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A STUDY OF FLUID AND STRUCTURE INTERACTION IN A CAROTID BIFURCATION

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ABSTRACT

In this work a study of the deformation of a carotid bifurcation model subjected to blood pressure is presented. A realistic model of the carotid bifurcation wall consisted of two layers to represent the multiplayer structure (intima/media/adventitia) of a typical elastic artery was developed. The flow inside the carotid bifurcation and the blood pressure on the arterial wall were altered by an obstacle located in the large bulb and blocking 20% of the cross-section area. A three-dimensional finite element model of the carotid bifurcation was created. The relative motion of the layers upon bifurcation deformation was investigated in this study.

INTRODUCTION

Atherosclerosis is a major cause of morbidity and mortality in our society representing up to 50 % of all deaths in the western world.[1] The risk factors for the development of atherosclerosis are well recognised[2] as is the inflammatory process within the arterial wall.[3,4,5,6] However, the initiating process and ongoing stimulus which perpetuates the repair process and leads to the formation of the atherosclerotic plaques still requires investigation. Better understanding of the precursors is the key to improving prophylactic and management options.

There are various hypotheses put forward to explain the development of atherosclerosis such as the organised thrombus hypothesis, the monoclonal cell growth hypothesis, the infection-inflammation hypothesis and the response to injury hypothesis.[3] The cause of atherosclerosis is most likely multifactorial with contributions from genetics the environment and local factors at the arterial wall interface including haemodynamics.

It is thought that haemodynamics contribute significantly to atherosclerosis as the major areas of plaque formation are at vessel branches. At these branch points there is prolonged turbulent flow[1] and low wall shear stress.[7,8] These factors allow for prolonged exposure to the damaging effects of turbulence and of atherogenic agents to the endothelium such as lipoproteins which may, when trapped and incorporated into the vessel wall, contribute to the formation of atheromatous plaques.[9]

The underlying cause for instability and acute changes to plaques is likely to be part of a cycle or an ongoing mechanism from plaque formation to plaque disintegration and clinical events. This study is based upon the hypothesis that the known susceptible sites are subject to intramural shear stresses that lead to wall injury, akin to fatigue fractures, that cause bleeding from the penetrating vasa vasorum leading from the adventitial layer to the intima. The shear stress causes micro-hemorrhages in the sub-intimal planes and it is the healing of these that leads to lesions subject to repeated insult and growth. The integrity and inherent health of the wall of the intima involving all of the above theories of atherogenesis, then contributes to acceleration of the lesions or rate modification with therapy.

Some plaques are stable and asymptomatic whilst others are more likely to produce symptoms due to acute local changes such as plaque rupture ulceration or hemorrhage.[10] Fatigue fractures due to wall stress leading to intramural haematoma formation for plaque fracture play a role in clinical events. Progression of the atheroma at the plaque site represents an underlying cause for instability and acute change to plaque morphology. Shear stress points, for example the carotid bifurcation, the origin of vessels and the adductor canal, are recognised sites for the formation of occlusive atheroma related to clinical events and common sites for treatment (Figure 1).

This study looks at the effect of an obstacle which is initiated inside a carotid bifurcation on the flow and the biomechanical stress strain behaviour to determine its influence on atherosclerosis and plaque instability. The obstacle represents a stage of the process growth of This phenomenon is studied using finite element computational fluid dynamics (CFD) and stress/strain analysis techniques commonly used in engineering which has been adapted for the evaluation of biomechanical and haemodynamic stresses on the cardiovascular system.

The carotid bifurcation was selected as it is a common region for atherosclerosis with often serious consequences. It also represents a region of which we have intimate knowledge as it is frequently imaged and surgically explored.

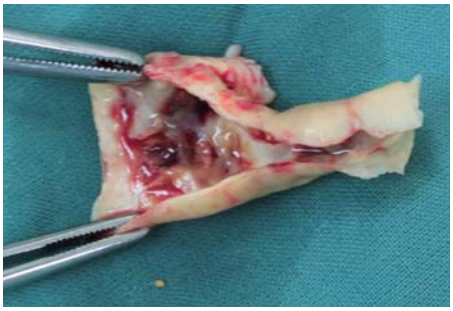

Figure 1: Subintimal haemorrhages at the carotid bifurcation - causal consequence of atheroma (Image courtesy of Professor John Harris, Royal Prince Alfred Hospital, Sydney, Australia).

MODEL DESCRIPTION

The analyses of the flow and the structure in this work were conducted with use of the so-called 'one-way coupling' fluid /structure interaction (FSI) model. Initially, a fluid-dynamics simulation was conducted to obtain the stress distribution on the carotid wall. The obtained fluid stresses was subsequently imported into and used in a structural analysis.

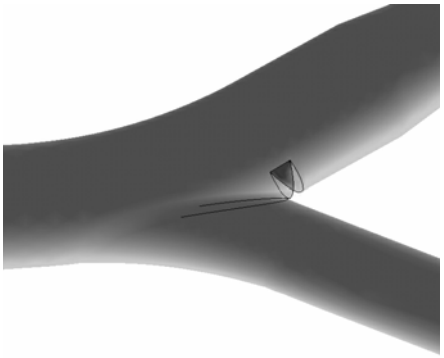

Figure 2: Carotid bifurcation model with an obstacle blocking the flow in the large bulb.

Typical arterial walls consist of three layers: the intima, media and adventitia, where the predominant connective tissue components are smooth muscle, collagen and elastin. The influence of smooth muscle in determining the form of stress strain relationship is relatively small. At different levels of stress, the elastin and collagen fibers contribute differently to the total structural function of the artery wall, which leads to the so called "biphasic" artery structural behaviour. That is linear isotropic stress-strain dependency at low stress and highly non-linear and anisotropic (viscoelastic) behaviour at high stress (Figure 2). The mechanical behaviour of a typical abdominal aorta exhibits this " biphasic" nature.[13]

To qualitatively describe the blood vessel mechanical properties, the vessels can be modelled as purely elastic, pseudoelastic, or viscoelastic material. The purely elastic (linear isotropic) assumption is most commonly used to reduce the model complexity, but it can not describe non-linear mechanical behaviour of the artery wall. The pseudo-elastic material model is usually based on the use of hyperelastic strain energy functions, which are given in terms of either Green-Lagrangian strain components or strain invariants. Many experimental measurements (both *in-vivo* and simulated) have been conducted to determine the parameters of the strain energy correlations, which were found to change significantly through aging, disease and change in mechanical load.

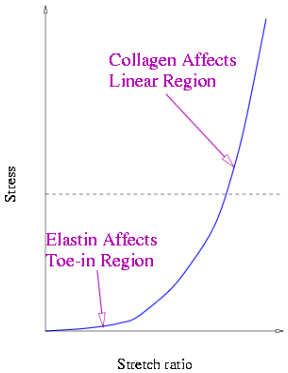

Figure 3: Typical structural behaviour of a blood vessel.

At very high stress/strain above the toe (low strain) and stiff (medium strain) regions, individual collagen fibers of the blood vessel begin to rupture and the vessel deformation becomes irreversible (plastic deformation). The stress/strain values at which the blood vessel rupture begins are referred to as ultimate stress/strain. Like the stress/strain relationship, the ultimate stress/strain vary depending on age, disease history, and mechanical load. The ultimate stress/strain also depends on the dynamics of the mechanical load.

Uniaxial tension tests by Mohan *et al.* (1982) (performed on human thoracic aorta) gave an approximate value of tensile failure strength in the range from 1.47 MPa to 5.07 MPa. In the work by Monson (2001), ultimate stretch (ratio) of 1.35:1.5 and failure stress of 1.06 to 3.4 MPa were reported for cerebral blood vessels).

The finite element model of the carotid bifurcation has been built based on the measurement data reported by Delfino et al., 1997:

- The diameter of the common carotid is 8.92 mm;
- One branch of the carotid bifurcation has a large bulb with inner diameter of 9.24 mm. To the outlet of this branch, the artery diameter gradually decreases to 7.08 mm;
- In the smaller branch, the carotid diameter slightly decreases from 6.46 mm to 6.38 mm;
- The angle between the axes of the internal and external carotids is 50^{o};
- The total arterial wall thickness is 0.7 mm throughout the carotid bifurcation;
- The carotids are "thin-walled" and no *residual* stress in the carotid wall is present in unloaded state.

The modelling and numerical analyses have been carried out using the commercial finite-element ANSYS 6.0 code. The fluid-dynamics simulation was carried out using 53,705 FLUID142 elements. The carotid bifurcation model consists of two layers: the inner one to represent the combination of tunica intima and media, and the outer one to represent tunica adventitia. The inner layer of the carotid bifurcation wall which includes the obstacle was built with 6720 elastic SHELL63 elements. The outer layer was represented by 6652 SHELL63 elements. SHELL63 is a 4-node finite strain element with six degrees of freedom at each node - translation in the x,y,z directions and rotation about the x,y,z axes. This type of element has both bending and membrane capabilities and is able to deal with large (geometrically non-linear) displacement. The contact between the two layers was modeled using 13,304 ANSYS contact element pairs. In total, 26,676 finite elements were used to model the carotid bifurcation wall and obstacle.

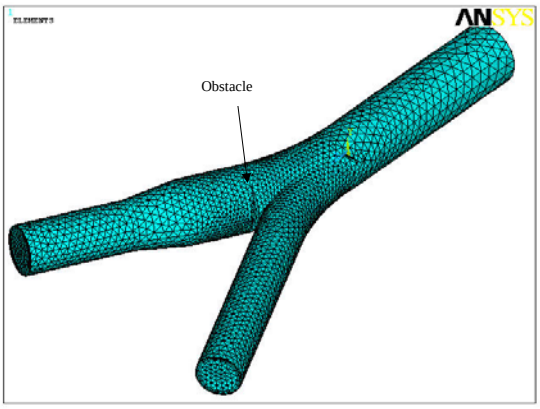

Figure 4: Computational mesh used in the CFD study.

Appropriate longitudinal displacement constrains are also applied at the ends of the carotid bifurcation section. The pressure load was initially equal to 1% of the full load and was gradually ramped up to 100%.

RESULTS

CFD simulation

The average blood velocity in the common carotid was assumed to be 1 m/s. Constant systolic pressure of 24 kPa (180 mmHg) was set at the branch outlets. The blood density and viscosity were specified as 1060 kg/m^3 and 0.0036 Pa.s, respectively. The flow Reynolds number was about 2650.

The computational results are shown in Figures 5-7. The obstacle is seen to have a significant effect on the flow and the distribution of wall pressure. In figure 5, significant pressure build-up is observed in the flow before the obstacle. Lower pressure is seen in the flow behind the obstacle which can be explained by the formation of an area with slow recirculation. The computational results indicate that the wall shear stress is small compared with the wall pressure. Therefore, only fluid pressure was used in the following structural analysis.

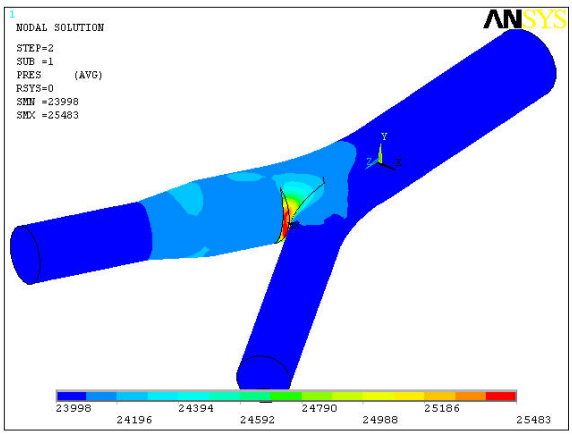

Figure 5: Distribution of the blood pressure on the carotid bifurcation wall.

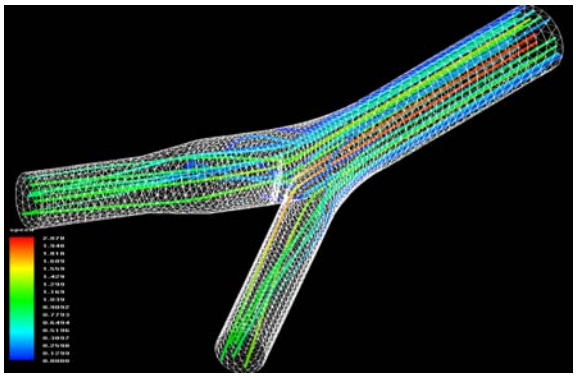

Figure 6: Flow streamlines

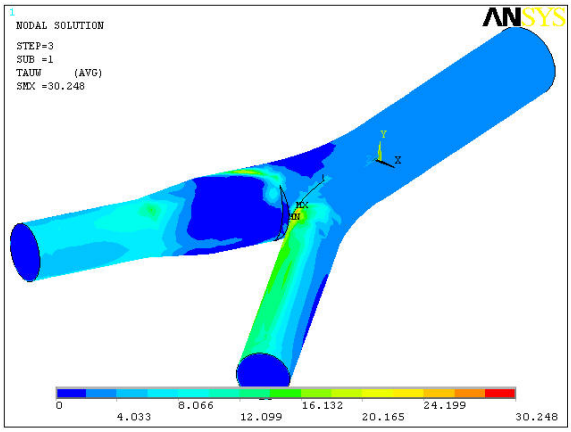

Figure 7: Distribution of the wall shear stress.

Structural analysis

In the realistic elastic arteries (aorta, carotids), the thicknesses of the media and adventitia layers are comparable. In this analysis, the thicknessses of both inner and outer layers are assumed to be equal to 0.35mm which is a half of the total thickness (0.7 mm). In this work, media and adventitia were modeled as isotropic, linear elastic materials. The structural properties reported in the work by Finet et al., 2002, and Ohayon et al., 2005, were used and listed in Table 1. In this analysis, the carotid bifurcation is modeled using thin (and plane) shell elements and, hence, the effect of cross-plane deformation is ignored. The stiffness of the obstacle is set to a relatively high value so that its deformation is small.

	Young's modulus, kPa	Poisson's ratio
Adventitia	850	0.27
Media	150	0.27
Estimated equivalent single layer properties	500	0.27

Table 1: Structural properties used in the analysis (Finet et al., 2002).

Two simulations were conducted: one with different (case 1) and the other with equivalent single-layer (case 2) structural properties of the wall. Appropriate displacement constrains were applied at the ends of the carotid bifurcation section to prevent its movement. The pressure load was initially set equal to 1% of the full load and gradually ramped up to 100%. Careful control of ramping step was required to obtain convergence. To accurately predict the deformation of the thin shells the option of "Large Displacement solution" was activated in all simulations.

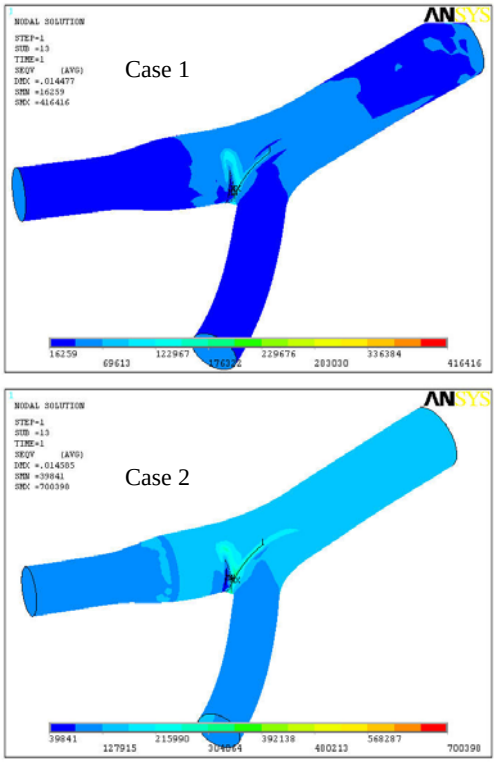

Figure 8: Von-Mises stress[1] in the inner layer.

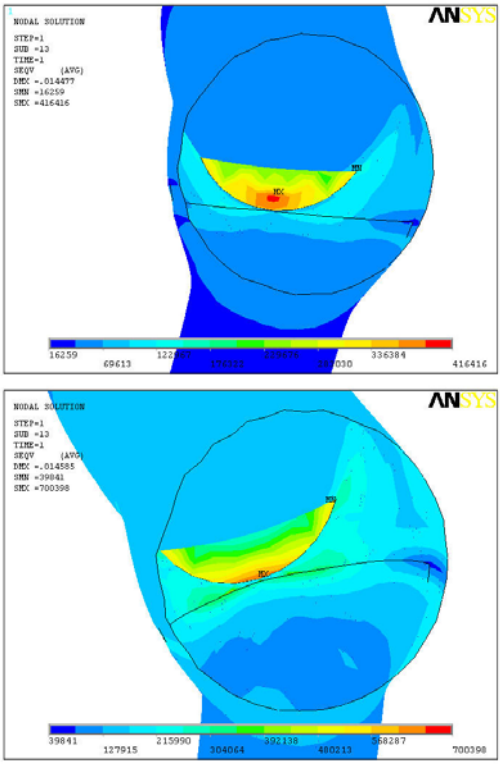

Figure 10. Von-Mises stress in the obstacle.

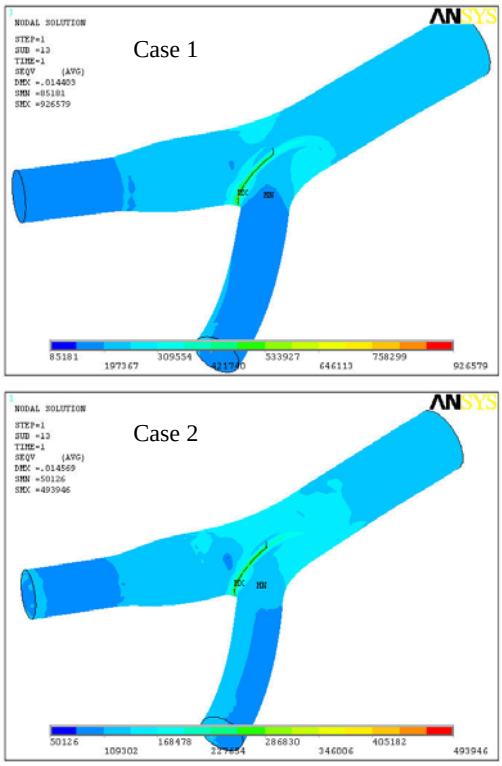

Figure 9. Von-Mises stress in the outer layer**.**

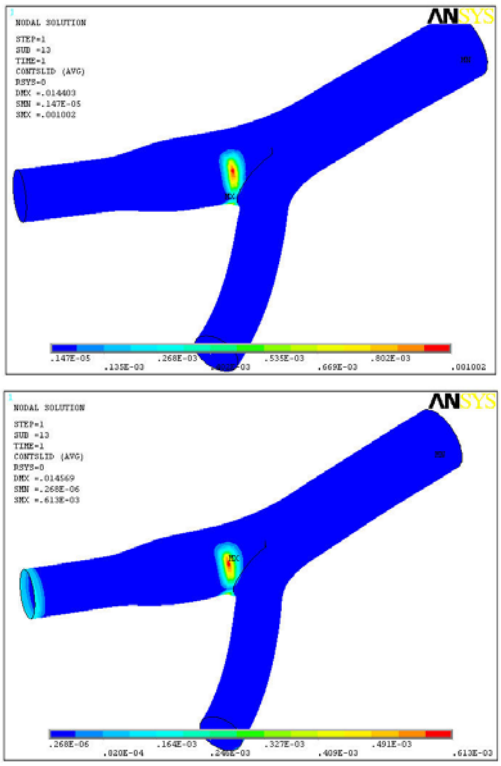

Figure 11. Sliding distance**.**

The structural analysis results shown in Figures 7-11 can be summarized as follows:

- The results show that the stress distributions in the layers are different and depend on the layer structural properties. In case 2 where the layers have similar stiffness, deformation of the outer layer is small compared to the inner layer and, hence, the stress in

[1] The von-Mises equivalent stress, σ_e is defined from the three principal stresses σ_1, σ_2, and σ_3, as follows:

$$\sigma_e = \frac{1}{\sqrt{2}}\left[(\sigma_1-\sigma_2)^2+(\sigma_2-\sigma_3)^2+(\sigma_3-\sigma_1)^2\right]^{1/2}$$

the outer layer is lower than that in the inner layer. In case 1, the outer layer is much stiffer than the inner one and the stress in the outer layer is seen to be higher than that in the inner layer.

- The presence of an obstacle in the large branch of the carotid bifurcation significantly alters the flow in the surrounding area. The obstacle is seen to be subjected to a force resulted from the difference in the flow pressure before and after the obstacle. This force which is much greater than the normal shear stress is seen to transfer to the inner layer wall and causes much higher contact stress and larger sliding between the layers compared to the case without an obstacle. The contact stress and sliding distance are bigger in case 1 when the stiffness of the inner layer is an order of magnitude smaller than that of the outer layer.

CONCLUSION

In this work a combined study of the flow inside and structural behaviour of a carotid bifurcation with an obstacle initiated in its large branch was conducted. This combined CFD and a finite element stress/strain study was carryout out with use of a realistically-scaled three-dimensional model of carotid bifurcations and 'one-way' coupling of the fluid and structure analyses. A two layer arterial wall model was devised to represent the interface between the adventitia and media and then exposed to finite element stress/strain analysis.

This model revealed significant pressure difference in the flow before and after the obstacle which in turn subjected the obstacle to a force much larger than the normal shear stress. This force is seen to cause increased stress in and between the arterial wall layers. In addition, the presence of an obstacle which narrowed the artery could also elevate the flow speed and the wall shear stress in the vicinity of the obstacle. Therefore, an obstacle which is initiated by a damage in the blood vessel can cause increases of the mechanical load on the arterial wall around the obstacle which may lead to further damage of the wall (and consequent further growth of the obstacle) both by its direct effect on wall structure and also by producing shearing forces between vessel wall layers. The stress/strain may also exceed the vessel threshold leading to vessel fracture, which may result in endothelial disruption or plaque rupture. Alternatively or in addition, shearing between adventitia and media may tear the *vasa vasorum*[2] producing haemorrhage into a plaque which may produce embolisation and/or local thrombosis all of which may lead to an acute event.

Success of this model allowed us to expand its application to try to more accurately represent the *in vivo* arterial bifurcation.

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