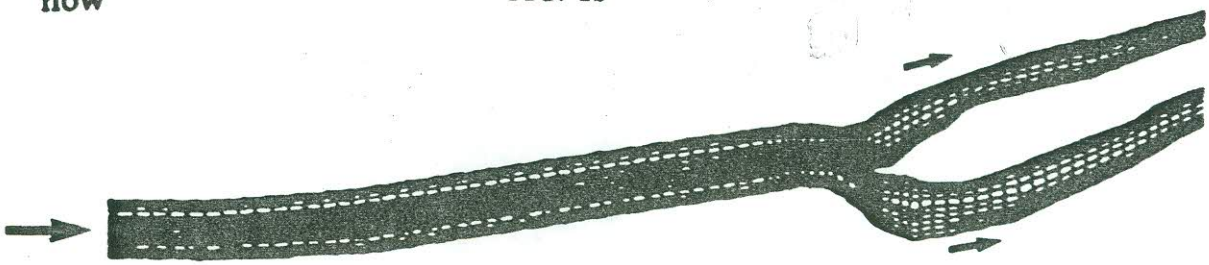

FIG. 1c