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# Blood Flow Simulation in an Atherosclerotic Coronary Artery

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*Abstract-* *Introduction:* Atherosclerosis is highly linked to heart attacks and strokes which are two leading cause of death in the United States according to the Center of Disease Control and Prevention (CDC). The increasing severity of atherosclerosis comes from the instance that symptoms usually appear at late stages of the disease. In addition, the mechanisms by which atherosclerosis forms, develops, and triggers the onset of myocardial infarction is not fully understood. In this study, we simulated the effect that atheromas have on the blood flow physics and the correlation that these flow disturbances may have on the onset of myocardial infarction.

*Materials and Methods:* A 3D virtual geometry of an idealized coronary artery with a hemispherical obstruction was created using human anatomical dimensions. All Ansys simulations performed in this study used laminar flow conditions with density = 1060 kg/m3, viscosity = 3.5 centipoise. We applied a physiological velocity waveform at the inlet and a zero relative-pressure condition at the outlet. No slip boundary conditions were prescribed to the coronary artery walls.

*Keywords:* *atherosclerosis; coronary artery disease; vascular erosion; computational fluid dynamics; heart attack; stroke.*

*GJMR-I Classification:* *NLMC Code: WG 595*


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Joshua Bennett [α] & Manuel Salinas [σ]

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*Results and Discussion:* We observed that the obstruction in the blood flow caused severe flow disturbance downstream from the atheroma. These time dependent cyclical flow profiles cause oscillatory velocities, pressures and shear stresses. These flow alterations have been linked to vessel erosion and may be a key factor on the onset of heart attacks.

*Conclusions:* In this study, we coupled an anatomically relevant time dependent velocity waveform with a segment of a coronary artery blocked by an atheroma. We have demonstrated that coronary arteries afflicted with atherosclerosis causes recirculation areas immediately downstream from the occlusion which are areas linked to vessel erosion and thrombus formation. Our focus in our future work will be to incorporate vessel elasticity and movement. In addition, we hope to be able to correlate our results with tissue culture and small animal studies.

*Keywords: atherosclerosis; coronary artery disease; vascular erosion; computational fluid dynamics; heart attack; stroke.*

## I. Introduction

Atherosclerosis is highly linked to heart attacks and strokes which are two leading cause of death and morbidity worldwide[1-17]. In the United States alone, 8.917 million people worldwide died in 2015 due to atherosclerosis complications. The annual burden on the United States Healthcare system is estimated to be $351.2 billion in 2014-2015, with $213.8 billion in direct costs[18]. There is no cure for atherosclerosis and the underlying mechanisms that cause it have not been fully and exhaustibly delineated[1,4,15,19-21].

There are numerous studies dedicated to understanding how atherosclerosis develops. Some studies state that atherosclerosis is correlated to the infiltration of the artery by lipids and proteins[19]. Efforts elsewhere state that viral infections may play a role in the activation of atherosclerosis[11,22]. Other experiments suggest that blood flow disturbance aids in the onset and progress of the disease[10,21,23].

Choi et al.[24] screened 217 patients who each had a chronic coronary total occlusion (CTO) due to atheromas. They reported varying degrees of myocardial scarring in most myocardial tissue downstream from the atheroma. In another study by Franck et al.[21], a cuff was used to constrict a rat's aorta. They also reported increased number of inflammatory cells and vessel erosion at zones of oscillatory shear stress.

Despite these and many other studies, the underlying mechanisms by which atheromas contribute to the onset of heart attacks is not well understood and pose a major challenge to the cardiovascular scientific community[21]. In this study, we investigated the effect that atheromas have on the flow physics of blood and the possible correlation that these flow disturbances may have on the onset of myocardial infarction.

## II. Mesh Construction, Simulation Set-up

A 3D virtual geometry of an idealized coronary artery with hemispherical atheroma was created using Ansys Design modeler as depicted in Fig. 1 (Ansys Inc., Canonsburg, The vessel diameter equaled 3.1 mm with a length of 30 mm and a hemispherical obstruction of radius 1.55 mm centered 10 mm from the inlet. A standard mesh with 77,220 elements was constructed. Computational fluid dynamics (CFD) simulations were conducted in CFX (Ansys Inc., Canonsburg, PA). All simulations performed used constants and methods previously validated in other studies[25-28]. Briefly, we prescribed laminar flow conditions with density = 1060 kg/m$^3$ and viscosity = 3.5 centipoise. A zero relative-pressure condition was also applied at the outlet. At the

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inlet, we applied a time dependent physiologically relevant velocity waveform[29] shown in Fig. 2. No slip boundary condition was applied to the vessel wall. Simulations were conducted using a Lenovo Desktop workstation with Intel® Core™ i7-7500U CPU, 2.70 GHz (1 processor) with 16 Gb installed memory and 64-bit Windows 10 operating system.

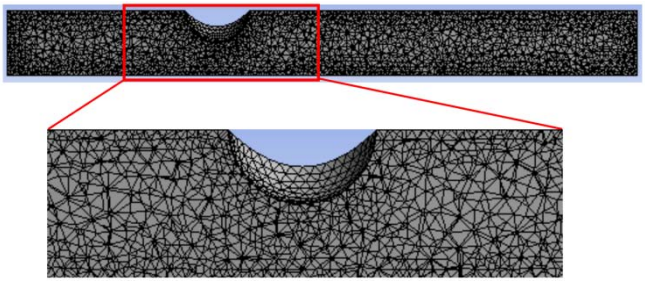

*Fig. 1:* Cross-sectional look of the virtual geometry and mesh of the arterial wall and occlusion. The complete mesh consists of 15976 nodes and 77220 elements.

## III. Results and Discussion

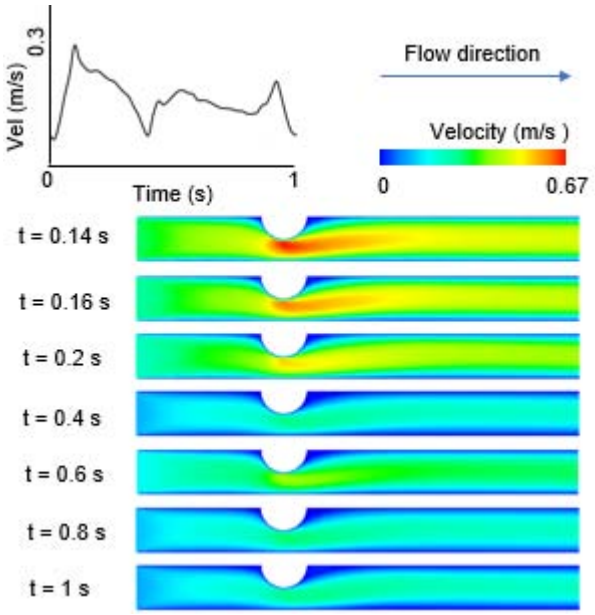


*Fig. 2:* Contour of velocity magnitudes at specified time intervals along a longitudinal central cross-sectional plane. Notice the higher velocities at the apex of the occlusion, and lower velocities near the vessel wall.

The velocity magnitudes of blood flow were plotted on a longitudinal cross-sectional plane down the geometry's center as seen in Fig. 2. In the obstructed and unobstructed regions of the coronary artery, velocity of blood decreased near the vessel walls. Also, fluid velocity increased at the apex of the occlusion. The maximum velocity occurred at 0.16 seconds.

A longitudinal cross-sectional plane at the same location was used to measure flow pressure as seen in Fig.3. Highest blood pressure surrounding the obstruction was reported when velocity was at its maximum corresponding to a time of 0.14 seconds. On the other hand, the lowest pressure corresponded to times of 0.16 seconds in the regions next to the occlusion.

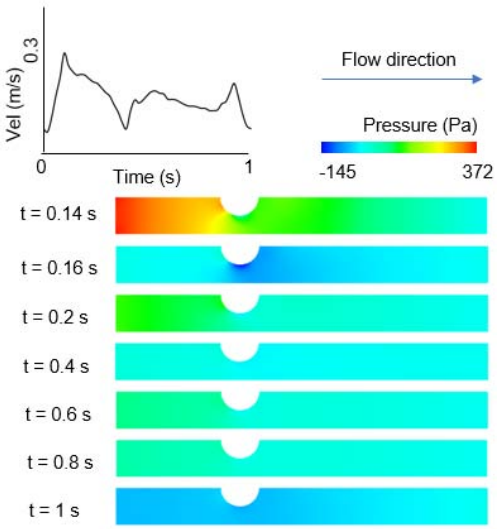


*Fig. 3:* Contour of pressure at specified time intervals on cross-sectional longitudinal plane. Simulation shows low to medium pressure throughout most timesteps. Timestep t = 0.14 s showed highest pressure throughout the system. Timestep t = 0.16 s showed lowest pressure surrounding the obstruction.

Axial velocity vectors were plotted on the same plane as velocity as seen in Fig. 4. Recirculation was observed downstream from the occlusion particularly at 1 second. This results agree with findings in other studies that suggest atheromas cause abnormal blood flow[10,21,30]. These findings also agree with larger scale animal models developed by Frank et al.[10] where they reported an increase flow disturbance activity downstream from an artificial occlusion in a rat artery and reported increased number of inflammatory cells and vessel erosion.



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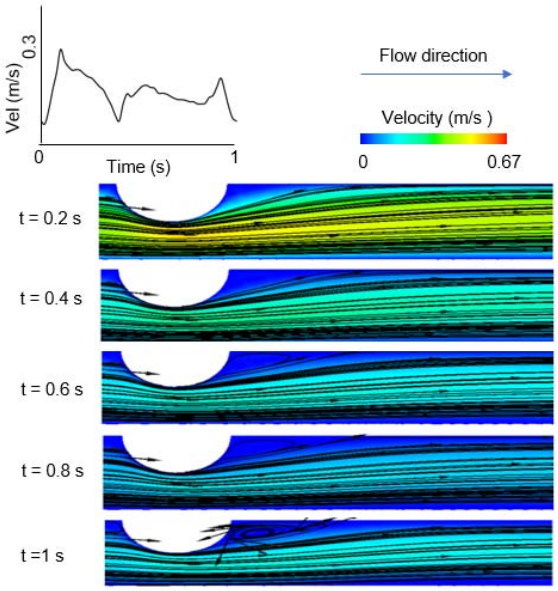


*Fig. 4:* Contour of velocity magnitudes and streamlines at specified time intervals on longitudinal plane focused on the immediate surrounding of the hemispherical obstruction. Recirculation occurs at 1 second into the cycle.

## IV. Conclusion

One of the principle achievements of this paper is the coupling of an anatomically relevant time dependent velocity waveform with a segment of a coronary artery blocked by an atheroma. We have demonstrated that coronary artery afflicted with atherosclerosis causes recirculation areas immediately downstream from the occlusion. Our focus in our future work will be to incorporate vessel elasticity and movement. In addition, we hope to be able to correlate our results with tissue culture and small animal studies.

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