BIFURCATION ANGLE AND STENOSIS INFLUENCE ON CAROTID ARTERY HEMODYNAMICS

Tej Jolly

INTRODUCTION

Atherosclerotic plaque formation is strongly linked to the hemodynamic parameter wall shear stress (WSS). Atherosclerotic plaque forms in areas of the vasculature subject to low WSS. The carotid bifurcation produces regions of both low and high wall shear stress.

Because the form of the carotid artery is broadly consistent, hemodynamic studies on a generalized carotid model are useful for developing a map of hemodynamic variables of clinical interest, e.g., wall shear stress, pressure, residence time, oscillatory index. Such studies have shown that the bifurcation region is more susceptible to the hemodynamic factors that contribute to atherosclerosis [1]. Although the carotid configuration is broadly consistent, geometric variations do occur in bifurcation angle and diameter ratios. Furthermore, plaque is simulated as a stenotic narrowing. The current study varies bifurcation angle and stenosis severity to quantify their impact on carotid hemodynamics, bridging generic models and patient-specific assessment.

METHODS

GEOMETRY The same generalized model of the carotid artery used in a previous study [1] was used in this study as the base model to which geometric variations and a stenotic narrowing would be applied. A 3-D model of the carotid artery comprising the common carotid artery (CCA), the external carotid artery (ECA), and the internal carotid artery (ICA) was created in Rhino (version 8).

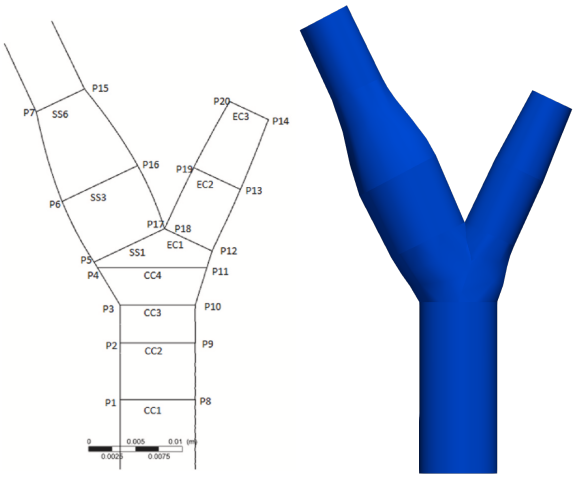

Figure 1 (a) – a top-down diagram of the model used and (b) a top down view of the Rhino model

The base model was varied to increase the angle of the bifurcation and to introduce a stenosis. The bifurcation angle, α, of the base model is 50°. The bifurcation angle was increased from 50° to 70° in another model. The stenosis narrows the ECA lumen diameter by 30% at its most severe point.

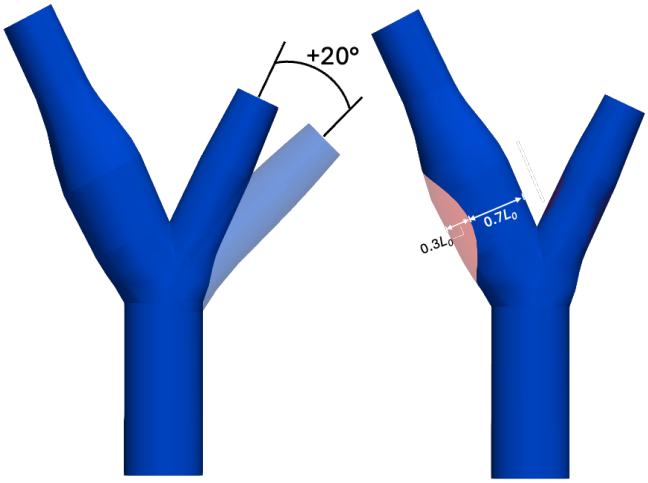

Figure 2 - (a) bifurcation angle variation and (b) stenosis variation

MESH The model was discretized into a mesh using the SimVascular (Version 4.2.2) mesher. The mesh consisted of 1.7 x 10^5 tetrahedral volumes. Three boundary layers at half the length of a cell and a reduction rate of 80% were applied at the vessel wall to better resolve near-wall hemodynamics. Element size near the bifurcation and stenosis was reduced relative to the average element size in anticipation of more complex flow patterns near those two areas.

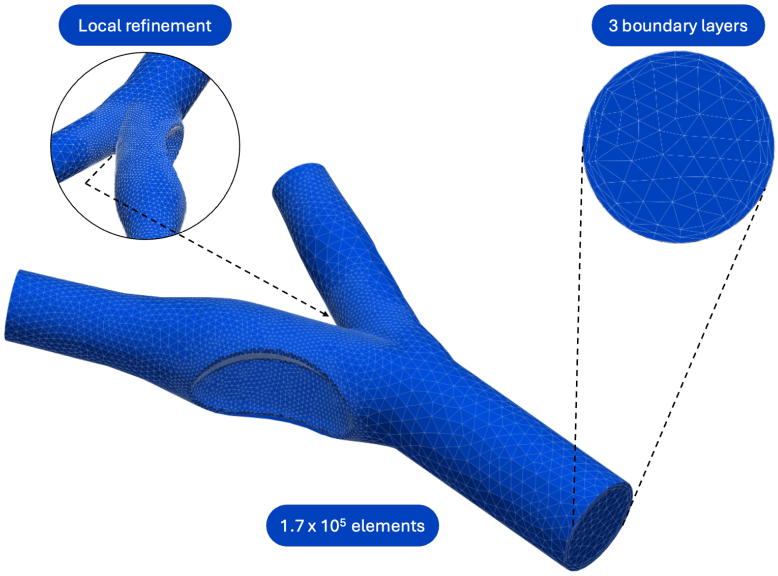

Figure 3 - Meshed of the carotid artery

BOUNDARY CONDITIONS A Dirichlet boundary condition specifying the velocity normal to the face and into the model domain was imposed on the face of the CCA. Resistance boundary conditions were applied at the faces of the ICA and ECA. Vessel walls were assumed to be rigid and no-slip.

A previously published velocity waveform (period = 0.857 seconds) was prescribed to simulate pulsatile flow [2] . A parabolic flow profile was applied at the inlet.

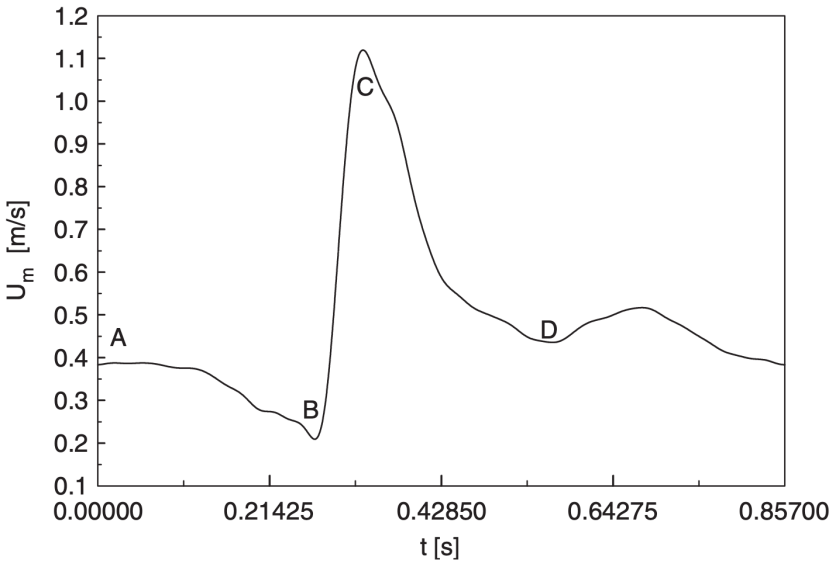

Figure 4 – velocity waveform at the inlet of the model

FLUID PROPERTIES Blood was simulated with the following properties:
Viscosity model: Newtonian
ρ = 1050 kg/m3 [1]
μ = 0.0035 kg/m-s [1]

EXPERIMENTS Four models are developed as combinations of models with α=50°, α=70°, a 30% stenosis, and an non-stenotic geometry. The first experiment, which uses the base model (α=50°, non-stenotic) was used to calibrate the resistor boundary condition values so that (a) outflow would match a previous study, i.e., 70% of the outflow to the ECA and 30% to the ICA, and (b) a peak systolic pressure at the inlet of 120 mmHg [1]. The simulations ran for four cardiac cycles to allow the solver to reach equilibrium with respect to velocity and pressure compared cycle to cycle.

RESULTS

The model outlet balance was calibrated to within 1.5% of the target outflow values when averaged with respect to time over one period. The inlet pressure was calibrated to within 1% of the target inlet peak systolic pressure value. Note that the minimum inlet pressure falls far below a realistic physiological value to nearly 20 mmHg.

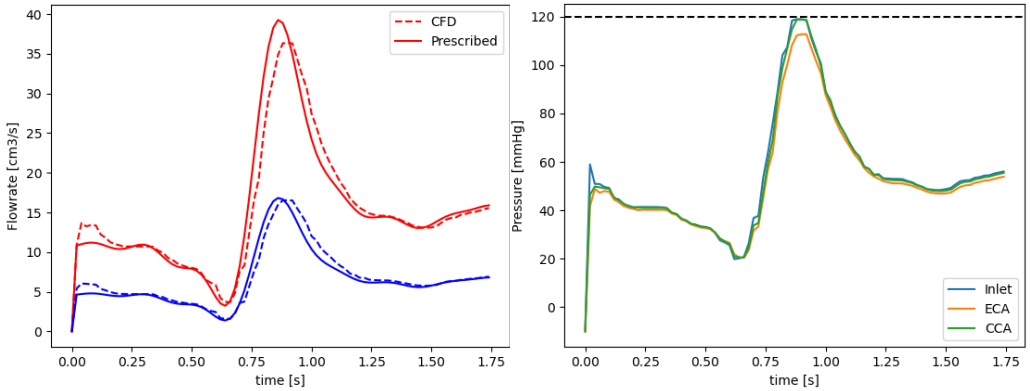

Figure 5 – (a) target flow splits versus simulated flow splits and (b) target peak inlet pressure and pressure waveform

The normalized area of the model under high WSS (> 70 dynes/cm^2) at the time of peak systolic flow, t_1, (point B in Figure *x*) and the normalized area of the model under low WSS (< 5 dynes/cm^2) at the time of a local minimum of systolic flow, t_2, (point D in Figure *x*) were measured for each simulation.

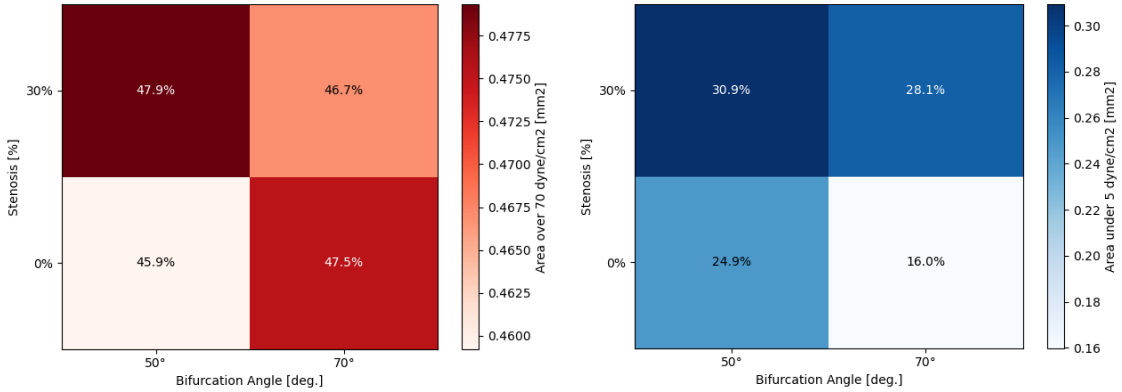

Figure 6 – Heatmaps of the normalized surface area subject to (a) high WSS and (b) low WSS

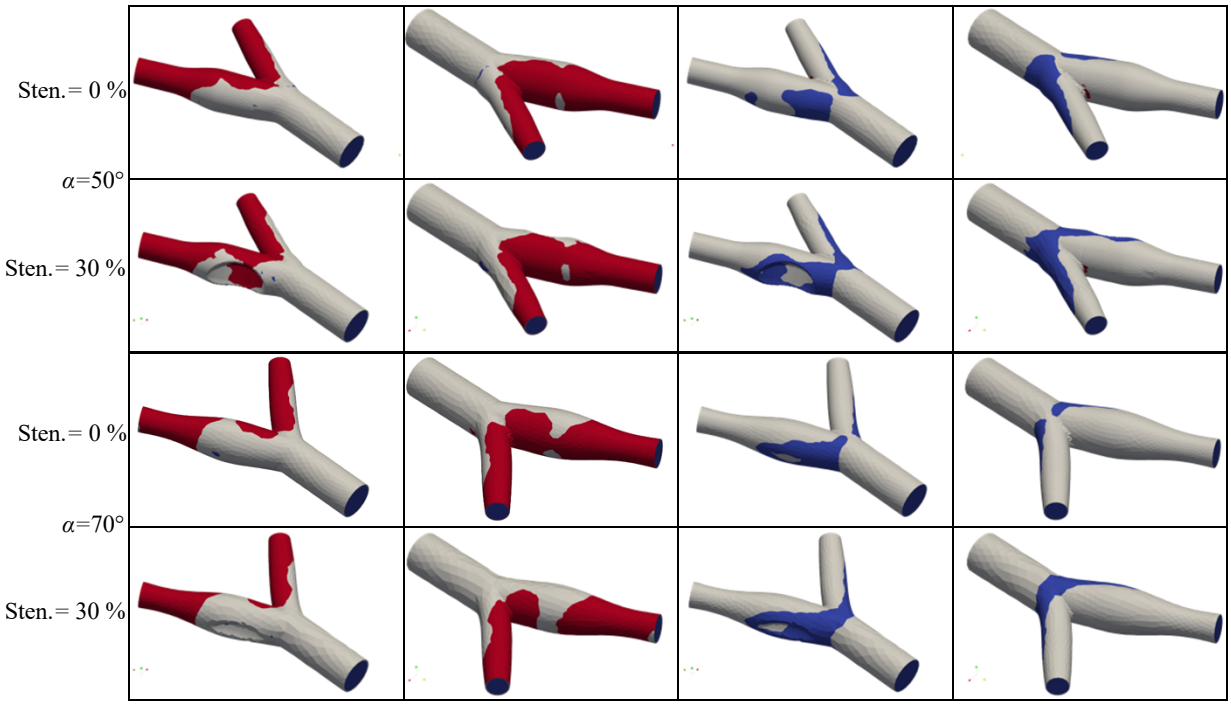

Figure 7 – Isometric views (front and back) of areas of high WSS (red) at time t_1 and low WSS (blue) at time t_2.

DISCUSSION

The results show that, for a given bifurcation angle, the presence of stenosis increases the normalized area subject to low wall shear stress by roughly 10%. Furthermore, for a given stenosis, increasing the bifurcation angle reduces the area subject to low wall shear stress.

Aside from these two findings, the results show contradictory trends. In the case of an non-stenotic artery, increasing bifurcation angle slightly increases the area subject to high WSS; however, for the stenotic artery, increasing the bifurcation angle slightly decreases the area subject to high WSS. Similarly, at a bifurcation angle of 50°, introducing a stenosis increases the area subject to high WSS, but *vice versa* for the 70° case.

The results above are dubious and require a more rigorous reproduction with respect to mesh element sizing and sample size. Due to an act of God, computing resources were limited, and the current mesh was the most granular mesh that could be computed in a reasonable time. It is suspected that the current results are not mesh independent.

The small number of samples means that any trends or mathematical relationships derived from the data will have a p - value $\gg 0.05$. However, if the current results happen to be mesh independent, the results of the current study are useful as indicative of a general trend for low WSS vis-à-vis bifurcation angle and stenosis.

Several aspects of the current model stand out as those that might be improved upon or extended in a future study. The boundary condition at the outlets should be replaced with a multiple-element Windkessel to improve fidelity to physiological values, especially with respect to the pressure waveform. Additionally, a fluid-structure interaction study may be valuable in investigating the interplay of pressure and wall-distensibility and their effects on the rest of the hemodynamics of the vessel.

BIBLIOGRAPHY

[1] Bashar MdS, Hossain R, Rahman MdH, et al. CFD analysis of non-Newtonian blood flow through human carotid artery bifurcation: Carotid sinus susceptible to atherosclerosis. *Heliyon* 2024; 10: e40286.

[2] Valencia A, Zarate A, Galvez M, et al. Non-Newtonian blood flow dynamics in a right internal carotid artery with a saccular aneurysm. *Int J Numer Methods Fluids* 2006; 50: 751–764.