Fluid-Structure Analysis of an Atherosclerotic Coronary Artery

A. Gholipour¹, M. H. Ghayesh¹, A. Zander¹, S. J. Nicholls^{2, 3}, and P. J. Psaltis^{2, 3}

¹School of Mechanical Engineering

University of Adelaide, Adelaide, South Australia 5005, Australia

²Vascular Research Centre, Heart Health Theme,

South Australian Health & Medical Research Institute (SAHMRI), Adelaide, South Australia 5000, Australia

³Department of Medicine, University of Adelaide, Adelaide, South Australia 5005, Australia

Abstract

A theoretical fluid-structure interaction (FSI) model for the stress field of atherosclerotic coronary arteries are obtained and the influence of various characteristics on the stress distribution in diseased coronary arteries is highlighted. A reliable model is developed (and hence accurate heart attack prediction), the following factors are incorporated: (1) non-Newtonian blood flow; (2) artery's tapered shape; (3) the micro-calcification of the plaque; (4) blood pulsation. Incorporating these factors in the model makes it possible to accurately predict plaque ruptures. The system is modelled based on a 3D fluid-structure interaction analysis via the finite element method (FEM). Experimental data from previous studies are used to generate a realistic material model. The generated model is utilised as a predictive model for plaque rupture and to determine high risk situations in the coronary arteries. It is shown that incorporating the physiological flow rate in the model, the wall shear stress (WSS) (stresses impose to the plaque from blood) and von Mises stresses (stresses in the plaque) are predicted accurately. Also it is shown that microcalcification increases the von Mises stress substantially in the plaque, when the WSS remains the same. Considering tapered shape of the artery is also shown to be important for predicting correct values of both shear and von Mises stresses.

Introduction

Atherosclerosis is one of the main causes of death worldwide [1]. In Australia, the same figure is reported by approximately 19,000 deaths due to coronary arteries diseases [2]. The aforementioned leading diseases reflect situations in which the blood supply for the heart or brain is disturbed in a way. Among the reasons affecting the blood supply, atherosclerosis is the most common one for obstructing the fresh and oxygenated-blood. In the progress of this disease a plaque is being shaped mainly because of LDL deposition. Figure 1 shows an angiography imaging of a diseased left coronary artery. The plaque shaped at the indicated location. The high risk situation is happened when the plaque ruptures. In order to prevent this disease, a biomechanical model is required to be developed to predict the high risk situation or plaque rupture.

The literature on modelling atherosclerotic coronary arteries can be divided into three groups namely structure, fluid, and fluidstructure analysis.

Structural analysis

In the structural analysis, studies have been conducted based on histology, IVUS, MRI, hrMRI, and OCT of the arteries; real shape of arteries were used for theoretical modelling and other features in arteries were investigated; these features are acoustic analysis and prediction of strains [3], sensitivity to material properties [4] via isotropic non-linear and anisotropic linear, cracks and fracture analysis based on MRI in-vitro [5], initial stress in the arteries [6], viscoelastic and hyperelastic analysis [7], and non-linear isotropic and piecewise homogeneous material [8]. The gap of modelling WSS on the wall of plaque is still obvious in this class of investigations.

Fluid analysis

Different imaging methods have been used to examine the fluidic behaviour of arteries and plaques. Most of the examinations chose Newtonian and incompressible fluid for blood modelling. Unsteady and pulsatile flow [9], 3D shear stress distributions [10], turbulence models [11], and hemodynamics [12] are the characteristics of blood and flow which have been examined. Moreover, theoretical models of fluid micro-hemodynamic and flow reversal of an artificial artery vessel were developed [13]. Fluid analysis does not provide stress field analysis in the solid regime of the system which seems important in atherosclerosis.

Fluid-structure analysis

Prominent investigations have been conducted considering a hyperelastic solid and Newtonian fluid models that consider different characteristics such as superficial arterial vessel [14], hydrogel model [15], MRI-based imaging [16], IVUS-based modelling [17], and histology-based modelling [18]. The contribution of the present investigation is to incorporate various features of the problem such as physiological blood flow, artery's tapered shape, non-Newtonian flow, and micro-calcification in the asymmetric 3D fluid-structure analysis of atherosclerotic coronary artery.



Figure 1. Angiography of diseased left coronary artery [19].

Method of Investigation

The FSI models of atherosclerotic coronary artery has been constructed via FEM in ANSYS. It is shown that all of these factors (blood physiological pulsation, tapered artery, non-Newtonian blood flow, micro-calcification inside the plaque) can alter the predicted stress field, which in many cases, ignoring them results in drastic errors and hence an incorrect prediction of the plaque rupture (and hence the heart attack). For all the numerical results presented in the following sections the mesh convergence has been obtained using finer elements.

Geometry and Material Properties

The schematic of diseased artery is shown in Fig. 2. This shape is adopted form Ref. [20], and modified according to experimental data of Ref. [21]. The artery is assumed to be fixed at the inlet side. The material properties for artery's several layers i.e. media, adventitia, intima, and lipid are applied to the model based on hyperelastic [21] and viscoelastic [22] model of Mooney-Rivlin and Prony Shear Relaxation using ANSYS [23, 24].

In the fluid model, the non-Newtonian, incompressible, turbulent blood flow with the physiological pulsation [25] is considered. The non-Newtonian model of Carreau constitutive law is also adopted and used from Ref. [20].

(a)



(b)



Figure 2. Schematic of atherosclerotic left main coronary artery (a) 3D asymmetric shape; (b) different parts of problem: intima, media, adventitia, plaque, lipid core, and blood.

Results and Discussion

The plaque rupture could happen due to damaged endothelial cells due to WSS from blood (fluid part) and material failure of the intima layer on the plaque (solid part); determining both shear and von Mises stress field is essential.

Illustrated in Fig. 3 is the time-dependent variation of WSS at its maximum. The WSS has the peak of 53.3 Pa at 0.42 s during 0.8 s of the heartbeat (sub-figure (a)). In panel (b), WSS distribution is depicted throughout the artery. The cap of the plaque is the most vulnerable region from shear-stress prospective.

As an index to analyse the stress field in the artery and plaque, von Mises stress is calculated in the model. As shown in Fig. 4, von Mises stress trend with time is the same as the WSS and has the peak of 749.8 Pa at 0.42 s (sub-figure (a)). Sub-figure (b) shows the von Mises stress distribution is high at the plaque shoulder towards up-stream of the flow. Due to complexity and nonlinearity of the problem, there are some local maximum values in other regions of the artery.











Figure 4. (a) von Mises stress change in time; (b) von Mises stress at t=0.42 s.

Pulsatile Flow

In order to compare the effect of pulsation, a comparison of the physiological and simplified sine wave velocity pulsation of the blood is considered in this section. In Fig. 5 (sub-figure (a)), a comparison between physiological and sine wave related WSS shows that even if the sine wave is composed from the physiological data it still cannot predict neither of minimum nor maximum values of the real WSS. As a result, in order to determine the right values of WSS, the physiological pulsation is important to be considered. The same trend for the von Mises stress occurs in the region where the von Mises stress is high.

Micro-calcification effect

In most recent studies from the literature, it was shown that microcalclification results in stress concentration and hence plaque rupture [26]. Figure 6 illustrates this effect; the system characteristics are the same as those of the previous sections. Modelling micro-calcification created a stress concentration which increases von Mises stress significantly to 3780.6 Pa (sub-figure (b)); however, the WSS remains almost the same (sub-figure (a)).



Figure 5. (a) WSS based on two models: sin wave velocity pulsation and physiological velocity pulsation; (b) von Mises stress based on two models: sin wave velocity pulsation and physiological velocity pulsation.







Figure 6. (a) WSS using two models: with and without microcalcification in plaque; (b) von Mises stress using two models: with and without micro-calcification in plaque.

Inclusion of Tapered Shape

Another important factor is tapered shapes of arteries; according to Ref. [27]. As seen in Fig. 7, considering artery's tapered shape changes the stress distribution (and hence the initiation of the plaque rupture) by increasing both the shear or von Mises stress fields.



Figure 7. (a) WSS using two models: straight and tapered artery; (b) von Mises stress using two models: straight and tapered artery.

Conclusions

The WSS and von Mises stress fields have been determined for an atherosclerotic coronary artery via developing a finite element biomechanical model using ANSYS. The 3D biomechanics of the system was analysed incorporating the blood pulsation, artery's tapered shape, non-Newtonian flow theory, and microcalcification; all of these factors are vital in the model development of both normal and atherosclerosis coronary arteries. The following conclusions can be drawn from the investigation: (i) maximum WSS occurs at the plaque's cap when the pulsation velocity is maximum; (ii) maximum of the von Mises stress occurs at the shoulder of the plaque when the blood velocity is the highest; (iii) the exact time trace of blood pulsation to be employed in the model, as small deviation from that ends up in inaccurate results; (iv) stress concentration due to micro-calcification increases the von Mises stress significantly; (v) The overall values (for both shear and von Mises stresses) become larger when the tapered shape of the artery is incorporated.

References

- W. H. O. WHO, "The top 10 causes of death," pp. <u>http://www.who.int/mediacentre/factsheets/fs310/en/</u>, 2017.
- [2] A. I. o. H. a. W. AIHW, "Leading causes of death," <u>https://www.aihw.gov.au/reports/life-expectancy-death/deaths-in-australia/contents/leading-causes-of-death</u>, 2017.
- [3] A. I. Veress, J. A. Weiss, G. T. Gullberg, D. G. Vince, and R. D. Rabbitt, "Strain measurement in coronary arteries using intravascular ultrasound and deformable images," *Journal of Biomechanical Engineering*, vol. 124, no. 6, pp. 734-741, 2002.
- [4] S. Williamson, Y. Lam, H. Younis, H. Huang, S. Patel, M. Kaazempur-Mofrad, and R. Kamm, "On the sensitivity of wall stresses in diseased arteries to variable material properties," *Journal of biomechanical engineering*, vol. 125, no. 1, pp. 147-155, 2003.
- [5] A. Ferrara, and A. Pandolfi, "Numerical modelling of fracture in human arteries," *Computer methods in biomechanics and biomedical engineering*, vol. 11, no. 5, pp. 553-567, 2008.
- [6] L. Speelman, A. Akyildiz, B. Den Adel, J. Wentzel, A. Van der Steen, R. Virmani, L. Van der Weerd, J. W. Jukema, R. Poelmann, and E. van Brummelen, "Initial stress in biomechanical models of atherosclerotic plaques," *Journal of biomechanics*, vol. 44, no. 13, pp. 2376-2382, 2011.
- [7] V. M. Heiland, C. Forsell, J. Roy, U. Hedin, and T. C. Gasser, "Identification of carotid plaque tissue properties using an experimental–numerical approach," *Journal of the mechanical behavior of biomedical materials*, vol. 27, pp. 226-238, 2013.
- [8] J. Lu, W. Duan, and A. Qiao, "Finite element analysis of mechanics of neovessels with intraplaque hemorrhage in carotid atherosclerosis," *Biomedical engineering online*, vol. 14, no. 1, pp. 1, 2015.
- [9] Y. Fu, A. Qiao, and L. Jin, "The influence of hemodynamics on the ulceration plaques of carotid artery stenosis," *Journal* of Mechanics in Medicine and Biology, vol. 15, no. 01, pp. 1550008, 2015.
- [10] J. Brunette, R. Mongrain, J. Laurier, R. Galaz, and J. Tardif, "3D flow study in a mildly stenotic coronary artery phantom using a whole volume PIV method," *Medical engineering & physics*, vol. 30, no. 9, pp. 1193-1200, 2008.
- [11] F. Tan, G. Soloperto, S. Bashford, N. Wood, S. Thom, A. Hughes, and X. Xu, "Analysis of flow disturbance in a stenosed carotid artery bifurcation using two-equation transitional and turbulence models," *Journal of biomechanical engineering*, vol. 130, no. 6, pp. 061008, 2008.
- [12] K. Bhaganagar, and C. Moreno, "Modeling of Stenotic coronary artery and implications of plaque morphology on blood flow," *Modelling and Simulation in Engineering*, vol. 390313, pp. 1-14, 2013.
- [13] L. Ai, L. Zhang, W. Dai, C. Hu, K. K. Shung, and T. K. Hsiai, "Real-time assessment of flow reversal in an eccentric arterial stenotic model," *Journal of biomechanics*, vol. 43, no. 14, pp. 2678-2683, 2010.

- [14] D. Tang, C. Yang, S. Kobayashi, J. Zheng, and R. P. Vito, "Effect of stenosis asymmetry on blood flow and artery compression: a three-dimensional fluid-structure interaction model," *Annals of biomedical engineering*, vol. 31, no. 10, pp. 1182-1193, 2003.
- [15] D. Tang, C. Yang, S. Kobayashi, and D. N. Ku, "Effect of a lipid pool on stress/strain distributions in stenotic arteries: 3-D fluid-structure interactions (FSI) models," *Journal of biomechanical engineering*, vol. 126, no. 3, pp. 363-370, 2004.
- [16] X. Huang, C. Yang, J. Zheng, R. Bach, D. Muccigrosso, P. K. Woodard, and D. Tang, "Higher critical plaque wall stress in patients who died of coronary artery disease compared with those who died of other causes: a 3D FSI study based on ex vivo MRI of coronary plaques," *Journal of biomechanics*, vol. 47, no. 2, pp. 432-437, 2014.
- [17] M. H. Kural, M. Cai, D. Tang, T. Gwyther, J. Zheng, and K. L. Billiar, "Planar biaxial characterization of diseased human coronary and carotid arteries for computational modeling," *Journal of biomechanics*, vol. 45, no. 5, pp. 790-798, 2012.
- [18] H. Yamada, and N. Sakata, "Low pressure condition of a lipid core in an eccentrically developed carotid atheromatous plaque: a static finite element analysis," *Journal of biorheology*, vol. 27, no. 1-2, pp. 9-17, 2013.
- [19] J. A. Tremmel, T. Koizumi, A. O'Loughlin, and A. C. Yeung, "Intramural hematoma appearing as a new lesion after coronary stenting," *JACC: Cardiovascular Interventions*, vol. 4, no. 1, pp. 129-130, 2011.
- [20] M. Cilla, I. Borrás, E. Pena, M. Martínez, and M. Malve, "A parametric model for analysing atherosclerotic arteries: On the FSI coupling," *International Communications in Heat and Mass Transfer*, vol. 67, pp. 29-38, 2015.
- [21] G. A. Holzapfel, G. Sommer, C. T. Gasser, and P. Regitnig, "Determination of layer-specific mechanical properties of human coronary arteries with nonatherosclerotic intimal thickening and related constitutive modeling," *American Journal of Physiology-Heart and Circulatory Physiology*, vol. 289, no. 5, pp. H2048-H2058, 2005.
- [22] A. Karimi, A. Shojaei, and R. Razaghi, "Viscoelastic mechanical measurement of the healthy and atherosclerotic human coronary arteries using DIC technique," *Artery Research*, vol. 18, pp. 14-21, 2017.
- [23] "ANSYS® Academic Research Mechanical, Release 18.0, Help System, ANSYS Documentation/ Mechanical Applications/ Explicit Dynamics Analysis Guide Overview/ Material Models Used in Explicit Dynamics Analysis/ Hyperelasticity/ 5-Parameter Mooney-Rivlin Model, ANSYS, Inc.."
- [24] A. Gholipour, M. H. Ghayesh, A. Zander, and R. Mahajan, "Three-dimensional biomechanics of coronary arteries," *International Journal of Engineering Science*, vol. 130, pp. 93-114, 2018.
- [25] F. Chabi, S. Champmartin, C. Sarraf, and R. Noguera, "Critical evaluation of three hemodynamic models for the numerical simulation of intra-stent flows," *Journal of biomechanics*, vol. 48, no. 10, pp. 1769-1776, 2015.
- [26] N. Maldonado, A. Kelly-Arnold, Y. Vengrenyuk, D. Laudier, J. T. Fallon, R. Virmani, L. Cardoso, and S. Weinbaum, "A mechanistic analysis of the role of microcalcifications in atherosclerotic plaque stability: potential implications for plaque rupture," *American Journal* of Physiology-Heart and Circulatory Physiology, vol. 303, no. 5, pp. H619-H628, 2012.
- [27] S. Chakravarty, and P. K. Mandal, "Two-dimensional blood flow through tapered arteries under stenotic conditions," *International Journal of Non-Linear Mechanics*, vol. 35, no. 5, pp. 779-793, 2000.