

FLUID-STRUCTURE INTERACTION MODELING OF BLOOD FLOW IN A NON-STENOSED COMMON CAROTID ARTERY BIFURCATION

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ABSTRACT

The rupture of atherosclerotic plaques is known to be associated with stresses that act on or within the arterial wall and is one of the leading causes of mortality and disability worldwide. In this study a fluid-structure interaction modeling of blood flow in a non-stenosed common carotid artery bifurcation is carried out using vascular geometries reconstructed from Doppler ultrasound images. Comparison of results between rigid wall model and fluid-structure interaction model is presented.

Keywords: carotid artery bifurcation, blood flow, fluid-structure interaction.

INTRODUCTION

Carotid atherosclerosis is the process of vascular wall injury resultant from the build-up deposition of fatty substances, which cause the formation of plaque and consequently stenosis. Stenosis alters blood pressure, resistance, and blood flow can lead to critical flow alterations such as high flow velocities, high shear stress, cyclic artery compression and flow recirculation. These conditions may be linked to thrombus formation and excessive artery fatigue leading to stroke (Wootton, 1999). The rupture of carotid plaques is a major cause of cerebrovascular thrombotic events, including transient ischemic attacks and stroke and the mechanism for plaque rupture is not fully understood. Coupling solid mechanics models with computer fluid dynamics codes it is possible to determine flow and wall stress in arteries with branches, stenoses and plaques allowing investigate the effect of stresses on carotid plaques.

Fluid-structure interaction (FSI) analysis has been employed as a powerful tool to enhance our understanding of how hemodynamic factors are involved in atherosclerotic disease. It combines blood flow simulation through computational fluid dynamics with wall stress study. Li et al. investigation (Li, 2006; Li, 2007) correlate stress levels with inflammatory activity in the plaque. Recent studies (Tang, 2005; Gao, 2009) suggest that a local increase in stress/strain could be a cause of plaque rupture, and that stress in the plaque region can be used for plaque rupture risk assessment.

A patient specific 3D fluid-structure interaction (FSI) simulation is carried out using ANSYS commercial software throughout its coupling system feature. The main purposes of this study are to investigate the hemodynamic characteristics on the distribution of wall shear stress (WSS) and flow patterns induced by the arterial wall deformation. Rigid and FSI models of a healthy common carotid artery (CCA) bifurcation are presented and compared.

METHODS

Model definition was performed using a set of Doppler images of the CCA, its bifurcation and proximal segments of internal (ICA) and external (ECA) carotid arteries acquired during daily medical routine. The longitudinal and transversal set of B-mode images was recorded at end-diastole, to control physiologic variations of diameter within cardiac cycle. The segmentation of ultrasound images was obtained using an in-house semi-automatic algorithm implemented in MATLAB software (Henriques, 2015). The segmentation method is based on the hypoechoic characteristic of the lumen and allows the lumen contour extraction in 2D B-mode images.

To establish a computational geometry of the arterial region of the CCA bifurcation, 2D smooth lumen contours were placed in the axial direction according to each image location obtained during data acquisition (Fig. 1). After smoothing of the obtained surface, three diameters cylindrical extensions upstream and downstream of the carotid bifurcation were performed, in order to minimize flow modeling inaccuracies due to the imposition of the boundary conditions.

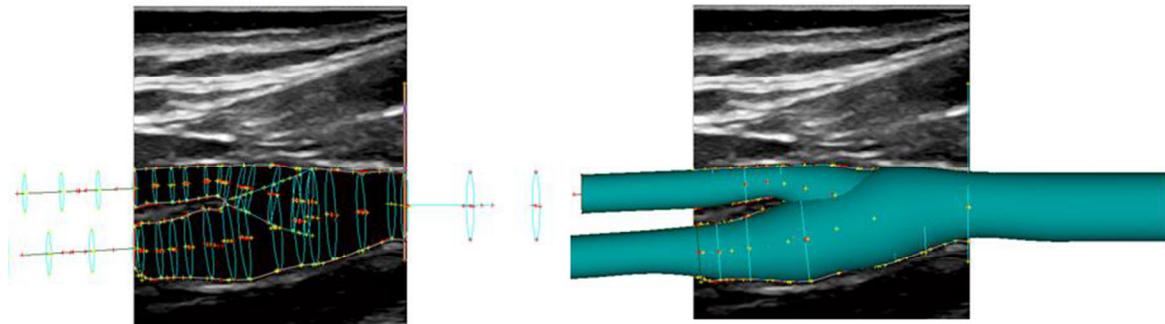


Fig. 1 - 3D surface generation

Blood flow simulation was obtained iteratively, using the SIMPLE algorithm and a second-order upwind scheme for the convective terms. Blood was considered an incompressible homogeneous Newtonian fluid, with a density of 1060 kg/m^3 and dynamic viscosity of 0.0035 N.s/m^2 . The vessel lumen is approximately circular at the CCA inlet and an equivalent circular radius was calculated in order to impose a Womersley velocity profile based on Doppler ultrasound measurements (Sousa, 2014).

A proposed-developed code implemented in MatLab software was used to calculate time-dependant Womersley profile. For outflow boundary conditions a constant in time flow rate division between ICA and ECA was defined, equal to $\text{ICA/ECA} = 60/40$.

The arterial wall was modeled as an isotropic elastic material with a Young's modulus of 1.06 MPa , a Poisson's ratio $\nu=0.45$ and a density equal to 1120 kg/m^3 . The thickness of the blood vessel wall was considered equal to 1 mm (Mulani, 2015). All nodes on the inlet and outlet planes were constrained in the local direction of the lumen centerline direction. In order to prevent possible rotation of the entire model, which could increase convergence time, one

edge of one element was fully constrained at the inlet and outlets. The remaining nodes were left free to undergo displacement in any direction (Zhao, 2000). FSI simulation was performed in an Intel Core I3 computer of 2.4GHz processor and 4GB of RAM.

Three cardiac cycles were simulated considering a constant time step equal to 0.008 seconds. The flow velocities and the WSS vectors obtained from the last cycle were recorded for post-processing. Mesh density sensitivity analyses were performed on both fluid and solid domains until stress differences between solutions from two consecutive meshes were negligible (less than 2.5% of the absolute value).

An iterative solution (Fig. 2) between fluid solver (FLUENT) and structural solver (Transient Mechanical) is used until convergence is attained (Khader, 2014).

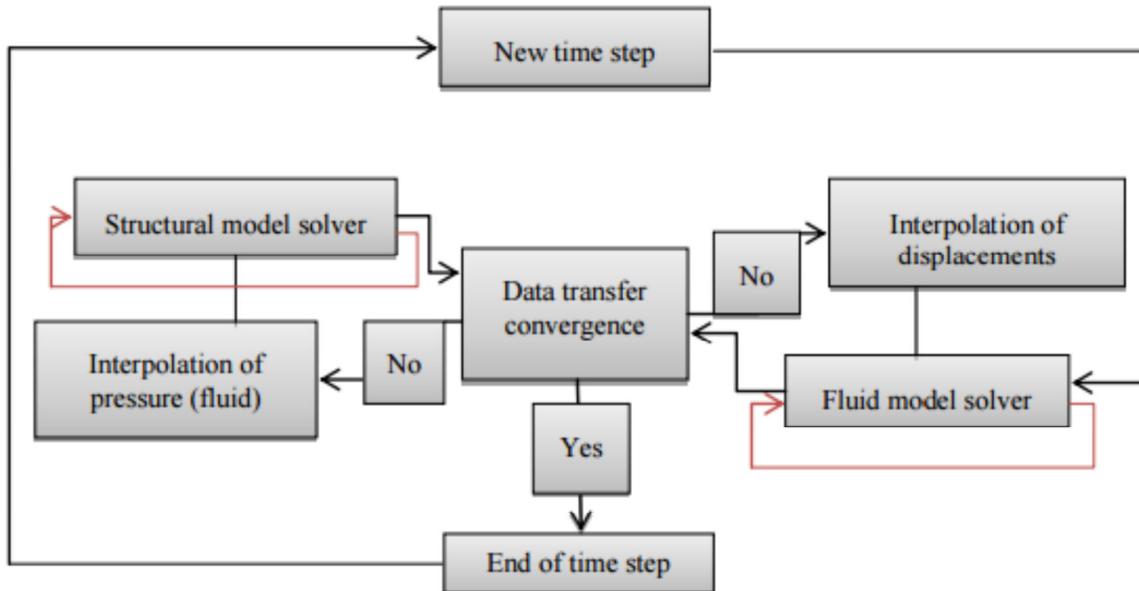


Fig. 2 - Two way system coupling process

RESULTS AND DISCUSSION

Fig. 3 shows velocity fields obtained at systolic peak for the two studied models. Both simulations present high velocities within stenosis and low velocity values in the outer wall region of the carotid bulb due to the enlargement of the vessel. This study shows that FSI analysis enables a reliable 3D blood-artery-plaque simulation that mimics a realistic deformable vessel. As the flow begins to decelerate after the peak flow, a larger recirculation zone develops at the non-divider wall of internal carotid artery in FSI model due to the elastic energy stored in the expanding compliant wall.

Fig. 4 shows wall shear stress distributions for the two considered studies. In both simulations maximum WSS values occur in the bifurcation apex due to a natural narrowing of the vessel. Low WSS patches in CCA are contiguous with the carotid bulb low WSS region prone to atherosclerosis.

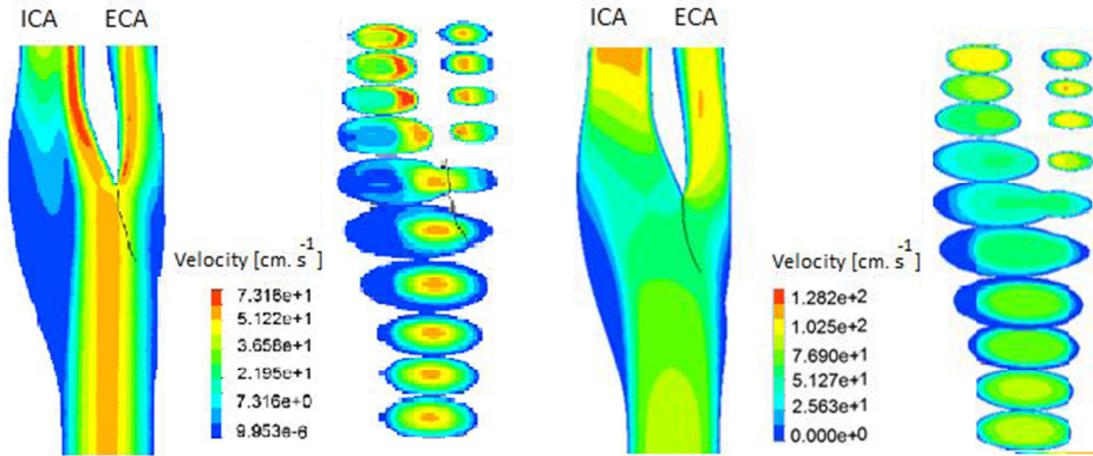


Fig. 3 - Velocity fields: FSI simulation (left) and Non-FSI model

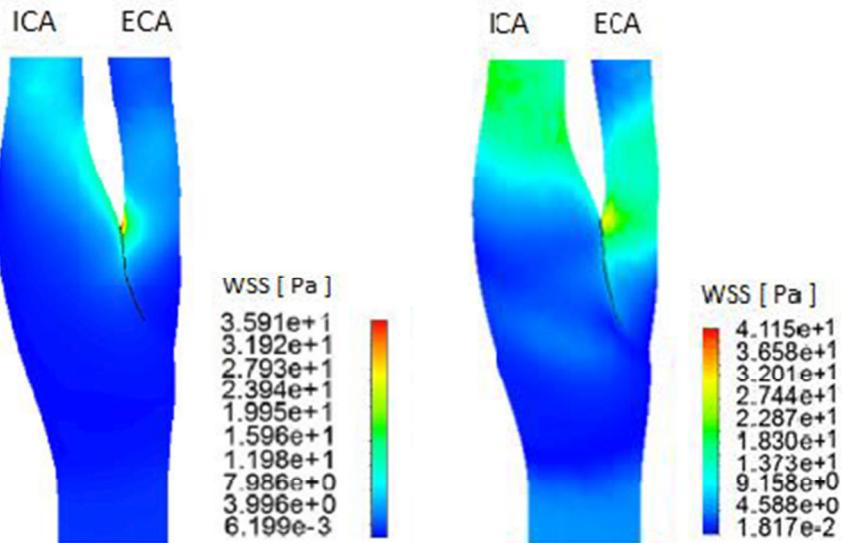


Fig. 4 - Wall shear stress fields: FSI simulation (left) and Non-FSI model

As expected, considering the FSI simulation, the maximum WSS value equal to 35 Pa is lower than the obtained for the non FSI simulation (41 Pa), where deformation of the arteries was not considered.

CONCLUSIONS

In this work a FSI analysis was performed in order to investigate the influence of artery compliance on the CCA bifurcation hemodynamics and wall shear stress distribution. This model has some limitations: it does not account for residual arterial stresses, patient-specific material properties and as available imaging data could not provide information on material anisotropy, it was not considered. Nevertheless, as expected, velocity and WSS distributions are similar and FSI model presents larger recirculation area and lower maximum WSS value.

We can conclude that compliance does not affect local hemodynamic in the proximal region of the studied non-stenosed CCA bifurcation. Further studies, simulating blood flow in stenotic carotid arteries, should be performed in order to better understand the development and rupture of carotid plaques.

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